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Heightened Pelvic Floor Muscle Tone and Altered Contractility in Women With Provoked Vestibulodynia

Mélanie Morin, PT, PhD,[1] Yitzchak M. Binik, PhD,[2] Daniel Bourbonnais, OT, PhD,[3] Samir Khalifé, MD,[4] Stéphane Ouellet, MD,[5] and Sophie Bergeron, PhD[6]

ABSTRACT

Background: Pelvic floor muscle (PFM) dysfunctions are reported to be involved in provoked vestibulodynia (PVD). Although heightened PFM tone has been suggested, the relative contribution of active and passive components of tone remains misunderstood. Likewise, alterations in PFM contractility have been scarcely studied.

Aims: To compare PFM tone, including the relative contribution of its active and passive components, and muscular contractility in women with PVD and asymptomatic controls.

Methods: Fifty-six asymptomatic women and 56 women with PVD participated in the study. The PVD diagnosis was confirmed by a gynecologist based on a standardized examination.

Outcomes: PFM function was evaluated using a dynamometric speculum combined with surface electromyography (EMG). PFM general tone was evaluated in static conditions at different vaginal apertures and during repeated dynamic cyclic stretching. The active contribution of tone was characterized using the ratio between EMG in a static position and during stretching and the proportion of women presenting PFM activation during stretching. Contribution of the passive component was evaluated using resting forces, stiffness, and hysteresis in women sustaining a negligible EMG signal during stretching. PFM contractility, such as strength, speed of contraction, coordination, and endurance, also was assessed during voluntary isometric efforts.

Results: Greater PFM resting forces and stiffness were found in women with PVD compared with controls, indicating an increased general tone. An increased active component also was found in women with PVD because they presented a superior EMG ratio, and a larger proportion of them presented PFM activation during stretching. Higher passive properties also were found in women with PVD. Women with PVD also showed decreased strength, speed of contraction, coordination, and endurance compared with controls.

Clinical Implications: Findings provide further evidence of the contribution of PFM alterations in the etiology of PVD. These alterations should be assessed to provide patient-centered targeted treatment options.

Strengths and Limitations: The use of a validated tool investigating PFM alterations constitutes a strength of this study. However, the study design does not allow the determination of the sequence of events in which these muscle alterations occurred—before or after the onset of PVD.

Conclusion: Findings support the involvement of active and passive components of PFM tone and an altered PFM contractility in women with PVD. **Morin M, Binik YM, Bourbonnais D, et al. Heightened Pelvic Floor Muscle Tone and Altered Contractility in Women With Provoked Vestibulodynia. J Sex Med 2017;14:592–600.**

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[1]School of Rehabilitation, Faculty of Medicine and Health Sciences, Université de Sherbrooke; Research Center, Centre hospitalier de l'Université de Sherbrooke, Sherbrooke, QC, Canada;

[2]Department of Psychology, McGill University, Montréal, QC, Canada;

[3]School of Rehabilitation, Faculty of Medicine, Université de Montréal; Institute of Rehabilitation Gingras-Lindsay of Montreal; Centre for Interdisciplinary Research in Rehabilitation of Greater Montreal (CRIR), Montréal, QC, Canada;

[4]Department of Obstetrics and Gynecology, Jewish General Hospital, Montréal, QC, Canada;

[5]Department of Obstetrics and Gynecology, Université de Montréal and CHUM, Montréal, QC, Canada;

[6]Department of Psychology, Université de Montréal, Montréal, QC, Canada

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INTRODUCTION

Vulvodynia is a common gynecologic pain condition with a population prevalence rate as high as 7% to 8% by 40 years of age.[1] The most common type of vulvodynia, provoked vestibulodynia (PVD), is characterized by a burning pain at the entry of the vagina during the application of pressure or while attempting vaginal penetration.[2] Although the etiology of PVD remains poorly understood, pelvic floor muscle (PFM) dysfunctions, most notably heightened PFM tone, have been proposed among key pathophysiologic mechanisms.[3] It has been hypothesized that increased PFM tone could act as an initiator of vestibular pain and/or a perpetuating factor, which results in a vicious cycle involving pain and further muscle tensions.[4–7] However, the comparison of PFM tone in women with PVD with that in asymptomatic controls has generated conflicting results.[7–15] This can stem from insufficient understanding of muscle tone physiology and limitations of current assessment tools. Muscle tone (also referred to as general PFM tone) is defined as the resistance provided by an innervated muscle when stretching is applied. Muscle tone is composed of an active (electrogenic) component and a passive (viscoelastic) component.[16] No studies to date have considered these components of PFM tone in women with PVD. Several studies have used digital palpation, a general but subjective approximation of muscle tone.[17,18] Others have used resting electromyography (EMG) amplitude as an index of the active component (electrogenic), although this measurement has several limitations when used for inter-subject comparison.[19] Because confounding factors such as vaginal lubrication, thickness of the vaginal tissue, and contact between the electrodes and the mucosa can influence EMG amplitude, it is recommended in musculoskeletal research to use ratio or normalized EMG values or muscle activation.[20–22] A methodology combining EMG and dynamometry has been developed to overcome the limitations of current pelvic floor assessment tools and has been validated to investigate general PFM tone and the relative contribution of active and passive components.[23,24] Further, alterations in PFM contractility, such as strength, speed of contraction, coordination, and endurance, have rarely been studied in women with PVD[8–10,14,15] and should be investigated with direct and validated tools such as a dynamometer. Not only is this is important for a better understanding of PVD pathophysiology, but it also has significant clinical implications for treatment. For instance, the active component that encompasses the electrogenic spasms would be specifically addressed by teaching proper muscle relaxation and control with biofeedback, whereas heightened viscoelastic properties (passive component)[21] would require stretching. Thus, the goals of this study were to (i) assess and compare PFM tone, including general muscle tone and the relative contribution of active and passive components, in women diagnosed with PVD and asymptomatic controls and (ii) compare PFM strength, speed of contraction, coordination, and endurance between the two groups.

METHODS

Participants

Participants were recruited through posters in universities and affiliated hospitals, health professional referrals, and newspaper and website advertisements. For the PVD group, 87% were recruited through posters and advertisements, 9% from health professional referrals (gynecologists and psychologists), and 4% from another PVD study in which women expressed an interest in being re-contacted for participating in similar studies. For the control group, 73% were recruited through posters and advertisements and 27% by word of mouth. Eligibility criteria were verified by a telephone screening interview and the physiotherapist's assessment. In addition, women with PVD had their diagnosis confirmed by a gynecologist from our team (S.K. and S.O.) based on a standardized and validated protocol consisting of an interview and a physical examination.[25] As part of the protocol, a cotton-swab test was performed to evaluate the sensitivity of the vestibule by applying random pressure at 3, 6, and 9 o'clock. The women had to report a pain intensity of at least 5 on the numerical rating scale (from 0 to 10) and to express similar pain to the one perceived during vaginal intercourse. The gynecologic assessment was conducted on a different occasion than the PFM assessment. Other inclusion criteria for women with PVD were (i) pain during intercourse that was subjectively distressing and occurred during 80% of intercourse attempts in the past 6 months and (ii) pain limited to intercourse and other activities involving vestibular pressure (eg, bicycling). Asymptomatic women had to be sexually active (ie, vaginal penetration in the past 6 months) and report no history of vulvovaginal pain and no difficulties with sexual activity, gynecologic examinations, or tampon insertion. The exclusion criteria for the two groups were (i) deep dyspareunia; (ii) postmenopausal status; (iii) current or previous pregnancy that lasted longer than 18 weeks; (iv) urogynecologic symptoms such as urinary or anal incontinence or urinary urgency; (v) pelvic organ prolapse (stage $>$ 1 at the pelvic organ prolapse quantification)[26] and active urinary or vaginal infection (or in the past 3 months); (vi) previous vulvovaginal surgery or pelvic floor physical therapy treatment; (vii) ongoing treatment for dyspareunia; and (viii) age younger than 18 or older than 45 years. The present study was approved by the institutional review boards of the Centre hospitalier de l'Université de Montréal and McGill University and all women gave written informed consent.

Instrumentation

The PFM function was evaluated with a dynamometric speculum according to a previously described and validated methodology (Figure 1).[27,28] The psychometric properties related to the assessment of PFM tone during stasis and repeated cyclic stretching show strong reliability and validity and have been presented elsewhere.[23,24] To allow the assessment of women with vulvovaginal pain, the speculum's upper and lower

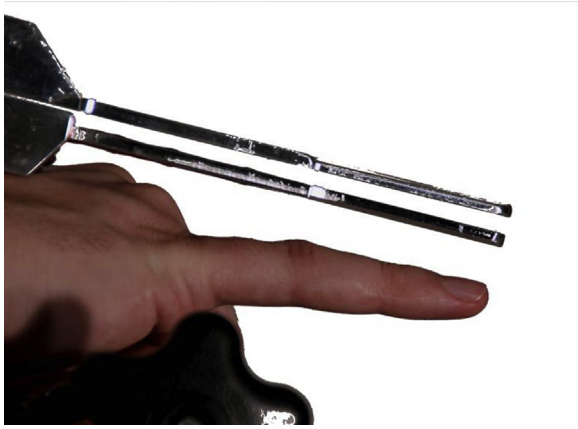

Figure 1. Dynamometric speculum. Figure 1 is available in color online at www.jsm.jsexmed.org.

branches were reduced to the size of a pediatric speculum (width = 11 mm, anteroposterior diameter when the two branches are closed = 10 mm). This reduced speculum was used for all participants in the present study. The lower branch, equipped with strain gauges, can be moved downward to gently increase the vaginal aperture. The linear position transducer (MLT-38000-101; Honeywell, Morris Plains, NJ, USA) enables real-time monitoring of the vaginal anteroposterior aperture. EMG recordings were used to monitor muscle activity (active component) during PFM tone measurements (Bagnoli-8 EMG system; Delsys, Natick, MA, USA). After disinfection of the speculum branches and covering with the finger part of a surgical sterile glove, four pairs of Medtronic disposable electrodes (9013L4611; Medtronic, Minneapolis, MN, USA) were stuck on the lower branch with a 4-mm distance between each pair and therefore covered the entire length of the lower branch inserted in the vaginal cavity. The pair of electrodes recording the highest EMG amplitude was considered for analysis.[24] A bipolar configuration was used to maximize the selectivity of the EMG recordings. The ground electrode (Kendall Medi-Trace; Medtronic) was positioned over the great trochanter area. The EMG signals were amplified (gain = 1,000), bandpass-filtered using a fourth-order zero-lag Butterworth filter of 10 to 400 Hz, and notch-filtered at 59 to 61 Hz (Butterworth fourth-order zero-lag) to remove ambient noise and then the root mean square was computed using 500-ms windows (±250 ms around each point). The EMG signals, dynamometric speculum, and position transducer driven by DC amplifier signals (Analog Devices, Norwood, MA, USA) were simultaneously recorded using a laptop computer equipped with an acquisition card (NIDAQCard-6024E; National Instrument Corporation, San Diego, CA, USA).

Procedures and Outcome Measures

The participants convened at a PFM assessment session conducted by an experienced pelvic floor physiotherapist (M.M.) in an adapted examination room at McGill University. Before the examination, the women were asked to empty their bladder to ensure their comfort during the assessment and avoid any potential influence of bladder fullness on PFM activity.[29] The women adopted a supine lying position with hips and knees flexed and their ability to properly contract the PFMs was verified by digital assessment. The lubricated branches of the speculum were inserted into the vaginal cavity to a depth of 5 cm along the natural orientation of the vagina. Participants were asked to perform two unrecorded contractions to ensure comfort and proper device positioning. The evaluator had access to force and raw EMG curves displayed on the computer screen in real time to instruct the participants to properly relax or contract the PFMs, depending on the condition assessed. A 2-minute period between each condition was used to allow tissue viscoelasticity restoration or prevent muscle fatigue.[23,27] The PFM function was assessed in six conditions, the first three evaluating PFM tone and the others evaluating PFM contractility. (i) For initial passive resistance, women were asked to relax their PFMs as much as they could with the force output displayed as feedback. Mean resting forces and root mean square of EMG activity were recorded over 5 seconds with the speculum branches closed (ie, in stasis at a 10-mm vaginal aperture). The mean of two trials was considered. (ii) For passive resistance at the maximal aperture, the aperture was slowly increased to the maximal aperture, which was determined according to the participant's tolerance.[24] Mean resting forces were evaluated in stasis at the maximal aperture and the average of two trials was calculated. (iii) For repeated lengthening and shortening cycles, the two speculum branches were separated at a constant speed of 5 mm/s to stretch the PFM and surrounding tissues until the maximal vaginal aperture (as previously determined; lengthening phase). Then, the branches were closed back to the minimal aperture at the same constant speed according to a template displayed on the screen (shortening phase). As preconditioning, five stretch-relax cycles were performed at 80% of the maximal aperture. Thereafter, five stretch-relax cycles were carried out at 100% of the maximal aperture. Resting forces, EMG, and apertures were monitored and recorded in real time. To minimize the thixotropic effects, the following parameters were extracted from the curves and averaged for cycles 3 through 5: (a) resting forces (N) at minimal aperture, maximal aperture, and a common aperture of 15 mm; (b) passive elastic stiffness (PES; change in forces/change in vaginal aperture; N/mm) at minimal aperture, maximal aperture, and a common aperture of 15 mm; (c) aperture at a common resting force of 2 N; and (d) hysteresis (N · mm), which is the area between the lengthening and shortening curve. General muscle tone, which encompasses the active and passive components, was evaluated from these parameters regardless of the EMG evaluation. The relative contribution of the active component was investigated by comparing the ratio between the EMG amplitude in stasis (condition i) and during repeated stretching (condition iii) and the percentage of participants presenting muscle activation during stretching, defined as EMG

signals 2 SD larger than the mean EMG value recorded in condition 1.[30] The contribution of the passive component was evaluated using the aforementioned parameters in women presenting negligible EMG signals at rest (EMG < 2 SD).[21,23,24] (iv) For maximal strength, women were instructed to contract their PFMs maximally for 10 seconds. The peak force value minus the baseline value (rest) was considered. (v) For speed of contraction and coordination, women were asked to contract maximally and relax as fast as possible for 15 seconds. The parameters evaluated were the mean slope of the ascending curve of the first contraction, the speed of relaxation estimated by the mean slope of the descending curve, and the number of contractions performed. (vi) For endurance measurements, participants were asked to maintain a maximal contraction for 90 seconds to capture the decrease of forces over time.[27] The normalized area under the force curve ([area/maximal force] × 100) was computed.

Statistical Analyses

Data analyses were performed using PASW Statistics 18.0 (SPSS Inc, Chicago, IL, USA). The deviation from the normal distribution was verified graphically. The baseline characteristics of women with PVD and asymptomatic healthy controls were compared using χ^2 test and Student t-test. The data analyst in charge of extracting the dynamometric and EMG parameters from the raw data was blinded to participant group allocation. Multivariate analyses of variance (MANOVAs) were used to compare PFM function parameters between the two groups to take account for their potential interrelations. Subsequent univariate ANOVAs were used to ascertain which individual parameters differed significantly between the two groups. The proportion of participants with EMG silent muscles between the two groups was compared using a χ^2 test. All P values presented were two-tailed, with a P value no higher than .05 considered statistically significant. To better appreciate the significance of the data, effect sizes were evaluated with Cohen d (where 0.20 indicates a small effect, 0.50 indicates a moderate effect, and ≥0.80 indicates a large effect).[31]

RESULTS

Sample Characteristics

Of the 145 women who met the eligibility criteria, 12 women with PVD and 21 controls refused to participate, resulting in a sample size of 56 women with PVD and 56 asymptomatic controls. As presented in Table 1, no statistical differences were found for the baseline characteristics between women with and those without PVD. Women with PVD had a mean pain duration of 5.5 years (range = 0.5−27.0, SD = 5.2) and a mean pain intensity of 6.5 of 10 using a numerical rating scale (range = 2−10, SD = 1.8). Of these women, 30 (54%) had primary PVD and 26 (46%) had secondary PVD.

Table 1. Baseline characteristics of women with PVD (n = 56) and asymptomatic controls (n = 56)*

	PVD	Control	*P* value
Age (y), mean ± SD	26.2 ± 5.9	25.4 ± 5.7	.489
Education level (y), mean ± SD	16.0 ± 2.9	16.7 ± 2.3	.146
Age at first vaginal intercourse (y), mean ± SD	17.7 ± 2.6	17.3 ± 3.9	.449
Frequency of vaginal intercourse in past month, mean ± SD	6.2 ± 6.8	5.2 ± 6.2	.409
Place of birth, n (%)			.629
North America	36 (69)	41 (73)	
Europe	7 (13)	6 (11)	
Latin or South America	8 (14)	4 (7)	
Other	5 (9)	5 (9)	
Religion, n (%)			.181
Catholic	39 (70)	32 (57)	
Other	8 (14)	16 (29)	
None	9 (16)	8 (14)	
Place of birth, n (%)			.629
North America	36 (64)	41 (73)	
Europe	7 (13)	6 (11)	
Latin or South America	8 (14)	4 (7)	
Other	5 (9)	5 (9)	
Annual income, n (%)			.759
$0−$19,999	31 (55)	35 (63)	
$20,000 −$39,999	8 (14)	7 (13	
$40,000 −$59,999	8 (14)	9 (16)	
>$60,000	7 (13)	4 (7)	
Hormonal contraceptive use, n (%)	50 (91)	45 (80)	.188

PVD = provoked vestibulodynia.
*Continuous variables are expressed as mean ± SD and categorical variables are expressed as frequency (percentage).

PFM Function

Before the dynamometric evaluation, digital palpation examination confirmed that all participants could perform a correct PFM contraction. Moreover, all women completed the dynamometric assessment. Results from MANOVA, with the participant group as the independent variable and the general PFM tone parameters as the dependent variables, showed significant differences between women with PVD and asymptomatic controls on at least one of these parameters ($F_{12,96} = 9{,}770$, $P < .0005$; Wilks $\Lambda = 0.450$, partial $\eta^2 = 0.550$). Follow-up univariate analyses showed significant differences between women with and without PVD (Table 2). These data on PFM tone are presented regardless of muscle activation; hence, they

Table 2. General pelvic floor muscle tone in women with PVD and asymptomatic controls

Conditions	Parameters	PVD (n = 56), mean ± SD	Control (n = 56), mean ± SD	*P* value	Cohen d
Initial passive resistance	force (N)	1.36 ± 0.75	0.94 ± 0.55	.001	0.647
Passive resistance at maximal aperture	force (N)	3.75 ± 1.48	4.61 ± 1.72	.007	0.543
	aperture (mm)	21.31 ± 3.74	28.70 ± 4.59	<.001	1.787
Dynamic stretching (lengthening and shortening cycles)	force at minimal aperture (N)	0.85 ± 0.75	0.52 ± 0.40	.005	0.561
	PES at minimal aperture (N/mm)	0.77 ± 0.32	0.55 ± 0.16	<.001	0.851
	maximal aperture (mm)	18.96 ± 3.46	24.94 ± 4.17	<.001	1.575
	force at maximal aperture (N)	4.11 ± 1.68	4.61 ± 1.47	.099	0.320
	PES at maximal aperture (N/mm)	0.36 ± 0.13	0.31 ± 0.11	.020	0.453
	aperture to have a passive force of 2 N (mm)	14.01 ± 2.05	16.53 ± 2.89	<.001	1.014
	force at aperture of 15 mm (N)	2.69 ± 1.13	1.83 ± 0.80	<.001	0.884
	PES at aperture of 15 mm (N/mm)	0.34 ± 0.13	0.27 ± 0.10	.005	0.561
	hysteresis (N · mm)	9.27 ± 8.25	16.15 ± 8.02	<.001	0.866

PES = passive elastic stiffness; PVD = provoked vestibulodynia.

represent the general PFM tone consisting of active and passive components. Women with PVD showed greater resting forces during the initial passive resistance condition compared with asymptomatic controls ($P = .001$). Moreover, because of their pain, women with PVD did not tolerate as much aperture as the asymptomatic women ($P \leq .001$) and their resistance at maximal aperture was lower ($P = .007$) as evaluated at smaller apertures. During dynamic stretching, women with PVD showed increased forces and PES at minimal and 15-mm aperture with a Cohen d from 0.561 to 0.884, indicating a moderate to large effect size ($P \leq .007$). Also suggestive of an increase in general muscle tone in women with PVD, the aperture required to obtain 2 N of passive force was smaller and the hysteresis was higher in this group (large effect size, d ≥ 1.015, $P < .001$). As with resistance at the maximal aperture, women with PVD did not tolerate as much aperture during the dynamic stretching, resulting in a non-significant difference in force between the two groups.

PFM Tone—Contribution of Active Component

The relative contribution of the active component in PFM tone can be investigated by evaluating the ratio between the EMG amplitude in stasis during the initial passive resistance condition and during the repeated stretching. Women with PVD showed significantly greater muscle activation during repeated stretching than asymptomatic controls (ratio PVD 1.28 ± 0.49; controls 1.02 ± 0.25; $P = .001$). Similarly, the percentage of women presenting muscle activation was significantly larger in the PVD group compared with the control group (PVD 29 of 55, 53%; controls 8 of 55, 15%; $P = .001$). It should be mentioned that the EMG signal could not be interpreted because of artifacts in one woman with PVD and in one control.

PFM Tone—Contribution of Passive Component

To appreciate the relative contribution of the passive component to PFM tone, the passive properties of women showing a quiescent EMG signal during dynamic stretching (ie, presenting no muscle activation, EMG activity < 2 SD) are presented in Table 3. Controlling for EMG activation, a MANOVA ($F_{9,62} = 11.890$, $P < .0005$; Wilks $\Lambda = 0.367$, partial $\eta^2 = 0.633$) and follow-up univariate analyses showed significant differences between the two groups, supporting the contribution of the passive component to PFM tone in women with PVD.

PFM Contractility

For PFM contractility, there was a statistically significant difference between women with and without PVD ($F_{5,106} = 8.621$, $P < .0005$; Wilks $\Lambda = 0.711$, partial $\eta^2 = 0.289$). As seen in the results of follow-up univariate analyses presented in Table 4, women with PVD showed less PFM strength and endurance than asymptomatic controls ($P \leq .028$). They also presented a decreased speed of contraction as evaluated by the slope of the curve at the beginning of the contraction and an altered coordination as reflected by the number of contractions performed (with large effect size, d = 1.134, $P < .001$).

DISCUSSION

The aim of the present study was to investigate and compare PFM tone and contractility in women with and without PVD using an innovative methodology combining simultaneous dynamometric and EMG measurements. Confirming our main hypothesis, women with PVD showed heightened general PFM

Table 3. Evaluation of pelvic floor muscle tone during repeated stretching in women with electromyographically silent pelvic floor muscles

Parameters	PVD (n = 26), mean ± SD	Control (n = 46), mean ± SD	*P* value	Cohen d
Force at minimal aperture (N)	0.93 ± 0.84	0.51 ± 0.37	.021	0.733
PES at minimal aperture (N/mm)	0.82 ± 0.32	0.53 ± 0.15	<.001	1.270
Maximal aperture (mm)	18.57 ± 2.79	25.25 ± 4.10	<.001	1.839
Force at maximal aperture (N)	4.24 ± 1.82	4.58 ± 1.33	.363	0.228
PES at maximal aperture (N/mm)	0.36 ± 0.14	0.31 ± 0.11	.078	0.445
Aperture to have passive force of 2 N (mm)	13.58 ± 1.88	16.79 ± 2.92	<.001	1.255
Force at aperture of 15 mm (N)	2.88 ± 1.19	1.77 ± 0.81	<.001	1.171
PES at aperture of 15 mm (N/mm)	0.36 ± 0.14	0.27 ± 0.10	.007	0.761
Hysteresis (N · mm)	8.91 ± 8.18	16.12 ± 6.61	<.001	1.015

PES = passive elastic stiffness; PVD = provoked vestibulodynia.

tone. When evaluating more specifically the contribution of the passive and active components of PFM tone, results showed significant involvement of the two components. Moreover, findings showed altered PFM contractility such as a decreased strength, speed of contraction, coordination, and endurance in women with PVD.

Analysis of the PFM dynamometry at rest showed that women with PVD present greater resting forces and stiffness at different apertures, greater hysteresis, and smaller aperture to obtain a resting force of 2 N compared with asymptomatic controls. These measurements taken regardless of EMG activation are reflexive of heightened general PFM tone (ie, summative contribution of active and passive components) in women with PVD. This finding concurs with those of other studies using palpation measurements[8–10] and with those using objective evaluations such as manometry[11] and ultrasonography.[7] Although manometry and ultrasonography are indirect measurements of PFM tone by evaluating intravaginal pressure and geometric measurements, dynamometry provides a direct and comprehensive evaluation of PFM tone using forces and stiffness parameters. Furthermore, less flexibility or tolerance to stretching in women with PVD was observed when evaluating the maximal aperture. This also was reported by Gentilcore-Saulnier et al[9] in a smaller sample, as measured subjectively by separating two fingers to approximate latero-lateral hiatus diameter. Therefore, our results provide strong evidence using an objective assessment that women with PVD present increased general muscle tone and decreased flexibility. This re-enforces the role of PFM tone in PVD, as highlighted in current terminology and guidelines[2,3] that, thus far, had relied on subjective or indirect assessment.

Regarding the contribution of the active component of PFM tone, our findings showed that women with PVD had greater muscle activation during stretching than controls. These findings are in line with those of a number of studies,[12–14] although others reported a non-significant difference in resting EMG amplitude in women with vs without PVD.[9–11,15] This divergence of results can be attributed to methodologic limitations related to EMG amplitude analysis used in these studies, which were mainly overcome in the present study by using a ratio or proportion of muscle activation.[21] It should be noted that some studies of skeletal muscle proposed to normalize resting EMG amplitude using signals recorded during maximal voluntary contractions.[32] This was not suitable in the present study because of strength differences between the two groups. The active component captured with the EMG measurement can be attributed to normal electrogenic contraction and electrogenic spasms.[16] Normal electrogenic contraction implies resting activity in normally relaxed muscle and myotatic reflex during stretching,[33] which appears unlikely considering the stretch velocity set at minimal speed. An electrogenic spasm is probably of greater clinical relevance because it includes unintentional muscle contraction.[12] In addition to pain, an electrogenic spasm can be related to psychological distress (eg, anxiety), muscle overload or overuse (eg, related to an inadequate posture), and

Table 4. Pelvic floor muscle contractility in women with PVD and in asymptomatic controls

Conditions	Parameters	PVD (n = 56), mean ± SD	Control (n = 56), mean ± SD	*P* value	Cohen d
Maximal strength	maximal force (N)	2.35 ± 1.25	2.98 ± 1.55	.021	0.456
Speed of contraction	Contractions (n)	7.18 ± 1.65	9.64 ± 2.62	<.001	1.134
	slope of ascending curve (N/s)	3.58 ± 2.43	5.35 ± 3.84	.005	0.556
	slope of descending curve (N/s)	−2.82 ± 2.14	−3.22 ± 2.66	.346	0.165
Endurance	normalized area under force curve (% · s)	1,816.27 ± 761.68	2,168.34 ± 830.93	.028	0.446

PES = passive elastic stiffness; PVD = provoked vestibulodynia.

inefficient use (eg, failure to fully relax after contraction).[16] Although it is not possible to identify the exact cause of muscle activation, our results support the contribution of electrogenic spasms in women with PVD. This could serve as the basis for orienting treatment toward enhancing muscle control and relaxation (eg, with biofeedback),[34,35] decreasing pain behaviors such as protective muscular reactions,[9] and correcting other potential contributors to electrogenic spasm.

To our knowledge, no studies to date have investigated the passive component of the PFM tone in women with PVD. Findings show that women with PVD sustaining quiescent muscle activity during stretching still presented greater passive forces, stiffness, and hysteresis than asymptomatic controls. Therefore, the results demonstrate that women with PVD not only showed muscle over-activation but also presented persistent alterations in muscle viscoelastic properties that encompass several structures[21]: (i) the extensibility of actin-myosin cross-bridges (EMG silent); (ii) non-contractile cytoskeleton proteins; and (iii) conjunctive tissues surrounding the entire muscle (epimysium), muscle fascicle (perimysium), and muscle fiber (endomysium). These alterations can contribute to the onset of pain or be a consequence of a chronically increased active component (eg, due to immobilization in a shortened muscle position). Most importantly, these alterations directly influence treatment because specific modalities should be selected to target the passive component of tone. For instance, control and relaxation techniques might not be sufficient in these women and stretching, dilator, and manual techniques should be added to restore viscoelastic alterations. It also should be emphasized that adequate muscle relaxation is a prerequisite to act on the passive component.[36] Our methodology using repeated dynamic stretching and real-time monitoring of forces and vaginal apertures offers key advantages. Passive properties can be evaluated at several apertures, which is relevant considering the force-muscle length relation.[37] The measurements taken during stretching also allowed us to capture time-dependent viscoelastic tissue behaviors.[37] Moreover, it considered the thixotropic effect in women with PVD. Recognized as an important phenomenon that interferes with the reliability of measurements,[38] thixotropy refers to the loss of resistance to movement occurring after the initial movement of a muscle or the stiffening that occurs in a muscle with periods of inactivity.[16,38] This is the first study on PVD that controls for the thixotropic effect while analyzing the last three cycles of fives stretches.[24] Overall, our results enhance the current conceptualization of PVD[2,3] by showing a novel mechanism by which PFMs could contribute to its etiology.

Regarding PFM contractility, findings show that women with PVD present contractile alterations such as a significant decrease of strength, endurance, and speed of contraction compared with asymptomatic women. These results are in line with those of some studies showing decreased strength,[8,10,14] speed of contraction,[14] and endurance[10,15] as evaluated with palpation and EMG. Conflicting results also have been reported[9,10,15] which could be attributed to methodologic limitations related to the reliability and validity of a measurement. An important consideration for EMG amplitudes recorded during maximal voluntary contraction is that they can be indicative of, but are not equivalent to, the force output of muscles. In sum, results suggest that the assessment of the PFM should go beyond PFM tone to evaluate the contractile properties.

A possible limitation of this study is that no specific measurements were collected for myofascial trigger points. Although Simons and Mense[16] included this endogenous contracture in the active component, Masi and Hannon[39] considered them a different component because they are EMG silent. In the present study, the contribution of trigger points was globally evaluated in the general tone measurement. Another limitation is the potential presence of pain or discomfort that could be related to any of the current intravaginal assessment methods in women with PVD (eg, dynamometry, palpation, manometry, intravaginal EMG). Therefore, the potential influence of pain during measurement cannot be ruled out. However, it should be emphasized that the results of a morphometric study using ultrasound, a pain-free method, also were suggestive of these contractile alterations.[7] Furthermore, the cross-sectional design of the study does not allow one to discriminate whether PFM dysfunctions are involved in the onset of pain or contribute to perpetuate pain in women with PVD. A longitudinal trial would be required to investigate the sequence of events.

CONCLUSIONS

This study provides sound evidence that active and passive components of PFM tone are involved in the pathophysiology of PVD. In addition to heightened PFM tone, women with PVD present contractile alterations. Regarding the clinical implications, these findings highlighted the importance of a comprehensive assessment of the PFM alterations to guide intervention and provide adapted treatment modalities. Future studies should focus on assessing physical therapy treatment as it specifically targets PFM dysfunctions.

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Corresponding Author: Mélanie Morin, PT, PhD, School of Rehabilitation, Faculty of Medicine and Health Sciences, University of Sherbrooke, 3001 12th Avenue North, Sherbrooke, QC J1H 5N4, Canada. Tel: 819-346-1110, ext 13818; Fax: 819-820-6864; E-mail: Melanie.M.Morin@usherbrooke.ca

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STATEMENT OF AUTHORSHIP

Category 1

(a) Conception and Design
Mélanie Morin; Yitzchak M. Binik; Daniel Bourbonnais; Samir Khalifé; Stéphane Ouellet; Sophie Bergeron

(b) Acquisition of Data
Mélanie Morin

(c) Analysis and Interpretation of data
Mélanie Morin, Daniel Bourbonnais, Sophie Bergeron

Category 2

(a) Drafting the Article
Mélanie Morin

(b) Revising It Critically for Intellectual Content
Mélanie Morin; Yitzchak M. Binik; Daniel Bourbonnais; Samir Khalifé; Stéphane Ouellet; Sophie Bergeron

Category 3

(a) Final Approval of the Completed Article
Mélanie Morin; Yitzchak M. Binik; Daniel Bourbonnais; Samir Khalifé; Stéphane Ouellet; Sophie Bergeron

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