Myofascial pain syndrome in the pelvic floor: etiology, mechanisms, symptoms, diagnosis, and treatment.

Review article

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Abstract

Myofascial pain syndrome in the pelvic floor is a common condition, originally described by Drs Janet Travell and David Simons, involving the musculoskeletal system; there is a high percentage of misdiagnosis and a high failure rate of medical interventions and, therefore, of frustrated specialists and patients. The etiology includes urologic, gynecologic, gastrointestinal, proctologic, neurological, and musculoskeletal problems. Muscle injury can occur when soft tissues are exposed to single or persistent episodes of muscular overload. Myofascial pain syndromes are characterized by the development of myofascial trigger points that are locally painful when stimulated. These trigger points are hyperirritable spots within a taut band of hypercontracted extrafusal muscle fibers. The trigger point and the taut band can be palpated, which provokes the typical referred pain. Affected muscles are usually shortened and have an increased tone and tension. Diagnosis is done based on the history, physical examination, and neurophysiologic tests. Treatment requires medical, psychological, and physical therapies, including local infiltrations with lidocaine, dry needling, botulinum toxin injections, and several physiotherapeutic and psychological techniques.

Keywords

Pelvic Floor, Trigger Points (TrPs), Myofascial Pain, Referred Pain, Infiltrations, Physiotherapy.

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"The only true wisdom is knowing you know nothing"
Socrates

1. INTRODUCTION

Myofascial syndrome in general, and in the pelvic floor in particular, is a well-differentiated entity thanks to the studies of Janet Travell and David G. Simons, published since 1983. With the passage of time, the concept of myofascial pain has been approached in different ways. In 1952 Dr. Travell published the first compendium of individual pain patterns that are characteristic of each of the muscles that most often develop this discomfort throughout the body [3, 22].

Later she herself recognized the multiplicity of factors that perpetuate the trigger points of this pain, which transform a simple muscle pain syndrome into a chronic, complex, and debilitating pain.
It is the most common cause of musculoskeletal pain, yet its diagnosis is often overlooked. Treatment is poor or ineffective, probably due to insufficient training of professionals in this pathology.
Moreover, chronic prostatitis, a condition that caused and keeps on causing concern for professionals and patients alike, seems to be closely related to myofascial pain and dysfunction. This was how Zermann et al. understood it in 1999 in their article "Chronic Prostatitis: A Myofascial Pain Syndrome?" This study involved 103 men, and 92.2% of those who had chronic pelvic pain/chronic prostatitis presented with pelvic floor dysfunction and negative microbiological tests, and a significant number had neurological dysfunction [18].
Similarly, in the same period Anderson et al. presented a paper recommending physiotherapy treatment for myofascial release of type III chronic prostatitis. They concluded that this treatment might be effective and lasting [19].
Earlier, in 1977, Sinaki et al. of the Mayo Clinic had published an interesting study on pelvic floor tension myalgia in 94 patients, in which a combined treatment of diathermy, Thiele massage, and relaxation exercises gave good results [20].
Other authors have defined this type of symptomatology as the short pelvic floor syndrome, due to loss of muscle length and the consequent functional problems [35,36].
As we shall see, we will come across a number of terms that we are very familiar with and that are interrelated: myofascial pain, muscle hypertonicity, trigger points, chronic prostatitis, physiotherapy, referred pain, sensitization of the nervous system, etc. In all these, the muscle and the nervous system form the central axis.

We will also encounter a number of disease entities which, although very different in origin, have a common aspect: the myofascial pain syndrome. Thus, it can be deduced that treatment will be very similar in all these cases. Among these entities are chronic bacterial prostatitis [30,31], chronic nonbacterial prostatitis/chronic pelvic pain syndrome [30,31,100,101], interstitial cystitis [30,32,35,36,49,50,70, 99,100], levator ani syndrome [26,34,70], urgency-frequency syndrome [35,36, 49], prostatodynia [32,35,36], endometriosis [33,50,70], pyramidal syndrome [35,36], vulvodynia [35,36,49,70,92], coccydynia [35,36], irritable bowel syndrome [70,98], and abdominal scar pain [53].

Today we know that the myofascial syndrome is a regional pain disorder that affects the muscles and fascia, in such a way that the muscles involved have a trigger point (TrP) or trigger points as associated components.

The muscles involved have the following characteristics [21, 22]:

Pain produced and maintained by one or more active TrPs.

The TrP is located within a taut band of the muscle or its fascia.
The taut band and the TrP can be palpated, which provokes referred pain.
The ability of the affected muscle to stretch is restricted, and often the muscle cannot stretch completely. The muscle is contracted (Figure 1).
The pattern of referred pain is specific for each muscle.
The muscles **adjoining the affected muscles are also tense on palpation.**
There is a spasm response to firm pressure on an active TrP, because of a transient contraction of the muscle fibers in the taut band. This is called the local twitch response.
Moderate but sustained palpation of a TrP tends to accentuate the pain.
The maximum force of contraction of the affected muscle is decreased, with muscle weakness and increased fatigability, but without atrophy or muscle fibrosis.
The TrPs are activated by direct trauma, pressure, and/or muscle overload.
Autonomic regional and segmental changes coexist with the above symptoms: local changes in the skin, increased sweating, changes in local temperature, and sometimes small local edemas.

In some cases suprapubic edema, sacral edema, or edema in the vaginal fornices is especially important. In rare cases, the swelling is so pronounced that it can lead to misdiagnosis and to the belief that it is the only cause of the problem.

Sometimes there is no myofascial trigger point, but what is known as a Tender Point; in such a point there is no local twitch response nor a taut band, and the pain is localized and intense.

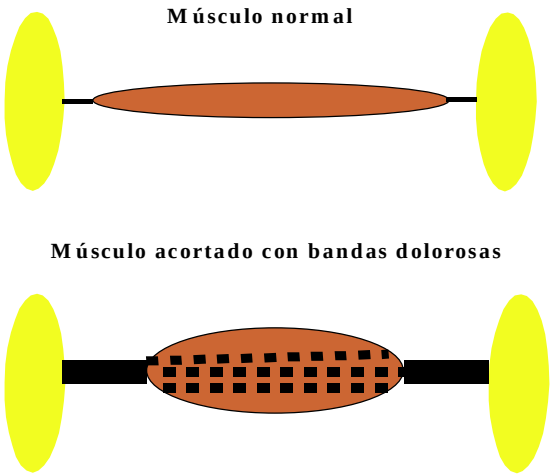


Figure 1: Normal Muscle
Contracted muscle with tender bands

2. BRIEF OVERVIEW OF THE ANATOMY OF PELVIC FLOOR MUSCLES

The pelvic floor or perineum is that part of the trunk that is located below the pelvic diaphragm. It is located in the inferior outlet of the pelvis [3, 4, 5].

The word perineum is derived from the Greek word perineos, which means the space between the anus and the scrotum.

It is of great importance in certain medical specialties such as urology, gynecology and obstetrics, proctology, neurology, and muscular and fascial disorders, although the latter are not covered by a single specialty. Physiotherapy is a closely related discipline that is concerned with the problems of this complicated and complex area of our body.

The boundaries of the pelvic floor are: the anal triangle or posterior part containing the anal canal, the ischiorectal fossae on each side, and the external anal sphincter; the urogenital triangle or anterior part containing the external genitalia and terminal portions of the urogenital ducts; the inferior side of the pelvis is closed, but the anal canal, urethra and, in women, the vagina, pass through it.

The posterior part is closed by the pelvic diaphragm, and the anterior part of the inferior part of the pelvis is closed by the urogenital diaphragm.

The two levator ani muscles and the two coccygeus muscles form the pelvic diaphragm and close the narrow inferior part, or the pelvic outlet, forming a large funnel. The pelvic diaphragm divides the pelvic cavity into two parts: the superior part containing the pelvic viscera and the inferior part, called the ischiorectal fossa, containing fat.

The urogenital diaphragm is a thin sheet of striated muscle that lies between the two faces of the pubic arch lining the anterior inferior part or the pelvic outlet where the most anterior and the most posterior fibers follow a transverse course (transversus muscle), while the medial fibers surround the urethra (external urethral sphincter muscle).

The muscles of the pelvic floor or perineum are: the external anal sphincter, the levator ani, the coccygeus muscle, the bulbospongiosus muscle, the ischiocavernosus muscle, the urethral sphincter, the superficial transverse muscle, and the deep transverse muscle.

The most important ones and the internal obturator muscle because of its important relationship with the perineum and referred pain [3,4,5] are described below.

Levator ani muscle

- Location: Posterior perineum. Consists of the puborectalis, pubococcygeus, iliococcygeus, and levator prostatae muscles.
- Insertions: Into the descending and horizontal rami of the pubic bone and into the anococcygeal raphe.
- Innervation: Pudendal Nerve and S2-S3-S4.
- Action: As pelvic diaphragm and as elevator and constrictor of the anus.

Obturator internus muscle

- Location: Interior and exterior of the pelvis.
- Insertions: Inside into the inner faces of the obturator membrane, descending branch of the pubis, and ischium; outside into the trochanteric fossa.
- Innervation: Main branch of the sacral plexus.
- Action: External rotator of the thigh.

Deep transverse perineal muscle
- Location: Dorsal part of the urogenital diaphragm.
- Insertions: Outside into the posterior surface of the ischial pubic ramus; inside into the anterior part of the median perineal aponeurosis.
- Innervation: Dorsal nerve of the penis.
- Action: compressor of the urethra as part of the urogenital diaphragm. Contributes to erection.

Ischiocavernosus muscle
- Location: In the perineum.
- Insertions: At the back into the face of the ischium and ischial pubic ramus; in the front into the root of the corpus cavernosum.
- Innervation: Pudendal Nerve.
- Action: Erector of the penis.

Bulbospongiosus muscle
- Location: In the anterior perineum.
- Insertions: At the back into the median raphe; in the front into the superior aspect of the bulb and the fibrous capsule of the dorsal aspect of the cavernous body.
- Innervation: Pudendal Nerve.
- Action: Accelerates or propels urine and semen.

External anal sphincter muscle
- Location:

-Subcutaneous part: surrounds the anal canal, lacks bony attachments.
-Superficial part: perineal body.
-Deep part: surrounds the anal canal, lacks bony attachments.

- Insertion: Coccyx
- Innervation: Inferior rectal nerve and perineal branch of S3.
- Action: Together with the puborectalis muscle it forms the voluntary sphincter of the anal canal.

Coccygeus muscle
- Location: In the posterior perineum.
- Insertions: On the outside into the ischial spine and sacrociatic ligament; on the inside into the margin of the coccyx.
- Innervation: Coccygeus.
- Action: Uncertain.

3. ANATOMICAL-CLINICAL CORRELATION

After the initial studies by Travell and Simons, Dr. Anderson and his team at Stanford University pioneered the study and detailed analysis of the pelvic floor muscles, their TrPs, and the symptoms of each one of them. Obviously, in many cases these symptoms overlap and it will be up to us to uncover this to be able to make an accurate diagnosis and take the most appropriate therapeutic measures [1, 2, 24].

3.1 Internal TrPs of the pelvic floor, typical referred pain, and resulting symptoms.

Levator ani muscle

Superior or puborectal portion

- Most important location of TrPs in males.
- Responsible for pain in the head and body of the penis. Probably caused by a TrP of the levator prostatae (in our experience).
- Sensation of fullness and pressure in the prostate.
- Pain referred to the urethra and the bladder.
- Pain or discomfort in the lower abdomen.
- Increased urinary frequency and urgency.

Inferior portion

- Pain referred to the perineum and the penis.

Middle portion, iliococcygeus muscle

- Pain referred to the lateral wall, perineum, and anal sphincter.

Posterior portion

- Sensation of a golf ball in the rectum.
- Pain during and after ejaculation.
- Pain after defecation.

External anal sphincter muscle

- Pain in the anus.
- Pain in the anterior part of the pelvis close to the pubis.
- Pain in the posterior part of the anal sphincter.
- Tingling and burning in the anal area (in our experience).

Coccygeus muscle

- Pain around the tailbone.
- Pain in the gluteus maximus.
- Pain during bowel movements.
- Intestinal fullness.
- Anal pressure and pain, and sensation of a golf ball in the rectum.

Internal obturator muscle

- Pain referred to the hip.
- Vulvar pain.
- Urethral pain in women.
- Pain in the entire pelvic floor.
- Sensation of a golf ball in the rectum.
- May simulate a pudendal nerve entrapment (in our experience), and since the nerve and the muscle are intimately related, palpation of the area causes a burning and intense pain.

Bulbospongiosus and ischiocavernosus muscles

- Pain in the base of the penis and perineum.
- Pain in the ventral aspect of the penis (in our experience).

3.2 External TrPs of the pelvic floor, typical referred pain, and resulting symptoms.

Quadratus lumborum muscle

- Inguinal pain.
- Pain in the lower abdomen.
- Pain referred to the hip.
- Low back pain.

Iliopsoas muscle

- Low back pain.
- Inguinal pain.
- Pain in the front of the leg.

Rectus abdominis muscle

- Pain radiating to the prostate area.
- Pain inside the penis.
- Pain in the lower abdomen.
- Low back pain.

Abdominal oblique muscle

- Pain radiating to stomach.
- Pain radiating to ribs.
- Inguinal pain.
- Testicular pain. This is a cause of testicular pain that is often overlooked.

Pyramidalis muscle (Figure 2)

- Pain in the bladder and urethra.
- Erectile dysfunction.
- Pain around the pubic bone.
- Pain referred to the sacroiliac joint, buttock and hip that increase when standing and sitting.
- Pinched sciatic nerve pain with neurological compression symptoms.

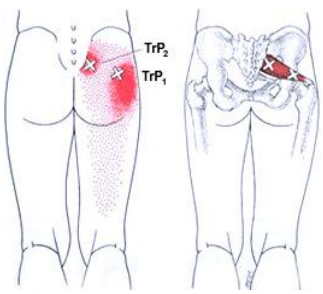


Figure 2

Gluteus maximus, medius, and minimus muscles

- Pain traveling down the leg.
- Testicular pain.
- Pain around the tailbone.
- Pain in the sacrum.
- Pain in the hamstrings.
- Pain in the pelvic girdle.
- Pain in the buttocks.

4. DEFINITION OF THE TRIGGER POINT (TrP) [21,22]

A TrP is a tiny area (it palpates like a grain of "crispy rice") with a diameter between 5 and 10 mm; it is highly irritable, located inside a muscle, rigid (taut band) on palpation, has a restricted stretching amplitude, and shows noticeable weakness. There is no atrophy or fibrosis and normally there is no neurological involvement, although there may be; in fact, there usually is neurological involvement to a greater or lesser extent when the TrP has been evolving for a long time.

It causes a decrease in the elasticity of the muscle involved and its fascia. With chronicity it causes shortening of the muscle and of adjacent structures.

4.1 Types of trigger point

- **Active TrPs:** these are painful without stimulation. They are always sensitive; the patient experiences them as constantly painful spots. The pain increases on palpation, pressing, mobilizing, and stretching the muscle.
- **Secondary TrPs.** These usually appear in response to the contraction of agonist and antagonist muscles that attempt to compensate for the injured muscle.
- **Latent or satellite TrPs.** These develop within the reference area of the original TrP. They are only painful on palpation and when activated they can behave as active TrPs.

5. ETIOLOGY AND MECHANISMS OF PRODUCTION OF THE LESION

- Chronic muscle tension patterns since childhood (sexual abuse [96], chronic constipation [95], dance training, stress, etc.).
- Small repeated traumas such as constipation, recurrent urinary tract infections, impact sports or sports with risk of perineal injuries, even if no injury occurs (bicycling, jogging, horseback riding, athletics, gymnastics, ballet, etc.).
- Small acute injuries when practicing sports.
- Direct physical trauma as a consequence of bicycling, labor, or urological or gynecological surgery.

- Inflammation of the pelvic organs: prostatitis, cystitis, urethritis, endometriosis, vaginitis, proctitis, hemorrhoids, or anal fissures
- Pain referred from other muscle groups, viscera, or nerves.

6. EPIDEMIOLOGY

- The condition is very common, but often it is not diagnosed as such. Of 283 consecutive admissions to a pain clinic, 85% were diagnosed as suffering from myofascial syndrome somewhere in the body [81]. The highest incidence is found among people between 30 and 50 years old. Latent TrPs become more frequent with increasing age and decreasing physical activity.
- In a multicenter study done in urological outpatient centers of 28 hospitals in Italy with 5,540 patients, Bartoletti et al. found that 746 of them had chronic pelvic pain. The prevalence of the syndrome was 13.8% and the estimated incidence was 4.5%. The syndrome is closely related to among others lifestyle, diet, smoking, sexual dysfunction, and anorectal disorders. Therefore, this phenomenon is much more significant than expected [95].
- Other authors have found results along the same lines [97, 99, 101].
- The relationship between sexual dysfunction and pelvic floor muscle problems (prostatitis) is very common and frustrating for the quality of life of patients, both men and women [102,103].
- A study by Krieger in 2004 on the classification, epidemiology, and implications of chronic prostatitis in the U.S., Europe, and Asia is worth reading, since it clarifies a number of uncertainties such as its actual frequency: it affects 2-10% of adult men and 15% suffer symptoms of prostatitis at some point in their lives [104].
- It is more common in women than in men.
- It is also more common in patients who perform tasks involving repetitive use of the muscles of the pelvic girdle, pelvic floor, and lower limbs, and who must also, because of the nature of their work, adopt incorrect, antiphysiological, or non-functional postures.
- Ninety-five percent of men diagnosed with chronic prostatitis do not show evidence of inflammation or infection [112,113].

7. PHYSIOPATHOLOGY

The physiopathology is largely unknown.
The most widespread theories are:

1. The energy crisis theory: This concept was developed to explain four significant facts: first, the absence of action potentials of the motor unit in the taut band of a TrP when the muscle is at rest; second, the fact that TrPs are activated by muscle strain; third, the sensitization of nociceptors in the TrPs; and fourth, the effectiveness of almost any therapeutic technique that leads the muscle from a shortened state to its proper length [73, 74].
2. Theory of dysfunctional muscle spindles: Hubbard and Berkoff concluded in an interesting paper that the EMG activity of TrPs was caused by dysfunctional use [77].

3. Pain-spasm-pain cycle hypothesis: This old concept does not stand up to experimental evidence, be it from the physiological point of view or from the clinical point of view [75, 76].

TrPs are thought to arise from a neuromuscular dysfunction and to evolve into well-defined histological lesions.
Any trigger can activate pathogenic mechanisms which give rise to the fascia, the muscle it contains, and sometimes other neighboring flexible and elastic connective tissues losing their own elasticity and the ability to glide between the different layers, which is vital for their proper function.
As a consequence, the fascia and muscle shorten and ache, and generate in an area of the muscle a taut band within which there is a hyperirritable point, the so-called "Trigger Point".
When not treated adequately and early, this creates a situation that can be compared with the wave that forms when you throw a stone into the water: the initial active spot creates hypomobility and dysfunction in adjacent tissues and viscera which, in the end, is sometimes greater than the problem that caused it. For this reason, myofascial dysfunction is sometimes quite distant from the point where it originated.

Myofascial TrPs cause chronic pelvic pain by three mechanisms:

- **Through local tension around the affected organs and referred muscle patterns.**

The striated muscles of the pelvic floor adhere closely to the visceral structures (urethra, bladder neck, prostate, vagina, and rectum) for support and sphincter control.
Because the afferent nerves of the viscera and deep muscles connect to the medial thalamus, they cannot locate noxious stimuli as do the nerves of the skin, which go from the lateral thalamus to the somatosensory cortex.
Therefore, patients with active TrPs and pelvic floor spasms do not perceive that their symptoms originate in the pelvic muscles.
They also experience varying degrees of emotional stress.

- **Viscerosomatic and somatovisceral reflexes.**

The effect of visceral pain on somatic structures was shown by Vecchiet et al. (1989) [7] and by Giambernardino et al. (1994) [8]. They found that between 30 and 64% of patients who had suffered repeated episodes of renal colic experienced lumbar muscle hyperalgesia years after the original pain.
The second group postulated that colic pain produced plastic neural changes in spinal or supraspinal levels that were perpetuated after the visceral impulses ceased.
These studies may actually have found myofascial TrPs created by visceral pain via the viscerosomatic reflex. This repetitive pain model may be applicable to any inflammation of a pelvic organ.

- **Central sensitization**

TrPs are not only sources of pain, but can also sensitize neurons of the CNS, thus leading to a more intense neuropathic pain that is resistant to treatment.

A review of the neurophysiologic bases of pain [9] suggests that afferent nerve branches in the spinal cord join with many dorsal horn cells in many upper, lower, or contralateral segments of the spinal cord.

Taken together, the nerves of muscles, organs, or skin may converge in the dorsal horn neurons and interfere with each other at supraspinal levels as a second-order neuronal pathway that is in close contact with the brainstem and the thalamus on their way to the cerebral cortex [10].

This interaction prevents a normal and coordinated communication between the organs. Thus, any damage in dorsal horn neurons and thalamic cells, which are the heart of the nervous communication system, may cause a general dysfunction.

Its normal activity is altered by chronic pain stimuli from the peripheral nerves that join with them.

These harmful impulses can have many causes: active trigger points, visceral pain, inflammation of the skin, etc.

And when they reach the cells in the spinal dorsal horn via C-fibers, neuropeptides are released which produce physical, chemical, and genetic changes that facilitate abnormal connections [11].
These plastic changes may alter pain perception in a variable way and cause pain by non-painful stimuli (allodynia); the pain can also be spontaneous, intensify (hyperalgesia), extend, or increase in duration, thus contributing to the worsening of the initial disease [11].

What begins as myofascial, visceral, or superficial may, with a sufficiently noxious stimulus, be transformed into neuropathic pain affecting a wider area and more organs [7, 8, 9].

If the dorsal horn cells remain in a state of sensitization, the initial pain may be reactivated by a noxious stimulus that reaches them from an organ that shares their nervous fields.

The sensitivity of these cells may be influenced by many changing factors: depression, hormonal changes during menstruation, sleep alterations, diet, etc. [12].

Pathophysiological considerations related to the treatment

Can these nerve disorders or sensitized neurons return to normal?
We can treat the trigger points, but can we treat the newly established reflex patterns or sensitization of the CNS?
Gracely et al. [13] suggest that changes in the central process cannot be sustained without the continuation of the painful focus. In their study, when they produced an

anesthetic block of a painful point near the elbow, chronic pain in the distal part of the arm and the hand disappeared.
Other authors, such as Koltenburg et al. [14], Cohen and Arroyo [15], Bach et al. [16], and Bonica (1990) show that the central processing mechanism reverts to normal when nociceptor activity is reduced to below critical levels.
Thus, if we provoke a block of the sensitized nerves and achieve a symptom-free period, we make recovery possible since there are no noxious stimuli at that time.

8. Perpetuating factors

These do not receive sufficient attention. If they are not eliminated, treatment will not last.
They are also predisposing factors. The **"overload"** factor of the muscles is constant. Elimination may lead to the disappearance of active TrPs.

8.1 Mechanical stress

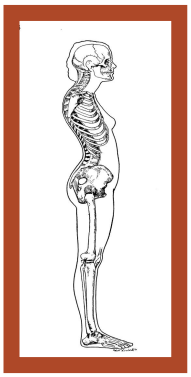
Lower extremity dysmetria (LED). If one lower extremity is shorter than the other, this can cause pelvic tilt while standing, lead to compensatory scoliosis, and to a perpetuation of the TrPs. This can be corrected with heel elevation. It is a key perpetuating factor. LED is strongly associated with back pain. It is worth to mention not only dysmetria of the LE but also changes in foot support that require correction with arch support and that can also create perineal dysfunction, especially when associated with risk factors such as sports or a standing profession. The impact that is cushioned at the pelvic floor is transmitted by the legs from the supporting base, which is the foot, while the lower limb is merely the transmitter of the impact [65,66].

8.2 Postural dysfunctions and abnormalities

Dysfunction of the sacroiliac joint, the sacrococcygeal joint, and the lumbosacral hinge may be aggravating causes for the TrPs of the pelvic floor [53].
The TrPs in the levator ani and coccygeus are perpetuated by postural tension caused by inadequate furniture, defective postures (both standing and sitting), overuse of muscle groups, prolonged immobility or sitting, and repetitive overload [67,68,69]. This leads in most cases to hypotonia of the subumbilical abdominal girth and a hypertonia of the thoracic diaphragm that generates hyperpressure on the pelvic diaphragm, which responds with hypertonia or hypotonia; both situations are possible but in either case harmful, because they produce pudendal neuropathy by compression in the first case and by overstretching in the second case. In the latter case it is also an aggravating and/or triggering factor for genital prolapse in women.

In an interesting article, Slocumb said that other coexisting diseases such as ovarian cysts or pelvic adhesions do not prevent a satisfactory response to local infiltration of the TrPs of the levator ani, coccygeus, or in scars of the vaginal cuff after hysterectomy [71].
King et al defined a typical posture of patients with chronic pelvic pain consisting of lordosis and anterior pelvic tilt, which occurs in 75% of the cases seen [72] (Figure 3).

Figure 3



8.3 Nutritional disorders [78, 79]

The water-soluble vitamins B1, B6, B12, folic acid, vitamin C, and the trace elements calcium, iron, and potassium play a role in myofascial pain syndrome.
Vitamin C, which is particularly important in this syndrome, is an essential co-factor in eight different enzymatic reactions such as norepinephrine and serotonin synthesis, both of which are involved in the central modulation of pain transmission. It is also involved in collagen synthesis and amino acid degradation. Collagen makes up one fourth of total protein in organic tissues, and therefore vitamin C deficiency leads to muscle and ligament disorders that may eventually cause or perpetuate TrPs [82].
There are two levels of vitamin deficit: **insufficiency**, which requires some metabolic adjustment by the organism, and **deficiency** which is a gross functional impairment with serum levels below the limit accepted as normal and associated with full-blown clinical disease.

- **Prevalence**

In a hospital-based study of 120 patients, 105 (88%) had low levels of one or more vitamins out of eleven, and 60 (50%) had low levels of two or more vitamins.
Folic acid was low in 45% of the patients, but only 38% had clinical manifestations [83].

- **Toxicity**

Fat-soluble vitamins A, D, and E have a much greater toxicity than the water soluble B complex vitamins.
Hypervitaminosis A can cause bone and joint pain and a severe throbbing headache, which can be confused with myofascial symptoms related to vitamin A deficiency.

8.4 Metabolic and endocrine disorders

- Hypothyroidism, which may be subclinical [84].
- Growth hormone deficiency.
- Hyperuricemia.
- Obesity.
- Hormonal changes: menopause.

8.5 Psychological factors

- Stress [93].
- Hyper-responsible personalities.
- Depression.
- Anxiety-depression syndrome [93].

8.6 Chronic infections and infestations

- Chronic prostatitis. Interstitial cystitis.
- Repeat cystitis.
- Oophoritis, salpingitis.

8.7 Other factors

- Sleep disturbance [85, 86, 87]. This is a very important factor because if the patient does not recover at night, chances of developing or perpetuating TrPs are very high.
- Pinched nerve (peripheral entrapments and radiculopathies). There is a significant correlation between its existence and the existence of TrPs [88, 89, 90, 91].
- Exhaustion or generalized fatigue.
- Total or partial abrupt cooling.
- Hemorrhoids, anal fissures (both the pathology and its surgical correction).
- Endometriosis.

9. DIAGNOSIS

Trigger points of the LE are deceptive and often go undetected. The discomfort they produce often leads to diagnostic errors because it is thought to stem from a different source.
To determine the cause of musculoskeletal pain, it is much safer to let oneself be guided by other features than the location of the discomfort and the hypersensitivity.
Diagnosis of myofascial pain is done by checking the clinical history, pain measurement, manual/digital examination of the musculature, and electromyographic findings [44].

9.1 Clinical history

Chronic or repetitive acute muscle overload is always involved in the onset of pain and always contributes to chronic pelvic pain.
The intensity of the pain depends on the posture or movement, and can be continuous when severe.

Pain (anorectal, perineal, in the penis, etc.), local dysfunction (muscle weakness due to hypertonia, loss of coordination), sleep disorders, etc.
The referred pain for each muscle was already discussed in the section "anatomical-clinical correlation" above.
The patientôs history might reveal a possible beginning in the prostate, progressing with pain in the urethra followed by increased urinary urgency and frequency, anal pain, lumbar pain, headaches, anxiety, stress, fatigue, and finally sexual dysfunction and depression.

9.2 Measuring pain

Pain can be measured with the frequently used visual analog scale (VAS).
The McGill questionnaire is reliable and valid to measure pain as a multidimensional experience because it assesses sensorial and affective aspects, as well as the intensity of pain.
The pain diagrams originally described by Travell and Simons are very useful because they accurately reflect the location and extension of the pain.

9.3 Physical examination

TrPs are identified through palpation, first superficial and then deep. In addition to the TrPs, the basal tonus of the thoracic diaphragm, the subumbilical abdominal wall, the pelvic floor, and lastly, the mobility and texture of the connective tissue in all these areas should be assessed. Finally, the standing posture must be assessed (symmetry of the folds, breathing, bone reference points, etc.).

- **Deep palpation:** When exploring the area in search for a TrP and the taut band around it, the following may be found: hyperirritability, immobility, tenderness, edema, tension, and muscle contracture.

The ñjump signò is typical, and constitutes a valuable indication of a TrP. All Berger tender points should be palpated (Figure 4).

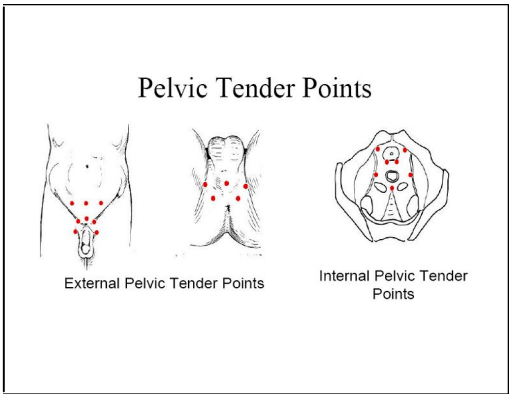


Figure 4

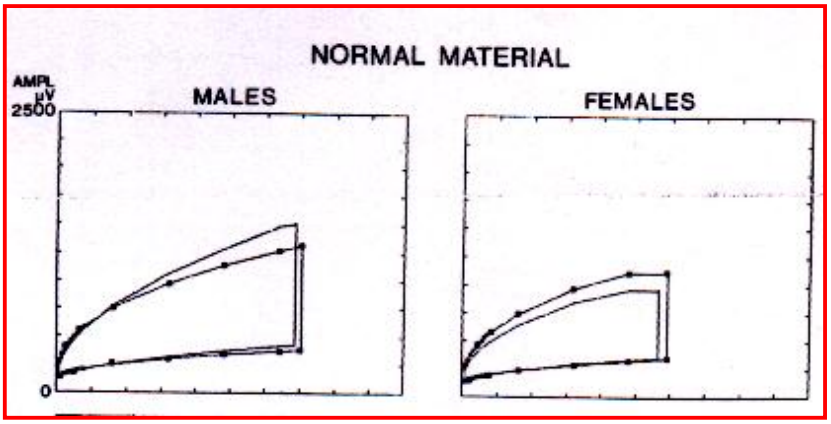
- **The administration of a muscle relaxant** two hours before enables a more accurate detection of active TrPs because it temporarily reduces the pain of secondary and satellite TrPs.
- **Diagnostic dry needling.** A needle is inserted into the TrP, causing a local twitch response (spasm). Very typical.
- **Local anesthetic block.** Local and referred pain disappear; as we shall see, this procedure can also be therapeutic.
- **Pressure using an algometer.** To measure the pain pressure threshold of the muscles having TrPs.

9.4 Electrophysiological studies

The electrodiagnostic features of the TrPs were first described by Weeks and by Travell in 1957. Hubbard and Berkoff reported a similar electrical activity in myofascial TrPs and according to them only high-frequency spike potentials are characteristic [77].
Later, Simons and Hong detected another component in the form of low-amplitude noise, which was always present. This noise was called spontaneous electrical activity [29, 94].

In our experience, it is common to find an increased basal muscular activity at rest which is related to the pathogenesis of the process; this can be quantified by averaging the turn/amplitude obtained in electromyographic analysis. Essentially, the number of turns of the EMG signal is measured during a unit of time, and the mean amplitude of the turns obtained during this period; the values are then compared to those obtained with healthy subjects.

Basically, the test consists of recording the EMG activity in various muscle sites (between 6 and 10), preferably in the area halfway between the motor point and the tendon. Each point represents the automatic analysis of a period or "epoch". Between 20 (minimum) and 30 tests are done. Under normal conditions, the points are distributed in a "cloud" shape, where 95% of the obtained points are found. When at least 10% of the points are outside the cloud, this is considered pathological.



This tool can be useful when assessing the progress of patients after several therapeutic interventions [119,120,121].

9.5 Thermography.

Thermographically, a TrP appears as a small area that has a temperature between 0.6 and 1°C higher than the surrounding tissue or its contralateral area [109,110,111].

10. Differential diagnosis

The three most common musculoskeletal disorders that require special attention are myofascial pain, fibrositis or fibromyalgia, and joint disorders.

For none of the three disorders there are radiological or laboratory tests that help to strengthen the diagnosis.

Therefore, diagnosis is made based on a thorough clinical history and a detailed physical examination, especially of the muscles.

Too often these three pathologies are misdiagnosed because the clinical history and physical examination are not carried out systematically.

To avoid this situation, the examiner should know exactly what to look for and develop the manual dexterity to find it. The examination should be guided by a presumptive diagnosis [117].

Confirmatory findings of Myofascial Syndrome

- Local twitch response induced by palpation or by inserting a needle through a TrP, and which is evidenced by detection of movement by sight, palpation, or ultrasound imaging.
- Referred pain patterns for each muscle.
- Motor endplate noise when an EMG needle is inserted into the TrP.

Inconveniences when palpating a trigger point

- Obesity.
- Interposed muscles.
- Interposed aponeurosis.
- Tense and deep subcutaneous tissue.
- Lack of palpatory skill.

11. TREATMENT

The fundamental principle of therapy is based on myofascial release through inactivation of the TrPs and muscle re-education.
Pain of musculoskeletal origin is more likely to be controlled if its cause is identified and corrected.
Effective treatment is difficult to attain and relatively slow. A multidisciplinary team is required for chronic pain syndromes with complications.
According to a study by Giubilei et al. [118], aerobic exercise seems to be beneficial for the recovery of patients with chronic pelvic pain. This must be evaluated in each case by the professionals who treat the patient; in early stages of treatment it is usually harmful. Recommendation of exercise must be individually tailored to each case.

11.1. Medical treatment

- Physical trauma during bicycling, labor, or urological, gynecological, or colpoproctological surgery.

- Inflammation of pelvic organs or structures: prostatitis, cystitis, urethritis, endometriosis, vaginitis, proctitis, hemorrhoids, or anal fissures.

11.1.1 Treatment of chronic bacterial prostatitis

Treatment with antibiotics: when, how, for whom?
Repetitive prostatic massage: when, how, for whom? According to Dr. Felicianoôs Manila protocol [115,116].
Intraprostatic injections: when, how, for whom? According to Dr.Guercini's protocol [114].

11.1.2 Infiltration with local anesthesia.

In a single-blind study comparing lidocaine, botulinum toxin, and dry needling to inactivate TrPs, Kamanli et al showed in 29 patients with myofascial pain that lidocaine injection is faster, more effective, and causes less discomfort than dry needling, and is more cost-effective than botulinum toxin [25]. Many other authors reach the same conclusions, especially regarding postoperative discomfort and greater therapeutic efficacy [45, 46, 64].

On the other hand, Langford et al injected a mixture of lidocaine, bupivacaine, and triamcinolone to treat TrPs in the levator ani muscle in 18 women. They obtained improvement in thirteen patients after the first injection (72%). Six women (33%) became completely pain-free. The authors were surprised by the high efficacy of the treatment and the underutilization by other professionals [26].

However, there is no evidence that corticoids combined with anesthetics improve the clinical response compared to local anesthetics alone [37]. Moreover, it is known that the repeated use of steroids can cause degenerative lesions and even rupture of the muscles [38, 39].

11.1.3. Corticosteroid infiltration in areas with enthesopathy or tendinitis.

Since myofascial pain syndrome involves muscle shortening, there will obviously be abnormalities in the areas of muscle insertion, be it tendinitis or enthesopathies. Kang et al used transanal infiltration with lidocaine and triamcinolone every two weeks for a maximum of three sessions. The mixture was injected in the most tender areas. There was significant improvement at 3ï 6 months of follow-up.

The authors concluded that the procedure is sufficiently simple, safe, and effective to be recommended as a first-line therapy [28].

11.1.4 Infiltration with botulinum toxin.

In Spain, we work with Botox or Botulinum toxin type A: it is a neurotoxin produced by Clostridium Botulinum. It works by inhibiting the release of acetylcholine at the neuromuscular junction, which results in the chemical denervation of the latter, thus paralyzing the treated muscle.

Botulinum toxin has been recognized by many authors as a good treatment for myofascial syndrome, and is used successfully in any area of the body [54]. It reduces the tone of contractured muscles [108].
In a pilot study, Sherin et al. injected 12 women suffering from chronic pelvic pain associated with spasm of the levator ani with Botox. Their results were promising, since they obtained alleviation of pain and a reduced hypertonicity [55, 56].

Other authors recommend the toxin only when other simpler measures, such as dry needling, have failed [57, 58, 59, 60, 61].
However, Göbel et al conducted a prospective, randomized, double-blind study about the safety of this treatment, and reported good tolerance, fast resolution of side effects, and significant improvement of pain 4ï 6 weeks after treatment [62].
Zermann et al. observed that pelvic and prostatic pain are accompanied by motor and sensory disorders, which led them to hypothesize that prostatic pain stems from a changed processing of afferent and efferent information in the central nervous system. They concluded that the injection of Botox around the urethral sphincter can lower its tone by blocking acetylcholine and interrupting negative efference from the CNS, leading to pain relief and improvement of the symptoms [63].

11.1.5 Acupuncture and electroacupuncture.

In their study on acupuncture, Chen and Nickel concluded that this is a safe, effective, and lasting method to improve symptoms and the quality of life of patients with pelvic pain. As a neuromodulatory and minimally invasive treatment, this is an option when traditional therapies fail [105,106,107].

11.1.6 Scar treatment

Abdominal, perineal, or lumbosacral scars can cause abdominal pain or pelvic pain and limit the mobility of muscles and fascias. Kuan et al treated 221 painful scars with a mixture of 0.5% bupivacaine, 2% lidocaine, and betamethasone infiltrated into the fibrous tissue, and obtained a high rate of success. The pain had

disappeared in 86.5% of the patients after three months of follow-up [27]. The procedure is supplemented with specific physical therapy techniques, both manual and instrumental (hyperthermia, laser, ultrasound).

11.2 Physiotherapeutic treatment

11.2.1 Physical therapy

Physical therapy consists fundamentally of analytical and global techniques: massage, stretching, release of TrPs by acupressure and dry needling, the release of mobility limitations in the fascias (myofascial induction techniques), and craniosacral rhythm regulation in cases with scars; when initial assessment determines a need, hypertonicity of the diaphragm and the abdominal musculature as the main regulators of the transmission of intra-abdominal pressure should be treated.
Time is a critical factor. The release of fibers is a slow process.
Clinical experience shows that slowly sustained stretches are much more effective than rapid stretches. Unfortunately, as soon as the muscles relax, the sarcomeres return to their initial state unless something more is done. We must continue with post-isometric relaxation or contract-relax. Contraction alone is an inadequate treatment. Gentle contraction must follow (10% of maximum). This would be contract-release or post-isometric relaxation and release [21].

For the rehabilitation of the short pelvic floor, Fitzgerald and Kotarinos recommend ten weekly one-hour sessions, and have been generally successful with the following regime [35, 36]:

1. Avoiding activities that may exacerbate the problem, such as Kegel exercises, abdominal exercises, and wearing tight clothes.
2. Correcting poor posture and recommending other more common ergonomic postures.
3. Detecting anatomical abnormalities such as a small pelvis.
4. Treatment of connective tissue disorders, especially panniculitis.
5. Treatment of abdominal and perineal scars that produce restrictions on normal mobility.
6. Treatment of extra-abdominal TrPs.
7. Stretching exercises of the abdominal muscles.
8. Release of the pelvic floor TrPs by means of pressure, post-isometric relaxation, proprioceptive neuromuscular facilitation, and reciprocal inhibition.
9. If required, anesthetic infiltration of the TrPs.
10. Pudendal blocks, if required.
11. Home maintenance programs of stretching exercises for the abdominal muscles and relaxation of the pelvic floor in a squatting position and pushing the knees (Figure 6).

Figure 6: Adductor (péctineo): Adductor (pectineus)
Glúteos: Glutes
Iliopsoas: Iliopsoas
Caudrado Lumbar: Quadratus Lumborum
Piriforme y rotadores laterales: Piriformis and lateral rotators
Recto abdominal: Rectus abdominis
Pelvis: Pelvis

11.2.2 Release methods

The immediate elongation of the muscle promotes equilibrium with respect to the length of the sarcomere; when this is done slowly, it helps to reconfigure the new sarcomere length, which thus tends to stabilize.
In any case, complete relaxation of the patient is a prerequisite for effective release.

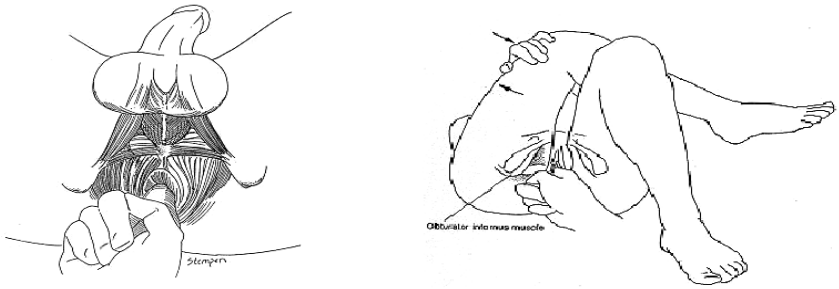
11.2.2.1 Release through pressure on the TrP

This consists of applying a gentle and gradually increasing pressure on the TrP until an increased tissue resistance is found by the finger.
This maneuver normally causes bearable pain. Pressure should be maintained until the clinician notices a reduction in the tension under the palpating finger.
At this point, the finger increases the pressure enough to reach the next barrier: the finger ñfollows the tissues that are being relaxedò. Again, the clinician sustains a gentle pressure until the tension yields under the finger [3,22,24].
Sexual dysfunction is common among patients with refractory pelvic pain; application of myofascial release and paradoxical relaxation is very helpful in the treatment of pelvic pain, urinary symptoms, libido, ejaculatory pain, erectile dysfunction, and postejaculatory pain [2].
Weiss reported successful outcomes in 52 patients with interstitial cystitis and urethral syndrome treated with manual therapy. About 70% and 83%, respectively,

experienced moderate or marked improvement. Electromyographically a decreased muscle tone was observed. Duration of the symptoms had been 6ï 14 years [51].

Acupressure of the levator ani and obturator internus muscles

Digital examination in men digital examination in women



TACTO MASCULINO **TACTO FEMENINO**

11.2.2.2 Release through dry needling

This TrP release method is increasingly popular among physical therapists and is also practiced by physicians. Its therapeutic usefulness has been recognized by such prestigious organizations as the Cochrane Collaboration [43].

On the other hand, the false popular and professional belief that Chinese acupuncture and medical acupuncture or dry needling are the same thing leads to error and confusion. However, there is a 71% overlap in the location of traditional acupuncture points and dry needling points [40, 41, 42].

Gunn et al. developed a method for deactivation of TrPs called intramuscular stimulation, which involves introducing a needle into the TrP as is done in dry needling [48]. This can be combined with electrical stimulation through the inserted needle.

A Korean study from 2007 seems to confirm the results of Gunn et al. and highlights the better results obtained with this technique compared with infiltration of 0.5% lidocaine [52].

11.3 Psychological treatment

Relaxation techniques: Jacobsonôs progressive relaxation, Wiseôs paradoxical relaxation, yoga, meditation, etc.
Dr. Wiseôs paradoxical technique combined with point release is proving to be a more effective method to alleviate pain and urinary dysfunctional symptoms than traditional methods. Thus, marked improvement is achieved in 72% of cases [1, 23].

12. Evolution and prognosis

Clear signs of improvement are seen in most cases after three or four months of treatment with medical and physical therapy, with two weekly one-hour sessions, a

daily program of specific stretches at home, body relaxation techniques, and stress management.
Up to two years may be necessary for a stable and permanent improvement of myofascial syndrome.

13. Conclusions

"Evidence based medicine is not ócookbook medicineô with a book where all formulas can be found. External clinical evidence can inform but can **never** replace individual clinical expertise, and it is this expertise that decides whether the external evidence is applied to the individual patient at all.ò David Sackett said this, and we must apply it to a complex problem that results in a large number of chronic disabilities, decreased quality of life, and much suffering among those who experience this condition.

The additive effect of patterns of constant pelvic tension, trauma, inflammation, or pelvic organ disease can overload the muscles and stimulate the development of TrPs and hypertonia of the pelvic floor.
Therapy should be aimed at total eradication of noxious stimuli transmitted from the body surface, viscera, trigger points, or mechanical alterations of the body to the sacral spine to allow periods free of noxious stimuli.

We must always keep in mind that a chronically shortened and hypertonic muscle will asphyxiate, catabolites will accumulate inside, and in time this can lead to degenerative histological changes. This is why the longer the duration of the dysfunction, the worse the prognosis for the muscle, the nerve, and the fascia. The key factor is time (one of the diagnostic errors is precisely not giving importance to myofascial dysfunction in very early stages and with very mild symptoms).

From the above, we should also conclude that early physiotherapy and neurotherapeutic treatment of postoperative scars is important, to prevent the development of myofascial dysfunction.
It should also be noted that patients with long-lasting myofascial pelvic floor dysfunction manifest complex sets of psychological symptoms (anxiety, depression).

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