addiction and psychosocial functioning (difficult to evaluate in patients who deny their abuse or who are not engaged in addictions treatment), possible overdosage in the presence of unaccustomed drug use, and precipitation of withdrawal through the use of medications such as pentazocine, which contain opiate antagonists. An example of a novel attempt to minimize such risks is the report by Kennedy and Crowley in 1990 of a pilot study of methadone maintenance for patients with chronic non malignant pain and addiction who responded to a treatment program that included weekly routine urinalysis; weekly psychotherapy; and the use of quarterly self-report tests of mood, pain, and function to evaluate change. A fourth goal of treatment is to reduce patient anxieties that are contributing to the experience of pain. Effective means of doing this include the use of adequate doses of opiates (which are higher for those who have been dependent on opiates) and of measures to enhance control when possible, such as the use of patient-controlled analgesia pumps, regularly scheduled versus as-needed medications for persistent pain, and the avoidance of an excessive focus on the details of pain medication. Pain medication should never be used as a bargaining tool.

Both Portnoy and the California Medical Association recently proposed guidelines for the prescription of controlled substances for the treatment of chronic pain, emphasizing the need for adequate evaluation, diagnosis, documentation, informed consent, objectives, periodic review, and modification of treatment. Adaptation of such guidelines to the treatment of patients with both chronic pain and addiction requires careful attention to the multiple needs of these complex patients, awareness of the emotional responses they engender in their caretakers, and effective collaboration among all of the clinicians caring for them.

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SECTION 5. GENERALIZED PAIN SYNDROMES

220. MYOFASCIAL PAIN SYNDROME

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Chronic and disabling pains are commonly of musculoskeletal origin. These may arise from a pathologic process involving the joints, muscles and their ligamentous attachments, or both. When pain seems to be arising from one or more joints and can be confirmed by appropriate radiologic studies and objective signs of localized inflammation, the diagnosis of arthritis can be easily established. Another common form of musculoskeletal pain is persistent, deep aching pain that is not localized to the joints. It is commonly labeled as myofascial pain syndrome. As the name implies, this is a syndrome that encompasses a spectrum of symptoms, primarily involving muscles and their ligamentous attachments. It can be persistent, severe, and disabling and afflicts women about five times more commonly than men. Often it arises from mild trauma or muscular overuse and, if not properly treated, can persist for prolonged periods of time. Frequently, this syndrome is known by other names, including fibromyalgia, fibrositis, muscular rheumatism, nonarticular rheumatism, and idiopathic myalgia, among others. Traditionally, it is subdivided into localized and diffuse myofascial pain syndromes. The International Association for the Study of Pain defines it as "diffuse, aching musculoskeletal pain associated with discrete predictable tender points and stiffness." These tender areas are commonly known as trigger points. They are considered of vital importance in maintaining the pain syndrome and, by analogy, in treating it. Simple techniques directed at disarming trigger points are the mainstay of therapy. They are easy to perform in the office setting once the diagnosis of myofascial pain syndrome has been established.

CLINICAL FEATURES

A thorough history and physical examination are absolutely essential in diagnosing myofascial pain syndrome. Patients frequently describe diffuse muscular pain. Most often it is deep, continuous, dull, and aching in character. Rarely is it reported as throbbing or burning. Often no inciting event is remembered

by the patient, but sometimes the pain begins abruptly, and an inciting event is precisely remembered. Mild trauma such as a whiplash injury from a motor vehicle accident may be noted. Simple increased physical activity, often found in "weekend athletes," can be the inciting event. Even increased office hours with resultant strain on back musculature from prolonged desk and computer work under the stress of approaching tests or deadlines can incite myofascial pain syndrome. Muscle spasm can be prominent, as can radiation of pain. Although most frequently located in the trunk and neck muscles, any muscle or group of muscles can be involved. The pain is frequently increased by stress; inadequate sleep; fatigue; and cold, humid weather. Continued use of the involved muscle group tends to aggravate the pain. Stiffness is an important feature, which is worse in the morning. Chronic exhaustion is a prominent symptom and may be associated with restless and unrefreshing sleep.

On physical examination, diffuse tenderness may be noted. Discrete areas of point tenderness overlying muscles and their ligaments are found. Although the patient is usually unaware of the presence and location of trigger points, they are exquisitely tender to palpation. Often they are approximately the size of the examiner's finger pad. A small lump of contracted muscle may be felt. These trigger points are usually located within taut bands of muscle, which can be rolled between the examiner's fingers. Usually, a "jump sign" will be elicited as the trigger point is palpated, and the patient may cry out and move away as the trigger point is located. A local muscle twitch is usually felt and occasionally seen during palpation of a trigger point. Reactive hyperemia to palpation may be seen, most commonly on the trunk. These trigger points are usually within the region of the patient's reported pain, but sometimes are not. Palpation of true trigger points, however, should reproduce the patient's typical pain. Well known patterns of radiation have been described. For example, trigger points over the neck and scapula tend to cause pain in the ipsilateral shoulder and arm. Those of the flank tend to cause pain in the ipsilateral buttock and those of the buttock, into the posterior thigh and calf. The radiation of pain should be regional and nondermatomal in nature. If the radiation pattern of peripheral nerve or root is observed, other causes such as herniated disc or nerve lesion should be sought out. It should be realized that the presence of trigger points is an essential feature of this syndrome but is not sufficient for the diagnosis of myofascial pain syndrome. Based on studies involving healthy and asymptomatic volunteers, up to 50 percent of the general population has been estimated to harbor latent trigger points, which are difficult to distinguish from true myofascial trigger points in patients suffering from myofascial pain. It is conceivable that a significant portion of the general population that harbors latent trigger points may be at increased risk for developing myofascial pain syndrome. These latent trigger points do not cause spontaneous pain at rest or with movement, as opposed to active trigger points. They are, however, painful to palpation, and they do cause radiation to typical reference areas. Latent trigger points may be activated by jerky movements or muscle overuse. They may persist for years after an episode of transient myofascial pain that resolves over time, leaving patients susceptible to painful relapse.

The appearance of the affected region tends to be normal but can occasionally show mild swelling and other signs of muscle spasm, particularly in the neck and shoulder region. There usually is no evidence of muscle wasting, although long-standing disuse of an extremity secondary to pain can be a cause of weakness, decreased range of motion, and atrophy. Heat and cold intolerance is not prominent. There should not be evidence of true allodynia (pain caused by a stimulus, e.g., light touch, which normally does not produce pain). If trophic changes and allodynia are present, a component of neuropathic and/or sympathetically maintained pain may be present, which may require further evaluation (see Ch. 222).

Myofascial pain syndrome is a common occurrence in pain clinic populations and is usually an easy and straightforward diagnosis, but many practicing physicians are not familiar with this syndrome. This, is at least partly due to the absence of objective signs, normal radiologic studies, and lack of any diagnostic laboratory tests in myofascial pain syndrome. This is further complicated by the fact that it occurs in varying intensity, duration, and location and cannot be accurately diagnosed unless the affected muscles are properly examined. The following case studies from our own clinical experience illustrate some key points about myofascial pain syndrome. Readers are encouraged to consult references at the end of this chapter for a detailed review of this topic.

CASE STUDIES

Case Study 1

A 27-year-old woman was referred for evaluation with a 6-month history of severe, daily headaches involving the right side of her head after hitting her head against a window while at work. She had no immediate symptoms, and it was not considered an extraordinary incident. Gradually, over the next few days, she developed worsening headache, which did not respond satisfactorily to the Fiorinal (Sandoz, E. Hanover, NJ) prescribed by her primary care physician. The headaches continued and she was referred to a neurologist. After a neurologic evaluation and a normal computed tomographic scan of her brain, a diagnosis of "post-traumatic headache" was established. Daily amitriptyline in increasing doses was prescribed, and the patient was instructed to use acetaminophen or ibuprofen on an "as-needed" basis. At the time of her evaluation, she reported improved sleep with a single night-time dose of 125 mg of amitriptyline but continued to complain of daily headache during the day. She also reported worsening of pain on chewing and combing her hair.

Physical examination showed a healthy and fit-looking young woman complaining of right-sided headache. Her neurologic examination was completely normal but she had an exquisitely tender point in her right temporalis muscle (Fig. 220-1). Palpation of that "trigger point" reproduced her usual pain.

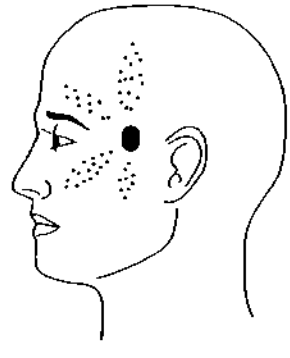

FIG. 220-1. The pattern of the myofascial pain syndrome in patient 1. The square represents the trigger point in the temporalis muscle, and the zones of stippling mark the areas of referred pain.

She initially declined to have an injection but could not tolerate occupational therapy manipulation. The tender spot was infiltrated with a 6-ml mixture of 1 percent lidocaine and 0.25 percent bupivacaine, which gave her lasting relief from headaches. Amitriptyline was slowly tapered.

Case Study 2

A manual laborer, aged 36, was referred for an evaluation. He had suffered for 8 months from low-back pain, radiating to his right hip and posterior thigh area. He could not recall a single precipitating event but remembered gradual worsening of his pain. He was referred to a local pain specialist who made a diagnosis of "lumbosacral radiculopathy" after a magnetic resonance imaging (MRI) scan of his spine showed disc bulges at L_4-L_5 and L_5-S_1 levels. The patient received two epidural steroid injections and bed rest over a 6-week period with partial and transient relief. His symptoms worsened, and he could not continue working. Physical examination showed a well-appearing and muscular man who walked with a slight limp. Neurologic examination was normal, but he had two tender spots in the quadratus lumborum muscle and one tender spot over the piriformis muscle (Fig. 220-2). Palpation over these trigger points reproduced his symptoms. The trigger points were infiltrated with a 20-ml mixture of 1 percent lidocaine, 0.25 percent bupivacaine, and 40 mg of triamcinolone, and he was referred for physical therapy. He was able to return to work in 2 months, during which he required repeat trigger point injections once and a course of physical therapy.

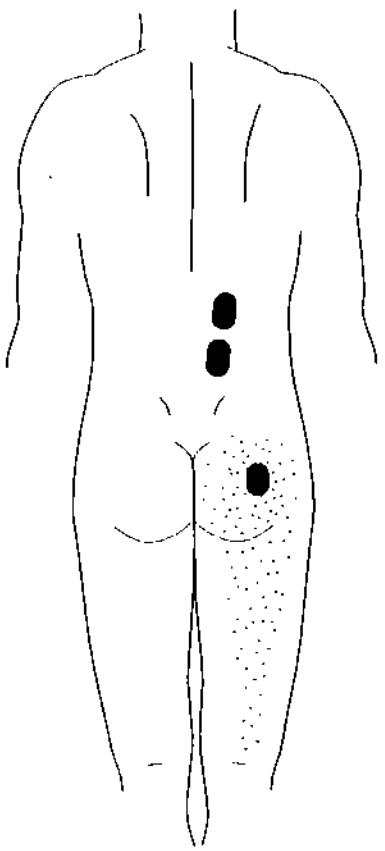

FIG. 220-2. The pattern of the myofascial pain syndrome in patient 2. The squares represent the trigger points in the quadratus lumborum and piriformis muscles, and the zones of stippling mark the areas of referred pain.

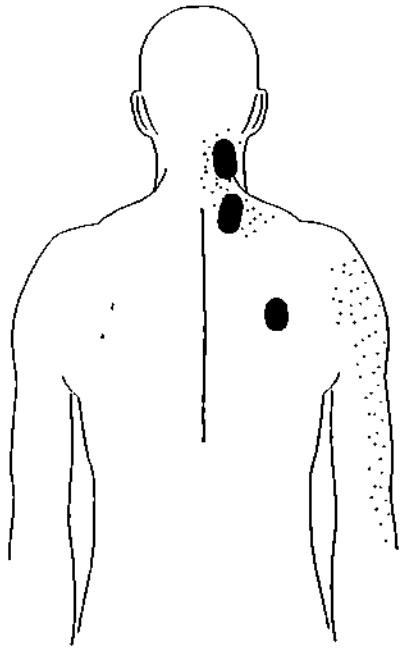

FIG. 220-3. The pattern of the myofascial pain syndrome in patient 3. The squares represent the trigger points in the levator scapulae and infraspinatus muscles, and the zones of stippling mark the areas of referred pain.

Case Study 3

A 29-year-old painter complained of progressive worsening of shoulder and neck pain, radiating to his right arm over a 3-month period. The pain was described as continuous, deep, and aching and was aggravated by any use of the right arm. He also complained of neck stiffness and had acute exacerbations of pain on any movement of his neck. He had well-defined trigger points in his right neck and scapular regions (Fig. 220-3). Palpation of these points produced referred pain in his neck, shoulder, and right arm. Over a 3-week period, he received multiple trigger point injections with local anesthetic and physical therapy three times per week. He reported significant pain relief for up to 3 to 4 hours after each session of trigger point injections followed by physical therapy. At this point, he was referred to our pain clinic. Physical examination showed a well-appearing muscular man, holding his head flexed to the right. All neck movements were greatly limited by pain. He did have two trigger points (Fig. 220-3). Palpation over these reproduced his typical referred pain but not numbness and the tingling sensation that was also felt in his hand and forearm on sudden movement of the neck. His neurologic examination was otherwise normal. We performed trigger point injections using long-acting local anesthetic and steroids in an attempt to provide him with long-lasting relief but also scheduled him to have an MRI scan of his cervical spine. This time, injections provided pain relief for 2 days. MRI scan showed a C6–C7 disc herniation impinging on the right neural foramen without other significant abnormalities. His pain finally responded to a combination of cervical traction, epidural steroid injections, and physical therapy.

PATHOPHYSIOLOGY

The exact mechanism of myofascial pain syndrome remains uncertain. Kellgren, who is credited for his pioneering work in this syndrome, demonstrated that injecting a particular muscle

with an irritating solution consistently produces pain, which is felt over a much larger region than the muscle injected. He described classic cases of myofascial pain with trigger points that referred pain in the same pattern as in his earlier experiments. A contracted, hyperirritable spot in the muscle, known as a trigger point, is an integral part of this syndrome. How it is formed and maintained remains unclear. It is believed that overuse of deconditioned muscle and acute trauma contribute to formation and activation of trigger points. It has been speculated that trauma or muscle overload causes microscopic tissue injury involving sarcoplasmic reticulum. There is a resultant release of tissue humoral factors, which cause inflammation and pain. Local instability of calcium channels may occur. Increased levels of extracellular calcium may play a part in initiating and maintaining local muscle spasm. This produces a region of local vasoconstriction, ischemia, and uncontrolled metabolism within the muscles. Central and sympathetic nervous system reflexes may also be involved, leading to persistent inflammation, spasm, and pain. This process results in shortened muscle fibers in an area of increased metabolism and decreased circulation, which are palpable as a taut band in the muscle and give rise to the phenomena typically associated with palpation of trigger points.

After muscle injury, afferent impulses from damaged tissue bombard the central nervous system. Stress response systems intercede, leading to increased sympathetic activity in the affected muscle. Generalized fatigue and anxiety feed into this system. Local vasomotor changes occur. An area of increased metabolic demand secondary to continued spasm coupled with a decreased blood supply caused by sympathetic response develops. Local ischemia results in further release of humoral factors such as histamine, serotonin, kinins, and prostaglandins. A vicious cycle of local trigger point muscle spasm ensues, leading to vasomotor constriction with resultant continued pain and inflammation. This cycle can perpetuate itself long after the inciting event.

DIAGNOSIS

The diagnosis of myofascial pain syndrome is based on an extensive history and physical examination. Often a recent or remote episode of tissue trauma is elicited. A contralateral injury may be found to cause asymmetric body mechanics with resultant muscular stress of the painful muscle groups. Chronic muscle overload may be found. If a history of trauma is lacking and the pain has been gradual in onset, a thorough exploration into the patient's personal life is warranted. Significant emotional stressors should be elicited. Sleep patterns are important, as exhaustion can be a cause of myofascial pain syndrome in susceptible people. Depression is frequently found in these patients. Whether depression is a cause of this illness or rather a symptom is controversial. Certainly, any emotional derangement augments the pain cycle.

The pain may have been present for months to years and is frequently debilitating. Many patients are out of work secondary to pain. A complete list of previous medication trials and therapies should be sought. Radiation of pain is typical but should be regional and nondermatomal in character. History of paresthesia and numbness should be absent.

Trigger points are pathognomonic for myofascial pain syndrome. Their inactivation with resultant long-lasting relief of pain should therefore be considered diagnostic. Palpation for typical trigger points should be performed. These may be outside of the patient's described area of pain. Upon palpation, however, the typical referred pain will be elicited. Local tenderness is not considered a trigger point. Frequently a jump response is obtained. As mentioned, a taut band of muscle with a discrete trigger point within it may be felt as the muscle is rolled between the examiner's fingers. Reactive hyperemia may be found after trigger point palpation. Each trigger point should be labeled with a skin marker for later treatment. Deep tendon reflexes and sensory examination should be normal. Motor function may be diminished in patients with long-standing myofascial pain syndrome. Thermography of the skin overlying trigger points may show increased temperature secondary to the increased metabolism of a muscle in spasm, but that is a nonspecific finding and is not required to make this diagnosis. In fact, there are no radiographic or laboratory test abnormalities that are considered diagnostic of myofascial pain syndrome. Some cases of polymyalgia rheumatica, other inflammatory muscle diseases, and rheumatologic disorders may mimic myofascial pain syndrome. In those patients, erythrocyte sedimentation rate, muscle enzyme levels, antinuclear antibody, rheumatoid factor, and so forth become essential screening tools, and further management plans depend on the results of these tests. For all practical purposes, it is essentially a clinical diagnosis and sometimes a diagnosis of exclusion.

TREATMENT

The treatment of myofascial pain syndrome generally involves a multimodality approach. Inactivation of trigger points with local anesthetic injections, dry needling, and stretch and spray techniques form the mainstay of therapy. Physical therapy, transcutaneous electrical nerve stimulation, and massage therapy have also been used. Pharmacologic trials of anti-inflammatory medications are reported to help some patients in combination with other modalities. Biofeedback and relaxation therapy have also been used.

We most frequently use local anesthetic injections of trigger points with or without steroids. Steroids are generally most useful when hyperemia is present or there is history of prolonged pain after injection and should not be used frequently. Trigger points are located and marked during the physical examination. The skin overlying the trigger points is prepared with alcohol or povidone-iodine. A mixture of 20 to 40 mg of triamcinolone in 10 ml of 0.25 percent bupivacaine is generally used. The total volume of the solution depends on the size of the muscles involved and the number of trigger points to be injected. If a shorter duration of action is desired 1 to 1.5 percent lidocaine can be substituted for bupivacaine. A 1.5-inch 25-g needle on a 12-ml syringe is typically used. The trigger point is isolated between the physician's first and second fingers. The needle is introduced into the muscle after anesthetizing the skin overlying the trigger point. Often a local muscle twitch is felt and occasionally seen when the needle enters the trigger point, and the patient's typical referred pain may be reproduced. A total volume of 3 to 5 ml of local anesthetic is typically placed within the trigger point. Then the needle is withdrawn almost to the surface and redirected to the same depth, injecting other areas of the muscle surrounding the original injection site, thereby ensuring complete inactivation of the trigger point. Shortly after injection of each trigger point, the patient's pain should resolve or become markedly diminished, and muscle spasm should abate. Dry needling can be performed in a similar manner without the aid of local anesthetic, but we do not recom-

mend dry needling because it is extremely painful and without any clear benefit over local anesthetic injections.

Stretch and spray can be performed when injection therapy is undesirable or contraindicated. The patient is placed in a comfortable position. The muscle to be treated is sprayed with a vapocoolant, such as ethyl chloride or chlorofluoromethane starting at the skin overlying the trigger point and continuing in the direction of the referred pain. The muscle is then very carefully stretched through its normal range of motion. This sequence of spray and stretch can be repeated a few times before rewarming. The involved muscle must reach its full stretch length in order to inactivate trigger points. This technique is particularly useful and preferred over trigger point injections in patients with diffuse myofascial pain.

Regardless of the technique chosen for trigger point inactivation, the patient should note prolonged pain relief. The relief should far outlast the duration of local anesthetic action. Frequently, patients may note prolonged pain relief after only one set of injections. Sometimes, however, a series of injections is performed as the effects of the last injections start to wane. Usually the interval between symptomatic recurrence will increase until the patient's myofascial pain syndrome has resolved. This may require a series of three to six sets of trigger point injections over a few weeks to months.

It is often useful to have patients treated with physical therapy after and between trigger point injections. Passive stretching and massage treatments can be instituted. Gradual increase in activity and gentle graduated exercise programs complement trigger point inactivation. Care must be used not to strain already compromised muscle groups. Especially vigorous physical therapy can aggravate the syndrome and often will alienate patients who either give up or go elsewhere for treatment. As patients improve, general exercise and body conditioning programs should be instituted. Transcutaneous electrical nerve stimulation may also be used during physical therapy. If patients respond, a portable unit may be taken home on a trial basis and later purchased or rented if effective.

Nonsteroidal anti-inflammatory medications are sometimes useful adjuncts in treating myofascial pain syndrome. They help reduce inflamation and are useful in treating the inherent soreness patients exhibit after the local anesthetic action of trigger point therapy abates. They are generally considered safe and offer the advantage of being nonaddicting. Other useful medications include tricyclic antidepressant medications. At relatively low doses, they certainly help with sleep and may, with time, help diminish pain. We would typically use amitriptyline or nortriptyline if anticholinergic side effects are especially undesirable. Ten- to 25-mg starting doses are generally well tolerated. This dose can then be titrated by the patient under strict guidelines every 4 to 7 days by the same starting increment. It must be remembered that nortriptyline is about twice as potent as amitriptyline and other tricyclic antidepressants commonly prescribed for chronic pain. Pain tends to improve within several days after reaching adequate dose of the medication with the maximum benefit found over weeks. Most patients respond to doses between 25 and 100 mg, although some may require much higher doses for effect. An adequate trial of these medications would require at least a few weeks of therapy at the highest dose tolerated. Biofeedback and relaxation techniques also afford significant pain relief in selected patients and should be considered as adjunct therapies.

PROGNOSIS

As with most pain syndromes, the prognosis is dependent on the chronicity of the pain. Patients with myofascial pain of short duration often respond to just a few trigger point injections. These patients tend still to be working and leading active, albeit dramatically modified lives. As the chronicity of the pain syndrome increases so, too, does the period of time needed to treat it and the modalities incorporated in its treatment. Frequently these patients are out of work and depressed. They might benefit from the antidepressant action of the tricyclic medications as much as from the pain relieving quality of it. Certainly physical therapy and psychological counseling with relaxation technique training is invaluable. With weeks to months of interdisciplinary therapy, these chronic pain sufferers tend to respond slowly.

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221. ARTHRITIC PAIN

SIMON M. HELFGOTT

Arthritic pain is one of the most common subjective complaints encountered in clinical medicine. The term is often used synonymously with *musculoskeletal pain*, although, in fact, arthritic pain is only one of several types of pain related to the musculoskeletal system (Table 221-1). Arthritis pain must be distinguished from other causes of musculoskeletal pain to establish an accurate diagnosis and initiate appropriate treatment. Some clinicians use the term *arthritis pain* as a diagnostic label. As there are more than 100 disease states associated with arthritis, it is important to avoid this practice.

EVALUATION OF MUSCULOSKELETAL PAIN

Arthritis implies joint inflammation and the associated clinical features, including joint swelling, tenderness on palpation, limitation of motion, and warmth. Other types of musculoskeletal