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PELVIC FLOOR MYOFASCIAL TRIGGER POINTS: MANUAL THERAPY FOR INTERSTITIAL CYSTITIS AND THE URGENCY-FREQUENCY SYNDROME

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ABSTRACT

Purpose: The effectiveness of manual physical therapy was evaluated in patients with interstitial cystitis and the urethral syndrome, that is urgency-frequency with or without pelvic pain. The rationale was based on the hypothesis that pelvic floor myofascial trigger points are not only a source of pain and voiding symptoms, but also a trigger for neurogenic bladder inflammation via antidromic reflexes.

Materials and Methods: From September 1995 to November 2000, 45 women and 7 men, including 10 with interstitial cystitis and 42 with the urgency-frequency syndrome, underwent manual physical therapy to the pelvic floor for 1 to 2 visits weekly for 8 to 12 weeks. Results were determined by patient completed symptom score sheets indicating the rate of improvement according to outcome parameters, including 25% to 50%—mild, 51% to 75%—moderate, 76% to 99%—marked and 100%—complete resolution. In 10 cases these subjective results were confirmed by measuring resting pelvic floor tension by electromyography before and after the treatment course.

Results: Of the 42 patients with the urgency-frequency syndrome with or without pain 35 (83%) had moderate to marked improvement or complete resolution, while 7 of the 10 (70%) with interstitial cystitis had moderate to marked improvement. The mean duration of symptoms before treatment in those with interstitial cystitis and the urgency-frequency syndrome was 14 (median 12) and 6 years (median 2.5), respectively. In patients with no symptoms or brief, low intensity flares mean followup was 1.5 years. In 10 patients who underwent electromyography mean resting pelvic floor tension decreased from 9.73 to 3.61 μV., which was a 65% improvement.

Conclusions: Pelvic floor manual therapy for decreasing pelvic floor hypertonus effectively ameliorates the symptoms of the urgency/frequency syndrome and interstitial cystitis.

KEY WORDS: bladder; urination disorders; pain, intractable; cystitis, interstitial; physical therapy

It is well established that dysfunctional pelvic floor muscles contribute significantly to the symptoms of interstitial cystitis and the so-called urethral syndrome, that is urgency-frequency with or without chronic pelvic pain.[1–6] However, it is also possible that these muscles act not only as a source of symptoms, but also as a trigger for neurogenic inflammation of the bladder wall, which is a source of the urothelial permeability characteristic of interstitial cystitis.

Schmidt and Vapnek performed urodynamics in patients with interstitial cystitis or severe urgency and frequency, and observed that pain episodes paralleled behavioral changes in the sphincter more than in the bladder.[2] Pressure applied to the pelvic floor muscles, especially the levators, elicited pain and continued compression referred pain to the suprapubic and perineal regions, rectum, glans penis and labia. Most patients had no voluntary control over the pelvic floor muscles. However, after the muscles were relaxed through biofeedback or neurostimulation symptoms rapidly improved.

Because the symptoms of interstitial cystitis and the urgency-frequency syndrome are similar, the etiology may also be similar. An association of dysfunctional pelvic floor muscles with voiding symptoms in the urgency-frequency syndrome has also been noted:[3,4] In 25 women with severe urinary urgency and frequency Schmidt and Tanagho noted hyperactivity of the voluntary muscles surrounding the distal third of the urethra.[3] In patients with the urgency-frequency syndrome Bernstein et al observed a high tonic level in the pelvic floor, poor ability to relax or tense and inadequate voluntary control.[5] In similar patients Kaplan et al diagnosed sphincter dyssynergia or pelvic floor hyperactivity.[6] What these reports imply is that the bladder may not be completely responsible for the symptoms of the urgency-frequency syndrome and interstitial cystitis.

Therefore, it is possible that in some patients interstitial cystitis represents the end of a spectrum on which the urgency-frequency syndrome symptoms are at the start.[7] It may follow that progression can occur along this spectrum as the result of years of chronic and/or progressive myofascial dysfunction. In support of this concept Held et al reported that 28% of patients diagnosed with interstitial cystitis recalled difficult voiding in childhood.[8] Likewise in my experience at the initial interview patients often report a long history of intermittent urinary symptoms that progressed to an association with pain. Pelvic floor myofascial trigger points may underlie the pathophysiology of this progression.

As noted by Schmidt and Vapnek,[2] pelvic floor findings on palpation are consistent with those of a myofascial trigger point, defined by Simons et al as a tender spot created by injury at the motor end plate as a result of acute, repetitive or sustained muscle overloading.[9] Depending on the severity of myofascial injury a trigger point can be latent and asymptomatic. The confusing aspect in the development of a symptomatic trigger point is that traumas leading to it may be additive, contributing to an injury pool.[10] Sometimes the

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final triggering event appears so trivial that it is not recognized as a cause.

Although the similarities of neurogenic inflammation and interstitial cystitis have been described,[11,12] to my knowledge the initiating event that causes bladder wall neurogenic inflammation has not been discovered. However, in my experience the majority of patients with interstitial cystitis report an early history that may result in pelvic floor muscle dysfunction and they have urethral or anal symptoms suggestive of increased pelvic floor tension. Accordingly as an integral part of the treatment regimen in these patients, normalization of these muscles is included by eradicating the trigger point and reeducating the pelvic floor with stretching and strengthening exercises.

PATIENTS AND METHODS

Patients. Between September 1995 and November 2000, 45 women and 7 men 26 to 80 years old underwent manual therapy for pelvic floor myofascial trigger points, including 6 women and 4 men in whom interstitial cystitis had been documented by hydrodistention under anesthesia, and 39 women and 3 men with the urgency-frequency syndrome, that is severe urinary urgency and frequency with or without urethral pain (the so-called urethral syndrome and prostatodynia, respectively). Some patients with the urgency-frequency syndrome who had symptoms suggestive of interstitial cystitis refused cystoscopy under anesthesia.

Patients were referred for manual therapy because of dissatisfaction with previous treatments. Administration of antibiotics, pentosan polysulfate sodium, tricyclic antidepressants, heparin/dimethyl sulfoxide and sacral neurostimulation had been ineffective in 3, 4, 4, 4 and 1, respectively, of the 10 patients with interstitial cystitis. Of the 42 patients with the urgency-frequency syndrome ineffective treatment included antibiotics in 55%, urethral dilation in 50%, anticholinergics in 30%, diazepam in 22%, tricyclic antidepressants in 15%, α-blockers in 12.5%, phenazopyridine hydrochloride in 10%, surgery in 5% and acupuncture in 10%.

At the initial visit patients were given a symptom score sheet. Questions on urethral, bladder and low back pain that were relevant to each gender were included as well as those specific to each on testicular, penile, prostatic and vaginal pain. Voiding questions involved urgency, frequency, nocturia, hesitancy, intermittency, stream strength and incomplete emptying. Patients were asked to rate symptoms on a scale of 0—none to 4—severe.

In each completed questionnaire scores represented symptoms present in the preceding 30 days. After comparing pretreatment and posttreatment scores the rate of improvement was recorded as 25% to 50%—mild, 51% to 75%—moderate, 76% to 99%—marked and 100%—complete resolution. In 10 cases the effect of myofascial manual therapy was evaluated by electromyography using a biofeedback unit and perianal/perivaginal sensor. Pretreatment and posttreatment measurements were compared.

Manual therapy. Initial physical examination done via the rectum or vagina with the patient in the lithotomy position consists of palpation of the whole pelvic floor, that is the urinary and anal sphincter, pubourethralis, vaginalis and rectalis, iliococcygeus, obturator internus and piriformis muscles. This examination identifies tightness, tenderness or taut bands and pain radiation that duplicates symptoms. Tenderness is usually evident in women in the muscles and connective tissue lateral to the urethra (urinary sphincter and pubourethralis), and in men in the puboprostatic muscle (pubococcygeus) lateral to the prostate and in the urogenital diaphragm. In addition, trigger points in the perineal body may be a significant source of perineal pain in men.

In contrast to external muscle groups that physiotherapists treat manually with 1 or 2 hands, internal muscle groups limit the practitioner to 1-finger treatment via the rectum or vagina. Tenderness, tightness or taut bands are located. They are then treated with compressing, stretching, strumming at right angles to the affected muscle bundles or allowing the finger to glide between fibers to seek the direction of least resistance, termed following the well. The simultaneous use of external muscle stretching (piriformis stretch or isometric stretches of the pubococcygeus) or external heat application facilitates greater muscle relaxation.

When these techniques are applied in women, the tender areas in the urinary sphincter, periurethral tissues and pubourethralis are compressed against the symphysis pubis, combined with lateral traction (fig. 1). Initial light pressure is steadily increased as the patient adjusts to the technique. If the first contact is too abrupt or firm, muscle spasm can result.

After this lateral stretching/compression maneuver is repeated several times posterior traction is applied via the vagina or rectum (fig. 2). The patient is then asked to contract the muscle after the pubovaginalis is demonstrated, while the examiner finger is held fixed. This maneuver results in an isometric contraction against resistance. This type of stretching has a reflex inhibitory effect on muscle tension and results in greater relaxation and muscle length. With the examiner finger in this new, more posterior position the patient again repeats the isometric contraction several times, which results in even more muscle elongation.

The goal of this maneuver is to lengthen these anterior contracted muscles by decreasing periurethral tension, eradicate trigger points in the levator muscles, reeducate the muscles to a normal range of motion and evoke patient awareness to muscle tension. When periurethral palpation is repeated, there is usually less tenderness, softening and thinning of the contracted tissue mass between the palpating

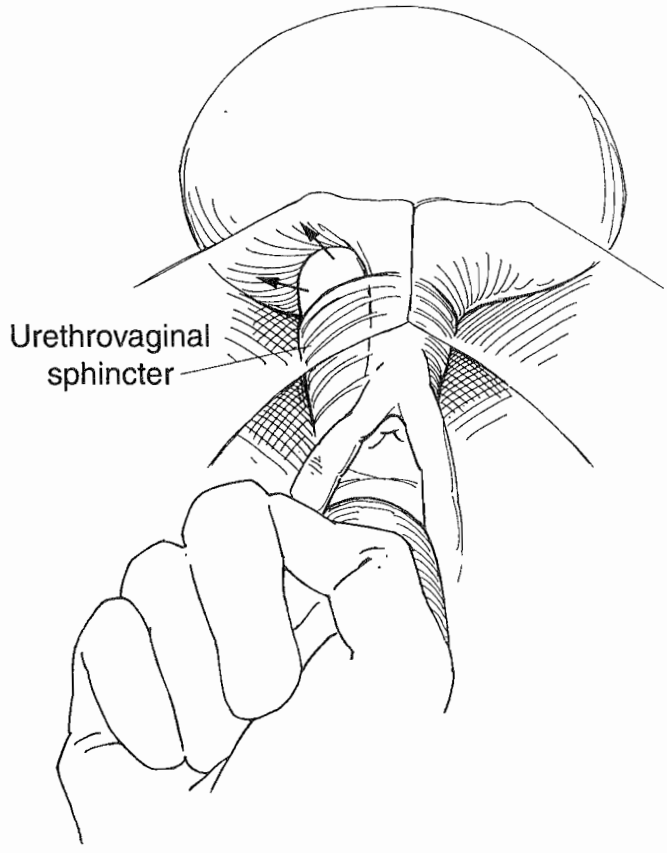

FIG. 1. Lateral stretching and compression of urinary sphincter.

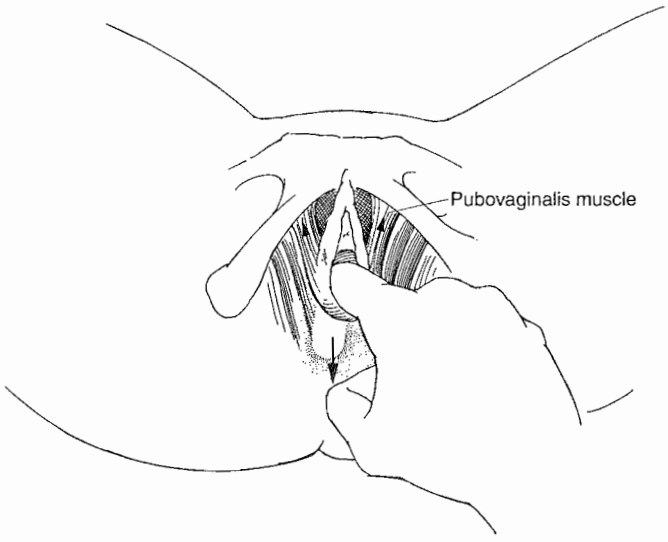

FIG. 2. Posterior stretching of pubovaginalis muscle

finger and symphysis pubis as well as improvement in patient capacity for muscle contraction and relaxation.

A similar technique is used in men but attention is directed to the endopelvic fascia and puboprostatic or pubococcygeus muscle lateral to the prostatic edge from bladder neck to membranous urethra (fig. 3). Repetitive compression loosens the tissue and decreases sensitivity. After these ends are achieved attention is directed toward the urogenital diaphragm and area of the urinary sphincter. The curved internal finger is pointed toward the examiner and the urogenital diaphragm is stretched internally. The posterior margin is readily identified by the superficial transverse perineal muscle, which limits posterior movement.

In addition to the urogenital diaphragm, the obturator internus is commonly involved. Involvement can be detected by having the patient abduct the thigh in the lithotomy position against resistance by pushing the knee lateral against the hand (fig. 4). This maneuver causes the obturator internus muscle to contract, shorten and widen under the levator muscle, which makes it identifiable. Any tender points are then eradicated by compression and stretching. Compression can be most effective when the patient brings the ipsilateral thigh toward the contralateral shoulder in the so-called piriformis stretch. The stretch can be enhanced with fluorimethane spray-stretch techniques and/or heat.[10] Trigger points in the levator muscles overlying the obturator internus can be identified by noting any increase in pain under the palpating finger when the patient contracts the pubococcygeus muscle.

If discrete myofascial trigger points remain after 6 to 8 weekly treatments or are so tender that manual therapy cannot be done, 0.5% bupivacaine/1% lidocaine injection can be added. The technique depends on location. If a 2 to 3.5 cm. 25 gauge needle directed through the perineum can reach the internal finger, it should be used. This technique is especially helpful in men with perineal pain secondary to trigger points of the urogenital diaphragm, pubourethralis or perineal body. If the affected points are deeper, a prostate aspiration biopsy guide placed on the examining finger is directed through the anus or vagina to the trigger point. After the fingertip is in contact with the trigger point a 21 cm. 22 gauge spinal needle is inserted through the guide and 1 to 2 ml. are injected at each trigger point site. The muscle is then put through a full range of motion with stretches. These injections can be performed every 1 to 2 weeks depending on progress. It is not uncommon for patients to have temporary symptom exacerbation before significant improvement.

Treatment should continue until tenderness and tightness have dissipated, which requires 1 to 2 visits weekly for 8 to 12 weeks depending on the duration and severity of symptoms. As trigger points and muscle tension decrease in severity, the frequency of therapy is decreased. In addition to office treatment, the patient is instructed in a home program consisting of biofeedback and Kegel instruction, external pelvic muscle stretches and strengthening, and stress reduction techniques.

RESULTS

The 42 patients with the urethral syndrome presented to an average of 24 visits in a mean of 15.6 months, while the 10

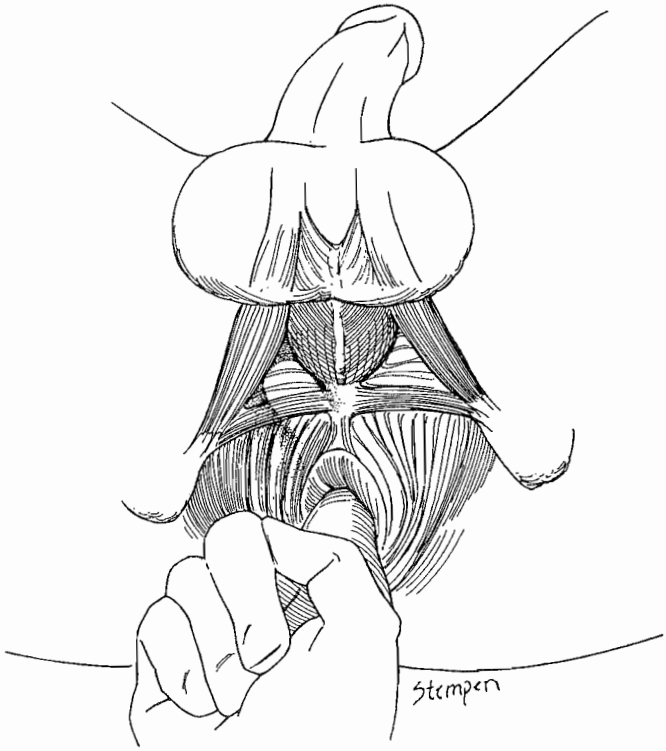

FIG. 3. Lateral and inferior stretching of puboprostate, urogenital diaphragm and urinary sphincter.

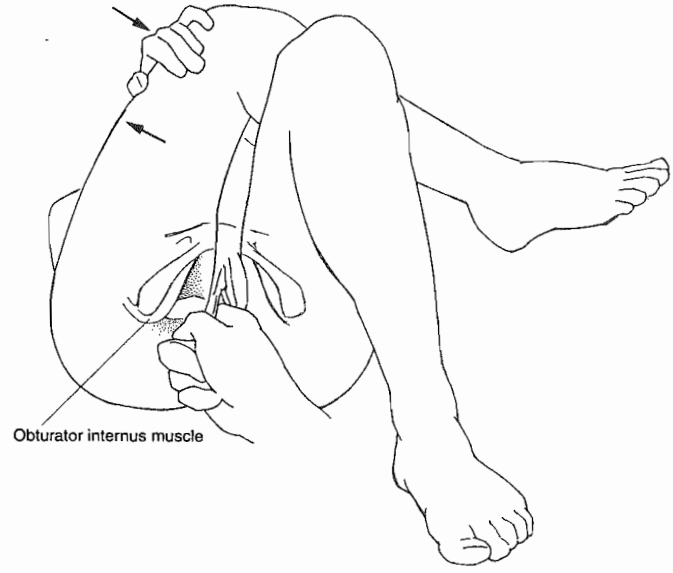

FIG. 4. Compression and stretching of obturator internus muscle assisted by external stretching.

with interstitial cystitis presented to an average of 18 visits in 19.7 months. No patient withdrew during the initial treatment period. Pretreatment symptom scores indicated a marked difference in those with interstitial cystitis and the urgency-frequency syndrome with a median score of 18.80 and 12.65, respectively. Patients with interstitial cystitis also had more tenderness, spasticity and pelvic floor muscle trigger points.

Of the 42 patients with the urgency-frequency syndrome 13 (31%) had moderate improvement, 13 (31%) had marked improvement and 9 (21.4%) had complete resolution of symptoms after the initial treatment period. Thus, complete resolution, marked and moderate improvement were achieved in 35 cases (83%). Only mild improvement was noted in 6 patients and symptoms were unchanged in 1. Urinary symptoms were most improved with an average 5.65 and 3.74 decrease in the symptom and pain scores, respectively.

Mean followup in these 42 patients was 20 months after the completion of therapy with 3 lost to long-term followup. The questionnaire showed a 9% decrease in improvement ratings. Thus, in the long term the improvement rate decreased to 74% (29 of 39 patients) from 83% at the completion of therapy.

After the initial treatment period 1 of the 10 patients with interstitial cystitis had marked and 6 had moderate improvement, while mild improvement was reported by 3. Thus, the combined rate of marked and moderate improvement was 70% (7 of 10 cases). No patients had complete resolution and none reported unchanged or worse symptoms. As in those with urgency-frequency, urinary symptoms improved most with an average 5-point decrease in the symptom score, although pain improved only slightly less, that is by 4 points. Mean followup in these cases was 19 months after the completion of therapy. Three patients were lost to followup but respondents reported no change in the symptom rating. In the 10 patients who underwent electromyography the mean pretreatment resting tension value of 9.73 μV. decreased after therapy to 3.61 μV. (fig. 5).

Many patients had periodic symptom flares that appeared to be related to stress, diet, sexual activity or other specific physical activity. However, after the treatment course was completed the flares were shorter and less intense. The need to treat patients periodically for these flares explains the 3 to 48-month range in followup to the completion of therapy. Generally the duration of symptoms before treatment correlated directly with symptom disappearance or decrease. In addition, patients who realized that life stresses were associated with symptom flares and, thus, habitually used stress decreasing techniques, or participated in support groups or psychotherapy generally responded better. After treatment was begun a symptom flare tended to respond much more rapidly (1 or 2 treatments) than at the start of therapy. During subsequent months flares were less readily initiated by activity that had triggered them previously. The decrease in symptom improvement after 6 to 12 months was largely attributable to patient failure to continue the home program, that is stress reduction techniques, Kegel and physical therapy exercises.

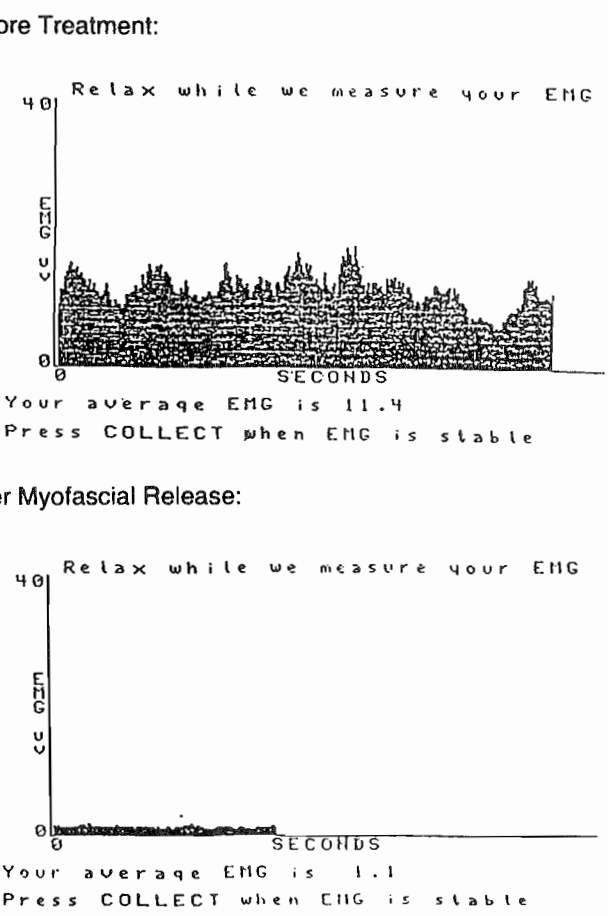

FIG. 5. Effect of myofascial release on pelvic floor hypertonus. *EMG*, electromyography.

DISCUSSION

Patients with urinary urgency and frequency with or without chronic pain present a frustrating clinical challenge. The enhanced urothelial permeability characteristic of interstitial cystitis has been well described,[13] although the pathogenetic mechanism is complex. Nevertheless, to provide relief from the vexing symptoms of urgency-frequency and interstitial cystitis practitioners must attempt to address their origin. The pelvic floor is particularly vulnerable to myofascial trigger points because of its central location, transmitting forces between the upper body and legs, constant supportive, sphincteric and sexual activity, characteristic eccentric or elongated type of contraction that places more stress on myofascial tissue[14] and significant response to stress.

The anatomical reason that these acute pelvic floor myofascial trigger points can influence the bladder lies in the close proximity within the spinal cord of the afferent nerve endings from the pudendal (pelvic floor) and bladder parasympathetic nerves. With enough painful input to the sacral spinal cord spinal dorsal horn neurons may possibly trigger antidromic transmission into the adjacent bladder nerves. The more severe pretreatment symptom scores of patients with interstitial cystitis in this series represent evidence of the painful consequences of bladder involvement.

Neurogenic inflammation has been observed after stimulating spinal cord nerves,[15] pelvic ganglia[16] and dorsal roots.[17] Jasmin et al experimentally produced neurogenic cystitis in rats by injecting the neurotropic virus into the abductor caudalis dorsal tail muscle.[11,18] They postulated that bladder related circuits were activated as a result of the invasion of neighboring circuits by this virus. Therefore, it is possible that pelvic floor myofascial pain may initiate antidromic transmission to the adjacent bladder nerves via the dorsal root reflexes,[19] axon reflexes from the peripherally bifurcating afferent fibers[20,21] or other unknown mechanisms that initiate the neurogenic inflammation described in these studies.

This antidromic propagation into the bladder afferent C-fibers may cause silent nociceptors in the bladder to increase in number and release substance P and calcitonin gene-related peptide, which degranulate mast cells.[22] Local axon reflexes can also release substance P at perivascular

sites, creating vasodilation. Increased substance P fibers and degranulated mast cells are present in the bladder submucosa in cases of interstitial cystitis.[12,23]

Mast cell degranulation releases histamine, serotonin and prostaglandin.[24] In turn this release causes pain, vasodilation, tissue damage and increased bladder lining permeability.[13,22] This process can then be perpetuated through central mechanisms when irritants released from the mast cells stimulate bladder afferents (fig. 6).[12,21]

Although it is possible that treatment directed to the bladder for decreasing the effect of local noxious stimuli can convert an adjacent active painful myofascial trigger point to an asymptomatic one, the latent point still represents a risk. It can be activated by stress, dietary irritants, improper exercise or movement, and trauma due to sexual activity, cold weather, hormonal shifts and viral infection.[9] These factors are known to cause flares in interstitial cystitis.

Symptoms that continue despite physical therapy to the dysfunctional pelvic floor can be partially explained by central sensitization. Chronic pain in the pelvic floor or bladder can affect the spinal and supraspinal neurons in a way that creates various sensory changes, such as a decreased pain threshold, so that a nonpainful stimulus such as touch or bladder filling causes pain (allodynia), increased pain intensity with a painful stimulus such as dietary irritants (hyperalgesia), spontaneous pain activity, expansion of the pain field, an increased degree and greater duration of pain with each repeated painful or nonpainful stimulus (windup) or sympathetically maintained pain.[25] Initially this mechanism appeared to be a major deterrent to successful interstitial cystitis treatment but others have reported that central sensitization cannot be maintained without ongoing painful input.[26–28] Therefore, this finding implies that if noxious input were disrupted for a period, central sensitization would decrease.

Because the sensitized sacral spinal cord is influenced not only by the bladder and pelvic floor, but also by other organs or muscles that converge on these neurons (somatovisceral convergence) or by general factors that increase nerve sensitivity, a comprehensive approach must be used to stop ongoing painful input from any source.[29] The external muscles most commonly involved have attachments to the bony pelvis or attach in close proximity to the urinary sphincter, namely the gluteal, iliopsoas, piriformis, quadratus lumborum, adductor magnus and rectus abdominis muscles. After they become dysfunctional, they may perpetuate pelvic floor pain and hypertonus.[30] Thus, it is essential that patients should undergo a physical therapy evaluation and treatment to correct any predisposing or perpetuating condition.

It has been suggested that treatment for interstitial cystitis and the urethral syndrome should include muscle relaxing techniques, such as biofeedback and neurostimulation.[2,3,5] However, such remote control methods are not the first line of therapy for other muscles and since these techniques are not usually included in the medical school curriculum, they may be bypassed in favor of other, more familiar techniques. Nevertheless, the treatment plan must consider a combination of options, including bladder directed, physical and mental therapy, pelvic floor rehabilitation and the avoidance of pain producing activity with diet and exercise.

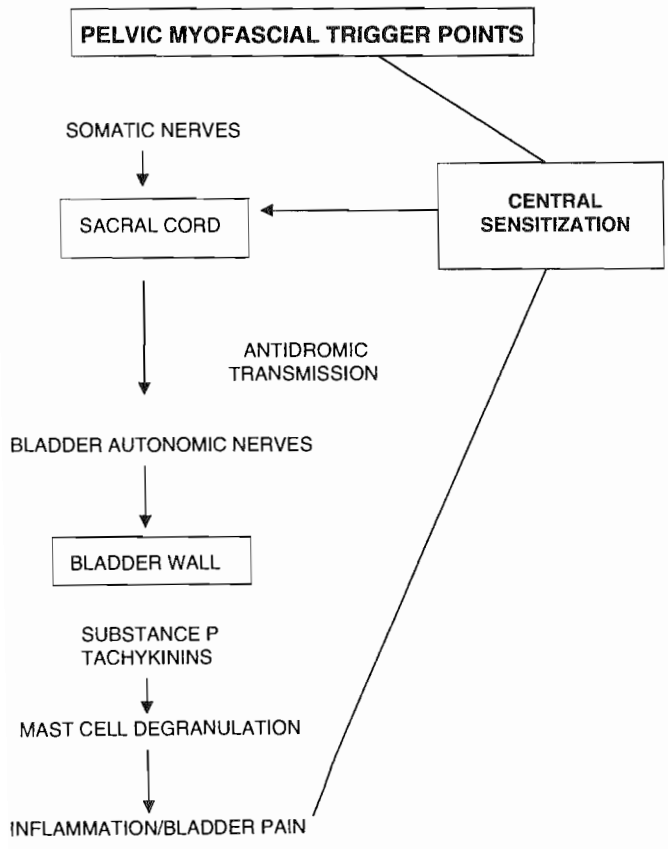

FIG. 6. Theoretical pathophysiology of interstitial cystitis

CONCLUSIONS

As part of a comprehensive treatment plan in patients with interstitial cystitis and the urgency-frequency syndrome, pelvic floor physical therapy arrests the neurogenic trigger leading to bladder changes, decreases central nervous system sensitivity and alleviates pain due to dysfunctional muscles.

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