Journal of Oral Science, Vol. 55, No. 2, 175-181, 2013

Original

Laser therapy and needling in myofascial trigger point deactivation

Luciana Uemoto[1,2], Marco Antonio C. Garcia[3], Cresus Vinicius D. Gouvêa[4], Oswaldo V. Vilella[4], and Thays A. Alfaya[4]

[1]Dentistry Graduation Program, Federal Fluminense University, Niterói, RJ, Brazil
[2]Dentistry Graduation Program, Salgado de Oliveira University, Niterói, RJ, Brazil
[3]Physical Education Graduation Program, Federal do Rio de Janeiro University, Rio de Janeiro, Brazil
[4]Dental Clinic Post Graduation Program, Federal Fluminense University, Niterói, RJ, Brazil

(Received October 22, 2012; Accepted May 11, 2013)

Abstract: The aim of this study was to evaluate different approaches to deactivating myofascial trigger points (MTPs). Twenty-one women with bilateral MTPs in the masseter muscle were randomly divided into three groups: laser therapy, needle treatment and control. Treatment effectiveness was evaluated after four sessions with intervals ranging between 48 and 72 h. Quantitative and qualitative methods were used to measure pain perception/sensation. The Wilcoxon test based on results expressed on a visual analog scale (VAS) demonstrated a significant ($P < 0.05$) decrease in pain only in the laser and needle treatments groups, although a significant increase in the pressure pain threshold was evident only for needling with anesthetic injection ($P = 0.0469$), and laser therapy at a dose of 4 J/cm² ($P = 0.0156$). Based on these results, it was concluded that four sessions of needling with 2% lidocaine injection with intervals between 48 and 72 h without a vasoconstrictor, or laser therapy at a dose of 4 J/cm², are effective for deactivation of MTPs. (J Oral Sci 55, 175-181, 2013)

Keywords: myofascial pain; trigger point; low-level laser therapy; anesthetic injection; dry needling.

Introduction

Myofascial pain syndrome (MPS) is one of the most frequent causes of pain involving the orofacial region, i.e., the head and neck (1). A key feature of this syndrome is the presence of myofascial trigger points (MTPs) in the affected muscles. When these points are active, patients often complain of pain distant from the site, e.g., in the head, ear, mandible, temporomandibular joint, teeth, eyes and cervical spine. This feature prompts patients to consult a wide range of health professionals, including otorhinolaryngologists, ophthalmologists and neurologists. Several tests are usually performed, but no abnormality is usually detected.

The signs and symptoms of this syndrome have not been clarified. However, certain diagnostic criteria have been reported, such as a palpable and hypersensitive taut muscle band – if the muscle is accessible – acknowledgement of pain by the patient when pressure is applied to the active MTP, and soreness when the affected muscle is stretched (2-7). Therefore, diagnosis is purely clinical, based on a detailed history and thorough physical examination performed by muscle palpation and observation of motor function (4). MTPs can be classified as active (single) or latent (multiple), depending on their clinical features. As previously stressed, there is local sensitivity in the taut muscle bands and pain at a distance, which causes the patient to respond by twitching, flinching, or showing facial expressions of discomfort during palpation (2,4,6). Moreover, any pain or tenderness is generally located ipsilateral to the detected MTPs. The latent MTPs are generally multiple and do not cause referred pain, but cause result in muscle shortening or weakness (4,6).

No consensus has yet been reached regarding the etiology of MPS. Direct or indirect factors may be

Correspondence to Dr. Thays Almeida Alfaya, R. Doutor Calandrine 235 A, São Gonçalo, Rio de Janeiro 24755.160, Brazil
E-mail: thalfaya@gmail.com

associated. The former consist of direct injury to the muscle (macrotrauma) or repeated microtrauma caused by parafunctional (2,3) and improper postural habits, or recreational or occupational activities that produce repetitive stress in a specific muscle or group of muscles (2). Indirect factors cause muscle weakness, predisposing the muscle to the development of trigger points. Contributing factors include nutritional deficiencies, structural disharmonies, such as occlusal disorder, physical inactivity, sleep disorders and joint problems (2-4) as well as any continuous source of deep pain and emotional distress (4,8,9).

Given its multifactorial etiology, no standard treatment protocol for MPS is currently available. Instead, several treatment alternatives have been suggested. The main objective has been to restore the normal length, position and full range of motion of the muscle(s), including the identification and removal of perpetuating factors, in addition to MTP deactivation (4,5).

Suggested treatments for deactivation of MTPs include ultrasound (6), application of pressure or massage (6), transcutaneous electrical nerve stimulation (TENS) (6,10), ethyl chloride spray and stretch techniques (6), acupuncture (6), dry needling, (4,6,11), or needling with injection of certain agents, (6) and low-level laser therapy (1,2,6,12-14).

In view of the above, it is of paramount importance to clinically assess and compare some of the therapies used for MTP deactivation with a view to establishing more effective alternatives for the treatment of MPS. Therefore, the aim of this study was to compare two different approaches, laser and needling therapy, in subjects presenting with MPS.

Materials and Methods

Subjects

The patients who sought treatment for temporomandibular joint disorders (TMJD) were first evaluated, and all those who manifested active MTPs in the masseter muscle were invited to participate in the present study.

Twenty-one women aged 20-52 years with a body mass index of 19.7-32.4 kg/m² volunteered for this study. They were randomly divided into three groups: laser therapy (LG), needle treatment (NG) and control (CG). Inclusion criteria for this study were a) being female and Caucasian, b) more than 20 years of age, and c) presence of active MTPs in both masseter muscles, previously identified by manual palpation. On the other hand, the exclusion criteria were a) use of pain killers, muscle relaxants, and/or anti-inflammatory medication and benzodiazepines, b) pregnancy, and c) receiving treatment for TMJD. All of the women were patients at two university hospitals (Universidade Federal Fluminense (UFF) and Universidade Salgado de Oliveira (UNIVERSO)), both in the city of Niterói, Rio de Janeiro, Brazil.

The present study was submitted to the local ethics committee (filed under number 024/2008) and was only conducted after each volunteer had given informed consent.

Procedures for data acquisition

After selection, a diagnostic questionnaire designed for research on temporomandibular joint disorders (TMD-RDC-Axis II) (15) was applied. Data on the location of MTPs in the masseter muscle and response to treatment were also recorded. MTPs were found by digital palpation using the index finger with the patient in horizontal recumbency. Active MTPs were identified by the presence of pain on palpation and referred pain. Pain was assessed before and after treatment in the following manner: use of a visual analog scale for spontaneous pain, a digital algometer (Model 20 DDK, KRATOS Equipamentos Industriais Ltd., São Paulo, Brazil) to determine the pain threshold to pressure, and measurement of maximum mouth opening without discomfort using a caliper. The surface electromyographic (sEMG) signals were then collected and subsequently therapy was delivered. All evaluations were performed by a single person trained for this task.

For evaluation of the pressure pain threshold, the tip of the device (~ 1 cm^2) was pushed perpendicularly onto the skin surface at a constant velocity until the patient notified the operator that she was beginning to feel a painful sensation. At this moment, the pressure was automatically recorded on the display and the values obtained from the spot being evaluated were noted. After checking all MTPs, the procedure was repeated once again, and the mean value of the two measurements was determined. During this procedure, each patient was instructed to keep her head in an upright and stable position with the aid of the hand contralateral to the side being treated.

To acquire the sEMG signals, an electromyographic device (EMG *System* do BRASIL Ltd., São Paulo, Brazil) with a bandpass filter (4th order) between 20-500 Hz, the gain and sampling frequency (Fs) set at 2,000 and 2,000 Hz, respectively, and a microcomputer, were used. The WinDaq software package was also used to acquire and process the sEMG signals.

The sEMG signal analysis was based on the root mean square (RMS) value, as follows:

$$RMS = \sqrt{\frac{1}{N}\sum_{i=1}^{N} x_i^2} \quad \text{(Equation 1)}$$

Where N is defined as the number of samples ($N = 8{,}000$), dependent on the total time for data analysis (T = 4 s), and X represents each one of the samples that contained a specific RMS value for the sEMG signal.

The RMS value was used because it reflects with greater accuracy any and all variations in sEMG signal amplitude.

All the patients underwent analysis of bilateral sEMG activity from the masseter muscle. For this purpose, each patient sat on a chair with a backrest keeping the Frankfort horizontal plane parallel to the floor (looking at the horizon), with feet flat on the floor and hands resting on the legs. Prior to placement of the surface electrodes (Ag/AgCl, Meditrace 100; 1 cm diameter; interelectrode distance 17 mm) the skin was cleaned with cotton soaked in 70º alcohol to remove any oily substances or bases that would in order to reduce local impedance. For placement of the surface electrodes, the patients were asked to bite into maximum intercuspation in order to locate the point of maximum muscle contraction. The electrodes were placed along the axis of the muscle fibers. The reference electrode was placed over the spinous process of the C7 cervical vertebra. Micropore adhesive tape was placed over the electrodes to reduce movement artifacts in the sEMG signal.

Surface EMG signal acquisition was performed by asking the patient to bite on a piece of Parafilm M tape placed bilaterally between the posterior teeth, and to remain in isometric contraction for 10 s. The initial and final 3 s were excluded from the sEMG signal to minimize variability at the beginning and end of the test.

Therapy sessions

The volunteers were divided into three groups as follows.

Laser group (LG): Seven patients who received application of an infrared laser (Model Three Light, Clean Line brand, São Paulo, Brazil) with a wavelength of 795 nm at 80 mW power. The MTPs located in the right masseter of each patient were irradiated with the laser at a dose of 4 J/cm^2. On the other hand, a dose of 8 J/cm^2 was applied to the left side.

Needling group (NG): Seven patients who underwent dry needling of MTPs located in the right masseter muscle. The same muscle on the left side was injected with 0.25 ml of 2% lidocaine without epinephrine (Lidostesim SV-Dentsply brand, São Paulo, Brazil). Dental carpules with reflux and short 30G (Unoject Nova DFL brand, Rio de Janeiro, Brazil) disposable needles were utilized. The needle was inserted to a depth of 1 to 2 cm at an acute angle of 30° to the skin, in various directions, with movement into the tissue. These patients were instructed to avoid pressing directly on the needling site for at least 2 min after the procedure to prevent bruising.

Control group (CG): Seven patients who comprised the control group. They received placebo treatment at trigger points located in the right and left masseter muscles. In this group laser therapy was simulated, i.e., no laser light irradiation was used.

At the end of each treatment session, all patients, including those in the control group, performed three 10-s sets of active muscle stretching with maximum mouth-opening.

Four treatment sessions were conducted with an interval of 72 h between the first and second sessions, 48 h between the second and third sessions, and 72 h between the third and fourth sessions.

After the 4th therapy session, the sEMG signals were collected, mouth opening was measured, and pain was assessed using a visual analog scale and a digital algometer.

To confirm that the laser energy and needling/injection were being applied to the same site in each session, anatomical landmarks (the tragus and mandibular angle) were adopted and a goniometer was used.

All patients were advised not to use any medication during the treatment phase. They were also advised to use ice compresses, stretch their neck and correct their posture during sleep, as well as using a proper pillow height and support for the arms and legs with pillows to stabilize the spine. They were also advised to monitor or, as far as possible, eliminate harmful habits throughout the day, such as clenching, onychophagy, chewing gum, and avoid biting or holding objects between the teeth (e.g., pens, pencils, clips).

Wilcoxon's signed-rank paired test was used to analyze the data (before and after treatments sessions) since they were not Gaussian, and evaluation was also conducted using the Shapiro-Wilk test. The level of significance (α) was set at 0.05.

Results

On the basis of the visual analog scale (VAS) data, the control group was the only one that failed to show a significant improvement ($P = 0.0781$) in pain. Dry needling therapies ($P = 0.0313$) (Fig. 1), injection of anesthetic ($P = 0.0313$) (Fig. 2), and laser therapy at a dose of 4 J/cm² ($P = 0.0156$) (Fig. 3) and 8 J/cm² ($P = 0.0313$) (Fig. 4) yielded statistically significant improvements in the degree of pain.

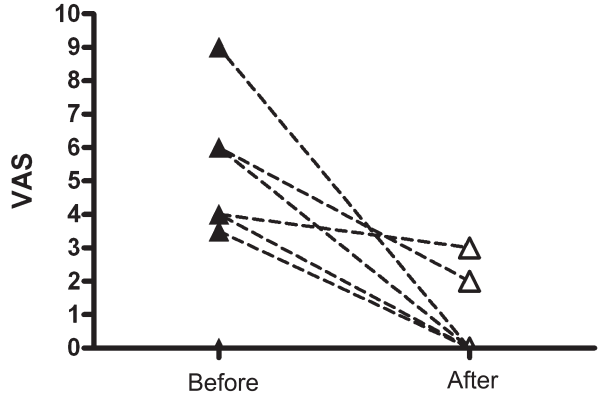

Fig. 1 VAS scores comparing the pain level before and after ($P = 0.0313$) dry needling in the NG group.

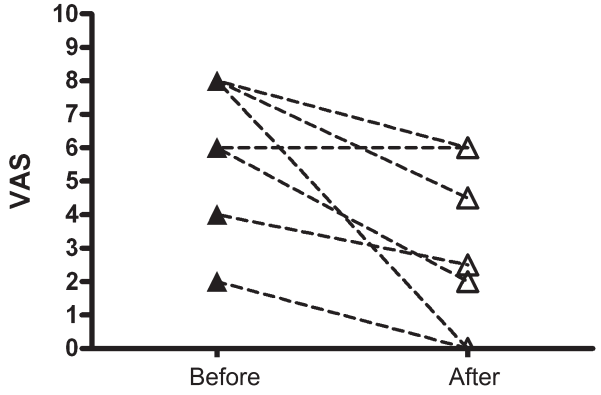

Fig. 4 VAS scores comparing the pain level before and after ($P = 0.0313$) laser therapy at a dose of 8 J/cm^2 in the LG group.

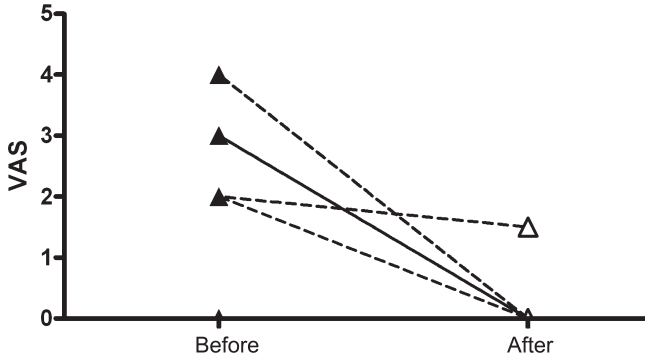

Fig. 2 VAS scores comparing the pain level before and after ($P = 0.0313$) injection of anesthetic in the NG group.

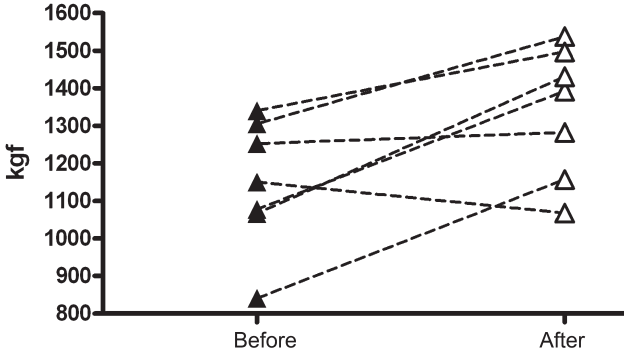

Fig. 5 Results of digital algometer evaluation comparing the pain level before and after ($P = 0.0469$) injection of anesthetic in the NG group.

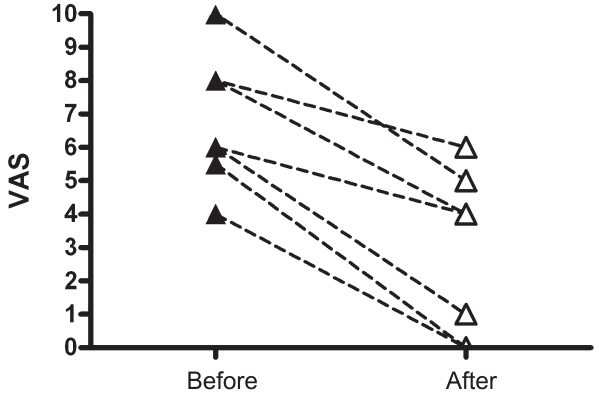

Fig. 3 VAS scores comparing the pain level before and after ($P = 0.0156$) laser therapy at a dose of 4 J/cm^2 in the LG group.

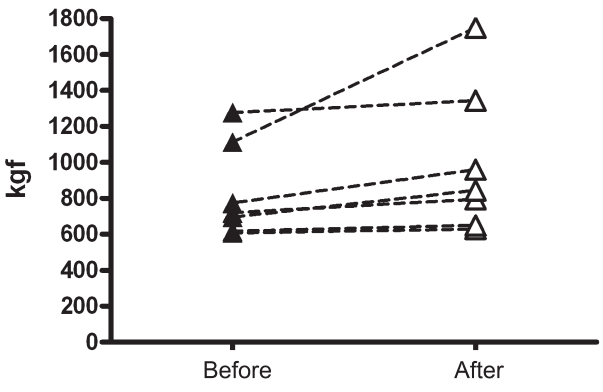

Fig. 6 Results of digital algometer evaluation comparing the pain level before and after ($P = 0.0156$) laser therapy at a dose of 4 J/cm^2 in the LG group

The digital algometer was used on all MTPs. A total of 69 MTPs in the right masseter vs. 71 in the left masseter were evaluated in this study. Significant improvements were observed only in the groups injected with anesthetic ($P = 0.0469$) (Fig. 5) and laser therapy with a dose of 4 J/cm² ($P = 0.0156$) (Fig. 6). There were no significant improvements in the control ($P = 0.4688$), dry needling ($P = 0.1094$) and laser therapy ($P = 0.4688$) groups, the latter irradiated with a dose of 8 J/cm².

There were also no significant changes (laser therapy with a dose of 4 J/cm²: $P = 0.8125$; laser with a dose of 8 J/cm²: $P = 0.3750$; dry needling: $P = 0.8125$; injection

with lidocaine: $P = 0.6875$, control: $P = 0.9375$) in the sEMG signals collected from the masseter muscle during maximum voluntary contraction before and after the therapy sessions.

Regarding mouth-opening, no comparison was made between the therapies employed for each masseter muscle, since the measurement was the same, regardless of whether it was on the right or left side. In this case, only the control, needling and laser therapy groups were compared, but no significant differences ($P = 0.1094$, $P = 0.1563$, $P = 0.1563$, respectively) were observed.

Discussion

Despite the fact that MPS is one of the most common causes of pain and disability in patients with musculoskeletal pain, patients and many health professionals do not acknowledge it, since diagnosis depends solely on clinical history and the findings of physical examination (2). This neglect means that the disease tends to become chronic (16) and to influence the affective-motivational variable. Measuring this internal, complex and personal experience objectively is considerably challenging. For this reason, different methods were used to measure pain perception/sensation in the present study. In several previous MPS-related studies (7,11,12,16-18), the VAS and pain threshold in response to pressure have been the most widely used instruments. Both are rapid and easy to apply, and reflect the subjective and objective evaluations, respectively. In the present study, the VAS proved to be a faster tool since the digital algometer was used in each of the MTPs.

When the focus of research is to evaluate therapies in patients with chronic TMJD, such as MPS, inclusion of a control group makes it possible to assess the extent to which pain relief is being influenced by the psychological components of a given treatment and/or professional care. Furthermore, special attention must be given to the therapeutic approach implemented for individuals who comprise this group. Tschoop and Gysin (17) and Ojala et al. (12) compared the effects of different substances injected into MTPs and chose 0.9% saline solution as their placebo of choice. It was found that injection of this substance was also an effective therapy for deactivating concealed MTPs (12,17,19). The action of different substances is not fully understood. One hypothesis suggests that substances (e.g., algogenic substances, Ca^{2+} ions) accumulated in a given spot can be "flushed out", thereby decreasing nerve sensitivity and uncontrolled muscle twitching, since even substances with no pharmacological action such as saline gave satisfactory results in comparison with local anesthetics and type A botulinum toxin (4). This theory, however, is not applicable to dry needling. This might be one possible explanation for the better clinical performance of anesthetic injection over dry needling found in the present study, unlike the findings of Cummings and White (19), but corroborating those of Hong (11) and Kamanli et al. (1). Therefore, if only needling therapies are being compared, the use of another type of control group is recommended. Laser therapy without emission of laser light appears to be an appropriate alternative (7,20), and this was the approach adopted in the present investigation.

When properly applied, low-level laser therapy (LLLT) has shown satisfactory effects in deactivating MTPs (16,17,20,21), although it is still a more costly procedure. In MTP areas, laser therapy improves the local microcirculation, thus increasing the supply of oxygen to hypoxic cells, and helping to remove cellular metabolic by-products. Once stabilized, the microcirculation breaks the vicious cycle of pain-spasm-pain (21).

The most suitable laser wavelengths for deactivation of MTPs are within the range 780-904 nm, corresponding to the infrared, since they have higher tissue penetration (20), as was seen in the present investigation. However, Ilbuldu et al. (7) used an electromagnetic spectrum below what is usually recommended (632.8 nm and 730 nm, respectively), achieving satisfactory results, which suggests that a suitable dosage is important for successful therapy. In fact, application of an optimal dose directly to the target area is even more important than the wavelength of the device, since under- or over-irradiation may be ineffective or even exert an inhibitory effect (21). This may help to explain the better result obtained in the laser group with a dose of 4 J/cm² than with 8 J/cm². The dose should also be adjusted according to the type of tissue. For swarthy skin, a 50% increase over the usual dose is recommended, since melanin absorption is greater on the surface, thus reducing the dose at the target depth. For patients with substantial subcutaneous fat, the dose should also be increased accordingly because the fat may cause reflection, leading to lower absorption of radiation by the tissue (21,22).

On the basis of the present results, we recommend that laser therapy be applied two to three times a week, in agreement with Simunovic (2, 21), and since this is a chronic disorder (> 6 months), a lower dose of about 4 J/cm² is advisable. Although four sessions yielded satisfactory results, Venancio et al. (20) recommended a greater number of sessions (> 30 sessions).

With regard to the extent of painless mouth opening, no significant improvement was found in the needling, laser or control group. The fact that therapy was applied only

to the masseter muscle may have inhibited relaxation of the other mandibular elevator muscles, and consequently relaxation of the muscles responsible for mouth opening.

In the present study, no significant differences in maximum contraction were identified on the basis of sEMG signals. However, it cannot be concluded that there was no improvement in muscle strength, because the pre- and post-therapy bite values were not standardized. Further controlled studies employing sEMG and MPS evaluations will be required.

Among the therapies evaluated in this study, needling with anesthetic injection and laser therapy at a dose of 4 J/cm² yielded the best results in terms of MTP deactivation. Both methods are quick and convenient. Although the former is more cost-effective, it requires greater technical mastery. Laser therapy does not offer as many risks as needling during application, and is a good option for patients who are afraid of needles.

Currently, the most widely accepted tool for standardizing the diagnostic criteria for TMJD is the Research Diagnostic Criteria for Temporomandibular Disorders-RDC/TMD, which is divided into two axes, i.e., axis I, corresponding to physical assessment for establishing diagnosis of muscles and/or joints, and axis II, for psychosomatic evaluation (15). Although this has improved research reliability and minimizes any variability in the examination methods and clinical judgment that may influence the sorting process, there are still limitations to its use. For muscle diagnosis, the only options available are myofascial pain and myofascial pain with limited opening, which do not match the diagnostic approach used in the present study, but cover a broad category of muscular disorders commonly found in TMJDs, which may or may not be painful. This explains why RDC has not been adopted in MPS-related research. Ultimately, this lack of standard criteria undermines work quality, and thus further controlled studies of MPS therapies are warranted.

We conclude that laser therapy at a dose of 4 J/cm² and needling with injection of 2% lidocaine without the use of a vasoconstrictor yield the best results for MTP deactivation as assessed on the basis of a VAS and the pain threshold to pressure. Laser therapy at 8 J/cm² and dry needling proved to be effective only when assessed by the VAS. None of the therapies yielded significantly better results than the mouth-opening and sEMG activity criteria, which might be related to the effectiveness of the conventional sEMG approach for mapping myoelectric activity from the masseter muscle. We believe that high-density surface EMG (HD-sEMG), which enables the collection of myoelectrical activity from different locations on the same muscle, might be an alternative approach. In HD-sEMG, multiple electrodes are placed over different regions of a muscle, even the smaller ones, and many signals can be obtained at the same time. On the basis of the present results, we conclude that HD-sEMG signal acquisition may be helpful to clinicians in the near future for evaluating and mapping MTPs, thereby replacing the current standard technique. Readers interested in this new approach can find details concerning methodological issues in Garcia and Vieira (23) and Merletti et al. (24,25).

References

1. Kamanli A, Kaya A, Ardicoglu O, Ozgocmen S, Zengin FO, Bayik Y (2005) Comparison of lidocaine injection, botulinum toxin injection, and dry needling to trigger points in myofascial pain syndrome. Rheumatol Int 25, 604-611.
2. Fricton JR, Kroening R, Haley D, Siegert R (1985) Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. Oral Surg Oral Med Oral Pathol 60, 615-623.
3. Han SC, Harrison P (1997) Myofascial pain syndrome and trigger-point management. Reg Anesth 22, 89-101.
4. Simons DG, Travell JG, Simons LS (1999) Myofascial pain and dysfunction: the trigger point manual; vol. 1 upper half of body. 2nd ed, Williams & Wilkins, Baltimore.
5. Wright EF (2000) Referred craniofacial pain patterns in patients with temporomandibular disorder. J Am Dent Assoc 131, 1307-1315.
6. Alvarez DJ, Rockwell PG (2002) Trigger points: diagnosis and management. Am Fam Physician 65, 653-660.
7. Ilbuldu E, Cakmak A, Disci R, Aydin R (2004) Comparison of laser, dry needling, and placebo laser treatments in myofascial pain syndrome. Photomed Laser Surg 22, 306-311.
8. Okeson JP (1995) Bell's orofacial pains. 5th ed, Quintessence, Chicago.
9. Manfredini D, di Poggio AB, Romagnoli M, Dell'Osso L, Bosco M (2004) Mood spectrum in patients with different painful temporomandibular disorders. Cranio 22, 234-240.
10. Rodrigues D, Siriani AO, Bérzin F (2005) Effect of conventional TENS on pain and electromyographic activity of masticatory muscles in TMD patients. Braz Oral Res 18, 290-295.
11. Hong CZ (1994) Lidocaine injection versus dry needling to myofascial trigger point. The importance of the local twitch response. Am J Phys Med Rehabil 73, 256-263.
12. Simons DG, Stolov WC (1976) Microscopic features and transient contraction of palpable bands in canine muscle. Am J Phys Med 55, 65-88.
13. Graboski CL, Gray DS, Burnham RS (2005) Botulinum toxin A versus bupivacaine trigger point injections for the treatment of myofascial pain syndrome: a randomised double blind crossover study. Pain 118, 170-175.
14. Ojala T, Arokoski JP, Partanen J (2006) The effect of small

doses of botulinum toxin a on neck-shoulder myofascial pain syndrome: a double-blind, randomized, and controlled cross-over trial. Clin J Pain 22, 90-96.
15. Dworkin SF, LeResche L (1992) Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. J Craniomandib Disord 6, 301-355.
16. Hakgüder A, Birtane M, Gurcan S, Kokino S, Turan FN (2003) Efficacy of low level laser therapy in myofascial pain syndrome: an algometric and thermographic evaluation. Lasers Surg Med 33, 339-343.
17. Tschopp KP, Gysin C (1996) Local injection therapy in 107 patients with myofascial pain syndrome of the head and neck. ORL J Otorhinolaryngol Relat Spec 58, 306-310.
18. Kiralp MZ, Ari H, Karabekir I, Dursun H (2006) Comparison of low intensity laser therapy and trigger point injection in the management of myofascial pain syndrome. Pain Clinic 18, 63-66.
19. Cummings TM, White AR (2001) Needling therapies in the management of myofascial trigger point pain: a systematic review. Arch Phys Med Rehabil 82, 986-992.
20. Venancio RA, Camparis CM, Lizarelli RFZ (2002) Laser in the treatment of temporomandibular disorders. J Brasileiro de Oclusão 2, 229-234.
21. Simunovic Z (1996) Low level laser therapy with trigger points technique: a clinical study on 243 patients. J Clin Laser Med Surg 14, 163-167.
22. Simunovic Z (2000) Lasers in medicine and dentistry: basic science and up-to-date clinical application of low energy-level laser therapy LLLT. Vitagraf, Rijeka.
23. Cavalcanti Garcia MA, Vieira TMM (2011) Surface electromyography: why, when and how to use it. Rev Andal Med Deporte 4, 17-28.
24. Merletti R, Aventaggiato M, Botter A, Holobar A, Marateb HR, Vieira TMM (2010) Advances in surface EMG: recent progress in detection and processing techniques. Crit Rev Biomed Eng 38, 305-345.
25. Merletti R, Botter A, Cescon C, Minetto MA, Vieira TMM (2010) Advances in surface EMG: recent progress in clinical research applications. Crit Rev Biomed Eng 38, 347-379.