

# Myofascial Pain: An Update

## Myofaszialer Schmerz: Ein Update

### Author

F. Timmermans

### Affiliation

Uplands Physiotherapy Clinic, CA-Penticton

### Key words

- myofascial pain
- trigger point
- physiotherapy

### Schlüsselwörter

- myofaszialer Schmerz
- Triggerpunkt
- Physiotherapie

### Abstract



**Background:** Myofascial pain, and its characteristic myofascial trigger points, is very common in physiotherapy practice.

**Objective/Method:** This article, an update from 2006, reviews the latest research and points out some potential clinical implications for daily physiotherapy practice.

**Results/Conclusions:** Myofascial pain is beginning to gain recognition as a neuromuscular disease and scientific studies of myofascial trigger points (MTrP) have found objective abnormalities that, together with observed changes in motor and sensory characteristics, implicate peripheral and central mechanisms in the development of myofascial pain. Beyond a primary muscle cause, predisposing and precipitating factors and remote lesions can be present, and need management as well.

### Zusammenfassung



**Hintergrund:** Myofaszialer Schmerz und seine charakteristischen Triggerpunkte sind in der physiotherapeutischen Praxis weit verbreitet.

**Ziel/Methode:** Dieser Artikel – ein Update von 2006 – gibt einen Überblick über die neueste Forschung und stellt einige mögliche klinische Schlussfolgerungen für die tägliche physiotherapeutische Praxis dar.

**Ergebnisse/Schlussfolgerungen:** Myofaszialer Schmerz beginnt als neuromuskuläre Erkrankung Anerkennung zu finden, und wissenschaftliche Studien haben objektive Anomalien gefunden, die zusammen mit beobachteten Veränderungen motorischer und sensorischer Eigenschaften periphere und zentrale Mechanismen in die Entstehung myofaszialer Schmerzen einschließen. Neben einer primären muskulären Ursache können prädisponierende und auslösende Faktoren sowie weiter entfernte Läsionen vorkommen, die ebenso behandelt werden müssen.

received 21.5.2014  
accepted 10.7.2014

### Bibliography

**DOI** <http://dx.doi.org/10.1055/s-0034-1384918>  
physioscience 2014; 10:  
106–114 © Georg Thieme  
Verlag KG Stuttgart · New York ·  
ISSN 1860-3092

### Correspondence

**Frank Timmermans, PT, CGIMS**  
Uplands Physiotherapy Clinic  
185 Middle Bench Road South  
Penticton B.C V2A 8S7  
Canada  
uplandsphysio@shaw.ca

### Introduction



There are more than 100 types of soft-tissue pain (STP), classified with respect to relevant painful conditions into three subgroups depending on the extent of body involvement: localized, regional and generalized [55]. In this system, focal muscle pain is localized pain, myofascial pain is regional pain, and fibromyalgia is generalized pain [55]. Myofascial pain syndrome (MPS) has been more recently termed “chronic myofascial pain” (CMP), and is starting to gain recognition as a neuro-muscular disease [43].

### Definition of Myofascial Pain and Classification of MTrPs



Myofascial pain is uniquely characterised by local regions of muscle hardness in a discrete taut band with myofascial trigger points (MTrPs) that can display localized tenderness and referred pain [28]. The taut band is a readily palpable group of contracted muscle fibres that are tender at the region of greatest hardness [33]. Referred pain occurs when spontaneous pain is referred to remote sites from an MTrP and is the result of central sensitisation in the spinal cord dorsal horn [33, 58]. Simons and Travell [59] defined a MTrP as the “most tender or hyperirritable circumscribed spot in a palpable taut band of skeletal muscle fibres”. An active MTrP (ATP) causes spontaneous pain or pain due to movement [59]. Spontaneous

pain occurs without any stimulus or provocation. A myofascial pain patient may have many ATPs. A latent MTrP (LTP) is a sensitive spot with pain or tenderness or sensitivity on mechanical stimulation such as stretch or pressure (also palpation) without spontaneous pain [33, 59].

CMP could begin with the patient having only one ATP in the affected muscle. If it is not appropriately treated or the associated underlying pathologic lesion is not eliminated, the pain region can expand to other regions and additional ATPs may develop [33]. There may be a spectrum of nociceptor irritability that distinguishes a normal muscle from a muscle with a latent or active trigger point. MTrPs are likely first formed as an LTP and then become activated and converted to ATPs by continuous detrimental stimuli such as prolonged or unaccustomed exercise, low-load repetitive muscle work, acute or mechanical trauma, continued stress or prolonged ischemia [33]. Therefore, diagnosis and management of LTPs are very important in the clinical settings [7].

### Prevalence of Myofascial Pain

Myofascial pain is considered a prevalent under-diagnosed and under-treated component of many acute and chronic pain complaints [27, 33, 59]. CMP is often noted as being the “most common cause of pain in the musculoskeletal system” [33, 59]. Age, gender and repetitive work are correlated with CMP [20, 21, 45, 65].

The true prevalence of CMP is difficult to measure because not many studies of CMP per se have been conducted. Myofascial pain affects approximately 44 million Americans [27, 43, 67]. Almost everyone will develop an MTrP at some time in their life as the lifetime prevalence in the general population is estimated at 85% [21].

Between 21 and 93% of patients with regional pain complaints have CMP, with the numbers varying with the patient population examined, the diagnostic criteria used to diagnose CMP and, in particular, the training and skill of the investigators ([27, 43, 67];

• **Tab. 1).**

German physicians conclude that almost every second person has ATPs at any given time [21]. Studies in the general population demonstrate that 25 – 54% of asymptomatic individuals can have LTPs [27, 43]. Lucas et al. [40] report that 90% of 154 healthy adults examined for the presence of LTPs in the scapular positioning muscles had at least one MTrP in these muscles. In addition, Celik et al. [7] detected LTPs in 60.5% of the scapular muscles: 47.8% on one side and 39.2% on both sides. Grieve et al.

[29] found multiple LTPs in trapezius, triceps surae and gastrocnemius muscles.

### Muscle Pain

Muscle pain, classified as nociceptive pain, has a unique neurobiology with distinctive characteristics that are critical in explaining the clinical presentation of myofascial pain [58, 68]. Clinical muscle pain is not simply the consequence of a “switching on” of the “pain system” in the periphery by a particular pathology, but instead reflects to a substantial extent, the state of excitability of central nociceptive circuits [68].

An important implication of early basic science studies was the possibility that the pain experience might not necessarily only reflect the presence of a peripheral noxious stimulus [68]. The ability of the central nervous system (CNS) to react to a short-duration input of noxious stimulation with a long-lasting deviation from normal synaptic function is particularly obvious in the case of muscle pain [33, 58]. Although the STP classification is convenient for clinical practice, it does not indicate the potential mechanisms behind muscle pain [33, 55].

### Neurophysiology of Muscle Pain

The primary peripheral sensing apparatus in muscle involves group III (thinly myelinated, low-threshold, fast conducting a-delta-fibres) and group IV (unmyelinated, high-threshold, slower c-fibres) afferent nerve fibres. These fibres cause aching, cramping pain that is deep and difficult to localize when stimulated with micro-neural techniques [46, 53].

Reduced spatial resolution due to a lower innervation density of muscle tissue makes localizing of muscle pain more difficult [45]. The dorsal horn cells receiving information from muscles are convergent neurons, both wide dynamic range and nociceptive neurons [33]. After the first synapse with the dorsal horn cell in the spinal cord, the nociceptive information from muscle is largely mixed with information from other tissues, such as skin, periosteum, bone, and viscera and this can blur the identification of the origin of the pain [46].

There is no evidence of the existence of an ascending tract that exclusively mediates muscle pain and a specific cortical centre for muscle pain does not seem to exist [47, 58]. In addition to activating the thalamus, muscle afferent input preferentially activates the limbic system, which plays a critical role in modulating muscle pain and the emotional or affective component to persistent pain [46, 58]. Increased activity in the limbic system leads to greater fear, anxiety, and stress [51].

### Persistent Myofascial Pain

Models for acute muscle pain have been developed and yielded information about the generation of local and referred pain [6, 45]. However, most clinically relevant muscular pain lasts far longer than the conditions studied in animals or even in humans studied under laboratory conditions. Therefore, there is great interest in investigating longer lasting and persistent pain in humans [6, 27]. In conditions with chronic pain the balance in pain modulation can be disturbed due to impaired pain inhibition and/or enhanced pain facilitation. The induction of activity-de-

**Tab. 1** Prevalence of myofascial pain in different USA based medical settings [43].

body region	medical institution	number studied	prevalence of CMP (%)
general	medical	172	30
general	pain medical centre	96	93
general	head and neck pain clinic	283	85
craniofacial	comprehensive pain centre	164	55
lumboglotal	orthopaedic clinic	97	21

CMP = chronic myofascial pain

This document was downloaded for personal use only. Unauthorized distribution is strictly prohibited.

pendent increases in synaptic function in these circuits, triggered and maintained by dynamic nociceptor inputs, shifts the sensitivity of the pain system such that normally innocuous inputs can activate it and the perceptual responses to noxious inputs are exaggerated, prolonged and widespread so called “central sensitisation” [6, 45, 68].

Central sensitisation can lead to a number of physiological changes, including increased spontaneous activity of dorsal horn neurons, apoptosis of inhibitory neurons at the segmental levels, reorganisation of spinal cord neurons and open up new synaptic connections, expansion of receptive field sizes and generate expanded referred pain regions, a lowered activation threshold for excitatory neurons, windup, which may lead to a 20-fold increase in neuronal sensitivity, prolonged after-discharges, increased activity of brain-orchestrated facilitatory pathways, which augment nociceptive transmission, dysfunctional endogenous analgesia and altered sensory processing in the brain [68]. Central sensitisation can also be enhanced and maintained by supraspinal processes involving cognition, attention, emotion and motivation –so called cognitive emotional sensitisation [6, 69]. These forebrain products can significantly contribute to the clinical pain experience in persistent conditions, including CMP and FM, the latter being characterised by widespread pain with allodynia, also outside the tender points, fatigue, abdominal symptoms, migraine, painful bladder, and irritable bowel [45, 48, 55].

The implications can be profound and persist for a prolonged period of time, leading to pain elicited by normally non-noxious stimuli (allodynia), pain evoked by an innocuous stimulus with enhanced nociception, an increase in responsiveness and prolonged after effects (mechanical hyperalgesia), and expanded pain patterns with increased size and number of referred pain areas with associated somatosensory changes [33, 45, 58]. Eventually, distinguishing CMP from fibromyalgia (FM) may become difficult [9, 23, 48].

The increased excitability of spinal neurons and the spread of excitation within the CNS are the first steps in the process of chronification of muscle pain [46, 47]. Such neuroplastic changes support the clinical observation that chronic muscle pain is often difficult to resolve [58]. MTrPs are now not merely a peripheral phenomenon, but rather they activate and sensitise the dorsal horn and higher brain centres and may, in turn, be dynamically modulated by these structures [58]. A sensitised CNS may lead to a lowering of the activation threshold of the peripheral nociceptors in a trigger point, inducing the transition from an LPT to an ATP [33].

When the pain intensity in a remote lesion is strong enough, the pain message can spread to the pain pathway of that muscle. This remote lesion can be at another muscle and other MTrPs could develop in the referred pain area of the original MTrP [27, 45, 47]. Other tissues (e.g. tendon, ligament, bursa, joint, bone and viscera) as well as psychological stress can contribute to this process [33, 35].

### Pathophysiology of CMP

Current literature still provides three hypotheses to explain the pathophysiology of CMP, as supported by scientific investigation of the motor and sensory characteristics of MTrPs ([27, 33, 39, 45, 59; • **Tab. 2**). Objective abnormalities support the assertion that MTrPs are a complex form of neuromuscular dysfunction, invol-

**Tab. 2** Characteristics of latent and active myofascial trigger points (modified from Hong [33]). The separation of ATPs into three categories is artificial. Referred pain, LTRs, and restricted ROM are not always elicited in a LTP. In a mildly ATP, the referred pain is often not obvious. In a moderately ATP, referred pain usually develops, which explains many of the pains seen in the clinic. Patients with severely ATPs typically show motor dysfunction and autonomic phenomena with enhanced sympathetic activation such as abnormal sweating or increased vasomotor and pilomotor responses.

characteristic of MTrP	LTP	mild ATP	moderate ATP	severe ATP
taut band*	+	++	+++	++++
local twitch response* (LTR)	+/-	+	++	+++
restricted range of motion (ROM)	+/-	+	++	+++
pain** (spontaneous)	-	+	++	+++
referred pain** (spontaneous)	-	+/-	+	++
motor dysfunction* weakness	-	+/-	+/-	+
autonomic phenomena**	-	-	-	+/-

\* = motor characteristic; \*\* = sensory characteristic; ATP = active trigger point; LTP = latent myofascial trigger point; MTrP = myofascial trigger point

ving skeletal muscles as well as peripheral and central sensitisation [27, 33, 39, 45, 46, 58].

Recent studies on myofascial pain show [33, 39, 46, 58]:

- ▶ Imaging techniques using magnetic resonance imaging (MRI) and ultrasound can demonstrate the physical features of the MTrP and provide morphologic evidence of taut band and contracture knot in the MTrP region.
- ▶ The multiple tiny sensory components of the MTrP are sensitised nociceptors that are responsible for tenderness, pain, referred pain, and local twitch responses.
- ▶ The motor components of the MTrP are dysfunctional endplates that are responsible for taut band formation as a result of excessive acetylcholine leakage.
- ▶ A drop in pH is probably one of the main activators of peripheral muscle nociceptors.
- ▶ Electromyographic changes can be recorded in the MTrP region.
- ▶ The concentrations of pain-related and inflammation-related substances are increased in an ATP region compared to a LTP or normal muscle tissue.
- ▶ This MTrP circuit is the connection among spinal sensory (dorsal horn) neurons responsible for the MTrP phenomena.
- ▶ Laser doppler flowmetry improves the imaging contrast between suspected MTrPs and surrounding muscles and shows abnormalities are not confined to discrete isolated nodules but instead affect the milieu of the muscle surrounding palpable MTrPs.

There is a need for more scientific knowledge concerning the pathophysiology and pathogenesis of myofascial pain [6]. With further refinement, ultrasound imaging may be a promising objective method in the clinical setting for characterising soft tissue abnormalities associated with MTrPs [58].

### Integrated Hypothesis of Myofascial Trigger Point

The integrated hypothesis, first put forward by Simons und Travell [59] and later expanded by Gerwin [27], is the most credited

local hypothesis for primary MTrP formation. In this hypothesis, an excess release of acetylcholine (Ach) at the motor endplate results in the creation of taut bands in the affected muscle that compress capillaries thereby decreasing local blood flow and causing ischemia. Ischemia limits the availability of oxygen and glucose, thereby creating an energy crisis in the working muscle. As a result, potassium, histamine, substance P and other excitatory substances that activate peripheral nerve nociceptive receptors are released, stimulating dorsal horn nociceptive neurons and causing pain [6, 7, 27, 33].

Furthermore, Shah's studies [58] demonstrate and confirm that both ATPs and LTPs are associated with significant objective differences in the local biochemical MTrP milieu, high concentrations of which have the ability to cause both peripheral and central sensitisation. These findings could help explain why ATPs can be acutely painful, tender, and a source of referred pain [58]. The excessive acetylcholine release, the sarcomere shortening, and release of these sensitising substances are the three essential features that relate to one another in a positive feedback cycle that is self-perpetuating once initiated [33].

### Muscle Spindle Hypothesis

The muscle spindle hypothesis was introduced in 1993 by Hubbard and Berkoff [34] and attributed the source of spontaneous electrical activity (SEA) to intrafusal muscle spindles located near MTrPs. Later, Simons hypothesised that SEA originates from dysfunctional motor endplates and defined it as "endplate noise" (EPN; [3, 24]). These miniature endplate potentials are thought to be the result of the spontaneous release of acetylcholine from motor nerve potentials, but this has not yet been demonstrated [27].

Various electromyography studies performed since then suggest that SEA/EPN has an important role within MTrPs in muscle pain and central sensitisation [3, 24, 27]. SEA represents a focal muscle fibre contraction, contributing to the formation of muscle tension and the taut band associated with MTrPs [26]. In humans, lower pain pressure thresholds (PPT) at MTrPs have been associated with higher SEA amplitudes. Thus, the irritability of an MTrP was highly correlated with the prevalence of EPN/SEA [33, 38]. The assessment of these phenomena in an MTrP region may be applied to evaluate the irritability of that MTrP [10, 38]. However, intramuscular needle electromyography (EMG) may not be appropriate due to patient discomfort [8].

### Radiculopathy Behind MrPs

A third hypothesis suggests that the formation of MTrPs is due to minor lesions in the peripheral nerve, especially in the nerve root [30]. The neuropeptides stored in the muscle nociceptors can be released when spinal nerves are compressed [46]. The formation of an LTP may be due to minor radiculopathy from minor repetitive stress to the spine [33]. Minor radiculopathy may cause excessive acetylcholine secretion at the neuromuscular junction, subsequently inducing endplate noise in the neuromuscular junction and causing the focal contractions of sarcomeres in the endplate zones [30, 33]. Other researchers suggest spinal segmental sensitisation (SSS) is consistently associated with musculoskeletal pain, underscoring its significance. Indeed de-

velopment or activation of more MTrPs is one of the clinical manifestations of SSS ([58]; [Fig. 1](#)).

### Predisposing, Precipitating and Perpetuating Factors Surrounding CMP

MTrP is a dynamic entity, undergoing transitions between a non-tender taut band to an LTP to an ATP and back again. Chronic or recurrent myofascial pain may not improve until the underlying precipitating or perpetuating factors are managed [4, 25, 33, 59, 64, 65]. Predisposing factors include sleep deprivation, genetics, age, nutrition and lack of movement. Precipitating factors can be present when MTrPs develop during occupational, recreational, or sports activities if muscle use exceeds muscle capacity and normal recovery is disturbed [5]. Muscle overuse/overload is hypothesised to be the result of sustained or repetitive low-level muscle contractions, eccentric muscle contractions, maximal or submaximal concentric muscle contractions or direct muscle trauma [5].

Perpetuating factors fall into two major categories: mechanical and systemic [25, 27, 33, 59]. Mechanical factors with a combination of static and awkward postures often cause repetitive minor trauma to muscles, tendons, and ligaments that can perpetuate the activation of MTrPs [27, 33, 59]. Ergonomic factors, such as time at the computer or habitual postures with sitting or driving, often play a vital role in myofascial pain problems [27]. Systemic factors include nutritional deficiency, vitamin deficiency, anaemia, and endocrine disorders such as hypothyroidism or estrogen deficiency [25, 28].

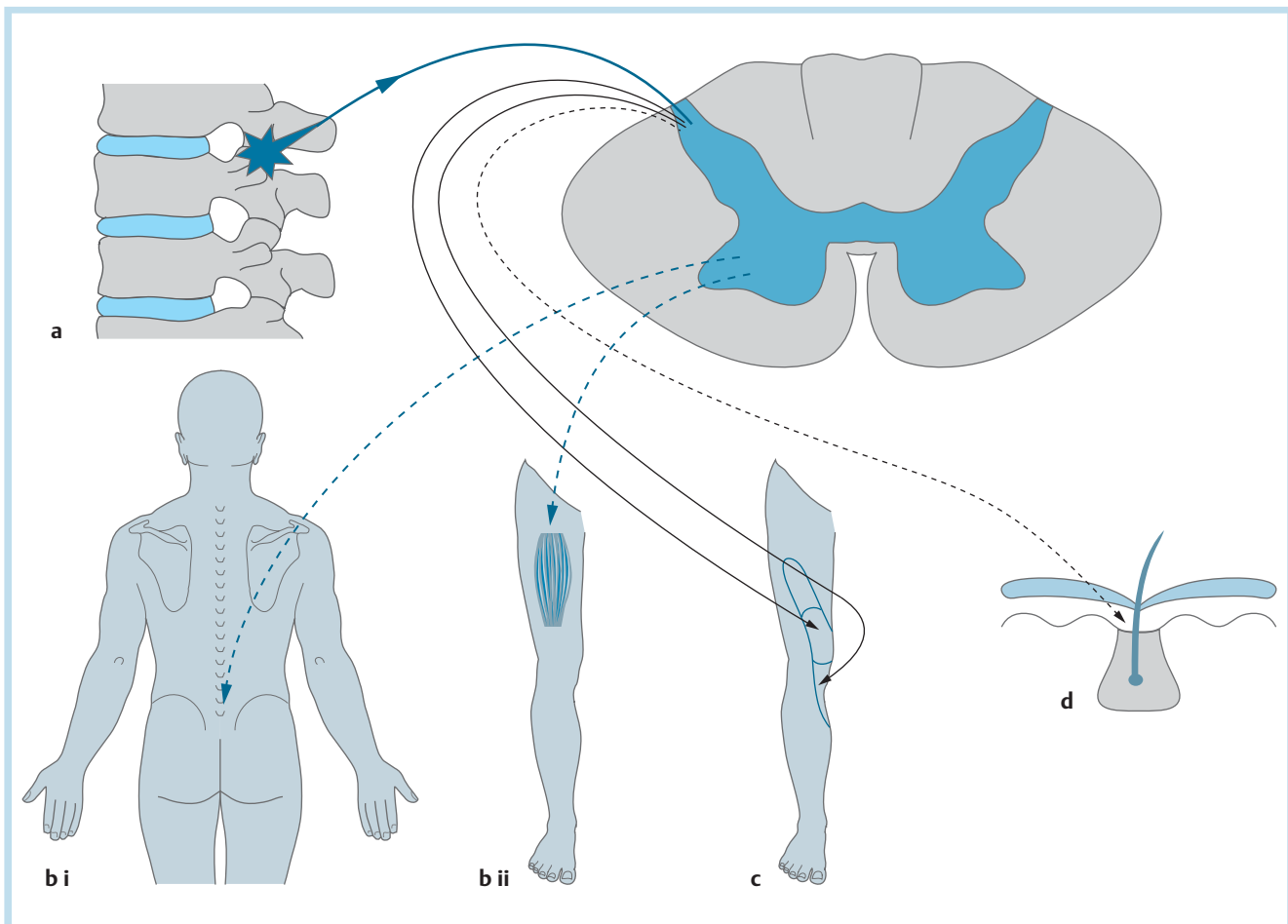
Beyond the primary muscular cause, persistent or recurrent MTrPs can also be related to other associated conditions such as cervical or lumbar disc herniation, lumbar disk lesions, cervical facet lesions, cervical radiculopathy, osteoarthritis of the knee, teres minor tendinitis, lateral epicondylitis, septic arthritis, herpes zoster or visceral disease [17, 25, 30, 33, 35, 50, 56, 57]. Referred pain can mimic other pains that range from joint disease and radiculopathies to visceral pain [27, 33, 35]. Thus, as part of the diagnostic process, clinicians should consider other conditions that may feature widespread pain [33].

### Diagnostic Criteria for Myofascial Pain

Despite the fact that muscle makes up more than half of the human body by weight, there has been limited organized focus on student training or research with respect to muscle pain [27, 33]. Myofascial experts consider MTrPs the most common yet misdiagnosed and inadequately treated component of non-articular musculoskeletal pain disorders [28, 33, 58, 59].

One of the main difficulties with diagnosing myofascial pain remains the subjective nature of the diagnosis [22, 32, 33, 62, 63]. Clinicians tend to only recognise and treat disorders that are diagnosed with objective tests, yet there exist no standardised objective measures to verify the presence of MTrPs [22, 63]. As a result of this lack of understanding, awareness and/or training, muscle pain is frequently overlooked and patients are often poorly diagnosed and inadequately treated thus perpetuating the chronic pain [22].

A number of studies have validated the physical examination of trigger points and a diagnosis of CMP should at a minimum be supported by a history of regional pain, a palpable taut band and



**Fig. 1 a–d** Facilitated spinal segment [58]. **a** Nociception originating in the L4 facet joint synapses on the dorsal horn. **b** A motor neuron within the ventral horn is activated on the L4 segmental level, causing a reflex spasm of muscles innervated by the same segment such as the paraspinal muscles (**b i**) and the rectus femoris muscle (**b ii**). **c** Dermatomal and sclerotomal

structures sharing the L4 segmental level may become sensitised and painful as a result of dorsal root reflexes. **d** Dorsal root reflexes in the L4 segment may also sensitise cutaneous structures, rendering them more painful.

tender spot(s) in this taut band containing specific sensitivity at the MTrP level and patient pain recognition on tender spot palpation with predicted pain referral expected from a trigger point in that muscle [22, 26–28, 33, 59, 64]. Other diagnostic features that are helpful, but not strictly required for CMP include local twitch responses (LTR), weakness, restricted range of motion and autonomic signs (e.g. in the skin; [28, 33, 59, 64]). Further research is necessary to test these diagnostic criteria. This is a critical step in the process of further establishing myofascial pain as a common, universally accepted and eminently treatable disease [22].

### Patient History

The regional distribution of pain in CMP described in a patient history is essential. Usually the patient is not able to locate the pain and it is described as deep, faint and aching [26–28, 33, 46, 48, 58, 59]. The diagnosis of CMP requires an awareness that the cause of pain may lay at a distance from the site of pain [27, 28, 33, 46, 58, 59]. The pain patterns give a good indication of the muscles that may be involved in CMP [27, 33, 59]. Each muscle

tends to have the same consistent and characteristic area of referred pain in different subjects [33, 59].

Referred pain from a muscle to another distant muscle has been demonstrated in animal studies [33, 45–48]. Sensory disturbance (e.g. paresthesias, dysesthesias, localised skin tenderness) may be noted in the same area where pain is referred [27, 28, 33, 46, 59]. Practitioners treating CMP should be familiar with the commonly affected muscles and the distribution of their referred pain patterns [27, 28, 33]. CMP is confirmed during careful examination applying the next three basic criteria: finding the taut band, identifying the specific spot tenderness, and pain recognition by the patient [27, 28, 33, 47, 59].

### Inspection

The most common mechanical maladaptations are: scoliosis or pelvic torsion, generalized or local joint laxity, leg length differences and foot structure issues. Characteristic posture changes can be apparent, such as head-forward and round shouldered, possibly matching particular muscle contractures [27, 33, 59]. Observation of the patient sitting, standing and walking is also important. Does the patient use a cane or a walker? Do certain



clothing items form a predisposing factor [27]? Scars from injuries or surgeries, changes in muscle volume or autonomic symptoms such as localized sweating, piloerection or goosebumps may also be noted [30, 33, 59].

### Range of Motion

Clinicians frequently recognise the relationship between CMP and joint hypomobility and several theories have been presented [19, 33]. First of all, increased tension of the taut muscular bands associated with a MTrP and facilitation of motor activity can maintain displacement stress on the joint [19, 30, 33]. Alternatively, the abnormal sensory input from the joint hypomobility may reflexively activate MTrPs [19]. It is also conceivable that MTrPs provide a nociceptive barrage to the dorsal horn neurons and facilitate joint hypomobility [19]. Practitioners are generally more inclined to relate ROM limitations to the joints. However, a quick improvement in joint mobility can often be measured after successful treatment of the MTrPs implying their importance [30, 64].

### Motor Features

Muscles affected by myofascial pain can show a loss of strength from 0.5 to 1 compared to the uninvolved side when measured on the Muscle Strength Grading Scale (Oxford Scale). Functional adaptation of muscle action occurs when muscle pain is present [52]. An ATP is a source of localized muscle pain and can result in a reduced activity of the painful muscle [58]. One possible explanation is that muscle contraction is simply limited to a degree below the threshold that can activate pain [27]. However, a MTrP in one muscle can inhibit effort or contractile force in another one, suggesting a role for central motor inhibition [27]. The most likely explanation is that the muscle containing the MTrP is relatively weakened as a result of reflex inhibition [27, 33]. This is rapidly reversible immediately on inactivation of the MTrP, also suggesting that the effect is caused by this inhibition of muscle action [27].

This common finding of muscle weakness associated with MTrPs is more frequently caused by inhibition from LTPs in the same or neighbouring muscles than ATPs [7, 27]. The LTP can also cause a disordered recruitment of muscles that work together to produce an action such as an orderly activation of muscles that produce abduction of the upper extremity [41]. Furthermore, motor and sympathetic output pathways are not only affected by nociceptive input, but afferent pathways (proprioception, somatosensory processing) are also influenced by tonic muscle nociception as well [52]. Along with the lack of reciprocal inhibition, this can cause co-contraction that reduces the movement quality and leads to clumsiness and an incoordination of fine movement [27]. Both ATPs and LTPs do not only interfere with host muscle efficiency, they can also have a reciprocal effect on the antagonist muscle [27]. Nociception-induced motor inhibition might prevent effective motor retraining and requires a shift in thinking: stop trying to restore normal motor control in case of chronic nociception [52]. This effect is increased with the chronicity of the condition and needs to be recognised and addressed during treatment [52]. Moreover, starting first with inactivation of the LTP cause of the weakness avoids the usual mistake of starting strength training, thus resulting in teaching the patient to use

substitutes instead of the inhibited muscle which, unfortunately, can make muscle function more abnormal [27]. In order to examine more closely the changed motor features in CMP patients further studies are needed [14, 27].

### Palpation

Careful palpation of the affected muscles is essential to confirm the diagnosis of CMP, first finding taut muscle bands and then localizing the MTrPs [27, 28, 33, 59]. LTrPs have the same clinical characteristics as ATrPs and therefore, their assessment is similar [8]. The taut band is a group of contracted muscle fibres, readily palpable, and tender at the region of greatest hardness [26, 27, 59]. A taut band in the distinguished muscle is found by palpating or by dragging the fingers perpendicular to the muscle fibres [27, 60]. Finding a taut band by palpation requires a combination of palpation skill, training, and critical clinical practice [27, 28, 30, 33, 58, 59].

Simons [61] suggests that an MTrP is always found in a taut band of skeletal muscle fibres and a taut band is the precursor of an MTrP. MTrP tenderness does not occur except in regions of muscle hardness, but regions of muscle hardness can occur without local or referred pain. Hence, muscle hardness or the taut band that occurs in the absence of pain may be the first abnormality. However, this sequence of events has not been systematically investigated [27].

The most important characteristic of an MTrP can be found within the taut band region: circumscribed spot(s) with pain or tenderness in a relatively small area that can be reasonably palpated using finger(s) or thumb [2, 3, 27, 28, 33, 59]. If the pain can induce or aggravate the patient's usual clinical complaint in response to digital compression, this indicates that the MTrP is responsible for (part of) those symptoms. It is very important to relate the found ATPs and LTPs to the patient's history [8, 27, 33, 59, 64].

The correct amount of pressure is also important; the pressure applied to the area should be consistent and approximately 4 kg/cm<sup>2</sup> [33]. The application of too much pressure can elicit pain in nearly all individuals [33, 45]. In human studies, the application of high pressure could also elicit referred tenderness from an LTP region or even normal muscle tissues [33, 45]. The pressure required to elicit referred pain from a compressed site is proportional to the degree of irritability (= amount of sensitised nociceptors) on that site [33].

In women PPTs are lower, signifying greater hypersensitivity to mechanical stimulation [27, 45]. There may also be increased central sensitisation in women, possibly related to weaker descending inhibition [45]. Certain effects of sex hormones on pain mechanisms are also known. However, the mechanism of gender differences in muscle pain remains to be identified [27, 45]. Typically, the patient has a positive "jump sign" when applying local pressure on the area. This jump sign should not be mistaken for a LTR [33]. The jump sign simply shows that the patient is reacting to pain or discomfort in the palpated area.

### Local Twitch Response

The taut band can contract sharply when stimulated with high pressure by strumming or needling the taut band – both resulting in a mechanical band deformation [27, 33, 59]. The LTR is a tran-

sient visual and/or palpable local contraction, when the tight muscle fibres are tensioning at the level of the MTrP [59]. LTR can rarely be elicited by low-pressure stimulation [33]. Often high pressure is required to elicit an LTR in an MTrP with low irritability, and vice versa [33]. The application of high-pressure compression during palpation cannot be recommended because patients are not able to tolerate it [33]. The occurrence of the LTR also depends on the irritability of the MTrP. It can be difficult to elicit an LTR in the case of a stimulated LTP [33].

Spinal mechanisms in the LTR have been documented extensively and require an intact spinal cord reflex arc [33, 38, 39]. EMG activity of an LTR elicited by stimulation of an MTrP can be recorded electromyographically in the taut band containing this MTrP. A needle tip can provide high pressure stimulation to the MTrP and can much easier cause an LTR than using finger palpation [33]. It is maximally elicited from the most tender region of the taut band, diminishes with increasing distance from this point, and is not elicited when recording from normal muscle as little as 1 cm from the taut band [27, 33]. Intramuscular needle EMG may not be used due to patient discomfort [8].

LTRs can also be monitored by sonography which was used to identify LTRs [27, 33, 62]. Rha et al. [54] suggest that ultrasonography was useful for detecting LTRs in the deep muscles where palpation is limited.

### Reliability and Limitations of Digital Palpation

Reliable identification of MTrPs is based on a high degree of skill following adequate training and sufficient clinical experience [2, 27, 33, 58, 59]. Numerous research studies and two systematic reviews have investigated the reproducibility of the MTrP examination for several muscles [3, 27, 33, 42, 49]. However, these studies focussed on the reliability of the MTrP diagnostic criteria without giving attention to the reliability of palpation protocols in identifying their exact location [3].

Best reproducibility of MTrPs has been reported for the upper trapezius, and an experienced physiotherapist can reliably identify MTrP locations in this muscle using a palpation protocol [3]. Considering that MTrP treatment requires the same MTrPs located and treated over repeated sessions, the intra-rater reliability of a palpation protocol could be relevant in supporting a clinical practice. The clinical relevance of the observed error is limited, and it should not influence standard treatment techniques, such as ischemic compression, ultrasound, or dry needling [3].

Although digital palpation is considered the gold standard for diagnosis, it has several limitations [22, 41, 49, 58]. Finger pressure techniques provide borderline reliability and require a standardised method accompanied by a calibration process to enhance reliability between raters [22]. Specifically, digital palpation does not provide an objective, reliable, and sensitive method of CMP diagnosis. It cannot objectively differentiate between ATPs, LTPs and palpably normal tissue or discriminate between superficial and deep MTrPs. Also, it does not produce quantitative comparisons of the tissue properties before and after treatment [22, 58].

In order to standardise the amount of applied pressure a pressure algometer can be used. It has been reported as a reliable and valid device to help assess myofascial pain [22, 33]. The scope of tenderness may be calculated as the mean PPT of all trigger point sites or as the percentage of sites that were tender using a standardised pressure; the latter measure appears to be the most predictive item [22]. The pressure algometer can also be useful for

**Tab. 3** Differences between radicular pain and trigger point-referred pain.

radicular pain	trigger point referred pain
specific dermatomal pattern may be present	map may extend across several dermatomes
loss of sensitivity in dermatome may be present – no local hyperalgesia	no loss of dermatomal sensitivity – local hyperalgesia
loss of motor power to the point of paralysis	weakness – no myotomal deficits
not induced by local muscle tissue pressure	induced with local muscle tissue pressure
loss of deep tendon reflex	no loss of deep tendon reflex

the assessment of the effectiveness of MTrP therapy as it provides quantitative comparisons of the tissue properties before and after treatment [33, 58].

### Neurological Examination

Myofascial pain can mimic radiculopathies, but there exist specific differences ([1, 16]; **Tab. 3**).

### Additional Assessment

No specific lab tests confirm a diagnosis of myofascial pain, but some tests can be helpful for finding predisposing conditions such as hypothyroidism, hypoglycemia, and potential vitamin deficiencies [25, 27, 59].

### Therapy Options for CMP

Clinical reasoning should always include all systems possibly participating in the patient's symptoms [33]. MTrPs in muscles innervated by the same nerve root could indicate a possible radiculopathy [30]. MTrPs surrounding joints could point out arthropathy or ligament lesions around this joint [50]. MTrPs in agonists are often accompanied by common tendon's lesion [30, 33]. MTrP pain during active contraction implies tendinopathy of the contracting muscle [33].

Sometimes activation of MTrPs can cause pain avoiding any movement that could interfere with the primary lesion's healing process. Muscle pain can be an important defence mechanism avoiding further injury before complete healing of the etiologic (e.g. neurological) lesion [33].

Comprehensive myofascial management should focus on removing all perpetuating factors (including all MTrPs!) and addressing sensitisation early in its development with centrally-acting pharmacologic agents, biofeedback and behavioral therapy [15, 58]. Non-steroidal anti-inflammatory drugs (NSAIDs) are less effective for CMP [46].

Physiotherapy can include non-invasive and invasive therapies for CMP [33, 60, 64]. Evidence-based practice established by 7 systemic reviews and 21 randomised controlled trials found in 2008, demonstrated that certain types of manual therapies and different kinds of modalities can serve as effective non-invasive treatments for MTrPs [4, 7, 13, 18, 33, 60, 64–66]. For physiotherapists dry needling is considered to be a clinically effective,

minimally invasive treatment [6, 11, 12, 30, 36, 37, 44, 58, 64, 65]. Patient education regarding CMP and home exercises should always be a part of a treatment programme [31, 64–67].

In summary, more research is necessary with respect to the different forms of (physio) therapy being applied to CMP [64, 65]. In addition, in order to develop standard guidelines and determine treatment outcome measures following CMP therapy clinical investigation is required [58].

## Conclusions



Physiotherapists should be aware that myofascial pain is often part of acute and chronic pain complaints which may originate away from the painful site.

One of the main difficulties in diagnosing myofascial pain remains the subjective nature of the diagnosis. Myofascial pain is diagnosed by applying four basic criteria: regional pain in patient history of finding the taut band, identifying the specific spot tenderness and the patient's pain recognition.

Reliable identification of myofascial trigger points is based on a high degree of skill following adequate training and sufficient clinical experience. Palpation within the clinical setting can reliably identify MTrP locations and can be relevant enough in supporting daily practice.

In addition to generating pain, myofascial pain can influence range of motion, strength and coordination, and hence prevent successful rehabilitation. Comprehensive myofascial management using non-invasive and invasive therapies, should primarily focus on removing all perpetuating factors including myofascial trigger points.

In summary, in order to adequately identify and treat a myofascial pain component better education in myofascial pain management and enhancing manual skills are necessary.

## Quintessence

Myofascial pain is often a component of acute and chronic pain. It may influence range of motion, strength and coordination.

Management of myofascial pain should include non-invasive and invasive therapies, with the central focus on removing perpetuating factors like myofascial trigger points.

## References

- 1 *Abd Jalil N, Awang MS, Omar M.* Scalene myofascial pain syndrome mimicking cervical disc prolapse: a report of two cases. *Malays J Med Sci* 2010; 17: 60–65
- 2 *Barbero M, Bertoli P, Cescon C et al.* Intra-rater reliability of an experienced physiotherapist in locating myofascial trigger points in upper trapezius muscle. *J Man Manip Ther* 2013; 20: 171–177
- 3 *Barbero M, Bertoli P, Cescon C et al.* Myofascial trigger points and innervation zone locations in upper trapezius muscles. *BMC Musculoskelet Disord* 2013; 8: 171–177
- 4 *Borg-Stein J, Iaccarino MA.* Myofascial Pain Treatments. *Phys Med Rehabil Clin N Am* 2014; 25: 357–374
- 5 *Bron C, Dommerholt JD.* Etiology of myofascial trigger points. *Curr Pain Headache Rep* 2013; 16: 439–444
- 6 *Cagnie B, Dewitte V, Barbe T et al.* Physiologic effects of dry needling. *Curr Pain Headache Rep* 2013; 17: 348–356
- 7 *Celik D, Kaya Mutlu E.* The relationship between latent trigger points and depression levels in healthy subjects. *Clin Rheumatol* 2013; 31: 907–911
- 8 *Celik D, Mutlu EK.* Clinical implication of latent myofascial trigger point. *Curr Pain Headache Rep* 2013; 17: 353–360
- 9 *Chandola HC, Chakraborty A.* Fibromyalgia and myofascial pain – a dilemma. *Indian J Anaesth* 2009; 53: 575–581
- 10 *Chou LW, Hsieh YL, Kao MJ et al.* Remote influences of acupuncture on the pain intensity and the amplitude changes of endplate noise in the myofascial trigger point of the upper trapezius muscle. *Arch Phys Med Rehabil* 2009; 90: 905–912
- 11 *Clewley D, Flynn TW, Koppenhaver S.* Trigger point dry needling as an adjunct treatment for a patient with adhesive capsulitis of the shoulder. *J Orthop Sports Phys Ther* 2014; 44: 92–101
- 12 *Cotchett MP, Munteanu SE, Landorf KB.* Effectiveness of trigger point dry needling for plantar heel pain: a randomized controlled trial; 2014, [www.ncbi.nlm.nih.gov/pubmed/24700136](http://www.ncbi.nlm.nih.gov/pubmed/24700136)
- 13 *Desai MJ, Bean MC, Heckman TW et al.* Treatment of myofascial pain. *Pain Manag* 2013; 3: 67–79
- 14 *Doraisamy MA, Anshul.* Effect of latent myofascial trigger points on strength measurements of the upper trapezius: a case-controlled trial. *Physiother Can* 2011; 63: 405–409
- 15 *Edwards J.* The importance of postural habits in perpetuating myofascial trigger point pain. *Acupunct Med* 2005; 23: 77–82
- 16 *Facco E.* Myofascial pain mimicking radiculars. *Acta Neurochir Suppl* 2005; 92: 147–150
- 17 *Fernández-Carnero J, Fernández-de-Las-Peñas C, de la Llave-Rincón AI et al.* Prevalence of and referred pain from myofascial trigger points in the forearm muscles in patients with lateral epicondylalgia. *Clin J Pain* 2007; 24: 353–360
- 18 *Fernández-de-las-Peñas CF, Sohrbeck Campo M, Fernandez Carnero J et al.* Manual therapies in myofascial trigger point treatment: a systematic review. *J Bodywork Movement Ther* 2005; 9: 27–34
- 19 *Fernández-de-las-Peñas C.* Interaction between trigger points and joint hypomobility: a clinical perspective. *J Man Manip Ther* 2009; 17: 74–77
- 20 *Fernández-de-las-Peñas C, Gröbli C, Ortega-Santiago R et al.* Referred pain from myofascial trigger points in head, neck, shoulder, and arm muscles reproduces pain symptoms in blue-collar (manual) and white-collar (office) workers. *Clin J Pain* 2013; 28: 511–518
- 21 *Fleckenstein J, Zaps D, Rüger LJ et al.* Discrepancy between prevalence and perceived effectiveness of treatment methods in myofascial pain: results of a cross-sectional, nationwide survey. *BMC Musculoskelet Disord* 2010; 11: 32
- 22 *Fricton J, Meister T, Seiroe M.* Scope of tenderness is best predictor of myofascial pain diagnosis. Defining diagnostic criteria for myofascial pain. *J Musculoskeletal Pain* [submitted]
- 23 *Ge HY.* Prevalence of myofascial trigger points in fibromyalgia: the overlap of two common problems. *Curr Pain Headache Rep* 2010; 14: 339–345
- 24 *Ge HY, Fernandez-de-las-Peñas C, Yue SW.* Myofascial trigger points: spontaneous electrical activity and its consequences for pain induction and propagation. *Chin Med* 2011; 6: 13
- 25 *Gerwin RD.* A review of myofascial pain and fibromyalgia – factors that promote their persistence. *Acupunct Med* 2005; 23: 131–134
- 26 *Gerwin RD.* The taut band and other mysteries of the trigger point: An examination of the mechanisms relevant to the development and maintenance of the trigger point. *J Musculoskeletal Pain* 2008; 16: 115–131
- 27 *Gerwin R.* Myofascial Pain: Here we are, where must we go? *J Musculoskeletal Pain* 2010; 18: 18
- 28 *Gerwin RD.* Diagnosing fibromyalgia and myofascial pain: a guide. *J Fam Pract* 2013; 62 (Suppl 1): S 19–S 25
- 29 *Grieve R, Barnett S, Coghil N et al.* The prevalence of latent myofascial trigger points and diagnostic criteria of the triceps surae and upper trapezius: a cross-sectional study. *Physiotherapy* 2013; 99: 278–284
- 30 *Gunn CC.* *The Gunn Approach to the Treatment of Chronic Pain.* New York: Churchill Livingstone; 1996
- 31 *Hanten WP, Olson SL, Butts NL et al.* Effectiveness of a home program of ischemic pressure followed by sustained stretch for treatment of myofascial trigger points. *Phys Ther* 2000; 80: 997–1003
- 32 *Harden RN, Bruehl SP, Gass S et al.* Signs and symptoms of myofascial pain: a national survey of pain management providers. *Clin J Pain* 2000; 16: 64–72
- 33 *Hong CZ.* Muscle pains. In: Braddom RL (ed) *Physical Medicine and Rehabilitation: Expert Consult – Online and Print.* Toronto: Elsevier; 2011



- 34 Hubbard DR, Berkoff GM. Myofascial trigger points show spontaneous needle EMG activity. *Spine* 1993; 18: 1803–1807
- 35 Jarrell J, Giamberardino MA, Robert M et al. Bedside testing for chronic pelvic pain: discriminating visceral from somatic pain. *Pain Res Treat* 2011; 2011: 1–6
- 36 Jayaseelan DJ, Moats N, Ricardo CR. Rehabilitation of proximal hamstring tendinopathy utilizing eccentric training, lumbopelvic stabilization, and trigger point dry needling: 2 case reports. *J Orthop Sports Phys Ther* 2014; 44: 198–205
- 37 Kietrys DM, Palombaro KM, Azzaretto E et al. Effectiveness of dry needling for upper-quarter myofascial pain: a systematic review and meta-analysis. *J Orthop Sports Phys Ther* 2013; 43: 620–634
- 38 Kuan TS, Hsieh YL, Chen SM et al. The myofascial trigger point region: correlation between the degree of irritability and the prevalence of endplate noise. *Am J Phys Med Rehabil* 2007; 86: 183–189
- 39 Kuan TS. Current studies on myofascial pain. *Curr Pain Headache Rep* 2009; 13: 365–369
- 40 Lucas KR, Rich PA, Polus BI. How common are latent myofascial trigger points in the scapular positioning muscles? *J Musculoskelet Pain* 2008; 16: 279–286
- 41 Lucas N, Macaskill P, Irwig L et al. Reliability of physical examination for diagnosis of myofascial trigger points: a systematic review of the literature. *Clin J Pain* 2009; 25: 80–89
- 42 Lucas KR, Rich PA, Polus BI. Muscle activation patterns in the scapular positioning muscles during loaded scapular plane elevation: the effects of latent myofascial trigger points. *Clin Biomech* 2010; 25: 765–770
- 43 Luo JJ, Dun NJ. Chronic pain: myofascial pain and fibromyalgia. *Int J Phys Med Rehabil* 2013; 1: e102
- 44 Mejuto-Vázquez MJ, Salom-Moreno J, Ortega-Santiago R et al. Short-term changes in neck pain, widespread pressure pain sensitivity, and cervical range of motion after the application of trigger point dry needling in patients with acute mechanical neck pain: a randomized clinical trial. *J Orthop Sports Phys Ther* 2014; 44: 252–260
- 45 Mense S, Simons DG. *Muscle Pain. Understanding its Nature, Diagnosis, and Treatment.* Philadelphia: Lippincott Williams & Wilkins; 2001
- 46 Mense S. Muscle pain: mechanisms and clinical significance. *Dtsch Arztebl Int* 2008; 105: 214–219
- 47 Mense S. How do muscle lesions such as latent and active trigger points influence central nociceptive neurons? *Journal of Musculoskeletal Pain* 2010; 18: 348–353
- 48 Mense S. Differences between myofascial trigger points and tender points. *Schmerz* 2011; 25: 93–103
- 49 Myburgh C, Larsen AH, Hartvigsen J. A systematic, critical review of manual palpation for identifying myofascial trigger points: evidence and clinical significance. *Arch Phys Med Rehabil* 2008; 89: 1169–1176
- 50 Nguyen BM. Myofascial trigger point, falls in the elderly, idiopathic knee pain and osteoarthritis: an alternative concept. *Med Hypotheses* 2013; 80: 806–809
- 51 Niddam DM, Chan RC, Lee SH et al. Central modulation of pain evoked from myofascial trigger point. *Clin J Pain* 2007; 23: 440–448
- 52 Nijs J, Daenen L, Cras P et al. Nociception affects motor output – a review on sensory-motor interaction with focus on clinical implications. *Clin J Pain* 2013; 28: 175–181
- 53 Patel N. Physiology of pain. In: Patel N, Kopf A (eds) *Guide to Pain Management in Low-resource Settings.* Seattle: IASP; 2010
- 54 Rha DW, Shin JC, Kim YK et al. Detecting local twitch responses of myofascial trigger points in the lower-back muscles using ultrasonography. *Arch Phys Med Rehabil* 2011; 92: 1576–1580
- 55 Russel LJ. Future perspectives in generalised musculoskeletal pains. *Best Pract Res Clin Rheumatol* 2011; 25: 321–331
- 56 Samuel AS, Peter AA, Ramanathan K. The association of active trigger points with lumbar disc lesions. *J Musculoskeletal Pain* 2007; 15: 11–18
- 57 Sari H, Akarirmak U, Uludag M. Active myofascial trigger points might be more frequent in patients with cervical radiculopathy. *Eur J Phys Rehabil Med* 2012; 48: 237–244
- 58 Shah J. New frontiers in the pathophysiology on myofascial pain. *Pain Practitioner* 2012; 22: 26–33
- 59 Simons DG, Travell JG. *Myofascial Pain and Dysfunction: The Trigger Point Manual.* Vol. 1. Baltimore: Williams & Wilkins; 1999
- 60 Simons DG. Understanding effective treatments of myofascial trigger points. *Journal of Bodywork and Movement Therapies* 2002; 6: 81–88
- 61 Simons DG. New views of myofascial trigger points: etiology and diagnosis (commentary). *Arch Phys Med Rehabil* 2008; 89: 157–159
- 62 Thomas K, Shankar H. Targeting myofascial taut bands by ultrasound. *Curr Pain Headache Rep* 2013; 17: 349
- 63 Though EA, Tough AR, White S et al. Variability of criteria used to diagnose myofascial trigger point pain-evidence from a review of the literature. *Clin J Pain* 2007; 23: 278–286
- 64 Timmermans F. Aktueller Stand der Überlegungen zum myofaszialen Schmerzsyndrom. *physioscience* 2006; 2: 25–32
- 65 Timmermans F. Een analyse van het myofasciale pijnsyndroom. Deel 2. Therapie. (Dutch). *Tijdschrift voor Kinesitherapie* 2008; 3: 19–27
- 66 Weiner DK. Office management of chronic pain in the elderly. *Am J Med* 2007; 130: 306–315
- 67 Wheeler AH. Myofascial pain disorders: theory to therapy. *Drugs* 2004; 64: 45–62
- 68 Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. *Pain* 2011; 152 (3): S2–S15
- 69 Zusman M. Forebrain-mediated sensitization of central pain pathways: “non-specific” pain and a new image for MT. *Man Ther* 2002; 7: 80–88