3 Trigger Point–Induced Disturbances

Both directly and indirectly, myofascial trigger points (mTrPs) can cause a multitude of disturbances in the neuromusculoskeletal system and therefore can be involved in the development and perpetuation of acute and chronic pain in a variety of ways.

3.1 Disturbances Induced Directly by Trigger Points

Directly caused by mTrPs:
- Pain
- Motor dysfunction
- Autonomic disturbances

3.1.1 Pain

MTrPs usually trigger **referred pain**, which often radiates to surprisingly widespread areas. (See, for example, the gluteus maximus, medius, and minimus muscles, ▶ Fig. 7.225 **a-b**). Occasionally, however, the pain also remains **locally** within the area of the mTrP itself (e.g., the deltoid muscle, ▶ Fig. 7.2).

The **quality of pain** is described by patients in a variety of ways: drawing, sticking, boring, burning, sharp or dull, clearly circumscribed or diffuse, superficial or "deep in the joint," etc. Trigger point activity often manifests itself in the form of **paresthesias**, **dysesthesias**, or **hypesthesias** (prickling or burning sensations, feeling of "ants crawling under the skin" [formication], feeling of heaviness, sensation of being "compressed by a surrounding band," or feeling that "something is swollen," or a numb sensation).

3.1.2 Motor Dysfunction

Both intermuscular and intramuscular **coordination disturbances** can be caused directly by mTrPs (Arendt-Nielsen and Graven-Nielsen 2008, Dejung 2009, Ibarra et al. 2011, Ivanichev 2007, Lucas 2008, Lucas et al. 2004, Travell and Simons 1999). Surface EMGs in muscles with mTrPs demonstrate delayed muscle activation, early fatigue, and slow recovery (Ge et al. 2012, 2014, Mense et al. 2001a, Travell and Simons 1999). By means of EMG measurements, Lucas et al. (2004, 2010) demonstrated that mTrPs in the scapular-stabilizing muscles cause significant changes in the timing of the activation pattern of all the shoulder muscles, and that treatment of the TrPs led to normalization of the muscle activation pattern (▶ Fig. 2.5). This shows that TrPs can cause motor system **dysfunction**. The altered movement pattern can result in inappropriate loading and overload, which can then cause pain and perpetuate chronic painful conditions.

MTrPs cause **weakening** of the affected muscles. The muscle weakness is primarily not associated with atrophy, but instead, it is reflexive or caused by pain, and can be detected by EMG (Travell and Simons 1999).

Motor disturbances caused by TrPs further manifest themselves in the form of **delayed muscle relaxation** (documented on EMG, ▶ Fig. 2.4), **deceleration** (▶ Table 2.1), and accelerated fatigability.

3.1.3 Autonomic Disturbances

mTrPs commonly result in vegetative (autonomic) phenomena (Travell and Simons 1999). They can manifest themselves in a variety of ways, both in the TrP itself as well as in the referred pain area, such as dizziness, nausea, increased sweating secretion, changes of the skin temperature and trophic skin changes in the area of the mTrPs (Fischer and Chang 1986, Haddad et al. 2012), and the referred pain areas (Haddad et al. 2012). They are interpreted as reflex responses of the sympathetic nervous system (Dejung 2009). Clinically, such vegetative-trophic disturbances occurring in the referred pain area of the mTrPs can manifest themselves in the form of insufficient capacity of the muscle tissue to regenerate (see Clinical Tip: Insufficient Capacity of the Muscle Tissue to Regenerate – Development of Satellite Trigger Points), Sudeck's disease (see Clinical Tip: Complex Regional Pain Syndrome), or carpal tunnel syndrome.

Clinical Tip

Insufficient Capacity of the Muscle Tissue to Regenerate — Development of Satellite Trigger Points

If the muscle tissue is not regenerating as quickly as expected, i.e., if pain persists after trigger point treatment for longer than 2 days, for example, this could be a clinical sign that autonomic disturbances impede appropriate response to the therapeutic stimulus of trigger point treatment (manual or dry needling). In these cases, it is very likely that a satellite trigger point, which originated in the referred pain zone of a primary trigger point, has been treated, and not the primary trigger point (see Trigger Point Chains, p. 188).

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Clinical Tip

Complex Regional Pain Syndrome (CRPS)
Myofascial disturbances can as well contribute to CRPS type 1, also known as reflex sympathetic dystrophy (RSD) and Sudeck's disease. For one thing, mTrPs are in the position to cause autonomic disturbances within their referred pain zone. For another, taut bands and connective tissue changes can cause neuromuscular entrapments with irritation of the neural structures, including the autonomic nerve fibers, and thus contribute to the development and perpetuation of CRPS. The therapeutic approach consists of locating and treating mTrPs in those muscles that radiate into the area affected by CRPS. For treatment of CRPS causing swelling, skin changes, and pain in the forearm and hand region, for example, the treatment area includes the subscapularis, supraspinatus, and serratus posterior superior muscles, among others. Treatment should also include treatment of the mTrPs responsible for the structures causing the entrapment of the neural structures involved — one such example is the treatment of the mTrPs responsible for taut bands in the anterior and middle scalene muscles that affect the brachial plexus. It is contraindicated to start local therapy at the site of the CRPS.

3.2 Disturbances Induced Indirectly by Trigger Points

MTrPs cause taut bands and connective tissue changes, which themselves can cause a range of problems.

3.2.1 Disturbances Resulting from Taut Bands

The taut bands induced by mTrPs are able to cause the following (see also ▶ Table 5.2):

- **Impaired intra- and intermuscular coordination:** If portions of a muscle are chronically shortened (taut bands), then intramuscular coordination is disturbed. Changes in intramuscular coordination cause alterations of the functional pattern of the affected muscle, which may possibly cause disruption of intermuscular coordination as well.
- **Muscle shortening:** If taut bands occur frequently and prominently, they dominate the total length of the muscle, and muscle shortening develops.
- **Restricted range of motion:** Muscle shortening is associated with restricted ROM.
- **Articular dysfunction:** Disturbance of intramuscular and intermuscular coordination result in malfunction and inappropriate loading of articular structures (Gunn 1996, Lewit 2007, Weissmann 2000). Two examples of such problems resulting from taut bands: (1) a shortened piriformis muscle contribute to dysfunction of the sacroiliac joint (SIJ); (2) a myofascially disturbed scapulohumeral rhythm can cause articular problems in the glenohumeral joint (▶ Fig. 3.1).
- **Faulty loading** and **muscle overload:** These result from impaired intra- and intermuscular coordination and muscle shortening.
- **Insertion tendinopathies:** As a result of shortening, taut bands produce inappropriate muscle loading or persistent overload at their insertion sites. This favors the formation and perpetuation of insertion tendinopathy. This can occur, for example, at the lateral epicondyle, the greater and lesser tubercle, the greater trochanter and the calcaneus. If the muscle attachment also lies within the referred pain area of other muscles, then the loading capacity of the insertion site is diminished because of the autonomic disturbances in the referred pain area, making the emergence of insertion tendinopathy even more likely (see Clinical Tips: Insertion Tendinopathy [p. 94], Search for Primary Trigger Points [p. 168, p.169] and Secondary TrPs and Satellite TrPs [p. 190]).
- **Disturbances of local perfusion and trophic disturbances:** Taut bands put increased pressure on the intramuscular blood vessels. Strangulation of the venous and arterial blood vessels develops, with resultant local perfusion disturbances and subsequent trophic changes.
- **Edema:** Taut bands also compromise extramuscular blood vessels (veins are particularly vulnerable) and lymphatics, thereby increasing the development of edema. In addition, taut bands inhibit the muscle's

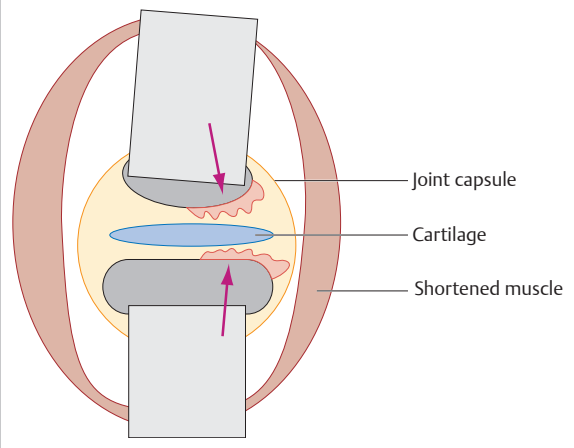

Fig. 3.1 Articular dysfunction due to muscle shortening: A shortened muscle resulting from mTrPs with taut bands can cause joint dysfunction (after Gunn 1996).

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ability to completely contract and relax. The pumping function of the muscles to transfer blood back toward the heart in the venous side of the circulatory system, primarily in the lower extremities (function of the muscles as a "peripheral heart"), is thus reduced, which further favors the development of edema. In this manner, trigger point–induced taut bands in the soleus muscle can reduce the efficiency of the muscle in its function as a "peripheral heart," while at the same time, the same taut bands present an increased resistance for venous return. The development of edema is thus significantly intensified.

- **Neuromuscular entrapment:** Neural structures penetrate the muscles in many places. If the muscle fibers at these locations are tensed as a result of mTrPs, they exert pressure on the nerve structures: The nerve tissue is then poorly perfused, and symptoms, such as dysesthesias and weakness, develop in the region supplied by the nerve (see also Entrapment Neuropathy, Chapter 6.1.3).

Clinical Tip

Insertion Tendinopathy — Formation and Approach to Treatment

As a result of shortening, taut bands produce inappropriate muscle loading or persistent overload at their attachment sites. This favors the formation and perpetuation of insertion tendinopathies; for example, in:

- The lateral epicondyle
- The deltoid tuberosity
- The greater or lesser tubercle
- The greater or lesser trochanter
- The calcaneus (heel spur)
- Tender spinous processes

If the insertion site (on the humeral lateral epicondyle, for instance) is additionally located within the referred pain area of a muscle with mTrPs (such as the supraspinatus muscle, ▶ Fig. 7.8), then the associated autonomic disturbance in the vicinity of the insertion site can reduce the site's loading capacity. Overload at the insertion site occurs more quickly and is more pronounced than it would otherwise have been if no autonomic malfunction were present, analogous to the development of Satellite Trigger Points, p. 188.

The causal treatment of the insertion problems (Gautschi 2012b) consists of:

- Deactivation of the primary trigger points (i.e., the same mTrPs that triggered the referred pain)
- Deactivation of the mTrPs in the local muscles that are responsible for the taut band, and therefore also for the persistent pathological loading at the insertion site
- Treatment of the insertion site (with technique II analog to a deep friction)

3.2.2 Disturbances Resulting from Connective Tissue Changes

The changes in connective tissue that develop in connection with the formation of mTrPs (including shortening, intra- and intermuscular adhesions and pathological crosslinks), for their part, can cause the following problems:

- **Restricted range of motion:** Fascial adhesions between adjacent muscles often restrict their mobility (e.g., the subscapularis muscle and the serratus anterior muscle).
- **Disturbances of intra- and intermuscular coordination:** Shortening and adhesions within the various muscle connective tissue layers interfere with the coordination in the muscle itself, on the one hand, and with the interaction of synergists and/or antagonists, on the other hand.
- **Articular dysfunction:** Inappropriate loading and irritation of the joints occur as a result of the disturbance of the intra- and intermuscular coordination and restricted ROM.
- **Peripheral chronification:** Connective tissue contractures (p. 58) superimpose the rigor complexes and thus fix them additionally structurally. This represents the peripheral chronification of myofascial pain (▶ Fig. 6.1 **b**).
- **Neuromyofascial entrapment:** Neural structures and the accompanying blood vessels penetrate the muscle fascia. If the resistance of the fascia is increased at these perforation sites, a "mini-entrapment" occurs. Motor, sensory, and autonomic nerve fibers as well as small arterioles and venous blood vessels are affected, thus limiting the optimal nerve and blood supply — and with it, the function — of the muscles (▶ Fig. 3.2; see also Chapter 8.4.3).
- **Disturbances of the neural and humoral transport pathways:** As a layer carrying blood vessels and nerves, the intramuscular connective tissue assumes an important supply function (Schünke 2000), which can be

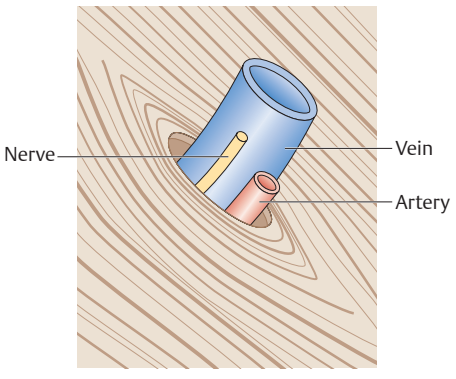

Fig. 3.2 Perforating triad at the passage point through the superficial muscle fascia: schematic representation of a fascial opening with a "perforating triad." At each opening, a perforating vein (blue), perforating artery (red), and perforating nerve (yellow) penetrate through the superficial muscle fascia (after Staubesand 1994, 1996).

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impaired by narrowing of the perimysial and endomysial structures [p. 58]).

- **Disturbances of the deep sensibility, interoception, and proprioception:** Dysfunction of the connective tissue of the muscle can change the functional capacity of the mechanoreceptors, such as the interstitial receptors (p. 42) and muscle spindles (p. 43), which are found in high numbers there. This local dysfunction then becomes a sustaining factor for dysfunction of the locomotor system overall, with lack of power and coordination. Adhesions between the subcutaneous functional gliding surface and the underlying muscle fibers causes a severe disturbance of the perception and transmission of stimuli (Sinz 2001). Disturbance of the sensory input results in altered motor output, and can thereby act as a sustaining factor for dysfunction of the locomotor system in terms of insufficient coordination and power.
- **Disturbances of nociception:** The nociceptive structures (p. 44) of the skeletal muscles are embedded in the connective tissue parts of the muscle (p. 41) and can become irritated by connective tissue disturbances.

3.3 Myofascial Syndrome

The sum of all the direct and indirect disturbances caused by mTrPs and fascial disorders is known as the myofascial syndrome (MFS). The terms "myofascial pain syndrome" (MPS), "myofascial dysfunction syndrome," and "myofascial pain and dysfunction syndrome" are used synonymously.

The clinical presentation for such functional, muscular-caused painful conditions and/or functional disturbances includes headaches, craniomandibular dysfunction (CMD), cervical syndrome with or without headaches, shoulder problems, elbow pain, lumbovertebral syndrome with or without leg pain, hip pain, groin pain, knee pain, achillodynia, foot pain, etc.

In everyday clinical practice, it is helpful to differentiate between primary and secondary MFS and between acute and chronic MFS (further information in Chapter 6.1.1).

- **Primary myofascial syndrome:** Here the cause for the development of mTrPs and fascial disorder lies within the muscle itself, so that a causal treatment is possible. The prognosis is good if the triggering and perpetuating factors are known and can be concurrently incorporated in the treatment concept.
- **Secondary myofascial syndrome:** Here the myofascial pain and its consequences are the result of another underlying disturbance, which may be arthrogenic, neurogenic, viscerogenic, or psychogenic. Causal treatment with trigger point therapy is therefore not possible. Whenever possible, causal treatment should be given; if causal therapy cannot be carried out, then symptomatic treatment to alleviate pain should be considered (see Clinical Tip, p. 196).
- **Acute myofascial pain syndrome:** Diagnosis and treatment are usually simple and can be performed without difficulty (Travell and Simons 1999, Dejung 2009).
- **Chronic myofascial syndrome:** Usually multiple active mTrPs and disorder of fascia are involved. Persistent activity of primary mTrPs favors the development of secondary TrPs in synergistic and antagonistic muscles as well as satellite TrPs in the referred pain region of the primary mTrP. In this manner, entire trigger point chains can develop. In chronic myofascial pain, the treatment of the reactive connective tissue changes is indispensable (see further information).

Summary

Trigger Point–Induced Disturbances

Both directly and indirectly, mTrPs cause a multitude of disturbances in the neuromusculoskeletal system and can therefore be involved in the development and perpetuation of chronic pain in a variety of ways.

- Disturbances that are induced directly by TrPs are pain (local and radiating), motor dysfunction, and autonomic disturbances.
- Disturbances that are indirectly induced by TrPs are caused by taut bands and/or connective tissue changes as follows:
  - Disturbance of intra- and intermuscular coordination: The economy of movement is hampered by taut bands and connective tissue changes. Inappropriate loading and overload of the muscles and joints occur as a result.
  - Taut bands cause shortening of the muscles, resulting in reduced mobility and articular dysfunction (Lewit 2007). Fascial adhesions between adjacent muscles often cause drastically restricted ROM.
  - If the taut bands compromise blood vessels, then perfusion disturbances (development of edema) and trophic disturbances develop.
  - Neuromuscular entrapment: Neural structures penetrate the muscles in many places. If the muscle fibers at these locations are tensed as a result of mTrPs, they exert pressure on the nerve structures: The nerve tissue is poorly perfused, and symptoms, such as dysesthesias and weakness, develop in the region supplied by the nerve.
  - Connective tissue contractures overlie and fixate the rigor complexes, which signifies the peripheral chronification of myofascial pain.
  - Disturbances of deep sensibility, proprioception, and nociception: The receptors located in the muscle connective tissue can become irritated by connective tissue dysfunction.

The sum of all the direct and indirect disturbances induced by mTrPs and associated fascial disorder is known as the myofascial syndrome (MFS).

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