Differences in pelvic floor muscle activation and functional output between women with and without stress urinary incontinence

by
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Abstract

**Introduction:** The primary purpose of this research was to determine whether women with stress urinary incontinence (SUI) demonstrate pelvic floor muscle (PFM) strength or endurance deficits and/or changes in the motor control patterns used during maximum voluntary PFM contractions (PFM MVCs) and coughing. A secondary purpose was to determine the effect of age on these parameters.

**Methods:** After first validating the use of vaginal pressure to study the functional output of the PFMs (Chapter 3), three studies were carried out to address these objectives. In two studies vaginal pressure and PFM and abdominal muscle electromyography (EMG) data were recorded simultaneously during PFM MVCs (Chapter 4) and maximum effort coughs (Chapter 5) in continent women, women with mild SUI and women with moderate to severe SUI in both supine and standing. In the final study (Chapter 6), the effect of continence status and age on PFM strength and endurance was measured with vaginal pressure. **Results:** Changes in vaginal pressure induced by PFM MVCs and coughing were found to reflect changes in urethral pressure. In Chapters 4 and 5, the women with SUI and the continent women were found to be equally able to produce peak PFM EMG and vaginal pressure amplitudes during PFM MVCs and coughs. Compared to the continent women, the women with SUI delayed activating their abdominal muscles during the PFM MVCs. During coughing, vaginal pressure and PFM EMG peaked simultaneously in the continent women, while in the women with SUI vaginal pressure peaked after PFM EMG. During both the PFM MVCs and the coughs, the EMG activity in all of the
muscles tested was higher at the onset of vaginal pressure generation in the women with SUI compared to the continent women. In Chapter 6, no difference was found in PFM endurance between the women with and without SUI. The ability to generate peak vaginal pressure during coughing decreased with age. **Conclusions:** PFM weakness does not appear to play a significant role in SUI. Rather, the results of this research suggest that a combination of motor control deficits and delays in pressure transmission are associated with SUI in women.
Co-Authorship

Stéphanie J. Madill was the primary author of all chapters within this thesis. Co-authors of this manuscript include: Marie-Andrée Harvey and Linda McLean. More specifically, the authors of Chapters 3 and 6 are Stéphanie J. Madill and Linda McLean and the authors of Chapters 4 and 5 are Stéphanie J. Madill, Marie-Andrée Harvey and Linda McLean. In each of these chapters, Ms. Madill designed the study procedures with the assistance of Dr. McLean and contributions from Dr. Harvey on Chapters 4 and 5. Ms. Madill completed all of the experimental work. Ms. Madill wrote the initial drafts of all the manuscripts and she participated in all subsequent editing with Dr. McLean and Dr. Harvey.

Chapter 3 is still being prepared for submission to a journal. Chapter 4 has been published by the International Urogynecology Journal (2009;20:447-459). Chapter 5 has been accepted by the Journal of Electromyography and Kinesiology and Chapter 6 is in press in the journal Neurourology and Urodynamics.
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“It’s a long road from conception to completion.” Molière

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It’s all I have to bring to-day,
This, and my heart beside,
This, and my heart, and all the fields,
And all the meadows wide.
Be sure you count, should I forget,-
Some one the sum could tell,-
This, and my heart, and all the bees
Which in the clover dwell.

Emily Dickinson
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Abbreviations

AD: analog to digital
ANCOVA: analysis of covariance
ANOVA: analysis of variance
ATFP: arcus tendineus fascia pelvis
ATLA: arcus tendineus levator ani
BMI: body mass index
CI: confidence interval
CV: coefficient of variation
cm: centimetres
cmH₂O: centimetres of water pressure
EMG: electromyography or electromyographic,
EO: external obliques muscle
Hz: hertz
IAP: intra-abdominal pressure
ICC: intraclass correlation coefficient
IO: internal obliques muscle
MRI: magnetic resonance image or magnetic resonance imaging
ms: milliseconds
MVC: maximum voluntary contraction
MVE: maximum voluntary electrical activity
PFM: pelvic floor muscle
POP-Q: Pelvic Organ Prolapse Quantification examination

RA: rectus abdominis muscle

RMS: root mean square

SD: standard deviation

SUI: stress urinary incontinence

UDI: Urinary Distress Index

UI: urinary incontinence

V: volt

VP: vaginal pressure
Chapter 1 Introduction

1.1 Prevalence of urinary incontinence

Urinary incontinence (UI) is defined by the International Continence Society as “the complaint of any involuntary leakage of urine” page 168.\(^1\) It affects more women than men and is very common, with industrialized countries reporting prevalences of 30 to 60% for their adult female populations and five to 15% of women reporting daily incontinence.\(^2\) A recent telephone survey of 1365 adult women in Canada found that 37% reported some degree of urine leakage, with 21.1% of these women reporting at least daily leakage episodes and 19.2% reporting one to three leakage episodes per week.\(^3\) The Fourth International Consultation on Incontinence reports one-year incidence rates of three to 11%, which increase with age.\(^2\) The prevalence of UI in women rises to about age 50 and then stabilizes to about age 70 when it rises again.\(^2\)

The International Continence Society defines stress urinary incontinence (SUI) as: “the complaint of involuntary leakage [of urine] on effort or exertion, or on sneezing or coughing” page 168.\(^1\) It is estimated to account for 50 to 60% of women with incontinence.\(^2\) The Canadian telephone survey discussed above found that 70% of 511 women who reported urine leakage reported leaking urine with laughing, sneezing or coughing and 28% reported leaking urine during physical activity or exercise,\(^3\) all of which fall under the definition of SUI. The incidence and severity of SUI increase with age until approximately 50 years of age, when the incidence of urge and mixed incontinence supersedes that of SUI.\(^2\)
SUI is associated with high-impact exercise, childbirth, obesity and smoking. Pregnancy increases the incidence of SUI but most women become continent again following delivery. It is thought, however, that incontinence associated with pregnancy and delivery may be a risk factor for incontinence in later life. Vaginal childbirth, as compared to only ever having had caesarean births, increases a woman’s risk of developing SUI, as do instrumented deliveries and increased maternal age at the time of the birth. Parity is strongly associated with SUI in young women but this association weakens with increasing age as the other risk factors become more predominant.

More severe SUI is correlated with lower activity levels, and women may limit their activities and social contacts as a way of coping with urine leakage. Alternatively, women may restrict their fluid intake as a way of decreasing urine leakage; however, there is no evidence that fluid consumption is related to the frequency or severity of SUI.

1.2 Impact of SUI

SUI can lead to feelings of embarrassment, shame and powerlessness over one’s body. It is associated with sexual dysfunction as well. Despite this, incontinence is underreported to health professionals. The Vigod survey of 511 Canadian women with UI found that while 40% reported that incontinence interfered at least somewhat in their daily lives and 9% reported that incontinence interfered a great deal with their daily lives, only 32% of the women had consulted their physician or any other health care professional about the problem. Some of the reasons given for not discussing urine leakage with a health care provider are:
embarrassment, the leakage is seen as normal, the expectation that treatment will not benefit them, the fear of surgery and the hope for spontaneous improvement.\textsuperscript{17, 21, 22} However, UI treatment studies that have used a no intervention control group have found that the degree of incontinence experienced by the control group was unchanged or worsened over the study period.\textsuperscript{23-25} Overall, one-year remission rates for UI ranged from zero to 13%; it is not clear if this remission is due to treatment, spontaneous resolution or both.\textsuperscript{2}

Urinary incontinence is also financially burdensome: in 1997 Canadian patients paid 75\% of the cost of treatment for UI out of pocket.\textsuperscript{26, 27} The direct costs of UI include costs associated with diagnosis, treatment and routine care.\textsuperscript{27, 28} There are also indirect costs relating to UI which include decreased productivity, absenteeism, treatment for falls and skin ulcers, treatment for urinary tract infections, increased length of stay in acute care and admissions to long term care. Incontinence also produces social costs resulting from being unable to participate in activities due to embarrassment, uncertainty about the location of bathrooms or the odour associated with urine leakage.\textsuperscript{27, 28} The total cost of treating UI in the United States in 1995 was $17.5 billion US, an amount greater than the cost of treating all forms of gynaecologic and breast cancers in the same year and roughly the same as the cost of treating all forms of arthritis that year.\textsuperscript{29}

1.3 \textbf{Conservative treatment for SUI}

Pelvic floor muscle exercise therapy is recommended, by the World Health Organization’s Fourth International Consultation on Incontinence, as “first line conservative therapy [for] women with stress, urge or mixed urinary incontinence”
Foote and Moore found that women treated for SUI by a nurse continence advisor achieved similar improvements in urine leakage and quality of life as did women who were treated surgically and that the conservative treatment was more cost effective. No differences in cure rate or cost of treatment have been found for conservative treatment between women with mild or moderate UI nor does age affect a woman’s potential to benefit from conservative treatment.

Pelvic floor muscle (PFM) exercise is one form of conservative treatment. There is strong evidence to suggest that PFM exercise is better than placebo or no treatment, that supervised exercise is more effective than an unsupervised home exercise programme and that more intense exercise is better than less intense exercise; however, the optimal number, frequency and type of PFM exercises remains unknown.

Correct contraction of the PFM, without muscle substitution, is essential for PFM training to be effective. A correct contraction constricts the vaginal lumen and lifts the perineum cranially; it does not increase bladder pressure. It has been reported that as many as 30% of women with SUI cannot initially voluntarily contract their PFM correctly and that self-awareness of PFM activity is low. No demographic or medical history predictors have been found to predict who will be able to correctly perform an effective, voluntary PFM contraction.

There are three theories for how PFM exercise improves continence in SUI. The first is behavioural, suggesting that women learn to intentionally contract their PFM prior to activities that they know will provoke urine leakage. This is supported by the finding that women who are taught to do a PFM precontraction can reduce
the amount of urine they leak with a cough by more than 70% in a week.\textsuperscript{43, 44} The second theory posits that strength training produces enduring PFM hypertrophy that provides structural support through increased PFM strength, endurance and resting tone.\textsuperscript{45-49} The third theory is that training the PFMs also improves contractile efficiency and trains the normal muscle synergies between the deep abdominal muscles and the PFMs. This theory is based upon observations that the PFMs and the abdominal muscles contract synergistically and that women who perform regular, moderate exercise have stronger PFMs and are less likely to develop SUI than women who do not exercise.\textsuperscript{50-54} The second and third theories suggest that with sufficient strengthening intentional PFM precontraction becomes unnecessary. Skilling and Petros\textsuperscript{55} found that women who regained continence with PFM exercise were continent even when they were caught unaware by increases in intra-abdominal pressure.

PFM training has been shown to decrease the volume of urine leaked and the frequency of urinary incontinence episodes.\textsuperscript{12, 36, 56} PFM resting tone, the ability to isolate and correctly perform a PFM contraction, PFM strength, urethral closure pressure and bladder neck support all improve with PFM exercise.\textsuperscript{45-48, 56-60} The ability to perform strenuous physical activity without leakage improves, as does overall participation in activity.\textsuperscript{6, 7, 61}

The improvements in continence that are achieved with PFM exercise are maintained for at least one year following treatment.\textsuperscript{62-65} Longer term, over five to 15 years, the improvement diminishes; however, women who continue to perform PFM exercises regularly may be more likely to maintain their improved continence
status.64,66-71

As well, there is large variation in the reported outcomes following PFM training,12,23,25,32,72-75 which may be due to difficulty in selecting those who will most benefit.76 Several studies have attempted to determine which demographic and medical history factors predict the women with the greatest likelihood of becoming continent with PFM exercise with inconsistent results for age, detrusor instability, intrinsic sphincter deficiency, urethral hypermobility and previous continence surgery.46,72,77-81 Dumoulin et al.82 are the only group to have reported predictors of PFM function; they found that low PFM passive force and high PFM endurance were predictive of becoming continent with PFM exercise. Compliance with the exercise programme has been found to be the single highest predictor of success.72,83,84 DeLancey85,86 has suggested that the success of an intervention for SUI is most probably dependent upon the intervention treating the underlying cause of the incontinence. Unfortunately, current diagnostic tests and imaging techniques are not able to reliably predict treatment outcome as the relationships between structural deficits and SUI are not well understood.76,86 This leads to the current situation in which the World Health Organization recommends PFM exercise as a cost-effective, first-line treatment for SUI, without being able to predict which women will most benefit or even understanding the mechanisms by which it works.87
1.4 Objectives of this thesis

This thesis involves the collation of four research studies on the biomechanics and motor control of the continence system in women with and without SUI. First, the current state of the literature as it pertains to the biomechanics and motor control of female continence and the failure of the continence mechanism is reviewed and presented in Chapter 2. Then the four studies are presented as individual chapters.

The aim of the first study, presented in Chapter 3, was to determine if vaginal pressure measurements are a suitable surrogate for urethral pressure measurements for biomechanical research and physiotherapy treatment purposes, and if there is an optimal location (anterior or posterior) in the vagina at which to make these measures. This work was an essential first step in order to validate the methods used throughout the thesis to investigate differences in motor control between women with and without SUI. The three objectives of this study were to: (I) determine if there are systematic differences between pressure recordings made adjacent to the anterior vaginal wall and those made adjacent to the posterior vaginal wall, (II) determine if vaginal pressure recordings made adjacent to the anterior and posterior vaginal walls are good approximations of urethral pressure recordings and if one of the vaginal pressures is a better approximation of urethral pressure than the other, and (III) determine how PFM and abdominal muscle activity contribute to urethral pressure generation. It was determined that vaginal pressure measures are highly correlated with urethral pressure allowing the use of
vaginal pressure as a surrogate measure for urethral pressure when studying women with and without SUI.

Following this, three studies were undertaken to better understand the differences in PFM function among women with mild SUI, women with moderate to severe SUI and women without SUI. In the first of these studies, presented in Chapter 4, the ability to perform voluntary PFM contractions in supine was examined. The objectives of this study were to compare the maximum PFM and abdominal muscle EMG activation amplitudes, vaginal pressure amplitudes and muscle activation patterns among the groups during voluntary PFM contractions.

In the second study, presented in Chapter 5, the differences in involuntary PFM contractions produced by coughing were compared among women with mild SUI, women with moderate to severe SUI and women without SUI in supine and standing. The objectives of this study were to: (I) compare the maximum PFM and abdominal muscle EMG activation amplitudes generated during coughing among the groups and the testing positions, (II) compare the peak vaginal pressure generated during coughing among the groups and the testing positions, (III) compare the duration of the vaginal pressure rise and the timing of the vaginal pressure peak among the groups and the testing positions, and (IV) investigate the relationship between the generation of vaginal pressure and EMG activity recorded from the PFM and abdominal muscles in each group and testing position, and then to compare the relationships among the groups.

In the final study, presented in Chapter 6, the generation of vaginal pressure during maximum voluntary PFM contractions in supine, coughing in supine and
standing and sustained voluntary PFM contractions in supine were compared among women with mild SUI, women with moderate to severe SUI and continent women. The objectives of this study were to: (I) compare the maximum vaginal pressure among the PFM maximum contraction and coughing tasks and the groups, (II) compare the maximum vaginal pressure and the vaginal pressure rise time during coughing between the positions and the groups, and (III) determine if women with and without SUI are equally able to hold a sustained PFM contraction.

An overall discussion is presented in Chapter 7 which links the knowledge gained through this work with current knowledge in the field and provides aims and direction for future work that will enhance our understanding of the deficits within the pelvic fascia and PFM seen in women with SUI such that improvements to diagnosis and management can be implemented in the future.
Chapter 2 Literature Review

2.1 The continence mechanism and normal PFM function

The continence mechanism is comprised of two principal components that are named according to their relationship to the urethra: the intrinsic continence mechanism which is composed of the urethra with its mucosa, vascular plexus and smooth muscle and the striated urethral sphincters, and the extrinsic continence mechanism including the PFM and fascial supports, which work together to ensure that urethral pressure is greater than bladder pressure at all times except when voiding.\textsuperscript{87}

The bladder is a hollow, muscular organ that collects and stores urine until the individual is in a socially acceptable place to void.\textsuperscript{88} Its walls are composed of the detrusor muscle, which is made up of parasympathetically innervated, randomly arranged, large diameter smooth muscle bundles.\textsuperscript{87} The detrusor maintains a low-pressure environment throughout bladder filling due to its broad length-tension relationship and neural inhibition from the intramural ganglia to ensure that the upper urinary tract drains freely.\textsuperscript{87,89} In the trigone, the triangular section of the bladder base that has the openings of the ureters and the urethra at its corners, the muscle bundles are smaller and form two distinct layers although the purpose of these layers is not clear.\textsuperscript{87} At the bladder neck the muscle bundles extend obliquely and longitudinally into the urethra.\textsuperscript{87,90} These muscle bundles receive primarily sympathetic innervation and assist in occluding the proximal urethra for urine storage.\textsuperscript{99,91,92} However, this mechanism does not seem to be a primary continence
mechanism as it has been found to be incompetent in 25 to 50% continent women.93, 94

In the urethra the smooth muscle forms two layers: an inner longitudinal layer and an outer circular one.95, 96 The purpose of the longitudinal layer is unclear but it has been proposed that it either contracts during micturition to open the urethra or that it is active during storage to stabilize the urethral wall.87, 95-97 The circular layer occludes the urethral lumen to assist urine storage.87, 95, 97 The density of the circular smooth muscle declines with age which may partially account for age-related declines in urethral closure pressure98 and increases in the prevalence of UI.2

The other significant muscular component of the urethra is the somatically innervated, striated external urethral sphincter, which surrounds the middle third of the urethra and receives input from the pudendal nerve.89, 95, 99, 100 The highest closure pressure is produced in this section of the urethra.34, 101, 102 The primary role of the striated urethral sphincter is to maintain urethral coaptation at rest,87 although it does contribute to urethral closure pressure during increases in intra-abdominal pressure (IAP) as well by contracting synergistically with the PFM's during both voluntary and involuntary PFM contractions.34, 37, 101-103 More than 85% of the muscle fibres in the external urethral sphincter are small diameter slow twitch fibres that are about one third the size of the fibres in other striated muscles.87, 95, 104

In women, three muscles form the striated external sphincter: the urethral sphincter, the compressor urethrae and the urethrovaginal sphincter.90, 97, 100 Contraction of these muscles constricts the urethral lumen and pulls the urethra
posteriorly against the anterior vaginal wall and the contracting PFMs. With age the striated urethral sphincter atrophies and becomes infiltrated with smooth muscle and connective tissue.\textsuperscript{95, 105} This atrophy involves the loss of both nerve and muscle fibres and is accompanied by a decrease in urethral closure pressure.\textsuperscript{97, 106, 107}

The pelvic fasciae are primary components of the extrinsic continence mechanism and they have several principal constituents. (See Figure 2.1.) The arcus tendineus fasciae pelvis (ATFP) attaches to the pelvic sidewall from the interior surface of the pubic bone to the ischial spine bilaterally.\textsuperscript{92, 97, 108} The endopelvic, pubocervical and rectovaginal fascia hang the pelvic organs off the pelvic sidewalls directly and through connections with the ATFPs.\textsuperscript{109-112} The urethra is held forward by the pubourethral ligaments, which run from the pubic bone to the inferior edge of the proximal third of the urethra.\textsuperscript{95, 110} The pelvic fasciae contain smooth muscle fibres that are active during urine storage and relax with micturition.\textsuperscript{109, 113}

The other primary component of the extrinsic continence system is the pelvic floor, a fibro-muscular sheet that runs from the symphysis pubis anteriorly to the coccyx and sacrum posteriorly, attaching laterally to the arcus tendineus levator ani (ATLA)\textsuperscript{88} and forming the inferior and dorsal walls of the pelvis.\textsuperscript{114} It is basin shaped anteriorly and becomes domed into the abdominal cavity posterior to the vagina.\textsuperscript{115, 116} The pelvic floor supports the pelvic organs and provides additional external forces to enhance sphincter closure of both the anus and the urethra.\textsuperscript{97}
The principal muscles of the pelvic floor (PFMs) are: the pubovisceral muscle: including the puborectalis and the pubococcygeus muscles, the iliococcygeus muscle and the coccygeus muscle. The pubovisceral muscle forms a U-shaped muscular band that originates on either side of the symphysis pubis, below the ATFP and loops around the rectum to attach to the ano-coccygeal raphé. It fuses with the lateral vaginal wall and contributes fibres to the perineal body.
iliococcygeus arises from the ATLA and the coccygeus muscle arises from the ischial spine, both attach to the lateral borders of the coccyx and the lower sacrum, and together they form the solid levator plate upon which the pelvic organs rest.\textsuperscript{111} Although the different fibre direction of each of the PFMs would indicate a different action, the muscles can only be contracted collectively and thus have an amalgamated action that lifts the pelvic organs and pulls the coccyx anteriorly.\textsuperscript{33, 87, 116, 118, 119} This squeezes the vaginal canal, compresses the urethra between the anterior vaginal wall and the symphysis pubis, puts the anterior vaginal wall under tension and relieves stress from the fascial support.\textsuperscript{91, 108, 111, 113, 118, 120-122} The PFMs are innervated by the nerve to the levator ani, which arises from the S3-5 nerve roots and travels directly on the superior surface of the PFMs.\textsuperscript{123}

The PFMs are functionally adapted to produce both tonic and phasic muscle activity and are composed of more than 70\% slow twitch fibres.\textsuperscript{104, 120} The PFMs also contain smooth muscle fibres and the proportion of smooth muscle in the pubococcygeus muscle increases from lateral to medial, which may contribute to or be a result of the tonic activity of the PFMs.\textsuperscript{122} The PFMs are tonically active in all positions, contracting phasically in reaction to bladder filling and increases in IAP, while ceasing activity with micturition.\textsuperscript{122, 124-126} Imperative urgency, coughing and sneezing are accompanied by intense activity in the PFMs.\textsuperscript{120, 125, 126} The increase in PFM activation, in response to increased IAP, is proportional to the intra-abdominal pressure and is independent of the volume of urine in the bladder.\textsuperscript{122, 127} Continent women maintain the ability to maximally activate their PFMs into their eighth decade;\textsuperscript{128} however, the ability to lift the pelvic organs with a voluntary PFM
contraction decreases with age.\textsuperscript{118}

Urethral pressure at rest is maintained by three factors in essentially equal proportions: the striated urethral sphincters, the submucosal vascular bed and the circular smooth muscle and connective tissue in the urethral and periurethral tissues.\textsuperscript{129} Maximum urethral pressure increases by an average of 23\% with a change from a lying to an upright position.\textsuperscript{130} As bladder volume increases, the circular smooth muscle of the urethral wall, the striated urethral sphincter and the PFMs increase urethral pressure.\textsuperscript{96} The urethral pressure increase is greater than the bladder pressure increase,\textsuperscript{131} resulting in maintained continence. The same pattern of urethral pressure increase in response to bladder filling is observed in sitting as in supine, however, in sitting the maximum urethral pressure is not as high relative to the bladder pressure as it is in supine, resulting in lower urethral closure pressure in sitting.\textsuperscript{131} Maximum urethral pressure increases from birth to age 20-25 when it begins to decrease, the decrease becomes significant in the mid-30s.\textsuperscript{132, 133} Unlike limb muscles, the diameter of the muscle fibres in the striated urethral sphincter does not decrease with age suggesting that nerve damage, not disuse atrophy, is responsible for age-related declines in urethral closure pressure.\textsuperscript{106, 134} Hormonal changes through the menstrual cycle and at menopause do not appear to affect maximum urethral pressure.\textsuperscript{132, 135}

Increases in IAP exert two types of opening forces on the urethra: shear forces produced by unequal separation of the anterior and posterior urethral walls (the anterior wall is restrained by the pubourethral ligaments while the posterior wall moves with the vagina) and expulsive forces caused by increased IAP acting on the
bladder and its contents.\textsuperscript{76,136,137} These forces are resisted by contraction of the external urethral sphincter, aided by the contraction of the PFMs.\textsuperscript{76} Urethral pressure and urethral sphincter electromyography (EMG) amplitudes increase during coughing, Valsalva manoeuvre and voluntary PFM contractions.\textsuperscript{34,37,97,101,102}

The PFMs form the bottom of the abdominal cavity and there appears to be a synergistic relationship between the abdominal muscles and the PFMs.\textsuperscript{138-141} Several authors have reported that women are not able to perform a PFM contraction without also activating their abdominal muscles,\textsuperscript{140-142} that abdominal muscle contractions are accompanied by PFM activation\textsuperscript{142,143} and that the strength of the abdominal muscle contraction and the synergistic PFM contraction are positively correlated.\textsuperscript{143} Vaginal pressure and PFM EMG activity have been found to increase prior to the increase in abdominal pressure or abdominal muscle EMG during both voluntary abdominal muscle contractions and postural tasks, suggesting that the PFMs contract in preparation for the increase in IAP.\textsuperscript{143-147} This precontraction of the PFMs tenses the anterior vaginal wall in preparation for an increase in IAP, which produces downward forces on the abdominal contents. The PFMs resist this downward force, limiting bladder neck descent and allowing the increased IAP pressure to compress the urethra against the anterior vaginal wall, thereby preventing urine loss.\textsuperscript{97,148}

To maintain continence during episodes of increased IAP the striated urethral sphincter contracts, constricting the urethral lumen and compressing the urethra against the anterior vaginal wall. The PFMs contract, compressing the urethra against the pubic symphysis and tightening the anterior vaginal wall, which allows
IAP to compress the urethra against the anterior vaginal wall. The pelvic fascia prevents the downward movement of the pelvic organs, further tightening the anterior vaginal wall and increasing the percentage of the IAP that is transferred to the urethra. The timing of these events is important; the sphincter and PFM contractions must occur before the increase in IAP in order to arrest the downward and expulsive forces that the pressure increase exerts on the bladder, urethra and vagina.

2.2 Aetiology of SUI

The aetiology of stress urinary incontinence is not well understood, although it has been suggested that the principal mechanism is the failure of the striated urethral sphincter, with the assistance of the PFMs, to adequately resist increases in IAP.\textsuperscript{87,97,149} Such failure may be compounded by the loss of fascial support.\textsuperscript{76,97,150} Damage to the proximal urethra, the external urethral sphincter, the pelvic fascia, the PFMs, the pudendal and levator ani nerves, and altered involuntary PFM and urethral sphincter activity are all implicated in the development of SUI.\textsuperscript{76,151,152}

2.2.1 Urethra

It has been suggested that the urethra and its sphincter can be injured by vaginal delivery, vaginal prolapse, pudendal nerve damage, chronic cough and/or chronic straining.\textsuperscript{76,137,151} Recently, DeLancey et al.\textsuperscript{149} reported that decreased urethral closure pressure was more predictive of SUI (d=1.47) than were measures of urethral support (d≤0.50) or PFM strength (d≤0.60). External sphincter activity has been found to be diminished or absent in women with SUI compared to continent
controls during voluntary PFM contractions,\textsuperscript{153-155} Valsalva,\textsuperscript{156} coughing\textsuperscript{156} and cystometric bladder filling.\textsuperscript{131} Women with SUI also have smaller urethral sphincters than do continent women and the size of the sphincter is negatively correlated with the severity of incontinence.\textsuperscript{157, 158} Pudendal nerve conduction time to the urethral sphincter has been found to be prolonged in women with SUI and the conduction time is negatively correlated with maximum urethral closing pressure.\textsuperscript{159}

2.2.2 Fascia

Trauma from vaginal delivery is considered to be the primary cause of fascial damage in women with SUI,\textsuperscript{76} however, genetic differences in collagen composition,\textsuperscript{160} damage from chronic cough or straining\textsuperscript{161} and high-impact athletic activities\textsuperscript{5, 7} have also been implicated in fascial damage. Damage to the pubourethral ligaments and the endopelvic fascia has been observed in women with SUI using magnetic resonance imaging (MRI).\textsuperscript{110} Shishido et al.\textsuperscript{162} found that women with SUI demonstrated nearly 50% more movement of the anorectal junction on Valsalva manoeuvre than did continent women (p<0.01) and that in continent women the anorectal junction moved in the same direction as the urethra while in women with SUI the movement was away from the urethra. This suggests that the women with SUI had less pelvic organ support and that the straining might put a stretch on the pelvic floor structures leading to possible ongoing injury. The increase in movement may delay or prevent the transmission of IAP to the urethra which potentially allows urine to leak.\textsuperscript{97}

Differences have been found in the composition of the connective tissue of
women with SUI compared to continent women. Women with SUI have less collagen,\textsuperscript{163-167} a decreased ratio of type I to type III collagen,\textsuperscript{164} fewer cross links,\textsuperscript{164, 168} and a higher rate of collagen replacement\textsuperscript{165, 169} in the skin, vagina and pelvic ligaments than continent women. It is not possible to determine whether these differences in the connective tissue of women with SUI predispose them to SUI or whether they result from previous trauma, as collagen is dependent upon loading to maintain its structure and strength.\textsuperscript{170} However, the finding that increased collagen content in the skin is correlated with higher closure pressure at rest in the proximal urethra along with higher maximum urethral closure pressure and maximum abdominal pressure transmission under stress\textsuperscript{171} and the finding that more than half of the variance in bladder neck descent is attributable to additive genes\textsuperscript{160} together suggest that altered collagen structure and metabolism predispose some women to SUI.

2.2.3 Pelvic floor muscles

Women with SUI have been found to have smaller and thinner PFMs and wider urogenital hiatuses than continent women using ultrasound and MRI.\textsuperscript{172-177} They have also been found to produce less lift with a PFM contraction\textsuperscript{178, 179} and to permit more bladder neck movement with coughing than continent women.\textsuperscript{150} Gaps in the PFMs have also been observed on MRI and ultrasound, and have been associated with de novo SUI in women post-partum.\textsuperscript{174, 177, 180} The damage is seen primarily in the most infero-medial portion of the pubovisceral muscle.\textsuperscript{181, 182} In older women, PFM defects seem to occur with equal frequency in women with and without SUI suggesting that in younger women SUI may be more related to urethral support and
PFM function, while in older women it may be more related to urethral function.\textsuperscript{149, 183}

Biopsies of the PFMs in women with SUI have found an increased proportion of pathological muscle cells and increased diameter of the slow twitch muscle fibres, as well as partial replacement of muscle fibres with adipose and connective tissue.\textsuperscript{176, 184, 185} The loss of muscle fibres found on PFM biopsies is associated with lower abdominal pressure transmission ratios and with lower urethral closing pressures during stress.\textsuperscript{186} These findings support the role of the PFMs in providing urethral support and augmenting urethral closure pressure, demonstrating that PFM damage is associated with the primary deficit in SUI: decreased urethral closure pressure with increased intra-abdominal pressure.

Along with damage to the PFMs and fascia, partial denervation of the PFMs is associated with SUI.\textsuperscript{187} Smith et al.\textsuperscript{159} found that women with SUI had significantly longer nerve conduction times to the pubococcygeus muscle compared to continent women. Weidner et al.\textsuperscript{188} found that concentric needle EMG signals from the PFMs of women with SUI displayed significantly fewer turns per second than those of continent women, indicating a loss of motor units. This finding is supported histochemically by the finding of fibre type grouping in the PFMs of women with SUI.\textsuperscript{185, 189} Nerve injury may result from both stretching and compression of the pudendal and levator ani nerves during vaginal delivery.\textsuperscript{109, 123} The denervation largely recovers spontaneously following delivery but signs of denervation may recur in later life, possibly due to ongoing injury from chronic coughing or straining\textsuperscript{151, 187, 190} or due to overuse of the remaining motor units.\textsuperscript{191} More
denervation is evident in women who are multiparous, have had instrumented delivery, long second stages of labour, third degree perineal tearing and/or heavier babies.161,192

2.2.3.1 Voluntary PFM function in women with SUI

There is disagreement in the literature about whether the ability to produce a voluntary PFM contraction is impaired in women with SUI. Some authors have found that women with SUI produce lower PFM EMG amplitudes and less intravaginal pressure than continent women when they attempt maximal voluntary PFM contractions,128,179,193-195 while others report no differences in the ability to generate maximum PFM activation between women with and without SUI.149,196 Interestingly, one study that found no differences in maximum PFM force between women with and without SUI did find that women with SUI were less able to generate force quickly,196 suggesting that fast twitch muscle fibres may be most affected in SUI and that solely measuring maximum pressure or EMG amplitudes may be insufficient to distinguish between women with and without SUI. This may be related to the nerve and muscle injuries discussed in the previous section.

The PFMs and the abdominal muscles work in synergy to generate intravaginal pressure during voluntary PFM contractions in continent women.139 Thompson et al.194 found that, like continent women, women with SUI contracted their abdominal muscles when they attempted voluntary PFM contractions. However, they had lower abdominal muscle EMG activation than continent controls, which may suggest that altered muscle activation patterns play a role in SUI or it may suggest that women
with SUI avoid activating their abdominal muscles in order to avoid generating IAP, thereby preventing urine leakage.

2.2.3.2 Involuntary PFM function in women with SUI

It is believed that the involuntary PFM contractions produced with increases in IAP are an important part of the continence mechanism.\textsuperscript{76} Coughing, voluntary abdominal muscle contractions and limb movements produce preparatory PFM contractions to counteract the increase in IAP.\textsuperscript{120,143,146,147} Coughing is accompanied by an increase in urethral pressure that rises approximately 240 ms prior to the rise in bladder pressure in continent women.\textsuperscript{34,197} A strong correlation has been found between the forcefulness of a cough and the EMG amplitude of the corresponding PFM contraction in continent women.\textsuperscript{127,198}

These involuntary PFM contractions have been found to be disrupted in women with SUI. The rise in urethral pressure is diminished and delayed,\textsuperscript{197,199-201} along with a delayed rise in PFM EMG activity.\textsuperscript{198,202} As well, the ability to modulate the strength of the PFM contraction to the forcefulness of the cough is diminished in women with SUI.\textsuperscript{203} In women with SUI, maximum urethral closing pressure decreases with repeated coughs while it remains unchanged in continent women,\textsuperscript{204} suggesting that PFM fatigue may play a role in SUI. Ultrasound imaging has shown that during coughing the anorectal junction moves ventrally in continent women, while in women with SUI the anorectal junction moves dorsally.\textsuperscript{205} This provides evidence that the PFM resist the dorsal-caudal movement of the pelvic organs produced by increased IAP and suggests that the PFM contraction is impaired in women with SUI.
Contrarily, using needle EMG, Bø et al.\textsuperscript{206} found that seven young women with SUI demonstrated similar synergistic PFM and urethral sphincter contractions in response to coughing, as did seven matched continent controls. This conflicting result may be due to the fact that there are different aetiologies for SUI in different populations; these women were nulliparous and, therefore, were unlikely to have traumatic nerve or muscle damage. This conflicting result also highlights the challenges to understanding the biomechanical basis for SUI.

2.2.3.3 Tonic PFM function in women with SUI

Tonic PFM activity has been found to be altered in women with SUI: they do not demonstrate the increase in PFM EMG amplitude with cystometric bladder filling that continent controls do,\textsuperscript{131} and they display increased tonic PFM activity during standing.\textsuperscript{207} Smith et al.\textsuperscript{207} found that the women with SUI demonstrated both greater centre of pressure movement and higher trunk and PFM EMG amplitudes during static standing than did continent women. This suggests that postural control may be impaired in women with SUI and may partly explain the increased risk of falls in women with incontinence.\textsuperscript{2} In another study, Smith et al.\textsuperscript{146} examined PFM EMG activation timing in response to rapid arm movements and found that continent women activated their PFMs prior to the onset of deltoid EMG activity while women with SUI activated their PFMs following the onset of deltoid activity (p=0.002). They also found that some of the women with SUI demonstrated a period of decreased PFM EMG activity that straddled the onset of deltoid EMG activity.

Morin et al.\textsuperscript{208} have reported that women with SUI produce lower passive forces in the vagina, both statically and during a constant velocity stretch, compared to
continent women. The passive forces are believed to be the result of a combination of tonic PFM activity and fascial support.

These recent studies of PFM activity therefore suggest that both the level of tonic PFM activity and the phasic timing relative to limb movements may be altered in women with SUI.

2.3 Instrumentation used to quantify the function of the continence mechanism

As the continence mechanism is not comprised of a single structure or tissue, but rather is a amalgamation of structures and tissues that, while not sufficient each unto itself to maintain continence, together ensure that urethral pressure exceeds bladder pressure and continence is maintained,87 no single instrument is capable of measuring the function of the full continence mechanism. Indeed, the International Continence Society recommends that researchers use tools that assess different aspects of continence in aggregate in order to better understand how the components of the continence mechanism complement one another and how they fail in women with SUI.209

2.3.1 Urethral Pressure

By definition, SUI occurs when urethral pressure does not increase sufficiently to exceed IAP,1 and recording urethral pressure allows for the effect of a PFM contraction or a cough on the urethra to be measured directly.101,210 In continent women, urethral closure pressure increases with bladder filling, moving from supine to sitting or standing and PFM contractions.101,131,155,211 It is not affected by hormonal fluctuations throughout the menstrual cycle.135 During coughing, the rise
in urethral pressure exceeds the rise in bladder pressure, due to abdominal pressure transmission and contraction of the PFM and the striated urethral sphincter. In women with SUI, the increase in urethral pressure with bladder filling is attenuated and there are smaller increases in urethral pressure when moving from supine to upright positions and with a PFM contraction compared to continent women. During coughing, the increase in urethral pressure is less than the increase in bladder pressure resulting in urine leakage.

Urodynamic testing, including urethral pressure recording, is the gold standard for diagnosing SUI. Urethral pressure is recorded with catheters that are either air-filled, fluid filled, fibre-optic or tipped with microtransducers. Although the normative values recorded with different types of urethral catheters are quite variable, the type of catheter used does not seem to impact the reproducibility of the measures. However, the instructions given to the volunteer, the testing position and the degree of bladder fullness can all affect the pressure measurement, therefore, the procedure must be rigidly standardized. Urethral pressure is measured either with the catheter pulled through the urethra at a steady rate (urethral profilometry) or with the pressure transducer positioned in the portion of the urethra of interest (usually the region of highest pressure).

Urethral pressure measurements are highly variable between testing sessions and there is so much overlap in resting urethral pressure, maximum urethral pressure and maximum urethral closure pressure (maximum urethral pressure minus bladder pressure) between women with SUI and continent women that it is not possible to use urethral pressure measurements alone to distinguish between
these groups.\textsuperscript{214} As well, urethral pressure alone does not provide much information about the biomechanical deficits underlying urine leakage because the striated urethral sphincter, the PFM and abdominal pressure transmission all act on the same section of the urethra.\textsuperscript{92, 216} Urethral pressure measurements are also technically more difficult to make than vaginal pressure measurements and there are risks of significant discomfort and infection inherent in measuring urethral pressure that are substantially reduced when vaginal pressure is measured instead.\textsuperscript{217} For these reasons, vaginal pressure is frequently used as a surrogate measure of urethral pressure; however, the relationship between urethral pressure and vaginal pressure is very poorly understood.\textsuperscript{218}

\textbf{2.3.3 Vaginal Pressure}

Pressure measured in the vagina does not include contributions from the striated urethral sphincter, so in measures of vaginal pressure the PFM and IAP are the main contributors. Vaginal pressure and force measures are the most frequently reported measures of PFM function in the literature.\textsuperscript{219}

Vaginal pressure is measured with commercially available vaginal probes,\textsuperscript{140, 194} vaginal balloons\textsuperscript{220, 221} and fluid filled bags.\textsuperscript{222} The probes and balloons are most frequently air-filled which may demonstrate an impulse transmission delay that must be removed if the timing of the pressure data is to be compared to the timing of data collected with other systems (e.g. EMG or fluid-filled pressure systems).\textsuperscript{138} Force within the vagina has been measured with dynamometers\textsuperscript{223, 224} or with custom instrumented probes.\textsuperscript{225} All of these measurement systems are affected by the location of the transducer in the vagina. The highest pressure and force signals
are recorded in the anterior-posterior direction, at a depth adjacent to the PFMs’ attachments to the lateral vaginal wall.\textsuperscript{222, 226, 227} Vaginal pressure and force measures have been reported to be reliable\textsuperscript{228-231} and to be unaffected by either bladder fullness or hormonal fluctuations during the menstrual cycle.\textsuperscript{232} There is no difference in the ability to produce vaginal pressure and force with a maximum voluntary PFM contraction between the supine and standing positions.\textsuperscript{224, 233} The primary limitation of vaginal pressure measures is that equivalent vaginal pressure can be produced by straining as with a PFM contraction, therefore, it is essential to confirm that PFM contractions are being performed correctly with other measures.\textsuperscript{141}

\textbf{2.3.3 PFM EMG}

Electromyography (EMG) is frequently used to quantify muscle activation.\textsuperscript{234} Surface EMG, with electrodes mounted on a vaginal probe or sponge, is the most common method of recording EMG activity from the PFMs but needle and fine-wire electrodes have been used as well.\textsuperscript{235} Surface EMG recordings have the advantage over recordings made with indwelling electrodes in sampling activity from a greater number of motor units and providing a picture of the overall muscle activity.\textsuperscript{236} There are several commercially available vaginal probes for recording PFM EMG, however, only one, the Femiscan™ (Mega Electronics Ltd., Kuopio, Finland), has a bipolar electrode configuration that meets accepted SENIAM standards\textsuperscript{237} and records from the right and left sides of the PFMs separately. Several groups have tested PFM EMG recordings made using the Femiscan™ probe for reliability and have found that PFM MVCs performed within a subject have moderate to high
interclass correlation coefficients (ICCs) (between 0.70 and 0.98) and coefficients of variation (CVs) between 8.5% to 14.3%. During coughing the ICCs have been repeatedly high (0.79 and 0.96) and the CVs good (12.7% to 17.3%), indicating that between-trial PFM EMG recordings made with the Femiscan™ vaginal probe demonstrate good reliability. The between-day reliability of PFM EMG amplitude data recorded during PFM MVCs and coughs with the Femiscan™ probe is not as good, with ICCs of 0.36 to 0.70. When the coughs were normalized to the PFM MVCs the reliability improved, suggesting that the position of the probe relative to the PFMs was not consistent between days and that only normalized data should be compared between testing sessions. Hallencreutz-Grape et al. have reported higher between day reliability with the Femiscan™ probe, however, they included within day comparisons in the between day comparison which artificially improved the reliability.

Crosstalk is a concern with any EMG recordings and particularly with recordings made from small muscles that are in close proximity to other muscles. As the PFMs are small muscles that attach laterally to the obturator internus fascia and that cannot be contracted maximally without also contracting the abdominal muscles, a preliminary study was undertaken to determine the effect of crosstalk on the PFM EMG signals recorded with the Femiscan™ vaginal probe. Eight women performed PFM contractions in isolation and in conjunction with maximum isometric contractions of the hip adductors, hip external rotators, rectus abdominis, transversus abdominis and the hip extensor muscles. Overall, there was no increase in PFM EMG amplitude or vaginal pressure with the addition of the
other contractions; although in two women adding the abdominal and hip external rotator contractions increased the EMG amplitudes recorded from the PFMs, but not the vaginal pressure, indicating that in most cases crosstalk does not greatly interfere with the PFM EMG signals recorded with the Femiscan™ probe; however, maximal hip rotator contractions can result in crosstalk in some women. Measures, such as confirming the volunteer’s ability to perform a PFM contraction correctly, providing specific instructions to the volunteer to avoid contracting the hip muscles and observing the volunteer for accessory muscle contraction or movement at the hip joints, should therefore be undertaken to reduce crosstalk during PFM EMG data collection.

A further limitation with EMG recordings is that PFM EMG amplitudes may be normal even though the transmission of PFM force to the vagina and urethra may be disrupted by PFM avulsion or fascial damage, which demonstrates the necessity for exploring continence function from multiple angles simultaneously in order to understand the continence mechanism in women with and without SUI.

### 2.3.4 Other measures

Imaging techniques, such as ultrasound and MRI, have recently been used to measure continence function. They have the advantage over surgical techniques of being non-invasive and do not subject women to the ionizing radiation risks that are present with roengenograms. Good correlations have been reported between ultrasound images and roentgenograms in the evaluation of the continence mechanism. Greater anatomical detail of the pelvic structures is visible in MRIs than is visible with any other imaging technique, however, MRI is more expensive.
and there are significant contraindications to its use compared with ultrasound imaging.\textsuperscript{243}

Ultrasound imaging and MRI are both used to measure the position of the pelvic organs\textsuperscript{,110,244} to estimate the volume of the urethral sphincter and PFMs\textsuperscript{,157,158,245} to assess the integrity of the PFMs\textsuperscript{,174,183,246} to measure the effect of PFM contractions, Valsalva manoeuvres and coughing on the PFMs and the pelvic organs\textsuperscript{,33,118,137,179,205,247}\textsuperscript{,247-249} and to determine the effect of PFM training on these measures.\textsuperscript{56,57}

Imaging has also been used to assess the integrity of the paravaginal and endopelvic fascia, for although they are multiplanar structures that cannot be seen directly on either ultrasound or MRI, damage to these structures can be inferred from observing the movement of the pelvic organs under stress conditions.\textsuperscript{59,76} More recently, functional MRI has begun to be used to determine which areas of the brain control continence,\textsuperscript{250} how this control is altered in women with SUI and the effect of PFM exercise programs on brain activation.\textsuperscript{59}

\section*{2.4 Concluding remarks}

Stress urinary incontinence is recognized as a significant problem for many women that has great social, emotional and economic costs. Its aetiology is multifactorial with growing evidence that PFM, urethral sphincter, fascia, nerve and motor control dysfunction all contribute.\textsuperscript{221,251} This research was undertaken to improve our understanding of whether PFM function and motor control deficits are present in women with SUI and how they reveal themselves during voluntary and involuntary PFM contractions.
Chapter 3 Vaginal Pressure Measurements Provide Valid
Representations of the Changes in Urethral Pressure that Occur
during Voluntary Pelvic Floor Muscle Contractions and during
Coughing

3.1 Abstract

Aim: The purpose of this study was to determine if vaginal pressure recordings
are a reliable surrogate for urethral pressure recordings for use in biomechanical
research and pelvic floor muscle (PFM) rehabilitation. Methods: Eleven women
participated. They performed three maximum voluntary PFM contractions (PFM
MVCs) and three 30 s series of repeated PFM contractions in supine and standing,
and three maximum effort coughs in standing. Pressure data were recorded in the
vagina and the urethra simultaneously with surface electromyography (EMG) data
recorded from the PFMs. Peak pressure amplitudes were compared with repeated
measures analyses of variance. Regression analyses and cross-correlation functions
were used to compare the peak pressure amplitudes, the patterns of pressure
generation and the timing of the pressure bursts. Ensemble average curves were
created to describe the relationship between vaginal pressure and urethral
pressure, and between PFM EMG and urethral pressure. Results: Peak urethral
pressure was higher than peak vaginal pressure during all tasks and the relationship
between them was linear in all tasks. During the repeated PFM contractions the
relationship between the urethral and vaginal pressure was linear and there was no
time delay. The ensemble average urethral pressure versus vaginal pressure curves
were curvilinear during the supine PFM MVCs and linear during the standing PFM MVCs and coughing. The ensemble average urethral pressure versus PFM EMG curves were curvilinear during all tasks. Conclusions: The results of this study suggest that vaginal pressure is a valid surrogate measure for urethral pressure during maximum PFM contractions and coughing.

3.2 Introduction

The primary impairment in stress urinary incontinence (SUI) is the inability of the urethral sphincters and the pelvic floor muscles (PFMs) to generate sufficient urethral pressure to overcome increases in intra-abdominal pressure, whether due to muscle defects, damage to urethral support structures or poor motor control patterns. PFM exercise is an effective, conservative treatment for SUI, which is thought to work by improving the PFMs’ ability to directly compress the urethra and also by providing a firm surface against which increases in intra-abdominal pressure can compress the urethra.

Vaginal pressure is frequently used as a measure of PFM function and as a surrogate measure for urethral closure pressure in physiotherapy research and practice. Efforts have been made to determine where in the vagina pressure measurements should be made, with recent research indicating that greater pressure is generated antero-posteriorly than laterally and that the highest pressure zone is adjacent to the vaginal attachments of the PFMs.

However, the relationship between vaginal and urethral pressure measurements during voluntary pelvic floor muscle contractions and functional tasks has not been established. In the only published accounts of the correlation between PFM strength
and urethral pressure generated during voluntary PFM contractions, the authors used vaginal palpation as their measure of PFM strength and they performed their assessments of urethral pressure and PFM strength on different days, both factors resulting in potentially serious limitations to the validity of the results. In addition, the correlation between vaginal and urethral pressure found during voluntary PFM contractions cannot be generalized to what occurs during more functionally relevant tasks such as coughing.

The purpose of this study was to investigate the relationship between vaginal and urethral pressure during both voluntary PFM contractions and coughing to determine whether changes in vaginal pressure during each task provide a reliable representation of changes in urethral pressure, and to determine the best place (anterior or posterior vaginal wall) to locate the pressure transducer within the vagina to achieve the optimal representation of urethral pressure.

A secondary purpose of this study was to evaluate the contribution of the PFMs and the abdominal muscles to the generation of urethral pressure.

### 3.3 Materials and methods

#### 3.3.1 Sample

This was a cross-sectional, observational study. Ethics approval was received from the Queen’s University Health Sciences and Affiliated Teaching Hospitals Research Ethics Board prior to subject recruitment. Women without neurological or rheumatological diagnoses, diabetes, prolapse ≥ POP-Q stage II or previous pelvic
surgery were recruited using posters and word of mouth. Women with symptoms of incontinence were included in the sample.

3.3.2 Instrumentation

Urethral pressure was measured using a saline-filled, 7 French triple lumen catheter (Laborie Medical Technologies, www.laborie.com) interfaced with a Becton Dickinson DTXTM Plus DT-12 pressure transducer (calibration 0.008 V=1 cmH2O, linear range 0-10 V). The volunteers were asked to empty their bladders and then the catheter was inserted into the urethra until a drop of urine was noted in the catheter to ensure that the tip of the catheter was positioned in the bladder. The position of the lateral catheter transducer was then adjusted manually, both longitudinally and by rotation, until the location was found that resulted in the highest pressure values during both a PFM contraction and a cough.

Vaginal pressure data were recorded adjacent to the anterior and posterior vaginal walls using two air-filled, 10 French rectal balloons (Laborie Medical Technologies, www.laborie.com) mounted on the anterior and posterior surfaces of a Femiscan™ vaginal probe (Mega Electronics Ltd., Kuopio, Finland), centered 3.5 cm from the vaginal introitus.226 (See Figure 3.1.) The pressure balloons were interfaced with Motorola MPX5010 Integrated Silicon pressure transducers (calibration 0.04 V=1 cmH2O, linear range 0-10 V). Because air-filled chambers are slower to transmit pressure changes than are water-filled chambers, there was a 5 ms delay in the pressure impulses recorded by the vaginal transducers. This delay was removed prior to all data processing. All of the pressure transducers were
interfaced directly with a National Instruments NIDAQ PCI-MIO-16XE-10 analogue to digital (AD) converter.

Figure 3.1 Modified Femiscan™ vaginal probe. Black arrow: anterior vaginal pressure balloon. Grey arrow: posterior vaginal pressure balloon. Fine arrows: left PFM EMG electrode pair. Striped arrow: superior or proximal vaginal pressure balloon, used as a surrogate for intra-abdominal pressure: these data were not included in this analysis.

Surface electromyography (EMG) data were recorded from the PFMs bilaterally and from the rectus abdominis (RA), external obliques (EO) and internal obliques (IO) muscles on the left side. EMG data were recorded from the PFMs with the surface electrodes on the Femiscan™ vaginal probe interfaced with Delsys D.E.2.1 pre-amplifiers. Similarly, EMG data were recorded from the abdominal muscles using Delsys™ D.E.2.1 active surface electrodes (Delsys Inc., Boston, Massachusetts). All of the preamplified EMG data were further amplified using a Delsys Bagnoli™ 16-channel EMG amplifier and had an overall gain of 1000.
The pressure and EMG data were sampled simultaneously through the AD converter at 1000 Hz and EMGworks® Acquisition software (Delsys Inc., Boston, Massachusetts) was used to acquire and store the data on a personal computer.

3.3.3 Data collection

After the volunteers provided written, informed consent (See Appendix A.), demographic data were recorded and the volunteers completed the Urogenital Distress Inventory (UDI, See Appendix B.).

The women were taught how to correctly perform a PFM contraction, confirmed by vaginal palpation and observation of cranial and anterior motion of the perineum with minimal contributions from the hip and gluteal muscles. The volunteers were allowed as many practice contractions as they needed to be able to consistently perform a PFM contraction correctly.

The skin over the abdomen was cleansed with rubbing alcohol and surface electrodes were centred over the following sites in line with the muscle fibres: RA 2 cm lateral and caudal to the umbilicus, EO over the tip of the eighth rib and IO 2 cm proximal to the midpoint of a line from the anterior superior iliac spine to the symphysis pubis. A common reference electrode was placed over the left anterior superior iliac spine.

The urethral catheter was inserted and adjusted as described above. The volunteer was then assisted to place the Femiscan™ probe in her vagina. The volunteer performed several PFM contractions to ensure that inserting the vaginal probe had not changed the position of the urethral catheter and adjustments were made if required.
In supine, data were collected with one pillow under the head and two pillows under the knees to ensure that the volunteer was comfortable. Resting data were collected first with the volunteer instructed to close her eyes and to relax as much as possible. Next, the volunteer performed three repetitions of a maximum voluntary PFM contraction (PFM MVC) with the instructions to “Pull up and in and squeeze around the probe”, followed by three, 30 s series of repeated PFM contractions with the instructions: “Contract your PFM’s maximally and then relax fully. Do not hold either the contraction or the relaxation and go at a comfortable pace.” If the volunteer held her breath or demonstrated visible accessory muscle use, the trial was stopped and repeated. The volunteer was then assisted to come to standing, taking care to avoid pulling on any of the catheters or wires. She stood barefoot with her feet comfortably apart in her normal posture. In standing, resting data were recorded first, with the instructions: “Stand comfortably and as relaxed as possible”, then the volunteer performed three PFM MVCs and three repetitions of the 30 s series of repeated PFM contractions with the same instructions as above. Lastly, the volunteer performed three maximum effort coughs, with the instruction to “Cough once, as hard as you can.” At least 60 s of rest were given between trials.

### 3.3.4 Data processing and analysis

#### 3.3.4.1 Peak pressure amplitudes

All pressure data from the PFM MVC and cough tasks were dual filtered with a third order, 5Hz low pass Butterworth filter. The filtered mean of the first 100 data points was subtracted from all remaining values to remove bias associated with differences in urethral and vaginal anthropometrics and vaginal pressure balloon.
inflation. The data were converted from voltages (V) to centimetres of water (cmH₂O).

The highest smoothed value for each pressure recording site during each repetition of each task: supine PFM MVC, standing PFM MVC and cough, was determined to be the peak pressure for that repetition. The peak pressures from each repetition were compared among the pressure recording sites and tasks using a two-way repeated measures analysis of variance (ANOVA) model (Minitab v.14, State College, Pennsylvania) in which the pressure recording site by task interaction was included. Post hoc tests were completed as appropriate using the Bonferroni method (α=0.05).

Linear regression analyses (α=0.05) of the peak urethral pressure versus the peak anterior and posterior vaginal pressure were also performed.

3.3.4.2 Repeated contractions

Regression analyses (α=0.05) of the urethral pressure versus the anterior and posterior vaginal pressures from the repeated contractions in supine and standing were calculated for each trial. The slopes of the regression lines and their respective coefficients of determination were described using the mean and standard deviation for each position. The slopes and coefficients of determination were compared between the urethral pressure versus the anterior vaginal pressure curves and the urethral pressure versus the posterior vaginal pressure curves using student t-tests (α<0.05).
The timing of the urethral pressure bursts generated during the repeated PFM contractions was compared to the timing of the anterior and posterior vaginal pressure bursts using the time lags computed from the cross-correlation functions performed on the filtered data.

3.3.4.3 Relationship between vaginal pressure and urethral pressure

To describe the relationship between vaginal pressure and urethral pressure during the PFM MVCs and coughs, ensemble average urethral pressure versus vaginal pressure curves were created by trial for each task. The data were filtered as described above. The rise in urethral pressure was divided into five percent increments and the amplitude of each of the vaginal pressures was determined for each urethral pressure increment. The vaginal pressure amplitudes were normalized to the maximum amplitude achieved by that pressure transducer during that repetition of that task.

The ensemble average urethral pressure versus vaginal pressure curves were tested for significance ($\alpha < 0.05$) using runs tests (GraphPad Prism™ v.4 software, La Jolla, California). The patterns demonstrated by the vaginal pressure recording sites were then compared within the tasks. The curves were considered to be significantly different if the same order equations produced the best fit and the 95% confidence intervals for the constants did not overlap, or if different order equations produced a best fit.
3.3.4.4 Contributions of PFM and abdominal muscle activation to urethral pressure

The same method of creating ensemble-average curves was used to describe the contributions of the PFMs and the abdominal muscles to the generation of urethral pressure. As there was no difference between the EMG recordings from the two sides of the PFMs, the data from the right and left sides were pooled. The curves were tested for significance as described above and the same method was used to determine if the curves were significantly different from one another.

The time of the peak smoothed PFM EMG was determined and subtracted from the time of the peak smoothed urethral pressure for each task. The relative timing was compared among the tasks using a one-way, repeated measures ANOVA ($\alpha=0.05$).

3.4 Results

3.4.1 Sample

Eleven women participated; all were able to perform a PFM contraction correctly. Their median age was 42 (range 29 to 68) years. The participants had a median body mass index of 26.7 (range 18.9 to 29.7) kg/m$^2$, a median UDI score of 3/19 (range 0 to 8), a median of 2 (range 0 to 4) children and a median of 2 (range 0 to 2) vaginal deliveries. The most commonly reported symptoms on the UDI were SUI (6 of 11), urgency (4 of 11) and urge incontinence (4 of 11).

Figure 3.2 provides a typical example of the data recorded from the PFMs and the vaginal and urethral pressure transducers. The peak pressure amplitude and
timing data were normally distributed, therefore parametric statistical tests were used.

Figure 3.2 Rectified PFM EMG, urethral pressure, anterior vaginal pressure and posterior vaginal pressure data recorded from one volunteer: a. Supine PFM MVC, b. Standing PFM MVC, c. Cough and d. Supine 30 s series of repeated PFM contractions. EMG data are presented in volts, pressure data are presented in cmH₂O. PFM: pelvic floor muscles, UP: urethral pressure, AVP: anterior vaginal pressure and PVP: posterior vaginal pressure.

3.4.2 Peak pressure amplitudes

The peak (mean ± standard deviation) rise over baseline in urethral pressure was higher during coughing (286.0 ±138.1 cmH₂O) than during either the supine or the standing PFM MVCs (145.8 ±79.3 cmH₂O supine, 176.8 ±136.9 cmH₂O standing, p<0.001 for both) and there was no difference between the peak rise over baseline
in urethral pressure generated during the supine and standing PFM MVCs (p=1.00). The baseline urethral pressure was higher in standing than in supine (p=0.035).

There was no difference in peak rise over baseline in vaginal pressure among the three tasks for either the anterior or the posterior pressure recording site (anterior vaginal pressure: 31.8 ±12.9 cmH₂O supine MVC, 40.6 ±16.8 cmH₂O standing MVC, 42.0 ±20.4 cmH₂O cough, posterior vaginal pressure: 28.6 ±17.2 cmH₂O supine MVC, 34.3 ±20.0 cmH₂O standing MVC, 26.9 ±13.4 cmH₂O cough, p=1.00).

The regression analysis found that women who generated higher urethral pressures increases also generated higher vaginal pressures increases (p<0.001 for all tasks).

3.4.3 Repeated contractions

The regressions between urethral pressure bursts and vaginal pressure bursts were linear for both the anterior and posterior vaginal pressure recording sites in both supine and standing. (See Table 3.1.) There were no significant differences between the anterior and posterior vaginal pressure recordings in either the slopes or the correlation-coefficients of the regressions between urethral pressure and vaginal pressure (p>0.6).

Table 3.1 Slopes and r² values for the urethral pressure versus vaginal pressure regression equations during the 30 s series of repeated PFM contractions.

<table>
<thead>
<tr>
<th>Urethral Pressure versus Position</th>
<th>Slope (SD)</th>
<th>r² (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior Vaginal Pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supine</td>
<td>2.07 (3.17)</td>
<td>48.77 (27.95)</td>
</tr>
<tr>
<td>Standing</td>
<td>0.62 (1.04)</td>
<td>32.92 (23.51)</td>
</tr>
<tr>
<td>Posterior Vaginal Pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supine</td>
<td>2.21 (3.64)</td>
<td>43.98 (27.42)</td>
</tr>
<tr>
<td>Standing</td>
<td>1.21 (1.84)</td>
<td>33.11 (20.87)</td>
</tr>
</tbody>
</table>
The cross-correlation analyses showed no measureable time delay between the urethral pressure bursts and either the anterior or the posterior vaginal pressure bursts (mean delay 0 ms, 95% confidence interval -0.0 to 0.0 ms for both).

**3.4.4 Relationship between vaginal pressure and urethral pressure**

The relationship between the increase in urethral pressure and the increase in vaginal pressure during the PFM MVCs and the coughs is presented in Figure 3.3. The urethral pressure rise versus vaginal pressure rise curves were curvilinear during the supine MVCs and linear during the standing MVCs and the coughs. The slopes were much lower during the standing MVCs (anterior slope = 0.252 ±0.049, posterior slope = 0.321 ±0.043) than during the coughs (anterior slope = 0.768 ±0.030, posterior slope = 0.772 ±0.092, p<0.001 for both).

The vaginal pressure was much higher at the onset of the urethral pressure rise during the MVCs as compared to during the cough, as indicated by the y-axis intercepts, while the anterior and posterior vaginal pressure had intercepts close to zero during the coughs. This indicates that during the PFM MVCs vaginal pressure increased before the onset of the rise in urethral pressure, however, during coughing the vaginal and urethral pressure rises began nearly simultaneously.
3.4.5 Contributions of PFM and Abdominal Muscle Activation to Urethral Pressure

The relationship between the generation of urethral pressure and PFM EMG activity was curvilinear for all three tasks, indicating that the PFMs were more strongly involved in generating urethral pressure during the initial part of the pressure rise than they were in the latter part of the pressure rise. (See Figure 3.4.) The y-axis intercept was lower during the cough than during the standing PFM MVC (p<0.05), there was no difference between the y-axis intercept during the supine PFM MVC and the other two tasks. The initial rise of the urethral pressure rise
versus PFM EMG curve was steeper during coughing than during either of the MVC tasks (p<0.05 for both).

The PFM EMG peaked before the urethral pressure peak for all three tasks. The time between the PFM EMG peak and the urethral pressure peak was different for each task (p=0.047, supine PFM MVC: 1048 ±1537 ms, standing PFM MVC: 36 ±1829 ms and cough: 142 ±1203 ms).

It was not possible to model the relationships between the generation of urethral pressure and RA and EO EMG activity during the supine and standing PFM MVCs, because the timing of the RA and EO activity bursts relative to the increase in urethral pressure differed so much among the women that the ensemble average curves had slopes that were not significantly different from zero. (See Figure 3.5.) The urethral pressure rise versus RA and EO EMG curves were curvilinear during coughing. The urethral pressure rise versus IO EMG curves were curvilinear for all three tasks. (See Figure 3.4.)
Figure 3.4 Ensemble average curves for urethral pressure rise versus PFM EMG 1 and IO EMG 2: a. Supine MVC, b. Standing MVC, c. Cough. The filled squares indicate the mean and the bars indicate the 95% confidence interval of the mean. The solid lines indicate the best fit regression, and the dashed lines indicate the 95% confidence interval for the regression line.
3.5 Discussion

The results of this study suggest that changes in both anterior vaginal pressure and posterior vaginal pressure are good surrogate measures for changes in urethral pressure measurements, particularly during coughing tasks. The results suggest that IO and PFM activity make clear contributions to urethral pressure generation. They also suggest that while women use contributions from RA and EO to generate urethral pressure, the motor patterns employed during voluntary PFM contractions vary greatly among individuals.
3.5.1 Sample

This study included parous women and women with symptoms of SUI and urgency, which may mean that the results are not representative of the optimal relationship between urethral pressure and vaginal pressure which would be found in nulliparous, continent women. However, when the data were plotted by UDI score, women with higher UDI scores did not produce data that were outliers compared to the women with UDI scores of 0 to 2. As well, by including women with incontinence the results of this study may be more generalizable to women seeking exercise treatment for incontinence. Further research studies using larger sample sizes to compare women with and without incontinence would be necessary to determine if the relationship between change in urethral pressure and change in vaginal pressure is the same irrespective of diagnosis.

3.5.2 Peak Pressure Amplitudes

As expected based on Lose et al., women generated greater increases in urethral pressure during coughing than during the PFM MVCs. Women were equally able to generate increases in urethral pressure in supine and standing. Also as expected, baseline urethral pressure was higher in standing than it was in supine, which accounts for there being no difference in peak urethral pressure between the two testing positions. Although lower baseline pressure may be a factor in urinary incontinence, it was subtracted from the absolute values in order to isolate the contributions of the urethral sphincters and the PFMs to the continence mechanism.
There was no difference in peak increase in vaginal pressure among the three tasks, which is consistent with our previous work\textsuperscript{256} and the results of Constantinou et al.\textsuperscript{225,227} The location of the pressure transducers within the vagina (anterior or posterior) appears to be of no consequence on experimental results when peak pressures are recorded, which is again consistent with Constantinou et al.\textsuperscript{225,227}

The rise in peak urethral pressure was much higher in amplitude than were the rises in peak vaginal pressures. This is consistent with the literature\textsuperscript{139,212,233} and it may describe a real difference in pressure between the urethra and the vagina, which occurs at least partially because urethral pressure includes contributions from the urethral sphincters that do not contribute to vaginal pressure.\textsuperscript{87} It may also be related to using different pressure recording systems in the urethra and the vagina, since the pressure recorded is a function of the surface area of the transducer.\textsuperscript{214,257} The relationship between the peak increase in urethral pressure and the peak increase in vaginal pressure was linear for both vaginal pressure recording sites and all three tasks indicating that the peak increase in vaginal pressure is a valid surrogate for the peak increase in urethral pressure during both PFM MVCs and coughing.

3.5.3 Repeated contractions

The relationship between the variations urethral pressure and the variations in vaginal pressure was linear for both vaginal pressure recording sites throughout the repeated PFM contractions in both supine and standing, indicating that vaginal pressure tracked the rises and falls in urethral pressure.
The $r^2$ values ranged from approximately 0.33 to 0.49, which indicates that less than half of the variability in the change in urethral pressure was accounted for by the changes in vaginal pressure. This may be because the urethral pressure often did not return to its initial baseline during each relaxation, rather the baseline rose with each relaxation, while the vaginal pressure did return to its initial baseline between contractions. This may be due to contributions to the urethral pressure from the urethral smooth muscle, which has a longer relaxation time.\textsuperscript{87} In subsequent studies it might be useful to allow longer delays between PFM contractions to allow the smooth muscle urethral sphincter time to relax between contractions.

3.5.4 Relationship between vaginal pressure and urethral pressure and the contributions of PFM and abdominal muscle activation to urethral pressure

There was no difference in the ensemble averaged urethral pressure rise versus anterior and posterior vaginal pressure rise curves for any of the three tasks. The location of the pressure transducers within the vagina (anterior or posterior) does not appear to matter when vaginal pressure generation is used as a surrogate for urethral pressure generation.

The urethral pressure rise versus vaginal pressure rise ensemble average curves during the PFM MVCs all have y-intercepts that are greater than zero indicating that vaginal pressure generation began before urethral pressure generation. This delay may be the result of the time required after the initiation of the PFM contraction to take up slack in the pelvic tissues before pressure can be transmitted to the urethra.\textsuperscript{87, 258} The y-intercepts for the urethral pressure rise versus vaginal pressure
rise curves generated during coughing were close to zero, indicating that there was less of a delay between the initiation of vaginal pressure and the initiation of urethral pressure rises. This lower intercept in the coughing data as compared to the voluntary contraction data may be related to the stereotypical motor patterns employed during coughing. Coughing presumably requires a more powerful contraction of the PFMs; such an abrupt activity may first recruit fast-twitch fibres of the PFMs, and as such, any slack in the paravaginal tissues would be taken up more quickly than it would be during voluntary contractions which would recruit the PFMs in a more orderly fashion.\textsuperscript{259, 260} The curves do not reach 100\% on the $y$-axis because of dispersion: the individual volunteers’ curves reached 100\% at different points on the urethral pressure axis.

Vaginal pressure rise is more closely correlated with urethral pressure rise during coughing than during PFM MVCs. This again may be due to differences in neuromuscular activation and/or motor control. Because coughing produces involuntary contractions which may follow stereotypical muscle activation patterns, there is likely less variability in PFM fibre recruitment order; whereas, not only are PFM MVCs voluntary, demonstrating greater variability in the muscle activation patterns used to generate them, they are not a practiced skill and, therefore, women do not likely have fixed motor programs.\textsuperscript{37, 261} The variation in the contributions of the RA and EO muscles to urethral pressure during the voluntary contractions support this theory. There was no clear pattern of activation in these muscles, unlike the PFMs and IO, which demonstrated significant curvilinear relationships when plotted against the urethral pressure rise.
Dispersion played a large role in the urethral pressure rise versus PFM EMG and urethral pressure rise versus abdominal EMG ensemble average curves. With regard to RA and EO, muscle activation timing differed so markedly among the women that the slopes of the ensemble average curves during the PFM MVCs were flat even though there was a distinct rise in EMG activation for each muscle on the individual curves.

3.6 Conclusions

While it must be recognized that urethral pressure includes components generated by the urethral sphincters and passive tissue stiffness that do not contribute to vaginal pressure, this study has demonstrated that changes in vaginal pressure recorded adjacent to both the anterior and posterior vaginal walls are representative of changes in urethral pressure. As such vaginal pressure change can be used as a surrogate for urethral pressure change measures in biomechanical studies of the continence system and as a functional outcome measure in physiotherapy treatment incontinence.
Chapter 4 Women with SUI Demonstrate Motor Control Differences during Voluntary Pelvic Floor Muscle Contractions

4.1 Abstract

Introduction and Hypothesis: To compare maximum abdominal and pelvic floor muscle (PFM) electromyographic (EMG) and vaginal pressure (VP) amplitudes, and muscle activation patterns during voluntary PFM contractions between women with and without stress urinary incontinence (SUI). Methods: Twenty-eight continent women and 44 women with SUI performed single and repeated PFM contractions in supine. Surface EMG data were recorded simultaneously with VP. Maximum EMG and VP amplitudes and ensemble average VP versus EMG curves were determined from the single contractions. Muscle activation timing was determined with cross-correlation functions from the repeated contractions. Results: The continent group produced higher PFM EMG amplitudes than the SUI group; there were no between group differences in VP. The women with SUI demonstrated delays in rectus abdominis activation. The VP versus EMG curve shapes were similar between the groups, however, the SUI group had higher abdominal muscle y-intercepts than the continent women. Conclusions: These findings suggest that women with SUI demonstrate altered motor control strategies during voluntary PFM contractions.

4.2 Introduction

The pelvic floor muscles (PFMs) have a baseline level of tonic activity that only disappears during voiding, plus they increase their contraction phasically during
increases in intra-abdominal pressure\textsuperscript{122} and critical urgency.\textsuperscript{120,125} Tonic PFM activity supports the pelvic organs, whereas phasic activity assists in occluding the urethral lumen by providing a firm surface against which the urethra can be compressed during increases in intra-abdominal pressure to provide urinary continence.\textsuperscript{120,262}

Electromyography (EMG) is a means of measuring the PFMs ability to contract. Increasing EMG amplitude is correlated with increasing force, however, EMG amplitude cannot be used to quantify the force of the contraction.\textsuperscript{219} Vaginal pressure measurements may be more valuable in quantifying the force of a PFM contraction, but are not able to isolate the contribution of the PFMs to this system.\textsuperscript{219} When recorded simultaneously, PFM EMG and vaginal pressure provide a more complete picture of how effectively this part of the continence mechanism is functioning than either measure can provide alone.

The associations among PFM EMG activation, vaginal pressure and continence status are unclear. Some authors have found that, during voluntary PFM contractions, continent women have higher PFM EMG activation amplitudes and/or vaginal pressures than do women with SUI,\textsuperscript{128,131} while others have found no differences between continent and stress incontinent women.\textsuperscript{196} Moreover, it is not known whether continence is achieved through PFM exercise therapy by returning the continence mechanism to its previous state or through training the PFMs to compensate for other deficits,\textsuperscript{42} which could require stronger PFMs than are usual in women who have not experienced urine leakage. As well, the PFM deficits seen in
women with SUI may be different depending on the severity of the leakage\textsuperscript{147} and/or on the underlying pathophysiology.\textsuperscript{85}

Synergistic relationships among the PFMs and abdominal muscles are thought to exist in order to allow women to maintain urinary continence in situations of elevated intra-abdominal pressure and co-activation of the abdominal muscles appears to contribute to the generation of vaginal pressure in urinary continent women through the transfer of intra-abdominal pressure to the vagina.\textsuperscript{140,263} In continent women, a rise in intra-abdominal pressure is preceded by contraction of the PFMs\textsuperscript{127} and the striated external urethral sphincter,\textsuperscript{37} while the failure of the PFMs to adequately resist increases in intra-abdominal pressure is thought to underlie the pathology of SUI.\textsuperscript{262} Women with incontinence are less likely than continent control subjects to demonstrate a coordinated pattern of PFM and abdominal muscle contraction during coughing,\textsuperscript{264} defined as upward movement of the perineum and contraction of the deep abdominal muscles. In a previous study on urinary continent women, it was shown that the abdominal muscles were activated during maximum voluntary PFM contractions performed in supine, and that the activation of the external oblique muscles preceded the simultaneous activation of the PFM, rectus abdominis, internal obliques and transversus abdominis by 27 ±60 ms.\textsuperscript{139} A comparison of the activation timing between women with and without SUI might elucidate motor control deficits that contribute to urine leakage in women.

The author has also found that, during a voluntary PFM contraction, the initial vaginal pressure rise is generated by the combined activity of the PFMs and the
abdominal muscles. The subsequent rise in vaginal pressure was associated primarily with the PFMs until the pressure reached approximately 70% of its maximum. The final rise in vaginal pressure was generated almost exclusively by the abdominal muscles. Understanding the nature of the abdominal-PFM synergy is particularly important given that increases in intra-abdominal pressure associated with abdominal muscle contraction appear on the one hand to be responsible for and, on the other hand, to prevent the urine leakage seen in SUI.

The objectives of this study were: (I) to compare maximum PFM and abdominal muscle EMG, as well as proximal and distal vaginal pressure amplitudes among women with no, mild and moderate to severe SUI during voluntary PFM contractions, and (II) to determine if the abdominal and PFM activation patterns and their relationships with distal vaginal pressure generation differ among women with and without SUI.

4.3 Materials and Methods

Ethics approval for the study was received from the Queen’s University Health Sciences Research Ethics Board (REH-184-03), and each volunteer provided informed consent prior to participating (See Appendix C). Continent and stress incontinent women aged 21 to 70 years who were not pregnant, had not given birth in the previous twelve months, and who were in good general health were invited to participate. Potential participants were excluded if they had undergone previous gynaecological or continence surgery, had genital prolapse greater than POP-Q stage 2, had intrinsic sphincter deficiency, had a history of diabetes, neurological conditions, autoimmune connective tissue disorders, or used any medications to
treat or known to exacerbate urinary incontinence. Continental women were included if they had no self-reported history of incontinence and had no incontinence episodes recorded on a three-day bladder diary.

Volunteers were recruited via newspaper advertisements. They were initially screened for inclusion by telephone and then attended a screening session with a registered nurse. During the screening session, women provided baseline demographic data and were assessed for prolapse, intrinsic sphincter dysfunction and the ability to perform a PFM contraction. Women who could not perform a palpable PFM contraction with visible cephalad movement of the perineum were excluded. All women who met the inclusion criteria were provided with a three-day bladder diary. (See Appendix D.) The women who had histories that fit the criteria for SUI underwent confirmational urodynamic testing.

The EMG and pressure testing occurred approximately one week after screening and data were acquired by a researcher blinded to group assignment.

4.3.1 Instrumentation

A custom modified Femiscan™ (Mega Electronics Ltd. Kuopio, Finland) surface EMG probe was designed to simultaneously measure PFM EMG and proximal and distal vaginal pressure. (See Figure 3.1.) The pressure sensors were constructed using 10 French rectal balloon catheters (Laborie Medical Technologies, Mississauga, Ontario, Canada) and were air filled. Proximal vaginal pressure was used as a surrogate measure of abdominal pressure and distal vaginal pressure was measured at the level where the PFMs attach to the vagina.
Surface EMG data were acquired from the abdominal muscles using Delsys™ DE-2.1 surface electrodes (Boston, Massachusetts). The electrodes were oriented along the line of action of the underlying muscle fibres on the left side and centred at the following locations: rectus abdominis (RA) 2 cm lateral and 2 cm caudal to the umbilicus, external obliques (EO) over tip of the eighth rib and internal obliques (IO) 2 cm proximal to the midpoint of a line from the anterior superior iliac spine to the symphysis pubis. A common reference electrode was located over the left anterior superior iliac spine. Crosstalk among pairs of muscles was found to be minimal in a previous study and the reliability of EMG and pressure data collected with the custom Femiscan™ probe has been shown to be adequate.

All EMG electrodes were interfaced with a Delsys Bagnoli™ 16-channel EMG amplifier system (band pass filter 20-450 Hz, CMRR 92 dB at 60 Hz, input impedance >10 POhm, gain 1000) and a 16-bit National Instruments analog to digital (AD) converter (NIDAQ PCI-MIO-16XE-10). The pressure transducers were interfaced directly with the AD converter. The pressure and EMG data were acquired simultaneously at 1000 Hz and stored on a personal computer using EMGworks® Acquisition software (Delsys Inc. Boston, Massachusetts). Impulse response testing of the pressure transducers revealed that there was a five ms delay in the pressure recordings, which was most likely the result of transmission delays caused by using air as the transmission medium. This delay was removed from all pressure data prior to data processing and analysis.
4.3.2 Data collection

Upon arrival, the volunteer returned her completed bladder diary in a sealed envelope. After instruction, each volunteer was left in the private laboratory area to insert the probe into her vagina; the researcher visually verified the probe’s position afterward. With the probe in situ, the volunteer performed PFM contractions while pressure and EMG data were acquired. These data were used to provide feedback to achieve a correct PFM contraction. A correct PFM contraction involved squeezing around the probe with visible cephalad movement of the perineum and without evidence of breath holding or palpable contraction of the hip or gluteal muscles. A rise in the peak pressure and EMG data that was at least five times higher than the baseline level was indicative of a correct PFM contraction and acceptable signal fidelity. Data collection began once the subject could achieve such a result. Each volunteer performed as many practice contractions as were needed to achieve a correct PFM contraction.

All data were recorded with the volunteers in supine: pillows were placed under the volunteers’ heads and knees and their hips were slightly abducted. The subjects performed three maximal voluntary isometric contractions (MVCs) of each of the abdominal muscles tested in supine. The RA contraction involved pushing maximally into a webbing strap fastened across the shoulders and around the plinth (i.e. resisted abdominal crunch exercise). The IO and EO MVCs involved repeating the crunch exercise and adding ipsilateral and contralateral trunk rotation respectively. Five seconds of baseline (resting) data were acquired from all sensors while the subjects attempted to keep the hip, pelvic and abdominal muscles
completely relaxed. The volunteers then performed three repetitions of a maximum PFM contraction and of a 30 s series of repeated maximum PFM contractions at a comfortable pace with complete relaxation between contractions. The participants all performed the manoeuvres in the above order.

4.3.3 Data processing and analysis

4.3.3.1 Group assessment and sample

The severity of urine leakage in the women with SUI was determined from the three-day bladder diaries. The total number of leakage episodes was divided by three to obtain the mean number of leakage episodes per day. Ingelman-Sundberg’s\textsuperscript{269} criteria for classifying the severity of SUI were used to categorize the intensity of provocation required to cause urine leakage. Women who only had leakage during intense physical activity received a score of one, those who had leakage during sneezing, coughing or laughing received a score of two and those who had leakage with very light activity: sit to stand, walking or bending over, received a score of three. The severity score was based on the mean number of leakage episodes per day multiplied by the minimum stress that provoked a leakage episode. Women with a severity score of one to two were classified as mild, while women with a severity score of three to five were classified as moderate and women with a score of six or greater were classified as severe. The amount of leakage experienced by the volunteers during their daily activities was not considered in the severity score as no objective measures of urine loss were made and the estimations of the volume of leakage on the bladder diaries were not standardized.
One-way analyses of variance (ANOVA) were used to test for differences among the groups in terms of age, BMI, number of deliveries (parity) and number of vaginal deliveries ($\alpha=0.05$). The Bonferroni method was used to conduct post hoc tests when main effects were found. $\chi^2$ tests were used to test for differences among the groups in menopausal status and the number of nulliparous women in the groups ($\alpha=0.05$). When differences among the groups were found, $\chi^2$ tests between each pair of groups were used to conduct post hoc tests ($\alpha=0.016$, to control for multiple comparisons).

**4.3.3.2 EMG activation levels**

Pelvic floor muscle EMG data were acquired bilaterally; however, the two sides did not always show equal signal amplitude. The two sides were, however, highly correlated (cross-correlation coefficient=0.90, 95% confidence interval: 0.89 to 0.92) and synchronous (time lag=0 ms). Therefore, only data from the side with the larger signal amplitude were included in the statistical analyses. Although unilateral PFM defects are also common, side-to-side differences in signal amplitude are likely due to the rigid nature of the probe allowing it to make optimal contact with only one vaginal wall and underlying pubovisceral muscle at any time.

To determine the relative levels of muscle activation among the groups, the EMG data were smoothed by computing root mean square (RMS) amplitudes using a 200 ms moving window which was incremented across each data set by one millisecond steps. Resting data were subtracted from each data file to remove any bias (differences in electrode impedance, charge build up, et cetera), instrumentation
noise and tonic PFM activity. The highest unbiased and smoothed EMG value was considered to be the maximum voluntary electrical activity (MVE) attained for a given muscle during a given contraction. The data were normalized by dividing the peak EMG activity for each abdominal muscle during the PFM contraction by the highest MVE computed for that muscle during the three repetitions of its MVC. A two-way repeated measures ANOVA was used to test for differences in maximum EMG activity among the groups using group and muscle recording site as factors. The peak values from all three repetitions were used in the ANOVA and the interaction was considered in the model (α=0.05). The Bonferroni method was used to conduct post hoc tests when significant interactions or main effects were found. To ensure that any differences among the groups were not due to significant differences in demographic factors (e.g. age or BMI), the EMG amplitude data were analyzed using that demographic factor as a covariate.

4.3.3.3 Vaginal pressure amplitudes

To determine the relative differences in vaginal pressure amplitude among the groups, the pressure data were smoothed by computing RMS amplitudes using a 200 ms moving window, as described for the EMG data. Resting data were subtracted from each data file to remove any bias (differences in balloon pre-inflation level, et cetera), instrumentation noise and resting vaginal pressure. The peak RMS values for both proximal and distal vaginal pressure were not normalized, but were converted from voltages to centimetres of water (cmH₂O) (0.04 V =1 cmH₂O).
One-way ANOVAs were used to test for differences in maximum pressure at each pressure-recording site (∝=0.05); the peak pressure values from the three repetitions were included in the analysis. The Bonferroni method was used to conduct post hoc tests when significant main effects were found.

4.3.3.4 Muscle activation timing

In order to determine the activation patterns of the abdominal muscles relative to the PFMss, the data from the 30 s series of repeated contractions were used. The number of contractions performed during each series was determined by counting the number of PFM EMG bursts during the 30 s. To be included both the rise and the fall of the burst had to be complete within the recording window. The EMG data were full wave rectified and filtered (forward and back) with a third order low-pass Butterworth filter with a five Hz cut off. Cross correlation functions were used to compare the PFM EMG signals to the EMG signals recorded from each abdominal muscle. The time lags of each muscle burst relative to the PFM burst were used as outcome measures. Two-way repeated measures ANOVAs were used to test differences among the groups, with group and muscle as factors. The interaction was considered in the model. The Bonferroni method was used to conduct post hoc tests when significant interactions or main effects were found.

4.3.3.5 Relationship between vaginal pressure generation and EMG

To describe the relationship between distal vaginal pressure and muscle activation, ensemble average pressure versus EMG curves were created using the individual trials. Specifically, the EMG activity of each muscle was plotted against the
distal vaginal pressure generated during each repetition of the PFM MVC. The data were full wave rectified and filtered as described above. The rise in distal vaginal pressure was divided into five percent increments and then the amplitude of the smoothed EMG signal was determined at each distal vaginal pressure increment. The EMG data were normalized to the maximum smoothed value attained by that muscle during that PFM contraction. Similar curves were generated to investigate the relationship between distal and proximal vaginal pressure.

The ensemble average distal vaginal pressure versus EMG curves for each muscle and each continence group were tested for significance (α<0.05) using runs tests (GraphPad Prism™ v.4 software, La Jolla, California). The patterns demonstrated by the groups were then compared. The curves were considered to be significantly different if the equations were linear and the 95% confidence intervals for the intercepts or slopes did not overlap, or if different order equations produced a best fit. The same method was used to test and compare the ensemble average distal versus proximal vaginal pressure curves.

The timing of the proximal vaginal pressure peak relative to the distal vaginal pressure peak was determined by subtracting the time of the peak proximal vaginal pressure during the PFM MVC from the time of the peak distal vaginal pressure during the PFM MVC. Positive values indicate that the proximal vaginal pressure peak occurred after the distal vaginal pressure peak.
4.4 Results

4.4.1 Sample

One hundred and forty women were screened for this study. Sixty-eight women were excluded from the study for various reasons. (See Table 4.1.)

Table 4.1 Number of volunteers excluded by each criterion.

<table>
<thead>
<tr>
<th>Reason for Exclusion</th>
<th>Number of Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Declined for personal or scheduling reasons</td>
<td>22</td>
</tr>
<tr>
<td>Prior pelvic organ surgery</td>
<td>14</td>
</tr>
<tr>
<td>Medication on exclusion list</td>
<td>12</td>
</tr>
<tr>
<td>Neurological deficits found on screening</td>
<td>2</td>
</tr>
<tr>
<td>Prolapse &gt; POP-Q stage II</td>
<td>3</td>
</tr>
<tr>
<td>Unable to perform a PFM Contraction</td>
<td>5</td>
</tr>
<tr>
<td>Detrusor instability of urodynamic testing</td>
<td>1</td>
</tr>
<tr>
<td>Bladder diary not consistent with SUI</td>
<td>9</td>
</tr>
<tr>
<td><strong>Total:</strong></td>
<td><strong>68</strong></td>
</tr>
</tbody>
</table>

Seventy-two women participated in the experimental protocol, 28 were continent and 44 had SUI. Among the women with SUI, 33 had mild SUI, seven had moderate SUI and four had severe SUI. The women with moderate and severe SUI were grouped together (n=11) because they displayed similar patterns of distal vaginal pressure generation in the vaginal pressure versus EMG curves and to improve statistical power. The women in all three groups were of similar age (p=0.083). There were significant differences among the groups in BMI and parity: both groups of women with SUI had larger BMIs than the continent women (p=0.03) and more of the women with SUI were parous ($\chi^2 = 11.95$ p=0.001). (See Table 4.2.)
Table 4.2 Demographic data by continence status.

<table>
<thead>
<tr>
<th></th>
<th>Continent (n=28)</th>
<th>Mild SUI (n=33)</th>
<th>Severe SUI (n=11)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td>46.8 (7.6)</td>
<td>46.5 (7.4)</td>
<td>52.8 (8.4)</td>
</tr>
<tr>
<td><strong>Body Mass Index (kg/m²)</strong></td>
<td>24.3 (3.4)</td>
<td>27.0 (5.3)*</td>
<td>28.4 (5.7)*</td>
</tr>
<tr>
<td><strong>Menopause Status</strong></td>
<td>pre: 17, peri: 4, post: 7</td>
<td>pre: 16, peri: 8, post: 9</td>
<td>pre: 5, peri: 1, post: 5</td>
</tr>
<tr>
<td><strong>Parous</strong></td>
<td>17/28</td>
<td>31/33**</td>
<td>11/11**</td>
</tr>
<tr>
<td><strong>Parity (#)</strong></td>
<td>1.4 (1.2)</td>
<td>2.1 (1.1)</td>
<td>1.9 (0.6)</td>
</tr>
<tr>
<td><strong>Vaginal Deliveries (#)</strong></td>
<td>1.25 (1.1)</td>
<td>1.9 (1.1)</td>
<td>1.8 (0.8)</td>
</tr>
<tr>
<td><strong>Leakage Episodes (#)</strong></td>
<td>0 (0)</td>
<td>4.6 (4.3)</td>
<td>11.1 (5.8)+</td>
</tr>
</tbody>
</table>

Continent: continent group, Mild SUI: mild SUI group, Severe SUI: moderate to severe SUI group, BMI: body mass index, pre: premenopausal, peri: perimenopausal, and post: postmenopausal. All of the data are given as the mean and (standard deviation) except Menopause Status and Parous, which are the number of women in the group. * indicates a significant difference compared to the continent group (p<0.05). ** indicates a significant difference compared to the continent group (p<0.016, to control for multiple comparisons). + indicates a significant difference compared to the group with mild SUI (p<0.05).

4.4.3 EMG Activation Levels

The women with moderate to severe SUI produced higher MVEs during the abdominal MVCs than did either of the other groups (p<0.001). As such it was felt that normalization to the MVE would bias the results; therefore, subsequent analyses were performed with non-normalized data using a nested model (subject nested in group) to account for inter-individual differences in EMG amplitude among subjects.

In the analyses of covariance, the group by BMI interaction was not significant for any of the muscles (PFM p=0.19, RA p=0.37, EO p=0.67 and IO p=0.09), nor did parity affect the EMG amplitudes (p=0.63). For the EMG data recorded during the maximal PFM contraction, the interaction between group and muscle was not significant (p=0.28). The main effects for group and muscle were significant (p=0.002 and p<0.001 respectively). The continent group produced higher PFM
EMG amplitudes than both of the groups with SUI, and higher EO and IO EMG amplitudes during maximum PFM contractions than the women with moderate to severe SUI. The women with mild SUI produced higher EO EMG amplitudes than did the women with moderate to severe SUI. (See Figure 4.1.)

Figure 4.1 Maximum EMG amplitudes by group and muscle. C: continent group, M: mild SUI group, S: moderate to severe SUI group, PFM: pelvic floor muscles, RA: rectus abdominis, EO: external obliques and IO: internal obliques. The bars indicate the mean and the whiskers indicate the 95% confidence interval for the mean. The * indicate significant differences between groups (α<0.05).

4.4.3 Vaginal pressure amplitudes

There were no significant differences among the groups in distal or proximal vaginal pressure amplitudes recorded during the maximum voluntary PFM contraction (p=0.21 and p=0.48 respectively). (See Figure 4.2.)
Figure 4.2 Maximum pressure amplitudes by group: a. distal vaginal pressure and b. proximal vaginal pressure. Continent: continent group, Mild SUI: mild SUI group, Severe SUI: moderate to severe SUI group. There were no significant differences among the groups for either pressure recording site.

4.4.4 Muscle Activation Timing

The continent women performed a mean of 16 (±10) contractions in the 30 s series of PFM contractions, while the women with mild SUI performed 13 (±7) and the women with moderate to severe SUI performed 10 (±3). These differences in the number of contractions performed in 30 s were not significantly different among the groups (p=0.12).

There was a significant interaction between group and muscle for the time lags between PFM activation and abdominal muscle activation, indicating that the groups displayed different muscle activation patterns (p=0.015). In the continent women IO was activated 14 (SD 35) ms prior to the activation of the PFM, RA and EO. In the women with mild SUI all of the muscles were activated simultaneously. In the women with moderate to severe SUI the PFM, EO and IO were activated simultaneously followed by RA being activated 40 (SD 66) ms afterwards. The RA time lag was the only one that was significantly different among the groups (p<0.001 for both continent versus moderate to severe SUI and mild SUI versus
moderate to severe SUI). (See Figure 4.3.) The data were tested and found to be normally distributed using the Anderson-Darling Normality test.

Figure 4.3 EMG cross-correlation time lags between the abdominal muscles and the PFMs. Continent: continent group, Mild SUI: mild SUI group, Severe SUI: moderate to severe SUI group, RA: rectus abdominis, EO: external obliques and IO: internal obliques. Negative numbers indicate that the EMG burst occurred prior to the PFMs’ EMG burst and positive numbers indicate that the EMG burst occurred after the PFMs’ EMG burst. The bars indicate the mean and the whiskers indicate the 95% confidence interval for the mean. * indicate time lags that are significantly different between groups, + indicate time lags that are significantly different from the PFMs.

4.4.5 Relationship Between Vaginal Pressure Generation and EMG

The ensemble average distal vaginal pressure versus EMG curves for each group are represented in Figure 4.4. Each of the curves has an intercept that is greater than zero, which is consistent with the expected electromechanical delay (60-120 ms in slow twitch muscle\(^\text{270}\)) in which a muscle becomes electrically active before measurable force is generated. As well, although each volunteer’s curve for each muscle did attain 100% EMG activation, different curves reached their peak EMG activation at different percentages of their maximum pressure: therefore the
averaged curves do not reach 100% on the EMG axis. There was no difference in the percentage of maximum pressure at which the groups reached their maximum EMG activation (p=0.22).

Figure 4.4 Distal vaginal pressure versus EMG and proximal vaginal pressure ensemble average curves by group: a. distal vaginal pressure versus PFM EMG, b. distal vaginal pressure versus internal obliques (IO) EMG and c. distal vaginal pressure versus proximal vaginal pressure. Both EMG and pressure are given as a percentage of the maximum achieved during the contraction. Continent: continent group, Mild SUI: mild SUI group, Severe SUI: moderate to severe SUI group, The shapes indicate the mean and the whiskers indicate the 95% confidence intervals of the mean. The curves for IO were typical of the curves for all of the abdominal muscles tested.

The distal vaginal pressure versus PFM EMG curves were best described in the continent group by a linear equation, while in the two groups of women with SUI the PFM curves were best described with second order polynomial equations. However,
on visual inspection, the curves did not appear to be substantively different and the second order equations were not notably better at predicting the total variance in the model in the women with SUI (R² values less than 4% greater in the 2nd order equations). Therefore, in order to compare the curves among the groups, linear equations were used so that the slope and intercept coefficients could be compared. (See Figure 4.4a.) Using the linear equations, the intercepts and the slopes of the lines were not significantly different among the groups (p=0.11 and p=0.59 respectively).

The curves describing distal vaginal pressure relative to EMG activation for RA, EO and IO were best described by linear equations in all three groups. (See Figure 4.4b for the IO curves.) For RA, there were no significant differences among the three groups for either the intercepts or the slopes of the regression lines (p=0.34 and p=0.27 respectively). For EO, there were no differences in the intercepts of the regression lines between the continent and the mild SUI groups (p=0.17), however, the intercept was higher in the moderate to severe group than in the continent group (p=0.001) and the mild SUI group (p=0.044), indicating that EO was active to a larger degree by the time pressure began to rise in the women with moderate to severe SUI than it was in the other two groups. There were no differences in the slopes of the regression lines among the groups (p=0.28). For IO, the intercepts of the regression lines were higher in the mild and the moderate to severe SUI groups than in the continent group (p<0.001 for both), but there were no significant differences between the mild and the moderate to severe SUI groups (p=0.43). The slope of the regression line was lower in the moderate to severe SUI group than in
either the continent group (p=0.001) or the mild SUI group (p=0.037). There was no difference between the continent group and the mild SUI group (p=0.14), indicating that in the women with moderate to severe SUI there was less contribution from IO to the increase in distal vaginal pressure than in either of the other groups.

The curves describing distal vaginal pressure relative to proximal vaginal pressure were best described by a second order polynomial equation in the continent group and by linear equations in the groups with mild and moderate to severe SUI. Again, linear equations were used to compare the groups because, for the continent women, the second order equation was not notably better than the linear equation at predicting the curve’s variability (R^2 <2% larger in the second order equation). (See Figure 4.4c.) All of the resulting regression lines had y-axis intercepts that were near or below zero because on most trials there was a drop in proximal vaginal pressure below baseline as the distal vaginal pressure began to rise. This intercept was significantly lower in the group with moderate to severe SUI compared to the group with mild SUI (p<0.05) indicating a larger drop in proximal vaginal pressure prior to the rise in distal vaginal pressure. However, there were no significant differences in the intercepts between the continent group and either of the other groups (p>0.05 for both). There was no difference in the slopes of the curves among the groups (Continent: 0.2815, ±0.2734, Mild SUI: 0.2347, ±0.2332, Moderate to Severe SUI: 0.3734, ±0.2480, p>0.05) indicating that the increase in proximal vaginal pressure relative to the distal vaginal pressure was similar in the three groups.

The peak proximal vaginal pressure occurred after the peak distal vaginal
pressure in all three groups, which is why the distal versus proximal vaginal pressure curves do not reach 100% on the y-axis. The proximal vaginal pressure peak occurred more quickly after the distal vaginal pressure peak in the group with mild SUI than it did in the other two groups (proximal vaginal pressure peak timing relative to distal vaginal pressure peak timing: Continent: 1126 ±1615 ms, Mild SUI 753 ±1896 ms, Moderate to Severe SUI 1169 ±1240 ms, p=0.049). Proximal vaginal pressure was used as a surrogate measure for intra-abdominal pressure, so this demonstrates that intra-abdominal pressure continued to rise after distal vaginal pressure had begun to fall during a voluntary PFM contraction and that intra-abdominal pressure peaked sooner in the women with mild SUI than in the other two groups.

4.5 Discussion

This study allowed the investigation of the relationships between PFM and abdominal muscle activity during voluntary PFM contractions in women with and without SUI. Through recording EMG and vaginal pressure simultaneously, it was possible to describe the muscle activation strategies that are used by continent and stress incontinent women to generate vaginal pressure, which provides a richer portrait of the extrinsic continence mechanism than can be seen through either pressure measures or EMG activity alone.

It can be hypothesized that if SUI in women is due to PFM weakness, then women with SUI would display lower PFM activation and lower distal vaginal pressure amplitudes than a control group of continent women. If SUI is due to fascial
deficits, then PFM EMG activation amplitudes may be the same or higher than those seen in continent women but vaginal pressure amplitudes would be lower, as the PFMs would be trying to compensate for their mechanical disadvantage. Thirdly, SUI may be due to altered motor control, not evidenced by any differences in PFM EMG or vaginal pressure amplitudes but by differences in muscle activation and vaginal pressure generation patterns and timing. The findings of this study suggest that a combination of muscular weakness, altered fascial support and altered motor control are associated with SUI. The theory that women with SUI demonstrate PFM weakness is supported by the finding of lower maximum PFM EMG amplitudes in the women with SUI; however, the finding that the women with SUI had similar distal vaginal pressure amplitudes to the continent women suggests that the women with SUI used different muscle activation patterns to generate this pressure. Altered fascial support is suggested by the finding of higher EO and IO intercepts in the distal vaginal pressure versus EMG curves displayed by the women with SUI, which indicate that these abdominal muscles were active at a higher level before distal vaginal pressure began to rise, suggesting that the initial force produced by these muscles was absorbed by the slack in lax vaginal support structures instead of being transferred directly to the vagina. Furthermore, this study demonstrates altered motor control in the women with SUI; the finding that distal vaginal pressure was the same in the three groups despite the lower maximum PFM and abdominal EMG amplitudes in the women with SUI suggests that the women with SUI may have been using other mechanisms to generate this pressure. As well, the women with moderate to severe SUI displayed delayed RA activation and a larger drop in
proximal vaginal pressure as distal vaginal pressure began to rise, which also suggests altered motor control strategies.

This work broadens our understanding of how the PFMs work in synergy with the abdominal muscles in urinary continent and stress incontinent women. In particular and consistent with our previous work, it was determined that the abdominal muscles in continent women contribute significantly to the development of distal vaginal pressure during voluntary PFM contractions, as indicated by the rise in abdominal EMG activation concomitant with the rise in distal vaginal pressure. This synergy seems to be preserved in women with SUI, however, some of the abdominal muscles (EO and IO) were active at a greater percentage of their maximum before the distal vaginal pressure began to rise, suggesting that the transfer of intra-abdominal pressure to the vagina may be impaired in the women with SUI.

4.5.1 Sample

Five women who could not perform a voluntary PFM contraction were excluded from participating in this study. This was approximately seven percent of the number of women excluded and approximately three and a half percent of the total number of women who volunteered. As this was such a small percentage, it was unlikely to have biased the results. Only one study has reported on involuntary PFM activity in one continent woman who was unable to perform a voluntary PFM contraction; future research should evaluate involuntary PFM function in larger groups of women, both with and without SUI, who are unable to perform voluntary
PFM contractions and should determine whether these women are able to learn to perform voluntary PFM contractions with training.

The sample of women recruited to participate in this study was similar in age and menopause status but not in BMI or parity. An attempt was made to match continent and stress incontinent subjects by BMI and whether or not they were parous, however, the limitations this posed to recruitment were challenging to the point that it was abandoned. The parity and BMI distributions of the final sample reflect the reality that larger BMIs and parity are known risk factors for SUI. Increased adipose tissue can lower EMG amplitudes through tissue filtering; however, the women with SUI produced higher EMG amplitudes during the abdominal MVCs so tissue filtering was not likely to have produced any differences among the groups. Since the women with SUI produced higher abdominal EMG amplitudes during the abdominal MVCs these contractions were not used for normalization. Instead, a nested model (subject nested in group) was used in all ANOVAs to account for inter-individual differences in EMG amplitude among subjects. The EMG and pressure amplitudes were normalized to the maximum amplitude achieved during that PFM contraction to create the vaginal pressure versus EMG curves, which again eliminates tissue filtering as a possible source of bias.

4.5.2 EMG Activation Levels

The continent women produced higher maximum voluntary PFM EMG amplitudes than did either of the groups of stress incontinent women. They also
produced higher EMG amplitudes for EO and IO during the PFM MVCs than did the women with moderate to severe SUI. The women with mild SUI produced higher EO amplitudes than did the women with moderate to severe SUI. By grouping together women with moderate and severe SUI differences in EMG amplitudes may have been obscured; however, the statistical power would have been diminished if the group had been divided. Future research should include larger numbers of women with more severe SUI and should include incontinence severity as a covariate in the analysis to determine if EMG amplitudes vary with continence status. It is unlikely that the differences in EMG amplitudes were due to differences in BMI or parity as the effects of these factors were not significant in the ANCOVAs.

In contrast to our findings, Smith et al.\textsuperscript{147} found that seven women with mild SUI demonstrated higher PFM EMG amplitudes in response to a postural perturbation than did 14 continent women, while the PFM EMG amplitudes from nine women with more severe SUI were not significantly different from those produced by the continent women. This difference may be due to differences between the voluntary PFM contractions used in this study and the involuntary PFM contractions investigated by Smith et al.\textsuperscript{147} or the standing task they used might have resulted in crosstalk from the deep abdominals and hip muscles being recorded by their monopolar vaginal EMG probe. Involuntary PFM contractions may be accompanied by different synergistic muscle activity than are voluntary PFM contractions or the standing task may be accompanied by muscle guarding in the women with mild SUI. Further research is needed to understand the differences between involuntary and voluntary PFM contractions as situations that increase intra-abdominal pressure are
accompanied by automatic PFM contractions\textsuperscript{262} and perhaps larger differences would be found in involuntary PFM contractions between women with and without SUI than have been found with voluntary PFM contractions. However, as voluntary PFM contractions are used to prevent urine leakage during episodes of increased intra-abdominal pressure\textsuperscript{43} and as the basis of exercise treatment for SUI,\textsuperscript{42} it remains important to understand how voluntary PFM contractions function in women with SUI.

That the abdominal muscles contracted with a voluntary PFM contraction in all three groups of women is consistent with the idea that the PFM and abdominal muscles work in synergy, as has been described by others.\textsuperscript{140,263} A possible explanation for the higher PFM EMG amplitudes being produced by the continent women, while the distal vaginal pressure amplitudes were not different among the groups, is that the PFM EMG recordings from the continent women may have included crosstalk from other intrapelvic muscles, such as obturator internus. This is unlikely, however, since all the volunteers were screened before testing for their ability to correctly perform a PFM contraction and they were observed during the data recording to ensure that no movement occurred at the hips. In addition, in previous work it was found that maximally contracting the hip external rotators and the PFMs at the same time did not result in an increased PFM EMG signal compared to an isolated PFM MVC recorded with Femiscan™ vaginal probe,\textsuperscript{267} so again crosstalk was not a likely factor in this puzzling result. Another possible explanation is that the women with SUI used a motor control strategy that included other muscles surrounding the abdominal cavity to generate distal vaginal pressure. All of
the women in the study were instructed to continue breathing during the PFM contractions and trials were excluded if a volunteer was bearing down; however, perhaps there was some unobserved difference in muscle activation patterns.

4.5.3 Vaginal Pressure Amplitudes

In the current study, the rises in proximal and distal vaginal pressures generated during PFM contractions did not differ among the groups. Similarly, using a vaginal dynamometer Morin et al.\textsuperscript{196} found that there was no difference in the maximal force generating capacity of the PFMs in 30 continent women compared to 59 women with SUI. Our inability to find a clear association between PFM EMG amplitudes and distal vaginal pressure generation may be due to vaginal pressure not being sufficiently specific or sensitive to measure of the impact of PFM contraction on the continence mechanism; perhaps urethral pressure should be measured directly in future studies.

Only pressure changes induced with PFM contraction were measured in this study, as the baseline pressures were highly variable among the groups. Other research has suggested that resting pressure or absolute pressure is better able to distinguish between women with and without SUI.\textsuperscript{208,227,253} Future research should include measures of baseline pressure in comparisons between women with and without SUI.

4.5.4 Muscle Activation Timing

Different muscle activation patterns were found in each of the groups studied and these patterns were different from the pattern that was found in previously
with continent women. This difference, particularly as compared to the current continent group, may be due to the smaller sample in the earlier study which may have displayed an atypical pattern or it may have been due to familiarity with contracting the PFM, as the women in the earlier study were mostly physiotherapists or physiotherapy students with an interest in PFM rehabilitation, while the women in this study were drawn from the general population. This difference may be not be of clinical significance as, in both this sample and the previous one, continent women activated one of the abdominal muscles prior to the PFMs while the women with SUI in this study delayed the activation of their abdominal muscles.

The three groups of women performed a similar number of contractions during the 30 s series of PFM contractions. Conversely, the Morin et al. study discussed above found the continent women were able to perform more rapid, repeated PFM contractions over 15 s than were the women with SUI (10.3 ±3.9 versus 8.5 ±3.0, p=0.01). This difference is likely due to the instructions given to the study participants. Morin et al. instructed their volunteers to do as many PFM contractions as possible during the 15 s, while the volunteers in this study were instructed to contract and relax maximally at a comfortable pace.

To the author’s knowledge, this is the first study to describe the order of abdominal muscle activation in response to a voluntary PFM contraction in women with and without SUI; others have described abdominal and PFM activation orders in response to coughing and postural disturbances. Understanding the differences in muscle activation timing during voluntary PFM contractions between
women with and without SUI may be important to improving treatment programs: if women with SUI demonstrate altered motor activation patterns then treatment goals may need to include restoring the normal order of abdominal and PFM activation.

The differences in abdominal muscle activation timing displayed by the women with SUI are also of interest. The delayed RA activation displayed by the women with moderate to severe SUI compared to both the women with mild SUI and the continent women is the most striking difference and is consistent with the greater drop in proximal vaginal pressure at the beginning of the distal vaginal pressure rise that was observed in the women with moderate to severe SUI. Both the women with mild SUI and those with moderate to severe SUI delayed activating their abdominal muscles, perhaps to avoid generating intra-abdominal pressure before the PFMs are active and thereby avoiding urine leakage. Another possibility is that by delaying the abdominal muscle activation the women with SUI prevented optimal activation of the PFMs. Further research is necessary to determine the effect of this delay as it may have treatment implications: if the delay helps to prevent urine leakage then teaching women to keep RA relaxed during voluntary PFM contractions may be an effective treatment strategy, however, if the delay prevents the optimal activation of the PFMs then teaching the optimal muscle activation order may improve treatment outcomes.

The cross-correlation method used does not compute the difference in muscle activation onset timing but rather the difference in the timing of the entire EMG burst, which may make it difficult to compare the muscle activation timing reported
here to the activation orders reported by others.\textsuperscript{146, 147, 202, 271} However, this method was chosen because muscle activation onset timing detection is highly unreliable.\textsuperscript{272}

\subsection*{4.5.5 Relationship between vaginal pressure generation and EMG}

While the association between muscle activity and the generation of distal vaginal pressure was different between the continent group and the two groups of women with SUI, in all three groups PFM EMG was strongly correlated with the rise in distal vaginal pressure. This suggests that the PFMs were active in the generation of vaginal pressure throughout the pressure rise in all three groups. As well, the three groups of women produced very similar patterns of abdominal muscle activity in the generation of distal vaginal pressure. There were differences among the groups in the intercepts and the slopes of the pressure versus EMG curves for EO and IO. These results indicated that EO and IO required more EMG activation to show a rise in distal vaginal pressure. It is hypothesized that their contributions to intra-abdominal pressure generation may not have been transmitted to the vagina as effectively in the women with SUI perhaps because there was more slack in their vaginal support tissues which absorbed the pressure before it could be transmitted to the vagina. The lower slope for IO in the women with more severe SUI suggested that these women may have been holding back or avoiding the IO contraction, which may be an attempt to avoid urine leakage or may indicate a motor activation pattern that limits their ability to generate a PFM contraction.

The distal vaginal pressure versus proximal vaginal pressure curves were also different among the groups: with the continent women producing a curve that was essentially flat initially and then rose steeply during the latter part of the distal
vaginal pressure rise, where as in the women with moderate to severe SUI the proximal vaginal pressure rose linearly with the distal vaginal pressure. The women with mild SUI produced a curve that was visually between the shapes of the other two groups but that was best modelled with a linear equation. In the women with moderate to severe SUI the proximal vaginal pressure dropped below baseline as the distal vaginal pressure began to rise; this appears as negative proximal vaginal pressure on the graphs because baseline pressure is designated as zero pressure, not because negative pressure was produced in the abdominal cavity. This drop may indicate an attempt to avoid urine leakage by delaying generating intra-abdominal pressure until the PFM s were active. The peak in the proximal vaginal pressure occurred after the peak in the distal vaginal pressure in all three groups; however, it was earlier in the women with mild SUI compared to the other two groups. These findings suggest that the women with SUI were not able to use their abdominal muscles to generate pressure in the distal vagina as effectively as the continent women: perhaps because it was absorbed by laxity in the vaginal support structures or that they were avoiding using their abdominal muscles in an effort to avoid urine leakage. Alternately they may have been using altered motor control strategies to generate distal vaginal pressure during voluntary PFM contractions.

This study does not take into consideration the possibility that the diaphragm or other trunk muscles participated in the generation of vaginal pressure. Attempts were made to minimize the contribution of the diaphragm by instructing the participants to breathe normally throughout the PFM contractions and by excluding any trials during which either the breath was held or the volunteer bore down. This
should also have limited the contribution of the thoracic muscles. It is unlikely that the trunk extensor muscles played a significant role in the generation of vaginal pressure as they are primarily spinal stabilizers during postural tasks and the supine testing position reduced the demands on them.²⁷³

These findings may indicate an adaptive pattern of muscle activation in which the women have learned to optimize the effect of a PFM contraction by delaying and then not fully activating their abdominal muscles. It is also possible that this pattern is maladaptive and that the altered motor control strategy used to maintain the ability to generate distal vaginal pressure may increase the likelihood of urine leakage. Further research with direct measurement of urethral pressure is needed to confirm the effects of PFM and abdominal muscle contraction on urethral closure in order to determine whether this is an optimal or a maladaptive strategy.

All data recording in this study was performed in supine, which may not be generalizable to sitting or standing. However, no differences in maximum EMG or vaginal pressure amplitudes have been found by others between supine and standing during maximum voluntary PFM contractions,¹⁹³,²²⁴,²⁷⁴ which suggests that the differences between women with and without SUI found in this study may be applicable to the upright positions. Further research is necessary to confirm this.

4.6 Conclusions

This study found that the women with SUI generated similar vaginal pressures to the continent women despite having lower maximum EMG amplitudes for the PFMs, EO and IO during PFM MVCs, which suggests that the women with SUI used
different motor control strategies to generate vaginal pressure. This was further evidenced by the fact that muscle activation timing was different among the groups, with a delayed use of RA in the women with more severe SUI, and that there were higher $y$-intercepts for EO and IO on the distal vaginal pressure versus EMG curves of the women with SUI as compared to the continent women. The higher intercepts may also indicate that women with SUI were not able to transfer intra-abdominal pressure to the vagina as effectively as the continent women. The patterns of distal vaginal pressure versus proximal vaginal pressure generation were different among the groups, with the continent women showing greater contributions from the proximal vaginal pressure during the latter part of the distal vaginal pressure rise, and the women with SUI demonstrating more consistent contributions from the proximal vaginal pressure throughout the distal vaginal pressure rise. These findings do not present a clear and simple picture; however, they do suggest that women with SUI are less able to recruit the PFMs during voluntary PFM contractions than are continent women, but that they remain able to generate similar distal vaginal pressure amplitudes by using different motor control strategies.
Chapter 5 Women with Stress Urinary Incontinence Demonstrate Motor Control Differences during Coughing

5.1 Abstract

Introduction: This study compared the patterns of pelvic floor muscle (PFM) activity during coughing between women with stress urinary incontinence (SUI) and continent women, using surface electromyography (EMG) and vaginal pressure (VP). Methods: Twenty-four women participated: eight continent, eight with mild SUI and eight with moderate to severe SUI. Volunteers performed three maximum coughs in supine and in standing. Maximum PFM EMG and VP amplitudes and the timing of the EMG peak relative to the VP peak were determined. Ensemble average VP versus EMG curves were created. Results: There were no significant differences among the groups in the maximum EMG or VP amplitudes. The EMG and VP peaked simultaneously in both positions in the continent group. In the mild SUI group, the EMG and VP peaked simultaneously in supine, but the EMG peaked before the VP in standing. In the moderate to severe SUI group, the EMG peaked before the VP in both positions. The shapes of the VP versus EMG curves were similar among the groups and positions; however, the SUI groups displayed higher EMG-intercepts than the continent women. Conclusion: These findings suggest that urine leakage during coughing in women with SUI may be related to delays in force transmission rather than PFM weakness.
5.2 Introduction

Coughing produces a rapid increase in intra-abdominal pressure (IAP) that commonly provokes episodes of stress urinary incontinence (SUI). To assist with continence, the pelvic floor muscles (PFMs) provide a firm surface against which the urethra can be compressed by the increase in IAP. In continent women, increases in IAP are preceded by a rise in urethral pressure produced by contraction of the PFMs and the striated external urethral sphincter.

The electromyographic (EMG) amplitude of the PFM contraction has been shown to be positively correlated with cough strength in continent women, suggesting that the force generated by the PFMs is modulated to task demands. PFM EMG activity during coughing appears to be different in women with SUI compared to continent women. Specifically, anal sphincter EMG amplitude is lower, PFM activation is delayed and the ability to modulate PFM contraction strength to the forcefulness of a cough is impaired. The rise in urethral pressure prior to the rise in IAP is also absent in women with SUI, which may explain the lower midurethral pressure found in women with SUI during coughing. Vaginal pressure is a convenient surrogate measure for intraurethral pressure as it poses a lower risk of infection, is more comfortable and easier to perform and yet still reflects the functional closure forces delivered to the urethra.

The precise relationship among PFM and abdominal muscle activation, the resultant vaginal pressure (VP) and continence status during coughing is unclear. It is thought that, during voluntary PFM contractions, continent women generate higher PFM EMG activation amplitudes and VP, and are able to contract the
PFMs more quickly than women with SUI. This may have implications for coughing as it requires a large and rapid increase in PFM activation in preparation for increases in intra-abdominal pressure.

PFM and abdominal muscle activation have been shown to differ between women with and without SUI during voluntary PFM contractions and postural perturbations; however, less is known about PFM and abdominal muscle activation during coughing. The purpose of this study was to compare patterns of abdominal and PFM activity associated with coughing between women with SUI and continent women in order to shed light on differences in contractile activity that may explain the urine leakage seen in women with SUI.

5.3 Methods

5.3.1 Objectives of the Study

This study was designed to compare cough-induced PFM and abdominal EMG activation and VP increases among three groups of women (continent, mild SUI and moderate to severe SUI) in two positions (supine and standing). This was done through comparing: (I) maximum abdominal and PFM EMG amplitudes, (II) maximum VP amplitudes, (III) the timing of the peak VP relative to peak IAP, (IV) the rate of the VP rise and (V) the contributions of the PFMs and the abdominal muscles to the generation of VP.

5.3.2 Sample and recruitment

Women with and without SUI were recruited for a larger study on the biomechanics of the extrinsic continence system using advertisements published in
a local newspaper. Ethics approval for the study was received from the university’s Health Sciences Research Ethics Board with each volunteer providing written, informed consent prior to participating (See Appendix C.). Volunteers were screened by a research nurse and were included if they were between 35 and 60 years old and either presented with symptoms of SUI or reported no urine leakage. Women were excluded if they were unable to perform a voluntary PFM contraction, were pregnant or had given birth in the previous twelve months, had auto-immune, connective tissue or neurological disorders, had intrinsic sphincter deficiency, had pelvic organ prolapse >POP-Q stage II or if they were taking medications that either treat or provoke urinary incontinence.\textsuperscript{262,265} All of the volunteers who qualified for the study were provided with a three-day bladder diary to be completed prior to the data collection session. (See Appendix D.) Women who reported SUI underwent urodynamic testing to confirm the absence of other types of urinary incontinence. The number of leakage episodes recorded on the diary was divided by three to obtain the mean number of leakage episodes per day. The mean number of leakage episodes was multiplied by the Ingelman-Sundberg score\textsuperscript{269} to determine the leakage severity score. All women with moderate to severe SUI (severity score >2) from the larger sample were included in this study and women with mild SUI (severity score =1 or 2) and continent women were matched to them by age (within 3 years), body mass index (BMI: within 3 kg/m\textsuperscript{2}) and parity (yes/no) retrospectively.

5.3.3 Instrumentation

EMG and VP data were recorded during one testing session. EMG data were
recorded from the rectus abdominis (RA), internal oblique (IO) and external oblique (EO) muscles using Delsys™ D.E.2.1 surface electrodes (Delsys Inc., Boston, Massachusetts). Surface EMG activity from the PFM was recorded using a Femiscan™ vaginal probe (Mega Electronics Ltd., Kuopio, Finland). The Femiscan™ electrodes were coupled to Delsys™ D.E.2.1 preamplifiers. All of the preamplified EMG signals were passed through a Delsys Bagnoli™ 16-channel EMG amplifier system. Pressure was recorded via two air-filled pressure transducers mounted on the vaginal probe to measure both proximal VP (located at the vaginal apex as a surrogate for IAP266) and distal VP, adjacent to the posterior vaginal wall centred 3.5cm proximal to the introitus,226 capturing vaginal closure pressure induced by abdominal and PFM contraction output. The pressure transducers were interfaced directly with the analog to digital converter.

The pressure and EMG data were sampled simultaneously at 1000 Hz using a 16-bit National Instruments analog to digital converter (NIDAQ PCI-MIO-16XE-10) and EMGworks® Acquisition software (Delsys Inc., Boston, Massachusetts) then stored on a personal computer. During coughing, expiratory flow rate was measured using a peak flow meter.

5.3.4 Data Acquisition

The completed bladder diary was returned to the researchers in a sealed envelope so that the researchers remained blinded to each volunteer’s continence status. The volunteer was instructed on how to use the peak flow meter so that a tight seal was maintained around the mouthpiece by the lips. A nose plug was used to prevent air leakage from the nares. The volunteer performed three practice
coughs to ensure that she could consistently achieve a peak flow of at least 250 l/min. After verbal instructions, the volunteer was left in private to insert the EMG and pressure probe into her vagina. Visual inspection of the perineum and the probe for cephalad movement, and of the EMG and pressure traces during a voluntary PFM contraction, confirmed correct placement of the probe and the volunteer's ability to correctly perform a PFM contraction. Each volunteer was permitted as many practice contractions as needed to achieve a correct PFM contraction and data collection began once the volunteer was able to perform such a contraction.

After skin preparation, the electrodes were adhered over the abdominal muscles on the volunteer's left side. The RA electrodes were centred 2 cm lateral and caudal to the umbilicus, the EO electrodes were centred over tip of the eighth rib and the IO electrodes were centred 2 cm proximal to the midpoint of a line from the anterior superior iliac spine to the symphysis pubis. A common reference electrode was placed over the left anterior superior iliac spine.

EMG and pressure data were recorded during coughing in supine and in standing; the order of these positions was randomized. In each position, the women were asked to relax completely so that tonic activity and noise could be recorded for five seconds. They then performed three coughs of maximal expulsion effort with their mouth sealed over the peak flow meter mouthpiece and with the meter held vertically. The instructions were: “When I say go, take a deep breath in and cough once as hard as you can. Be sure to keep your mouth tightly sealed around the peak flow meter.” At least 60 s of rest were given between repetitions.
5.3.5 Signal processing and data analysis

5.3.5.1 Peak flow during coughing

A two-way analysis of variance (ANOVA) ($\alpha < 0.05$), including group and position as factors, was used to compare the peak flow during coughing among the groups (Minitab™ v.14, State College, Pennsylvania). This analysis was done to ensure that differences in peak flow did not bias the VP or EMG results.

5.3.5.2 EMG amplitudes

EMG data were rectified and smoothed using a 200 ms sliding window (199 ms overlap) over which the root mean square (RMS) value was calculated. For each electrode channel during each cough, the lowest RMS value computed across the recording period was subtracted from the peak EMG value to remove tonic activation levels. This corrected peak value was recorded as the maximum EMG RMS for each channel and task. A three-way ANOVA (general linear model) was used to test for differences in peak PFM and abdominal EMG amplitudes among the groups. Each trial was included in the analysis. The model included participant, as a random factor nested in group, group and position as fixed factors and all two and three-way interactions. The interaction between participant and group was used as the denominator for each respective F-test and as such the statistical analysis accounted for the variability attributed to inter-individual differences in EMG amplitude among participants without normalizing to a reference level of activity that would be difficult to standardize. The $\alpha$ level for all tests was 0.05 and relevant post hoc comparisons were made using the Bonferroni method.
PFM EMG data were recorded bilaterally and there was no systematic difference in the peak EMG RMS amplitudes between the right and left sides (p>0.05). Therefore, the EMG data from the side of the PFMs that produced higher EMG activity was used in the statistical analyses.

**5.3.5.3 Vaginal pressure amplitudes**

The air-charged pressure sensors had an impulse response with a 5 ms delay that was removed from all pressure data prior to processing and analysis. The pressure data were smoothed and the maximum pressure was determined as described for the EMG data above. The maximum VP amplitudes for each pressure transducer, from each trial, were compared among the groups using a nested (subject in group) three-way ANOVA model including group, position and pressure transducer as factors with all possible interactions included in the model (α<0.05). Relevant post hoc comparisons were made using the Bonferroni method.

**5.3.5.4 Rate of vaginal pressure generation**

The slope of the distal VP rise was calculated from the time that the smoothed distal VP rose above two standard deviations over baseline to the time it reached its peak. The slopes of the distal VP rise from each trial were compared among the groups using a two-way nested ANOVA (participant nested in group) that included group and position as factors; the group by position interaction was considered in the model (α<0.05). Relevant post hoc comparisons were made using the Bonferroni method.
5.3.5.5 Timing of peak vaginal pressure generation

The time delay between the peak EMG amplitude for each muscle and the peak of the distal VP was calculated. Positive values indicated that the distal VP peaked before the peak in EMG activity. The delay between the proximal and distal VP peaks was computed similarly. The time delays between the distal VP peak and the proximal VP peak, and between the distal VP peak and the EMG peak for each muscle for each trial, were compared among the groups using a three-way nested ANOVA (subject nested in group) that included group, muscle and position as factors (α<0.05). Relevant post hoc comparisons were made using the Bonferroni method.

5.5.5.6 Muscle activation contributions to distal vaginal pressure

All of the EMG and pressure data files recorded during coughing were full wave rectified and smoothed using a dual-pass, third order, 5 Hz low pass Butterworth filter. All EMG and pressure data were normalized to the maximum smoothed amplitude computed from each channel during each cough. Ensemble average distal VP versus EMG curves were created using each trial by group for each muscle in each position by computing the percent maximum EMG activation at each 5% increment of percent maximum distal VP. The equations of the curves were determined and tested for significance (α<0.05) using a runs test (GraphPad Prism™ v.4, La Jolla, California). The patterns demonstrated by the groups were determined to be significantly different if the 95% confidence intervals for the
slopes and intercepts did not overlap or if different order equations produced the best fit.

All data were found to be normally distributed; therefore, the statistical analyses were performed using parametric tests.

5.4 Results

5.4.1 Sample

Data from 24 women: eight continent women, eight with mild SUI and eight with moderate to severe SUI, were included in this study. (See Table 5.1.)

Table 5.1 Demographic data

<table>
<thead>
<tr>
<th>Group</th>
<th>Continent (n=8)</th>
<th>Mild SUI (n=8)</th>
<th>Moderate to Severe SUI (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>51.6 ± 6.2</td>
<td>52.3 ± 7.0</td>
<td>53.5 ± 6.0</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>25.0 ± 3.7</td>
<td>27.1 ± 5.1</td>
<td>27.4 ± 5.4</td>
</tr>
<tr>
<td>Parity (# births)</td>
<td>1.9 ± 1.1</td>
<td>2.4 ± 1.2</td>
<td>2.4 ± 0.5</td>
</tr>
</tbody>
</table>

All data are given as mean ± standard deviation. The groups were matched by these factors.

5.4.2 Peak flow during coughing

Position affected the peak flow rate during coughing differently depending on continence status (p=0.003): women with mild SUI produced higher peak flows in standing than women with moderate to severe SUI. There were no other significant differences among the groups or positions. See Appendix E for the full results for peak flow.

5.4.3 Pelvic floor and abdominal muscle EMG amplitudes

There were no significant differences in maximum PFM EMG amplitudes produced during coughing among the groups or the positions. However, the
abdominal muscles reacted differently in the three groups (p<0.001). (See Figure
5.1.)

When supine, continence status affected the maximum cough-induced EO and IO
EMG amplitudes: continent women produced higher EO EMG amplitudes (p<0.001
vs. both mild SUI and moderate to severe SUI), while women with mild SUI
produced higher IO EMG amplitudes (p=0.008 vs. continent, p<0.001 vs. moderate
to severe SUI). When standing, even though the EMG activation patterns were
substantively the same as when supine, the differences among the groups were not
significant.

![Figure 5.1 Peak EMG amplitudes in supine (a) and standing (b). C: continent
continent women, M: women with mild SUI, S: women with moderate to severe SUI, PFM: pelvic floor
muscles, RA: rectus abdominis, EO: external obliques and IO: internal obliques. * indicates significant
differences between groups, p<0.05.](image)

Peak flow, when included in the analysis as a covariate, was not a significant
determinant of muscle effort either as a main effect or as an interaction with group
or position.

### 5.4.4 Vaginal pressure amplitudes

In both positions the proximal VP was higher than the distal VP for the continent
women and the women with moderate to severe SUI (p<0.001). This difference was
also present in the women with mild SUI but was only significant in standing. (See Figure 5.2.) The maximum cough-induced distal VP was not significantly different among the groups in either of the testing positions.

Maximum cough-induced proximal VP was significantly lower in the women with mild SUI compared to the other two groups in supine while there was no significant difference among the groups in standing. Peak expiratory flow did not influence the results when the analysis included this factor as a covariate.

![Figure 5.2 Maximum vaginal pressure amplitudes. C: continent women, M: women with mild SUI, S: women with moderate to severe SUI, Distal VP: distal vaginal pressure transducer and Proximal VP: proximal vaginal pressure transducer. * indicates significant differences between groups, p<0.05.](image-url)
5.4.5 Timing of peak vaginal pressure generation in relation to peak pelvic floor and abdominal muscle EMG

The effect of continence status on the relative timing of the peak PFM EMG and distal VP was influenced by position (p=0.021). (See Figure 5.3.) The continent women showed simultaneous PFM EMG and distal VP peaks in both positions. In the women with mild SUI, PFM EMG peaked 183 ms (±209) before the distal VP when standing but the peaks were simultaneous when supine. In the women with moderate to severe SUI, PFM EMG peaked before the distal VP in both positions (supine: 408 ms (±462) and standing 245 ms (±321)).

The timing of the abdominal muscle EMG peaks relative to the distal VP peak was disrupted in the women with SUI compared to the continent women. (See Figure 5.3.) The continent women generally reached their maximum abdominal muscle activation after the distal VP peak occurred (p<0.001). The women with mild SUI reached their maximum abdominal muscle activation prior to the distal VP peak (p<0.001); this pattern was clearest when standing. The women with moderate to severe SUI reached their maximum abdominal muscle activation before the distal VP peak in both positions, except for RA where the EMG activity peaked after the distal VP peak in standing (p=0.021).

There was a significant interaction between group and position in the timing of the proximal VP peak relative to the timing of the distal VP peak (p=0.005). In the continent women and those with mild SUI, the distal and the proximal VPs peaked simultaneously in both positions whereas in women with moderate to severe SUI,
proximal VP peaked before distal VP in both positions (supine: 370 ms (±554) and standing 179 ms (±265)).

Figure 5.3 Timing of the EMG and proximal vaginal pressure peaks relative to the time of the distal VP peak in supine (a) and standing (b). C: continent women, M: women with mild SUI, S: women with moderate to severe SUI, PFM: pelvic floor muscles, RA: rectus abdominis, EO: external obliques and IO: internal obliques and PVP: proximal vaginal pressure. The zero line indicates the timing of the lower vaginal pressure peak. * indicates that the timing of the EMG or proximal VP peak is significantly different from the timing of the distal VP peak time and + indicates significant differences between groups, p<0.05.

5.4.6 Rate of vaginal pressure generation

The effect of continence status on the rate at which women were able to generate distal VP was also influenced by position. In supine, all women demonstrated similar rapidity in the rise of the distal VP. In standing, continent women and those with mild SUI generated distal VP more rapidly than women with moderate to severe SUI (p=0.025 and p=0.022 respectively). (See Figure 5.4.)
Figure 5.4 Slope of the distal vaginal pressure rise. Continent: continent women, Mild SUI: women with mild SUI, Severe SUI: women with moderate to severe SUI. * indicates significant differences between groups, p<0.05.

5.4.7 Muscle activation contributions to distal vaginal pressure

The distal VP versus PFM EMG curves for each group in each position showed high variability. Outlying observations (defined as curves that did not follow the shape of the majority of curves over more than 50% of their length) were noted and re-analysis without these outliers was performed. The unusual observations were not consistently observed in a given group or individual. In supine, six trials in the continent group, four trials in the mild SUI group and three trials in the moderate to severe SUI group were excluded from the analysis. In standing, nine trials in the continent group, four trials in the mild SUI group and six trials in the moderate to severe SUI group were excluded from the analysis.
In both positions, and for all three groups, the primary distal VP versus PFM EMG curves were similar, demonstrating an initial steep rise followed by a plateau, which were best modelled with second order polynomial equations ($y=a+bx+cx^2$). This indicates that the PFMs were active generators of the initial VP rise and that they contributed less to the final portion of the VP rise. (See Figure 5.5.) In general, the excluded trials displayed PFM EMG activity that remained at a constant level throughout the increase in distal VP.

![Figure 5.5 Distal vaginal pressure versus PFM EMG ensemble average curves: left: supine, right: standing. The symbols indicate the mean and the whiskers indicate the 95% confidence interval of the mean. Squares: continent women, Triangles: women with mild SUI, Circles: women with moderate to severe SUI.](image)

The EMG-axis intercepts of the distal VP versus PFM EMG curves were all significantly greater than zero, reflecting the expected electromechanical delay between the EMG activation and the rise in VP. In supine, the EMG-axis intercept was significantly higher in the women with moderate to severe SUI compared to those with mild SUI ($p<0.001$). In standing, the women with mild SUI had a higher EMG-axis intercept than did either of the other groups ($p<0.001$ for both).

All of the distal VP versus abdominal EMG curves displayed an initial steep rise followed by a plateau. Figure 5.6 illustrates the ensemble average distal VP versus RA EMG curves, which are representative of the distal VP versus EMG curves
produced by each of the abdominals. Although the shapes of curves did not appear to be different among groups or positions there were significant differences in the EMG-axis intercepts among groups and positions. There was a pattern of higher intercepts for the women with SUI than for continent women with those with moderate to severe SUI displaying the highest intercepts. As well, all of the women displayed a pattern of higher EMG intercepts in standing compared to supine; it was only significant in the women with SUI.

![Figure 5.6 Distal vaginal pressure versus RA EMG ensemble average curves in standing. Continent: continent women, Mild SUI: women with mild SUI, Severe SUI: women with moderate to severe SUI. The symbols indicate the mean and the whiskers indicate the 95% confidence interval of the mean. The relationship was best modeled with a linear equation for the continent women and with second order polynomial equations for the women with mild and moderate to severe SUI, however, the shapes of the curves appear to be substantively similar.](image)

None of the EMG versus VP curves attained 100% on the EMG axis. This is because each volunteer attained her maximum EMG amplitude at a different point on the pressure axis, thus dispersing the peaks of the ensemble average curves.

5.5 Discussion

In this study, similar maximum PFM EMG and distal VP amplitudes were found between women with and without SUI. This suggests that a reduction in PFM activation secondary to muscle weakness is not a major factor associated with urine
leakage during coughing in women with SUI. There were, however, differences in the timing of muscle activation relative to VP and in the rate of VP generation among the groups. Relative to continent women and women with mild SUI, women with moderate to severe SUI demonstrated a delay in the distal VP peak relative to the proximal VP peak. They also generated distal VP more slowly than did either the women with mild SUI or the continent women. Compared to the continent women, all of the women with SUI demonstrated higher levels of EMG activation before a rise in distal VP was observed. This suggests that during coughing women with SUI have a slower PFM response and that they may have to activate their PFMs and abdominal muscles to a larger degree before changes in VP are observed. Further, the synchronous response of the PFMs and the abdominal muscles was not observed in women with SUI, which might explain why women with SUI experience urine leakage during coughing.

This study advances our understanding of the extrinsic continence mechanism in women with SUI during coughing. By including PFM and abdominal muscle EMG activity, the synergistic activation of these muscle groups can be observed. Furthermore, the muscle activation strategies used to generate VP can be described with the simultaneous recording of EMG and VP, revealing a more complete picture of the differences between continent and stress incontinent women.

5.5.1 Sample

The volunteers with SUI were stratified and matched by SUI severity retrospectively, therefore, the number of participants available for inclusion in this study was limited by the number of volunteers in the complete sample with
moderate to severe SUI. This likely has caused the study to be underpowered 
(\(\alpha=0.05, \beta=0.15\) for the maximum EMG amplitude ANOVA, and \(\alpha=0.05, \beta=0.16\) for 
the maximum VP amplitude ANOVA) and may have masked differences among the 
groups. However, to achieve \(\alpha=0.05, \beta=0.80\), 70 volunteers per group would have 
been required which suggests that any differences among the groups in maximum 
EMG or VP amplitudes were very small and perhaps not clinically relevant.

The decision to stratify the participants with SUI to mild and moderate to severe 
categories was based on the suggestion that involuntary PFM activity in women 
with SUI in response to postural perturbations may vary depending on the severity 
of their leakage.\textsuperscript{147} Consistent with Smith et al.\textsuperscript{147} this study found significant 
differences between women with mild SUI and those with moderate to severe SUI in 
some of our results. As such, this stratification was justified despite the fact that 
keeping the two strata together might have improved the statistical power of many 
of the analyses.

It is unlikely that differences in cough force affected our results. When the EMG 
and VP amplitudes were compared among the groups using peak flow as a covariate, 
no significant differences were found. In addition, despite the fact that women with 
mild SUI produced higher peak flows during coughing in standing, they generated 
equal PFM EMG and VP values.

5.5.2 Pelvic floor and abdominal muscle EMG amplitudes

There were no differences among the groups in the maximum PFM EMG 
amplitudes during coughing in either position. Because only the EMG from the side 
of the PFMs that produced larger signal amplitudes were included, the analyses
were repeated with the PFM EMG produced by the side with lower EMG amplitudes resulting in no difference in the results. This result suggests that the urine leakage seen during coughing in women with SUI is not related to difficulties in PFM activation (on either side) and is consistent with the literature for voluntary PFM contractions.\textsuperscript{193, 276, 277}

The continent women and the women with mild SUI produced their highest abdominal EMG amplitudes during coughing in supine. This may be related to it being more difficult to cough in supine than it is in standing as the diaphragm is less able to displace the abdominal contents.\textsuperscript{278} Whether or not these findings are of clinical significance is uncertain, as there were no concomitant differences in either maximum VP or peak flow.

5.5.3 Vaginal pressure amplitudes

There were no differences in the maximum distal VP rise among the groups in either position. This finding is consistent with the literature for voluntary PFM contractions\textsuperscript{196, 233} and is consistent with the EMG findings in this study. It suggests that urine leakage during coughing in women with SUI may not be due to inadequate VP generated through PFM contraction; it seems instead that women with SUI may not be able to effectively transfer VP to the urethra, perhaps because of damaged fascia or PFM tears or avulsion.\textsuperscript{149, 279}

Proximal VP was only significantly different among the groups in supine, which may be related to it being more difficult to cough in this position.

Distal VP was lower than proximal VP in continent women and women with
moderate to severe SUI. Since proximal VP was recorded above the PFMs’ vaginal attachments, it was assumed to be a measure of IAP. It was expected that distal VP would be higher than proximal VP reflecting contributions from both IAP and from the force generated by PFM contraction. That it was lower suggests either that measures of distal VP may not be as good a surrogate for urethral pressure in women as previously thought or that the transfer of IAP is less significant in the generation of VP than it is in the generation of urethral pressure and the relationship between VP and urethral pressure should be investigated further.

5.5.4 Timing of peak vaginal pressure generation in relation to peak pelvic floor and abdominal muscle EMG

PFM EMG and distal VP peaked simultaneously in the continent women; however, in both groups of women with SUI the PFM EMG peak preceded the distal VP peak in at least one position. One possible explanation is that women with SUI were using their PFMs to generate the initial rise in distal VP but were relying on other muscles to generate the final portion of the distal VP. Alternatively, there may be a delay in the transfer of force from the PFM contraction to the vagina, perhaps due to fascial damage.

The timing of the distal VP peak relative to the peak EMG amplitude for each abdominal muscle was different among the groups. In the continent group, the abdominal muscle EMG tended to peak after the distal VP. This may be due to their important role in generating IAP, which also tended to peak after VP. All of the women with SUI demonstrated abdominal EMG peaks prior to the distal VP peak; however, the patterns were different between women with mild SUI compared to
those with moderate to severe SUI. In women with mild SUI, both the abdominal muscles’ and the PFMs’ EMG activity peaked before the distal VP in standing which suggests that there may have been delayed transmission of pressure to the vagina, perhaps due to damaged fascial supports, because these muscles are primary generators of VP. Women with moderate to severe SUI demonstrated a pattern in which the PFM EMG activity peaked before the distal VP in both testing positions and there was a more variable pattern of abdominal muscle peaks. The variation in abdominal muscle activation may reflect that these women may have been bracing, perhaps in an attempt to avoid urine leakage, by globally activating their PFMs and abdominal muscles in preparation for the cough. This suggests that these women have spontaneously learned to voluntarily contract the PFMs prior to coughing and supports teaching women with SUI to perform a voluntary PFM contraction before coughing as a technique to reduce urine leakage. The exception to this bracing pattern is the delayed RA peak, relative to the both the proximal and the distal VP peaks, during the standing coughs. The functional implications of this delay are unclear and it was thought that the volunteers might be demonstrating individual adaptations. However, when the data were examined individually no patterns emerged to suggest such adaptations.

The timing of the proximal VP peak relative to the distal VP peak was different among the groups and positions. In the continent group, the distal VP peaked with the proximal VP in both positions. When standing, the timing of the distal versus proximal VP peaks was consistent with that found in the literature for the timing of the urethral pressure peak relative to the IAP peak during coughing in continent
women. In the groups with SUI, the proximal VP peaked before the distal VP in at least one position. This finding is consistent with the lower slopes in the VP versus time curves of women with moderate to severe SUI found in this study and suggests that the delayed VP peak may result from a decreased ability to generate pressure quickly. Such difficulty in generating pressure quickly may be related to differences in predominant muscle fibre type, a delayed onset of pressure generation (perhaps due to lax vaginal support structures or PFMs) or a combination of these two phenomena. The results of the current study are consistent with the literature where authors have reported delays in urethral pressure relative to IAP during coughing in women with SUI.

5.5.5 Rate of vaginal pressure generation

The rate of the pressure rise was different among groups and positions. These differences were most evident in standing where women with moderate to severe SUI generated VP the most slowly. Such a delay may contribute to urine leakage if it causes the urethral pressure peak to occur after the IAP peak. This slower VP generation is consistent with the findings of Morin et al. who reported that women with SUI were less able to perform rapid voluntary PFM contractions than continent women.

5.5.6 Muscle activation contributions to distal vaginal pressure

The shapes of the distal VP versus EMG curves were very similar among the groups and the positions. Likewise, the shapes of the distal VP versus EMG curves were similar among continent women, women with mild SUI and women with
moderate to severe SUI during voluntary PFM contractions performed in supine.
(See Chapter 4.) In the case of coughing, differences among the groups were seen in
the intercepts of the curves. The EMG-axis intercepts for the distal VP versus EMG
curves were the highest overall in the women with moderate to severe SUI. This
pattern was most apparent in supine. The same pattern was observed, to a lesser
extent, in the women with mild SUI. This finding suggests that women with SUI may
have to generate more muscle activity before they begin to generate distal VP. This
phenomenon might occur if, during the early phase of a cough, the force from the
PFM contraction and the increase in IAP are predominately absorbed by the slack in
the PFMs themselves or in the vaginal supporting structures, due to laxity or tears. If
this were the case, this phenomenon would be more evident in supine since there is
less gravitational pull on the pelvic tissues in this position and, therefore, there is
likely more laxity.

The findings of this work provide further evidence to support the
recommendation that women with SUI perform a voluntary PFM contraction prior
to coughing, as this pre-contraction may restore the normal order of the distal VP
peak preceding the proximal VP peak and may help to compensate for damaged
fascial supports. The findings also provide evidence that PFM exercise training for
SUI may work primarily by improving motor control as opposed to strengthening
as the deficits demonstrated by the women with SUI were in the rate and timing of
force production and not in PFM contractility.

5.6 Conclusions

Our results suggest that urine leakage in women with SUI during coughing does
not appear to be due to PFM weakness but is more likely due to fascial support defects and/or altered PFM and abdominal muscle co-ordination. This conclusion is based on the findings that: (I) even though the women with and without SUI were able to generate equal PFM EMG amplitudes and distal VP, the women with SUI generated distal VP more slowly than the continent women, (II) proximal VP peaked before distal VP in the women with SUI while proximal and distal VP peaked simultaneously in the continent women and (III) the women with SUI demonstrated higher EMG-axis intercepts on the distal VP versus abdominal EMG curves. The clinical implication of these findings is that PFM exercise therapy may work primarily through improving motor control by teaching women with SUI more effective motor patterns and training the PFMs to contract more quickly. The results also support the practice of teaching women with SUI to perform a PFM contraction prior to coughing as a practical way to reduce urine leakage.
Chapter 6 Vaginal Pressure Generated during Voluntary Pelvic Floor Muscle Contractions and During Coughing: The Effect of Age and Continence Status

6.1 Abstract

Aims: The purpose of this study was to determine if, compared to continent women, women with stress urinary incontinence (SUI) generate lower vaginal pressure (VP) during maximum voluntary pelvic floor muscle contractions (PFM MVCs) or coughing, slower contraction times or less ability to sustain VP, and if there are reductions in the ability to generate VP during these tasks associated with increasing age. Methods: Eighty-seven women participated (35 continent, 35 with mild SUI and 17 with severe SUI). VP data were acquired while participants performed PFM MVCs, maximum effort coughs and a sustained PFM contraction. The maximum VP amplitudes measured during the MVCs and coughs, the time for VP to reach a peak during coughing and the time for the VP to drop by 50% during the sustained contraction were compared among the groups using separate analyses of covariance, with age included as a covariate. Results: There were no differences in maximum PFM MVC VP or in endurance time among the groups. Women with SUI produced higher cough VP amplitudes than continent women. Age was associated with a reduction in VP and with slower contraction times during coughing. Conclusions: The data suggest that PFM weakness may not be an important factor in the pathophysiology of SUI. Women with SUI may intuitively use their PFMs to compensate for urethral sphincter or fascial deficits. The VP generated during
coughing and the speed at which peak VP is attained are, however, substantially reduced with increasing age, which might affect continence in older women.

6.2 Introduction

Stress urinary incontinence (SUI) is the most common type of urinary incontinence reported by adult women.\textsuperscript{262} Conservative treatment, including pelvic floor muscle (PFM) exercise, is recommended as the first line treatment for SUI in women.\textsuperscript{262} The goal of PFM exercise is to improve the PFM’s ability to respond to increases in intra-abdominal pressure by stabilizing the urethra and applying an external closure force to it;\textsuperscript{42} however, there is conflicting evidence as to whether or not PFM weakness is a contributing factor in many cases of SUI.\textsuperscript{196, 227, 264}

Studies comparing PFM function between women with and without SUI have produced equivocal results. Some studies have found that continent women have stronger PFMs than women with SUI,\textsuperscript{227, 264} while others have found no differences in PFM strength between women with and without SUI.\textsuperscript{196} Conflicting results have also been reported with respect to differences in PFM endurance between women with and without SUI.\textsuperscript{196, 271, 280}

Vaginal pressure (VP) amplitudes during coughing have been reported to be lower in women with SUI than in continent women.\textsuperscript{271} The relationship between VP generation during coughing and body position, lying versus upright, has not been reported in the literature and needs to be explored as urine leakage is reported more frequently in upright than in dependent postures.\textsuperscript{262}

Increasing age has been reported to be a contributing factor in the development of SUI.\textsuperscript{262} However, the effect of age on PFM function is unclear. Two studies have
reported that PFM strength decreases with increasing age in continent women,\textsuperscript{193} while others have reported that PFM strength is maintained with increasing age.\textsuperscript{107,128} Interestingly, one of the groups that found that PFM strength is maintained with aging in continent women also found that PFM strength decreases with increasing age in women with SUI.\textsuperscript{128}

In rehabilitation therapy for PFM dysfunction, assessment procedures normally include measures of PFM strength, endurance and contractile activity during coughing.\textsuperscript{219} However, research studies on PFM function in women with and without SUI have tended to focus on only one aspect of PFM function at a time (strength, endurance, co-ordination or involuntary activity) and have not combined these measures of PFM function in order to understand how the various aspects of PFM relate to one another and to SUI.

The purpose of this cross-sectional study was to compare four aspects of PFM function: (I) voluntary contractile strength, (II) contractile strength during coughing, (III) time required to reach peak VP during coughing and (IV) ability to sustain a contraction between women with and without SUI, and to determine whether these measures are affected by increasing age.

\textbf{6.3 Materials and methods}

Ethics approval for the study was received from Queen’s University’s Health Sciences Research Ethics Board (REH-184-03) and each volunteer provided informed consent prior to participating. (See Appendix C.) Continent and stress incontinent women aged 30 to 70 years, who were not pregnant, had not given birth in the previous year and who were in good general health were recruited using
newspaper advertisements. Women who demonstrated urine leakage during coughing in supine or urogenital prolapse greater than POP-Q stage II, who had prior diagnoses of either neurological or rheumatological conditions, who had a history of either gynaecological or continence surgery, who had ever participated in a formal PFM exercise program or who were taking medications known to alleviate or to exacerbate urinary incontinence were excluded. All of the women who responded to the advertisements were initially screened for inclusion by telephone and then, if appropriate, attended a screening session with a registered nurse to confirm that they met the inclusion criteria and that they were able to perform a voluntary PFM contraction. All women who met the inclusion criteria after screening were provided with a three-day bladder diary (See Appendix D.) and were asked to complete it over three days of typical activity during the next week. The diary was returned at the time of laboratory testing, which occurred approximately one week after screening. The women who reported symptoms of SUI underwent urodynamic testing and those who demonstrated urodynamic evidence of conditions other than SUI were excluded from the study. Based on previous literature, a sample size of 30 women per group was predicted to be adequate to detect differences in VP between women with and without SUI (α=0.05 and β=0.80).

Vaginal pressure data were acquired by a researcher blinded to group assignment. The custom vaginal probe had a pressure sensor mounted posteriorly that was centred 3.5cm proximal to the introitus. The pressure sensor was constructed from a 10 French rectal balloon catheter (Laborie Medical Technologies,
www.laborie.com) and was air filled. The pressure sensor was interfaced with a Motorola MPX5010 Integrated Silicon pressure transducer (resolution 0.04 V=1 cm H$_2$O, range 0-10 V). The impulse response of the pressure system demonstrated that there was a delay of 5 ms to peak pressure; however, this delay was not relevant to the measures made in this study. The reliability of pressure data collected with this probe was previously tested by our group and was found to be excellent ($r$>0.99).\textsuperscript{139}

The pressure transducer was interfaced directly with a 16-bit National Instruments analog to digital converter (NIDAQ PCI-MIO-16XE-10). The data were acquired at 1000Hz and stored on a personal computer using EMG Works® Acquisition software (Delsys®, www.delsys.com).

6.3.1 Data Collection

Vaginal pressure data were recorded in supine and in standing with the positions presented in random order. In supine, the volunteers performed three repetitions of a maximum voluntary PFM contraction held for less than three seconds, followed by three maximum effort coughs and then by a sustained PFM contraction. In standing, the volunteers performed three maximum effort coughs. For the PFM MVCs, the volunteers were instructed to: “Use your PFMs to pull up and in. Squeeze around the probe as hard as you can.” The instructions for the cough were to: “Cough once as hard as you can.” The instructions for the sustained contraction were the same as for the PFM MVCs with the added instruction to: “Hold the contraction for as long as possible.” The volunteers were coached throughout a 60 s period to maintain the contraction level at a maximum and then were told to relax their PFMs. The PFM MVCs and the sustained contraction were only performed
in supine as no difference has been found in the ability to generate maximum VP between supine and standing.\textsuperscript{233,274} As well, only supine was used because the performance of repeated contractions and/or a contraction sustained to fatigue was likely to affect the volunteer’s force generating capacity and endurance during subsequent trials.

6.3.2 Data processing and analysis

The severity of urine leakage in the women with SUI was determined from the bladder diaries. The leakage episodes were categorized by the lowest intensity of provocation required to cause urine leakage.\textsuperscript{269} The mean number of daily leakage episodes was multiplied by the provocation score to calculate the leakage severity score. Women with a severity score of zero were classified as continent, women with a severity score of one or two were classified as having mild SUI and women with a severity score of three or greater were classified as having moderate to severe SUI.

Demographic data for age and body mass index (BMI) were compared among the groups using one-way analyses of variance (ANOVAs). To determine if there were differences in the number of parous versus nulliparous women in each group a three-way Chi-Square ($\chi^2$) was used ($\alpha=0.05$).

6.3.2.1 Voluntary PFM contraction strength

Voluntary PFM contraction strength was computed from the PFM MVCs. The VP data were smoothed by computing root mean square (RMS) amplitudes using a 200 ms sliding window with a 199 ms overlap across all data files. The RMS values of
the resting data were subtracted from the peak RMS values to remove any bias due to differences in balloon inflation, vaginal anthropometrics, instrumentation noise or resting PFM activity. The highest remaining smoothed VP value for each MVC was considered to be the maximum rise in VP for that trial. An analysis of covariance model (ANCOVA) was used to compare the peak pressure during each trial of the MVC among the groups while considering age as a covariate. The interaction between group and age was included in the model. In all analyses, when significant group differences were found post-hoc tests were conducted using Tukey’s simultaneous tests.

In all ANCOVAs age was treated as a continuous variable. For clarity of graphical presentation, age was presented as means for each ten-year interval, but was not treated as a categorical variable in the analysis.

**6.3.2.2 Involuntary PFM contraction strength**

Contractile strength during coughing was used as a measure of involuntary PFM activity. The maximum VP during the coughs was calculated in the same way as the maximum VP during the PFM MVCs. An ANCOVA was used to compare the maximum cough VP from each trial among the groups and the positions while considering age as a covariate.

**6.3.2.3 Vaginal pressure rise time during coughing**

The VP rise time was used as a measure of the speed of PFM contraction. It was computed for the VP from the cough data files by subtracting the time at which the VP rose above two standard deviations over the baseline pressure from the time it
reached its maximum. An ANCOVA was used to compare the VP rise time from each trial among the groups and the positions while considering age as a covariate.

### 6.3.2.4 Duration of the sustained PFM contraction

The duration of the sustained PFM contraction was used as a measure of PFM endurance. To ensure that the comparison of hold time during the sustained contraction among the groups was not biased by the intensity of the contraction, the maximum VP from the sustained contraction was computed as described for the PFM MVCs. The maximum VP attained during the endurance task was then normalized to the maximum VP attained during the MVC trials. Both the raw and normalized values were compared among the groups using ANOVAs.

Hold time was calculated by subtracting the time at which the pressure rose above two standard deviations over the baseline level, from the time at which the pressure first dropped below 50% of the maximum raw VP recorded during the task. If the pressure did not drop below 50% of its maximum value over the 60 s recording, the contraction time was recorded as 60 s. Several women exceeded the 60 s hold time, creating a ceiling effect and therefore these data were not normally distributed. The contraction times were therefore compared among the groups using a Kruskal-Wallace test, $\alpha=0.05$.

### 6.4 Results

#### 6.4.1 Sample characteristics

Eighty-seven women (35 continent, 35 with mild SUI and 17 with moderate to severe SUI) participated. The groups were not different in terms of age ($p=0.95$).
The continent women had lower BMIs than the other two groups (p=0.014) and fewer of the continent women were parous (p=0.003). The continent women tended to have had fewer children (p=0.051). See Table 6.1 for the sample’s demographics.

Table 6.1 Demographic data by group.

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (years)</th>
<th>BMI (kg/m²)</th>
<th>Parous (#)</th>
<th>Parity (# births)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continent n=35</td>
<td>49.6 (9.6)</td>
<td>24.1 (3.3)</td>
<td>22</td>
<td>2 (0-4)</td>
</tr>
<tr>
<td>Mild SUI n=35</td>
<td>50.4 (9.9)</td>
<td>26.5 (5.1)</td>
<td>33*</td>
<td>2 (0-5)</td>
</tr>
<tr>
<td>Severe SUI n=17</td>
<td>50.2 (8.6)</td>
<td>27.9 (5.3)*</td>
<td>15</td>
<td>2 (0-4)</td>
</tr>
</tbody>
</table>

Values are given as mean (standard deviation) except for the values for Parous, which are the number of parous women in the group, and for Parity, which are given as median (range). * indicates that the group was significantly different from the continent group.

6.4.2 Voluntary PFM contraction strength

There were no significant differences among the groups for the maximum VP produced during the PFM MVCs (p=0.82). (See Figure 6.1.)

6.4.3 Involuntary PFM contraction strength

The maximum VP generated during coughing was higher than the maximum VP generated during the PFM MVCs (p<0.001). The continent women and those with mild SUI generated higher maximum VP amplitudes during coughing in standing than during coughing in supine (p<0.001 and p=0.011, respectively). There was no difference between the positions in the maximum VP amplitudes generated by the women with moderate to severe SUI during coughing (p=0.50).

There was a significant interaction between age and group on the maximum VP achieved during coughing in supine (p=0.028). (See Figure 6.2.) The group with mild SUI generated higher VPs (p<0.001), and demonstrated larger decreases in the
ability to generate VP with increasing age than the continent and moderate to severe SUI groups.

The effect of the interaction between age and group on the maximum VP achieved during coughing in standing was not significant (p=0.31), however, the main effects for age and group were both significant (p<0.001 and p=0.027 respectively) with older women generating lower VP during coughing in standing and the women with mild SUI generating higher VP than the other two groups (p<0.001 for both). (See Figure 6.3.)

Figure 6.1 Maximum vaginal pressure amplitudes during the PFM MVCs by group and age. The bars indicate the mean values and the whiskers indicate the 95% confidence interval for the mean. There were no significant differences among the groups or by age. Although age was a continuous variable in the ANCOVA, it was grouped into ten-year bins to improve the clarity of the graph. The number of subjects in each bin is given at the bottom of the corresponding bar.
Figure 6.2 Maximum vaginal pressure amplitudes during coughing in supine by group and age. The bars indicate the mean values and the whiskers indicate the 95% confidence interval for the mean. There was a significant age by group interaction, with the women with mild SUI producing the highest VP and being most affected by age (slopes: continent 0.059 (±0.116) cmH₂O/year, mild SUI -0.766 (±0.181) cmH₂O/year, moderate to severe SUI -0.181 (±0.183) cmH₂O/year). Although age was a continuous variable in the ANCOVA, it was grouped into ten-year bins to improve the clarity of the graph. The number of subjects in each bin is given at the bottom of the corresponding bar.
Figure 6.3 Maximum vaginal pressure amplitudes during coughing in standing by group and age. The bars indicate the mean values and the whiskers indicate the 95% confidence interval for the mean. The age by group interaction was not significant, but there were significant main effects for both group (* p<0.05) and age (slope −0.358 (±0.094) cmH₂O/year). Although age was a continuous variable in the ANCOVA, it was grouped into ten-year bins to improve the clarity of the graph. The number of subjects in each bin is given at the bottom of the corresponding bar.

6.4.4 Vaginal pressure rise time during coughing

The VP rise times were slower in supine than in standing (p=0.006); therefore the positions were analysed separately. The age by group interaction effect on VP rise time during coughing was not significant in either testing position (p=0.35 supine and p=0.19 standing). The main effect for group was also not significant (p=0.29 supine and p=0.34 standing). The main effect for age, however, was significant: supine: p=0.03, slope 7.92 (± 2.86) ms/year and standing: p=0.004, slope
9.72 (± 3.03) ms/year, indicating that older women took longer to generate maximum VP during coughing than younger women did. (See Figure 6.4.)

Figure 6.4 Vaginal pressure rise times during coughing in standing. The bars indicate the mean values and the whiskers indicate the 95% confidence interval for the mean. The age by group interaction and the group main effect were not significant, but there was a significant main effect for age (slope 9.72 (±3.03) ms/year). Although age was a continuous variable in the ANCOVA, it was grouped into ten-year bins to improve the clarity of the graph. The number of subjects in each bin is given at the bottom of the corresponding bar.

6.4.5 Duration of the sustained PFM contraction

There were no significant differences in the raw VP amplitudes achieved during the sustained contraction among the groups (p=0.22). There were significant differences among the groups in the intensity of the contraction that was achieved during the sustained contraction task relative to the PFM MVC: continent: 59.7% MVC (1.6 to 176), mild SUI 81.3% MVC (17.5 to 165.1) and moderate to severe SUI:
77.2% MVC (10.6 to 377.5), p=0.031. The women with SUI contracted at a higher level than did the continent women during this task. This difference was significant between the continent women and the women with mild SUI (p=0.045). There were no significant differences in the intensity of the held contraction between the women with moderate to severe SUI and either of the other two groups (p>0.96 for both).

There was no difference in the hold time among the groups: continent: 20.29 s (1 to 60), mild SUI: 12.08 s (1 to 60) and moderate to severe SUI: 32.56 s (1 to 60), p=0.22. Several women in each group were able to hold the contraction for the full 60 s (continent: 6/35, mild SUI: 5/35, moderate to severe SUI: 6/17).

6.4.6 Effect of age on PFM function

The correlation between maximum VP produced during the MVC and age was not significant. Although weak, the partial correlations between the maximum VP achieved during coughing in supine and standing and age were significant (r=-0.19, p=0.002 and r=-0.24, p<0.001, respectively), indicating that the ability to generate VP with a cough decreased with age. The Spearman’s correlation between age hold time was r_S= 0.05, indicating that the ability to hold a PFM contraction improved with increasing age.

6.5 Discussion

The key findings of this study are: women with SUI have PFMs that are as strong, if not stronger than those of women without SUI, women with SUI and continent women are equally able to generate VP quickly during coughing and the ability to
sustain a voluntary PFM contraction is not affected by continence status. These findings suggest that urine leakage in women with SUI is not, in general, related to PFM weakness or impaired static endurance. As women age, however, PFM strength and contraction speed appear to decline, which may increase the likelihood of SUI in older women.

6.5.1 Voluntary PFM contraction strength

The finding that the continent women and the women with SUI were able to generate similar rises in VP during PFM MVCs is consistent with the findings of Morin et al.\textsuperscript{196} but counter to the findings of Devreese et al.\textsuperscript{264} and Peng et al.\textsuperscript{227} A possible explanation for this discrepancy lies in the measurement procedures used. Devreese et al.\textsuperscript{264} measured VP using digital vaginal palpation, which may take other factors into account along with the squeeze force when a strength grade is assigned. The Peng et al.\textsuperscript{227} study did not subtract the resting or baseline activity from the VP. However, in a different study, this group did subtract the baseline pressure from their VP measurements and in that study they found no differences in maximum VP between women with and without SUI.\textsuperscript{253} It appears, therefore, that the total amount of PFM force (tonic and phasic) may be more important than the increase generated with contraction efforts (phasic only) when assessing differences in PFM function between women with and without SUI. In the measurement system used in the current study, baseline pressure is influenced by individual anthropometric differences in addition to differences in the resting pressure within the vagina and, for this reason, cannot be compared among the groups. Further research may
determine how the relative contributions of tonic PFM activity, PFM passive stiffness or reduced fascial support are implicated in SUI.

6.5.2 Involuntary PFM contraction strength

Both the continent women and the women with mild SUI generated higher rises in VP during the coughs performed in supine than the coughs performed in standing. This difference was not apparent in the women with moderate to severe SUI. It is possible that the normal response to coughing, in terms of VP generation, is different between supine and standing and that this response is maintained to some extent in the women with mild SUI but lost in women with moderate to severe SUI.

It was expected that the highest VP amplitudes would be generated during coughing in standing because of the added force of gravity acting on the bladder and of the higher peak flows that can be generated when coughing in standing, however, this was not the case. It may be that in standing, the effect of gravity tightens the fascial supports and, therefore, less phasic muscular activity is needed to maintain continence. It is clearly easier to cough in standing than it is in supine, therefore the women may also have exerted less expiratory force with their abdominal muscles when they coughed in standing than when they coughed in supine. Since abdominal muscle contraction, and the intra-abdominal pressure it generates, helps to create closure pressure by pressing the urethra against the anterior vaginal wall and the pelvic floor, if less forceful cough efforts were observed in standing the resultant rises in VP might be lower than that seen in supine. The volunteers were instructed to generate a maximum coughing effort on each trial in each position; however,
neither intra-abdominal pressure nor peak flow generated with each cough were recorded to confirm that coughs of equal force were achieved.

6.5.3 Vaginal pressure rise times during coughing

The pressure rise times observed during the coughs were significantly slower in supine than in standing but were not significantly different among the groups. This suggests that the influence of the PFM contraction and the fascial supports on the speed of VP generation during coughing may be different in supine compared to standing and that these responses are maintained in women with SUI.

The rise time calculation in this study did not take the time of the peak VP relative to the intra-abdominal pressure peak into consideration and so maintaining the ability to generate VP quickly does not mean that it is happening with optimal co-ordination. Others have found that women with SUI generate urethral pressure and VP later relative to the rise in intra-abdominal pressure as compared to continent women\textsuperscript{197,199} and such differences might explain why some women with SUI experience urine leakage.

6.5.4 Duration of the sustained PFM contraction

The women with mild SUI worked at a higher proportion of their maximal VP than did the continent women during the sustained PFM contractions. It is not clear why this occurred since all women were given exactly the same instructions for the task and the researcher was blinded to group assignment but this pattern may have confounded the results of the hold time analysis. The women with SUI held the sustained contraction at an intensity similar to that reported by Verelst and
Leivseth, who reported that both women with SUI and continent controls worked at 80% of their PFM MVC when asked to hold a sustained contraction and both groups held the contraction for similar durations.

Despite the potential bias caused by the differences in contraction level among the groups in the current study, there was no difference in PFM contraction time among the groups. This finding was not expected, especially so since the women with SUI were at a disadvantage because they were performing a stronger contraction.

Contrary to the results of the current study, Morin et al. and Deindl et al. found that continent women had significantly greater PFM endurance than did women with SUI, although the endurance tasks differed among the studies. In particular, Deindl et al. defined endurance as the time between the onset and the end of the EMG signal recorded from the PFMs using fine wire electrodes. As VP during a PFM contraction is the product of both abdominal and PFM activity, as well as passive stiffness, it is possible that the women in the different groups in the current study were using different motor control strategies to maintain VP. Concurrent intra-abdominal pressure and EMG measures would be needed to determine whether there were differences in the muscle strategies used: for example, whether women with SUI rely more heavily on their abdominal muscles than their PFMs to sustain VP during a sustained vaginal squeeze task.

There was a ceiling effect in the hold times recorded during the sustained PFM contraction in this study as a sizable proportion of women in each group were able to hold a PFM contraction for at least 60 s. One minute was chosen as the upper end
of our recording time because several authors had reported that women with SUI had PFM contraction endurance times of less than 15 (SD 2) s,\textsuperscript{65,284,285} and because a maximal voluntary contraction of most skeletal muscles cannot be sustained for more than five seconds.\textsuperscript{286} Despite this limitation, there was no apparent difference between women with and without SUI.

### 6.5.5 Effect of age on PFM function

The maximum VP generated during the PFM MVCs was not affected by age. These results are similar to those of Trowbridge et al.\textsuperscript{107} who also found that the ability to generate PFM force with a voluntary PFM contraction was not influenced by age.

During coughing, the correlations between age and peak VP were negative for all three groups in both positions. These negative correlations were expected given the strength declines seen with age in other skeletal muscles.\textsuperscript{287} Although these correlations were relatively low (r≤-0.25 for both positions), even a small decrease in the ability to generate VP may contribute to functional losses in women with other deficits in the continence mechanism.

The finding that age did not affect the ability to perform a PFM MVC, while it did impact the ability to generate VP during coughing, may be due to women not being able to generate a true maximum PFM contraction voluntarily. In the current study, the volunteers generated significantly higher maximum VP values during coughing than they did during the PFM MVCs.

In the context of the current study, the slower rise in VP seen with increasing age may be a reflection of changes in muscle twitch contraction time and consequently
may reflect differences in muscle fibre type composition as opposed to coordination changes with age. Slowed contraction times combined with reduced ability to generate high VP in older women is likely to contribute to urine leakage during coughing and, perhaps, other perturbations.

The correlation between the duration of the sustained contraction and age was weak but significant. Since with aging there is a gradual conversion of muscle fibre type towards having slow twitch characteristics,\textsuperscript{288} this result is not surprising. The functional relevance of this finding is, however, not known since this type of sustained contraction is not one that would be done during many functional activities. It should be noted that it is not known whether the contraction time measured in the current study was due to muscle fatigue, loss of attention or proprioceptive deficiencies so the interpretation of this result should be made with caution.

6.5.6 Limitations

It is likely that SUI has different aetiologies: SUI that is the result of urethral sphincter dysfunction or fascial damage may result in increased PFM activity and force, while SUI that is the result of PFM injury may result in decreased PFM activity and force.\textsuperscript{86} The sample recruited in this study may have had a predominance of women who have compensated for damage to other structures in the continence mechanism by increasing the strength of their PFMs. Future studies should incorporate imaging of the PFMs to determine whether or not there are PFM or fascial defects and participants should be stratified based on evidence of such damage.
It should be remembered that VP is generated by the combined efforts of the PFM and the abdominal muscles,\(^\text{139}\) and even the diaphragm.\(^\text{145}\) Therefore, as only vaginal pressure measurements were made in this study it cannot be assumed that there is no difference in PFM activity among the three groups. The women with SUI may be using their abdominal muscles to compensate for weak PFMs in the generation of VP and this adaptation may not effectively prevent leakage episodes.

### 6.6 Conclusions

There were no differences between women with and without SUI in their ability to generate VP during a maximum voluntary PFM contraction or to sustain a voluntary PFM contraction; however, women with mild SUI produce higher VP amplitudes during coughing than continent women. The women with SUI may have demonstrated a training effect. If women with SUI habitually use their PFMs to a larger extent than continent women in an attempt to compensate for other deficits and to limit urine loss under conditions of increased intra-abdominal pressure, this might result in training effects whereby they have an enhanced ability to generate VP and/or to sustain VP for longer periods of time.

Increasing age was associated with lower maximum VP s recorded during coughing and with slower rates of pressure generation during coughing. The implication of this decline in the PFM’s force generating speed and capacity may be functionally significant.

These findings suggest that PFM training may help to compensate for other defects (i.e. fascial damage) and may improve the speed at which VP can be generated through strengthening the neural pathways and improving the metabolic
efficiency of the contraction. The findings of the present study support the recommendations that PFM exercise programs include specific training for coordination and power.42,44
Chapter 7 General Discussion and Future Perspectives

Taken together, the findings of the studies presented in this thesis suggest that a decreased ability to activate the PFMs and diminished PFM endurance may not be primary features of SUI. Although voluntary PFM activation levels were lower in the women with SUI, there was no difference in PFM activation levels between the women with and without SUI during coughing nor was there a difference between the women with and without SUI in the ability to sustain a PFM contraction. Rather, these studies suggest that delayed pressure transmission: evidenced by higher EMG-axis intercepts on the vaginal pressure versus EMG curves, and altered motor control: evidenced by altered muscle activation timing, may play a greater role in SUI. Such differences may be the result of fascial tearing, muscle damage or changes in muscle fibre type related to prior injury or aging.

The results of the first study in this thesis (Chapter 3) showed that changes in urethral pressure were reflected in proportional changes in vaginal pressure during both voluntary PFM contractions and coughing. This relationship was found in a mixed group of women that included women with stress and urge urinary incontinence as well as in continent women. It was therefore concluded that vaginal pressure measures are a valid surrogate for urethral pressure measures during these manoeuvres. The results of the first study were consistent with the findings of two earlier but methodologically poorer studies.\textsuperscript{41,218} The earlier studies measured vaginal pressure and urethral pressure on different days and measured vaginal
pressure with digital palpation, which is less precise and may take other factors into account when assigning a strength grade.

The subsequent three studies (Chapters 4 to 6) used vaginal pressure and abdominal and PFM EMG to compare the biomechanics and motor control of the extrinsic continence system between women with and without SUI during voluntary PFM contractions and coughing. The results of these studies suggest that PFM weakness, although it has been contended by some,\textsuperscript{128, 179, 193-195} is not the primary deficit in SUI, as these studies showed that the women with SUI were able to generate peak vaginal pressure and peak PFM EMG amplitudes that were not different from those generated by the continent women. Instead, the results suggest that alterations in motor control contribute to SUI as the order of abdominal and PFM activation was altered and that pressure transmission to the vagina was delayed and more gradual in the women with SUI compared to the continent women. These findings are consistent with those reported by Barbic et al.,\textsuperscript{202} who found that women with SUI had delayed PFM activation and reduced pressure transmission to the urethra. They are also consistent with the findings of Morin et al.,\textsuperscript{196} who found that women with SUI were equally able to generate maximum PFM force but less able to generate PFM force quickly, and Smith et al.,\textsuperscript{146, 147, 207} who found that PFM EMG activation was delayed during postural tasks in women with SUI but that they generated PFM EMG activation levels that were the same as or higher than continent women.

These studies are unique in that they are the first to measure PFM EMG and vaginal pressure simultaneously. This simultaneous measurement allowed us to
describe how the abdominal muscles and the PFMs are active generators of vaginal pressure and how pressure transmission to the vagina is delayed in women with SUI. These findings add detail to the descriptions of the synergistic abdominal muscle activity that occurs with PFM contractions previously described by Bø et al., Sapsford and Hodges and Neumann and Gill. These findings are consistent with research on the respiratory and postural roles of the PFMs that found that the PFMs and abdominal muscles contract synergistically during breathing, rapid arm movement and balance tasks and that PFM activation is delayed in women with SUI. These results also support the recent anatomical research that suggests that fascial defects, rather than PFM damage, predominate in women with SUI.

A strength of the studies described in this thesis is the use of both voluntary and involuntary PFM contractions to assess differences between women with and without SUI. Voluntary PFM contractions are the most common clinical measure of PFM function and are the major component of PFM exercise therapy for SUI. However, as they are not performed during activities of daily living, the muscle activity patterns generated during these contractions may not be generalizable to functional tasks. Coughing is associated with involuntary PFM contractions that are proportional to the forcefulness of the cough. Therefore, by asking the participants in these research studies to cough maximally and to perform PFM MVCs, it was possible to compare intense voluntary and involuntary PFM contractions. Studies two through four (Chapters 4 to 6) found the delays in vaginal pressure generation were greater in women with SUI than in continent women.
These findings may reflect fascial defects in women with SUI,\textsuperscript{162, 183, 248} which may result in there being more slack in the PFMs that requires a certain amount of muscle contraction to tighten, resulting in longer times before contractile output results in force generation. The PFM MVC and cough studies also provide evidence that different motor control strategies are used to generate vaginal pressure during voluntary and involuntary PFM contractions as the muscle activation timing and vaginal pressure versus EMG curves were different during the two types of contraction. The results support our decision to examine both types of contraction and suggest that future research examining PFM synergies should look at a broad range of functional tasks and should take care not to generalize from the tasks examined to other tasks.

The utility of the different measures used in this work to evaluate the function of the extrinsic continence mechanism is discussed in detail below.

\textbf{7.1 Measures used for evaluating the function of the extrinsic continence mechanism}

\textbf{7.1.1 Urethral pressure}

In women with stress urinary incontinence, the primary deficit leading to urine leakage is reduced urethral closure pressure, particularly during conditions that provoke involuntary PFM contractions;\textsuperscript{101, 149, 200} however, the aetiology of this reduction in urethral closure pressure remains unclear. Damage to both the intrinsic continence mechanism (urethral smooth muscle and striated urethral sphincter) and the extrinsic continence mechanism (PFMs and pelvic fascia) have been reported in the women with SUI.\textsuperscript{155, 157, 162, 172, 173, 176} Damage to the extrinsic
continence mechanism manifests as a decreased ability of the PFMs to compress the urethra\textsuperscript{150,179} and as a diminished ability of the PFMs and pelvic fascia to resist increases in intra-abdominal pressure (IAP) resulting in decreased IAP transmission to the urethra.\textsuperscript{87} DeLancey et al.\textsuperscript{149} have reported that maximum urethral closure pressure is the single greatest predictor of SUI and that adding measures of fascia support, PFM function and cough strength did not significantly improve the predictive ability of the model. The authors suggest that this indicates that the dysfunction in SUI is primarily in the intrinsic continence system. However, urethral closure pressure is not solely a measure of urethral smooth and striated muscle sphincter function; on the contrary, it is a measure of the combined function of the intrinsic and extrinsic continence systems. Therefore, the results of this study do not suggest that that the primary dysfunction in SUI is in the urethral sphincters, rather they support the idea that SUI results from a combination of deficits in the intrinsic and extrinsic continence systems.

Despite being routinely performed to assess SUI in women, the measurement of urethral pressure to study continence mechanics is problematic on several levels: urethral pressure measurements are highly variable and have poor test-retest reliability;\textsuperscript{213,289} the procedure is uncomfortable and it carries a not insignificant risk of urinary tract infection,\textsuperscript{213,290} and the procedure makes the recruitment of research volunteers more difficult,\textsuperscript{291} which was borne out by our experience recruiting volunteers for the first study (Chapter 3). Inserting a urethral catheter is also beyond the scope of practice of physiotherapists in Ontario,\textsuperscript{292} making it impractical for clinicians and requiring either additional staff (nurses or physicians)
or extensive training and delegation from a physician for researchers. Despite these difficulties, it was essential to know how urethral pressure is related to vaginal pressure to determine if vaginal pressure is a suitable surrogate for urethral pressure measures in biomechanics research and PFM rehabilitation. This was of particular importance in the current work for two reasons: (I) many physiotherapists measure vaginal pressure to determine whether women can perform adequate PFM contractions to increase urethral pressure and (II) much of the research literature reports the use of vaginal pressure to study PFM contractions based on the assumption that vaginal pressure reflects urethral pressure.219

There are three principal types of catheters used to measure urethral pressure: fluid filled, microtip or fibre-optic, and balloon catheters.214, 255, 289 The primary advantages of fluid filled catheters are: they are less prone to movement artifact, they are less affected by the orientation of the ports and they are relatively inexpensive. As well, as long as a relatively small (10 French or smaller) catheter is used, the measured pressure is very close to the actual urethral pressure.255 The primary advantage of microtip and fibre-optic catheters is that they are able to measure very rapid pressure changes. The disadvantages of microtip and fibre-optic catheters are that the measured pressure is influenced by the stiffness of the catheter and the orientation of the transducer, and that they are expensive. As well, they do not measure urethral pressure directly, rather they measure the normal stress component on the surface of the transducer.289 Balloon catheters or air-filled catheters are also expensive and if they are over inflated initially they dilate the
urethra resulting in an overestimation of urethral pressure. In addition, if the balloon is too long, pressure variations along the urethra are diminished because the measured pressure is the average pressure along the entire section of the urethra that surrounds the balloon.\textsuperscript{255}

In Chapter 3, 7 French, fluid-filled catheters were used to measure urethral pressure. This system was chosen because it is able to measure pressure in a small section of the urethra with relatively little risk of either dilating the urethra or of movement artifact, it was less expensive than other systems and the impulse response was relatively rapid. The system was well tolerated by the volunteers; although most found the insertion of the catheter somewhat uncomfortable, once the catheter was in place the majority of the volunteers reported that any irritation from insertion resolved.

\textbf{7.1.2 Vaginal Pressure}  

As noted above, vaginal pressure is the most commonly used measure of PFM strength in women, both in PFM rehabilitation and research,\textsuperscript{219} as the PFMs attach to the lateral vaginal walls\textsuperscript{293} and the vagina is the most convenient orifice to access.\textsuperscript{294} Vaginal pressure measurements have an advantage over anal pressure measures because the external anal sphincter can maintain normal anal pressure levels without contributions from the PFMs.\textsuperscript{295}

Vaginal pressure is produced by the PFMs and through IAP transmission;\textsuperscript{219} therefore, it is not possible to distinguish between a properly performed PFM contraction and a Valsalva manoeuvre using the magnitude of the pressure rise alone.\textsuperscript{141} The vaginal pressure increases seen with a Valsalva manoeuvre may be
due to PFM contraction in the effort to resist the caudal movement of the pelvic organs, or due to compression of the vagina by IAP against the resistance to movement produced by passive tension in the PFMs and the pelvic fascia. Therefore, in the studies described in this thesis the volunteers were observed during all PFM contractions, both voluntary and involuntary, to ensure that they were not bearing down.

7.1.3 Vaginal pressure as a surrogate for urethral pressure

In study 1 (Chapter 3), no difference in peak vaginal pressure between anterior and posterior vaginal recording sites were found during either PFM MVCs or coughs. The fact that significant linear correlations were found between vaginal and urethral pressure changes during all tasks suggests that rises in vaginal pressure are valid representations of the influence of PFM contraction on urethral pressure. It was concluded that the PFMs’ role in the extrinsic continence mechanism could validly be measured by intravaginal pressure transducers in PFM rehabilitation and biomechanics research. It should be noted, however, that the change in pressure induced by PFM contraction does not reflect the overall pressure in the urethra as the baseline values were removed to be consistent with the vaginal pressure measurements and because baseline urethral pressure is difficult to standardize.

Other recent studies have used urethral closure pressure (urethral pressure minus bladder pressure) to assess continence function. Urethral closure pressure is also very difficult to standardize and there is so much overlap between the closure pressures produced by women with and without SUI that they cannot be used to classify the groups. As well, urethral closure pressure does not include
IAP, because IAP also acts on the bladder and is, therefore, subtracted out. As vaginal pressure includes contributions from IAP, and abdominal pressure transmission is important to urinary continence, it was decided to leave the IAP contributions to urethral pressure in the measure.

7.1.4 PFM EMG

The Femiscan™ vaginal EMG probe was chosen for this research because it was the only commercially available vaginal EMG probe with a bipolar electrode configuration to record from each side of the PFM. Several groups have found that it demonstrates good to excellent between-trial reliability. The between-day reliability is not always as good, which may explain why no differences in PFM EMG amplitudes were found between the groups in Chapter 5. The Femiscan™ probe was also chosen as it maintains a constant intra-electrode distance and, as long as the flange at the bottom of the probe was in contact with the perineum, a standard electrode depth.

The cylindrical barrel of the Femiscan™ probe does make the probe susceptible to movement during coughing and tasks performed in standing. Probe movement was minimized by having each volunteer wear her underpants and by having the volunteer hold the probe with her hand if movement artifact was observed in the tracings. Sensory cues provided by the probe may have increased awareness of the PFM contraction and may have increased PFM contraction levels. This is unlikely to have influenced the results, however, because it would likely have systematically affected all of the participants.
There was a difference in PFM EMG amplitudes between the two sides of the PFM; this finding replicates the findings of Brown\textsuperscript{239} who recorded PFM EMG data using both the Femiscan™ probe and fine-wire EMG electrodes. This was most likely due to the position of the electrodes relative to the PFM as no difference in the patterns or timing of activation between the two sides was found (Chapters 4 and 5), or it may have been related to differences in PFM thickness between the two sides.\textsuperscript{175}

Normalization is often considered necessary to be able to compare EMG amplitudes among individuals because the EMG amplitude can be affected by electrode placement and anthropometric differences.\textsuperscript{296-298} In the studies presented in this thesis, the initial intent was to normalize the abdominal EMG data to the peak EMG amplitude achieved for each muscle during tasks chosen to selectively activate each muscle. This ended up not being appropriate because there were systematic between-group differences in the peak activations achieved: the women with SUI demonstrated higher peak abdominal EMG activation, despite having higher body mass indices (BMIs), than the continent women. Under this circumstance, normalization would have lowered the relative activation of the abdominal muscles in the women with SUI that, in turn, could have biased the group differences found during statistical testing. Therefore, throughout this work abdominal EMG data were not normalized; instead, subject was included as a random factor in the mixed-model analyses of variance (ANOVAs) to account for inter-individual differences.\textsuperscript{299}

Cross-talk is the extent to which EMG electrodes record muscle activation from muscles other than the muscle of interest. Most reports of studies using PFM EMG
have not commented on whether or not the PFM EMG signals were tested for crosstalk. Sapsford et al. observed that surface EMG activity recorded from the gluteal and hip adductor muscles during PFM MVCs decreased or remained constant and concluded that the PFM EMG signals were unlikely to include crosstalk. During the pilot stages of this research, the extent to which the Femiscan™ vaginal EMG probe recorded crosstalk during voluntary PFM contractions was investigated by asking eight healthy, continent volunteers to perform PFM contractions, both in isolation and in combination with maximal, isometric abdominal and hip muscle contractions. Overall, there was no increase in the maximum PFM EMG amplitude with the addition of contractions of the hip musculature, although in two of eight volunteers adding the abdominal muscle and hip external rotator contractions did increase the maximum EMG amplitudes recorded from the PFMs, which likely indicated that crosstalk was being recorded.

Auchincloss and McLean recorded PFM EMG activity simultaneously with the Femiscan™ probe and fine-wire electrodes during rest and during three resisted, isometric hip contractions: adduction, external rotation and internal rotation. The results of this study showed increased PFM EMG activity compared to rest during the hip adduction and hip external rotation tasks with the Femiscan™ probe, while increased PFM EMG activity was recorded only during the hip external rotation task with the fine-wire electrodes. The normalized activation amplitudes (relative to a PFM MVC performed in isolation) were much higher in the EMG recordings made using the Femiscan™ probe as compared to the fine-wire electrodes. This suggests that the Femiscan™ probe recorded crosstalk during both the hip adduction and
external rotation tasks. Both of these studies suggest that crosstalk is recorded by the Femiscan™ probe during relatively high intensity contractions. Therefore, during all data collection sessions performed in this body of work the volunteers were observed for breath holding, straining, visible abdominal or hip girdle muscle contraction, or movement of the hip joints. When signs of extraneous muscle contraction were observed the volunteer was coached to avoid the extra contraction and the trial was repeated. As well, the effect of recording crosstalk would have been the same in the three groups (continent women, women with mild SUI and women with moderate to severe SUI) so the differences between the groups in PFM EMG are not likely to be the product of crosstalk.

Another possibility is that obturator internus and the PFMs contract synergistically. This is suggested by the PFM fine-wire EMG recordings reported by Auchincloss and McLean in the study discussed above, since even the fine-wire EMG recordings made from the PFMs demonstrated an increase in activation amplitude during the hip external rotation contraction. It is logical that contraction of the obturator internus muscle might facilitate PFM contraction, because the PFMs attach laterally to the obturator internus fascia. It is not possible to determine whether this increase in PFM EMG activity is the result of co-contraction or crosstalk from the research that is currently available.

It is only possible to conclusively determine if crosstalk is being recorded by individually electrically stimulating each of the hip muscles while recording PFM EMG. However, the individual hip external rotators are very difficult to reach to stimulate as both the muscles and their nerve supplies lie deep to larger muscles or
within the bony pelvis. It is also not possible to stimulate any of the hip external rotators in isolation at the spinal root level (L3 to S1), as they share spinal innervation levels with the PFMs (S1 to S4), and, unlike most skeletal muscles, the PFMs are connected to multiple reflex arcs that involve the low thoracic to the sacral spinal nerve levels. Therefore, the PFMs may still receive reflex stimulation even if the nerve root stimulated is above the levels that directly innervate the PFMs. Even so, studies have found differences in PFM EMG amplitudes between women with and without SUI, and increases in EMG amplitudes following PFM rehabilitation are associated with improved continence status, suggesting that PFM EMG gives sufficient information about the PFMs to be useful for biomechanical research into the continence mechanism.

7.1.5 Intra-abdominal pressure

In the studies presented in Chapters 4 and 5, intra-abdominal pressure was not measured directly, rather proximal vaginal pressure was used as a surrogate, as Al-Taher et al. have reported that pressure measured in the posterior vaginal fornix is a reliable surrogate for IAP measured in the rectum. Direct measurement of IAP would have required that a pressure transducer be situated in the stomach via a tube or a swallowed sensor, or in the rectum, but it was felt that requiring the volunteers to have an IAP transducer inserted via another orifice would make it more difficult to recruit volunteers. The shape of the Femiscan™ probe facilitated positioning a pressure transducer in the proximal, posterior vagina as the angle of the probe relative to its flange ensured that the posterior surface of the probe rested against the posterior vaginal wall and the length of the probe was such that, when
inserted, the tip rested in the proximal third of the vagina. Therefore, the decision was made to mount a pressure transducer on the top of the Femiscan™ probe to measure proximal vaginal pressure as a reflection of IAP in order to assist the recruitment of volunteers without compromising the information obtained.

7.2 Differences in voluntary, involuntary and endurance functions of the PFM among women with mild SUI, women with moderate to severe SUI and continent women

7.2.1 Voluntary PFM contractions

Both groups of women with SUI produced lower peak PFM EMG activation during the supine PFM MVCs than did the continent group (Chapter 4); however, they were still able to produce the same peak vaginal pressures (Chapters 4 and 6), suggesting that women with SUI may use different motor control strategies to generate vaginal pressure during PFM MVCs than do continent women. The women with moderate to severe SUI produced lower EO and IO EMG amplitudes than the continent women. They also produced lower RA amplitudes; however, it did not reach statistical significance. The women with mild SUI produced a similar pattern of abdominal muscle activation. This suggests that the women with SUI were not using IAP transmission to compensate for weak PFMs. Histological findings suggest that most of the damage to the PFMs in women occurs medially. It is possible that the women with SUI used the larger, more lateral segments of the PFMs to generate vaginal pressure, which are presumably able to generate more force output than the smaller, medial segments. Since the lateral segments of the PFMs do not attach to the vaginal wall and the EMG signals from this portion of
the PFMs may be subject to more tissue filtering, this may explain why lower PFM EMG amplitudes were recorded from the women with SUI.

In the vaginal pressure versus EMG ensemble average curves there were no differences in the shapes of the curves among the groups for each muscle during the PFM MVCs (Chapter 4). The women with moderate to severe SUI had higher EMG-axis intercepts than did the continent women for both EO and IO, while the women with mild SUI had higher EMG-axis intercepts only for IO, suggesting that, although the overall motor activation pattern is preserved, the women with SUI had to contract their abdominal muscles to a greater degree before the pressure was transferred to the vagina than did the continent women. This finding may reflect structural damage to the supporting fascia in many of the volunteers with SUI. To the author’s knowledge, the only other published research on PFM and abdominal muscle EMG activation patterns during voluntary PFM contractions found that the women with SUI had lower PFM EMG activation levels and higher abdominal muscle activation levels. These results contradict the results of our study and they may be due to different analyses or they may suggest that the volunteers in the two studies may have had different aetiologies for their SUI. In future studies, either ultrasound or MRI should be employed to determine the role of structural defects in delaying the transmission of pressure generated by the abdominal muscles to the vagina.

7.2.2 Involuntary PFM contractions

There was no difference in the peak PFM EMG or vaginal pressure amplitudes produced among the groups during coughing (Chapters 5 and 6). Since involuntary
contractions more accurately reflect PFM activity during activities of daily living that normally result in urine leakage than do voluntary PFM contractions, it is clear from these findings that not all women with SUI demonstrate functional PFM weakness. This is consistent with recent ultrasound imaging studies that suggest that SUI is not associated with major PFM abnormalities.\textsuperscript{183}

In the vaginal pressure versus EMG ensemble average curves there were no differences in the shapes of the curves among the groups during the coughs. For the vaginal pressure versus PFM EMG curves, the women with moderate to severe SUI had higher EMG-axis intercepts than the continent women and the women with mild SUI in supine. The women with mild SUI had the higher EMG-axis intercepts than the other two groups in standing. This suggests that involuntary PFM activation patterns differ with the severity of the incontinence and is consistent with the findings of Smith et al.\textsuperscript{147} who found that PFM activation levels differed between women with mild and more severe SUI during a load catching task. For the abdominal muscles, the women with moderate to severe SUI had the highest EMG-axis intercepts and the women with mild SUI had higher EMG-axis intercepts than the continent women. This suggests that, although the overall abdominal motor activation pattern is preserved, compared to the continent women the women with SUI had to contract their abdominal muscles to a greater degree before pressure was transferred to the vagina. As the severity of the incontinence increases so does EMG-axis intercept which may reflect a delay in pressure transmission to the vagina.

In Chapter 5, the importance of the unusual distal vaginal pressure versus PFM EMG curves that were not included in the primary ensemble average curves is
difficult to determine. Unusual patterns were not consistently performed by any given volunteer, but instead reflect different control patterns occurring occasionally within volunteers, suggesting that the motor patterns were highly variable even in this involuntary task. Overall, the unusual curves were linear with slopes of nearly zero, indicating that the PFM contraction was maintained at a given level throughout the vaginal pressure increase. Unfortunately, the volunteers were not asked to report urine leakage during the coughing tasks so it was not possible to determine if these unusual curves were produced when women were fearful of urine leakage or had the sensation of urine leakage. It might be that the unusual curves may represent trials during which the volunteers were bracing a pre-contraction to avoid urine leakage. This should be included in future work. Smith et al.\textsuperscript{147} reported that women with SUI leaked urine during a load catching task but they did not report whether the PFM activation patterns were different between trials during which leakage occurred compared to those trials in which it didn’t.

7.2.3 PFM endurance

In Chapter 6, the women with SUI held the sustained PFM contraction at a higher percentage of their PFM MVC than did the continent women, which should have put them at a disadvantage for being able to hold the contraction for an extended time. However, there was no difference in the ability to hold a PFM contraction among the groups. This suggests that there may be a training effect in the women with SUI: they may habitually use their PFMs to compensate for fascial damage or sphincter dysfunction in an attempt to prevent urine leakage. Smith et al.\textsuperscript{147, 207} found that women with SUI had higher PFM activation levels during static standing and load
catching tasks than did continent women, suggesting that women with SUI use their PFM s to compensate for other defects. Further investigation is required to describe these strategies and to determine if they are effective compensatory strategies.

Contrary to the results of the current study, other studies have found that women with SUI had significantly less PFM endurance than continent women.\textsuperscript{179, 196, 280} This may be the result of several factors: the use of different endurance tests, varying SUI aetiologies among the participants or volunteers using different motor control strategies to maintain the contraction. Further research, using different tasks and multiple measures of continence function (abdominal and PFM EMG, vaginal pressure and imaging), is necessary to determine how PFM endurance is involved in SUI.

\textbf{7.2.4 Effect of age on PFM function}

Previous research has found that continent women maintain the ability to generate PFM EMG and vaginal pressure during PFM MVCs with increasing age;\textsuperscript{107, 128} however, this ability decreases with increasing age in those with SUI.\textsuperscript{128, 193, 281} Our finding that women with SUI maintained the ability to produce peak vaginal pressure with PFM MVCs (Chapter 6) is, therefore, inconsistent with the literature and may be due to effort or to the underlying pathophysiology in the study groups. The ability to perform PFM MVCs may be maintained in women in whom the primary deficit is damage to the pelvic fascia and it may decline in women in whom PFM damage or denervation is more apparent. Although, to the author’s knowledge, there have been no reports of the rate of conversion of PFM fibres from type II to type I with age in women with or without SUI, it is known that PFM denervation
followed by reinnervation is a feature of SUI,\textsuperscript{159, 176, 184, 185, 187-189, 288} and that in individuals with post-poliomyelitis syndrome, another condition marked by denervation followed by reinnervation, the rate of conversion from type II to type I muscle fibres is accelerated compared to age matched controls.\textsuperscript{307} Further research, including the use of imaging to differentiate between women with PFM defects and those with fascial defects and the use of PFM biopsies to describe the muscle fibres, is necessary to explain this finding.

It was also demonstrated in Chapter 6 that the peak vaginal pressure produced during coughing decreased with age. This effect was most evident in the women with mild SUI. This is consistent with the literature in terms of the effect of age on voluntary PFM contractions\textsuperscript{107, 118, 128, 193, 281} and, as involuntary PFM contractions more accurately replicate PFM activity during activities of daily living, it may help to explain why SUI tends to become more severe as women age.\textsuperscript{2} This decline in the ability to generate vaginal pressure was seen during coughing and not during the PFM MVCs because, with aging, there is a shift towards having more type I muscle fibres in other skeletal muscles,\textsuperscript{287} which would particularly affect tasks, such as coughing, that require that the rapid generation of large forces. Since most women generate higher PFM activation during coughing than during PFM MVCs, the effect of age may not have been observed during the PFM MVCs because they may not have been true maximum contractions. Another possibility is that coughing generates large IAPs that are normally transmitted to the urethra and the vagina.\textsuperscript{87} If the pelvic fascia were damaged and could not provide effective resistance to the downward force induced by the IAP increase, the pelvic organs would descend and a lower
percentage of the IAP would be transferred to the urethra and vagina, resulting in lower peak vaginal pressure.\textsuperscript{97, 110, 162}

### 7.3 Application of thesis findings to exercise therapy for SUI

The findings in this thesis suggest that the reason PFM exercise has been shown to be effective in the reduction of the signs and symptoms of SUI (Level of Evidence I)\textsuperscript{12} is not because the PFMs are weak to begin with. Rather, PFM exercise may be effective through enhancing neuromuscular efficiency to compensate for fascial defects that delay pressure transmission\textsuperscript{202} and training or retraining motor patterns. Improved motor control may explain why performing a voluntary PFM contraction prior to coughing, “the Knack”, reduces urine leakage:\textsuperscript{43, 44} such pre-contractions may support the bladder neck, reducing the opening forces acting on the urethra and increasing abdominal pressure transmission. Teaching women with SUI to contract their PFMs quickly and forcefully, and practicing these contractions to encourage functional carryover, may retrain neuromuscular efficiency. Skilling and Petros\textsuperscript{55} finding that women who became continent with PFM exercise were continent even without performing a voluntary PFM contraction prior to sneezing or coughing supports this theory.

Pelvic floor muscle strength training may be necessary to compensate for laxity in the pelvic support structures,\textsuperscript{162, 183, 248} decreased resting pressure\textsuperscript{208, 227} and delays in pressure transmission.\textsuperscript{197, 199} For if resting vaginal pressure is low, the PFMs may need to generate more force, more quickly to generate sufficient pressure to exceed the increase in intra-abdominal pressure and prevent urine leakage.
The author has previously developed a theoretical model of SUI and the rationale for treating SUI with exercise that postulated that deficits in fascial support and motor control, along with damage to the PFMs and the pudendal and levator ani nerves, may be implicated in SUI and that PFM exercise might achieve its effect through strengthening the PFMs to compensate for fascial deficits and through training more effective motor control patterns. The research presented in this thesis supports these concepts. Further research is necessary to determine how the effects of PFM exercise on resting pressure, PFM EMG amplitudes, PFM strength and morphology are related to the effects of PFM exercise on urethral opening under stress conditions and on the signs and symptoms of SUI in order to understand the mechanism through which PFM training reduces urine leakage in women with SUI.

7.4 Study limitations

7.4.1 Sample

One limitation of the studies comparing women with and without SUI (Chapters 4 through 6) was that the groups were determined based on symptoms (presence and severity of SUI symptoms) and not on the deficits underlying the UI symptoms. Therefore, it is not possible to know the underlying cause of SUI in the volunteers who participated in these studies. Some may have had fascial defects, some may have had muscular defects, some may have had sphincteric deficits and some may have had a combination of these deficits, which may have increased the variability in the data. Future research should stratify women by the type of deficit present as determined by ultrasound or MRI.
structural damage may affect the performance of the PFMs and the compensatory strategies employed.85

7.4.2 PFM function tests

A second limitation of these four studies was the selection of tasks: PFM MVC, cough, repeated PFM contractions and sustained PFM contraction, used to test PFM function. They were chosen to conform with the tasks used in the literature, and to represent various aspects of PFM function: voluntary and involuntary strength, endurance and to assess activation timing.272 However, each task had limitations related to standardization and generalizability to activities of daily living.

The PFM MVC is used in biomechanical research on PFM function as if it is analogous to the isometric MVCs produced by limb muscles. However, the PFM MVC does not seem to be either truly maximal, as PFM contraction during coughing produces higher EMG activation amplitudes and urethral and vaginal pressures,101,148 nor is it isometric, as the contraction produces flexion of the coccyx and the whole muscle lifts to a higher position within the pelvis.33,116,118,249 PFM MVCs are also not functional. Although they are commonly used as a strengthening exercise and as a compensatory technique to prevent urine leakage in women with SUI,12 they are not normally performed during activities of daily living and some continent women are not able to voluntarily contract their PFMs even though they produce normal involuntary activation.37,39 It is also difficult to define at what point a maximum PFM contraction is achieved: should it include the contractile activity of the PFMs only to the point when synergists are recruited, or should the full PFM-abdominal-hip muscle synergy be included? The results in Chapter 4 found that the
continent women activated IO prior to the PFMs, suggesting that it may not be possible to initiate a PFM contraction without also activating at least the abdominal synergists. Although the preliminary studies have suggested that performing a voluntary accessory muscle contraction with a PFM contraction does not increase vaginal pressure over the PFM contraction alone, there are theories that co-contraction of the deep back and abdominal muscles, the diaphragm and the hip rotator and adductor muscles with the PFMs is necessary to support the lumbar spine, maintain postural stability, transfer forces between the axial skeleton and the lower extremities, and to support the pelvic organs. Initial studies have demonstrated that PFM activity is coordinated with breathing and postural tasks and that the postural responses of the PFMs and balance are altered in women with SUI. This suggests that, like the diaphragm, the PFMs may be components of multiple muscle synergies that may be automatically recruited to meet the demands of the task at hand and that it may be neither possible nor desirable to prevent the use of the accessory muscles. Further research is necessary to fully describe the synergies between the PFMs, the trunk muscles, the hip girdle musculature and the diaphragm, and to determine how they differ between women with and without SUI.

Voluntary coughs are frequently used to test involuntary PFM function. Voluntary coughs have the advantage of being very easy to perform; however, it is not known if the PFMs respond the same way to a voluntary cough as they do to a spontaneous cough. It is also difficult to standardize the forcefulness of a cough and, as the response of the PFMs is
proportional to the forcefulness of the cough, differences in PFM contraction between women with and without SUI may result if one group coughs less forcefully than the other. Also some women with SUI naturally perform a voluntary or spontaneous PFM pre-contraction to limit urine loss, despite not being instructed to do so.

It would be ideal to test involuntary PFM function during the functional tasks that most commonly provoke urine leakage: spontaneous coughing, sneezing and laughing, running, jumping and lifting. Unfortunately, there are technical difficulties with eliciting spontaneous coughs and sneezes and, as people do not all respond to a stimulus in the same manner, it would be very difficult to standardize the task. Eliciting spontaneous laughter also presents technical challenges in providing the appropriate stimulus for each person’s sense of humour and standardizing the laughter “effort” would be even more difficult. Running and jumping are used to provoke urine leakage during some pad tests; however, not all women who report symptoms of SUI demonstrate leakage during the tests and the physical demands of the running and jumping tasks may prevent some women, who would otherwise be appropriate, from participating in PFM research. Postural perturbation, involving catching a set mass, dropped from a standard height in a box, has also been used to study PFM function. This task has the advantage of being highly standardized and of simulating the suddenness of situations that typically provoke SUI but it only elicits relatively low amplitude PFM activation and, like the pad test, it may not be appropriate for investigating the roll of the PFMs in SUI in some populations.
Various tasks have been used to test the endurance of the PFM: a maximum PFM contraction held for 90 seconds or to exhaustion, repeated PFM contractions, repeated coughs and intense whole body exercise.\textsuperscript{179, 196, 203, 204, 314} These tasks have produced mixed results in determining differences between women with and without SUI, likely because, although the tasks are all described as endurance tasks, they actually test different aspects of PFM function and, overall, they do not represent the habitual functional demands placed on the PFM. Although it has been recognized clinically for many years that women with SUI leak more frequently in the evening than in the morning, that leakage is more likely to occur with repeated rather than single coughs, and that women with SUI who run tend not to leak until later in the run;\textsuperscript{83, 204} it is still not clear what type of task is best for elucidating differences in PFM endurance between women with and without SUI. Further research is necessary to systematically describe the point at which leakage actually occurs during activities that provoke SUI in order to be able to determine the underlying functional deficits.

All of the tasks currently employed to evaluate PFM function have limitations and care should be taken when generalizing from them to functional tasks performed in the “real world”. There are also significant limitations in implementing more functional PFM tests. Future research is needed to determine which tests of PFM function best reflect the underlying structural and functional deficits in order to be able to predict who will benefit from which type of treatment and to guide the development of future treatments.
7.5 Future directions

7.5.1 Motor control

Motor control and muscle synergies involving the PFMs appear to be more
involved in SUI than do simple measures of PFM function such as strength or
endurance and future research should attempt to describe these differences. The
first three studies in this thesis (Chapters 3 through 5) have begun this work by
describing the timing of abdominal muscle activation along with how these muscles
are involved in the generation of both urethral and vaginal pressure during
voluntary PFM contractions and coughing. Further descriptive studies are needed to
establish how women with SUI differ from continent women in terms of motor
control in areas such as: PFM, abdominal muscle and hip muscle activation patterns
and timing, muscle activation timing relative to the timing of urethral, vaginal and
intra-abdominal pressure onset and peak, and the effect of multiple task demands
(e.g. respiratory, postural and continence) on these activation patterns. The
intensity of the tasks should be graded to determine if the motor control patterns
are consistent to the task or if they change with the effort required. Longitudinal
studies are necessary to determine if the changes in motor control seen in women
with SUI cause or are adaptations to the incontinence. Intervention studies are also
needed to determine if normal motor control strategies can be relearned and if
restoring the normal motor control patterns results in improved continence.

7.5.2 Structural deficits

Along with studying motor control patterns, imaging techniques, such as MRI
and ultrasound, should be employed to define study groups into those with PFM
tears or avulsion and those with altered pelvic organ motion, indicative of fascial
damage.\textsuperscript{248} Determining the study groups in this way may help to resolve some of
the contradictions in the literature, with respect to PFM strength and endurance, as
the contradictory results among studies may be due to volunteers having different
aetiologies for their SUI. The underlying pathology may also determine the motor
control strategies that women with SUI demonstrate during individual tasks and
this should be investigated. Intervention studies should consider the underlying
structural defects present in their volunteers, as it is likely that the defects will have
an effect on the response to treatment.

7.6 Conclusion

The four studies presented in this thesis further our understanding of the
extrinsic continence mechanism in continent women and how the mechanism is
altered in women with SUI. The results provide support for the theory that the
aetiology of SUI is multifactorial as both delays in pressure transmission and motor
control differences between continent women and women with SUI were found. The
results also suggest that PFM damage is not likely to be a primary cause of SUI. The
findings that the abdominal muscles play a role in generating vaginal pressure
during both voluntary and involuntary PFM contractions provide further evidence
of an abdominal-PFM synergy and Chapter 3 demonstrates that the deep abdominal
muscles play a role in generating urethral pressure during both voluntary PFM
contractions and coughing. Further research using imaging techniques is needed to
link structural to functional deficits and to fully understand the synergies between
the PFMs and the back, abdominal, respiratory and hip muscles in both continent women and women with SUI.
References:
51. Lee D, Lee L-J. Stress urinary incontinence - a consequence of failed load transfer through the pelvis? 5th Interdisciplinary World Congress on Low Back and Pelvic Pain; 2004; Melbourne, Australia.


144. Smith MD, Coppieters MW, Hodges PW. Postural response of the pelvic floor and abdominal muscles in women with and without incontinence. XVI Congress of the International Society of Electrophysiology and Kinesiology; 2006; Turin, Italy.
146. Smith MD, Coppieters MW, Hodges PW. Postural activity of the pelvic floor muscles is delayed during rapid arm movements in women with stress urinary incontinence. Int Urogynecol J Pelvic Floor Dysfunct. 2007;18:901-11.


292. Physiotherapy Act, 1991, SO 1991, c. 37, as amm’d, Ontario, Canada


Appendix A: Consent form - Comparison of anterior vaginal, posterior vaginal and urethral pressures in women.

Background information:

You are invited to participate in a research study directed by Stéphanie Madill and Dr. Linda M’Lean to evaluate how the pressures measured in the vagina and in the urethra are related to each other and how abdominal and pelvic floor muscle activity affect these pressures. Ms. Stéphanie Madill, a physiotherapist and graduate student, will read through this consent form with you and describe the procedures in detail and answer any questions you may have.

Details of the study:

The aim of the study:

The purpose of this study is to determine the best way to measure the strength of pelvic muscle contractions and how these contractions work to prevent urinary incontinence. You will be asked to participate in this study if you are female between the ages of 35 and 70 and are generally healthy. You will be excluded from participating if you have a nerve disorder that might affect your pelvic floor muscles. Your sensation and reflexes will be checked for this reason.

Description of visits, dosage, tests to be performed as part of the study:

If you agree to participate in this study you will be asked to attend one session of about one hour. You will be in a private room for all procedures. First you will be asked a series of questions about your medical history and any medications that you are taking and have a brief examination to test your ability to perform a pelvic floor muscle contraction, to make sure that you do not have evidence of prolapse and to ensure that your reflexes are normal. This information will only be used to determine if you qualify for participation in the study.

Next, the skin over your abdomen will be cleaned with rubbing alcohol and pairs of self-adhesive recording discs will be placed on three areas of skin overlying muscles in your lower abdomen to record muscle activity. An urodynamic catheter that measures pressure in the urethra will be inserted by a nurse specialized in urodynamics. You will also be asked to insert a probe into your vagina. The probe measures the muscle activity and pressures produced by your pelvic floor muscles. Verbal instructions on how to insert the probe will be given to you and you will be left alone to place it appropriately and to change into a pair of loose shorts. You will be asked to leave the connectors accessible outside your clothes. Once you are ready, you will indicate to the investigator that you are ready for her to return.
you feel any discomfort with the probe inserted, please inform the investigators. You may remove the probe and stop the session at any time without any negative consequences.

You will be asked to perform a series of pelvic floor muscle contractions in two different positions: standing up and lying down. The tasks are as follows:

1. You will be asked to assume a comfortable and relaxed position and data will be recorded as you do so.
2. You will be asked to perform three repetitions of the strongest pelvic floor muscle contraction that you can perform.
3. You will be asked to perform three repetitions of a 30-second series of pelvic floor muscle contractions.
4. You will be asked to perform three series of five maximal coughs.
5. You will be asked to hold your breath and bear down (Valsalva manoeuvre) three times.

You will be given a short break and asked to switch positions. The same recording procedure will be repeated.

Risks/Side-Effects:

There are no known negative side effects reported to occur with pelvic muscle exercises, nor with the data recording devices used in this study. Should the probe cause any unusual feelings or symptoms, please report these immediately to the investigator.

Benefits:

While you may not benefit directly from this study, results from this study may improve our understanding of how a commonly used assessment tool, intravaginal pressure, relates to the functional problem of urinary incontinence. It will also increase our knowledge about how the pelvic floor muscles and abdominal muscles interact to maintain continence. This information may benefit women with urinary incontinence by allowing us to design treatment programs that provide maximal benefit to them.

Exclusions:

You will not be considered for this study if you have nervous system problems that could affect your bladder or pelvic floor muscles, if you are pregnant or have delivered in the previous six months, if you have urogenital prolapse, if you have had pelvic floor surgery or if you are taking medications that could affect your bladder or pelvic floor muscles. You will also not be considered if you are allergic to adhesive tape.
Confidentiality:

All information obtained during the course of this study is strictly confidential and your anonymity will be protected at all times. You will be identified by subject number only, not your name. Data will be stored in locked files and will be accessible only to the investigators. Any use of your data for teaching purposes, publication or reports will not reveal your identity.

Voluntary nature of the study/Freedom to withdraw or participate:

Your participation in this study is voluntary. You may withdraw from this study at any time and your withdrawal will not affect your future medical care with any physician, physiotherapist or nurse at any hospital or clinic.

Withdrawal of subject by the principal investigator:

The investigator may decide to withdraw you from this study if you are unable to contract your pelvic floor muscles, if the screening examination suggests any neurological problem that could affect your bladder or pelvic floor, or if you have signs of urogenital prolapse. In this event, you will be informed as to why you are being excluded and you will be advised to follow up with your family physician regarding our findings.

Liability:

In the event that you are injured as a result of taking part in this study medical care will be provided to you until the resolution of the medical problem. By signing this consent form, you do not waive your legal rights nor release the investigator(s) and sponsors from their legal and professional responsibilities.

Payment:

You will receive $50.00 for participating in this study. You will be given a parking pass for the time that you are involved in the study.
Subject statement and signature section:

I have read and understand the consent form for this study. I have had the purposes, procedures and technical language of this study explained to me. I have been given sufficient time to consider the above information and to seek advice if I chose to do so. I have had the opportunity to ask questions which have been answered to my satisfaction. I am voluntarily signing this form. I will receive a copy of this consent form for my information.

If at any time I have further questions, problems or adverse events, I can contact:

Ms. Stéphanie Madill (Principal Investigator) at 531-2600
or
Dr. Linda McLean (Faculty Advisor) at 533-6101
or
Dr. Sandy Olney (Director) at 533-6103

If I have questions regarding my rights as a research subject, I can contact:

Dr. Albert Clark, Chair, Research Ethics Board at 533-6081

By signing this consent form, I am indicating that I agree to participate in this study.

________________________________  __________________________
Signature of Subject                  Date

________________________________  __________________________
Signature of Witness                  Date

STATEMENT OF INVESTIGATOR:

I, or one of my colleagues, have carefully explained to the subject the nature of the above research study. I certify that, to the best of my knowledge, the subject clearly understands the nature of the study and the demands, benefits and risks involved to participants in this study.

________________________________  __________________________
Signature of Principal Investigator or Representative                  Date
Subject statement and signature section:

I have read and understand the consent form for this study. I have had the purposes, procedures and technical language of this study explained to me. I have been given sufficient time to consider the above information and to seek advice if I chose to do so. I have had the opportunity to ask questions which have been answered to my satisfaction. I am voluntarily signing this form. I will receive a copy of this consent form for my information.

If at any time I have further questions, problems or adverse events, I can contact:

- **Ms. Stéphanie Madill (Principal Investigator)** at 531-2600
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- or **Dr. Sandy Olney (Director)** at 533-6103

If I have questions regarding my rights as a research subject, I can contact:

- **Dr. Albert Clark, Chair, Research Ethics Board** at 533-6081

By signing this consent form, I am indicating that I agree to participate in this study.

________________________________  ______________________
Signature of Subject                 Date

________________________________  ______________________
Signature of Witness                 Date

STATEMENT OF INVESTIGATOR:

I, or one of my colleagues, have carefully explained to the subject the nature of the above research study. I certify that, to the best of my knowledge, the subject clearly understands the nature of the study and the demands, benefits and risks involved to participants in this study.

________________________________  ______________________
Signature of Principal Investigator  Date
or Representative
Appendix B: Urogenital Distress Inventory Form

The following symptoms have been described by women who experience accidental urine loss and/or prolapse. Please indicate which symptoms you are now experiencing. Be sure to answer all items.

Do you experience frequent urination?
☑ Yes ☐ No

Do you experience a strong feeling of urgency to empty your bladder?
☑ Yes ☐ No

Do you experience urine leakage related to the feeling of urgency?
☑ Yes ☐ No

Do you experience urine leakage related to physical activity, coughing or sneezing?
☑ Yes ☐ No

Do you experience general urine leakage not related to urgency or activity?
☑ Yes ☐ No

Do you experience small amounts of urine leakage (that is, drops)?
☑ Yes ☐ No

Do you experience large amounts of urine leakage?
☑ Yes ☐ No

Do you experience nighttime urination?
☑ Yes ☐ No

Do you experience bedwetting?
☑ Yes ☐ No

Do you experience difficulty emptying your bladder?
☑ Yes ☐ No

Do you experience a feeling of incomplete bladder emptying?
☑ Yes ☐ No

Do you experience lower abdominal pressure?
☑ Yes ☐ No
Do you experience pain while urinating?
☐ Yes   ☐ No

Do you experience pain in the lower abdominal or genital area?
☐ Yes   ☐ No

Do you experience heaviness or dullness in the pelvic area?
☐ Yes   ☐ No

Do you experience a feeling of bulging or protrusion in the vaginal area?
☐ Yes   ☐ No

Do you experience bulging or protrusion you can see in the vaginal area?
☐ Yes   ☐ No

Do you experience pelvic discomfort when standing or physically exerting yourself?
☐ Yes   ☐ No

Do you have to push on the vaginal walls to have a bowel movement?
☐ Yes   ☐ No

Other symptoms
☐ Yes   ☐ No
If yes, please describe:
________________________________________________________________________________________
________________________________________________________________________________________
________________________________________________________________________________________
Appendix C: Consent Form - Comparing synergistic pelvic floor and abdominal muscle contributions to rises in intravaginal pressure and abdominal and pelvic floor muscle activation patterns in response to coughing in urinary continent and stress incontinent women.

Background information:

You are being invited to participate in a research study directed by Drs. Marie-Andrée Harvey and Linda McLean to evaluate how the abdominal muscles contract with the pelvic floor muscles. Ms. Stéphanie Madill or Ms. Cindy Brown, both physiotherapists and graduate students, will read through this consent form with you and describe the procedures in detail and answer any questions you may have.

Details of the study:

The aim of the study:

The first purpose of this study is to determine the patterns of interaction between the abdominal and pelvic floor muscles during different types of pelvic floor muscle contraction. The second purpose of the study is to compare pelvic floor muscle activity in the women with urine leakage to pelvic floor muscle activity in women without urine leakage problems. You will be asked to participate in this study if you are female between the ages of 35 and 70 and have stress urinary incontinence or have had no problems with leaking urine. You will be excluded from participating if you have a nerve disorder that might affect your pelvic floor muscles. Your sensation and reflexes will be checked for this reason.

Description of visits, dosage, tests to be performed as part of the study:

If you agree to participate in this study you will be asked to attend three sessions of about one hour each. You will be located in a private room for all procedures. On the first visit you will respond to a questionnaire to assess symptoms of incontinence
and you will have a brief examination for prolapse, intrinsic sphincter deficiency and reflexes. A nurse will administer the examination and the questionnaire so that the investigators do not know whether you are in the continent or the incontinent group. You will be given a bladder diary to complete over three days to record any episodes of urine leakage. The diary will be returned to the investigators in a sealed envelope on the second visit. If you have urinary incontinence you will be referred for an urodynamic evaluation.

On the second visit, pairs of self-adhesive recording discs will be placed on three areas of skin overlying muscles in your lower abdomen to record muscle activity. You will also be asked to insert a probe into your vagina. The probe measures the muscle activity and pressures produced by your pelvic floor muscles. Verbal instructions on how to insert the probe will be given to you and you will be left alone to place it appropriately and to change into a pair of boxer shorts. You will be asked to leave the connectors accessible outside your clothes. Once you are ready, you will indicate to the investigator that you are ready for her to return. If you feel any discomfort with the probe inserted, please inform the investigators. You may remove the probe and stop the session at any time without any negative consequences.

You will be asked to perform a series of abdominal exercises and a series of coughs in two different positions: standing up and lying down. The tasks are as follows:

1. You will be asked to assume a comfortable and relaxed position and data will be recorded as you do so.
2. You will be asked to perform three repetitions of maximal abdominal manoeuvres:
   - Curl your body forward
   - Curl your body to the right side
   - Curl your body to the left side
3. You will be asked to perform three repetitions of the strongest pelvic floor muscle contraction that you can perform.
4. You will be asked to cough into a mouthpiece as forcefully as you can. The force of the cough will be shown to you so that you can adjust your strength to a true maximum. Once you have mastered this, data will be recorded as you perform three series of coughs. You will be given a short break and asked to switch positions. The same recording procedure will be repeated.

The third session will involve applying self-adhesive recording discs to the same locations on your abdomen and having you insert the vaginal probe again. You will be asked to perform the following tasks in three different positions: lying on your back, sitting and standing.

1. You will be asked to perform three repetitions of maximal abdominal and pelvic floor muscle contractions:
   • Pelvic floor muscle contraction
   • Curl body forward
   • Curl body to the right
   • Curl body to the left
   • Bearing down
   You will hold each contraction for two seconds and will rest for 60 seconds between contractions.

2. You will be asked to assume a comfortable and relaxed position and data will be recorded as you do so.

3. You will be asked to cough forcefully into a mouthpiece. The force of the cough will be shown to you such that you can adjust its strength. Once you have mastered the coughing force, data will be recorded as you perform it three times.

4. You will be asked to perform three repetitions of the strongest contraction of your pelvic floor muscles that you can perform. You will hold each contraction for five seconds and rest for 60 seconds between contractions.

5. You will be asked to perform three trials of contracting and relaxing your pelvic floor muscles repeatedly for 30 seconds.
After this you will be given a short break and asked to switch positions. The same recording procedure will be repeated in the other two positions. Once the tasks have been completed in the three positions, you will be asked to perform one sustained maximum contraction, holding it for as long as possible up to a maximum of 60 seconds. Immediately upon completion of this contraction you will be asked to repeat the coughing manoeuvre. Data will be collected while you are holding the contraction, and again while you are coughing.

**Risks/Side-Effects:**

There are no known negative side effects reported to occur with pelvic muscle exercises, nor with the data recording devices used in this study. Should the probe cause any unusual feelings or symptoms, please report these immediately to the investigator.

**Benefits:**

While you may not benefit directly from this study, results from this study may improve the understanding of how the muscles of the pelvis are influenced by nearby muscles. This information may benefit women with urinary incontinence by allowing us to design treatment programs that provide maximal benefit to women.

**Exclusions:**

You will not be considered for this study if you have nervous system problems that could affect your bladder or pelvic floor muscles, if you are pregnant or have delivered in the previous six months, if you have urogenital prolapse, if you have had pelvic floor surgery or if you are taking medications that could affect your bladder or pelvic floor muscles. You will also not be considered if you are allergic to adhesive tape.
Confidentiality:

All information obtained during the course of this study is strictly confidential and your anonymity will be protected at all times. Subject number only, not your name, will identify you. Data will be stored in locked files and will be accessible only to the investigators. Only the principal and student investigators will have access to your name. Any use of your data for teaching purposes, publication or reports will not reveal your identity.

Voluntary nature of the study/Freedom to withdraw or participate:

Your participation in this study is voluntary. You may withdraw from this study at any time and your withdrawal will not affect your future medical care with any physician, physiotherapist or nurse at any hospital or clinic.

Withdrawal of subject by the principal investigator:

The investigator may decide to withdraw you from this study if you are unable to contract your pelvic floor, if the screening examination suggests any neurological problem that could affect your bladder or pelvic floor, if you have signs of urogenital prolapse or if the urodynamic evaluation suggests that you have an incontinence problem that is not purely stress incontinence.

Liability:

In the event that you are injured as a result of taking part in this study medical care will be provided to you until the resolution of the medical problem. By signing this consent form, you do not waive your legal rights nor release the investigator(s) and sponsors from their legal and professional responsibilities.

Payment:

You will receive $25.00 payment for each visit that you attend (maximum $75.00) in compensation for participating in this study.
Subject statement and signature section:

I have read and understand the consent form for this study. I have had the purposes, procedures and technical language of this study explained to me. I have been given sufficient time to consider the above information and to seek advice if I chose to do so. I have had the opportunity to ask questions which have been answered to my satisfaction. I am voluntarily signing this form. I will receive a copy of this consent form for my information.

If at any time I have further questions, problems or adverse events, I can contact:

Dr. Marie-Andrée Harvey (Principal Investigator) at 548-6115
or
Ms. Stéphanie Madill (Student Investigator) at 531-2600
or
Dr. Linda McLean (Faculty Advisor) at 533-6101

If I have questions regarding my rights as a research subject, I can contact:

Dr. Albert Clark, Chair, Research Ethics Board at 533-6081

By signing this consent form, I am indicating that I agree to participate in this study.

__________________________________________________________
Signature of Subject _______________________________________
Date

__________________________________________________________
Signature of Witness _________________________________________
Date

Statement of investigator:

I, or one of my colleagues, have carefully explained to the subject the nature of the above research study. I certify that, to the best of my knowledge, the subject clearly understands the nature of the study and the demands, benefits and risks involved to participants in this study.

__________________________________________________________
Signature of Principal Investigator ______________________________
or Representative
Date
Subject statement and signature section:

I have read and understand the consent form for this study. I have had the purposes, procedures and technical language of this study explained to me. I have been given sufficient time to consider the above information and to seek advice if I chose to do so. I have had the opportunity to ask questions which have been answered to my satisfaction. I am voluntarily signing this form. I will receive a copy of this consent form for my information.

If at any time I have further questions, problems or adverse events, I can contact:

Dr. Marie-Andrée Harvey (Principal Investigator) at 548-6115
or
Ms. Stéphanie Madill (Student Investigator) at 531-2600
or
Dr. Linda McLean (Faculty Advisor) at 533-6101

If I have questions regarding my rights as a research subject, I can contact:

Dr. Albert Clark, Chair, Research Ethics Board at 533-6081

By signing this consent form, I am indicating that I agree to participate in this study.

________________________________  ______________________
Signature of Subject                  Date

________________________________  ______________________
Signature of Witness                  Date

Statement of investigator:

I, or one of my colleagues, have carefully explained to the subject the nature of the above research study. I certify that, to the best of my knowledge, the subject clearly understands the nature of the study and the demands, benefits and risks involved to participants in this study.

________________________________  ______________________
Signature of Principal Investigator or Representative Date
Appendix D: Bladder Diary Form

Date____________________

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<th>Time of day</th>
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<th>Activity engaged in when leakage occurred</th>
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Appendix E: The Effect of Peak Flow during Coughing on Peak EMG and Vaginal Pressure Amplitudes

A two-way ANOVA, including group and testing position as factors, was performed to compare the peak flow during coughing among the groups ($\alpha<0.05$). There was a significant group by position interaction in the peak flows ($p=0.003$) produced during coughing: the women with mild SUI produced higher peak flows in standing than the women with moderate to severe SUI. The main effects for group and position were not significant ($p\geq0.06$ for both). See Figure E.1.

Figure E.1 Peak flow during coughing in supine and standing. C: continent group, M: mild SUI group, S: Moderate to severe SUI group. * indicates significant differences between groups ($p<0.05$).