Review article

Understanding Chronic Pelvic Pain

MAREK JANTOS

School of Psychology, University of Adelaide

Abstract: Chronic pain is a complex phenomenon characterized by an interaction of sensory and emotional variables. Effective patient care can be facilitated by an understanding of the differences between acute and chronic pain and by recognition of its origins. The lack of visible pathology to explain the severity of symptoms should not be the basis for seeking psychological explanations or questioning the reality of the patient's pain. The article examines some of the psychophysiological mechanisms evident in chronic pain syndromes especially when mediated by myofascial pelvic dysfunction.

Key words: Myofascial; Trigger point; Pelvic pain syndrome; Vulvodynia.

INTRODUCTION

The importance of pain management in patient care is reflected in the recognition of pain as the fifth vital sign. As a results pain needs to be assessed and charted together with temperature, respiration, pulse, and blood pressure whenever a patient undergoes medical review. However, unlike the other vital signs, pain is difficult to define in ways that provide the clinician with practical, workable options. The challenge with pain is that there is no localised centre in the body for its control nor is there a single intervention for its effective management. Although there are various diagnostic labels to identify the location of pain and describe its qualities, these have little practical value. Labels often disguise the fact that little is known about the cause of pain its mediating mechanisms and have little bearing on the treatment, which in most instances ends up being the same. For these reasons pain poses a complex set of difficulties and the complexity increases manifold in relation to chronic pain. This paper considers some of the clinical challenges in understanding and managing chronic pain and recommends a multidisciplinary approach which recognises the importance of psychophysiological variables.

DEFINITION OF PAIN

The International Association for the Study of Pain defines pain as an unpleasant sensory and emotional experience associated with actual or potential damage to tissue.[1] This succinct definition introduces one of the most important qualities of pain, namely that it has both a sensory and an emotional component. To the ancient Greeks, pain was an affective feeling state rather then a sensory experience. In the 20th century greater emphasis was placed on the sensory component of pain.[2] With technological advances and the use of imaging techniques to map brain areas associated with the experience of pain, knowledge has taken on a new level of complexity with a recognition that pain has both sensory and emotional components.[3, 4]

It may seem highly implausible to both the patient and practitioner that the interplay of sensory and emotional variables is relevant to post surgical pain or to chronic pain syndromes involving pelvic, perineal and urogenital regions. Yet, most of these complex pain syndromes can serve to illustrate the intricate interaction between physiological, psychological and behavioural variables. Urogenital pain conditions such as vulvodynia, a form of chronic vulvar discomfort,[5] can serve to illustrate this point. This condition is characterised by burning which can vary in severity but has a disabling effect on intimate sexual behaviour and compromises the quality of life of women.[6] Vulvodynia occurs in the absence of any clinically identifiable physical or neurologic findings. Biopsies taken from the vulvar vestibule of sufferers revealed unique physiological characteristics such as increased immunoreactivity, nociceptor sensitivity, and even increased density of superficial nerve endings.[7-10] These physiological markers appear to form a constitutional predisposition to this complex pain syndrome. Evidence points to psychological traits such as anxiety as modulating the severity of pain experienced.[11, 12] Tests utilizing quantitative sensory testing in female genital sensation consistently confirmed that differences in pain thresholds between patients and controls were mediated by anxiety.[11] From these findings it is evident that enhanced pain perception, greater emotional response and increased autonomic reactivity are closely related to measures of anxiety. Anxiety and cognitive schemas, such as catastrophizing (the tendency to focus on pain and to pessimistically assess one's coping ability) not only contribute to higher levels of pain, but account for up to 31% of the variance in pain ratings.[13] Clinically, anxiety and catastrophizing serve as reliable predictors of the severity of the patient's experience of pain and should be considered in clinical assessments.

Acknowledging the subtle interaction between physical and psychological variables has other important clinical implications; it enables the clinician to discuss with the patient the need for a multi-disciplinary approach to pain management it helps the patient to be more open to accepting psychological support and encourages compliance with the use of prescription medications, which often include psychotropic medications. On a cautionary note, however, assessments of chronic pain should avoid any dualistic concepts by which the clinician attempts to determine how much of the pain is physical and how much stems from emotional factors. Attributing proportionality or labelling the physical components of pain as "real" and the emotional as "unreal" is of no clinical value and may compromises the assessment and treatment of the patient.[14]

ACUTE AND CHRONIC PAIN

Differentiating between acute and chronic pain is important in understanding chronic pelvic pain syndromes. Acute pain is most common, often experienced by patients after surgery or other soft tissue traumas. It tends to be immediate, severe and short lived however, pain that extends beyond a normal recovery period and lasts longer then 3-6 months constitutes chronic pain.

Chronic pain is more difficult to understand and often exists where there is no visible pathology. Pain continues long after soft tissue damage has occurred and persists well beyond the time of healing. In simple terms, chronic pain occurs when there is little if any reason for it to exist. Yet the pain is real and can significantly affect the patient's quality

of life, limiting their daily physical activities and disrupting their ability to rest and sleep.

When acute pain enters the chronic phase, normal sensory processes are affected by progressive sensitization of the peripheral and central nervous system. Sensitization is an important property of nociceptors and manifests itself in:

- decreased thresholds to nociceptor stimulation
- increased field of nociceptor reception (progressing from localised to generalised)
- increased nociceptor responsiveness to normally non-noxious stimuli (allodynia)
- increased intensity of response (hyperalgesia)
- prolonged post-stimulus sensations (hyperpathia), and
- the occurrence of unexplained spontaneous pain.[15]

Such sensory changes are the defining characteristics of chronic pain syndromes and require management strategies that are different to those used in the management of acute pain.

When seeking medical help, chronic pain patients often hope that tests will uncover some form of pathology or produce sufficient evidence to explain their pain. With most chronic syndromes however pain is not proportional to pathology findings. In discussing chronic pelvic pain, Steege comments on this important chronic pain anomaly:

"I'm not aware of a single chronic clinical problem associated with pain in which pain is seen as proportional to tissue damage… most clinicians intuitively or by training look for enough pathology to explain the pain. With pain this proportionality simply does not exist…the intensity of pain is not consistently related (either directly or inversely) to the apparent degree of tissue damage".[14]

Patients often hope for positive pathology findings and inadvertently look for easy answers. It is essential to inform patients about the distinctions between acute and chronic pain.

Medical reviews of chronic pain syndromes can also be influenced by individual specialty bias. Each specialty may look for information which supports their preconceptions. In relation to pain, a surgeon will focus on structural issues, a neurologist may focus on neuropathic origins, a gynaecologist may be inclined to see it as secondary to endometriosis, while a psychologist may look for unresolved and repressed emotional issues as possible explanation of pain. Likewise, patients may come in for a consult with a set of assumptions and preconceived ideas as to what the cause of their pain might be and this will not only influence their selection of specialists but also their preference of treatment. Those who seek out the services of a surgeon may do so because of their own personal belief that surgery is the best option. The need to be objective in assessing chronic pain patients is essential.

IDENTIFYING THE CAUSE OF PELVIC PAIN

The most important question for the patient and the clinician is to identify the cause of pain? In general the three most common sources of pain include:

- *Somatic origin* - arising from skin, muscles and bone tissue. Patients describe this type of pain as a throbbing, stabbing or burning.
- *Visceral origin* - coming from internal organs. This type of pain tends to be diffuse and more generalised, with patients frequently describing it in more emotive terms.
- *Neuropathic origin* - arising from damaged nerve fibres. The pain is described as numbness, pins and needles and produces electric current-like sensations.[15]

Identifying the source of pain is not always reliable but can facilitate the patient's accounts of symptoms. Of the three sources of pain, myofascial Trigger Points (TrPs) are the most common.

MYOFASCIAL PAIN

Myofascial Pain Syndrome refers to regional pain of soft tissue origin. Studies estimate that in 75-95 per cent of cases, myofascial pain is a primary cause of regional pain.[16] Myofascial pain is associated with muscle tenderness that arises from TrPs, focal points of tenderness, a few millimetres in diameter, found at multiple sites in a muscle and the fascia of muscle tissue. Biopsy tests found that trigger points were hyperirritable and electrically active muscle spindles in general muscle tissue.[17-19]

TrPs are defined by several primary characteristics:[2]

- A TrP has a clear and consistent referred pain pattern. Pain from TrPs can be felt not only at the site of its origin but also in areas remote from it. Since the pain originating from a given muscle tends to exhibit a relatively consistent pattern of pain referral, it is often possible to identify the muscle from which the pain originates if the pattern of pain is clearly delineated
- TrPs can arise in response to acute and chronic overload, or repetitive overuse of the muscle in which it occurs. Such muscle overuse can arise from muscle wind-up following physical trauma or as result of sympathetically mediated tension (anxiety related bracing and guarding/splinting)
- TrPs contribute to motor dysfunction by causing increased muscle tension (the primary function of the muscle spindles is the regulation of tension in muscle tissues), spasm of neighbouring muscles, loss of coordination in affected muscles, substitution patterns in recruitment of muscles and a weakening of affected muscles
- TrPs cause weakness and limited range of motion. In most cases the patient is only aware of the pain but not of the other dysfunctional aspects of muscle function
- The intensity and extent of the pain depends on the degree of irritability of the TrPs and not on the size or location of the muscle
- TrPs disturb the proprioceptive, nociceptive and autonomic functions of the affected anatomical region.

Pain from TrPs can go unrecognized unless the clinician is prepared to actively look and identify the source by palpating the suspected muscles. Palpation of the tender spot always evokes discomfort and assists the patient to immediately recognize and identify "their" pain. This simple and reliable means of identifying the pain confirms in the patient mind that the pain is of muscular origin and not arising from treatment related complications such as infections or scar tissue. Such specificity of diagnosis reduces the anxiety of the patient and immediately provides options for treatment. In most instances myofascial pain will respond to stretching of the muscle, message of the area, injection of TrPs and management of perpetuating factors.[16] Pelvic musculature is structurally and functionally predisposed to developing Myofascial TrPs due to its work load supporting abdominal and pelvic viscera, maintaining posture and facilitating movement.

MYOFASCIAL PAIN IN PELVIPERINEOLOGY

The presence of TrPs in pelvic muscles has been well documented.[20] TrPs in specific muscles of the posterior half of the pelvic floor can be the source of poorly defined pain in the perineal region and discomfort in the anus, rectum, coccyx and sacrum and is commonly labelled as coccygodynia or levator ani syndrome. TrPs in muscles in the anterior half of the pelvic floor refer pain to genital structures (vagina, penis and scrotum). Active TrPs in these muscles can interfere with intercourse by causing entry dyspareunia and aching pain in the perineal region. Myofascial TrPs in the deeper pelvic muscles can effect bowel and bladder

function, contributing to urethral syndrome symptoms and clitoral pain.

Myofascial TrPs and dysfunctional pelvic muscles have been frequently linked to symptoms of interstitial cystitis, urgency and frequency, pelvic pain and dyspareunia.[21-23] Palpation of pelvic muscles in patients with chronic pain symptoms not only elicits discomfort but refers pain into the suprapubic, perineal regions, rectum and labia.[24] In all reports the TrPs appeared to be linked with hypertonicity of pelvic muscles and an inability of patients to relax and exercise adequate voluntary control.[21] In reducing pelvic floor hypertonicity and manually releasing myofascial trigger points, Weiss found an 83 per cent reduction in symptoms, including a reduction in neurogenic bladder inflammation.[22] Weiss concluded that a comprehensive treatment plan for patients with chronic interstitial cystitis and urgency-frequency syndrome can decrease central nervous system sensitization and their symptoms.

Hypertonic pelvic muscles and spasm leading to chronic pelvic and perineal pain can arise in response to a range of triggers, including deep somatic or visceral disease, distressed viscera or the trauma of surgery.[2] Triggers of chronic pain can be of an acute nature, but lead to progressive neuromuscular wind up with muscle tissue not only responding to nociceptive triggers, but progressively becoming the primary "initiator of nociception" and the site of chronic pain.[27] Acute triggers such as irritants, inflammation and trauma tend to provoke a reflex-mediated muscle response which eventually leads to chronic over-activation and pain.

A PSYCHOPHYSIOLOGICAL PERSPECTIVE ON CHRONIC PAIN

The absence of visible pathology in chronic pain syndromes should not form the basis for either seeking psychological explanations or questioning the reality of the patient's pain. Instead it is essential to approach the complexity of chronic pain from a psychophysiological perspective which recognises the importance of the mind-body interaction. Some of the mechanisms by which the limbic system impacts on pain, and in particular myofascial pain, have been clarified by research findings in neurology and psychophysiology.[17, 28, 29]

Muscle tissue contains nociceptive nerve endings (pain receptors) in two specific structures. The first of these is located in the adventitia of the vascular system and the second in small organs of the muscle known as muscle spindles. Nociceptors in both of these structures are highly sensitive to pressure and are responsible for the perception of pain.[19] The early work of an Italian research team led by Pastorre, highlighted the involvement of the sympathetic nervous system in the innervation of muscles and in particular of the muscle spindle.[30] The primary function of the muscle spindle is to regulate the action of the muscle by monitoring muscle velocity, length and work load, ensuring smooth function and preventing damage to muscle tissue. It is important to note that muscle tension is sympathetically maintained through the activity of intrafusal fibers to the muscle spindle.[17] Laboratory experiments have shown sympathetic stimulation can activate muscle spindles in even curarized animals (curare is a cholinergic blocker, preventing the voluntary recruitment of muscles through the activation of motor units), while intramuscular injections of phentolamine (a sympathetic antagonist) eliminates activity in the spindle.[18]

The chronic over-activation of intrafusal fibers within the muscle spindle appears to be the underlying mechanism in the aetiology of myofascial trigger points.[17, 19] It is also noteworthy that psychological distress significantly increases the activity of TrPs [17, 28] while physiological quieting through relaxation training lowers sympathetic activity within the trigger points. This has significant implications for therapeutic interventions, where the patient suffering from chronic pain syndrome will benefit most from a psychophysiological approach to management.

Various triggers can give rise to neuromuscular wind-up. Triggers such as soft tissue injury, surgical trauma, infections and visceral disease can potentially lead to progressive sensitization in the pelvic-perineal region. The emotional disposition of the patient and their coping mechanisms can, in turn, impact on the perception of pain. Various models have been proposed elucidating the mechanisms by which this can occur. With anxiety as the best predictor of pain thresholds, the psychophysiological model provides the most rational approach to the care of the chronic pain patient.

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Correspondence to:
MAREK JANTOS M.A.
School of Psychology, University of Adelaide
Adelaide, South Australia 5005
Ph: 61 8 8221 7070 - Fax: 61 8 8221 7071
E-mail: marek.jantos@adelaide.edu.au

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Set-up and statistical validation of a new scoring system for obstructed defaecation syndrome. *Altomare DF, Spazzafumo L, Rinaldi M, Dodi G, Ghiselli R, Piloni V. Colorectal Dis. 2007 Apr 18; epub.* A disease-specific index to quantify severity to allow assessment of the results of treatment in clinical trials was validated studying 76 patients with obstructed defaecation syndrome (ODS) and 30 healthy controls. The ODS score was the sum of all points with a maximum possible of 31 points. Agreement between two operators, coefficient of repeatability, internal consistency were all evaluated. There was a significant difference between the mean ODS score for patients and controls and cluster analysis on each clinical finding showed a different profile between cluster 1 (a nonhomogenous group including rectocoele, intussusception or perineal descent), and cluster 2 (pelvic dysynergia). The ODS score offers a validated severity of disease index in grading the severity of disease and monitoring the efficacy of therapy.

6 – INCONTINENCES

Burch colposuspension versus fascial sling to reduce urinary stress incontinence. *Albo ME, Richter HE, Brubaker L et al.N Engl J Med. 2007;356:2143-55.* Among many procedures available for urinary stress incontinence, few randomized clinical trials provide a basis for treatment recommendations. This multicenter randomized trial compares the pubovaginal sling with autologous rectus fascia (n 326) and the Burch colposuspension (n 329) in women with positive stress test and urethral hypermobility, the primary outcomes being negative pad test, no urinary incontinence in a 3-day diary, negative cough and Valsalva stress test. At 24 months success rates were higher for women who underwent the sling procedure, however they had more urinary tract infections, difficulty voiding, and postoperative urge incontinence.

Current and future trends in the management of overactive bladder. *Wagg A, Majumdar A, Toozs-Hobson P et al. Int Urogynecol J Pelvic Floor Dysfunct. 2007;18:81-94.* Overactive bladder (frequency-urgency syndrome) is the commonest bladder problem in late life, affecting up to 41% of over-75-year-old individuals, and the elderly experience more severe disease. The current state and future developments in pharmacological therapy are outlined.

Surgical treatment of stress urinary incontinence using the tension-free vaginal tape-obturator system (TVT-O) technique. *Jakimiuk AJ, Maciejewski T, Fritz A et al. Eur J Obstet Gynecol Reprod Biol. 2007 Apr 25;epub.* TVT-O surgery was performed in 35 patients followed up for 12 months: total cure was achieved in 42.8%, significant improvement in 17.1%, SUI symptoms abated in 11.4%, no improvement in 20%, and QoL deteriorated in 8.7%. Additional patients should be analysed for a longer period of time.

Re: Reevaluating occult stress incontinence. *Twiss C, Triaca V, Raz S. Eur Urol. 2007;51:850-1. Gastroenterol Clin North Am. 2007;36:145-59.*

The relationships among measures of incontinence severity in women undergoing surgery for stress urinary incontinence. *Albo M, Wruck L, Baker J, Brubaker L et al. J Urol. 2007;177:1810-4.* Urinary incontinence severity measures correlate moderately with each other at best. While medical, epidemiological and social aspects of aging demonstrated stronger correlations with the other measures of severity and quality of life, Valsalva leak point pressure did not. Supine empty bladder stress test did not demonstrate a clinically significant association among severity measures.

The effect of surgery on quality of life in patients with faecal incontinence of obstetric origin. *Pla-Marti V, Moro-Valdezate D, Alos-Company R et al. Colorectal Dis. 2007;9:90-5.* Surgical treatment of faecal incontinence of obstetric origin achieves good results in a high percentage of patients and has a positive effect on their quality of life. The existence of prolonged preoperative pudendal nerve motor latency indicates a poor prognosis.

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