Original article

Mapping chronic urogenital pain in women: insights into mechanisms and management of pain based on the IMAP Part 2

MAREK JANTOS¹, SHERIE JOHNS², ANNA TORRES³, EWA BASZAK-RADOMAŃSKA⁴

- ¹ Behavioural Medicine Institute of Australia, South Australia & Department of Human Anatomy, Medical University of Lublin, Poland
- ² School of Nursing and Midwifery, University of South Australia ³ Department of Human Anatomy, Medical University of Lublin, Poland

Abstract: Objective: To introduce the integrated mapping and assessment protocol (IMAP), designed to systematically localise the origins of pain in chronic urogenital pain (CUP) syndromes. Method: A retrospective analysis of IMAP data, from 82 participants, comparing pain scores of two CUP subgroups; vulvodynia and bladder pain syndrome (BPS), with asymptomatic controls. A further analysis was carried out to assess the use of the IMAP as a tool for evaluating the impact of interventions. Results: The IMAP scores showed significant differences between CUP syndromes and asymptomatic controls. Pain scores from the two CUP subgroups confirmed that pain of pelvic muscle origin is characteristic of chronic pain conditions, and does not represent the normal state. The paraurethral area was shown to be the source of the highest pain scores, when compared with other maps. Analysis of pre and post intervention pain scores indicated a statistically significant reduction of pain scores, demonstrating the IMAP's sensitivity to intervention-related change. Conclusion: The IMAP is a promising tool in localizing the origins of pain, and providing insight into the role of peripheral pain mechanisms in CUP syndromes. Mapping will assist in the development of evidence-based interventions, and provides an objective means of evaluating their impact.

Keywords: Chronic urogenital pain (CUP); Vulvodynia; Bladder pain syndrome (BPS); Integrated mapping and assessment protocol (IMAP); Ischemia.

INTRODUCTION

The integrated mapping and assessment protocol (IMAP) was specifically developed for the mapping of chronic urogenital pain (CUP). CUP syndromes represent a group of functional pain disorders, which, in contrast to structurally related pain problems, lack well-defined pathophysiology and consequently are poorly understood. Mapping, whether in the mathematical, medical or geographical sense, is the process of delineating, linking, matching and establishing relationships between different sets of points, spaces, regions, themes, functions or symptoms. Scientists have mapped the brain, chromosomes, and peripheral nervous, circulatory and lymphatic systems but little work has been done on the mapping of pain disorders. This pilot study aims to systematically map the origins of CUP.

The science of mapping is referred to as cartography, or in the context of pain, as cartography of pain, and is commonly known as pain mapping. 1 In keeping with the current paradigm shift in the study of CUP, where greater emphasis is being placed on the need to understand the mechanisms of pain, innovative methods are required to develop evidence-based management strategies for disorders such as vulvodynia and bladder pain syndrome (BPS).

The IMAP as a tool is designed to localize the origins of pain, assess its severity, characteristics and spatial distribution. As a process, it engages the patient in delineating pathways that link the source of pain with "their" symptoms, and empowers and reassures them that their pain is not linked to sinister causes. Likewise, it enables the clinician to be discerning in the choice of suitable interventions. Intuitively, pain mapping, using the IMAP, shifts the focus to peripheral mechanisms involving dysfunctional muscle states as discussed in Part 1 of this series.2

Structurally and functionally the pelvic floor muscles (PFM) provide support to the pelvic organs; maintain urinary and faecal continence; enable sexual intercourse; facilitate parturition; provide postural support and assist with movement. The PFM form a musculoskeletal complex that is one of the most intricate in the human body,3 consisting of a large number of individual muscles, numerous ligaments and fascia that hold in place all of the pelvic viscera. The pelvic muscles are organised in several layers which span the pelvic cavity, and form a horizontally oriented platform.4 These layers of soft tissue are penetrated anteriorly by the urethra, centrally by the vagina and posteriorly by the anorectum, in an area known as the urogenital hiatus or pelvic outlet. Because the pelvic muscles relate to more than one organ system, their dysfunction can impact multiple systems at the same time, and each system can be a potential source of pain and symptoms. For purposes of pain mapping the PFM are accessible to digital and Q-tip palpation.5 This form of assessment is in keeping with the guidelines for the study of vulvodynia, BPS and pelvic pain dis-

The meaningful interpretation of pain maps requires comparison with normative data from asymptomatic women, who have no history of lower urinary tract dysfunction. A recent study assessed pelvic muscle tenderness in nulliparous, asymptomatic women, 18-30 years of age.¹⁰ The level of pelvic muscle tenderness was measured using the visual analogue scale (VAS). Using a cut off pain rating score of 3, pain \leq 3 was considered clinically not significant, whereas pain scores >3, were deemed to be significant. The study found that in all of the 17 participants, there was no tenderness. Furthermore, none of the subjects had high-tone PFM. The study concluded that pain on palpation could not be considered a variation of normal. By contrast, the incidence of pelvic muscle tenderness in bladder pain patients was reported to be as high as 94%, with the majority showing dysfunctional, hypertonic pelvic muscles.11 These studies show that in asymptomatic, nulliparous women, without lower urogenital tract symptoms, the pelvic muscles should be painless and of normal tone, and that a pain score > 3 should be considered as an uncommon finding.10

There is general agreement that pain, commonly attributed to painful end-organs such as the bladder, bowel and external genitalia, arises from high-tone pelvic muscles.² Furthermore, that dysfunctional pelvic muscles account for

⁴ Terpa Clinic, Lublin Poland

urinary urgency, constipation, dyspareunia, and neurogenic inflammation of the bladder. ¹² These conclusions are consistent across studies that have integrated muscle assessment into their research designs.²

While the role of dysfunctional pelvic muscles in CUP symptoms is acknowledged and better understood, there are no detailed pain maps localizing the origins of symptoms and pain. Earlier reports demonstrated a high degree of specificity with which certain symptoms can be linked to focal areas of pelvic anatomy.^{13, 14} This study, using the IMAP, undertook a comprehensive analysis of CUP mapping of the external urogenital area, internal pelvic muscles, and the paraurethral/bladder region with the aim of localizing the source of pain and symptoms.

METHOD

This study is based on a retrospective review of pain maps from 82 patients who were assessed by the authors. Patient data was obtained from two study centres, a CUP clinic in Adelaide, South Australia, and the Terpa Clinic in Lublin, Poland, in collaboration with the Medical University of Lublin.

The inclusion criteria for the CUP group consisted of consecutive patients diagnosed with CUP, aged 18-60, who attended urogenital pain consultations between January and December 2014. The exclusion criteria were: concurrent illness with overlapping symptoms; history of pelvic reconstructive surgery, and any pelvic surgical procedures, pregnancy, birth or lactation within the last 3 months. The control group consisted of 28 asymptomatic women who met the inclusion criteria, who were undergoing a regular gynaecological examination.

Of the 82 participants, only 70 were retained for the final analysis. A total of 9 CUP participants were excluded: 7 on account of coexisting infections and medical problems, and 2 due to prolapse. Of the remaining 45 CUP participants, 27 were diagnosed with vulvodynia and 18 with BPS. Of the 28 participants in the control group, 25 were retained for the final analysis. Two were excluded on account of lower urinary tract symptoms disclosed during assessment, and one on account of severe pelvic muscle tension and pain during internal mapping. This case was of interest as she presented as asymptomatic, but reported adhering to a rigorous, daily pelvic muscle exercise program, which resulted in hypertonic and painful pelvic muscles, which were identified during mapping.

Pain mapping was performed in the lithotomy position on an empty bladder. For each palpation point the patient was asked to provide three items of information; pain rating on a 0-10 numerical rating scale (NRS), pain characteristics, using up to three adjectives from a modified McGill Pain Questionnaire list; 15 and description of the spatial distribution of pain. Only pain scores were analyzed in this study; pain descriptors and spatial distribution will be reported in a separate study. A therapeutic intervention was performed on a subgroup of CUP participants (n=13) in order to evaluate the IMAP's ability to detect change in pain scores. Pain maps were completed prior to the commencement of therapy, and at its conclusion. The intervention consisted of electromyographic (EMG) assisted relaxation training, myofascial therapy, pelvic massage and desensitization of the paraurethral points.

The three pain maps were developed on the basis of clinical work, research studies and cadaver dissections. The cadaver studies specifically focused on the anatomical, physiological and functional relationships between the vulvar, urethra and bladder. While validating the three pain maps,

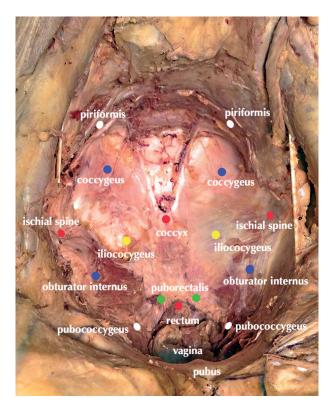


Figure 1a. – Cadaver image showing anatomical reference points (marked red), and internal pelvic muscle palpation sites (marked white).



Figure 1b – Pelvic Floor Image of Cadaver - palpation.

reference and mapping points were identified for internal pelvic muscles, these are marked in Figure 1a, and palpation is demonstrated in Figure 1b.

Mapping the external urogenital area (Map 1). The first map focused on the external urogenital area as shown in Figure 2.

Muscles marked with a solid black dot were palpated digitally, while points marked with a white dot were mapped using a moistened Q-tip. The pressure used ranged

UROGENITAL PAIN MAP (Inferior View) (I

Figure 2. - Urogenital pain map (Map 1), identifying external points for pain mapping.

from 0.1 - 0.2 kg/cm² for Q-tip palpations, and 0.4 - 0.5 kg/cm² for digital palpation of superficial muscles as based on an earlier study. 16 Several devices have been tested by the authors, but were considered not conducive to the task of intravaginal pain mapping. 17,18 The clinician's skill and training in locating pelvic muscles and applying correct and consistent pressure are a requisite for quality mapping. The urogenital pain map labelled points around the clitoris, ure-

thra, vestibule and anus on the basis of the perineal clock. However, the order of palpation did not follow the clock sequence in order to minimise the patient's anticipatory reactions.¹⁹

Mapping internal pelvic muscles (Map 2). The second map consisted of the palpation of internal pelvic muscles as shown in Figure 3.

PELVIC MUSCLE PAIN MAP

(Superior View)

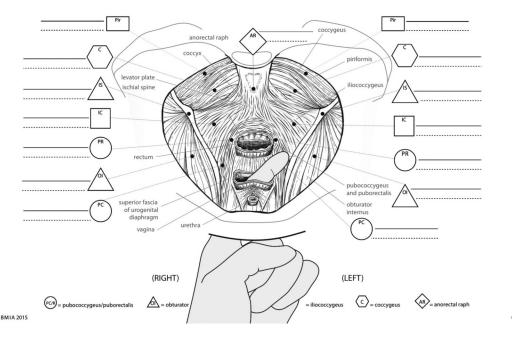
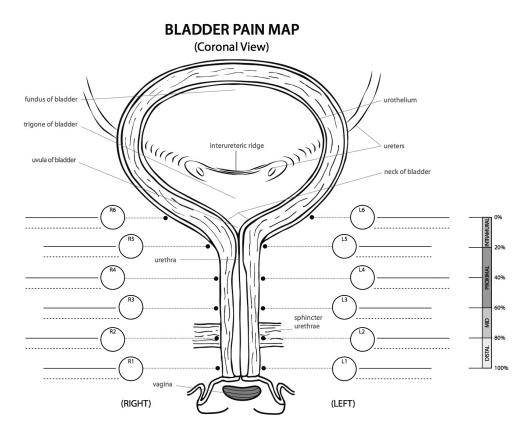


Figure 3. - Pelvic muscle map (Map 2), identifying internal points for pain mapping.

The pressure used during assessment of the internal muscles ranged from 0.4 to 0.5 kg/cm². A single digit was gently inserted into the vaginal canal to palpate the levator ani, and muscles lateral and posterior to the introitus. Each participant was then asked to voluntarily contract and relax the PFM, to assess general muscle strength using the six-point Oxford Scale.²0 In CUP cases the PFM are frequently in a non-relaxing state, revealing a functional contracture, which has also been shown by EMG assessments.²1 A healthy pelvic muscle should feel bulky, but well elasti-

cized, maintaining appropriate pressure around the examiners finger, with no pain on contact.²²

Mapping the para-urethral area (Map 3). When palpating the paraurethral area, a maximal pressure of 0.4 — 0.5 kg/cm² was applied, always starting with very light pressure and progressively increasing to maximal pressure. The paraurethral points were palpated lateral to the urethra, as shown in Figure 4.



3MIA 2015 v2

Figure 4. - Bladder pain map (Map 3), identifying paraurethral points for pain mapping.

Due to the fact that the paraurethral area is not normally examined as a source of pain, there are no protocols in literature for its assessment. The rationale for the protocol used in this study is based on the anatomical structure of the urethra. The urethra is a multilayered hollow tube of approximately 4 cm in length. It is surrounded by striated and smooth muscle layers organised in circular and longitudinal formations. For much of its length the urethra is fused with the anterior vaginal wall. At the level of the levator ani muscle the urethra is "incorporated into the pelvic floor rather than piercing a layer of muscle tissue". Since the urethra, bladder and supporting structures of the pelvis are all part of the pelvic floor, the tone and sensitivity of the urethra are closely linked to pelvic muscle tone.

Anatomically the urethra can be divided longitudinally into percentiles, with the opening of the internal vesical neck representing point 0, and the external meatus representing the 100th percentile.^{4, 23} Detrusor muscle fibres extend as far as the 15th percentile and the striated urogenital sphincter muscle begins where the detrusor muscles fibres end and extend to the 64th percentile.⁴ For mapping purposes palpation points are separated in 20th percentile increments, beginning at the external meatus around the 100th

percentile, then moving up to the 80th, 60th 40th, 20th, and 0 at the level of the vesical neck. An additional palpation point, estimated at a 20th percentile above the bladder neck, is recommended to assess pain originating from the trigone.

Analyses. Total pain scores were computed for each pain map for the three groups. The following analyses were undertaken; calculations of demographic data; means of pain scores across three maps and nine anatomical regions for the three groups; a comparison of pre and post treatment scores for CUP groups; and comparison of post treatment scores of the two diagnostic groups and the control group. The aim of the pre and post comparison was to show the sensitivity of the IMAP to changes in muscle pain scores.

As the study data consisted of a retrospective review of de-identified patient information, based on the written consent of each participant, no formal institutional approval was required.

RESULTS

A total of 82 participants were enrolled in the study, and data from 70 of the participants was used in the final statis-

tical analysis. Following 12 exclusions, 27 women remained in the vulvodynia group, 18 in the BPS group, and 25 in the control group. Statistical analysis was performed using SPSS v22.0.

There were no significant differences between the age means for vulvodynia, BPS and control group (Age Means = 34.4, 31.0, 33.9, respectively, F(2,67) = 0.70, p = 0.50). A significant positive correlation was noted between age and parity (r = .53, p < .001). A significant negative correlation was found between age and external urogenital pain scores (Map 1), for the vulvodynia and BPS groups, (r = .28, p = .02). There was no significant negative correlation between age and internal pelvic muscle pain (Map 2), for either diagnostic group (r = .18, p = .14); but there was a significant negative correlation between age and paraurethral pain (Map 3) for the combined diagnostic groups (r = .26, p = .03).

One-way ANOVAs were applied to test for differences between the vulvodynia, BPS, and control groups, in the average pain scores derived from the three pain maps. There were significant group differences for all three maps: external urogenital map (Map 1), F(2, 35.55) = 28.1, p <.001); internal pelvic muscle map (Map 2), F(2, 35.01) =

Pain Means by Maps

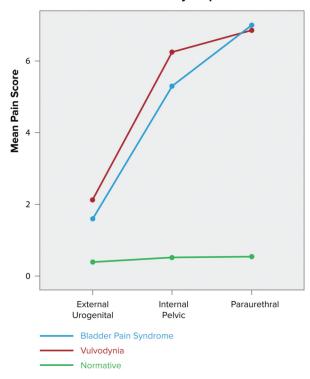


Figure 5. – Comparison of three pain map means for vulvodynia, BPS and asymptomatic controls.

88.3, p<.001; paraurethral map (Map3), F(2, 39.12) = 100.79, p<.001) as shown in Figure 5.

Post-hoc pairwise t-tests revealed that there were significant differences between controls and vulvodynia groups, and control and BPS groups, for all three pain maps. There were no significant differences in pain scores on each of the pain maps between the vulvodynia and BPS groups. The vulvodynia and BPS group experienced the least pain with the external urogenital pain points (Map 1), significantly more pain with internal pelvic muscles (Map 2) and most pain with the paraurethral points (Map 3).

An analysis of pain by nine specific anatomical regions, showed the lowest pain scores to be from the suprapubic, clitoral, anal and superficial muscles (as per Map 1).

Higher scores were provided for the urethral and vestibular regions (as per Map 1). A significant increase in pain scores for the two internal pelvic muscle regions - the levator muscles and obturator internus, and piriformis and coccyx (as per Map 2). Maximal pain scores were recorded in the paraurethral area (as per Map 3). The regional pain scores are shown in Figure 6.

Pain Means by Anatomical Region

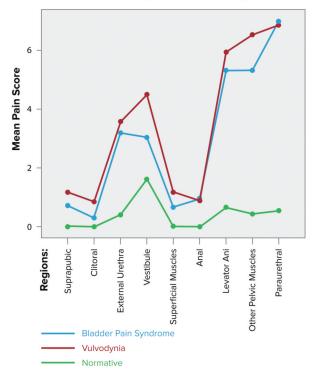


Figure 6. – Comparison of means of nine anatomical regions for vulvodynia, BPS and asymptomatic controls.

There were no significant differences in regional pain scores between the vulvodynia and BPS groups. Both groups showed the same pain score trends on all three maps.

 \bar{A} 3 x 2 x 2 mixed ANOVA was applied to investigate the relationship between the pain maps, treatment, and diagnosis. This three-way interaction was non-significant, F(2, 11) = 1.96, p = .16). The main effect of treatment was significant, F(1, 11) = 166.05, p < .001). Patient pain scores were significantly lower after treatment. However, the treatment by diagnosis interaction was non-significant, F(1, 11) = 0.02, p = .90). This suggests that the effect of the treatment was the same for both the vulvodynia and BPS groups, for each of the pain maps as shown in Figure 7.

A 3 x 2 x 2 mixed ANOVA was applied to investigate the relationship between the nine pain regions, treatment, and diagnosis. This three-way interaction was non-significant, F(8, 11) = 1.23, p = .29). The main effect of treatment was significant, F(1, 11) = 96.14, p < .001). Patient pain scores were significantly lower after treatment. However, the treatment by diagnosis interaction was non-significant, F(1, 11) = 0.06, p = .81). This suggests that the effect of the treatment was the same for both the vulvodynia and BPS groups, across all the pain regions as shown in Figure 8.

In the final analysis, the post treatment pain scores for the vulvodynia and bladder pain group showed no significant differences to the asymptomatic, control group pain scores for all three pain maps (external urogenital map, F(2, 35) = 0.25, p = .78); internal pelvic, F(2, 35) = 0.77, p = .77; and paraurethral, F(2, 35) = 0.79, p = .79).

Pain Map Means Pre & Post Treatment

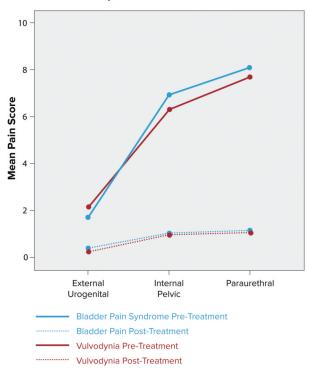


Figure 7. – Comparison of pain map means for pre and post treatment scores (three maps) for Vulvodynia and BPS.

Regional Pain Means Pre & Post Treatment

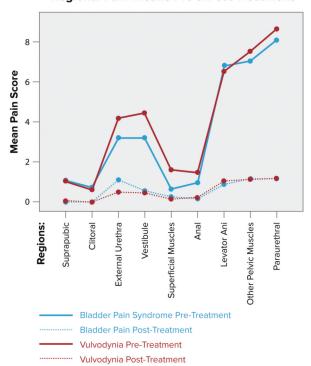


Figure 8. – Comparison of regional means for pre and post treatment scores (nine anatomical regions) for vulvodynia and BPS..

In the final analysis, the post treatment pain scores for the vulvodynia and bladder pain group showed no significant differences to the asymptomatic, control group pain scores for all three pain maps (external urogenital map, F(2, 35) = 0.25, p = .78); internal pelvic, F(2, 35) = 0.77, p = .77; and paraurethral, F(2, 35) = 0.79, p = .79).

DISCUSSION

This is the first study to provide a full urogenital pain map for the two CUP syndromes of vulvodynia and BPS. The results show that pain of pelvic muscle origin is characteristic of chronic pain syndromes. It is not a feature of asymptomatic controls, and does not constitute a normal state. This finding is consistent with a recent study assessing tenderness of pelvic muscles in asymptomatic nulliparous women.¹⁰

The results showed a significant negative correlation between age and pain on the urogenital and bladder pain map (Maps 1 & 3), indicating that pain was not linked to ageing. Quite to the contrary, it is the younger women who more frequently present with CUP symptoms. This is consistent with earlier findings showing that the prevalence of CUP disorders peaks at an early age.^{24, 25}

When the pain scores for vulvodynia and BPS groups were compared on the three pain maps, and across the nine anatomical regions, there were no significant differences between the two groups. This raises the important question of whether these two pain syndromes constitute different pain disorders, or form the same syndrome with variations in symptom presentation.²⁶ The data supports the later view and is consistent with the conclusions of other studies.^{27, 28}

The highest pain map scores were reported in the paraurethral area (Map 3). However, the areas traditionally mapped as part of diagnostic assessments for vulvodynia and BPS focus only on the external urogenital and perineal area. This study demonstrates that the paraurethral area may be of greater diagnostic and therapeutic relevance than the external urogenital region. This was consistent with the earlier findings of the authors. 13, 14 Only two other studies have made mention of bladder base tenderness, one in the context of dyspareunia, the other in the context of BPS.^{29, 30} In this study, paraurethral pain provoked by palpation was commonly described by patients as sharp, piercing, stabbing and burning pain, reproducing sensations of urge, suprapubic pressure, low abdominal pain, groin discomfort and in some cases, sensations of burning in the soles of the feet, as shown in Figure 8. Vulvodynia and BPS patients commonly use these pain descriptors, and palpation of the paraurethral points completely and accurately reproduced each patients symptoms.

An earlier study reported that BPS patients, on average, identified 2.1 pain sites.²⁷ Suprapubic pain was most commonly reported (83%), followed by urethral (36%), nongenital (29%) and genital (23%) pain. Pain was described as throbbing, tender, piercing and aching. Since 84% to 90% reported worsening of pain with bladder filling, it was hypothesized that the bladder was the generator of pain.²⁷ While the results of pain sites and descriptors are consistent, the current study showed that it is the paraurethral area, not the bladder that is in fact the generator of pain and symptoms.

The proximal paraurethral points associated with urge and frequency may be linked to the descent of the detrusor muscle fibres that extend down as far as the 15th percentile (refer to Figure 4). The sensation of urge increased in intensity with proximity to the bladder neck and the trigone. However, there were exceptions where urge was also reproduced by mid urethral points. These observations tend to confirm earlier reports that myofascial trigger points (TrPs) in the paraurethral area are linked to symptoms of frequency and urge as well as antidromic inflammation of the interstitium, as seen with BPS/IC.³¹

Some participants sought to differentiate between discomfort associated with urge and the experience of pain. They reported lower pain scores, yet experienced the most

PAIN of PARAURETHRAL ORIGIN

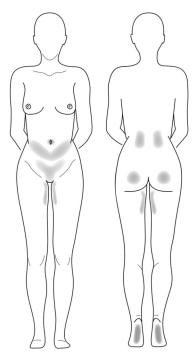


Figure 9. – Distribution of pain originating from the paraurethral

intense and distressing sense of urge when the proximal paraurethral points were palpated. Future studies need to differentiate between pain and the distress associated with an intense urge to void. During the course of therapy, as symptoms would abate, the rigidity of the urethra was reduced and the paraurethral area became more supple and markedly less sensitive. When the paraurethral area was successfully desensitized, urge triggered by palpation was eliminated and patients reported a reduction in voiding frequency. Even patients without voiding problems commented on reduced hesitancy and improved flow post desensitization.

Paraurethral hypersensitivity noted in this study is consistent with earlier studies by the authors which showed that palpation of paraurethral points reproduced bladder pain and voiding symptoms in 100% of BPS cases.¹³ The authors also found that in a subgroup of patients with clitorodynia and persistent genital arousal disorder, the upper paraurethral points reproduced clitoral pain and symptoms of arousal. Upon desensitization these symptoms would also subside.¹⁴

The extreme sensitivity of the paraurethral area appears to be a trigger for the bracing and guarding response of the deeper pelvic muscles. Such muscle reactions could be misdiagnosed as defining the involuntary muscle spasm noted in vaginismus. Yet, the reaction appears to be a normal pain reflex. In time, such a pain reflex may develop into an anticipatory protective reaction and become associated with anxiety, fear and phobia.

Lack of clinician's awareness of the existence of paraurethral hypersensitivity gives rise to a range of costly and invasive tests in search of pathology that might explain the persistent pain. Despite repeated laparoscopies and ultrasounds being performed, results are usually negative or pathology is disproportionate to the severity of pain. During the course of mapping the majority of patients stated that their pain and symptoms were accurately reproduced. Identifying the source of their pain was reassuring and reduced the perceived threat of more ominous causes

of pain.

Analysis of data from the deeper pelvic muscles map (Map 2) showed this to be the second most-tender area. These muscles have been the subject of various research reports in literature. ^{32,33} The levator muscle, as it supports the pelvic organs and encloses the hiatus, is structurally one of the most susceptible muscles to functional change. The over-activation of the levator ani muscle leads to a functional contracture resulting in the narrowing of the introitus and ischaemic hypersensitivity. ²¹ During pain mapping, palpation of the vestibule reproduced the sharp burning pain that women reported experiencing when wearing tight clothing, sitting or attempting penetration. Vestibular hypersensitivity is typically assumed to arise from peripheral nerve damage, yet appears to be the result of the hypertonus state of the levator ani muscles.

During pain mapping, the puborectalis was found to refer pain into the anorectal area and cause a sense of bowel urge. The coccygeus and piriformis muscles referred pain into the lower buttocks, coccyx and lower back. The obturator internus muscle accounted for intense pain radiating into the hip, lower back, abdominal quadrants and thigh region.

Mapping scores from the urogenital area (Map 3) provided the lowest overall pain scores for the vulvodynia and BPS groups. The pubic area, perineal body and superficial muscles, including the bulbocavernosus, ischiocavernosus and the transverse muscles, were not found to be generators of pain. The pain scores from the external urethral area and the vestibule received the highest pain ratings on the urogenital pain map. Pain in the anal sphincter area was rated low and appears to occur in conjunction with fissures, haemorrhoids, constipation and coccygeal pain.

The analysis of the pre and post treatment scores demonstrated the value of the IMAP to assess the effectiveness of a therapeutic intervention. According to patient's subjective estimates, the conclusion of therapy was marked by an 80-95% reduction in pain and voiding symptoms. The post mapping pain scores verified the patient's estimates by showing a statistically significant reduction in pain. The post therapy scores for vulvodynia and BPS patients showed no significant difference with the scores the control group. Some of the patients expressed the view that the treatment had provided them with a complete cure. A patient who previously suffered severe pain and voided at 15-minute intervals, noticed that she was voiding at 2.5-hour intervals and experienced no pain at the conclusion of therapy. Patients who suffered constipation reported restored regularity. Most patients were able to comfortably resume daily activities. The IMAP validated the patient's subjective estimates of improvement, and provided an objective means for evaluating the impact of a therapeutic intervention.

Given that pelvic muscles relate to more than one organ system, normalising the function of PFM appears to affect multiple systems simultaneously. If dysfunctional pelvic muscles lead to inflammation and tenderness of the urogenital viscera, they may have a similar impact on the gastrointestinal system. With comorbidities such as irritable bowel syndrome being common in CUP patients, future research should examine common links and mechanisms. Specifically, studies should evaluate what impact the normalizing of pelvic muscle function may have on reversing comorbidities.

In terms of potential mechanisms, several changes within muscles arise in association with muscle over-activation, commonly referred to as "wind-up". These are discussed in more detail elsewhere.³⁴ However, there are two primary mechanisms recognised in literature. The first of these is ischemia, or reduced blood perfusion, with hypoxia, reduced

oxygen supply during increased demand. Ischemia is associated with muscle over-activation and leads to deep tissue pain of moderate to high intensity.³⁵⁻³⁷ Ischemic pain is most often described as "stabbing", "burning", "heavy" and "exhausting" pain and leads to lower pain thresholds consistent with peripheral sensitization.^{35, 37, 38}Given that peripheral sensitization can be reversed through muscle normalization, the mechanisms are clearly different to those of central sensitization which require different management strategies.^{39, 40} Ischemic pain characteristics appear to be totally consistent with symptoms of vulvodynia and BPS, making ischemia one of the likely mechanisms.^{21, 41} Impaired microcirculation due to vasoconstriction, tissue hypoxia and tissue acidosis have been previously suggested as a cause of chronic pelvic and urogenital pain.⁴²

The second mechanism of pain that arises from muscle over-activation is mediated by TrPs which can give rise to myofascial pain syndromes. 43, 44 Some estimate that 85% of chronic pain conditions are muscle mediated, giving rise to regional pain. 45 Pain from pelvic muscle TrPs is well documented but can go unrecognized unless the clinician is prepared to actively look for, and identify the source of pain, by palpating muscles and soft tissue that harbour these points of tenderness. 31, 46

In summary pain mapping provides a range of benefits, the most important being its systematic approach to localising and identifying the source of pain. The localising of pain within pelvic muscles and soft tissue of the urogenital area further disproves the hypothesis of the end-organ being the cause of CUP. Based on the outcomes of this study and the clinical experience of the authors, it is the dysfunctional pelvic muscles that act as the generators of pain, and the end-organs appear to be the "innocent bystanders".⁴⁷ The wrongful attribution of pain to organs arises on account of the similarities in myofascial and visceral pain characteristics.¹² It is only through a systematic assessment of the urogenital area, using a tool such as the IMAP, that the true origins of pain can be established.

CONCLUSION

Pain mapping removes some of the perplexity associated with CUP syndromes. The systematic approach of the IMAP enabled pain to be localised, and peripheral mechanisms to be identified. Pain mapping directly links dysfunctional pelvic muscles to CUP symptoms. The use of the IMAP provided pertinent information that may be central to the development of evidence-based interventions. With further validation, the IMAP can serve as an important tool in the assessment and management of CUP.

REFERENCES

- 1. Schott GD. The cartography of pain: The evolving contribution of pain maps. European Journal of Pain. 2010; 14(8): 784-91.
- 2. Jantos M, Johns S, Torres A, Baszak-Radomanska E. Mapping chronic urogenital pain in women: rationale for a muscle assessment protocol the IMAP Pelviperineology. 2015;34(1):?
- Dickinson RL. Studies of the levator ani muscle. Am J Dis Wom. 1889; 22:897-917.
- Delancey J. Functional anatomy of the pelvic floor and urinary continence mechanisms. In: Schussler B, Laycock J, Norton P, Laycock J, editors. Pelvic floor re-education: principles and practice. London: Springer-Verlag; 1994. p. 9-27.
- Kaufman R, Friedrich E, Gardner H. Non-neoplastic epithelial disorders of the vulvar skin and mucosa; miscellaneous vulvar disorders. Benign diseases of the vulva and vagina Chicago Yearbook, Chicago, IL. 1989: 299-360.

- Bachmann GA, Rosen R, Pinn VW. Vulvodynia: A state-ofthe-art consensus on definitions, diagnosis and management. J Reprod Medicine. 2006; 51:447-56.
- Messelink B, Benson T, Berghmans B, Bo K, Corcos J, Fowler C, et al. Standardization of terminology of pelvic floor muscle function and dysfunction: report from the pelvic floor clinical assessment group of the International Continence Society. Neurourology and urodynamics. 2005; 24(4):374.
- 8. Hanno PM, Burks DA, Clemens JQ, Dmochowski RR, Erickson D, FitzGerald MP, et al. Amerian urological association guideline: diganosis and treatment of interstitial cystitis/bladder pain syndrome: American Urological Association; 2011 [cited 2011 23rd March]. Available from: http://www.auanet.org/content/guidelines-and-quality-care/clinical-guidelines.cfm.
- Engeler D, Baranowski AP, Elneil S, Hughes J, Messelink E, Oliveira P, et al. Guidelines on chronic pelvic pain: EAU guidelines. Presented at: 27th European Association of Urology annual congress. 2012; Feb 24-28, 2012; Paris, France.
- Kavvadias T, Pelikan S, Roth P, Baessler K, Schuessler B. Pelvic floor muscle tenderness in asymptomatic, nulliparous women: topographical distribution and reliability of a visual analogue scale. Int Urogynecol J. 2013; 24(2):281-6.
- Peters KM, Carrico DJ, Ibrahim IA, Diokno AC. Characterization of a clinical cohort of 87 women with interstitial cystitis/painful bladder syndrome. Urology. 2008; 71(4):634-40.
- Brookoff D. Genitourinary pain syndromes: Interstitial cystitis, chronic prostatitis, pelvic floor dysfunction, and related disorders. In: Smith H, editor. Current Therapy in Pain. Philadelphia: Saunders Elsevier; 2009. p. 205-15.
- 13. Jantos M, Johns S. A new perspective on interstitial cystitis / painful bladder syndrome. International Society of Pelvic Perineology; October, 2011; Sydney, Australia 2011.
- Jantos M, Johns S. Clitorodynia: causes, comorbidities and pain mechanisms. Journal of Lower Genital Tract Disease. 2013; 17(6s):e85-e115
- 15. Melzack R. The McGill Pain Questionnaire: Major properties and scoring methods. Pain. 1975; 1(3):277-99.
- Giesecke, M.D. J, Reed BD, Haefner HK, Thorsten C, Daniel J, et al. Quantitative sensory testing in vulvodynia patients and increased peripheral pressure pain sensitivity. Obstetrics & Gynecology. 2004; 104(1):126-33.
 Östgaard H, Zetherström G, Roos-Hansson E. The posterior
- Ostgaard H, Zetherström G, Roos-Hansson E. The posterior pelvic pain provocation test in pregnant women. European Spine Journal. 1994; 3(5):258-60.
- Oyama IA, Rejba A, Lukban JC, Fletcher E, Kellogg-Spadt S, Holzberg AS, et al. Modified thiele massage as therapeutic intervention for female patients with interstitial cystitis and hightone pelvic floor dysfunction. Urology. 2004; 64(5):862-5.
- Haefner HK, Collins ME, Davies GC. The vulvodynia guidelines. Journal of Lower Tract Genital Disease. 2005; 9:40-51.
- Laycock J. Clinical evaluation of the pelvic floor. Pelvic Floor Re-education London, United Kingdom: Springer-Verlag. 1994; 42-8.
- 21. Jantos M. Vulvodynia: a psychophysiological profile based on electromyographic assessment. Applied Psychophysiology and Biofeedback. 2008; 33:29-38.
- Fitzgerald CM, Hynes CK. Female perineal/pelvic pain: the rehabilitation approach. In: Smith H, editor. Current Therapy in Pain. Philadelphia: Saunders Elsevier; 2009. p. 227-33.
- Ashton-Miller JA, Delancey J. Functional anatomy of the female pelvic floor. Annual New York Academy of Science. 2007; 1101:266-96.
- Jantos M, Burns N. Vulvodynia. Development of a psychosexual profile. Journal of Reproductive Medicine. 2007; 52(1):63-71
- 25. Harlow BL, Stewart EG. A population-based assessment of chronic unexplained vulvar pain: have we underestimated the prevalence of vulvodynia? Journal of the American Medical Women's Association. 2003; 58(2):82-8.
- Peters K, Girdler B, Carrico D, Ibrahim I, Diokno A. Painful bladder syndrome/interstitial cystitis and vulvodynia: a clinical correlation. Int Urogynecol J. 2008; 19(5):665-9.
- 27. Warren JW, Langenberg P, Greenberg P, Diggs C, Jacobs S, Wesselmann U. Sites of pain from interstitial cystitis/painful

- bladder syndrome. The Journal of urology. 2008; 180 (4): 1373-7.
- Khan BS, Tatro C, Parsons L, Willems JJ. Prevalence of interstitial cystitis in vulvodynia patients detected by bladder potassium sensitivity. Journal of Sexual Medicine. 2010; 7:996-1002.
- Seth A, Teichman JM. Differences in the clinical presentation of interstitial cystitis/painful bladder syndrome in patients with or without sexual abuse history. The Journal of urology. 2008; 180(5):2029-33.
- Nourmoussavi M, Bodmer-Roy S, Mui J, Mawji N, Williams C, Allaire C, et al. Bladder base tenderness in the etiology of deep dyspareunia. The Journal of Sexual Medicine. 2014; 11(12):3078-84.
- Weiss JM. Pelvic floor myofascial trigger points: manual therapy for interstitial cystitis and the urgency-frequency syndrome. Journal of Urology. 2001; 166:2226-31.
- Tu FF, Holt J, Gonzales J, Fitzgerald CM. Physical therapy evaluation of patients with chronic pelvic pain: a controlled study. American journal of obstetrics and gynecology. 2008; 198(3):272. e1-. e7.
- Fitzgerald MP, Payne CK, Lukacz CC. Randomized multicenter clinical trial of myofascial physical therapy in women with interstitial cystitis/painful bladder syndrome and pelvic floor tenderness. The Journal of Urology. 2012; 187:2113-8.
- Jantos M. Understanding chronic pelvic pain. Pelviperineology. 2007; 26:66-9.
- Mense S, Simons DG, Russell IJ. Muscle pain: understanding its nature, diagnosis, and treatment: Lippincott Williams & Wilkins; 2001.
- 36. Graven-Nielsen T, Jansson Y, Segerdahl M, Kristensen JD, Mense S, Arendt-Nielsen L, et al. Experimental pain by ischaemic contractions compared with pain by intramuscular infusions of adenosine and hypertonic saline. European Journal of pain. 2003; 7(1):93-102.
- 37. Coderre TJ, Xanthos DN, Francis L, Bennett GJ. Chronic post-ischemia pain (CPIP): a novel animal model of complex regional pain syndrome-Type I (CRPS-I; reflex sympathetic dystrophy) produced by prolonged hindpaw ischemia and reperfusion in the rat. Pain. 2004; 112(1):94-105.

- 38. Seo H-S, Kim H-W, Roh D-H, Yoon S-Y, Kwon Y-B, Han H-J, et al. A new rat model for thrombus-induced ischemic pain (TIIP); development of bilateral mechanical allodynia. Pain. 2008; 139(3):520-32.
- 39. Jantos M. Surface electromyography and myofascial therapy in the management of pelvic pain disorders. In: Santoro GA, Wieczorek P, Bartram C, editors. Pelvic Floor Disorders: Imaging and a Multidisciplinary Approach to Management. Milan: Springer Verlag Italia; 2010.
- Jantos M. Electromyographic assessment of female pelvic floor disorders. In: Criswell E, editor. Cram's introduction to surface electromyography. 2nd ed. Sudbury: Jones & Bartlett Publishers; 2010. p. 203-30.
- 41. Bergeron S, Binik YM, Khalifé S, Pagidas K, Glazer HI. Vulvar vestibulitis syndrome: reliability of diagnosis and evaluation of current diagnostic criteria. Obstetrics & Gynecology. 2001; 98(1):45-51.
- 42. Dwyer PL. Chronic pelvic pain in urogynecological practice: a personal view. Int Urogynecol J. 2011; 22(4):413-8.
- Simons DG, Travell JG, Simons LS. Travell & Simons' myofascial pain and dysfunction: upper half of body: Lippincott Williams & Wilkins; 1999.
- 44. Ge H-Y, Zhang Y, Boudreau S, Yue S-W, Arendt-Nielsen L. Induction of muscle cramps by nociceptive stimulation of latent myofascial trigger points. Experimental brain research. 2008; 187(4):623-9.
- Slomski AJ. How groups successfully manage pain patients. Medical Economics. 1996; 15:114-27.
- 46. Travell JG, Simons DG. Trigger point manual: lower half of body. Baltimore: Williams and Wilkins; 1992.
- 47. Peters KM. Reply to letter-to-the-editor: Prevalence of pelvic floor dysfunction in patients with interstitial cystitis. Urology. 2008; 71(6):1232.

Correspondence to:

Marek Jantos

L9 118 King William Street, Adelaide 5000 SA, Australia E-mail: marekjantos@gmail.com