

Trigger points – Diagnosis and treatment concepts with special reference to extracorporeal shock waves

Myofascial trigger points (MTrPs) have had an unsteady history in terms of the evaluation of their medical significance since they were first described 70 years ago [73]. Despite its prominence, the trigger point theory, originally developed for medical diagnosis and therapy, used to be disputed by medical professionals for many years on grounds of lack of objective verifiability and scientific evidence. Among doctors, trigger point therapy was only performed by a small group of specialists who were highly skilled in manual techniques and focused on functional treatments, improving the procedure on the basis of various therapy approaches [1, 10, 25, 32, 53, 71]. At the same time, trigger point therapy became a standard procedure in paramedical symptom-oriented treatment, which has achieved widespread acceptance among patients as a result of its excellent success rates [24].

Renaissance of trigger point therapy

During the last decade, trigger point therapy has been increasingly used by orthopaedists practising conservative treatment as a new procedure with shock wave application. This is the result of two parallel scientific developments. One is muscle pain research, which during the last 30 years has come to consider muscle pain as a form of pain of its own [46] that is distinctly different from nerve and organ pain.

The other is the wider use of extracorporeal shock wave therapy to include the treatment of the most diverse types of tissue: tendons [5, 18, 51, 56], bones [7], skin [59], cardiac muscle [80] and, more recently, skeletal muscles.

As a result, shock wave treatment of skeletal muscles represents an empirically extended indication for regenerative shock wave therapy. During the last few years, it has come to be referred to as "trigger point shock wave therapy" because shock waves – better than any other method – are able to induce the referred pain that is characteristic of trigger points and treat the clinical symptoms associated with these trigger points [4, 22, 47].

Myofascial trigger points (MTrPs) and muscle pain

Trigger points are hyperirritable painful sites in a muscle that occur in the form of mostly palpable local indurations in a taut band (■ Fig. 1).

Trigger points are a specific type of ischaemic local muscle pain and are frequently associated with referred pain. Contrary to normal muscle pain, muscular trigger points have a limited self-healing capacity.

Pathophysiology of muscle pain

Muscle pain is mediated through the excitation of nociceptors by vaso-neuroactive substances (bradykinin, prostaglandin, serotonin, histamine) and high concentrations of potassium and H⁺.

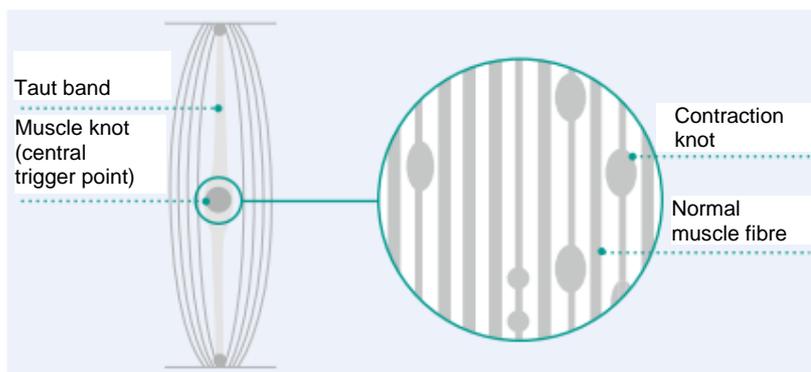


Fig. 1 ▲ Trigger point complex. *Left:* Macroscopic muscle knot (central trigger point) located in the taut band. *Right:* Magnified detail with contraction knot resulting from local sarcomere contractures within individual muscle fibres. (modified from [66], courtesy of Level10 Buchverlag, Heilbronn)

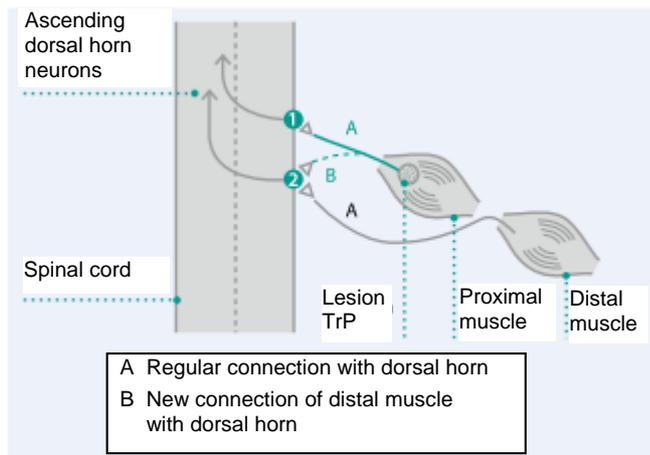


Fig. 2 ▲ Convergence-projection theory. By opening ineffective synapses (B), the nociceptive excitation of a proximal muscle affected by trigger points and normally connected with the spinal cord via dorsal horn neuron 1 is centrally transmitted via dorsal horn neuron 2 of the distal muscle. *TrP* trigger point. (modified from [46], courtesy of Level10 Buchverlag, Heilbronn)

The vasoneuroactive substances are released by subjectively painful and objectively tissue-damaging stimuli.

Muscle nociceptors, once activated, release neuropeptides (substance P, calcitonin-gene-related peptide [CGRP] and somatostatin), which lead to the formation of local tissue oedema. This phenomenon, which is referred to as neurogenic inflammation [79], does not cause any severe spontaneous pain, but rather dysaesthesia and a feeling of weakness.

In most cases, painfulness ends with the formation of local tissue oedema, and the lesion is repaired. In adverse conditions, however, the oedema may grow in size and thus cause a vicious circle. According to Travell and Simons [72], this mechanism is co-responsible for the formation of trigger points.

Longer-lasting sensitisation of muscle nociceptors gives rise to two clinical phenomena: hyperalgesia and allodynia. Persistent muscle pain is characterised by a high degree of subjective suffering. If it persists for over six months, it often becomes therapy-resistant and frequently results in pain chronification [46].

Referred pain

A peculiarity of muscle pain is its ability to induce referred pain in remote deep subcutaneous tissue (muscles, tendons, fasciae, joints – excluding the viscera) or in the skin without any primary nociceptive irritation. Pain referral is characteristic of muscular trigger points. It is explained on the basis of the convergence-projection theory [57] which stipulates that afferent nociceptive information from the muscle takes a wrong path in the spinal cord and reaches somatotopically unrelated dorsal horn neurons (■ Fig. 2). In patients, this results in erroneous pain localisation in the brain. There are characteristic pain referral patterns for almost all muscles of the musculoskeletal system.

» A peculiarity of muscle pain is its ability to induce referred pain.

Referred pain primarily occurs when local muscle pain is very intense or when it has persisted for a long period of time or has been triggered repeatedly.

Referred pain is primarily induced in the trigger point itself. However, it can also be evoked at a distance of up to 4 cm from the trigger point, more frequently around active trigger points (47 %) than around latent trigger points (23 % [29]). The degree of local and referred pain perceived and its extension depend on the extent of trigger point irritation and not on the size of the muscle. Moreover, referred pain is also determined by the external pressure applied during the examination.

Referred pain is not specific to the myofascial pain syndrome. It can also originate in tissue other than muscles, such as large joints, facet joints, ligaments, periosteum, fasciae, tendons, scars and especially inner organs.

This means that muscular referred pain is a characteristic but unspecific symptom, which frequently requires extensive differential diagnosis [46, 60].

Pathophysiology of myofascial trigger points

According to the acknowledged trigger point hypothesis [66], trigger points originate from local sarcomere contracture caused by an excessive release of acetylcholine at the motor end-plate due to overuse or trauma (■ Fig. 3). Strong muscular contraction causes intramuscular vascular compression and, consequently, local ischaemia, which releases vasoneuroactive substances and causes severe local pain at the trigger point. The resulting neurogenic inflammation with tissue oedema aggravates the energy crisis, preventing the uptake of calcium into the sarcoplasmic reticulum and, consequently, the elimination of the sarcomere contracture. This results in a vicious circle of persisting trigger points.

Recent in-vivo examinations of human tissue have revealed increased levels of vasoneuroactive substances in the immediate vicinity of trigger points, which can be considered to confirm the trigger point hypothesis [61].

Activators

Trigger points are found to a higher degree in people who engage in little physical activity in their everyday lives, but have occasional intervals of severe physical strain. Muscles primarily affected by trigger points include the postural muscles in the neck, shoulder and pelvic girdle.

In most cases, trigger points are activated by acute, persistent or repeated mechanical overstrain. In addition to this, there are perpetuating and aggravating factors, which are frequently different from trigger point activators.

Acute muscular overstrain occurs as a result of direct trauma (sprain, torn muscle fibres, crush) or eccentric and concentric contractions and causes damage to the muscle cell membrane. Persistent overstrain is caused by postural anomalies. Repeated overstrain occurs in case of weak muscle contractions (repetitive strain injury). It is explained with the "Cinderella hypothesis" [26] which postulates that selective overloading of the small type 1 muscle fibres occurs.

Moreover, trigger points are a concomitant of the following disorders:

- postural anomalies,
- osteoarthritis,
- tendinitis,
- discopathy,
- radiculopathy,
- peripheral nerve compression syndromes,
- blood circulation disorders.

Other activators and perpetuating factors include visceral, rheumatic, endocrinological and oncological disorders, nutritional deficits (lack of vitamins B1, B6, B12, folic acid, vitamins C and D, iron, magnesium, zinc) as well as general pain syndromes. Moist and cold climatic conditions have an additional negative effect. Since the severity of trigger point symptoms is primarily modulated by the autonomic nervous system [16], stress, lack of sleep and psychological and psychiatric disorders also play a key role.

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Abstract

The 70-year-old trigger point theory has experienced a growing scientific confirmation and clinical significance as a consequence of recent muscle pain research. The trigger point pain formation is caused by high levels of vaso-neuroactive substances. Depending on intensity and duration of the muscle stimulus the central pain processing is modified and leads to characteristic referred pain patterns. The most effective conventional forms of treatment are aimed at a direct mechanical manipulation of the trigger point as are new forms of therapy with focused and radial shock waves. By using high pressures the focused shock waves in particular are suitable to provoke local

and referred pain and thus simplify the trigger point diagnosis. The empirically found therapeutic effect of shock waves on muscles is hypothetical and can be explained in analogy with validated reactions of shock waves in non-muscle tissues. Overall, the shock wave therapy on muscles represents a confirmation and extension of the existing trigger point therapy. It seems to be suitable for treating functional muscular disorders and myofascial pain syndromes within the locomotor system.

Keywords

Trigger point · Extracorporeal shock wave · Muscle · Myofascial pain · Referred pain

Differentiation of trigger points

Different types of trigger points are distinguished by the clinical symptoms they present.

- Active trigger points are symptom-producing trigger points that may cause spontaneous local or referred pain and paraesthesia. Active trigger points generally develop at middle age and cause pain.
- Passive (latent) trigger points are clinically inapparent trigger points, but have the same potential as active trigger points when provoked by pressure or tension. They usually develop at an advanced age in life and manifest primarily through limited mobility.
- Satellite trigger points develop in the area of pain referred by a primary trigger point.

- Secondary trigger points occur in muscles that are adjacent to a primary trigger point location (synergists, antagonists). They develop as a result of a motion disorder caused by a primary trigger point.

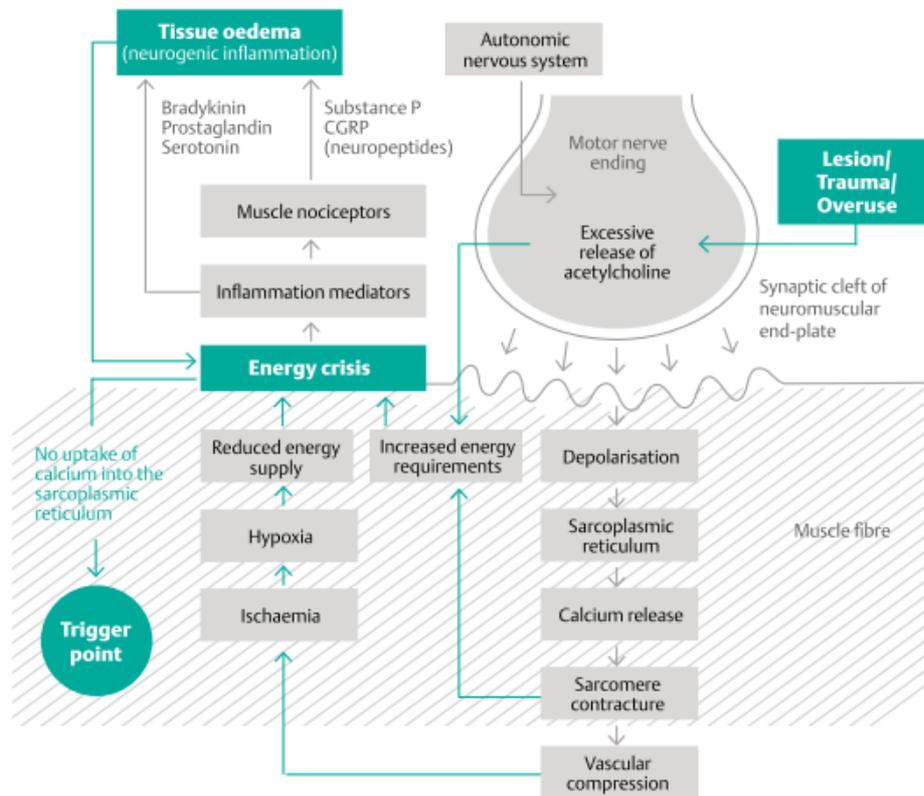


Fig. 3 ◀ Integrated hypothesis of trigger point formation: this hypothesis postulates that the energy crisis causes nociception by the release of sensitising substances and prevents actin-myosin separation. *CGRP* "calcitonin-gene-related peptide". (modified from [66], courtesy of Level10 Buchverlag, Heilbronn)

Symptoms

The clinical symptoms of trigger points are manifold. Most importantly, they cause pain perceived as deep local tissue pain or referred pain, which may be partly disabling and which is similar in intensity to a heart attack, bone fracture or colic. Besides pain, trigger points also cause sensory, motor and autonomic disorders. Sensory disturbances imitate neurological loss with temporary numbness and paraesthesia (dysaesthesia, hypaesthesia). Owing to central and peripheral sensitisation, trigger points also lead to hyperalgesia and allodynia.

Motor deficits manifest as hypertonia, stiffness, muscle shortening, muscle weakness (reflex inhibition without atrophy), coordination disorders, excessive activity, early fatigue and delayed recovery after strain [46].

In very rare cases, autonomic disorders may be accompanied by changes in skin temperature resulting from vasoconstriction or vasodilation, lacrimation, piloerection and proprioceptive deficits with unsteady gait, vertigo and tinnitus. Finally, direct pressure on trigger points during sleep may cause sleep disturbance.

Long-term sequelae

If the disorder persists for a long time, the trigger point may experience structural, partly irreversible alterations. Biopsies of muscular contraction knots have revealed a loss of myofibrils and an empty sarcolemma [54, 77].

Disorders resulting from muscle overload occur in the areas surrounding the trigger points. These functional deficits spread from one muscle to another and thus give rise to muscle chain reactions [40].

Where tendons are affected, the overload will cause insertional tendinitis [25] and attachment trigger points [50].

The point in time at which these sequential changes occur is impossible to predict.

Prevalence

Muscle pain, besides joint pain, is one of the main causes of musculoskeletal diseases requiring treatment [58]. It is the main reason why people need to see a doctor, take sick leave or file disability claims [67] and affects up to 85 % of the population [64]. Owing to its high prevalence, the treatment of muscle pain poses both a medical and a socioeconomic challenge. Myofascial trigger points play a key role in the genesis of muscle pain [65].

Diagnosis

Muscular trigger points are diagnosed primarily by history and on the basis of clinical criteria [10, 32, 53]. Laboratory diagnosis, currently available imaging techniques and electromyography are not suitable for identifying trigger points in daily clinical practice.

So far, no uniform international standards have been established for the diagnosis of muscular trigger points [70]. The criteria generally adopted are the localisation of trigger points by palpation of painful spots in taut bands and the recognition of the induced pain. Characteristics include referred pain, twitch response, restriction of mobility by muscle shortening, muscle weakness without neurological loss and diagnosed autonomous disorders.

Owing to the subjective nature of palpatory examination, the majority of examiners find it difficult to diagnose trigger points [30]. This has also been confirmed by recent review articles which criticise the quality of studies that paint a positive picture of the reliability of manual palpation [42, 48]. Trigger point specialists, however, attribute high intertester reliability to the manual examination method [11].

After the introduction of extracorporeal shock wave therapy in the treatment of myofascial syndromes, diagnostic options have also increased. According to the experience gathered by the authors of this article, especially focused shock waves, which can reach peak pressures of between 10 and 100 megapascals (MPa), enable easy and precise induction of local and referred pain. This is attributable to the fact that the intensity of local muscle pain and the strength of the pressure applied determine the severity of referred pain (see "Referred pain" section for details).

Trigger points and acupuncture

Over 90 % of trigger points coincide anatomically with acupuncture points treated for musculoskeletal pain relief. The clinical correspondence of trigger points and acupuncture points in terms of the local pain indication is 70 to 80 % [12, 45].

Muscles affected by pain referral from trigger points and the paths of acupuncture meridians have an over 80 % overlap rate [13]. These investigations suggest that the modern approach to myofascial pain and the 2000 years old system of meridians describe an identical pain mechanism [13, 32, 53].

Classic MTrP therapies

Trigger point therapy has a twofold purpose: to alleviate pain and to permanently eliminate the trigger point complex. To achieve these objectives, trigger point therapy uses treatment modalities aimed at breaking the vicious circle described in the integrated trigger point hypothesis through

- separation of fixed actin-myosin links [71],
- elimination of contraction knots [46],
- improvement in local blood circulation through reactive hyperaemia and resolution of the ischaemia-induced energy crisis [63],
- reduced concentration of vaso-neuroactive substances [61],
- muscle relaxation.

Among the non-invasive procedures employed, manual therapy techniques, including massage, transverse friction, ischaemic compression, release, spray and stretch and auto-stretching, play a dominating role [9, 39, 71].

Invasive treatments performed are trigger point injections with local anaesthetics [73], normal saline solution [15], corticosteroids [52], botulinum toxin A [78] and dry needling [69]. Invasive treatments are described to be more effective than non-invasive methods [35]. Injections generally give better results than dry needling [36]. A combination of lidocaine with corticosteroids provides fast pain relief [74]. As far as dry needling is concerned, the deep method has shown to be more effective than superficial dry needling [33].

The methods used to reduce pain symptoms include ultrasound, electrotherapy (TENS, microcurrent) and laser.

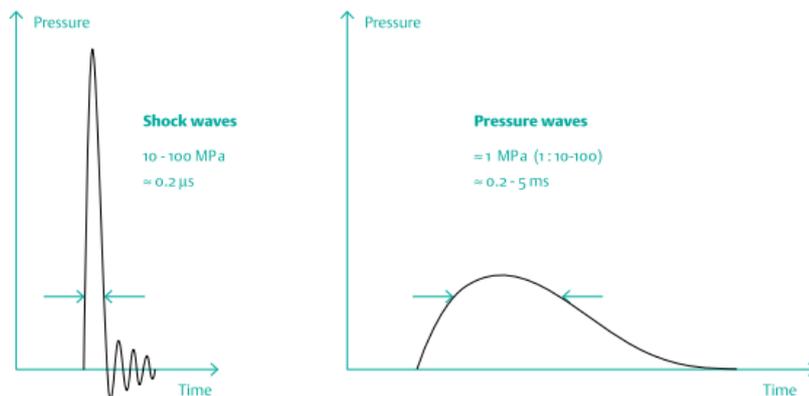


Fig. 4 ▲ Pressure graph of different types of shock waves. *Left*: focused shock waves (f-ESWT). *Right*: radial shock waves (r-ESWT). (courtesy of Level10 Buchverlag, Heilbronn)

Medication-based treatment with paracetamol, NSAIDs, metamizol, muscle relaxants, tricyclic antidepressants and opioids is effective as adjuvant therapy, but alone is not sufficient to ensure successful trigger point treatment.

Various follow-up measures are recommended: repeated slight movement over the full range of motion, moist heat, electric stimulation aimed at slight cyclic muscle contraction and biofeedback.

Overall, the clinical evidence of the various treatment options for trigger point therapy is not strong enough to allow evidence-based guidelines to be established. Judging by the results of a questionnaire survey, the majority of medical therapists are not satisfied with the therapy options available at the moment [14].

Shock wave application to muscles

Muscle treatment with focused shock waves (f-ESWT) was first discussed in individual publications in the late 1990s [37, 41]. This method was developed as an alternative to manual trigger point treatment, which basically consisted in applying external mechanical energy to the muscle. The objective of these treatments was to alleviate pain and to reduce muscle tone and muscle shortening.

The consistent use of shock waves in orthopaedics for the treatment of myofascial syndromes started in early 2000. Initially, radial

shock waves (r-ESWT) were used for these applications [2, 19], but focused shock waves (f-ESWT) followed shortly after [3, 4, 22, 47].

The shock wave systems employed in muscle treatment are the same as those originally developed for traditional indications of shock wave therapy. Based on the results of scientific studies into the tissue compatibility of shock waves, the recommended energy level for shock wave application should be below 0.5 mJ/mm². All other treatment parameters and application techniques have been determined empirically so far [20, 21].

In order to maximise treatment safety and standardise shock wave therapy, DIGEST e.V. (German-speaking International Society for Extracorporeal Shockwave Therapy, www.digest-ev.de) has established guidelines for the treatment of different types of tissue, including muscles. These guidelines have also been adopted by the ISMST (International Society for Medical Shockwave Treatment, www.ismst.com).

Hypothetical mechanisms of action

The mechanisms of action of shock waves in skeletal muscle treatment have not been fully established. To explain the clinical success of this therapy, the validated effects of shock waves on non-muscular tissue are taken as a basis [17, 55] and associated with the pathophysiological mechanisms of painful spots in muscles.

Based on the knowledge and information gathered to date, the following mechanisms of action of shock waves in muscle treatment can be discussed:

- separation of fixed actin-myosin links by the input of mechanical energy perpendicular to the muscle fibre direction [61, 71],
- improvement of blood circulation through reactive hyperaemia and angiogenesis [38, 61, 76],
- dilution of vasoneuroactive substances as a result of reactive hyperaemia [46, 61],
- pain modulation through release of substance P [28, 43] and CGRP [68],
- pain modulation through release and synthesis of nitric oxide [44, 49],
- selective degeneration of C-fibres [27],
- pain modulation according to the gate control theory [23, 75],
- biological mechanotransduction [31, 34, 49].

Research conducted by Shah et al. [61, 62] provides extensive information on the trigger point phenomenon and indirectly confirms the previously cited theories. In fact, these studies revealed a significant reduction in vasoneuroactive substances and neuropeptides after dry needling. These changes were similar to those associated with the pain modulation effect of focused shock waves applied to non-muscular tissue [27, 28, 43, 49, 68, 76].

Focused (f-ESWT) and radial shock waves (r-ESWT)

Shock waves are acoustic waves characterised by high peak pressures (10–100 MPa for f-ESWT; 0.1–1 MPa for r-ESWT), short rise times (10–100 nanoseconds [ns] for f-ESWT; 0.5–1 milliseconds [ms] for r-ESWT), short duration (0.2–0.5 microseconds [µs] for f-ESWT; 0.2–0.5 milliseconds [ms] for r-ESWT) and a low tensile wave component (negative pressure) which is 10 % of the peak pressure (■ Fig. 4). Shock waves propagate through a medium without any loss and without any changes in the acoustic impedance and release their energy at acoustic interfaces.

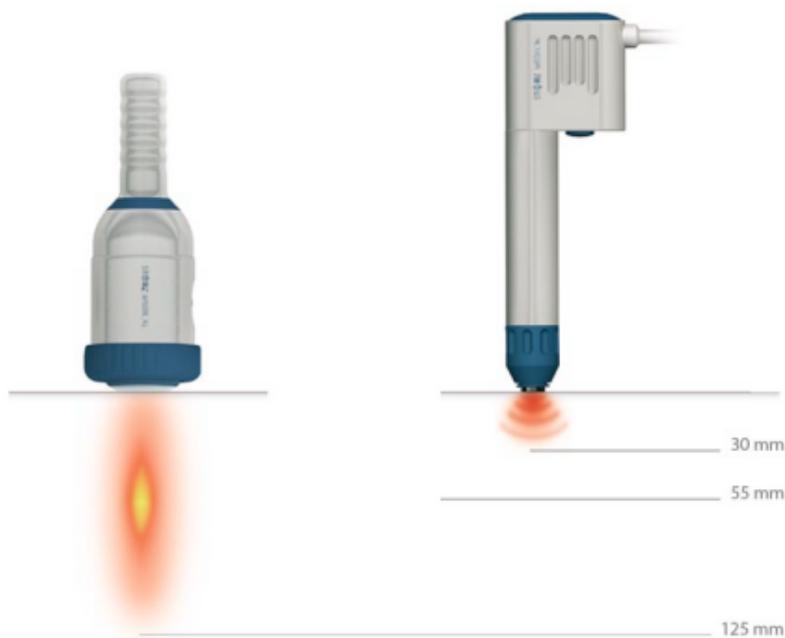


Fig. 5 ▲ Comparison between focused (f-ESWT) and radial (r-ESWT) shock waves. *Left:* f-ESWT with deep focus in tissue. *Right:* r-ESWT with radial shock wave propagation, small penetration depth and maximum energy at the skin surface which decreases with depth. (courtesy of Storz Medical AG)

Focused and radial shock waves differ in the way they are generated. Focused shock waves are generated electrically in a therapy head (electrohydraulic, electromagnetic or piezoelectric principle) and then concentrated in a therapeutic focus located in the tissue by optical reflection (■ Fig. 5 left).

This explains the beneficial property of focused shock waves to irritate trigger points with high local tissue pressures and to induce local and referred pain. Their disadvantage is that they can only be applied to a small treatment zone.

In physical terms, radial shock waves are ballistic pressure waves. They are generated by the collision of solid bodies. A projectile is accelerated by compressed air and hits a shock transmitter. At the skin surface, this transmitter transmits its kinetic energy in the form of a radial pressure wave which propagates divergently in the tissue. The energy intensity reduces with the square of the distance from the skin surface (■ Fig. 5 right; [8]).

Radial shock waves can be used on extensive muscle regions.

The disadvantages are their minimal penetration depth and the fact that they cannot be focused in the tissue, which explains the lower frequency of pain referral.

Extracorporeal shock wave therapy of skeletal muscles

Focused shock waves can be used for both diagnosis and therapy. After initial palpation, the muscle to be treated is examined with shock waves to identify local and referred pain. The objective is to enable patients to recognise the induced pain as part of the pain that has caused them to seek medical treatment. The actual therapy session takes place immediately afterwards to keep track of the identified pain regions. Treatment is performed applying 300 to 600 shock waves at an energy level of 0.1 – 0.35 mJ/mm² and a frequency of 4 Hz. The selected energy level depends on the pain intensity perceived by the patient in the target area. Pain is rated on the basis of a VRS (verbal rating scale) from 0 to 10, and the energy level used should not produce pain exceeding levels 5 to 8 on the VRS.

After pain has been reduced by over 50 %, treatment is continued in the same manner on the next painful spot. Treatment frequency varies between 1 or 2 sessions a week for chronic disorders and daily sessions for acute pathologies. In general, 4 to 10 sessions are necessary per muscle region.

Radial shock waves are used primarily for therapy purposes, especially for the local treatment of trigger points at high energy levels and for large-area muscle smoothing at lower energy settings.

After preliminary palpation, hardened muscle regions are treated locally with 500 to 1000 shock waves. Again, the energy level used depends on the patient's level of perceived pain intensity. Smaller muscles are treated with 2.0 to 2.6 bar, larger muscles with 3.0 to 4.0 bar. The shock frequency is 4 to 20 Hz.

Large-area muscle treatment in muscle fibre direction after the local treatment of several painful spots is performed at low energy levels of between 1.2 and 1.8 bar. Depending on the size of the muscle, 1000 to 4000 shock waves are applied by moving the shock transmitter slowly in muscle fibre direction from the distal to the proximal end. The shock frequency is 10 to 20 Hz. The treatment frequency per muscle region is the same as for f-ESWT.

Contraindications and diagnosis

According to DIGEST (www.digest-ev.de) and ISMST (www.ismst.com) guidelines, the structures listed below should not be in the shock wave focus for longer periods and are therefore to be considered absolute contraindications to shock wave therapy:

- malignant tumours,
- pulmonary tissue,
- epiphysial plate
- large vessels,
- nerves.

Antithrombotic therapies represent relative contraindications.

In view of the cited contraindications, recommended basic diagnostic procedures include an X-ray of the target region and ultrasound examination of the local soft tissue. If necessary, additional imaging techniques must be used.



Fig. 6 ◀ Haematoma colour changes in gluteal muscle region after radial shock wave application (r-ESWT)

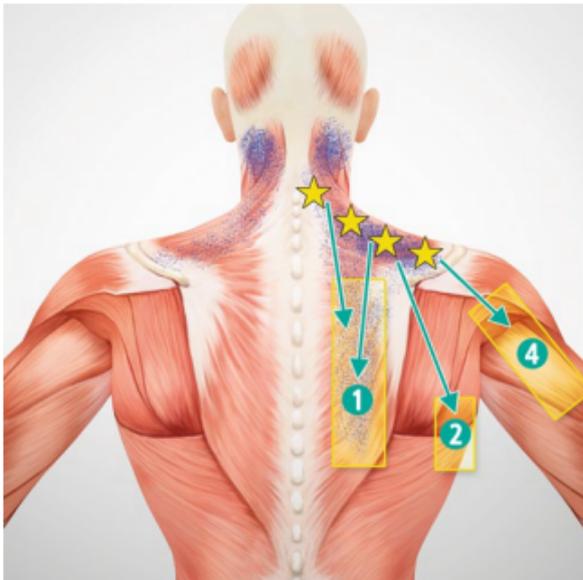


Fig. 7 ◀ Dorsal pain referral from descending part of trapezius muscle. *Yellow stars*: trigger point sites frequently identified with shock waves. *Yellow rectangles*: typical regions of referred pain identified with shock waves. *Blue clouds*: regions of referred pain described by Travell. (courtesy of Level10 Buchverlag, Heilbronn)

Side effects and complications

If the energy level selected for shock wave therapy is too high and localisation is incorrect, complications such as pneumothorax, tissue and organ bleeding or neural damage may occur. This applies especially to focused shock wave therapy.

Still, serious complications are unlikely to develop during shock wave therapy if the correct application technique, energy level and penetration depth are used.

However, temporary side effects have been found to occur in many cases, and patients should be made aware of them before treatment is started. During the first 1 or 2 days after the treatment, local pain may worsen temporarily.

In many patients, the application of radial shock waves causes immediate local skin reddening, pressure marks caused by the shock transmitter and haematoma colour changes (■ Fig. 6) which usually persist for 1 to 2 weeks. When shock waves are applied to the upper cervical region, headache and temporary ear sounds may occur or worsen.

Concomitant therapies

In general, no concomitant muscle treatments are necessary during shock wave therapy. Whenever additional treatment methods beyond shock wave application are used, any risk of muscular "overtherapy" should be avoided. This is crucial because shock waves already produce a strong tissue stimulus.

Adjuvant stretching treatments should be performed after the shock wave therapy session in order to maintain the reduction in muscle tone. In the treatment of joint blockage, manual therapy may prove beneficial after the first 2 to 3 shock wave treatments and initial muscle tone reduction. Moreover, many colleagues suggest combination with acupuncture and osteopathy.

Medical strengthening therapy (MST) performed 1 to 2 days after a shock wave treatment session is another possible option. However, it is important that during the entire shock wave therapy period and up to six weeks after its termination patients should not train at their maximum strength level and maximum muscle shortening to avoid activation of new trigger points [6].

Insertional tendinitis, which frequently develops in chronic syndromes, requires separate treatment because its nociceptive activity would interfere with the muscle therapy.

Patients suffering from chronic pain syndromes and severe pain should use NSAIDs or paracetamol shortly before and the day after shock wave therapy. Where necessary, adjuvants such as myorelaxants, antidepressants or neuroleptics can be used.

Local anti-inflammatory infiltrations at the target sites prior to shock wave therapy should be avoided.

Clinical examples

According to the experience gathered by the authors of this article, shock wave therapy has proved to provide good results in the treatment of all acute and chronic conditions of muscular dysfunction with increased muscle tone and muscle shortening, provided that there is no dominant pathology that permanently irritates the muscle and that the muscle does not present any structural damage. Indications include:

- chronic and acute medial and lower cervical syndrome with brachialgia, dorsalgia and cephalalgia,
- periarticular shoulder pain with restricted mobility,
- tendomyopathy of forearm extensors and flexors with and without radial epicondylopathy,

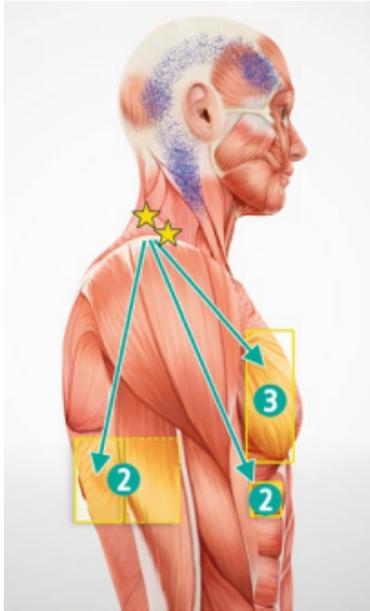


Fig. 8 ▲ Pain referral pattern from descending part of trapezius muscle to the lateral and ventral aspects. (courtesy of Level10 Buchverlag, Heilbronn)

- dorsalgia,
- chronic and acute lumbago with and without pseudoradicular irradiation,
- adductor tendinopathy and shortening,
- shortening of thigh extensors and flexors in cases of patellar chondropathy and patellar tendinitis,
- peroneal muscle and anterior tibial stress syndrome,
- shin splint,
- calf muscle shortening with tendency to cramp, with and without achillodynia,
- shortening of plantar foot muscles with and without plantar fasciitis,
- metatarsalgia,
- sports injuries without structural muscular discontinuity.

Cervical syndrome with brachialgia, dorsalgia and cephalgia

Cervical syndromes are among the most frequent indications for successful shock wave therapy. Muscular disorders associated with these syndromes manifest as pain and restricted mobility.

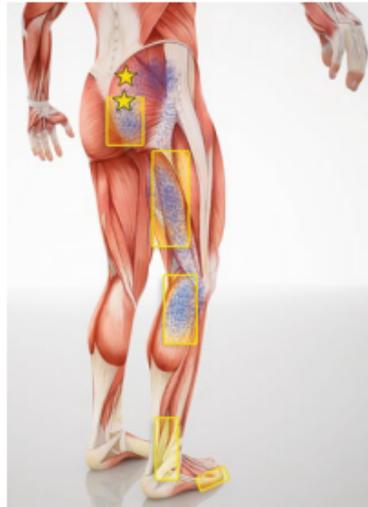


Fig. 9 ▲ Pain referral from dorsal gluteal muscles. (courtesy of Level10 Buchverlag, Heilbronn)

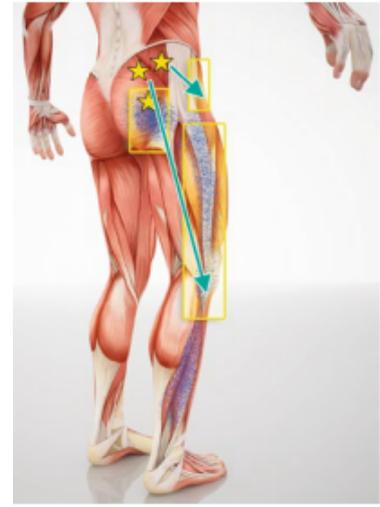


Fig. 10 ▲ Pain referral from lateral gluteal muscles. (courtesy of Level10 Buchverlag, Heilbronn)

» Cervical syndromes are frequent indications for successful shock wave therapy.

The referred pain induced by shock waves is mostly consistent with the pain information provided by patients and has a high level of recognition. Shock waves are applied both locally, using focused shock waves, and to larger target areas, using radial shock waves.

Owing to the great number of potential target regions, the muscles to be treated must be selected carefully. The descending part of the trapezius is among the most frequently affected muscles. It is responsible for local pain as well as for pain referral to the thorax, arm and head. Trapezius muscle shortening causes restriction of rotation on the affected side and reduced lateroflexion on the opposite side.

Localisation of trigger points. In the neck angle and in lateral direction. Paravertebral region at the C4-7 level and in lateral direction up to the acromioclavicular (AC) joint. In the entire ventral muscle portion. Since trigger points are distributed over the entire muscle, systematic muscle scanning with shock waves is necessary.

Referred pain induced by shock waves is different from the pain resulting from manual provocation. Contrary to reports in many medical publications dealing with the trigger point phenomenon, pain is hardly ever referred to the head. In many cases, the severity and frequency of occurrence of headache diminish as a result of the muscle treatment.

Pain referral patterns. (see [■] Figs. 7, 8).

The most frequent pain referral is to the dorsal interscapular region up to the level of the medial and inferior scapula, occasionally up to the thoracolumbar junction. Potential referred pain can be induced in all mentioned locations.

The second most frequent pain referral, which primarily originates from the ventral aspect of the trapezius muscle, is to the medial lateral thorax along the axillary line and ventrally to this line.

The third most frequent pain referral is to the supraclavicular and subclavicular regions of the ventral thorax, to the medial aspect of the pectoralis muscle and to the parasternal region. Pain referral to the thorax is suspected to originate primarily in the free ventral muscle margin and in the dorsal paravertebral region at the C7 level.

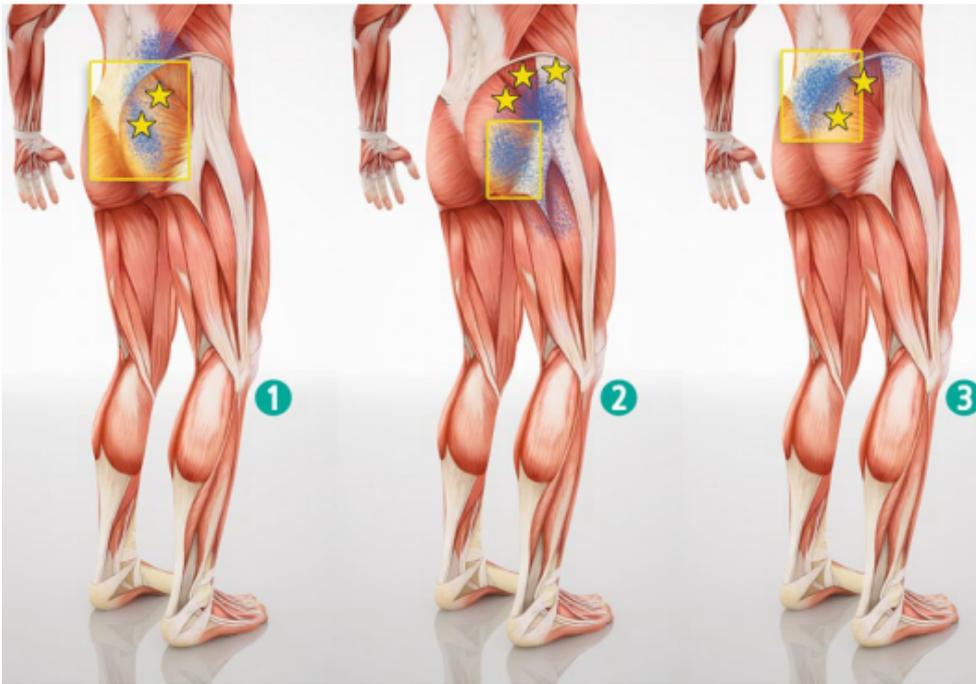


Fig. 11 ◀ Regional pain referral from the gluteus muscles: to the iliosacral joint (1), ischial bone (2) and inferior lumbar spine (3). (courtesy of Level10 Buchverlag, Heilbronn)

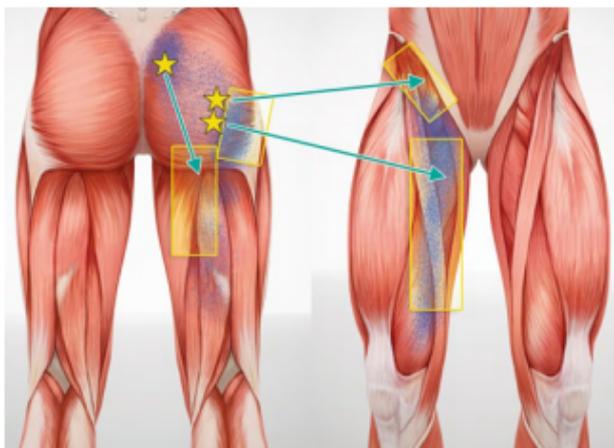


Fig. 12 ◀ Pain referral from external rotator muscles. (courtesy of Level10 Buchverlag, Heilbronn)

The fourth most frequent pain referral is to the dorsolateral upper arm at the level of the posterior part of the deltoid muscle or at the infraspinatus muscle level. In general, it can be induced in the lateral aspect of the trapezius muscle from the ventral or dorsal side. In many cases, pain is referred up to the ulnar or radial epicondyle, rarely up to the wrist on the same side.

Treatment example. *Anamnesis:* pain in the neck and temple regions, occurring regularly during computer work and aggravated by stress.

High sensitivity to cold and draft. Occasional pain radiation to the arm, not dermatome-related. Previously interpreted as tension headache and treated with muscle relaxants and massage without any significant improvement of symptoms.

Clinical findings: severe restriction of cervical spine rotation, soft touch at range of motion limit, myogelosis in the free margins of the trapezius muscle in a palpable taut band. Strong pain when affected area is pinched; pain radiates to the side of the head. Pain is recognised by patient as the pain complained of. No neurological loss.

Clinical diagnosis: muscle-related headache with trigger points in the descending part of the trapezius muscle.

Therapy: in this case only r-ESWT with a total of 6000 shock waves (3000 per side) to the free margins of the horizontal and descending parts of the trapezius muscle, applied at a pressure of 2.4 bar. 400 to 500 shock waves are applied to the spots in which pain is most severe (possible induction of referred pain) until pain has been reduced by about 50%. Followed by large-area muscle smoothing in fibre direction. Total number of therapy sessions is 4 to 6 at weekly intervals.

Results: reduced pain and improved mobility already after the first session; complete pain relief after 6 sessions.

Concomitant therapies: optional acupuncture, thermotherapy. Strengthening exercises after completion of treatment.

Lumbago and glutealgia with and without pseudoradicular sciatica

Chronic lumbago has shown to respond exceptionally well to shock wave therapy. In general, shock waves are applied to the upper gluteal region.

These muscles are involved in most chronic pain syndromes in the lumbar/pelvic/hip region. Their irritation is attributable to diverse causative factors, among which functional disorders or structural pathologies of the lumbar spine and hip joint. Clinically speaking, the pain discussed here is gluteal pain, which causes pain referral to the inferior lumbar spine and to the leg, primarily in laterodorsal direction and therefore referred to as "pseudoradicular" pain. Referred pain can be induced with a high degree of recognition.

Localisation of trigger points. Primarily along and ventrally to a connecting line between the posterior superior iliac spine and the greater trochanter and in the upper third of the gluteus medius and minimus muscles towards the iliac crest. Additional trigger points in the gluteus maximus muscle in the parasacral region and above the ischial bone. If pain is perceived at the greater trochanter, the trigger points are located in the dorsal region of the gluteus medius and maximus muscles and in the external rotators.

Pain referral patterns. Pain is referred from the dorsal aspect of the gluteus medius muscle (■ Fig. 9) to the buttocks, iliac bone, dorsolateral thigh up to the knee and from there in distal direction along the dorsolateral lower leg down to the ankle and foot. Pain referral is also from the middle gluteus medius and minimus muscles to the buttocks and lateral aspect of the thigh to just below the knee (■ Fig. 10), while pain radiates from the anterior gluteus medius and minimus muscles to the inguinal region and adductors. Only in very rare cases is referred pain regionally confined to the centre of the inferior lumbar spine, ischial bone and iliosacral joint (■ Fig. 11). The external rotators (■ Fig. 12), which are always included in the treatment of the dorsal gluteus muscles, often refer pain to the inguinal region and ventrally to the hip, thigh and adductors.

Treatment example. *Anamnesis:* pain in the lower lumbar spine area, in the paravertebral and gluteal regions and occasionally in the lateral aspect of the thigh, radiating approximately to the knee region.

No loss of sensitivity. Pain caused by strain while standing or after prolonged sitting or lying down (in supine or lateral position). Severe sleep disturbance.

Clinical findings: basically no restriction of lumbar spine mobility, slight restriction of internal rotation of hip joints, negative facet provocation. Deep palpation by exerting thumb pressure on the gluteus muscles cranially from the greater trochanter (gluteus minimus muscle) and along a connecting line between the greater trochanter and the posterior superior iliac spine (gluteus medius muscle) causes strong local pain which radiates towards the sacrum distally to the greater trochanter and towards the inguinal region. Upon provocation with f-ESWT, the patient recognises the radiation and perceives maximum pain. No neurological loss.

Diagnostic imaging: the lumbar MRI image reveals slight degenerative changes of the facet joints and intervertebral disks without nerve root or spinal canal compression.

Clinical diagnosis: lumbago with pseudosciatica caused by trigger points in the gluteal muscles.

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Therapy: f-ESWT for diagnosis with provocation of pain referral, immediately followed by therapy. Thanks to the penetration depth of up to 12 cm, the deep external rotators (primarily the piriformis muscle) are treated at the same time. Treatment with f-ESWT with 1000 to 2000 shock waves (0.15 to 0.25 mJ/mm²), of which about 200 to 300 per trigger point area until pain has been reduced by about 50 %. Followed by r-ESWT. Total number of shock waves per side: 3000, with large-area gluteal muscle smoothing in fibre direction. r-ESWT reaches primarily the superficial muscles (gluteus maximus and medius muscles). Total number of therapy sessions is 4 to 6 at weekly intervals.

Results: almost complete elimination of sleep disturbance after the first session and substantial reduction of pain radiation to the leg. Complete absence of symptoms after 4 sessions.

- **Effective treatment requires adequate differential diagnosis, precise local diagnosis and early trigger point therapy.**

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Conflict of interests. Dr Gleitz is a consultant to Storz Medical AG, Lohstampfstrasse 8, 8274 Tägerwil, Switzerland, providing therapy recommendations for the use of focused and radial shock waves. Dr Hornig declares that there is no conflict of interests.

Conclusions for practice

- **Muscle pain research conducted during the last few years and the experience gathered in the use of shock wave therapy of skeletal muscles confirm the important role of myofascial trigger points.**
- **The use of shock wave therapy allows the diagnostic and therapeutic options of conservative orthopaedics to be extended because it is primarily concerned with functional disorders and pain syndromes.**
- **Knowledge of muscle-specific pain referral is important to ensure correct differential diagnosis of disorders manifesting as dysaesthesia or muscle weakness, which in many cases cannot be diagnosed neurologically, and bring the muscle in the focus of medical treatment.**
- **Surgeons can refine indications for surgery and improve post-operative results by treating residual muscle pain.**

(English translation of the article in German: "Triggerpunkte – Diagnose und Behandlungskonzepte unter besonderer Berücksichtigung extrakorporaler Stosswellen", M. Gleitz, K. Hornig, in: Orthopäde 2 / 2012)

References (Listed in German)

1. Baldry P (1993) Acupuncture, trigger points and musculoskeletal pain. 2. Aufl. Churchill Livingstone, Edinburgh
2. Bauermeister W (2003) Die Behandlung myofaszialer Schmerzsyndrome durch Trigger-ESWT am Beispiel chronischer Lumbalgien und Lumboischialgien. Paper presented at the 51. Jahrestagung der Vereinigung Süddeutscher Orthopäden e. V.
3. Bauermeister W (2005) Diagnose und Therapie des myofaszialen Triggerpunkt-Syndroms durch Lokalisierung und Stimulation sensibilisierter Nozizeptoren mit fokussierten elektrohydraulischen Stosswellen. MOT 5:65–74
4. Bauermeister W (2007) Myofasziales Triggerpunkt-Syndrom: Diagnose und Therapie durch Stosswellen. Extracta Orthop 5:12–19
5. Buch M, Knorr U, Fleming L et al (2002) Extracorporeal shockwave therapy in symptomatic heel spurs. An overview. Orthopade 31(7):637–644
6. Byrne C, Twist C, Eston R (2004) Neuromuscular function after exercise-induced muscle damage: theoretical and applied implications. Sports Med 34(1):49–69
7. Cacchio A, Giordano L, Colafarina O et al (2009) Extracorporeal shock-wave therapy compared with surgery for hypertrophic long-bone nonunions. J Bone Joint Surg [Am] 91(11):2589–2597
8. Cleveland RO, Chitnis PV, McClure SR (2007) Acoustic field of a ballistic shock wave therapy device. Ultrasound Med Biol 33(8):1327–1335
9. Cyriax J (1977) Deep massage. Physiotherapy 63(2):60–61
10. Dejung B, Gröbli C, Colla F, Weissmann R (2003) Triggerpunkt-Therapie. 1. Aufl. Huber, Bern
11. Dommerholt J, Bron C, Franssen J (2006) Myofascial trigger points: an evidence-informed review. J Manu Manipul Ther 14(4):203–221
12. Dorsher PT (2008) Can classical acupuncture points and trigger points be compared in the treatment of pain disorders? Birch's analysis revisited. J Altern Complement Med 14(4):353–359
13. Dorsher PT (2009) Myofascial referred-pain data provide physiologic evidence of acupuncture meridians. J Pain 10(7):723–731
14. Fleckenstein J, Zaps D, Ruger LJ et al (2010) Discrepancy between prevalence and perceived effectiveness of treatment methods in myofascial pain syndrome: results of a cross-sectional, nationwide survey. BMC Musculoskelet Disord 11:32
15. Frost FA, Jessen B, Siggaard-Andersen J (1980) A control, double-blind comparison of mepivacaine injection versus saline injection for myofascial pain. Lancet 1(8167):499–500
16. Ge HY, Fernandez-de-las-Penas C, Arendt-Nielsen L (2006) Sympathetic facilitation of hyperalgesia-evoked from myofascial tender and trigger points in patients with unilateral shoulder pain. Clin Neurophysiol 117(7):1545–1550
17. Gerdesmeyer L, Weil L Jr (2007) Extracorporeal shockwave therapy. Data Trace Publishing Company, Towson
18. Gerdesmeyer L, Wagenpfeil S, Haake M et al (2003) Extracorporeal shock wave therapy for the treatment of chronic calcifying tendonitis of the rotator cuff: a randomized controlled trial. JAMA 290(19):2573–2580
19. Gleitz M (2003) Stosswellen zur Behandlung myofaszialer Schmerzen in der Orthopädie: Eine neue Therapiemöglichkeit. Paper presented at the 51. Jahrestagung der Vereinigung Süddeutscher Orthopäden e. V.
20. Gleitz M (2011) Myofasziale Syndrome und Triggerpunkte – Stoßwellentherapie in der Praxis. 1. Aufl. Level-10-Buchverlag Daniela Bamberg, Heilbronn
21. Gleitz M, Rädcl R (2008) Die orthopädische Stosswellentherapie bei myofaszialen Erkrankungen. MOT 4:218–223
22. Gleitz M, Dreisilker U, Rädcl R (2006) Die Orthopädische Trigger-Stosswellen-Therapie mit radialen und fokussierten Stosswellen: Eine Standortbestimmung. Orthop Praxis 42(5):303–312
23. Gregor M, Zimmermann M (1972) Characteristics of spinal neurones responding to cutaneous myelinated and unmyelinated fibres. J Physiol 221(3):555–576
24. Gröbli C, Dejung B (2003) Nichtmedikamentöse Therapie myofaszialer Schmerzen. Schmerz 17: 475–480
25. Gunn C (1996) The Gunn approach to the treatment of chronic pain: IMS for myofascial pain of radiculopathic origin. 2nd edn. Churchill Livingstone, Edinburgh
26. Hägg G (2003) The Cinderella hypothesis. In: Johannson H et al. (eds) Chronic work-related myalgia. Gävle University Press, Gävle, Sweden, pp 127–32
27. Hausdorf J, Lemmens MA, Heck KD et al (2008a) Selective loss of unmyelinated nerve fibers after extracorporeal shockwave application to the musculoskeletal system. Neuroscience 155(1):138–144
28. Hausdorf J, Lemmens MA, Kaplan S et al (2008b) Extracorporeal shockwave application to the distal femur of rabbits diminishes the number of neurons immunoreactive for substance P in dorsal root ganglia L5. Brain Res 1207:96–101
29. Hong CZ, Chen Y, Twehous D (1996) Pressure threshold for referred pain by compression on the trigger point and adjacent areas. J Musculoskelet Pain 4(3):61–79
30. Hsieh CY, Hong CZ, Adams AH et al (2000) Interexaminer reliability of the palpation of trigger points in the trunk and lower limb muscles. Arch Phys Med Rehabil 81(3):258–264
31. Ingber DE (2006) Cellular mechanotransduction: putting all the pieces together again. FASEB J 20(7): 811–827
32. Irnich D (2009) Leitfaden Triggerpunkte. Urban & Fischer Elsevier, München

33. Itoh K, Katsumi Y, Hirota S, Kitakoji H (2007) Randomised trial of trigger point acupuncture compared with other acupuncture for treatment of chronic neck pain. *Complement Ther Med* 15(3):172–179
34. Jaalouk DE, Lammerding J (2009) Mechanotransduction gone awry. *Nat Rev Mol Cell Biol* 10(1):63–73
35. Kalichman L, Vulfsoms S (2010) Dry needling in the management of musculoskeletal pain. *J Am Board Fam Med* 23(5):640–646
36. Kamanli A, Kaya A, Ardicoglu O et al (2005) Comparison of lidocaine injection, botulinum toxin injection, and dry needling to trigger points in myofascial pain syndrome. *Rheumatol Int* 25(8):604–611
37. Kraus M, Reinhart E, Krause H, Reuther J (1999) Low energy extracorporeal shockwave therapy (ESWT) for treatment of myogelosis of the masseter muscle. *Mund Kiefer Gesichtschir* 3(1):20–23
38. Kuo YR, Wang CT, Wang FS et al (2009) Extracorporeal shock-wave therapy enhanced wound healing via increasing topical blood perfusion and tissue regeneration in a rat model of STZ-induced diabetes. *Wound Repair Regen* 17(4):522–530
39. Lange M (1931) *Die Muskelhärten (Myogelosen)*. Lehmann's, München
40. Lewit K (1987) Chain reactions in functional disorders of the locomotor system. *Cas Lek Cesk* 126(42):1310–1312
41. Lohse-Busch H, Kraemer M, Reime U (1997) A pilot investigation into the effects of extracorporeal shock waves on muscular dysfunction in children with spastic movement disorders. *Schmerz* 11(2):108–112
42. Lucas N, Macaskill P, Irwig L et al (2009) Reliability of physical examination for diagnosis of myofascial trigger points: a systematic review of the literature. *Clin J Pain* 25(1):80–89
43. Maier M, Averbek B, Milz S et al (2003) Substance P and prostaglandin E2 release after shock wave application to the rabbit femur. *Clin Orthop Relat Res* 406:237–245
44. Mariotto S, Prati AC de, Cavaliere E et al (2009) Extracorporeal shock wave therapy in inflammatory diseases: molecular mechanism that triggers anti-inflammatory action. *Curr Med Chem* 16(19):2366–2372
45. Melzack R, Stillwell DM, Fox EJ (1977) Trigger points and acupuncture points for pain: correlations and implications. *Pain* 3(1):3–23
46. Mense S, Simons DG (2001) *Muscle pain: understanding its nature, diagnosis, and treatment*. Lippincott Williams & Wilkins, Philadelphia
47. Müller-Ehrenberg H (2005) *Diagnose und Therapie myofaszialer Schmerzsyndrome mit fokussierten Stosswellen*. MOT 5:1–6
48. Myburgh C, Larsen AH, Hartvigsen J (2008) A systematic, critical review of manual palpation for identifying myofascial trigger points: evidence and clinical significance. *Arch Phys Med Rehabil* 89(6):1169–1176
49. Neuland H, Duchstein H, Mei W (2004) Grundzüge der molekularbiologischen Wirkung der extrakorporalen Stosswellen am menschlichen Organismus – In-vitro- und In-vivo-Untersuchung. *Orthop Praxis* 40(9):488–492
50. Otten E (1988) Concepts and models of functional architecture in skeletal muscle. *Exerc Sport Sci Rev* 16:89–137
51. Petrone FA, McCall BR (2005) Extracorporeal shock wave therapy without local anesthesia for chronic lateral epicondylitis. *J Bone Joint Surg [Am]* 87(6):1297–1304
52. Porta M (2000) A comparative trial of botulinum toxin type A and methylprednisolone for the treatment of tension-type headache. *Curr Rev Pain* 4(1):31–35
53. Reilich P, Gröbli C, Dommerholt J (2012) *Myofasziale Schmerzen und Triggerpunkte*. 1. Aufl. Urban & Fischer Elsevier, München
54. Reitinger A, Radner H, Tilscher H et al (1996) *Morphologische Untersuchung an Triggerpunkten*. *Man Med* 34:256–262
55. Rompe JD (2002) *Shock wave applications in musculoskeletal disorders*. Thieme, Stuttgart New York
56. Rompe JD, Decking J, Schoellner C, Nafe B (2003) Shock wave application for chronic plantar fasciitis in running athletes. A prospective, randomized, placebo-controlled trial. *Am J Sports Med* 31(2):268–275
57. Ruch T (1949) *Visceral sensation and referred pain*. In: Fulton J (ed) *Howell's textbook of physiology*. 16th edn. Saunders, Philadelphia, pp 385–401
58. Salaffi F, De Angelis R, Grassi W et al (2005) Prevalence of musculoskeletal conditions in an Italian population sample: results of a regional community-based study. I. The Mapping Study. *Clin Exp Rheumatol* 23(6):819–828
59. Schaden W, Thiele R, Kolpl C et al (2007) Shock wave therapy for acute and chronic soft tissue wounds: a feasibility study. *J Surg Res* 143(1):1–12
60. Schoser B (2008) *Muskel und Schmerz – Ein Leitfaden für die Differentialdiagnose und Therapie*. 1. Aufl. Uni-Med, Bremen
61. Shah JP, Danoff JV, Desai MJ et al (2008) Biochemicals associated with pain and inflammation are elevated in sites near to and remote from active myofascial trigger points. *Arch Phys Med Rehabil* 89(1):16–23
62. Shah JP, Phillips TM, Danoff JV, Gerber LH (2005) An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. *J Appl Physiol* 99(5):1977–1984
63. Sikdar S, Ortiz R, Gebreab T et al (2010) Understanding the vascular environment of myofascial trigger points using ultrasonic imaging and computational modeling. *Conf Proc IEEE Eng Med Biol Soc* 2010:5302–5305
64. Simons DG (1996) Clinical and etiological update of myofascial pain from trigger points. *J Musculoskele Pain* 4:97–125
65. Simons DG (2004) Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. *J Electromyogr Kinesiol* 14(1):95–107
66. Simons DG, Travell J, Simons LS (1999) *Travell and Simons' myofascial pain and dysfunction: the trigger point manual (upper half of body)*. 2nd edn, vol 1. Williams & Wilkins, Baltimore

67. Skovron ML (1992) Epidemiology of low back pain. *Baillières Clin Rheumatol* 6(3):559–573
68. Takahashi N, Wada Y, Ohtori S et al (2003) Application of shock waves to rat skin decreases calcitonin gene-related peptide immunoreactivity in dorsal root ganglion neurons. *Auton Neurosci* 107(2):81–84
69. Tough EA, White AR, Cummings TM et al (2009) Acupuncture and dry needling in the management of myofascial trigger point pain: a systematic review and meta-analysis of randomised controlled trials. *Eur J Pain* 13(1):3–10
70. Tough EA, White AR, Richards S, Campbell J (2007) Variability of criteria used to diagnose myofascial trigger point pain syndrome – evidence from a review of the literature. *Clin J Pain* 23(3):278–286
71. Travell J, Simons DG (1983) Myofascial pain and dysfunction. The trigger point manual. The upper extremities, vol 1. Williams & Wilkins, Baltimore
72. Travell J, Simons DG (1992) Myofascial pain and dysfunction. The trigger point manual. The lower extremities, vol 2. Williams & Wilkins, Baltimore
73. Travell J, Rinzler S, Herman M (1942) Pain and disability of the shoulder and arm: treatment by intramuscular infiltration with procaine hydrochloride. *JAMA* 120:427–432
74. Venancio Rde A, Alencar FG, Zamperini C (2008) Different substances and dry-needling injections in patients with myofascial pain and headaches. *Cranio* 26(2):96–103
75. Wall PD, Cronly-Dillon JR (1960) Pain, itch, and vibration. *Arch Neurol* 2:365–375
76. Wang CJ (2003) An overview of shock wave therapy in musculoskeletal disorders. *Chang Gung Med J* 26(4):220–232
77. Windisch A, Reitingner A, Traxler H et al (1999) Morphology and histochemistry of myogelosis. *Clin Anat* 12(4):266–271
78. Zhang T, Adatia A, Zarin W et al (2011) The efficacy of botulinum toxin type A in managing chronic musculoskeletal pain: a systematic review and meta analysis. *Inflammopharmacology* 19(1):21–34
79. Zieglgänsberger W, Berthele A, Tolle TR (2005) Understanding neuropathic pain. *CNS Spectr* 10(4):298–308
80. Zimpfer D, Aharinejad S, Holfeld J et al (2009) Direct epicardial shock wave therapy improves ventricular function and induces angiogenesis in ischemic heart failure. *J Thorac Cardiovasc Surg* 137(4):963–970