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## Published Article

### Chronic Pelvic Pain and Myofascial Trigger Points

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#### INTRODUCTION

The additive effect of tense pelvic-floor holding patterns, trauma, inflammation, or pelvic organ disease can overload the muscles, stimulating the development of myofascial trigger points and pelvic floor hypertonus. The increased tenderness and tension in these muscles may refer pain into the lower back, abdomen, or perineum, or it may cause urethral, vaginal, or anal symptoms by compression. The noxious stimuli created by this self-perpetuating process can alter the central nervous system in a manner that magnifies and spreads the symptoms.

A stimulus-free period has been shown to reverse central sensitization, and thus all noxious input entering the relevant sacral spinal cord region must be eradicated. Therefore, a comprehensive approach must be followed, with attention paid to the skin, viscera, myofascial structures, the stressed or depressed mind, hormonal imbalances, poor nutrition, and sleep disturbances.

#### CASE REPORT

Ms. M., a 38-year-old woman, gave a 14-year history of right lower quadrant pain, urinary urgency, frequency, suprapubic pressure, and deep right-sided dyspareunia. These symptoms had arisen after a Caesarean section that followed two days of traumatic labor. Her pelvic pain symptoms had been treated elsewhere with nine laparoscopic procedures for lysis of adhesions, a right oophorectomy, and total abdominal hysterectomy. When she presented to our clinic complaining of continued pain, external pelvic examination revealed myofascial trigger points of the right rectus abdominus muscles and a tender suprapubic scar. Internal pelvic examination disclosed myofascial trigger points of the right obturator internus, pubourethralis and urinary sphincter. She was treated with internal myofascial release techniques, which eradicated the trigger points and hypertonus of the pelvic floor, and trigger point injections of 0.5% bupivacaine into the rectus abdominus trigger points and scar.

She became completely asymptomatic after 12 treatments.

#### DISCUSSION

As in the case of Ms. M., dysfunctional muscles of the pelvic floor are a frequently overlooked contributor to chronic pelvic pain. For example, it is not unusual to see regional pain appear to metastasize: what begins as lower abdominal pain progresses to urethral pain with urinary urgency and frequency, vulvar pain, anal pain and constipation, lower and upper back and neck pain, headache, anxiety, stress and fatigue. The classical analytic process used in medical diagnosis would attempt to implicate one source for these symptoms, not a dozen. The common denominator may be a myofascial trigger point, described as "a hyper-irritable spot, usually within a taut band of skeletal muscle or in the muscle fascia, that is painful on compression and that can give rise to characteristic referred pain, tenderness and autonomic phenomena."<sup>1</sup>

A myofascial trigger point is the end result of muscle injury at the motor end plate by overloading, whether acute, sustained or repetitive.<sup>1</sup> The tender trigger point can then refer pain along that muscle or to surrounding and distant muscles (see Figure), set off autonomic nervous system symptoms in the reference zone, weaken the muscles so that they cannot accomplish a full range of motion, and increase their sensitivity. The affected muscle and fascia contract, establishing a shortened position and causing surrounding muscle groups to compensate. These in turn become so overloaded that they too develop trigger points,<sup>1</sup> thereby spreading the symptoms.

The increased vulnerability of the pelvic floor muscles can be attributed to their anatomically central location, which transmits forces between the upper and lower body. Their constant functional activity (supportive, sphincteric and sexual) and their eccentric or elongating contractions are additional factors making these muscles a major target of stress.

To visualize how the pelvic floor responds to stress, one need only look at the movement of a dog's tail: when the dog is happy, the tail wags loosely from side to side; when the dog is stressed, the tail is pulled tightly under its legs. It is the pelvic floor muscles that control the tail. They were also the tail-waggers in man before the evolutionary loss of the tail and the assumption of the upright position made them supporting muscles. In fact, the pelvic floor muscles are still attached to the rudimentary tail, the coccyx, which is pulled forward when contracted, thereby compressing its penetrating organs. Therefore, man's pelvic muscles, as the dog's, may be the ultimate representation of the mind/body connection, for they are constantly responding to fluctuations in feeling. As a result, they may become overloaded and develop self-sustaining dysfunctional patterns.

The most common events that lead to injury are:

1. chronic tense holding patterns that develop in childhood as a result of sexual abuse, traumatic toilet training, abnormal bowel patterns, guilt surrounding sexual feelings, dance training or stress;
2. repetitive minor trauma or straining with constipation or urinary obstruction;

3. sudden brief severe strain sustained in sports, dance or gymnastic accidents;
4. direct physical trauma from bicycling, childbirth, urologic or gynecologic instrumentation or surgery;
5. inflammation of pelvic organs such as prostatitis, cystitis, urethritis, endometriosis, vaginitis, proctitis or anal fissures;
6. referred pain from other attaching muscle groups or viscera or nerves.

The intensity of the force required to create injury depends on the baseline muscle integrity. Common predisposing biomechanical problems that create pelvic muscle imbalance are skeletal deformities (scoliosis, articular dysfunction of the back, hips or SI joint) or hormonal (hypothyroidism, estrogen deficiency), nutritional (iron, vitamins B and C) or genetic deficits.

Depending on the severity of the myofascial injury, a trigger point can be latent (asymptomatic) or active (symptomatic). The development of a symptomatic trigger point can cause diagnostic confusion, as the traumatic events leading to it may be additive. With the final trauma, the muscle exceeds its stress tolerance.<sup>2</sup> Sometimes this triggering event is so minor that its contribution is not even recognized, as in the case of Ms. M.

#### **DIAGNOSTIC CONSIDERATIONS**

Myofascial trigger points create chronic pelvic pain or mimic visceral disease by three mechanisms: local tension around the penetrating organs and muscle referral patterns; viscerosomatic and somatovisceral reflexes; and central sensitization.

##### Local Effect

Unlike other striated, voluntary muscles, those of the pelvic floor surround and are intimately attached to visceral structures (urethra, bladder neck, vagina and rectum) for support and sphincteric control. Because the afferent nerves of viscera and deep muscles (i.e., those of the pelvic floor) go to the medial thalamus, they cannot localize noxious stimuli as well as nerves from the skin, which go from the lateral thalamus to the somatosensory cortex. Therefore, patients with active myofascial trigger points and spasm of the pelvic floor may not perceive their symptoms as originating in the pelvic musculature. Not only can they have urinary urgency and frequency, vaginal or anal pain, referred pain to the low back, suprapubic or perineal areas, but—because of neural pathways to the limbic centers via the medial thalamus—they can also experience varying degrees of emotional distress.

##### Viscerosomatic and Somatovisceral Reflexes

The effect of visceral pain on somatic structures was shown by Vecchiet et al.<sup>3</sup> and Giamberardino et al.,<sup>4</sup> who found that 30 to 64% of patients who had had repeated episodes of renal colic experienced hyperalgesia in the lumbar muscles years after the original pain. The latter group postulated that the stone pain triggered plastic neuronal changes at the spinal or supraspinal levels that were sustained after visceral input stopped, although they did not rule out tissue alterations in the referred zone. One of the tests, striking the muscle with the ulnar portion of the hand until contact with the tender point prompted the patient to jump, can be compared to the jump sign in the diagnosis of myofascial trigger points. Their study may actually have found myofascial trigger points created by visceral pain via the viscerosomatic reflex.

This model of repeated episodes of renal colic causing chronic muscular flank pain can be applied to recurrent endometriosis or other pelvic organ inflammation causing chronic pelvic myofascial pain. Wesselmann and Lai<sup>5</sup> demonstrated the viscerosomatic referral pattern from a gynecologic organ by experimentally inducing uterine inflammation in the rat. Using Evans blue as a plasma marker, they found that neurogenic inflammation was produced in the trunk, perineum, bilateral thighs, saddle area and proximal tail.

The pain distribution from this experimental uterine inflammation is similar to that seen clinically in women. In addition, once myofascial structures become physically involved in the pain process, they can develop myofascial trigger points that maintain pain, in this case even after the visceral inflammation has subsided.<sup>1</sup> This may be the mechanism underlying gynecologic surgical failure, as in the case of Ms M: in actuality the pain is related to pelvic myofascial trigger points, not visceral disease.

##### Central Sensitization

Myofascial trigger points not only can be a source of pain, but also can sensitize CNS neurons and thereby lead to the much deeper and treatment-resistant neuropathic pain. A review of basic pain neurophysiology indicates that an afferent nerve branches in the spinal cord to synapse with many dorsal horn cells in many spinal cord segments, above, below,<sup>6</sup> or contralaterally.<sup>7</sup> In addition, nerves from multiple organs (muscle, viscera and skin) can converge on a single dorsal horn neuron and affect each other in the supraspinal levels as the second-order neurons travel in close proximity in the brain stem and thalamus on the way to the cerebral cortex.

The obvious importance of this intermingling is to enable the organs to communicate for normal and coordinated body functions. However, since the dorsal horn neurons and thalamic cells are the heart of the communication system, any damage to them can cause widespread dysfunction. Their normal activity is most commonly disrupted by a severe or chronic painful stimulus from the peripheral nerves that synapse with them. This noxious input can have many causes, e.g. active myofascial trigger points, visceral pain, or skin inflammation. When these noxious impulses reach the dorsal horn cells in the spinal cord via the C-fibers, neuropeptides are released, initiating physical, chemical and genetic changes that activate low-efficiency synapses or neurons with a wide dynamic range and facilitate abnormal connections. The manner in which pain is processed is thus altered. This increased excitability of the spinal cord, or altered central processing, is termed neuroplasticity to signify a change that lasts longer than the triggering event.<sup>8</sup>

These plastic changes can variously alter the sensation of pain: the threshold may be lowered, whereby a non-painful stimulus will cause pain (allodynia); pain may be spontaneous or intensified (hyperalgesia) or its field may be expanded; or the degree and duration of pain may be enhanced with each repeated painful or non-painful

stimulus (windup).<sup>8</sup> These are all of the pain qualities we begin to observe as the CNS becomes unstable and the symptoms escalate—not from worsening of the disease, but rather from its chronicity.

In addition to these generalized phenomena, symptoms specific to the spinal cord segment are involved. Dorsal horn or thalamic cells sensitized by myofascial pain can cause sensitivity in other organs that converge on them, and painful viscera (e.g., kidney,<sup>3,4</sup> and uterus<sup>5</sup>) can cause sensitivity in myofascial structures in their reference zone. What begins as myofascial, visceral or skin pain can, with enough noxious stimuli, become neuropathic pain, affecting a wider area and more organs.<sup>8</sup>

If the dorsal horn cells remain in this sensitized state, the original pain can be reignited by a noxious stimulus that reaches them from any of the converging organs in their shared receptive fields. The studies of Vecchiet and Giamberardino et al.<sup>3,4</sup> indicated that the renal colic left the thoracic dorsal horn cells sensitized after its subsidence, and therefore pain anywhere within that thoracic receptive field could reproduce the original colic. The degree of sensitivity in these dorsal horn cells can be influenced by a multitude of changing factors, e.g. depression, hormonal fluctuations during the menstrual cycle,<sup>9</sup> sleep disturbance, or diet. Therefore, the pain experience may fluctuate may be that a summation of these factors, rather than a single factor, is necessary to exceed the threshold for the perception of pain.

### TREATMENT

From a practical standpoint, the question arises: How can these altered or sensitized neurons become normal again? We have methods to eradicate myofascial trigger points, but how do we treat sensitized nerves?

Gracely et al.<sup>10</sup> suggest that altered central processing cannot be sustained without ongoing input from a painful focus. In their study, when an anesthetic block was administered to a painful point near the elbow, the chronic pain in the distal arm and hand subsided. Koltzenburg et al.<sup>11</sup> have stated that "central sensitization cannot be perpetuated by central processes alone." They suggest that, when nociceptor activity is blocked or reduced below a critical level, the central processing mechanism quickly reverts to normal. Cohen<sup>12</sup> has also indicated that ongoing remote but related nociceptors can maintain neuropathic pain. In a study with Arryo,<sup>13</sup> their patient's knee pain was not significantly helped until the ipsilateral dysfunctional SI joint was treated.

The success of blocking painful input to the spinal cord to allow altered central processing to return to normal is described by Bach et al.,<sup>14</sup> who reported that pain memories in phantom limbs appeared less common when an anesthetic block created a pain-free interval between the onset of pain and the amputation. Bonica<sup>15</sup> found that closely spaced anesthetic blocks yielded pain relief of progressively greater duration and magnitude. This indicates that the block allowed the sensitized nerves a stimulation-free period in which to recover.

Therefore, effective therapy must be widespread and comprehensive to identify and correct any ongoing painful input that originates anywhere in the receptive fields. Therapy must be directed at eradicating all noxious stimuli transmitted to the sacral spinal cord from, for example, the skin, viscera, myofascial trigger points, or abnormal body mechanics, to allow a stimulation-free period. General factors should also be treated, as these disrupt normal pain modulation: e.g., hormonal and nutritional abnormalities and sleep disturbances.

Chronic stress, a contributor to neural sensitivity and increased symptoms, must also be addressed.

Psychotherapy can play an important role in identifying old traumas that sustain muscle hypertonus. In addition to deep-seated psychological problems, day-to-day stress can create or increase muscle tension and decrease the pain threshold. Therefore, the daily practice of a stress-reduction technique is essential to lower the overall muscle tension and keep it below a symptomatic level.

The failure to use a broad therapeutic approach to search out and eradicate all incoming noxious stimuli that maintain nerve sensitization will result in continued or recurrent pain. Therefore, it follows that the successful treatment approach must be holistic and comprehensive.

### REFERENCES

1. Travell J, Simons, D. Myofascial pain and dysfunction: The trigger point manual, Vol 1, Baltimore: Williams & Wilkins, 1992.
2. Sola AE, Bonica JJ. Myofascial pain syndrome, in Bonica JJ. The management of pain, Vol 1, 2nd ed, Philadelphia: Lea and Febiger, Chap 21, 1990, p. 354.
3. Vecchiet L, Giamberardino MA, Dragani L, Albe-Fessard P. Pain from renal/urethral calculosis: Evaluation of sensory thresholds in the lumbar area. *Pain* 1989;36:289-295.
4. Giamberardino MA, Bigontina P, DeMortegiani C, Vecchiet L. Effects of extracorporeal shock-wave lithotripsy on referred hyperalgesia from renal/urethral calculosis. *Pain* 1994;56:77-83.
5. Wesselmann V, Lai J. Mechanisms of referred visceral pain: Uterine inflammation in the adult virgin rat results in neurogenic plasma extravasation in the skin. *Pain* 1997;73:309-317.
6. Field HL. *Pain*, New York: McGraw-Hill, 1987, pp. 42-44.
7. Linderotin B, Brodin E. Mirror pain and indications of bilateral dorsal horn activation in response to unilateral nociception. *Pain* 1994;58:277.
8. Coderre TJ, Katz J, Vaccarino AL, Melzack R. Contribution of central neuroplasticity to pathological pain: Review of clinical and experimental evidence. *Pain* 1993;52:259-285.
9. Giamberardino MA, Berkley KJ, Iezzi S. Pain threshold variations in somatic wall tissues as a function of menstrual cycle, segmental site and tissue depth in non-dysmenorrhic women, dysmenorrhic women, and men. *Pain* 1997;71:187-197.
10. Gracely RH, Lynch SA, Bennett GJ. Painful neuropathy: Altered central processing maintained dynamically by peripheral input. *Pain* 1992;51:175-194.
11. Koltzenburg MK, Torebjork HE, Wahren LK. Nociceptor modulated central sensitization causes mechanical hyperalgesia in acute chrogenic and chronic neuropathic pain. *Brain* 1994;117:579-591.
12. Cohen ML. Comment on Gracely et al. Painful neuropathy: Altered central processing maintained dynamically by peripheral input. *Pain* 1992;51:194.

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## What is the cause?

### **What causes Pelvic Pain and Organ Dysfunction?**

The sensation and the need to urinate frequently and urgently, pain in the vagina, bladder, anus or pelvis may be caused by pelvic floor muscle spasm. Since this muscle tightness may not always be felt, it is only its effect on body structures that captures our attention.

As the major pelvic organs, the urethra, vagina and rectum, pass through these tight muscles of the pelvic floor, they may respond by becoming painful and irritable. The attachment of muscles to the pubic bone in front and the tailbone behind, can lead to a variety of pelvic pain patterns. Skin or bladder lining inflammation may result when irritating substances are released by the nerves as they are stimulated or compressed by these spastic muscles.

The underlying cause of the muscle spasm can usually be attributed to the development of hypersensitive trigger points or tender knots, which occur when the pelvic muscles become repeatedly strained or overloaded.

Emotional or physical stress, chronic straining, recurrent bladder infections, surgery, childbirth and muscle holding patterns developed during childhood are a few of the events that can cause the formation of trigger points and the resultant muscle tension.

A particularly deceptive feature of trigger points is that their presence may not be known until a series of muscle stresses exceeds the limit of trauma for that muscle. A spine or hip problem, poor posture or abnormal holding patterns may weaken the pelvic floor muscles. At a much later time, bladder or vaginal infections, trauma or surgery may add to the already stressed muscle and cause acute pain or abnormal functioning. In other words, a trigger point may become active as a result of a seemingly inconsequential event.