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Original Research

Pelvic floor muscle activation amplitude at rest, during voluntary contraction, and during Valsalva maneuver—a comparison between those with and without provoked vestibulodynia

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Abstract

Background: The neuromuscular contribution to increased tone of the pelvic floor muscles (PFMs) observed among those with provoked vestibulodynia (PVD) is unclear.
Aim: To determine if PFM activity differs between those with provoked PVD and pain free controls, and if the extent of PFM activation at rest or during activities is associated with pain sensitivity at the vulvar vestibule, psychological, and/or psychosexual outcomes.
Methods: This observational case–control study included forty-two volunteers with PVD and 43 controls with no history of vulvar pain. Participants completed a series of questionnaires to evaluate pain, pain catastrophizing, depression, anxiety and stress, and sexual function, then underwent a single laboratory-based assessment to determine their pressure pain threshold at the vulvar vestibule and electromyographic (EMG) signal amplitudes recorded from three PFMs (pubovisceralis, bulbocavernosus, and external anal sphincter).
Outcomes: EMG signal amplitude recorded at rest, during maximum voluntary contraction (MVC), and during maximal effort Valsalva maneuver, pressure pain threshold at the vulvar vestibule, and patient-reported psychological (stress, anxiety, pain catastrophizing, central sensitization) and psychosexual (sexual function) outcomes.
Results: Participants with PVD had higher activation compared to controls in all PFMs studied when at rest and during Valsalva maneuver. There were no group differences in EMG amplitude recorded from the pubovisceralis during MVC (Cohen's d = 0.11), but greater activation was recorded from the bulbocavernosus (d = 0.67) and the external anal sphincter(d = 0.54) among those with PVD. When EMG amplitudes at rest and on Valsalva were normalized to activation during MVC, group differences were no longer evident, except at the pubovisceralis, where tonic EMG amplitude was higher among those with PVD (d = 0.42). While those with PVD had lower vulvar pressure pain thresholds than controls, there were no associations between PFM EMG amplitude and vulvar pain sensitivity nor psychological or psychosexual problems.
Clinical implications: Women with PVD demonstrate evidence of PFM overactivity, yet the extent of EMG activation is not associated with vulvar pressure pain sensitivity nor psychological/psychosexual outcomes. Interventions aimed at reducing excitatory neural drive to these muscles may be important for successful intervention.
Strengths and limitations: This study includes a robust analysis of PFM EMG. The analysis of multiple outcomes may have increased the risk statistical error, however the results of hypothesis testing were consistent across the three PFMs studied. The findings are generalizable to those with PVD without vaginismus,
Conclusions: Those with PVD demonstrate higher PFM activity in the bulbocavernosus, pubovisceralis, and external anal sphincter muscles at rest, during voluntary contraction (bulbocavernosus and external anal sphincter) and during Valsalva maneuver; yet greater activation amplitude during these tasks is not associated with greater vulvar pressure pain sensitivity nor psychological or psychosexual function.

Keywords: provoked vestibulodynia; vulvodynia; pelvic pain; electromyography; muscle tone; overactivity; pelvic floor.

Introduction

Provoked vestibulodynia (PVD) is a primary cause of vulvovaginal pain during insertional sexual activities (dyspareunia) and affects approximately one in 20 women [1]. An evidence synthesis of PVD pathophysiology[2] indicates that local neuroproliferation[3,4] and inflammatory mediators[5,6] are closely associated with vulvar pain sensitivity.[7] Changes in central pain processing[8,9] and impaired descending pain modulation[10] also appear to be implicated in symptom persistence. While high pelvic floor muscle (PFM) tone and/or overactivity[11,12] is identified as another feature of PVD, empirical evidence for PFM involvement in PVD pathophysiology is limited. Further, it is not clear whether there is an association between pain sensitivity, regardless of whether it is nociceptive or nociplastic in nature, and PFM tone or overactivity observed among those who experience PVD.

"Overactivity" implies excessive muscle activity at rest or during tasks,[12] or impaired ability to relax a muscle after

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a task has ended.[12,13] While it is poorly understood, overactivity is neuromuscular in nature and thus can only be measured directly using electromyography (EMG). Muscle tone, in contrast, assessed as resistance to passive elongation, can be influenced by both neuromuscular (active) and connective tissue (passive) elements.[13] Ultrasound imaging and dynamometry findings suggest there are shorter and stiffer connective tissues in the PFMs among individuals with PVD compared to pain-free controls,[14-17] which has been attributed to higher muscle tone. It appears that both active and passive elements influence muscle tone among those with PVD.[17] There may indeed be overactivity in the PFMs that goes beyond a neuromuscular influence on passive elongation.

There is a clear need for better methods to understand impaired PFM activation among those with PVD. While studies have reported higher resting (tonic) PFM EMG activity among those with vulvar pain,[18-23] findings regarding PFM EMG during contraction tasks have varied, with some reporting lower activity[18,19] and others reporting higher activity.[21-23] There may also be differences in activation among the different muscular components of the PFMs. Gentilcore-Saulnier et al. found that tonic activation of the bulbocavernosus muscle was significantly higher among those with PVD compared to controls, while tonic activation of the pubovisceralis muscle did not reach statistical significance.[20] Using needle electrodes, Frasson[21] reported greater tonic EMG activity in the external anal sphincter, and paradoxical activation of the external anal sphincter during straining among those with compared to those without PVD, but did not evaluate the deep layer of the PFMs. EMG activity during straining has not been evaluated in the other PFM components. While, on balance, it appears that the PFMs among those with PVD may have higher tonic EMG activation, it is not clear if there are differences in EMG activation during other tasks, if observed patterns are specific to certain muscles within the pelvic floor group, if findings may have been influenced by the use of large intravaginal probes that caused pain during contraction, and/or if findings have been influenced by signal processing methods, particularly EMG signal normalization. These gaps in our understanding of PFM involvement in PVD were identified in a recent systematic review.[24]

Another recent systematic review[25] identified that the relationship between pelvic pain and PFM tone remains unknown. Also, while it seems clear that those with PVD experience changes in pain physiology[9,10] and emotional/psychosexual dysfunction,[26,27] the interaction of these factors with PFM activity remains unclear.

The aim of this study was to determine if PFM activity differs between those with provoked PVD and pain free controls, and if the extent of PFM activation at rest or during activities is associated with pain sensitivity at the vulvar vestibule, psychological and/or psychosexual outcomes.

The primary objective was to compare PFM activation at rest, during maximum voluntary contraction, and during maximal effort Valsalva maneuver (straining) between those with and without PVD. We hypothesized that those with PVD would demonstrate PFM overactivity manifested as higher PFM activation during all tasks. The secondary objective was to determine whether EMG activation of the PFMs among those with PVD is associated with pain sensitivity (vulvar pressure pain threshold), psychological or psychosexual outcomes. We hypothesized that higher PFM activation amplitudes would be associated with greater vulvar pain sensitivity,[28] more psychological indicators of centralized pain,[9,10] more stress, anxiety and depressive symptoms, and lower sexual function.[26-28]

Design

This was an observational case–control study.

Participants

Participant recruitment occurred between March 2020 and June 2022. Pre-menopausal females were recruited from the local community using posters and social media posts, from local pelvic health physiotherapy clinics and from local physician-led vulvovaginal pain clinics. Separate posters were used to recruit cases (ie, PVD) based on a history of recurrent vulvar pain experienced on touch/pressure of the vulvar vestibule during sexual or non-sexual activities, for example, tampon insertion, gynaecologic exam, and wearing tight clothing, which had lasted more than 6 months, and controls with no history of vulvar pain or painful vaginal insertion activities (sexual or non-sexual). Eligibility was established through telephone screening based on the criteria outlined in Table 1. Eligible participants attended a gynaecological examination which included a cotton swab test to rule in/out PVD (updated Friedrich's criteria by Bergeron et al.[29]), visual inspection of the genitals to rule out other causes of vulvar pain, recording vulvar pain intensity on provocation with the cotton swab (numeric rating scale: 0 to 10) and digital palpation including inserting a single gloved and lubricated digit into the vagina, during which the presence/absence of a palpable PFM contraction was noted. The examination ended with instruction on how to correctly contract the PFMs, which was confirmed through visual inspection (inward motion of the perineum) and digital palpation of closure around the introitus and lifting of the perineum. Without observable extra-pelvic muscle contraction (ie, gluteal muscles).[30,31] All eligible participants were identified as being able to tolerate a finger inserted into the vagina and to perform a correct PFM contraction.

Protocol

Participants completed five on-line questionnaires: The Female Sexual Function Index[32]- Revised (FSFI),[33] the McGill Pain Questionnaire (MPQ),[34] the Pain Catastrophizing Scale (PCS),[35] the Central Sensitization Inventory (CSI)[36], and the Depression, Anxiety, Stress Scales (DASS).[37,38] A laboratory assessment was scheduled within the projected luteal phase of each participant's menstrual cycle (within one week of the anticipated onset of their menstrual period), where relevant, to account for potential cyclic variations in motor and/or sensory thresholds.[39]

The laboratory-based assessment was performed by an academic pelvic health physiotherapist (>10 years of clinical experience assessing and treating female pelvic health conditions including vulvar pain) who remained blinded to group (ie, PVD, Control). This physiotherapist received >40 hours of one-on-one training by the senior author on the acquisition of high-quality EMG data following the CEDE[40] recommendations and SENIAM[41] guidelines, and had over 40 hours of practice inserting and removing the intravaginal electrodes

Table 1. Inclusion/Exclusion criteria.

Group	Inclusion Criteria	Exclusion criteria
Provoked vestibulodynia	• history of recurrent vulvar pain experienced on touch/pressure of the vulvar vestibule lasting >6 months, • PVD could be primary (pain experienced since the first attempts at vaginal insertion) or secondary (pain experienced after some period of pain-free vaginal insertion activities).	• no history of pelvic pain beyond the vulva, and no signs or symptoms of vaginal infection or dermatological condition at the time of recruitment • pregnant or within the first post-partum year • previous surgery that altered pelvic anatomy • known neurologic, metabolic or cardiovascular disease • current or past gynaecologic conditions that may cause dyspareunia (eg, endometriosis, uterine fibroids, vulvar or vaginal fissures or dermatologic conditions, etc.) • expressed anxiety related to the study methods.
Control	• no history of vulvar pain or painful vaginal insertion activities (sexual or non-sexual).	

prior to beginning the study. The physiotherapist also received >30 hours of instruction and practice using the custom V-QueST device[42] to measure pressure pain threshold at the vulvar vestibule.

Height and weight were measured using standardized techniques. Participants were then positioned supine on an examination table. The physiotherapist first assessed levator ani muscle (LAM) strength (Modified Oxford Scale, 0-5) through digital palpation,[30,31] then evaluated vulvar pressure pain threshold using a custom device, the V-QueST.[42] The cotton swab tip of the V-QueST was pressed perpendicularly into the skin at the vulvar vestibule using incremental force. The participant pressed a button when they first perceived the pressure as painful, and the force (g) was recorded as the pressure pain threshold (PPT). The median of three trials was retained.

We developed custom, recessed intravaginal suction electrodes to record EMG activity for the purposes of this study. These electrodes consist of a stainless-steel surface electrode (surface area 0.65cm^2) embedded within a circular casing. Once inserted into the vagina, the electrode surface is secured to the vaginal wall using a suction force generated through removing air from a syringe that is connected to the casing via catheter tubing (Figure 1). These electrodes, once inserted, do not apply pressure to the vulvar vestibule nor strain the levator ani or introitus, as only a flexible catheter extends out of the vagina. Our differential version of this electrode has been found to record EMG amplitudes during maximum voluntary contraction of the LAMs with acceptable within- and between- day reliability,[43] while being less affected by crosstalk contamination[44] than typical commercial EMG probes that have larger surface electrodes mounted on an intravaginal insert. The research physiotherapist first instrumented the deep PFMs by adhering the intravaginal suction electrodes. Over the pubovisceralis muscle belly laterally and the anterior vaginal wall, just lateral to the pubic symphysis on the same side to create a differential pair (Figure 1). Pairs of pre-gelled surface electrodes were then trimmed and adhered to the skin overlying the bulbocavernosus and external anal sphincter muscles. A common reference electrode was located over the right anterior superior iliac spine. All EMG electrodes were interfaced with Delsys Inc (Boston, United States) D.E 2.1 differential preamplifiers and Bagnoli-8 EMG Amplifiers, a 32-bit Analog to Digital Converter (NIDAQ USB3086, National Instruments, Texas, United States) and LabChart Pro Acquisition Software (AD Instruments, Colorado, United States). EMG data were sampled at 2 kHz.

Three 10s samples of resting (tonic) EMG activation were recorded from all EMG channels while participants paused their breathing at end exhalation and relaxed. Next, three PFM MVCs were performed; contractions were ramped to a maximum over one second, sustained for two seconds then let go. Lastly, participants performed three maximum effort Valsalva maneuvers whereby they used their abdominal muscles to increase intra-abdominal pressure while they exhaled strongly against a closed glottis and bore down, attempting to allow their perineum to descend. EMG data were recorded from one second before the command to inhale until 20 seconds after the command to bear down. Trials were repeated in cases where motion artifact was visualized during the task. A rest of two minutes was given between trials to minimize the effect of fatigue and allow the muscles to return to their rest state. Other assessment approaches were performed, including the evaluation of PFM responses to pressure at the vulvar vestibule and to transcranial magnetic stimulation (TMS), however these outcomes are beyond the scope of this report.

EMG time series data were full-wave rectified and low pass filtered (4th order dual-pass Butterworth filter with 3 dB cut-off of 5 Hz).[45] For activation at rest and Valsalva maneuver, the mean smoothed EMG amplitude was retained over the duration of the task, while for voluntary activation, the peak of the smoothed EMG amplitude was retained. Following international consensus guidelines, EMG data were normalized to account for high inter- and intra- individual variability in EMG amplitudes.[40,45,46]As such, resting and Valsalva EMG amplitudes were divided by the EMG activity recorded during the MVC and multiplied by 100 to generate a percentage of maximal voluntary activation (%MVC) for separate analysis.

Data analysis

Demographic and questionnaire outcomes were described and compared between controls and participants with PVD. All EMG data (resting, MVC, maximum effort Valsalva maneuver as recorded in microvolts, and normalized resting and Valsalva maneuver presented as a percentage of MVC were tested for normality using the Shapiro–Wilks' test. Separate General Linear Models were computed to determine the effect of group (PVD, control) on the EMG amplitudes recorded from each muscle during each task. Effect sizes (d) were computed and considered in the interpretation of the results. Further, to evaluate the impact of normalization to MVC on EMG amplitude during rest and straining, Pearson correlation

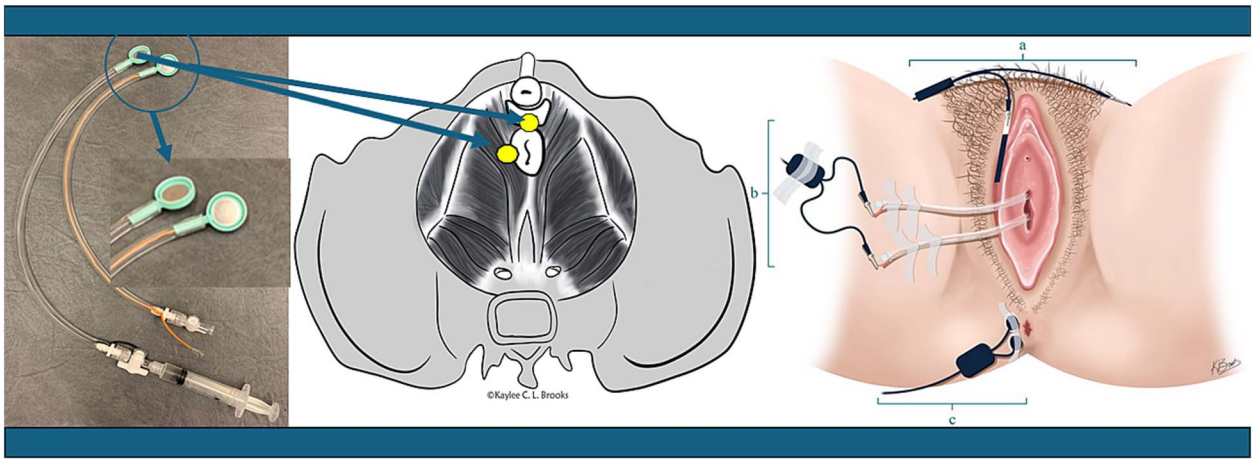

Figure 1. Electrode configurations. Recessed stainless steel electrodes (left panel) were placed on the vaginal wall overlying the pubovisceral (PV) muscle (locations indicated in middle panel) and held in place through removing 0.8-1 cc of room air using a syringe and stopcock assembly (seen (b) exiting the introitus in the right panel). Self-adhering surface electrodes (Ag/AgCl) were located over the (a) bulbocavernosus (BC) and (c) external anal sphincter (EAS) muscles on the right side as illustrated in the right panel. A common reference electrode was adhered to the skin overlying the right anterior superior iliac spine. All electrodes were interfaced with Delsys D.E.2.1 pre-amplifiers using adapted inputs (alligator clips).

Table 2. Demographic and Descriptive Data Presented by Group.

	Control (n = 43)	PVD (n = 42)	p
Age (years)	28 (7)	26(6)	0.23
BMI (kg/m^2)	24.4 (5.0)	24.5(5.3)	0.93
Parous (n, %)	3 (7.3%)	2(4.8%)	
PPT (g)	370.7 (138.3)	128.1(91.1)	**0.00**
DASS	6.7 (7.9)	11.1(9.7)	**0.04**
	6.4 (6.1)	9.4(6.4)	0.08
	11.4 (9.4)	16.7(9.2)	**0.03**
PCS	16.3 (10.8)	20.3(10.0)	0.12
FSFI	27.5 (6.3)	18.0(6.8)	**0.00**
CSI	33.5 (13.0)	41.9(13.3)	**0.01**
MPQ	2.8 (9.9)	26.8(13.3)	**0.00**

Abbreviations: PVD (Provoked Vestibulodynia), BMI (Body Mass Index), PPT (Pressure Pain Threshold), DASS (Depression Anxiety Stress), PCS (Pain Catastrophizing Scale), FSFI (Female Sexual Function Index), CSI (Central Sensitization Inventory), MPQ (pain reported on the McGill Pain Questionnaire).

coefficients (r) were computed between normalized and non-normalized outcomes (tonic and maximum effort Valsalva maneuver).

Pearson's correlations were also computed between EMG amplitudes and pressure pain threshold, and between EMG amplitudes and all patient reported outcomes (the McGill Pain Questionnaire, the Female Sexual Function Index, the Pain Catastrophizing Scale, the CSI and the Depression-Anxiety-Stress Scale). to address the second aim. Lastly, as a secondary analysis, outcomes were compared between those in the PVD group who did and did not demonstrate a palpable vaginal contraction on digital insertion general linear models ($\alpha = 0.05$).

The sample size was determined a priori based on Gentilcore-Saulnier et al.,[20] in which participants with PVD demonstrated significantly higher normalized (%MVC) tonic activation amplitudes of their bulbocavernosus muscle (mean = 2.97uV; sd = 0.24uV) compared to those without PVD (mean = 2.38uV; sd = 0.15uV), suggesting a required sample size of n = 5 per group would detect significant group differences (1-ß = 0.80; $\alpha = 0.05$). In the same study, the between-group difference in normalized (%MVC) tonic activation of the pubovisceralis muscle (mean difference = 0.52uV; effect size = 0.94) suggested that a sample size of 19 per group would be adequately powered to detect a difference. Because it was unclear whether the presence of a palpable contraction of the PFMs during intravaginal palpation might reflect greater PFM overactivity and thus influence outcomes, and because we expected that approximately half of the sample with PVD would have a palpable PFM contraction during digital insertion,[47,48] allowing for data loss, we aimed to recruit 42 participants with no history of vulvar pain and 42 participants with PVD, to have n = 20 with and 20 without a palpable PFM contraction during digital palpation. Using G*Power software (v.3.1.9.2), sample sizes of n = 20 per group were also deemed to be adequate to detect significant moderate correlations (r = 0.5) between EMG amplitudes and pain sensitivity and questionnaire outcomes.

Results

Flow of participants

Two hundred and six volunteers were screened, ultimately leading to a sample size of 85 (n = 43 control; n = 42 PVD), among which n = 7 were identified as having a palpable contraction of the PFMs during the insertion of a digit into the vagina (Figure 2). Sample demographic information and descriptive outcomes are presented in Table 2.

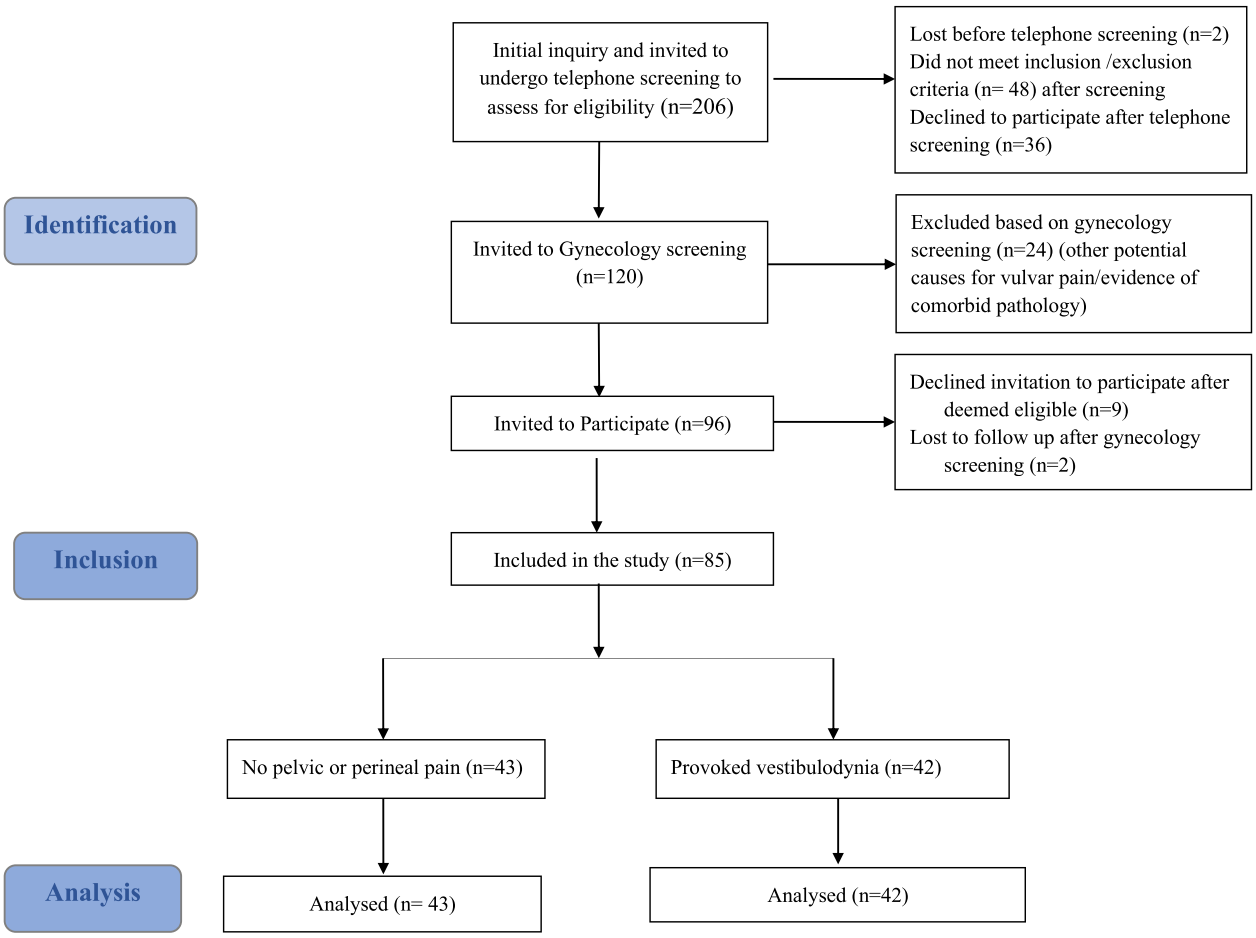

Figure 2. Participant flow diagram.

EMG amplitudes

EMG amplitudes presented in microvolts, thus non-normalized, are presented in Figure 3. In all muscles, PFM EMG amplitudes recorded at rest from participants with PVD were higher than controls ($0.40 < \text{Cohen's } d < 0.50$). Higher activation was also observed in the bulbocavernosus ($d = 0.44$) and external anal sphincter ($d = 0.46$) muscles during maximum effort Valsalva maneuver among those with PVD, with a trend toward higher activation observed in the pubovisceralis muscle as well ($d = 0.45$). During the MVC, there were no significant differences in EMG amplitude recorded from the pubovisceralis muscle ($d = 0.11$), but significantly greater activation was recorded from the bulbocavernosus ($d = 0.67$) and external anal sphincter ($d = 0.54$) muscles among those with PVD compared to controls.

No group differences were evident in the EMG amplitudes recorded at rest or during Valsalva when the data were normalized by the MVC, except at the pubovisceralis, where resting activity was higher among those with PVD compared to controls ($d = 0.42$). Normalized and non-normalized resting EMG amplitudes were uncorrelated [ie, resting activation of the pubovisceralis, $r = 0.278$ ($P = 0.074$), resting activation of the bulbocavernosus, $r = 0.184$ ($P = 0.244$), resting activation of external anal sphincter, $r = 0.279$ ($P = 0.063$)] while normalized and non-normalized EMG amplitudes during the maximum effort Valsalva maneuver were moderately correlated for pubovisceralis ($r = 0.397$, $P = 0.009$)] and external anal sphincter ($r = 0.337$, $P = 0.029$) but not for bulbocavernosus ($r = -0.038$, $P = 0.811$).

There were no significant differences in any EMG outcomes between participants with PVD who presented with and without a palpable contraction of their PFMs on digital insertion.

Relationship between EMG amplitude and pain sensitivity, psychosocial outcomes

No significant correlations were found between PFM EMG activation amplitude and pain sensitivity (Table 3). For pubovisceralis, the only significant correlations were between normalized resting EMG amplitude and Central Sensitization Index score ($r = -0.313$, $P = 0.044$), between EMG amplitude during Valsalva maneuver and Central Sensitization Index total score ($r = -0.324$, $P = 0.036$), and between EMG amplitude during MVC and the Stress score on the Depression-Anxiety-Stress Scale ($r = 0.343$, p-0.026). For the bulbocavernosus, the only significant correlation was between normalized EMG activity recorded during the Valsalva maneuver and the PCS Score ($r = -0.319$, $P = 0.040$). No significant correlations were found between EMG activation of the external anal sphincter and any patient reported outcomes.

Participants with a palpable PFM contraction on digital insertion ($n = 7$) were not different from those with PVD who demonstrated no palpable contraction of the PFMs on digital insertion in terms of demographic characteristics. They

Table 3. Associations Between Pelvic floor muscle activity recorded at rest and vulvar pain sensitivity and psychological/psychosexual outcomes.

	Pubovisceral muscle activation rest (uV)	Bulbocavernosis muscle activation at rest (uV)	External anal sphincter muscle activation at rest (uV)	Pubovisceral muscle activation at rest (normalized to MVC)	Bulbocavernosis muscle activation at rest (normalized to MVC)	External anal sphincter muscle activation at rest (normalized to MVC)
PPT (g)	0.079	0.097	0.051	−0.024	0.130	0.018
	(0.619)	(0.501)	(0.751)	(0.879)	(0.412)	(0.909)
MPQ	−0.033	0.270	−0.076	0.036	0.146	−0.149
	(0.839)	(0.088)	(0.637)	(0.822)	(0.363)	(0.352)
FSFI	−0.166	−0.054	0.259	−0.064	0.007	0.214
	(0.294)	(0.732)	(0.098)	(0.686)	(0.964)	(0.174)
PCS	−0.016	0.220	−0.034	−0.109	−0.206	−0.091
	(0.921)	(0.161)	(0.829)	(0.494)	(0.191)	(0.565)
CSI	0.090	0.247	0.186	**−0.313**	−0.048	−0.274
	(0.571)	(0.114)	(0.238)	**(0.044)**	(0.674)	(0.079)
DASS-D	−0.108	−0.075	0.035	−0.096	−0.052	−0.132
	(0.497)	(0.606)	(0.826)	(0.544)	(0.742)	(0.406)
DASS-A	0.096	0.028	−0.085	0.054	0.137	−0.001
	(0.547)	(0.861)	(0.592)	(0.736)	(0.386)	(0.997)
DASS- S	0.131	−0.107	−0.183	−0.118	0.003	−0.045
	(0.408)	(0.501)	(0.286)	(0.456)	(0.986)	(0.775)

Outcomes are Pearson correlation coefficients and associated p-values [r(p)]. Abbreviations: PPT (Pressure Pain Threshold), MPQ (pain reported on the McGill Pain Questionnaire), FSFI (Female Sexual Function Index), PCS (Pain Catastrophizing Scale), CSI (Central Sensitization Inventory), DASS (Depression Anxiety Stress Scale). Bolded values indicate significant correlations at $\alpha = 0.05$.

reported significantly more depressive symptoms on the DASS [(9.4(9.4) vs 19.6(6.7), $P = 0.001$], less catastrophizing on the PCS [21.7(10.1) vs 13.1(5.5), $P = 0.028$] and less pain severity on the MPQ [28.9(12.8) vs 16.7(11.8), $P < 0.001$] compared to those without PVD.

Discussion

Principal findings

PVD is associated with overactivity of the PFMs. In particular, the superficial (bulbocavernosus and external anal sphincter) PFMs demonstrate greater EMG activation at rest, during MVC and during maximum effort Valsalva maneuver. The deep (pubovisceralis) PFMs demonstrate greater activation at rest among those with compared to those without PVD, but not greater activity during MVC or maximum effort Valsalva maneuver. While pressure pain sensitivity at the vulvar vestibule was higher among those with PVD compared to controls it was not associated with EMG activation amplitudes recorded from any of the three PFMs during any of the tasks. As such, activity of the PFMs in those with PVD does not appear to be associated with the extent of local pain sensitivity. There were very few significant correlations between EMG activation recorded from the PFMs and psychological/psychosexual outcomes. The direction of the significant correlations between pubovisceralis activation and the Central Sensitization Index did not support the hypothesis that those with a more sensitized the nervous system have greater EMG activity of their PFMs. While pubovisceralis activation during MVC was positively correlated with higher levels of stress reported on the Depression-Anxiety-Stress Scale, given the large number of correlations computed and the lack of corroborating correlations, this finding may be spurious.

Results in the context of what is known

The findings of this study are in line with previous reports of greater tonic activation of the superficial[20,21] PFMs among women with PVD compared to controls. However, in Gentilcore-Saulnier et al.,[20] significant differences between groups were found in normalized tonic EMG amplitudes, while in the current study no group differences were found in the normalized tonic activation amplitude of the bulbocavernosus muscles, because EMG amplitudes were also higher among those with PVD during the MVC. In Gentilcore-Saulnier et al.,[20] the group difference in normalized tonic activation amplitude of the pubovisceralis muscle was not significant, whereas it reached significance in this larger sample.

The findings of this study are not consistent with reports by Næss and Bø[22] nor Polpeta et al[23] who reported no differences between women with and without PVD when reporting non-normalized tonic EMG activation of the PFMs, yet both of these groups used large electrode probes that were not specific to the pubovisceralis and failed to meet international standards.[40,41] Consistent with the bearing down task of Frasson et al.,[21] those with PVD in this study demonstrated greater EMG activation of their bulbocavernosus and external anal sphincter while performing the maximum effort Valsalva maneuver than was observed in the control group. A trend was also observed in the pubovisceralis muscle. This finding may be due to protective contractions[48,49] and/or greater muscle spindle excitability[21] in response to lengthening. The nature of this finding should be investigated further.

The low proportion of women with palpable PFM contraction during digital insertion into the vagina (7 of 42) was unexpected given the large overlap between genital pain, fear variables and vaginal muscle tension reported in the literature.[47-49] While we recognize that individuals with high levels of fear of intravaginal palpation (vaginismus) would not likely volunteer to participate in a study such as this, we expected that approximately half of our PVD sample would exhibit some degree of paravaginal muscle contraction on insertion of a digit into the vagina related to pain, fear of pain, a learned behavior or other psychological or psychosexual cause. We planned this disaggregation to provide additional insight into the nature of PFM involvement in PVD.[48] While the small number of participants with palpable PFM contraction during digital insertion precluded meaningful inferences about these

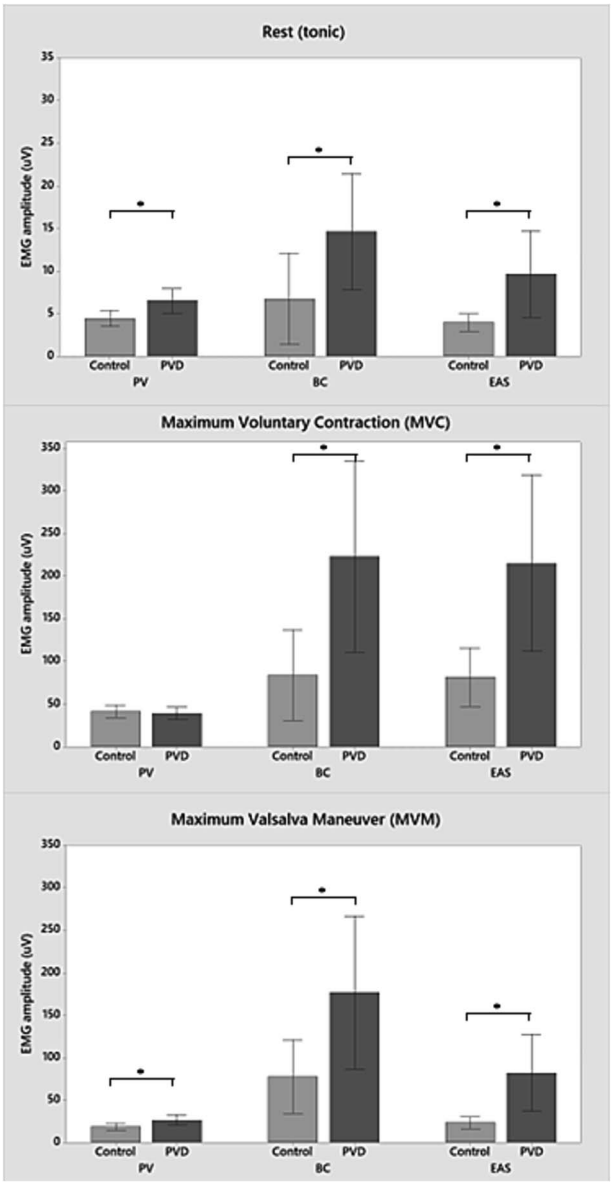

Figure 3. EMG amplitudes recorded, in microvolts, from the puboviscerali s (PV), bulbocavernosus (BC) and external anal sphincter (EAS) muscles at rest, during maximum effort Valsalva maneuver and during maximum effort voluntary contraction (MVC) performed by participants with (n = 42) and without (n = 43) provoked vestibulodynia (PVD). Note scale differences between panels on y-axes. Bars indicate mean values with associated 95% confidence intervals.

disaggregated groups, our preliminary analyses suggest that involuntary PFM contraction during digital insertion is not associated with greater pain catastrophizing nor lower sexual function.

The lack of association between pressure pain sensitivity and EMG activation of the PFMs is consistent with Benoit-Piau et al.[28] who found that, among a large sample of women with PVD (n = 173), PFM tone was not correlated with pain reported during sexual activities. Thus PFM overactivity and pain sensitivity likely require separate consideration in terms of evaluation and management.

Clinical implications

The findings of this study suggest that high PFM tone among those with PVD has, at least in part, a neuromuscular origin. And the finding that EMG activation was higher in all three PFMs studied and among different tasks (ie, MVC and maximum effort Valsalva maneuver) suggests that PFM overactivity is not only implicated in PFM tone, but is a more generalized neuromuscular feature of PVD. That said, PFM overactivity appears to be unrelated to pain sensitivity and psychosocial/psychosexual states or traits. These findings support physiotherapy approaches focused on relaxation and control of the PFMs as a component of multimodal and multidisciplinary therapy, but not as the only component of therapy.

Research implications

For the bulbocavernosus and external anal sphincter muscles, normalization to MVC masked the large between-group differences observed in the raw data because of larger EMG activation recorded during MVC among those with PVD. Indeed, there was a lack of correlation between the normalized and non-normalized EMG amplitudes. While caution is advised when evaluating non-normalized EMG data,[40] in this situation it is important to consider the impact of group differences in MVC values on normalization outcomes. The larger EMG amplitudes observed in the bulbocavernosus and external anal sphincter among those with PVD during all tasks (rest, bearing down, MVC) may reflect enhanced corticomotor drive to these muscles. Further research is needed to evaluate whether higher PFM activation among women with PVD may be associated with anticipatory or protective responses and/or other factors.

Strengths and limitations

The design of this study was superior to other research in this space. We used a blinded assessor, appropriate and specific EMG electrode configurations which did not touch the vaginal fourchette or create a lengthening force at the introitus or the levator hiatus, and quantitative assessment of pressure pain sensitivity. By analyzing both normalized and non-normalized EMG data, the findings shed light on the presence of PFM overactivity among women with PVD.

While including several outcome measures (three muscle sites, three tasks) may have increased the likelihood of Type II error, this appears to be unlikely since the model outcomes in aim 1 were consistent and Cohen's d effect sizes were moderate ($0.34 < d < 0.67$). For the second aim, as noted above, the few correlations that were found were mostly in the opposite direction to what we expected.

Despite the use of electrodes designed specifically to record PFM activity intravaginally, it is possible that the EMG signals included some crosstalk from nearby muscles, particularly the obturator internus muscle which shares an attachment with the levator ani. To mitigate this, the researchers observed the task performance and discarded trials where there was obvious contraction of the muscles around the hip, yet no formal evaluation of crosstalk was possible based on the study protocol. Methods were implemented to mitigate the impact of motion artifact on the outcomes through both the data collection and analysis phases.

As noted above, the sample is generalizable to those with genito-pelvic pain penetration disorder who exhibit signs and symptoms consistent with PVD without concurrent vaginismus. Further, while we attempted to incorporate a biopsychosocial perspective through the secondary objective, we did not fully evaluate potential confounding psychosocial factors

such as a history of sexual trauma or abuse, sociocultural influences gender identity or roles or other influences which are essential to our understanding of dyspareunia in general and vaginismus in particular.[50]

Conclusion

PVD appears to be associated with overactivity at rest of the superficial (bulbocavernosus and external anal sphincter) and deep (pubovisceralis) PFMs, and in the superficial PFMs during maximal contraction and Valsalva maneuvers. The extent of PFM overactivity is not associated with the extent of local hypersensitivity at the vulvar vestibule, nor with psychological or psychosexual outcomes.

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Conflicts of interest

Linda McLean is a Scientific Advisor for Cntrl+ Inc. (Canda) and Freyya Inc. (United States).

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