

Chronic Pelvic Pain: The Myofascial Component

Jennifer Gunter, MD

Chronic pelvic pain (CPP)—ie, pelvic pain of at least 6 months' duration—is a complex pain syndrome with a prevalence of 3.8% among women who present in a primary care setting.¹ Population surveys yield a much higher prevalence, with 14% to 24% of women reporting CPP; however, many women do not seek medical attention for this condition, and many of those who do never receive an actual diagnosis.¹⁻⁵ Chronic pelvic pain is estimated to account for 10% of referrals to gynecologists,⁶ so the gynecologist must have a thorough understanding of the complex interactions that may be involved in the pathogenesis of this enigmatic pain syndrome.

Chronic pelvic pain is seldom the result of pathology in a single organ; it most often involves a constellation of disease processes, and is therefore best described as a syndrome. Any structure in the pelvis may be involved in pelvic pain, and while gynecologists may be most familiar with visceral sources such as the reproductive, genitourinary, and gastrointestinal tracts, somatic sources such as the muscles, fascia, and ligaments are frequently overlooked as causes of persistent pain.^{7,8} Myofascial pain syndrome (MPS) is a musculoskeletal disorder characterized by spasm or hyperirritability, producing local and referred pain. It is estimated that up to 85% of all patients with chronic pain have MPS, and so it is a reasonable assumption that this high-tone muscle dysfunction may affect the pelvic floor muscles and contribute to CPP.^{9,10} While the incidence is unknown, pelvic floor MPS is believed to be a significant contributor to CPP for many patients, and failure to identify and treat this myofascial component may explain the high failure rate of various treatments for many women.^{7,8,11-13}

PATHOPHYSIOLOGY

Myofascial pain syndrome is characterized by tender, taut bands of contracted muscle fibers with associated

trigger points; manual stimulation of these trigger points reproduces the pain complaint, and is often associated with a "twitch response"—ie, a palpable or visual contraction of the taut band.^{9,14-16} Affected muscles have a decreased range of motion, are weaker, fatigue early, and may have associated autonomic phenomenon such as abnormal sweating, vasodilatation, and pilomotor activity (Table 1).^{9,14,16} Myofascial pain syndrome develops when sustained muscle-fiber contraction produces ischemia, resulting in a local "energy crisis" and release of neurotransmitters that sensitize sensory and autonomic nerves to produce extreme tenderness of the affected muscle.^{9,14,17} The chronic release of neuroactive substances and continued nociceptive input to the spinal cord produces central sensitization, essentially a remodeling of the central processing of pain signals.^{12,14} This phenomenon produces exaggerated pain responses to stimuli and converts an acute problem to a chronic one; a self-sustaining cycle of muscle spasm and pain ensues (Figure 1).¹⁶ Central sensitization is also responsible for referred pain, as efferent neurons that share affected spinal segments are also modified by these central changes.^{9,12,14,18} Perceived pain in MPS is most often a combination of both local and referred pain.

The pelvic floor is comprised of the endopelvic fascia,

TABLE 1: Symptoms Associated With Pelvic Floor Myofascial Pain

- Sleep disturbance
- Relationship to anxiety/stress
- Exacerbated with physical activity
- Exacerbated with prolonged sitting
- Increasing pain throughout the day
- Temporary improvement with short rests
- Dyspareunia
- Urinary hesitancy, urgency, frequency
- Pain with evacuation of bowels, constipation

Jennifer Gunter, MD, is assistant professor, Department of Obstetrics and Gynecology, University of Colorado Health Sciences Center, Denver.

Chronic Pelvic Pain

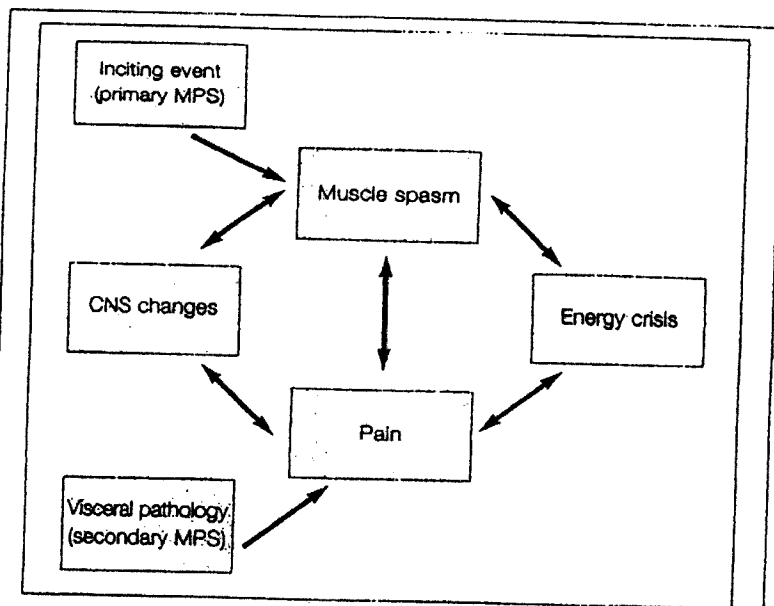


FIGURE 1. Self-sustaining pelvic myofascial pain cycle.

MPS = myofascial pain syndrome; CNS = central nervous system.

the levator ani (pubococcygeus, puborectalis, and iliococcygeus), the coccygeus, the obturator internus, and the piriformis muscles.^{11,13,19,20} The pelvic floor muscles are the only skeletal muscles with resting electric activity, and they provide a constant baseline tone that supports the pelvic viscera, maintains continence, and contributes to lumbopelvic stability.^{19,20} In addition, they contract to maintain closure of the pelvic floor with increasing abdominal pressure, and relax to allow micturition and defecation. These constant, complex, coordinated interactions result in chronic, repetitive muscle overload that may increase the vulnerability of the pelvic floor to MPS. In addition, hormones such as estrogen, progesterone, and relaxin may be involved in musculoskeletal pain through effects on muscle fibers, nociceptors, neuroinflammation, or some other mechanism.²¹⁻²³ Myofascial pain syndromes are more common among women compared with men, a predominance that starts with puberty and begins to decrease with menopause, and so it is reasonable to assume some relationship with sex hormones.^{21,24}

Risk factors specific to MPS of the pelvic floor include muscle trauma, either surgical or postdelivery; obesity, which increases the load on these muscles; and poor posture and a sedentary life-style, producing changes in muscle length, strength, and tone.²⁵ Daily vigorous physical activity may have a

protective effect against regional MPS; therefore, overall muscle conditioning may also affect risk.^{15,26}

Pelvic floor MPS may be a primary disorder in women with CPP, or may develop secondary to other pathology. The muscles of the pelvic floor are innervated by spinal nerves from L5 to S5, sharing spinal segments with the nerves that innervate the bladder, uterus, vagina, medial aspects of the fallopian tubes, and rectum; pain from pelvic visceral pathology may then be referred to the pelvic floor.^{18,27,28} The large number of visceral and somatic afferent and efferent nerve fibers in the pelvis converging over a relatively small number of spinal segments increase the likelihood that referred phenomena will occur between visceral and somatic structures. Gynecologists may be most familiar with acute manifestations of this viscerosomatic phenomenon, such as flank pain from ureterolithiasis or shoulder pain from diaphragmatic irritation. Depending on the underlying visceral pathology and the

resulting changes in the CNS, referred myofascial pain may outlast the underlying visceral cause and become a self-sustaining, secondary pain disorder (Figure 1).

EVALUATION

The history almost always yields clues that point to pelvic floor MPS among women with CPP (Table 1). The pain is usually localized to the lower quadrants of the abdomen, and is often described as dull, aching, cramping, or stabbing. The quality of musculoskeletal and visceral pain may be difficult to distinguish by history. The location of the pain as described by the patient is extremely important, as specific referral patterns have been identified for pelvic floor muscle pain (Figure 2). The muscles of the pelvic floor may refer pain to the flanks, suprapubic area, anococcygeal region, vagina, rectum, buttock, and posterior thigh.^{11,29,30} The diagnosis of pelvic floor MPS depends on the symptoms and characteristic findings on physical examination, and the suggested clinical criteria for the diagnosis of MPS can be adapted for the pelvic floor (Table 2).⁹

Myofascial pain is often severe, as muscles are rich in nociceptors and may be unresponsive to analgesics—including narcotics. Patients may report visits to an emergency department for exacerbations of pain during which they received intravenous narcotics with lit-

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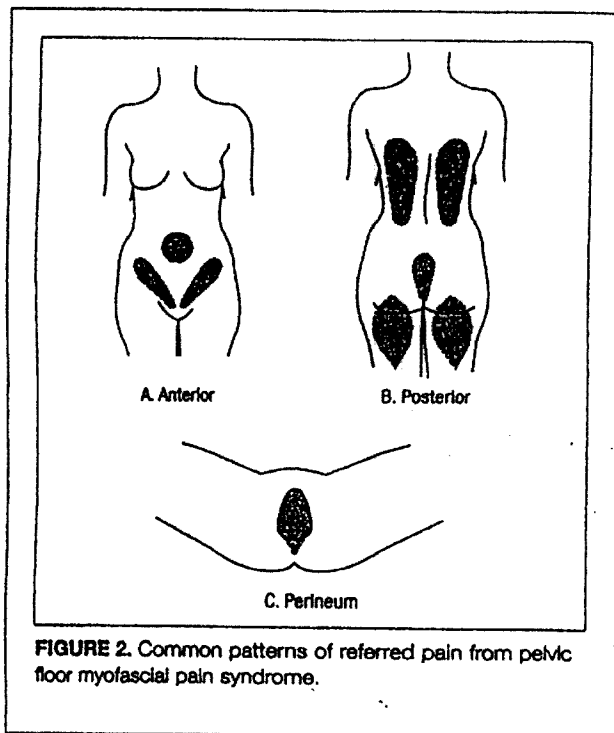


FIGURE 2. Common patterns of referred pain from pelvic floor myofascial pain syndrome.

tle or no relief. Musculoskeletal pain may interfere with sleep if the patient is unable to find a comfortable position, and patients may awaken with pain if movement during sleep increases strain on the affected muscles.^{16,29} The pain is often exacerbated by specific movements or by physical activity in general, and frequently increases throughout the day with the continuing visceral load on the pelvic floor. It may improve temporarily with short periods of rest or reclining. The presence of cyclic pain does not exclude a myofascial component, and it is important to distinguish severe dysmenorrhea—which may be associated with endometriosis or adenomyosis—from cyclic pain. Cyclic pain occurs with many pain syndromes, the classic example being menstrual migraines, and does not imply that endometriosis is a cause of the pain syndrome. Muscle hyperalgesia appears to be greatest perimenstrually, and dysmenorrhea appears to further lower the threshold for myofascial pain.³¹

The onset and duration of the pain should be recorded, and the patient should be asked specifically if there was a point in time when she was pain-free. Some patients may be able to associate the onset with a specific event or time period, while others may report a gradual onset. The chronology of the pain in relation to menarche, menopause, injury, surgery, or trauma to

the pelvis (including childbirth) should be recorded. A history of work and physical life-style habits may also be useful. Prolonged sitting, chronic abnormal posture, and/or a sedentary life-style produce changes in muscle length and tone, potentially contributing to MPS of the pelvic floor.^{16,29,30} The effect of emotions and stress on pain should also be noted, as stress and anxiety affect muscle tone, exacerbating pain.

Specific attention should be paid to a voiding history. Symptoms such as urinary frequency, urgency, nocturia, and increased pain with a full bladder are often reported. The short, contracted levator ani may limit the ability to inhibit the detrusor during bladder filling, and contractions of the pelvic floor to suppress urge symptoms may further increase pain.³⁰ Patients may also report urinary hesitancy, reflecting an inability to relax the high tone of the pelvic floor during micturition. A voiding diary and a questionnaire such as the pelvic pain and urgency/frequency patient symptom scale may help identify patients who may also have interstitial cystitis (IC).³² Interstitial cystitis is frequently associated with high-tone pelvic floor dysfunction and may affect both treatment options and prognosis. Difficulty defecating or pain with or immediately after defecation may also occur from the complex sequencing of muscles involved in evacuating the bowels; some patients may even develop anismus (paradoxical contraction of the pelvic floor during defecation). Deep dyspareunia and a history of painful pelvic examinations are common, as the affected muscles of the pelvic floor are exquisitely tender on palpation. Insertional dyspareunia may also be present, as central changes may affect efferent neurons to the vulva, producing vulvodynia.

Physical examination should include height, weight,

TABLE 2. Suggested Clinical Criteria for the Diagnosis of Pelvic Floor Myofascial Pain

- Pelvic pain
- Referred pain (distinct pattern)
- Exquisite local tenderness of pelvic floor muscles on palpation
- Presence of taut palpable band on pelvic examination
- Limited range of motion of pelvic floor muscles as evidenced by one of the following:
 - Urinary urgency and frequency
 - Urinary hesitancy
 - Inability to isolate and contract pelvic floor muscles on pelvic exam

Chronic Pelvic Pain

and calculation of the body mass index. Obesity not only increases the load on the pelvic floor, but it also exaggerates lumbar lordosis and the anterior pelvic tilt, further increasing muscular strain. Posture—both sitting and standing—should be noted, as many women with CPP have an abnormal posture; on standing, the back should be evaluated for a lumbar lordotic curve, kyphosis, and scoliosis. The iliac crest heights should be evaluated, with a difference of more than 0.5 cm considered significant.^{25,29,30} While the patient is standing, any evidence of unilateral standing (asymmetric weight-bearing, or the tendency to stand with one knee flexed or one leg externally rotated) should also be noted.²⁹ The patient should then be asked to bend at the waist, and on flexion, the lordosis should straighten and become slightly convex.²⁹ If any asymmetry or abnormalities are identified on this cursory musculoskeletal examination, referral to an experienced physical therapist for further assessment is indicated.

The muscles of the back and abdomen should also be evaluated for the typical referral patterns from the pelvic floor (Figure 2) and for trigger points. Trigger points are identified by the palpation of a painful, taut, or hard band within the muscle to produce a twitch response or contraction on plucking with a finger.^{9,14-17} The abdominal examination may reveal trigger points, especially in the lower abdomen at the insertion of the rectus abdominis and oblique muscles. The same muscle deconditioning that produces MPS of the pelvic floor may lead to changes in the anterior abdominal wall, or alternatively, these trigger points may be referred from the pelvic floor or visceral pathology.³³ The relation of the pain to any surgical scars should also be noted.

Prior to the internal pelvic examination, the vulva should be inspected for lesions or scars and evaluated with a cotton swab for areas of tenderness or pain, as vulvodynia frequently coexists with CPP. A neurologic examination should be performed to identify any areas of anesthesia or paresthesia. The anal-wink and bulbocavernosus-reflex tests should be performed to assess the sacral reflexes. The internal pelvic examination should first be performed with a single finger to look for focal pain on palpation, muscle spasm, and discrete trigger points. The base of the bladder should be palpated first, then the pubovaginal fascia on either side of the urethra. The examining finger should be moved laterally to palpate the levator ani. Taut bands of contracted muscle may be palpated, they will be extremely tender, and palpation will reproduce the referred pain.³⁰ The patient should then be asked to abduct the

flexed thigh against the examiner's other hand to contract the obturator internus; if this maneuver exacerbates the pain with internal palpation, then this muscle is also involved.^{11,30} Direct pressure posteriorly will evaluate the coccygeus and piriformis muscles. The patient should then be asked to contract the muscles of the pelvic floor to assess muscle strength, as affected muscles are weaker with a restricted range of motion, and many patients are unable to adequately recruit the pelvic floor muscles to perform a Kegel exercise. Many women with normal pelvic floor musculature are unable to contract these muscles after verbal instruction, but by asking the patient to squeeze the muscles surrounding the rectum as though they are holding in flatus, many patients will be able to recruit the puborectalis portion of the levator ani complex. Some patients who are unable to isolate and contract the pelvic floor muscles may inadvertently perform a Valsalva maneuver. If tolerated, a bimanual examination should be performed to assess the uterus and adnexa, although it is frequently difficult to identify the organs of the upper reproductive tract as the source of pain with this maneuver because other potential sources of pain (particularly the bladder and muscles of the anterior abdominal wall) are also compressed. Similarly, these referred phenomena may also affect the ability to reliably interpret painful findings at laparoscopic pain mapping under conscious sedation.

No laboratory studies are diagnostic of MPS, and muscle biopsies are not indicated. Pelvic floor surface electromyography, either transvaginal or transperineal, may confirm a high resting tone of the pelvic floor, with a resting baseline of less than 2.0 μ V considered normal (Figure 3).^{13,33} Other abnormal findings on surface electromyography include an inability to isolate and recruit the pelvic floor muscles and early fatigue on repetitive contractions. There are electrodiagnostic characteristics of trigger points, but needle electromyography is not practical or necessary. Diagnosis of high-tone muscle dysfunction can easily be confirmed by a physical therapist trained in assessing the pelvic floor, so referral to such a practitioner is invaluable when the diagnosis is in question.

MANAGEMENT

Management of pelvic floor MPS generally involves local techniques aimed at the taut bands of muscles and central therapies aimed at the plastic changes in the CNS that contribute to the pain cycle and referred pathology. Treatment options may include physical therapy, complementary approaches, medications, injections, and (most recently) neuromodulation. For



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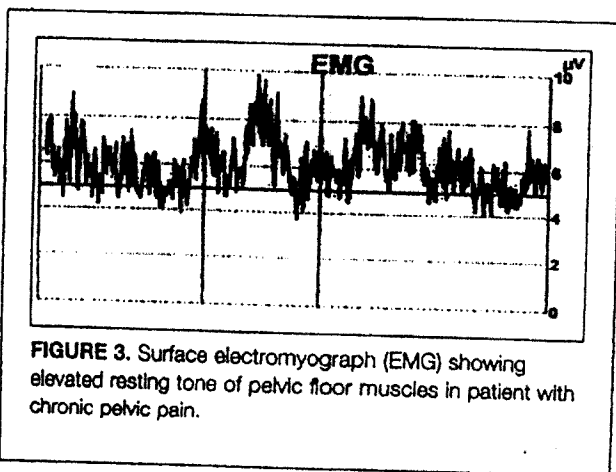


FIGURE 3. Surface electromyograph (EMG) showing elevated resting tone of pelvic floor muscles in patient with chronic pelvic pain.

patients with secondary MPS, treatment of the primary condition should be maximized. It is also important to remember that treating referred MPS will often improve symptoms associated with underlying visceral pathology, and so regardless of the primary diagnosis, treating MPS is always warranted. For the majority of women, an integrated approach using several therapies is the most effective.

Physical Therapy and Biofeedback

An appropriately trained physical therapist is essential for pain management in patients with high-tone pelvic floor dysfunction. Physical therapy is the most effective and least invasive technique for management of MPS, whether it is a primary or secondary phenomenon; more than 70% of women with CPP and a myofascial component will have significant improvement in pain with physical therapy.^{25,34-38} Techniques that may be employed by the physical therapist include internal massage, postisometric relaxation, and ultrasound in addition to correcting any postural problems.^{11,35-38} Intravaginal electrogalvanic stimulation (high-voltage direct current) and electrical stimulation (low-voltage alternating current) are both very effective, with moderate to significant improvements in pain for 50% or more of patients.^{11,13,39} Home use of these devices may offer a more practical alternative for patients who do not live close to an appropriately trained physical therapist. Biofeedback may be used to instruct patients to isolate and strengthen the pelvic floor muscles, and exercises to strengthen the core muscles of the abdomen may help correct posture and inappropriate loading on the pelvic floor. Many patients with CPP also develop kinesiphobia, therefore, encouraging ambulation and exercise is essential.⁷

Complementary Therapies

Acupuncture may be effective for a variety of chronic MPS conditions, and may be considered for pelvic floor pain.^{40,41} While this therapy has been practiced for more than 2,000 years in China, there are few prospective studies and results are highly practitioner-dependent, as there are many techniques and styles.⁴⁰ There is evidence to support a better analgesic effect with deep versus superficial stimulation.⁴¹ Relaxation and massage therapy may also be useful, as stress and anxiety can exacerbate MPS. Referral to a psychologist experienced in treating patients with chronic pain may also be beneficial in developing coping mechanisms and identifying and minimizing emotional factors that trigger acute exacerbations. However, most of these therapies are not covered by commercial insurance, and many patients do not have the financial resources to explore these options.

Pharmacologic Therapy

Myofascial pain is frequently severe, and so many patients may require analgesics, especially during the first month of physical therapy, as the manual release techniques many initially exacerbate pain. Analgesics such as acetaminophen and nonsteroidal anti-inflammatory drugs should be considered as first-line agents, although opioids may also be required. Methadone, an opioid that is also an N-methyl-D-aspartate (NMDA)-receptor antagonist, may be a particularly useful choice when opioids are required, as the NMDA receptor is involved in the central sensitization of chronic pain.⁴² Adjuvant analgesic medications such as tricyclic antidepressants or antiepileptic drugs should also be considered for chronic MPS, and have shown promise for CPP in women with IC.⁷ Venlafaxine—a nontricyclic norepinephrine reuptake-inhibitor antidepressant—may also be a useful adjuvant analgesic, but the selective serotonin reuptake inhibitors are not effective for this indication.^{7,43} Skeletal muscle relaxants may also be helpful, although generally these medications are more effective for acute muscle pain and spasm; tizanidine (an α_2 -adrenergic agonist) may be the best choice in this class, as α_2 agonists are also effective analgesics.^{7,44} The α_1 -adrenergic agonists have been used for functional bladder-neck obstruction in women, with improvement of voiding dysfunction in 50% of patients.^{45,46} These medications have also been used with some success for men with high-tone pelvic floor dysfunction and CPP, and further exploration of this class of drugs for women is warranted.

Chronic Pelvic Pain

Injections

Trigger-point injections have long been used for MPS; "needling" the taut band of muscle is believed to mechanically disrupt the trigger-point mechanism and improve both local and referred pain.^{15,17} Dry needling, sterile water, and a variety of local anesthetics have been used, and all appear to be effective; a small volume of local anesthetic administered at the time of needling may reduce postprocedural pain.⁴⁷ Treating abdominal-wall trigger points may help with pain referred from pelvic floor muscles or visceral pathology, and transvaginal injections of trigger points in the levator ani have also been described for patients refractory to physical therapy.^{11,33}

Botulinum toxins—ie, presynaptic neuromuscular blocking agents—are used for chronic MPS.⁴⁸⁻⁵⁰ Intramuscular injection with botulinum toxins is especially appealing for chronic MPS for several reasons. Not only does the needle mechanically disrupt the trigger point, but the paralytic effect on the muscle may also

reduce nociceptive input to the spinal cord, helping to break the cycle of pain and spasm.⁴⁹ In addition, retrograde uptake of metabolites by the nervous system may result in direct antinociceptive effects both peripherally and centrally.⁴⁹ A small prospective study of women with CPP and chronic spasm of the pelvic floor injected with a total of 40 U of botulinum toxin A into the pubococcygeus and puborectalis muscles suggests that high-tone pelvic floor dysfunction may respond to this therapy, although much more prospective research is needed.⁵¹

Neuromodulation

Patients with severe CPP and documented high-tone dysfunction of the pelvic floor who are refractory to all other treatments may be considered for sacral neuromodulation. The primary innervation to the pelvic floor is S2-S4, with 70% of the innervation to the levator ani from S3.^{11,20} Neuromodulation may affect afferent impulses to the spinal cord, and neuromodu-

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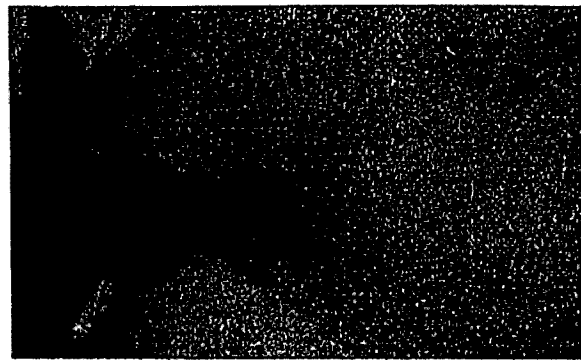
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Trigger-Point Injections

Trigger-point injections should be considered when non-invasive manual approaches have failed to inactivate the trigger point. The patient must be comfortable and the muscle relaxed, making it easier to distinguish the taut band of contracted muscle from neighboring relaxed muscle fibers.

The muscle is palpated with a finger; the finger slides over the muscle using constant pressure, and the trigger point will be felt as a ropy structure or taut band that rolls under the finger. Palpating the trigger point in this fashion should reproduce the patient's pain. If the patient is not obese, a localized contraction (twitch response) from the affected muscle will be palpated, or even seen, on localization of the trigger point.

Once the trigger point has been localized, the area should be marked with a pen, and then cleaned with antiseptic prior to injection. The skin may be anesthetized with a vapocoolant spray, the trigger point location reconfirmed by palpation, and the trigger point immobilized between two fingers. A 22- or 25-gauge needle is used to penetrate the trigger point, which should reproduce the pain and the twitch response. With practice, the firm knot of muscle may be felt with the needle. A small amount of local anesthetic (approximately 1 mL) may be injected at each location to reduce postprocedure discomfort, but dry needling is equally effective in treating trigger points. Pressure should be applied immediately if hemostasis is needed, and the affected muscle should be stretched through the full range of motion as soon as possible after the injection to promote release of the affected muscle fibers.²⁶



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lation at S3 may help patients with significant spasm and pain in the levator ani.^{7,13,52} The use of implantable neuromodulation devices for patients with high-tone pelvic floor dysfunction is still experimental, and it is important to remember that even with the most rigorous patient selection, a placebo response rate of up to 20% may be reported.⁵³

CONCLUSION

Pelvic floor MPS, or high-tone pelvic floor dysfunction, is an important contributor to the enigmatic CPP syndrome. Myofascial pain may be the primary cause of CPP, or may be a referred phenomenon from visceral pathology that eventually becomes a self-sustaining secondary pain syndrome. Central changes in the nervous system facilitate the development and maintenance of chronic MPS. Myofascial pain syndrome is diagnosed by clinical criteria, and integrated treatment approaches that direct therapy to the affected muscles and also address the associated changes in the nervous system have the best chance of success. All women with CPP should be evaluated for MPS of the pelvic floor, as failure to identify and treat this component may result in an incomplete response to therapy. Referred pain and pathology increase diagnostic challenges, and may help to explain the persistence of pain after maximal therapy for known somatic or visceral conditions.

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