Shock wave therapy of trigger points in the masticatory muscles

In the treatment of temporomandibular joint disorder (TMD), the application of shock waves to trigger points is a new therapy method with a precise effect. Especially focused shock waves enable reliable trigger point diagnosis by the induction of referred pain. These are the findings of Dr Danilo Jankovic, Head of the DGS Regional Pain Centre in Hürth/Cologne, Germany.

The cardinal symptoms of temporomandibular joint disorder (TMD) are pain in the temporomandibular joints (TMJs) and muscles of mastication, jaw joint sounds and impairment of mandible movement. In 1969, Laskin [9] presented the classic definition of the myofascial pain-dysfunction syndrome. According to that definition, the disorder is diagnosed if at least one of the following symptoms exists:

1. unilateral pain, generally in the ear or preauricular region;
2. tenderness to pressure of masticatory muscles;
3. clicking or popping sounds in the temporomandibular joint;
4. limited mouth opening.

The absence of organic alterations of the temporomandibular joint that would be detected by clinical or radiological examination is characteristic of the disorder. Myalgia of the masticatory muscles (myogenic temporomandibular dysfunction) is the regional manifestation of myofascial pain. Most patients with TMD suffer from myofascial pain or a combination of masticatory muscle and TMJ pain. Myalgias of the muscles of mastication are the most frequently reported non-infectious complaints in the orofacial region.

Such myalgias, along with temporomandibular joint arthralgia, are grouped under the term "myoarthropathies of the masticatory system" (MAP). Around 75 % of patients with painful MAP suffer from masticatory muscle pain without temporomandibular involvement [16].

The temporomandibular joint (TMJ)
The temporomandibular joint is the movable connection between the temporal bone and the mandible. From a topographical / anatomical perspective, there is a close relationship between the temporomandibular joint and the middle ear. Morphologically and functionally speaking, the TMJ belongs to the group of paired joints owing to the almost rigid connection between the right and left joint. This creates a continual reciprocal influence, as in a facet articulation.

Innervation of the masticatory system
The innervation of the masticatory system is very complex. Various cranial nerves are involved among which: trigeminal nerve (V), facial nerve (VII), glossopharyngeal nerve (IX), hypoglossal nerve (XII), vagus nerve (X) and accessory nerve (XI).

Muscles of mastication

The three mostly affected muscles of mastication (masseter, temporalis and lateral pterygoid muscles) will be discussed in the following sections.

Temporalis muscle ("temporal headache and maxillary pain")
The temporalis muscle is the strongest lever of the mandible. It originates from the temporal plane and temporal fascia. It connects with and attaches to the coronoid process of the mandible via a strong tendon.

Four trigger points (TrPs) are described in this context (Fig. 1). The TrPs are generally found along a horizontal line about one finger width above the zygomatic arch. Symptoms include temporal headache and toothache in the maxillary teeth [19].

Associated trigger points: TrPs in the temporalis muscle may be linked with TrPs in the ipsilateral masseter (deep portion) and with the contralateral temporalis muscle (less frequently with the lateral and medial pterygoid muscles).

Satellite trigger points: sternocleidomastoid and trapezius muscles [19].

Masseter muscle ("trismus muscle")
The anatomical attachments of the masseter muscle are located in the zygomatic arch and maxilla at the upper end and at the outer surface of the ramus of the mandible and jaw angle at the lower end. Symptoms of active trigger points in this muscle (jaw closer) are severe impairment of mouth opening (trismus), toothache (mandibular and maxillary molars) and unilateral tinnitus (deep portion of muscle) (Fig. 2).

Associated trigger points: in the ipsilateral temporalis and medial pterygoid muscles and in the contralateral masseter muscle (not regular).

Satellite trigger points: sternocleidomastoid muscle [19].

Lateral pterygoid muscle ("pain radiating deep into the temporomandibular joint")
The upper portion attaches to the sphenoid bone at the front end and to the articular disc and temporomandibular joint capsule at the rear end. The lower portion attaches to the lateral lamina of the pterygoid process at the front end and to the neck of the mandible at the rear end (Fig. 3).

Symptoms: the lateral pterygoid muscle (jaw opener) radiates pain deep into the temporomandibular joint up to the region of the maxillary sinus (Fig. 3). Pain is closely associated with the functional impairment of the joint. The trigger points in this muscle may be the major myofascial cause of pain referral into the region of the temporomandibular joint. This myofascial syndrome is frequently misinterpreted as arthritis of the temporomandibular joint.

Satellite trigger points: sternocleidomastoid muscle [19].
Etiology and pathogenesis
The etiology and pathogenesis of temporomandibular joint disorders are multifactorial [14, 16, 20]. Modern approaches distinguish between predisposing (e.g., structural, systemic, mental), initiating (e.g., microtraumas, macrotraumas, local ischaemia and overuse) and perpetuating (e.g., psychosocial) risk factors.

Besides traumatic, anatomical and neuromuscular causes, psychosocial factors and stress (increase in muscle tone) are also discussed in this context.

Diagnosis
One distinguishes between minimal diagnosis, standard diagnosis and extended diagnosis [18, 20, 21].

Minimal diagnosis comprises targeted, pain-related history, measurement of mandibular movement, assessment of tenderness to palpation of masticatory muscles and temporomandibular joints, pain-related psychosocial screening and panoramic radiography.

Standard diagnosis consists of pain-related history with standardised pain questionnaires, panoramic radiography, clinical examination and assessment of mandibular movement and tenderness to palpation of masticatory muscles (temporals and masseter muscles) and temporomandibular joints following a standardised protocol based on research diagnostic criteria for temporomandibular disorders (RDC/TMD) [18]. Added to these methods are occlusion diagnosis which determines dental status, tooth contact in habitual/maximum intercuspation (static occlusion) as well as attributes and V-shaped defects.

The manual functional analysis evaluates the end feel (comparison between active and passive mandibular movement) and the joint play (traction and translation movement in the temporomandibular joint region). The compression test and static and dynamic resistance tests round off the diagnostic spectrum. The main purpose of these special tests is to differentiate, in structural terms, between myogenic, arthrogenic and myoarthrogenic genesis of myoarthropathy.

The diagnosis also includes an assessment of the chronification stage and a psychological examination to determine psychosocial parameters, unspecific somatic symptoms and symptoms of depression.

Differential diagnosis
Differential diagnosis against the following conditions is necessary:
- episodic headache of the tension type,
- trigeminal neuralgia,
- Horton’s arteritis (giant-cell arteritis),
- atypical facial pain,
- polyomyositis (the most frequent form is traumatic ossifying myositis),
- rheumatic polymyalgia,
- fibromyalgia syndrome,
- malignant neoplasms,
- hypothyroidism, and others.

Therapy
Treatment of temporomandibular joint disorders comprises the following procedures [7, 17, 19]:
- biophysical therapy (occlusion splint, manual therapy, massage, TENS, physical autotherapy),
- therapeutic injections with local anaesthetics and corticosteroids [7],
- injection of botulinum toxin,
- osteopathic manipulations,
- intermittent cooling and stretching,
- acupuncture,
- pharmacotherapy, and
- behavioural therapy, biofeedback and progressive muscle relaxation.

Treatment of pain in masticatory muscles with focused shock wave therapy
Focused shock waves have been used with great success in the treatment of typical orthopaedic disorders [4] for about 15 years. Indications include calcific tendinitis [10], lateral and medial epicondylitis, heel spur [5], pseudoarthrosis [22] and muscular trigger points. Extracorporeal shock wave therapy (ESWT) was developed 30 years ago for urological applications and is still used for the non-invasive fragmentation of kidney stones. About 10 years ago, the therapy procedure was extended to include radial pressure waves, which provide similarly positive results in many indications.

Recently, extracorporeal – mostly planar – shock waves have also been used in dermatology. Wound healing disorders as in crural ulcers, burns or diabetic leg ulcers can now be treated with shock waves with remarkable success [8, 12, 15].

One generally distinguishes between focused shock waves and radial pressure waves [23]. Shock waves are characterised by high pressure amplitudes (approx. 1000 bar), short pulse duration of about 300 ns and extremely short pulse rise times of about 10 ns.
Treatment with focused shock waves is referred to as ESWT (extracorporeal shock wave therapy). Radial pressure waves are much slower (factor 1000), and their pulse amplitude is generally as low as 1 to 10 bar. Nevertheless, radial pressure waves have similar effects as shock waves in the treatment of many conditions. This is presumably due to the typical pulsatile and asymmetric pressure profile. Although different from shock waves [1], treatment with radial pressure waves is referred to as RSWT (radial shock wave therapy). But the acronym EPAT (extracorporeal pulse activation therapy), which has been increasingly used recently, seems to describe the principle much better [13]. Although its biological mechanisms of action are not yet fully known, shock wave therapy has been used with great success to improve perfusion and metabolic activity. It stimulates biological processes which eventually lead to permanent healing. The mechanisms of action that are known to provide the positive wound healing results we have observed include the following effects:

- immediate increase in blood flow, which, during focused shock wave application in particular, is not attributable to the pulse-massage effect of the shock transmitter vibrations, but to the documented release of nitric oxide (ENOS; endothelial nitric oxide synthase) [11]. Nitric oxide causes biochemical vasodilation and is a highly versatile messenger substance that is involved in the production of additional tissue factors;
- increase in cell membrane permeability [3] and, consequently, general improvement of metabolic activity;
- release of numerous tissue factors, among which the VEGF (vascular endothelial growth factor) is of crucial importance in wound healing processes as it promotes neovascularisation [6].

An additional key effect is the proliferation and differentiation of stem cells which lead to the formation of new healthy tissue almost without scarring [2, 8].

For most patients, an energy flux density of between 0.05 and 0.10 (0.15) mJ/mm² is used at a shock frequency of 2 to 4 Hz. About 2000 to 3000 shock waves are applied per therapy session (masseter and temporalis muscles and satellite TrPs) (Figs. 6 and 7). Therapists should make sure that the pain caused by shock waves is well tolerated by the patient. Muscle smoothing after shock wave application is done with the V-ACTOR handpiece (vibration therapy) or with radial pressure waves at a frequency of 18 to 21 Hz, applying 1000 to 1500 shocks at a pressure of 1.8 to 2 bar (equivalent to the stretch and spray technique) [19]. After completion of shock wave application, the treated area is massaged and passive muscle stretching is performed. After having applied a hot pack, the jaw should be moved actively.

The first three therapy sessions take place at intervals of about 10 to 14 days. Additional treatments are conducted at intervals of three weeks. In general, four to eight therapy sessions are necessary to achieve substantial pain relief. Experience has shown that in most cases the energy level can be increased from session to session because the pain caused by shock wave application decreases gradually if the therapy progresses as expected.

The trigger points in the lateral pterygoid muscle can be treated with additional targeted injections [7].

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A list of references can be obtained from the author or editorial office.