Fibromyalgia Syndrome

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Differential diagnosis: myofascial pain syndrome

CHAPTER CONTENTS

Jan Dommerholt and Tamer S. Issa

According to the American College of Rheumatology (ACR) criteria for the classification of fibromyalgia, a diagnosis of fibromyalgia (FMS) should be made when an individual presents with widespread pain lasting longer than 3 months and tests positive for the tender point count. Several associated conditions have been described and multiple aetiological hypotheses have been developed. Even though the ACR criteria are widely used, it remains debatable whether the tender point count is specific enough for diagnosing FMS, particularly in the presence of other clinical entities that may also feature many of the same symptoms (Dommerholt 2002, Gerwin 1999). One could argue that in these cases making the diagnosis of FMS may disregard the commonly applied medical differential diagnostic process. This chapter will highlight several other pain syndromes that should be considered in the differential diagnosis of FMS with special attention to the diagnosis of myofascial pain syndrome (MPS).

THE AMERICAN COLLEGE OF RHEUMATOLOGY CRITERIA

Following the 1990 publication of the ACR criteria, more than 1000 peer-reviewed articles on the subject of FMS have been published (Russell 2001, Wolfe et al 1990). In spite of the attention

FMS has received in both the popular and academic press, the diagnosis of FMS continues to be surrounded by controversy. The ACR criteria were deliberately referred to as ‘classification’ criteria to distinguish them from ‘diagnostic’ criteria. The term ‘classification’ was used to represent the minimal standard for entry of subjects into research and epidemiologic studies; however, the ACR criteria suggested that the criteria may be useful for clinical diagnosis as well (Wolfe et al 1990). A consensus document developed during the 1992 Second World Congress on Myofascial Pain and Fibromyalgia in Copenhagen supported using the ACR criteria as diagnostic criteria, even in absence of the required number of tender points (Copenhagen Declaration 1993). According to the Copenhagen Declaration, strict adherence to the tender point count is indicated in research protocols. However, when the ACR criteria are used as diagnostic criteria, the diagnosis of FMS can be made with fewer than 11 tender points, a sentiment later expressed in several other publications (Bennett 1999, Copenhagen Declaration 1993, Wolfe et al 1995). Following the publication of the ACR criteria and the Copenhagen Declaration, physicians and other health care providers worldwide started applying the classification criteria diagnostically in their clinical practices. Compared to other medical specialists, rheumatologists most frequently make the diagnosis (White et al 2000b).

Because the ACR criteria include only two parameters, namely widespread tenderness and duration of symptoms, there are potential pitfalls clinicians should be aware of when considering the diagnosis of FMS. First, the ACR criteria associate a single non-specific clinical feature, such as tenderness, with an entire pain syndrome and fail to distinguish between cause and effect (Cohen & Quintner 1993). Although several studies have confirmed the validity and inter-observer and intra-observer reliability of the tender point count, that does not necessarily mean that it is a valid tool to characterize a specific syndrome (Okifuji et al 1997, Tunks et al 1988, 1995). It has been established that a high number of tender points may depict a more general measure of distress, more somatic symptoms, more severe fatigue, and low levels of self-care. The use of the tender point count itself remains highly subjective (Croft 2000, Jacobs et al 1996, McBeth et al 1999, Wolfe 2000). MacFarlane and colleagues suggested a refinement of the ACR criteria with the revised Manchester criteria. The Manchester criteria require that pain is more genuinely diffuse and were found to be more strongly related to several associated features such as tenderness, fatigue and psychological distress (Macfarlane et al 1996).

Second, there is a substantial risk of circular reasoning. After patients have been diagnosed with FMS using the tender point count, they may still wonder why they have pain. Invariably, the clinician will answer something like ‘you have pain, because you have FMS’. This circular reasoning basically implies that patients have pain, because they have pain. By not distinguishing between cause and effect, circular reasoning is inevitable (Cohen & Quintner 1998).

Third, tenderness assessed by the tender point count does not distinguish a particular clinical entity, but may be an indication of allodynia, hyperalgesia, peripheral and central sensitization (Croft et al 1996, Graven-Nielsen et al 1999, Henriksson 2002). Central sensitization is not specific to FMS and is commonly seen with other chronic pain syndromes, including MPS, spinal cord injuries, burn injuries, post-herpetic neuralgia, phantom limb pain, trigeminal neuralgia, back and neck pain, whiplash associated disorders, temporomandibular pain, headache, etc. (Coderre et al 1993; Eide 2000; Eide et al 1994, 1996; Eide & Rabben 1998; Kavanagh et al 1991; Koelbaek Johansen et al 1999; Mense & Hoheisel 1999; Okifuji et al 1999b; Sessle et al 1999). There is much evidence that most chronic pain states feature a combination of central and peripheral mechanisms. Neuroimaging studies of patients with various chronic pain syndromes have shown similar alterations in functional brain activity independent of the specific diagnosis, that may contribute to allodynia, tenderness and other abnormal pain experiences (Bradley et al 2000, Bushnell et al 2002, Grachev et al 2000). The word fibromyalgia itself suggests that the pain would be limited to fibrous and muscular tissues, which is not the case. In patients with widespread pain, usually all tissues are equally painful (Vecchiet et al 1994). There is no evidence of any peripheral FMS-specific aberrations (Dommerholt 2000, Henriksson et al 1993,

Mengshoel 1998, Nørregaard et al 1994, Schrøder et al 1993, Simms 1996, Simms et al 1994, Yunus et al 1989).

One of the commonly described symptoms of FMS are sleep disturbances, even though impaired sleep patterns are not part of the ACR criteria. Sleep disturbances or insomnia are commonly observed not only in persons diagnosed with FMS, but also in healthy subjects, in persons diagnosed with AIDS, osteoarthritis, rheumatoid arthritis, MPS, depression, restless leg syndrome and obstructive sleep apnoea (Hirsch et al 1994, Korszun 2000, Kubicki et al 1989, Moldofsky et al 1987, Schneider-Helmert et al 2001, Scudds et al 1989, Von Korff & Simon 1996). It is often thought that persons with FMS have a disturbed sleep pattern with a characteristic alpha-delta anomaly or an interruption of the slow wave sleep phase. However, several recent studies do not always support this conclusion. The alpha-delta sleep anomaly was found in only one-third of persons diagnosed with fibromyalgia (Carette et al 1995). Clinicians usually consider insomnia a consequence of pain and patients also assume that they are awakened by night time pain, yet, in one study the type and degree of insomnia was equal in persons with chronic pain as in persons with primary insomnia, suggesting that night time pain may not be causally related at all to a lack of delta sleep and severe fragmentation of sleep (Schneider-Helmert et al 2001). Many of the secondary symptoms of FMS, including cognitive dysfunction, fatigue and poor attention span can be explained by insomnia, but are not specific either (Schneider-Helmert et al 2001). (See Ch. 3 for more on sleep and FMS.)

The most commonly cited neuro-endocrine and hormonal abnormalities in FMS include elevated levels of substance P and nerve growth factor and deficiencies of serotonin, growth hormone, cortisol, and dehydroepiandrosterone (Ackenheil 1998, Bennett 1998b, Crofford 1998, Dessein et al 1999, Giovengo et al 1999, Russell 1999a, Schwarz et al 1999, Værøy et al 1988, Wolfe et al 1997b). In spite of these studies, it is premature to conclude that the reported abnormal levels are specific for FMS. A recent study did not identify any significant differences in plasma tryptophan levels in patients with fibromyalgia (Maes et al 2000). Tryptophan is the metabolic precursor to serotonin. It has been established that serotonin, nerve growth factor and substance P are involved in the spinal regulation of nociception and one would expect disturbances in most chronic pain conditions (Coderre et al 1993, Eide & Hole 1991, Giovengo et al 1999, Yaksh et al 1999). High spinal cord levels of substance P and low levels of serotonin have indeed been reported in many chronic pain problems, including headache, low back pain, cervical pain, polyneuropathy, lumbo-ischialgia, osteoarthrosis, rheumatoid arthritis, etc. (Atkinson et al 1999, Cramer et al 1988, Stratz & Muller 2000, Stratz et al 1993). German researchers described high titres of specific antibodies to serotonin, which would suggest that an autoimmune process might contribute to the low levels of serotonin, but two subsequent studies were not able to demonstrate consistent antibodies to serotonin (Berg & Klein 1994, Klein & Berg 1995, Russell et al 1995, Vedder & Bennett 1995).

Last, in cases where a treatable medical diagnosis can be identified, it is questionable if and how patients benefit from an additional diagnosis of FMS. The ACR criteria suggest that the diagnosis of FMS is ‘a diagnosis by inclusion’. Dr I. Jon Russell, a foremost researcher of FMS, argues that the diagnosis of FMS should be made irrespective of other diagnoses and advocates the avoidance of physicians who believe that FMS is a diagnosis by exclusion (Russell 2001). Yet, there is an inherent risk in making the diagnosis of FMS by inclusion, especially when the majority of symptoms in a particular individual can be traced back to other medical conditions that feature similar symptoms, including widespread pain, sleeplessness and fatigue. Schneider & Brady refer to this category of patients as ‘pseudo FMS’ patients, or patients who were misdiagnosed with FMS (Schneider & Brady 2001). Among the diagnoses that may feature widespread pain and a positive FMS tender point count are hypothyroidism, adult growth hormone deficiency, other metabolic insufficiencies, MPS, myalgias secondary to medication use, parasitic infestations, myoadenylate deaminase deficiency, other rheumatic diseases, psychological diagnoses, hypermobility syndrome, whiplash syndrome and even widespread burns (Table 8.1) (Dommerholt 2001). There is evidence that the mere diagnosis of FMS may contribute to feelings

of hopelessness, depression, anger, anxiety and illness behaviour. Clinicians should be cautious with giving patients the diagnosis of FMS (Hadler 1996, Hellström et al 1999).

Table 8.1 Differential diagnosis of fibromyalgia syndrome

Fibromyalgia
Hypothyroidism
Adult growth hormone deficiency
Metabolic insufficiencies
Myofascial pain syndrome
Myalgias secondary to medication use
Parasitic infestations
Myoadenylate deaminase deficiency
Rheumatic diseases
Psychological diagnoses
Hypermobility syndrome
Whiplash syndrome
Widespread burns

When a diagnosis of FMS is made by inclusion, other diagnoses may fail to be identified; this potentially could result in withholding appropriate treatment options from the patient (Poduri & Gibson 1995). For example, when a cardiologist prescribes cholesterol-lowering medication, patients may develop widespread myalgia as a side-effect of the medication. All cholesterol-lowering medications in the so-called 'statin' family have widespread myalgia as a potential side-effect (Black et al 1998). The patient may not realize that the drug may be responsible for the relatively sudden onset of widespread muscle pain and may consult a general practitioner, rheumatologist or physiatrist, instead of the cardiologist who prescribed the drug. The patient may be diagnosed with FMS if the physician is not familiar with the potential side-effects of cholesterol-lowering medications, or if the physician follows the principle of a diagnosis by inclusion. It is safe to assume that the symptoms that are now ascribed to FMS will continue as long as the patient continues to take the cholesterol-lowering medication.

DIFFERENTIAL DIAGNOSES

In spite of the notion that, according to the ACR criteria, a diagnosis of FMS should be made irrespective of other diagnoses, a more logical approach would dictate following the commonly used medical differential diagnostic process and excluding other potential causes of widespread pain, fatigue and psychosocial distress. It is important to realize that even though the ACR criteria are used worldwide, similar criteria were developed in Germany and Switzerland. The German criteria feature more points at slightly different anatomical locations (24 vs 18), require less pressure to positively identify a tender point (2kg vs 4kg) and do consider other symptoms, such as autonomic features and functional limitations (Müller & Lautenschläger 1990a, 1990b; Müller & Müller 1991; Schmidt 1991). The German criteria consider the diagnosis of FMS a 'diagnosis by exclusion' and recommend making the diagnosis only after excluding all other dia-gnoses (Müller & Lautenschläger 1990a). A brief review of some common causes of widespread pain and associated symptoms pertinent for the differential diagnosis follows:

Hypothyroidism

Hypothyroidism is suspected clinically when there is a complaint of coldness, dry skin or dry hair, constipation, and fatigue. Hypothyroidism is commonly associated with widespread pain and in one study occurred in 10% of chronic MPS subjects with widespread myofascial trigger points (MTrPs) (Gerwin 1995). The thyroid-stimulating hormone (TSH) level may only be in the upper

range of normal, but, as shown by TRH stimulation tests, may still be abnormal for a given individual. Patients with hypothyroidism are commonly managed with medications such as levothyroxine (Singh et al 2000, Woeber 2000). However, not all tissues are equally able to convert thyroxine to triiodothyronine, the active form of thyroid hormone. The addition of triidothyronine to thyroxine has been shown to result in an improved sense of well-being, an improvement in cognitive function and mood, and an increase in serum levels of sex-hormone-binding globulins, a sensitive marker of thyroid hormone function (Bunevicius et al 1999; Bunevicius & Prange 2000). For more information about thyroid dysfunction, see Chapter 10 of this book, where John Lowe and Gina Honeyman-Lowe have provided an excellent review of their metabolic approach of patients with chronic widespread pain. For an even more in-depth review the reader is referred to Dr Lowe's comprehensive textbook on the subject (Lowe 2000). Since the clinical features of FMS and hypothyroidism are so similar, there is no real advantage to diagnosing patients with FMS as well, once hypothyroidism has been established.

Side-effects of statins

In patients with initial complaints of widespread pain a few weeks after they started taking any of the statin drugs, the cholesterol-lowering medications could be responsible for the pain complaint. These patients can be successfully treated by reducing the dosage of the medication or by switching to another cholesterol-lowering drug. When patients present with widespread pain after having started new medications, or after altering the dosage of current medications, a diagnosis of FMS is not indicated. Rather, these patients should be diagnosed with side-effects of medication use.

Parasitic disease

Parasitic infestations, such as amoebiasis, fascioliasis or giardia, can cause or contribute to widespread pain. Fascioliasis is perhaps the least known parasitic disease in this category even though it is endemic in 61 countries, including Thailand, Bolivia, Ecuador, Peru, Cuba, Egypt and Iran. It is also seen in many European countries, including France, Spain, Germany and Turkey. Fascioliasis is a common infectious disease of domestic herbivores, such as cattle, sheep and goats, due to liver flukes. Occasionally, humans can become a host especially in areas where sheep and cattle are raised and where humans consume raw watercress or other aquatic vegetables, such as kjosco and water caltrop (Laird & Boray 1992, Sapunar et al 1992). The World Health Organization estimates that 2.4 million people are infected worldwide, with at least 180 million at risk of infection. De Gorgolas and colleagues reported that the most common symptoms of fascioliasis were fever (83%), abdominal pain (100%), weight loss (83%), and generalized myalgia and joint pain (67%) (de Gorgolas et al 1992). Most parasitic infestations can be treated effectively with medications such as triclabendazole or praziquantel, eliminating the symptoms perhaps attributed to FMS (de Gorgolas et al 1992, Jamaiah & Shekhar 1999, Mannstadt et al 2000, Qureshi et al 1997, Richter et al 1999). Similarly, chronic candida yeast infections are a common contributing factor to widespread pain. They are particularly common in women who have been given courses of antibiotic therapy for recurrent urinary tract infections, suspected sinusitis, complaints of earache, or sore throat (Gerwin & Dommerholt 2002). (See also Chs 3 and 12.)

Myoadenylate deaminase deficiency

Myoadenylate deaminase deficiency is a syndrome of muscle enzyme deficiency that, in a few cases, may cause widespread pain for which there is no permanent solution. Patients with pain resulting from myoadenylate deaminase deficiency are best managed with common pain management strategies (Marin & Connick 1997).

Metabolic insufficiencies

In her work with pain patients, Dr Janet Travell was one of the first physicians to suggest that metabolic insufficiencies and deficiencies, including those for vitamin B_{12}, folic acid and ferritin, may cause or contribute to complaints of localized and widespread pain (Simons et al 1999). A deficiency is a value outside the normal range and is easily recognized; an insufficiency is within the normal range, but may be sub-optimal, and often receives little attention. Yet, insufficiencies may cause serious problems for individual patients (Simons et al 1999). Although there are few scientific studies to support Travell's claims, clinicians familiar with her work recommend paying close attention to metabolic insufficiencies or deficiencies when patients experience only temporary improvement following physical therapy intervention (Gerwin & Gevirtz 1995). Vitamin B_{12} and folic acid are closely related and function not only in erythropoieses, but also in central and peripheral nerve formation. Serum levels of vitamin B_{12} below 350pg/ml may be clinically significant and associated with a metabolic insufficiency manifested by elevated serum or urine methylmalonic acid or homocysteine (Pruthi & Tefferi 1994). Laboratories commonly indicate that the normal range for vitamin B_{12} levels is between 200 and 1200pg/ml. Gerwin found that 16% of patients with chronic MPS were either deficient in vitamin B_{12} or had insufficient levels of vitamin B_{12}. Of these, 10% had low serum folate levels (Gerwin 1995).

Ferritin represents the tissue-bound non-essential iron stores in the body that supply the essential iron for oxygen transport and iron-dependent enzymes. Serum levels of 15–20ng/ml indicate that storage sites for iron, such as muscle, liver and bone marrow, are depleted of ferritin. Many women with a chronic sense of coldness and chronic MPS have insufficient or deficient ferritin and iron levels either from excessive menstrual iron loss or from chronic intake of non-steroidal anti-inflammatory drugs. Iron insufficiencies in chronic MPS suggest that iron-requiring enzymatic reactions such as the cytochrome oxidase and NAD(H) dehydrogenase reactions may be limited, possibly resulting in a local energy crisis when muscles are exposed to excessive mechanical stress (Gerwin & Dommerholt 2002). Serum ferritin levels below 30ng/ml need to be corrected through iron supplementation. By correcting the insufficiencies and deficiencies, patients commonly experience either total elimination of their pain complaints or become able to respond to medical and physical therapy interventions (Simons et al 1999).

Rheumatologic disease

Several rheumatologic diseases, including sero-negative rheumatoid arthritis, ankylosing spondylitis, Lyme disease, polymyalgia rheumatica and lupus, feature widespread pain and can easily be mistaken for FMS (Poduri & Gibson 1995). Most rheumatic diseases are treated with medication and education combined with physical therapy and occupational therapy interventions (Bertin 2000, Clark 2000, Ramos-Remus et al 2000, Stucki & Kroeling 2000).

Adult growth hormone deficiency

Adult growth hormone deficiency has only recently been recognized as a distinct clinical entity. In the USA approximately 6000 new cases occur annually. Some 70000 adults are estimated to have adult growth hormone deficiency. The disease can be caused by pituitary tumours, adenoma, head trauma and certain infectious diseases such as HIV/AIDS. Growth hormone is an amino acid polypeptide hormone synthesized and secreted by the anterior pituitary. Its primary function is to promote linear growth. Growth hormone stimulates the release of somatomedin C in the liver, which is required for the maintenance of normal muscle homeostasis (Neeck & Crofford 2000). The symptoms of adult growth hormone deficiency are variable and not all patients have symptoms. Some of the more common symptoms of adult growth hormone deficiency include fatigue, muscle weakness, stiffness, joint pain, a reduced ability to exercise, reduced

cardiovascular function, depression, social isolation, osteoporosis and a weakened immune system. Several of these symptoms have been described for FMS.

Growth hormone deficiencies have been established in some subsets of patients with FMS (Bennett 1998b, Bennett et al 1992, Griep et al 1994, Leal-Cerro et al 1999). Bennett found growth hormone deficiencies in approximately 30% of patients with FMS (Bennett 1998a, 2002a). When compared to healthy subjects, FMS patients exhibited a reduced growth hormone response to exercise, which was thought to be the result of increased levels of somatostatin (Paiva et al 2002). Somatostatin is a growth hormone inhibiting hormone that is secreted under the influence of corticotrophin-releasing hormone and thyroid hormones (Sapolsky 1992). Other researchers did not find any significant growth hormone deficiencies (Dinser et al 2000, Nørregaard et al 1995). Bennett has established that administering growth hormone reduced or in some cases eliminated the symptoms (Bennett 1998a, 2002a). The question remains whether the symptoms of these patients are solely due to adult growth hormone deficiency or due to FMS. If the symptoms are part of the symptomatology of adult growth hormone deficiency, would there be any benefit to adding a diagnosis of FMS?

Psychological diagnoses

Patients with psychological diagnoses and widespread pain are appropriately treated with an interdisciplinary approach combining medications with psychological interventions, exercise and stress management techniques, emphasizing the psychosocial, behavioural and organic aspects of chronic pain (Turk & Okifuji 1999). Patients with FMS are reported to have higher rates of lifetime and current depression, notwithstanding a few studies that did not find any evidence of increased depression (Ahles et al 1991, Hudson & Pope 1996, Offenbächer et al 1998, Piergiacomi et al 1989, Yunus et al 1991). Depression, anger, anxiety and illness behaviour have a negative impact on patients' feeling toward themselves, which is reflected in poor expectations of patients and their health professionals and poor outcomes in physical therapy and rehabilitation (DeVellis & Blalock 1992, Jensen et al 1999, McCracken et al 1999, Okifuji et al 1999a). (See also Ch. 7.)

Several questions remain. Can depression cause or significantly contribute to FMS? Do patients with FMS get depressed because of pain or increased pain sensitivity, allodynia and hyperalgesia? There is some evidence that depression may be secondary to pain and may completely resolve once the pain has been eliminated. (Hendler 1984, Wallis et al 1997). Persons diagnosed with FMS routinely maintain that the psychological and emotional symptoms are the result of FMS and not the cause. Or are both disorders the result of a common underlying abnormality? Based on recent studies and theories, there are some indications that depression and FMS could be the result of an insufficient catecholaminergic or serotonergic neurotransmission or hyperactivity of corticotrophin-releasing hormone (Ackenheil 1998, Hudson & Pope 1996, Neeck & Riedel 1999).

It is likely that having a diagnosis of FMS combined with constant pain, poor expectations regarding recovery and a sense of hopelessness may also become perpetuating factors for depressive mood disorders. Fassbender and colleagues observed that patients with FMS had significantly more tender points than patients with depression (Fassbender et al 1997). Several studies have shown that patients with FMS demonstrated significantly higher lifetime prevalence rates of mood, anxiety and somatization disorders than patients with rheumatoid arthritis (Burckhardt et al 1993, Hawley & Wolfe 1993, Katz & Kravitz 1996, Walker et al 1997). Wolfe and colleagues found that people with FMS are more than four times as likely to be divorced compared to the general population without FMS (Wolfe et al 1995).

Rather than diagnosing these patients with FMS, a diagnosis based on the criteria of the Diagnostic and Statistical Manual (DSM-IV) of the American Psychiatric Association seems more

appropriate, and may include dysthymia, depression or somatoform pain disorder (APA 2000). A psychiatric diagnosis of depression or somatoform pain disorder may give some patients the impression that physicians do not take their pain seriously. Yet there is evidence that some patients with the typical fibromyalgia symptoms are so psychologically distressed that the syndrome may indeed become an excuse not to deal with their difficult life circumstances (Ford 1997, Hellström et al 1999).

Myofascial pain syndrome

A survey of members of the American Pain Society showed general agreement with the concept that MPS exists as an entity distinct from FMS (Harden et al 2000). MPS is often thought of as a localized problem, yet nearly half the patients with MPS in a chronic pain management centre featured pain in three or four body quadrants (Gerwin 1998). These patients may meet the ACR criteria and erroneously be diagnosed with FMS. Even though both diagnoses may represent central sensitization, there are distinct advantages of a diagnosis of MPS over a diagnosis of FMS. In most cases, MPS can be treated effectively. The rheumatology literature suggests that currently there is no treatment for FMS.

The FMS tender points do not represent any anatomic abnormality. Therefore, treatments to resolve the tenderness at these points are doomed to fail. The argument has been made that strain/counterstrain tender points can coincide with the FMS tender points. The strain/counterstrain approach can be successfully applied in the treatment of those with chronic pain; however, it is likely that the strain/counterstrain tender points are in fact unidentified myofascial trigger points (MTrPs). MTrPs are areas of severe muscle contraction that are painful and limit range of motion. The palpable contraction knots or MTrPs function as peripheral nociceptors that can initiate, accentuate and maintain the process of central sensitizaton (Borg-Stein 2002). As a source of peripheral nociceptive input, MTrPs are capable of unmasking sleeping receptors in the dorsal horn, which may result in spatial summation and the appearance of new receptive fields. Input from previously ineffective regions can now stimulate the neurons (Hoheisel et al 1993, Mense 1997). Patients with MPS are treated with specific trigger point therapy in combination with the elimination of various perpetuating factors. (Gerwin & Dommerholt 2002). Patients with widespread pain or FMS do not experience pain only at the tender point sites, but also throughout their entire bodies. (Vecchiet et al 1994). (See also Chs 6, 9 and 13)

Hypermobility syndrome

Many patients with hypermobility syndrome suffer from widespread myalgia, presumably because of muscle imbalances and constant compensation of muscles in an effort to stabilize unstable joints (Russek 1999). These patients commonly meet the ACR criteria, but there is no compelling reason to make the diagnosis of FMS in addition to the diagnosis of hypermobility syndrome. Patients with hypermobility syndrome are often difficult to treat. A comprehensive treatment programme emphasizing patient education, activity modification and a progressive strengthening regime can decrease the associated symptoms and improve functional abilities (Russek 1999, 2000). (See also Ch. 3.)

Whiplash associated disorders

In 1997, the Israeli physician Dan Buskila suggested that FMS is common following motor vehicle accidents (Buskila et al 1997). Other researchers have also suggested a relationship between trauma and FMS. In spite of the now commonly held belief that motor vehicle accidents frequently result in FMS, in subsequent publications Buskila and colleagues as well as White and

colleagues have concluded that there really is no scientific evidence of a causal relationship between trauma and FMS (Buskila & Neumann 2000, 2002; White et al 2000a).

It has been established that involvement in motor vehicle accidents may result in central sensitization and widespread pain, but there is no benefit to the additional diagnosis of FMS (Koelbaek Johansen et al 1999). In a retrospective review, Gerwin and Dommerholt found that all patients with chronic pain complaints following a motor vehicle accident had MPS (Gerwin & Dommerholt 1998). During the 2001 Fifth World Congress on Myofascial Pain and FMS in Portland, Orgeon, Dr Buskila acknowledged that in his initial study of the prevalence of FMS following whiplash, he had not considered the presence of MTrPs. However, even if MTrPs would have been considered, by applying the inclusive ACR criteria, the diagnosis of FMS would still have been made.

Some of those with whiplash injuries may also suffer from post-traumatic stress disorder, which can also feature many of the symptoms of FMS (Sherman et al 2000). Furthermore, it is conceivable that in some whiplash patients the thyroid gland may be injured. This may contribute to the development of post-traumatic hypothyroidism with widespread pain, fatigue and other symptoms commonly attributed to FMS (Sehnert & Croft 1996). (See also Chs 3 and 9 for further discussion of FMS and whiplash.)

Widespread burns

Patients with widespread burns may meet the ACR criteria for FMS. However, it is inconceivable that a physician would diagnose FMS in the presence of obvious signs of burn scars (Wolfe 1993).

There are no studies that indicate how frequently the diagnosis of FMS is made in the presence of other diagnoses, but it is very likely that published research studies on FMS include subjects with other clinical diagnoses responsible for the pain, sleep disorder and fatigue. This could contribute to the poor results of long-term outcome studies, that frequently show that patients diagnosed with FMS do not improve (Wolfe et al 1997a). Could it be that after making the diagnosis of FMS, physicians and patients may not consider any other causes of chronic widespread pain? In these cases, would that make FMS an iatrogenic syndrome, as the appropriate diagnosis and effective treatment options would not be entertained or implemented? Or is it appropriate to diagnose FMS and other diagnoses responsible for widespread pain simultaneously?

The question remains how an additional diagnosis of FMS improves the medical management, particularly when the rheumatology literature suggests that currently there is no treatment for FMS (Bennett 1999; Russell 2001). If that is true, than why are patients labelled with this diagnosis in the presence of another diagnosis that provides a mechanism for the reported symptoms and for which effective treatment options are available? While it is known that the diagnosis of FMS initially offers patients a meaningful confirmation of their pain syndromes, most of the other diagnoses that can cause similar symptoms accomplish the same. How does a diagnosis by inclusion influence the thinking about FMS, if these patients are included in research studies but do not receive the appropriate medical intervention for the possible underlying cause of pain and dysfunction?

Physicians who are willing to consider the common principles of differential diagnoses and accept that at best the diagnosis of FMS is a diagnosis by exclusion may not diagnose patients so rapidly with FMS syndrome and avoid risking the development of illness behaviour and feelings of hopelessness, depression, anxiety fear, and poor expectations (Dommerholt 2002, Gerwin 1999).

MYOFASCIAL PAIN SYNDROME

Schneider & Brady suggested that 'pseudo FMS' can be divided into three categories, namely organic diseases (e.g. Lyme disease and hypothyroidism), functional disorders (e.g. nutritional deficiencies and intestinal dysbiosis), and musculoskeletal disorders (e.g. MPS and undiagnosed disc and facet lesions among others) (Schneider & Brady 2001). After excluding organic diseases and functional disorders, the diagnosis of MPS offers a valuable approach to reduce or eliminate pain and other associated symptoms, and to restore function. MPS should be considered in the differential diagnosis not only of FMS, but also of radiculopathies, anginal pain, joint dysfunction, craniomandibular dysfunction, migraines, tension headaches, complex regional pain syndrome, carpal tunnel syndrome, repetitive strain injuries, whiplash injuries and most other pain syndromes. MPS is commonly associated with other musculoskeletal diagnoses including facet joint injuries, disc herniations, osteoarthritis, or as part of post-laminectomy syndromes. It can occur as a complication of certain medical conditions, including myocardial infarction or kidney disorders. There is no evidence that MPS develops into FMS, although this is frequently suggested in the literature (Meyer 2002, Russell 2001).

During the last few decades, MPS has received much attention in the scientific and clinical literature. Already during the early 1940s, Dr Janet Travell realized the importance of MPS and its hallmark feature, the MTrP. Recent insights in the nature, aetiology and neurophysiology of MTrPs and their associated symptoms have propelled the interest in the diagnosis and treatment of MPS worldwide (Bennett 2002b).

Historically pain from muscles has been described by multiple terms, including fibrositis, myofasciitis, muscular rheumatism, rheumatic myositis, muscle hardening, myogelosis, myofascial pain and myalgia (Simons 1975). The phenomenon of MTrPs was already described in 1816 by the British physician Balfour as 'nodular tumours and thickenings which were painful to the touch, and from which pains shot to neighbouring parts'. These nodules were considered a result of inflammation in the fibrous connective tissue in muscle (Stockman 1904). The term 'trigger point' was coined by the American physician Steindler in 1940 (Steindler 1940). Over the last 60 years several assessment and treatment approaches have emerged independently of each other both in Europe and in the USA, including myofascial trigger point therapy (USA), neuromuscular technique or NMT (UK), neuromuscular therapy, also abbreviated as NMT (USA), and manual trigger point therapy (Switzerland). It is intriguing that these approaches share many similarities and have common goals and objectives. These approaches are briefly summarized:

Myofascial trigger point therapy

The American physician Janet Travell (1901–1997) has been referred to as the pioneer in the treatment of musculoskeletal pain through the recognition of MTrPs. Travell coined the term 'myofascial pain syndrome' to describe pain as a result of trigger points in muscle, tendon, skin, fascia and ligaments. She published over 40 papers on MPS and MTrPs during her lifetime and, together with physician David Simons, wrote the classic two-volume text *Myofascial Pain and Dysfunction: the Trigger Point Manual*. The first volume, for the upper half of the body, was published in 1983, followed by a second volume, for the lower half, in 1992 (Travell & Simons 1983, 1992). The second edition of the first volume, published in 1999, includes contributions by experts in the growing field of myofascial pain and dysfunction. The second edition reported significant progress in the understanding of the pathophysiological basis of the clinical presentations associated with MTrPs (Simons et al 1999). Travell and Simons initially emphasized inactivation of MTrPs using manual compression, trigger point injections, vapocoolant spray and muscle stretching. Muscle stretching can be combined with muscle energy techniques or postisometric relaxation, during which the muscle is stretched after a brief sub-maximal isometric contraction. All techniques are used diagnostically and therapeutically. The second edition includes many other techniques and places MPS in a broader clinical context

whereby other structures, movement patterns and functions are also considered. Much emphasis is placed on determining and eliminating metabolic, structural and other perpetuating factors.

Neuromuscular technique

During the 1930s, European physicians Stanley Lief and Boris Chaitow developed a method of assessment and treatment of soft tissue dysfunction called 'neuromuscular technique' (NMT). The concept and technique was based on their background skills in chiropractic, osteopathic, and naturopathic medicine (Chaitow & DeLany 2000). NMT practitioners focus on modifying dysfunctional tissues, including MTrPs, by applying digital pressure and specific soft tissue mobilizations. Recently, Chaitow & DeLany have described several NMT techniques, such as the NMT thumb technique, the finger technique, trigger point compression, and the integrated neuromuscular inhibition technique, among others (Chaitow & DeLany 2000). Using NMT techniques, MTrPs are inactivated and tissue circulation is enhanced. NMT involves other therapeutic goals, including enhancing lymphatic drainage, muscle relaxation and facilitation of therapeutic exercise. The techniques are used both diagnostically and therapeutically. NMT techniques are applied as an end in themselves or to facilitate other therapeutic interventions such as exercise or manipulation.

Neuromuscular therapy

A similar concept to the European neuromuscular technique was fostered in America during the 1950s by chiropractic physician Raymond Nimmo (1904–1986). In the 1960s, Nimmo was joined by James Vannerson, also a chiropractic physician (Cohen & Gibbons 1998). Chiropractic physicians Schneider, Cohen and Laws have recently compiled Nimmo and Vannerson's writings into a comprehensive compendium, making their work and ideas more accessible to a broader audience (Schneider et al 2001). Nimmo described the presence of muscular 'noxious generative points' which he considered the basis of his receptor–tonus control method. These muscle points were thought to generate nerve impulses and eventually result in 'vasoconstriction, ischemia, hypoxia, pain and cellular degeneration' (Schneider et al 2001). Later in his career Nimmo adopted the term 'trigger point' after having been introduced to Travell's writings. Nimmo maintained that hypertonic muscles are always painful to pressure, a statement that later became known as 'Nimmo's law'. Like Travell, Nimmo described distinctive referred pain patterns and recommended releasing these dysfunctional points by applying the proper degree of manual pressure (Cohen & Gibbons 1998). Nimmo maintained that 3–8 seconds of gentle manual pressure would be sufficient to facilitate therapeutic changes via the nervous system. Nimmo's notions ('all work is specific to, and exclusive to, the nervous system,' and 'it does not make any difference where you begin the corrective treatment') are consistent with current manual therapy concepts based on peripheral and central nociceptive dysfunction (Schneider et al 2001).

Nimmo's basic concepts regarding the physiology and treatment of trigger points were complementary to the work pioneered by Travell. Nimmo introduced a new concept to chiropractors in the identification and treatment of pain. He was influential in developing the idea that joint mobility dysfunction can be treated through the soft tissue system. The receptor–tonus control method has become well recognized within the chiropractic community over the past 50 years. According to a 1993 report by the National Board of Chiropractic Economics, over 40% of chiropractors frequently apply Nimmo's techniques (National Board of Chiropractic Economics 1993). Two spin-offs of Nimmo's work are NMT St John method and NMT American version, which have become particularly popular among massage therapists. NMT American version specifically considers local hypoxia, MTrPs, neural interferences, postural distortions, nutrition and the patient's emotional well-being (DeLany 2001).

Manual trigger point therapy

Manual trigger point therapy was initially conceived by Swiss rheumatologist and psychologist Beat Dejung and further developed by Dejung and Swiss physical therapists Christian Gröbli and Ricky Weissmann, among others. Dejung is a teacher of manual medicine techniques associated with the Swiss Society of Manual Medicine. After attending a presentation by Dr David Simons in Zurich in 1983 on the diagnosis and treatment of MPS, Dejung became intrigued with the concepts of Travell and Simons. Through clinical experience and further analysis, Dejung realized that most musculoskeletal pain has a predominantly muscular origin. Dejung was also influenced by the work of Ida Rolf (1896–1979), who developed the Rolfing method to manually stretch shortened connective fascia, and by Jack Painter, who is best known for his Postural Integration® approach (Dejung 1987a, 1987b, 1988a, 1988b, 1994). Although he based his thinking primarily on the work of Travell and Simons, Dejung developed his own approach somewhat in isolation until 1995 when Gröbli and Dommerholt started combining Dejung's approach with the American myofascial trigger point therapy approach (Gröbli & Dommerholt 1997). Manual trigger point therapy consists of a combination of manual therapy techniques, dry needling, therapeutic stretches, and a home exercise programme (Gröbli 1997; Weissmann 2000).

The various schools of thought have more in common than they are different. Some techniques are slightly different and there is some disagreement about terminology and methodology. The terminology and definitions formulated by Simons, Travell and Simons are the ones most widely accepted and will be applied in this chapter: 'myofascial pain syndrome can be described as the sensory, motor, and autonomic symptoms caused by myofascial trigger points' (Simons et al 1999). A MTrP is clinically defined as 'a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is painful on compression and can give rise to characteristic referred pain, referred tenderness, motor dysfunction, and autonomic phenomena' (Simons et al 1999).

There are no laboratory or imaging studies available for the diagnosis of MPS or MTrPs. To make a diagnosis of MPS requires a systematic palpation of pertinent muscles across the direction of the fibres. Only by palpating perpendicularly to the fibre direction will a clinician be able to locate the taut band. A taut band feels like a rope or string of contracted fibres that may extend from one end of the muscle to the other, depending on the specific muscle architecture. Microscopic and electrodiagnostic research of muscles has revealed that many muscle bellies are divided into compartments of one or more fibrous bands or inscriptions. Each compartment has its own nerve supply and motor endplates. The number of inscriptions and compartments varies per muscle. For example, the biceps femoris and gracilis have each two compartments, the semitendinosus has three and the sartorius has four. Because of these inscriptions, the longest human muscle fibres are approximately 12cm, which corresponds to $5.5x10^4$ sarcomeres (McComas 1996). When palpating for taut bands, clinicians must be aware of these inscriptions, as different taut bands can be found throughout the muscle belly of one particular muscle. Palpation along a taut band may reveal a nodule that is exquisitely tender and that with firm pressure stimulation may produce referred pain sensations in typical patterns for each muscle. These painful spots are known as MTrPs. Patients often recognize the localized or referred pain as 'their pain', and this recognition of pain is now considered one of the diagnostic criteria for active MTrPs in addition to the presence of a taut band and the MTrP itself (Gerwin et al 1997, Simons et al 1999). Taut bands and MTrPs are found in asymptomatic individuals and are only considered clinically relevant when the patient recognizes the elicited pain or when the functional limitations imposed by the taut band contribute to mechanical dysfunction secondary to muscle shortening (Gerwin & Dommerholt 2002, Scudds et al 1995).

The minimum criteria that must be satisfied in order to distinguish a MTrP from any other tender area in muscle are a taut band and a tender point in that taut band. The presence of a local twitch response, referred pain or reproduction of the person's symptomatic pain increases the certainty and specificity of the diagnosis of myofascial pain syndrome (Gerwin et al 1997). The taut band,

trigger point and local twitch response are objective criteria, identified solely by palpation, that do not require a verbal response from the patient. A local twitch response is an indication of the presence of an active trigger point. It is a brief involuntary contraction of the taut band, that can be recorded electromyographically, felt with the needle during trigger point injection or needling, or observed visually or on diagnostic ultrasound. It is mediated primarily through the spinal cord without supraspinal influence (Hong 1999, Hong & Torigoe 1994). High resolution sonography was not sensitive enough to visualize the actual MTrP, but allowed researchers to visualize the twitch response of the taut band following stimulation of the trigger point by insertion of a hypodermic needle (Gerwin & Duranleau 1997, Lewis & Tehan 1999). The patient's body type, the skill level and experience of the clinician, and the nature of the muscle determine the ease of eliciting a local twitch response.

Several studies have considered the interrater reliability of the MTrP examination. However, it was only recently established by Gerwin and colleagues and by Sciotti and colleagues (Gerwin et al 1997, Lew et al 1997, Nice et al 1992, Njoo & Van der Does 1994, Sciotti et al 2001, Wolfe et al 1992). In Gerwin's study, a team of recognized experts could initially not agree. Only after developing consensus regarding the criteria did the experts agree, which indicates that training is essential for the identification of MTrPs (Gerwin et al 1997). The International Myopain Society (IMS) is currently conducting a worldwide multicentre study to establish reliable methods for the diagnosis of MPS, and to determine the interrater reliability of trigger point examination and the sensitivity and specificity with which classification criteria can distinguish patients with MPS from healthy control subjects. (Russell 1999b).

The integrated trigger point hypothesis

Combining all available supporting evidence of the existence of MTrPs, Simons has recently proposed a new 'integrated trigger point hypothesis' (Simons et al 1999). The integrated trigger point hypothesis has evolved through several steps since its first introduction as the 'energy crisis hypothesis' in 1981 (Simons & Travell 1981). The hypothesis builds on the finding that excessively released acetylcholine from the motor nerve terminal causes miniature motor endplates potentials that produce the endplate noise observed with needle EMG of MTrPs (Couppé et al 2001; Simons et al 2002). Endplate noise occurs more frequently in MTrPs than in the same endplate zone away from the trigger point. The excessively released acetylcholine maintains a sustained depolarization of the postjunctional membrane, which in turn results in an excessive release of calcium from the sarcoplasmic reticulum and sustained sarcomeric contractions. (Simons et al 2002). Shenoi & Nagler confirmed that an impaired re-uptake of calcium into the sarcoplasmic reticulum induced by calcium channel blockers may cause MTrPs (Shenoi & Nagler 1996).

The original energy crisis hypothesis assumed that the excessive release of calcium was due to some traumatic event, such as a mechanical rupture of the sarcoplasmic reticulum or of the muscle cell membrane. Now it is known that any muscle trauma that triggers excessive acetylcholine release is sufficient to initiate the vicious cycle. It appears that the excessive release of acetylcholine can be triggered by mechanical, chemical or noxious stimuli. The noted increase of endplate noise is not specific to MTrPs and is seen with other types of mechanical stimulation of the endplate structure (Liley 1956).

It is conceivable that the myosin filaments literally get stuck in the Z band of the sarcomere. During sarcomere contractions, titin filaments are folded into a gel-like structure at the Z band. In MTrPs, the gel-like titin may prevent the myosin filaments from detaching. The myosin filaments may actually damage the regular motor assembly and prevent the sarcomere from restoring its resting length (Wang & Yu 2000). The contracted sarcomeres may reduce the local circulation by compressing the capillary blood supply and cause a significant lack of local oxygen. One study has shown that the oxygen saturation in the centre of a MTrP is less than 5% of normal (Brückle

et al 1990). The combined decreased energy supply and increased metabolic demand possibly could explain the common finding of abnormal mitochondria in the nerve terminal, often referred to as ‘ragged red fibers’ (Henriksson 1999, Henriksson et al 1993). The local energy crisis would also impair the calcium pump and provide another mechanism of the sustained contractions. The calcium pump is responsible for returning intracellular calcium into the sarcoplasmic reticulum against a concentration gradient, which requires a functional energy supply.

Local hypoxia is associated with the release of endogenous inflammatory substances, such as prostaglandin, bradykinin, serotonin, capsaicin, histamine and interleukins. Bradykinin is known to activate and sensitize muscle nociceptors, which leads to inflammatory hyperalgesia, an activation of high threshold nociceptors associated with C fibres and an increased production of bradykinin. Furthermore, bradykinin stimulates the release of tumour necrosis factor (TNF-a), which activates the production of the interleukins IL-1 beta, IL-6 and IL-8. Especially IL-8 can cause hyperalgesia that is independent from prostaglandin mechanisms. Via a positive feedback loop, IL-1 beta can also induce the release of bradykinin (Poole et al 1999).

The combined high-intensity mechanical and chemical stimuli may cause activation and sensitization of the peripheral nerve endings and autonomic nerves, activate second order neurons, cause central sensitization, and lead to the formation of new receptive fields, referred pain, a long-lasting increase in the excitability of nociceptors, and a more generalized hyperalgesia beyond the initial nociceptive area. Sensitization of peripheral nerve endings can also cause pain through the activation of N-methyl-D-aspartate (NMDA) and neurokinin.

Stimulation of the autonomic system has been shown to increase the endplate potentials. For example an increase in psychological arousal resulted in an immediate increase of endplate spike rates (Lewis et al 1994, McNulty et al 1994). Autogenic relaxation and the administration of the sympathetic blocking agents phentolamine and phenoxybenzamine inhibited the autonomic activation (Banks et al 1998, Chen et al 1998a, Hubbard 1996). Induced autonomic nerve activity would explain the observed autonomic phenomena and further contribute to the abnormal release of acetylcholine, possibly by increasing the permeability of calcium channels in the cell membrane of the nerve terminal (Chen et al 1998b, Hou et al 2002). A recent study examined the effects of MTrP massage therapy on the cardiac autonomic tone in healthy subjects. The researchers observed that following MTrP therapy, there was a significant decrease in heart rate, and systolic and diastolic blood pressure, indicating a significant increase in parasympathetic activity (Delaney et al 2002).

The integrated trigger point hypothesis is summarized in Figure 8.1. The hypothesis is a ‘work in progress’ that is beginning to be subjected to rigorous scientific review and verification. If this hypothesis is basically correct, MTrPs are primarily a muscle disease with secondary but important sensory, motor and autonomic phenomena (Borg-Stein & Simons 2002).

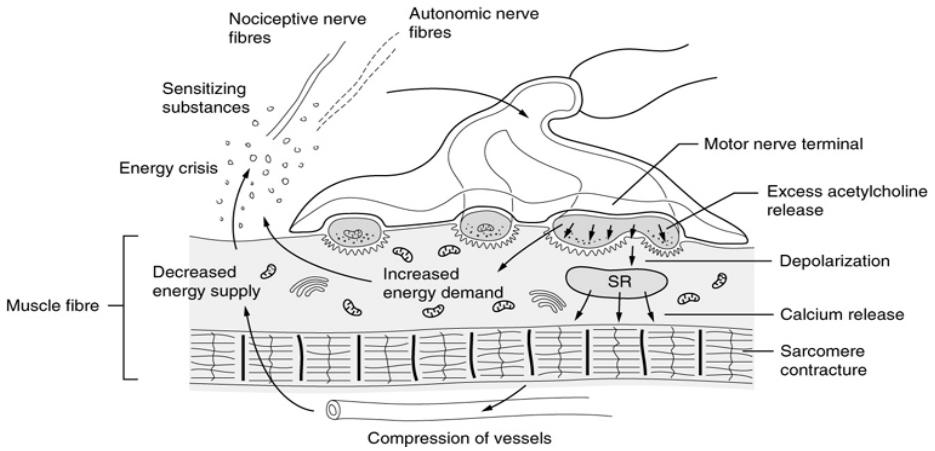

Figure 8.1 Integrated trigger point hypothesis (after Simons et al 1999; reproduced with permission from Chaitow & DeLany 2000, p. 68)

Clinical assessment

Any time a patient presents with a diagnosis of FMS or with any of its symptoms, a diagnosis of MPS should be suspected. As Schneider & Brady have outlined, a thorough differential diagnosis is critical and should include an assessment of the presence of organic diseases, functional disorders and musculoskeletal disorders (Schneider & Brady 2001). Several researchers have reported that MPS is the most common diagnosis responsible for chronic pain and disability, and possibly the most overlooked (Fricton et al 1985, Gerwin 1995, Hendler & Kozikowski 1993, Masi 1993, Rosomoff et al 1989, Skootsky et al 1989).

The initial task of the clinician in evaluating a patient is obtaining information regarding differential diagnostic characteristics, the cause of the problem, the patient's local tissue and global stress adaptability, and the prognosis. Clinicians must strive for completeness in their observations. A thorough examination requires a detailed patient history, observation, functional evaluation, palpation and drawing relevant conclusions (Materson & Dommerholt 1996). Following the initial assessment and formulation of diagnostic hypotheses, new data must be collected at each encounter. The initial hypotheses may need to be modified to facilitate the most efficient and effective management of patients with MPS (Jones 1994). A diagnosis of MPS does not exclude other diagnoses, such as joint dysfunction or metabolic insufficiency. At all times, the diagnostic process must consider all possible contributing factors to the pain syndrome.

Of particular importance in the evaluation of patients with chronic pain is the psychosocial assessment. This can provide insights into possible cultural influences of the patient's pain experience, family background and interpersonal dynamics, coping skills and perceived self-efficacy, and the presence of fear avoidance (Bandura et al 1987, 1988; Bates 1996; Bennett 2002c; Vlaeyen & Linton 2000). The chronicity of a pain problem may also be related to certain stressful work conditions, the work environment and the physical demand characteristics, or participation in leisure activities (Berg Rice 1995, Khalil et al 1994).

A sudden onset or a clear remembrance of the onset of pain may indicate an acute activation of MTrPs due to mechanical stress, but it may also indicate a sudden change in the patient's environment or habits. The mechanical stress may be the result of sudden or abrupt movements, motor vehicle accidents, falls, fractures, joint sprains or dislocations, a direct blow to a muscle or joint, excessive exercise or activity, or performing new or unusual activities. When the sudden onset of widespread pain occurs in close relation to a change in medication intake, the clinician should suspect that the change in medication intake may have triggered the pain response. In other cases, exposure to certain parasites may be the cause. For example, when a patient's local

drinking water well got polluted with giardia, several family members were subsequently diagnosed with FMS. The patient was told that FMS is known to be a familial disorder.

A slow insidious onset is usually the result of chronic overloading of tissue but may also be due to metabolic insufficiencies and parasitic infestations. Typical overload causes include postural imbalances, poor body mechanics, repetitive movements, and tension as a consequence of psychological or emotional stress. The symptoms of certain parasitic infestations, such as fascioliasis, may develop insidiously over a period of weeks, months and sometimes even years.

Questioning the patient regarding the nature of the pain and functional loss will give insight into which structures may be responsible. Myofascial pain caused by MTrPs tends to be dull, poorly localized and deep, similar to visceral referred pain and in contrast to the precise location of cutaneous pain (Gerwin 2002). It can present as a constant or intermittent deep ache, but rarely as throbbing or burning. Occasionally, patients describe pain from MTrPs as sharp or stabbing. The term ‘referred pain’ does not only encompass pain, but may also include other paraesthesias and dysesthesias. Referred sensations of MTrPs need to be distinguished from peripheral nerve entrapment and nerve root irritation. Functional limitations due to MTrPs include muscle weakness, loss of muscle coordination, fatigue with activity, decreased work tolerance and endurance and joint stiffness. Finally, limitations in active and passive range of motion are often due to MTrPs.

Once the possible cause has been identified, it is helpful to gain a better understanding of the course of the symptoms and previous diagnostic tests and treatments. Are there recurrent exacerbations and remissions, and if so, what are their triggers? Characteristically, MTrP pain is aggravated by strenuous use of the muscles, rigorous stretching of the muscles harbouring MTrPs, repeated trigger point compression, overloading and overcompensation of muscles during assumed prolonged postures, repetitive contractions of the involved muscle, cold and damp weather, viral infections and periods of increased stress, anxiety, and tension. Pain symptoms caused by MTrPs may be alleviated with periods of rest, gentle stretching, massage, use of heat, positional supports and activities that may induce relaxation, including breathing re-education and yoga.

What treatments have been administered previously and what were the outcomes and effects of those treatments? Often, acute problems develop into chronic problems due to poor insight and unawareness by the individual, inadequate management by medical professionals, and their inability to recognize MTrPs as the source of the problem or as a significant contributing factor. Due to the chronicity of the problem, muscle guarding and abnormal movement patterns persist, other muscles become involved, and latent MTrPs become active. Peripheral and central sensitization lead to other complications, including depression, anxiety, anger and other musculoskeletal problems. Clinicians who routinely consider MTrPs as part of the picture are often the last resort for patients who have been given endless diagnoses that do not explain or address the pain and associated dysfunction (Hendler & Kozikowski 1993). Patients often appear relieved when the practitioner can literally put the finger on the source of the pain, which usually results in instant rapport between patient and clinician.

Physical examination

The physical examination starts with a general impression of the patient’s physical expression, body type, static and dynamic posture and movement patterns. The patient’s breathing pattern may reveal potential overuse of accessory respiratory muscles, such as the scalene muscles, and indicate possible higher levels of stress. Structural abnormalities and asymmetries which result from a congenital or acquired movement impairment will invariably lead to persistent musculoskeletal pain and dysfunction and MTrPs. Pelvic obliquity, scoliosis, forward head posture, leg length discrepancy, small hemipelvis, short upper arm syndrome, long metatarsal

syndrome and scapular abnormalities are a few of the most common structural variations that can lead to MPS (Simons et al 1999). Leg length discrepancies are divided into structural and functional leg length discrepancies. Structural discrepancies are due to true anatomic differences in length of the femur or tibia, while functional discrepancies can be caused by hip adductor contractures, MTrPs in the quadratus lumborum muscles, hip capsule tightness or unilateral innominate rotation. Leg length discrepancies and pelvic asymmetries may produce muscle imbalances and postural adjustments and result in the development of MTrPs (Janda 1994). Leg length discrepancies may be due to congenital, developmental, traumatic, or pathological changes in one of the osseous links of the lower extremity kinetic chain.

Identifying the specific posture type from a thorough structural spine assessment in sitting and standing will offer the muscle imbalances that are present. The upper and lower crossed syndromes, described by Dr Vladimir Janda, recognize muscle imbalances on the basis of muscle fibre type and its inherent characteristics. Janda distinguished 'tonic or postural' muscles from 'phasic or dynamic' muscles. Tonic and phasic muscles are physiologically different in their oxidative ability and their ability to contract over a specified time period. Tonic muscles are slow twitch, slow oxidative and fatigue resistant posture (type I) muscles. Phasic muscles are divided into fast twitch, oxidative-glycolytic, and fatigue resistant movement (type II-a) muscles; fast twitch, glycolytic and easily fatigued movement (type II-b) muscles; and superfast (type II-m) muscles found primarily in the jaw muscles. (For more on muscle types, see Ch. 13.) MTrPs can develop in both tonic and phasic muscles. Tonic muscles include the hamstrings, rectus femoris, iliopsoas, quadratus lumborum, the erector spinae, the pectorals, the sternocleidomastoid, descending trapezius, and levator scapulae (Fig.8.2). Phasic muscles include the rectus abdominis, the serratus anterior, rhomboids, the ascending and transverse trapezius, the deep neck flexors, suprahyoid and mylohyoid (Janda 1983, 1993). Tonic muscles have a tendency to tighten in response to abnormal stress or dysfunction, while phasic muscles have a tendency to become weak. These typical response patterns will result in the upper and lower crossed syndromes (Figs 8.3 and 8.4). The upper crossed syndrome or forward head posture is the most common postural deviation in patients with MPS (Friction et al 1985, Janda 1994). (See also Fig.13.8, Ch. 13.)

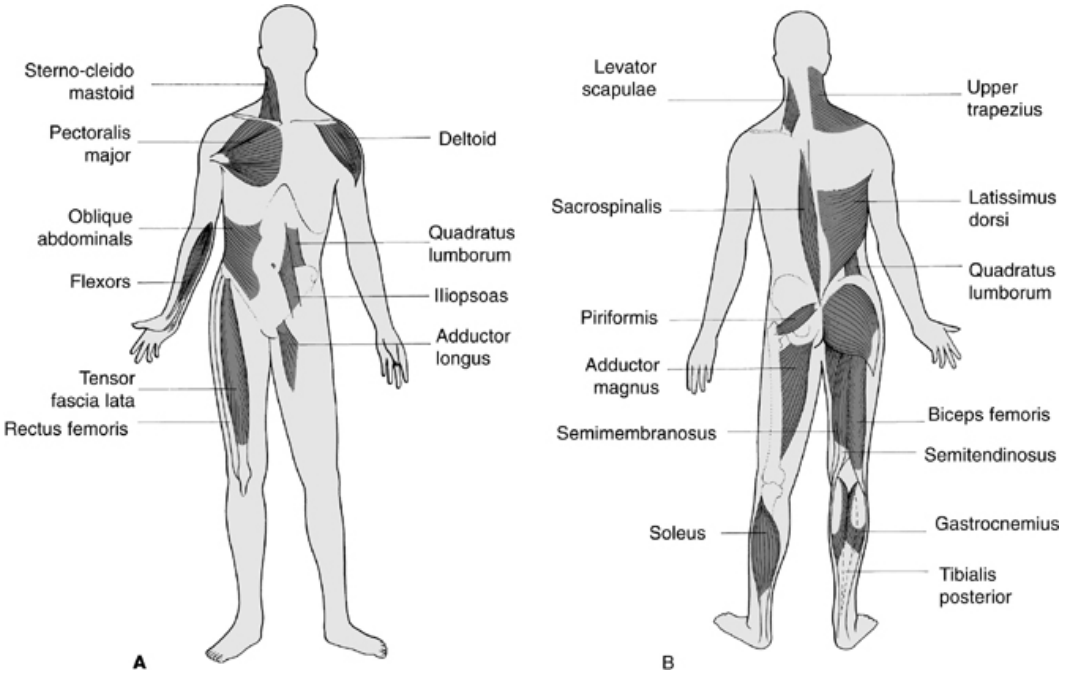

Figure 8.2 Major postural muscles **A** Anterior. **B** Posterior. (Reproduced with permission from Chaitow & DeLany 2000, p. 23.)

Forward head posture is usually associated with a posterior rotation of the cranium, an inversion of the cervical spine, and a protracted shoulder girdle. Myofascial restrictions and MTrPs are commonly seen in the suboccipital muscles, the cervical paraspinals, the splenius capitis and cervicis, the levator scapulae, the upper trapezius, the anterior and medial scalenes, the sternocleidomastoid, and the pectoralis minor and major muscles. Myofascial dysfunction also needs to be evaluated in the weak phasic musculature found in muscle imbalances. MTrPs will arise in any muscle that is functioning in a lengthened position or in a compromised position as a result of muscle imbalances and structural abnormalities.

Observing structural alignment and abnormal neuromuscular movement patterns during functional activities will also identify specific muscles or regions with myofascial restrictions. Much information can be gathered by watching how someone bends over and picks up an object from the floor, or how an individual walks down the hall, or balances on one leg. Someone with complaints of sciatica exacerbated with walking may exhibit an instability in hip rotation during a single leg stance due to poor neuromuscular control of the hip external and internal rotation muscles. MTrPs in the piriformis, gluteus medius and minimus and adductor muscles are likely.

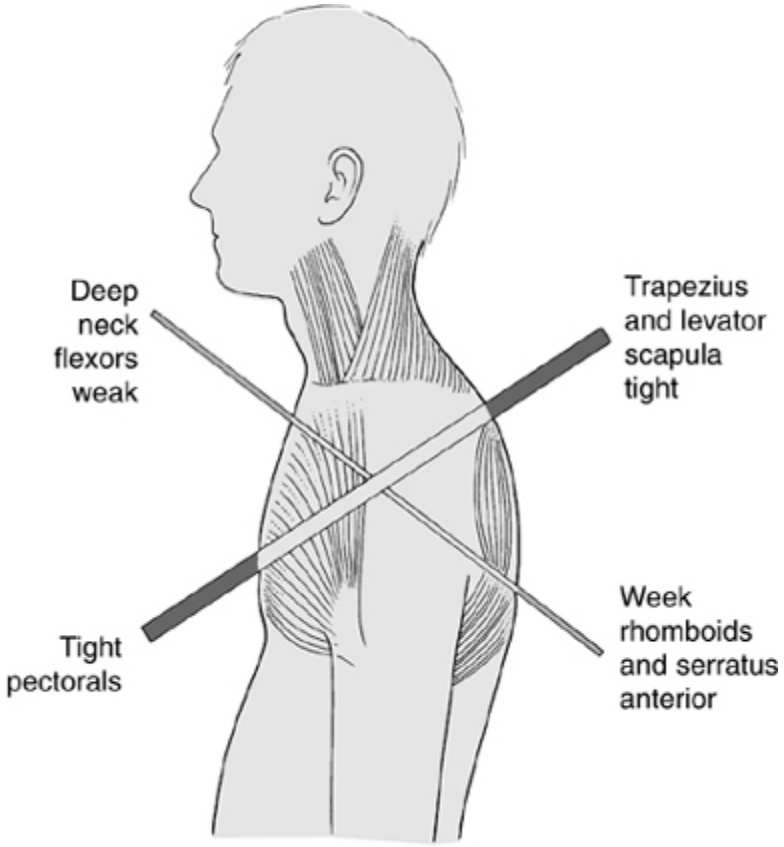

Figure 8.3 Upper crossed syndrome (after Janda). (Reproduced with permission from Chaitow & DeLany 2000, p. 56.)

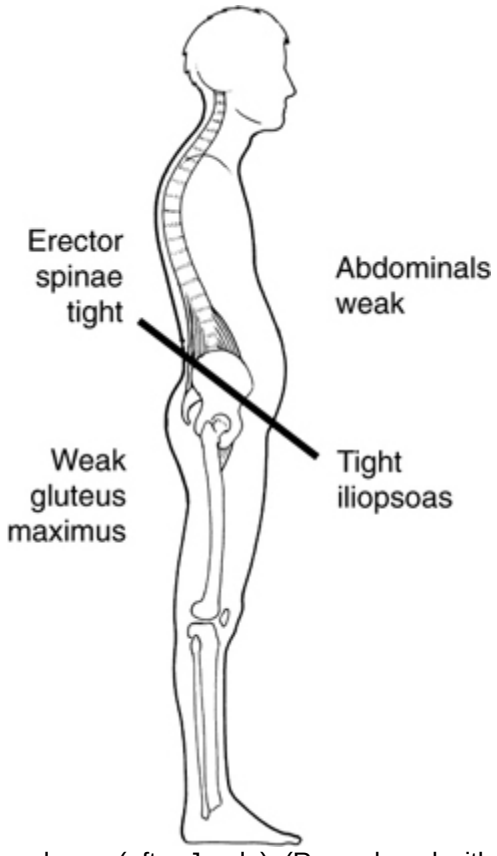

Figure 8.4 Lower crossed syndrome (after Janda). (Reproduced with permission from Chaitow & DeLany 2000, p. 56.)

As part of the physical examination, the clinician should include a thorough evaluation of MTrPs relevant to the patient's current pain presentation. The patient's current area(s) of pain can be visualized through patient pain drawings. MTrPs of each muscle have their own specific pain pattern. Active MTrPs are those trigger points that cause spontaneous complaints of pain. Latent MTrPs are not spontaneously painful, but may be tender to palpation. While patients communicate their pain patterns, the clinician can begin identifying those muscles and active MTrPs most likely involved in the pain problem. The localization of pain is not always the source of pain; hence the importance of a thorough differential diagnosis. Sources of referred pain are well known and include MTrPs, facet joints, intervertebral discs, nerve roots, peripheral nerve entrapments, viscera and sclera (Bogduk & Simons 1993, Giamberardino et al 1999, Simons et al 1999). Referred pain from MTrPs has been mistaken for pain from angina, radiculopathy, trigeminal neuralgia and thoracic outlet syndrome, among others.

In addition to considering the pain component, the mechanical aspects of MTrPs provide further insights. Both active and latent MTrPs may be associated with restricted range of motion and functional limitations. During the assessment, both the sensory and motor aspects of MTrPs must be considered. For example, in a patient who complains of temporal headaches, the pain complaint may direct the clinician to the sternocleidomastoid, trapezius, temporalis and inferior oblique capitis muscles. The patient's head posture in slight side-bending and rotation may implicate the scalene muscles, although the referred pain pattern from MTrPs in the scalene muscles do not include the head region. If the patient in addition presents with a paradoxical breathing pattern, it will be necessary to examine and treat the accessory breathing muscles as well, including the pectoralis minor, scalenes, sternocleidomastoid and upper trapezius musculature that may be overloaded due to increased demands. Teaching the patient a normal

diaphragmatic breathing pattern and fostering awareness of relaxation techniques for the upper chest and neck region will aid in the long term management of the headaches.

In the context of FMS, it is easy to understand how clinicians would conclude that a patient has a positive FMS tender point count when the pain is due to either localized or referred pain from MTrPs. A quick FMS tender point count will not reveal the cause of the increased sensitivity as the corresponding MTrPs frequently are not identified. Yet, the specificity of the diagnosis would increase dramatically and the prognosis would be far superior. The most common MTrPs, and their referred pain patterns that may be responsible for the tenderness at the FMS tender point locations, are summarized in Table 8.2. It is recommended that when a positive FMS tender point is identified, the clinician evaluates the patient for the presence of MTrPs that could cause the increased sensitivity at the FMS tender point location. MTrPs that correspond to the FMS tender points at the occiput, gluteal muscle, lateral epicondyle and knee are summarized in Figures 8.5 A–D. When a clinician identifies a positive FMS tender point, an examination of these MTrPs may direct the clinician to the cause of the pain and initiate an effective treatment approach. For the other FMS tender point locations, corresponding muscles should be examined for MTrPs (see Table 8.2)

The treatment of a patient with MPS falls beyond the context of this chapter. MPS can be approached from many perspectives, however, the therapy must address the various components of dysfunction. The local contraction knot or MTrP must be released, either manually or with dry needling or trigger point injections, to restore the local circulation, decrease pain and facilitate range of motion and functional movement patterns. There are many different manual techniques, including myofascial release techniques, compression, trigger point compression combined with active contractions of the involved muscle, muscle energy or postisometric relaxation, connective tissue stretches, and general massage therapy. Needling techniques include superficial and deep dry needling, and trigger point injections (Dr Peter Baldry has reviewed the various needling techniques in Chapter 6). The therapeutic programme must also address the various perpetuating factors, including metabolic insufficiencies, mechanical discrepancies, and psychosocial factors. The patient with chronic MPS may benefit from an interdisciplinary approach to include medical pharmacological management, psychosocial therapy, physical therapy, chiropractic or osteopathic mobilizations, massage therapy or more specific neuromuscular therapy. Patients with more acute MPS may only require treatment by a physician and either a physical therapist or neuromuscular therapist. (See Ch. 13 for MTrP treatment suggestions.)

The patient and the clinician need to identify appropriate goals and develop the means to implement them through therapy. Inactivation of MTrPs is a means to achieve relief of pain and improved biomechanical function, and thus to improve the ability of the patient to better perform whatever desired tasks have been selected as goals. Relief of pain or increased range of motion, both of which can be the result of MTrP inactivation, are not in themselves the final goals of treatment. For one patient an initial goal may be to simply sleep through the night. For another it may be walking the dog, or fastening a bra behind the back. For yet another it may be regaining sexual ability, or returning to work or to a recreational activity. Reasonable goals that can be achieved and measured as being reached or not are more important to focus on than simply the inactivation of a MTrP or an increase in the range of a particular movement (Gerwin 2000).

Table 8.2 Fibromyalgia tender point locations and overlapping myofascial trigger point areas and referred pain patterns

FMS TPs	Location of FMS TPs	Common overlapping MTrPs and referred pain patterns
Occiput	At the subocciptal muscle insertion	Suboccipitals, upper trapezius, splenius capitis, sternocleidomastoid, semispinalis cervicis, multifidi
Low cervical	At the anterior aspect of the intertransverse space at C5–C7	Upper trapezius, splenius cervicis, Levator scapulae multifidi, sternocleidomastoid
Trapezius	At the midpoint of the upper border	Upper trapezius, scalenes, levator scapulae supraspinatus, multifidi
Supraspinatus	At origin above the scapula spine near the medial border	Supraspinatus, levator scapulae, upper trapezius middle trapezius, iliocostalis thoracis
Second rib	At the second costochondral junction, just lateral to the junction on upper surface	Pectoralis major, pectoralis minor, sternalis
Lateral epicondyle	2cm distal to the epicondyle	Subscapularis, triceps, subclavius, scalenes, serratus posterior superior, supraspinatus, infraspinatus, brachioradialis, supinator, anconeus, extensor carpi radialis longus, extensor digitorum
Gluteal	In upper outer quadrant of the buttock in anterior fold of muscle	Quadratus lumborum, gluteus maximus, gluteus medius, gluteus minimus, piriformis, iliocostalis lumborum
Greater trochanter	Posterior to the trochanteric prominence	Quadratus lumborum, gluteus maximus, gluteus minimus, piriformis, iliocostalis lumborum, vastus lateralis
Knee	At the medial fat pad proximal to the joint line	Vastus medialis, rectus femoris, sartorius, adductors longus and brevis

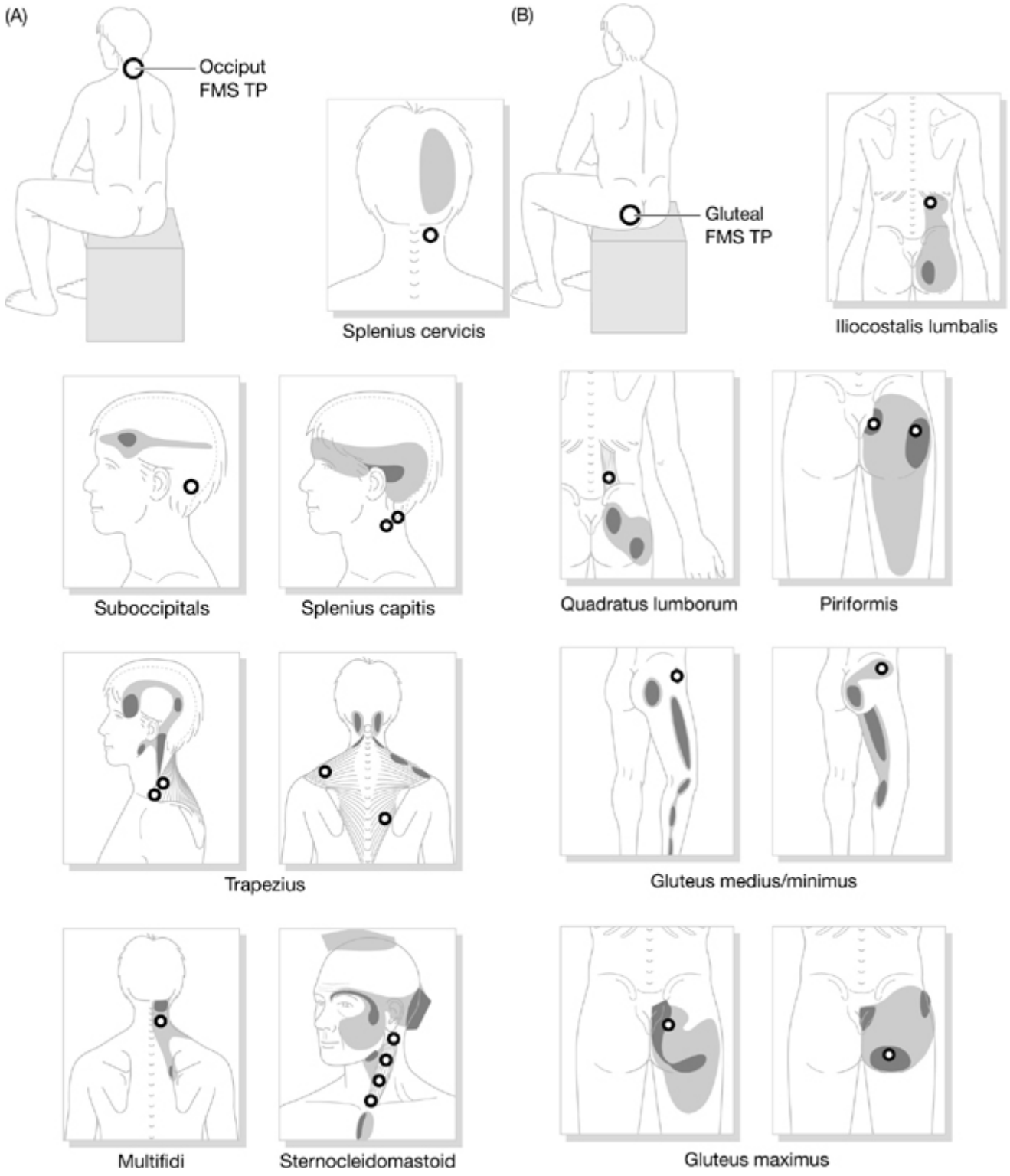

Fig 8.5 A Occiput FMS tender point and MTrPs. **B** Gluteal FMS tender point and MTrPs

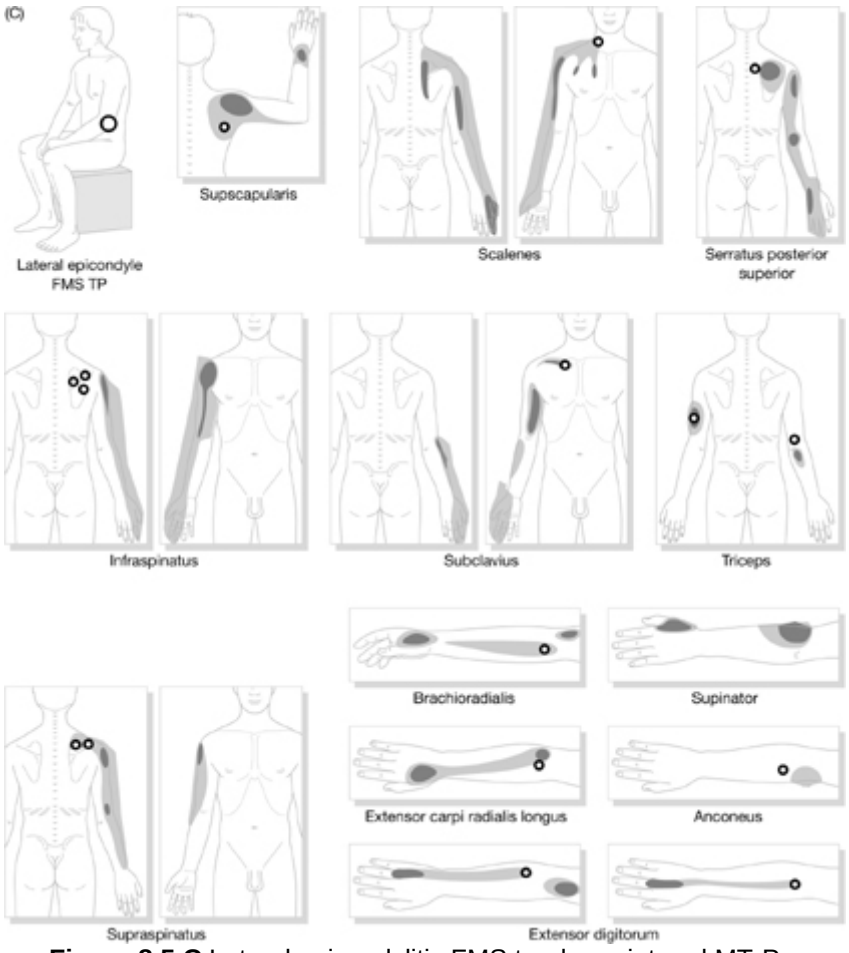

Figure 8.5 C Lateral epicondylitis FMS tender point and MTrPs.

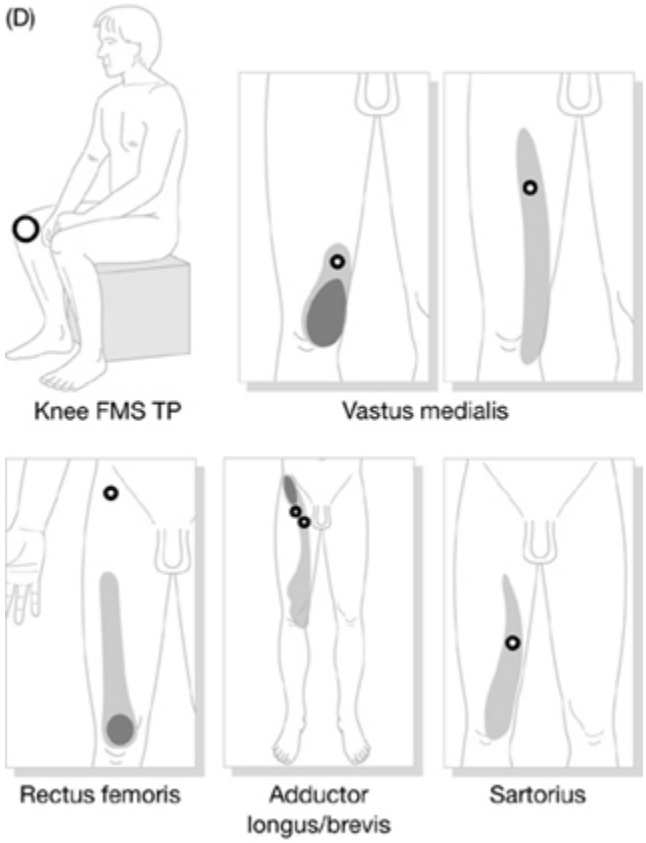

Figure 8.5 D Knee FMS tender point and MTrPs.

SUMMARY

Many arguments can be made to consider the normal differential diagnostic process in the diagnosis of individuals with widespread chronic pain. The notion that the diagnosis of FMS should be made irrespective of other diagnoses seems to be too simplistic and may actually deprive patients of necessary treatments, since clinicians and patients may not consider other possible causes of widespread chronic pain once the diagnosis of FMS has been established. Empirically, the diagnosis of MPS appears to be a reasonable alternative, especially when MTrPs are identified that mimic the patient's pain complaint. Other diagnoses, including organic diseases and functional disorders, must be ruled out. In agreement with the German criteria for FMS, patients with chronic widespread pain may be better off when the diagnosis of FMS is considered a diagnosis by exclusion.

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