Introduction

Trigger points correspond to a neuromuscular disorder referred to as Myofascial Trigger Point Syndrome (MTS). They are characterized by regional pain, sensory disorders and functional disorders of the musculoskeletal system which, in the initial phase, occur only when subjected to extraordinary stress. Later, in the chronification phase, pain is caused by performing activities of daily living, stress and even changing weather conditions. The final phase is marked by permanent pain, minimal stress threshold, increasing social isolation and reactive depression syndrome. This evolution coincides with an ever-expanding dysfunction of the motoric end plates of the muscle fibers. What is suspected is an excessive release of acetylcholine and a contraction of the Sarkomere[1]. An accumulation of these contracted Sarkomere is known as the trigger point complex (Figure 1) and brings about a shortening of the entire muscle. A pathological end plate noise (EPN) is identified electrophysiologically in the EMG and a histological exam of muscle biopsy shows the contraction of the Sarkomere as well (Figure 2)[2,3]. Based on the hypothesis of energy crisis, accompanying vasoconstriction releases sensitizing substances which affect the nociceptors and bring about a lowering of the pain. The development of a local pathological pain transfer system may bring about the muscular trigger point[4].

Depending on their clinical effect, active and latent trigger points can be distinguished. Active triggers shorten the muscle and cause transfer phenomena such as pain in another part of the body. This is know as referred pain. Like active
triggers, latent triggers also cause muscle structures to shorten but they do not bring about any referred phenomena causing patients to suffer. In muscles that are accessible for palpation, both types of trigger can be identified as a sharply painful knot in a hardened muscle. In case of active triggers, pressure applied to this knot causes significant pain. Applying pressure on a latent trigger causes no pain in general, or pain not known by the patient or pain not previously existing.

Figure 1. Trigger point complex.

Figure 2. Trigger point histology. Longitudinal cross-section of a canine M. The outlined area encompasses some 100 Sarkomere forming a knot. The Sarkomere on both sides of the knot appear to be longer as compensation.
The trigger point complex is located in the end plate zone and is referred to as central trigger points.

**Inclusion criteria**

Patients with chronic, therapy-resistant pain caused by active triggers.

**Exclusion criteria**

Patients with fibromyalgia, tumors, disc protrusion with neurologic deficits, peripheral nerve lesions or other significant diseases.

**Drop-out criteria**

Patients were required to get 6 treatments in total. Patients who refused the full treatment cycle were excluded for efficacy analysis.

**Patient selection**

For each group 20 patients were selected randomly.

**Evaluation criteria for response to treatment**

The evaluation criterion “Pain” was measured by the patients on the 100 mm Visual Analog Scale VAS. VAS PRE at baseline was documented before the first treatment, VAS POST (re-exam) at the end of the treatment. VAS FU was determined in a telephone follow-up.

**Trigger point diagnosis**

A first step consisted of measuring the range of motion (ROM) of the joint. A limitation in mobility or a right/left variation appears to indicate MTS. As a result of muscle shortening, there is restriction in the range of motion in mus-
cles (the antagonists) extended during the exam. Included in the study were the rotation, lateral flexion, flexion and extension of the neck spinal column; inner and outer rotation and abduction of the shoulder; rotation and lateral flexion of the lumbar spine; and internal and external rotation of the hip. Muscle groups responsible for limitations in range of motion (ROM) were examined for trigger-specific characteristics: taut muscle fiber bundle (Taut Band TB), sharply painful knot within the TB, trigger of patient-specific pain patterns (Referred Pain RP) by pressure stimulation with the TRIGGOsan key on the Trigger Points. TBs or Trigger Points could not be detected in deeper or very large muscles. In these muscles, diagnosis was limited to triggering RP by applying pressure with the TRIGGOsan key[5].

**Treatment**

The muscles identified through trigger point diagnosis were treated with radial extracorporeal shock waves using the Swiss DolorClast®. The number of impulses per treatment depended on the effect of the treatment as well as on increased mobility and decrease in pain. Treatment intensity was just below the pain tolerance threshold and no local anesthesia was required.

**Results**

A summary of the results is shown in Table 1.

**Cervical spine and shoulder**

VAS PRE (Chart 1) and VAS POST (Chart 2) were compared to evaluate the effect of the treatment. The comparison revealed a subjective improvement of about 56.6%. A follow-up (Chart 3) was conducted in 15 patients of this group. The subjective improvement was 43.7% in a mean follow up period of 135 days. The mean treatment number was 6.8 within a period of 71 days.

**Shoulder**

A comparison of VAS PRE (Chart 4) and VAS POST (Chart 5) in 20 patients showed a subjective improvement of about 68%. A follow-up (Chart 6) con-
ducted in 8 patients of this group showed an improvement of about 74.5% on the VAS. The treatment number was 4.45 within a treatment period of 39 days.

<table>
<thead>
<tr>
<th></th>
<th>Cervical n=20</th>
<th>Shoulder n=20</th>
<th>Lumbar n=20</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Mean value</td>
<td>Mean value</td>
<td>Mean value</td>
</tr>
<tr>
<td>1 VAS PRE</td>
<td>8.40</td>
<td>7.1</td>
<td>7.0</td>
</tr>
<tr>
<td>2 VAS POST</td>
<td>3.65</td>
<td>2.27</td>
<td>2.65</td>
</tr>
<tr>
<td>4 VAS FU</td>
<td>4.73 (n=15)</td>
<td>1.81 (n=8)</td>
<td>2.81 (n=16)</td>
</tr>
<tr>
<td>3 VAS PRE/VAS POST</td>
<td>56.6</td>
<td>68.0</td>
<td>62.1</td>
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<tr>
<td>Change in %</td>
<td></td>
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</tr>
<tr>
<td>5 VAS POST/VAS FU</td>
<td>43.7</td>
<td>74.5</td>
<td>59.8</td>
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<td>Change in %</td>
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<tr>
<td>6 Days FU</td>
<td>135</td>
<td>187</td>
<td>112</td>
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<tr>
<td>8 Total Impulses</td>
<td>30,400</td>
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<td>27,250</td>
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<tr>
<td>7 Number of Treatments</td>
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<td>4.45</td>
<td>6.1</td>
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<td>9 Impulses/Treatment</td>
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<td>2957</td>
<td>4467</td>
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<td>11 Treatment Period (Days)</td>
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<td>39</td>
<td>64</td>
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<td>10 Duration of Pain (Months)</td>
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<td>30</td>
<td>76</td>
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<td>12 Age (Years)</td>
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<td>54.2</td>
</tr>
</tbody>
</table>

**Table 1.** Results of treatment.
Low back pain

A comparison of VAS PRE (Chart 7) versus VAS POST (Chart 8) in 20 patients showed an improvement of about 62%. A follow-up (Chart 9) in 16 patients of this group showed a subjective improvement of 59.8% on the VAS. The treatment number was 6.1 within a period of 64 days.

Discussion

The results indicate that the application of radial extracorporeal shock waves may affect the symptoms of myofascial trigger point syndrome even over a longer period of time. The results for the cervical spine/shoulder area appear poorer and
this observation coincides with our experience in practice. Treatment results may be improved by increasing the number of impulses per treatment and by refining diagnosis of MTS in muscles with difficult access. A combined use of ballistic shock waves and mechanical pressure could further improve the treatment’s success. Finally, the treatment parameters (number of shock waves, energy flux density, interval, etc.) have to be specified by prospective controlled studies, but rESWT could be indicated in patients suffering from trigger point related musculoskeletal disorders as shown in our preliminary study.

Conclusion

These studies could be the basis for further evidence-based research to bring scientific evidence of the efficacy of the application of radial shock waves for the treatment of myofascial trigger point syndrome.

Literature

5. Diagnosis, and Treatment. Lippincott, Williams &Wilkins, Philadelphia. (Endplate hypothesis, pp.240 –259)