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Laser Phototherapy As Primary Treatment in Acute Trismus

KEYWORDS: low-level laser therapy, temporomandibular joint disorders, mandibular trauma, masseter muscle, temporomandibular joint

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ABSTRACT ***Background and Objective:*** *This study aimed to assess the effectiveness of laser phototherapy (LPT), administered within 48 hours after an acute mandibular trauma, on restricted mouth opening and accompanying pain.*

Study Design/Materials and Methods: *From 86 patients, 43 females and 43 males, which sustained a jaw trauma within the preceding 240 hours, 54 were treated exclusively by LPT at ≤ 48 hours after injury, and 32, seen 49 to 240 hours after the injury, continued with the pharmacotherapy already set (control group). Myalgia and arthralgia diagnoses were established accordingly to the diagnostic criteria of temporomandibular joint disorders (DC/TMD). The unassisted mouth opening (MUO) and the self-reported pain assessed on a visual analogic scale (VAS) were recorded at baseline and at 15 minutes after LPT or after 15 days of pharmacotherapy. The pain was additionally assessed at day 7 and 15 for the LPT group. A 25 cm²-area, covering the masticatory muscles, the temporomandibular joint and their innervation was scanned by a laser beam during 60 seconds (10 mm/s speed). An optic fibre of 600 µm delivered, at 5 Hz, a double wavelength: 635 nm (125 mW, 29.6 W/cm², 5 J) and 810 nm (1.3 W, 52 W/cm², 52 J).*

Results: *Regardless of gender or age, the posttreatment MUO was increased by 39 % (18.24 ± 4.63 mm) in the LPT group and by 29 % (10.22 ± 4.25 mm) in the control group ($p < 0.0001$). The VAS score diminished from 8.06 ± 1.25 to 2.37 ± 1.25 at 15 minutes after LPT and from 7.78 ± 1.45 to 5.66 ± 1.47 after 15 days of pharmacotherapy ($p < 0.0001$). At day 7 after LPT, the VAS score showed another significant reduction but remained unchanged at day 15.*

Conclusion: *when applied within the 48 hours after a facial trauma, LPT alone can resolve, in 15 minutes, both restricted mouth opening and pain in acute posttraumatic painful trismus, for 15 days.*

Introduction

The most common cause of facial pain is a group of musculoskeletal conditions called temporomandibular joint disorders (TMDs) [1] responsible of pain and dysfunction in the temporomandibular joint (TMJ) and masticatory muscles, affecting individuals most often between the ages of 20 and 40 and of feminine gender [2].

Acute trauma has been incriminated as the precipitating event in 43 % of TMDs patients [3]. In 720 patients (1,151 joints) arthroscopically treated for displaced disc, 60 % had sustained a TMJ acute trauma, more than a year before; only 10% had had concomitant mandibular fracture(s) [4]. In other 20 patients with facial trauma responsible of mandibular fracture(s) and TMJs injury, systematic bilateral arthroscopy performed within 2 to 10 days after trauma, noted in most cases greater degrees of TMJ damages on the side without fracture [5]. These reports support the commonness and severity of the TMJ damages occurred during a blow to the jaw. However, the masticatory musculature is the main source of pain and dysfunction in posttraumatic TMDs [5,6]; in these patients, compared to those without trauma history, the maximal mouth opening was significantly more reduced [8].

The term trismus defines any muscular dysfunction responsible of restricted mouth opening [9]. Orofacial traumas are common etiologies of trismus, due to the nociceptive inputs they generate; these are processed in the spinal trigeminal nuclear complex (STNC), inducing sustained bilateral increase in electromyographic (EMG) activity of both elevators and depressors of the jaw [10-11]. Animal experiments have shown that the inflammation of orofacial deep tissues produces stronger central neuronal activation in the STNC than does cutaneous inflammation [12-13]. Also, more widespread neuronal excitation is induced by inflammation in TMJ than in perioral skin [14]. Moreover, neuronal activation, as indicated by Fos protein expression, is induced bilaterally by the masseter inflammation, while it is ipsilateral following a skin-cut over the masseter muscle [15-16]. When injected into the TMJ, mustard oil induces greater activation of masticatory muscles, as compared to cutaneous injections [17]. Inflammatory substances injected into masticatory muscles [18] or TMJ [19] increase the excitability and expand the receptive fields of trigeminal nociceptive neurons; as early as at 30 minutes afterwards, reactive astrocytes were seen in the STNC and lasts for about one week after inflammation [20]. Glial hyperactivity induced by masseter inflammation was correlated to the hyperalgesia onset [21-22]. Noxious stimulation emanating from injured craniofacial tissues triggers a cascade of cellular events in the central nervous system, including the activation of neurotransmitter receptors and neuron-glia-cytokine interactions, which leads to long-term increases in excitability and plasticity, referred to as central sensitization; this might explain some unusual patterns of pain referral in myogenous TMDs [23] and underlies the mechanisms of persistent pain [24-25].

Volume : 6 | Issue : 10 | October 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

Acute TMJ traumas [26] can cause: a) discal ligaments elongation, inducing discal displacement or dislocation; b) overloading of the articular surfaces, resulting in acute synovitis; c) capsular abusive strain, inducing acute capsulitis; d) acute retrodiscitis, when the condyle is forced posteriorly.

Clinically, acute arthralgia is always accompanied by a bilateral tonic contraction of the masticatory muscles, called protective co-contraction [27] , which is not a pathologic condition in itself. Actually, reflex trismus is not always associated with myalgia [28]; in this case, mouth opening is limited, but when the patient is asked to open slowly, full opening is achieved. However, prolonged protective co-contraction may lead to muscle soreness, a primary, non-inflammatory, myogenous pain disorder characterized by the release of algogenic substances, such as bradykinin, substance P and histamine, which activate and sensitize the muscle nociceptors [29].

Laser phototherapy (LPT) has been used for more than three decades for the treatment of musculoskeletal disorders [30], including those of the masticatory system [31]. The LPT efficacy in chronic TMDs treatment is supported by many studies [32-37], although unsuccessful results have also been reported [38-41]. Globally, LPT appeared less effective in myogenous than in arthrogenous TMDs [42]. Functional outcomes were found poorer than pain relief [43-45] or vice versa [46-47]. Very few studies [48-49, 36] addressed the LPT efficacy in acute (< 30 days) or recent (< 6 months) TMDs.

Inappropriate irradiation dose was identified as a major factor for LPT negative outcomes in musculoskeletal disorders [50]. Given the mechanisms of reflex trismus, hyperalgesia, neurogenic inflammation and central sensitization, which develop very soon after a trauma, another possible reason could be an inappropriate irradiation surface, which does not cover at the same time all the pathologically-involved tissues, namely TMJs, masticatory muscles (most often bilaterally involved), and their afferent/efferent innervation.

Some findings support the idea that the period of treatment may be critical: a) after an acute ankle sprain, the pain increases between day 3 and day 14 [51]; b) during the initial 7 days (inflammatory phase) of tendon repair LPT induced much greater stimulatory effects than during the following next 7 days [52]; c) in muscular and articular TMDs, the same LPT protocol was successful in chronic cases and totally unsuccessful in recent TMDs [48]; d) LPT yielded better outcomes in acute than in chronic TMDs [36].

The aim of our study was to assess the LPT effectiveness in resolving the acute posttraumatic restricted mouth opening and the accompanying pain, when applied simultaneously to all the involved deep tissues, within the first 48 hours after injury.

Materials and Methods

Study population

One-hundred consecutive patients presenting acute post-traumatic painful trismus were recruited for this study, which was conducted with respect to the recommendations of our university ethic committee.

The including criteria were: direct jaw trauma within the prior 240 hours.

The exclusion criteria were: TMD history, systemic diseases of any etiology, and psychiatric disorders.

Finally, 86 patients have been enrolled, 36 of ages between 14 and 40 and 50 of ages between 41 and 68; 43 were females (41 ± 15 years old) and 43 were males (43 ± 11 years old). Their trauma was a consequence of car, bicycle or horse accident, falling-down, sports, violent attack or iatrogenic procedures (jaw overextension). No patient alleged cervical hyperextension/hyperflexion (whiplash) injury.

According to the time elapsed between trauma and clinical examination, the patients were distributed as follows: 54 patients (LPT group) with a jaw trauma occurred within the prior 4 to 48 hours (22.9 ± 1.8 hours), having no treatment since then, except for paracetamol taken more than 6 hours prior to the examination and 32 patients (control group) seen at 49 to 240 hours (106.6 ± 52.5 hours) after trauma, and having an exclusive, uninterrupted pharmacotherapy by a single drug: an analgesic (21 patients), a nonsteroidal anti-inflammatory drug (NSAID) (8 patients), a muscle relaxant (2 patients) or a corticosteroid (1 patient).

All the participants gave written, informed consent. The LPT patients agreed to the use of LPT alone and to avoid any other treatment, excepting in case of more intense pain than they would experience at the end of the procedure. They also consented to be telephonically contacted for pain assessment at 7 and 15 days after LPT. The control patients agreed to continue with the medication already set up and to avoid any other drugs or therapies until the next evaluation, scheduled after 15 days of pharmacotherapy.

Clinical examinations

The patients completed a symptom questionnaire consisting in items with established reliability (53, 54) focused on acute TMDs. It was designed to collect data related to trauma circumstances, pain and jaw function, TMD history and general health, as well as to assess pain intensity on a visual analogue scale (VAS), where 0 indicated "no pain" and 10 indicated "the worst imaginable pain".

The clinical examination of all the patients was performed by the same examiner (A. F-G), according to the examination protocol of diagnostic criteria of TMD (DC/TMD) (55). Because of the intense pain experienced by most of the participants the maximum assisted opening could not always be measured, so the maximum unassisted opening (MUO), including incisor overbite, was chosen as the representative measurement of the jaw mobility. Each recorded value was a mean of three measurements, read to the nearest millimeter on an interincisally-placed ruler.

The pain-related TMDs diagnoses (Table 1, Table 2) were established according to the Axis I DC/TMD (56). A panoramic radiograph was taken for each patient and it ruled out any fracture or visible TMJ abnormality.

The clinical outcomes were assessed with regard to the MUO and the VAS score, either the same day (D0), 15 minutes after LPT, or 15 days after the baseline examination (control patients). Follow-up contacts were conducted for the LPT patients and noted that none of them needed any other treatment during the study.

Volume : 6 | Issue : 10 | October 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

LPT protocol
The Digilase PDT 5W250 laser (Biophoton, Saint Alban, France, 2013), integrating two emitters, simultaneously delivered two wavelengths of 635 nm (125 mW, 29.6 W/cm², 5 J) and 810 nm (1.3 W, 52 W/cm², 52 J), at a frequency of 5 Hz, by using an optic fibre of 600 µm in a quasi-contact mode, perpendicular to the skin at 1 mm (Table 3). During irradiation, all the persons present put on the safety glasses provided by the manufacturer.

An area of 25 cm² (6.5 x 4 cm), including the TMJ lateral aspect and part of the auriculotemporal nerve, the masseter muscle and its innervation, as well as the lower part of the temporalis fossa, including the muscle and its innervation (Figure 1), was scanned by the laser beam during 60 seconds, at a speed of 10 mm/s (lower values provoked burning sensations).

LPT was administered in a unique session, bilaterally in case of bilateral myalgia. If in opposite sides, myalgia was treated as before and arthralgia was treated by irradiating only the TMJ area (Figure 1, area 1).

Statistical analyses
The statistics and their graphs were performed by Prism® software version 6 (GraphPad Software, Inc., San Diego, USA). The threshold of significance was set at $p < 0.05$ for all the tests.

Mann-Whitney test served to compare the control and LPT groups and subgroups, as regards with ages, MUO and VAS score at the baseline assessments.

Two-way analysis of variance (ANOVA) in conjunction with Tukey's multiple comparisons test served to compare the MUO before and after treatment, as well as the MUO gain, between the control and LPT patients, in all the subgroup counterparts. The same tests were applied for the VAS score before and after treatment, as well as for the VAS score reduction. The variances' homogeneity was confirmed by the Brown-Forsythe test.

In the LPT patients, the within-subject VAS score evolution has been analysed by repeated-measures ANOVA (RM ANOVA) with Greenhouse-Geisser correction.

Results
There were no statistical differences between the control and LPT groups as regarding with ages and genders distribution.

At baseline, the self-reported pain was equally intense in all the subgroups of control and LPT patients, while the MUO was significantly more limited in all the subgroups of the control patients. Posttreatment assessments indicated that both MUO (Table 4 and Figure 2) and self-reported pain (Table 5 and Figure 3) were significantly poorer in all the control subgroups ($p < 0.0001$), compared to the laser-treated counterparts.

At 15 minutes after LPT, the MUO has increased by 39 % (18.24 ± 4.63 mm), regardless of gender or age. Meanwhile, the self-reported pain diminished by almost three quarters, from 8.06 ± 1.25 to 2.37 ± 1.25 VAS score. Equally strong significant difference was noted within each subgroup. Then, the pain relieving progressed until the day 7, when the VAS score mean showed again a significant reduction compared to the D0 post-LPT assessment. At day 7, most of the patients (83.3%) reported to be pain-free. At day 15, the VAS score was 1 in 5 patients. No significant difference was noted within the LPT subgroups between the VAS score means at D7 and D15. For a given time point, the between-subgroups VAS score analysis showed no significant difference.

Discussion
The baseline clinical examination revealed that: a) arthralgia was present in all the patients who sustained a blow to the mandible; b) the chief complaint was always a painful trismus occurred within hours after trauma; an opening lesser than 40 mm, considered as limited [57], was noted in 100 % of cases, with the poorest values associated to longer durations of symptoms (control group); c) myalgia of myofascial type, simultaneously affecting the masseter and temporalis muscles, has been noted in most of the patients, incriminating neurogenic inflammation and sensitization mechanisms at peripheral and central levels;

d) neuroplasticity manifestations, including opposite side/bilateral myalgia of myofascial type and heterotopic pain, such as referral masseteric pain in the TMJ area and headache attributed to TMD, were common, with a stronger prevalence in the patients sustaining symptoms for longer durations (34 % in the control group vs. 15 % in the LPT group). In order to limit the risk of developing chronic posttraumatic TMDs, which are much more resistant to treatments [6-8], relieving as soon as possible an acute posttraumatic painful trismus should be considered of the utmost importance [29].

The rational for any therapy is based on a pathophysiological understanding of the complaints; now the underlying pathophysiological mechanisms in TMDs remain unclear, especially concerning the muscles' involvement, mostly explored in animal experiments [58]. Human studies showed that: a) the local anaesthesia of one painful TMJ in TMD patients reduces the increased stretch reflex response, bilaterally in masseters and unilaterally in temporalis muscles [59]; b) an increased intramuscular pressure during contraction causes blood vessels compression, responsible for metabolic debt, which induces compensatory postcontraction vasodilation; studies on healthy volunteers demonstrated that contraction in the jaw-elevator muscles as low as 5 % of the maximal voluntary contraction (MVC) is enough to induce postcontraction hyperaemia; during contraction, the venous occlusion is stronger and lasts more in masseter than in temporalis muscles [60]; c) in healthy subjects, painful stimulation induced by hypertonic saline infusion has shown that the muscular pain results in slowing and/or de-recruitment of one population of motor units, while a new population of units is recruited, so as to maintain the jaw-closing force [61]; d) similar experiments in the left masseter showed increased EMG activity of the contralateral masseter and both temporalis muscles, to compensate for the EMG decrease in the painful muscle, as to preserve the functional demands; rather than MVC at 100 %, low-level clenching such as 50% MVC, a force level required to break down food items [62], or 5% MVC, required during swallowing or talking [63], can cause extra-fatigue in non-painful muscles [64] and, finally, a widespread myogenous pain limiting the mouth opening. These data demonstrate that studies on TMD therapies assessing functional parameters or global jaw functions during MVC may falsely conclude to poor functional outcomes; this pitfall wasn't avoided in some studies on LPT efficiency in TMDs.

Beyond the discrediting conclusions due to methodologi-

Volume : 6 | Issue : 10 | October 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

cal issues, the LPT remains controversial because: a) the understanding of its underlying mechanisms at cell and tissue levels is still limited, so its use is largely empirical; b) any of its applications requires optimal irradiation parameters and a less than optimal choice yields poor outcomes [65]. To generate favorable clinical outcomes, the LPT has to induce biomodulation in an appropriate tissue volume. The tissue penetration depth depends on the wavelength: an 810-nm laser beam can penetrate several centimeters into tissue; a 632.8-nm wavelength penetrates to a depth of 8-10 mm [66]. The latter is attenuated by muscle and cartilage tissues up to 35 % [67], mostly by scattering than by absorption [68]. This attenuation is not significantly affected by the overlying skin, is directly proportional to the muscle thickness and not related to the average power of the light source [67]. In relaxed state, the mean thickness is 10.6 - 13 mm for masseters [69] and 4 - 7.7 mm for temporalis muscles [70]. From the skin, the maximal TMJ depth is 19-32 mm [71].

Studies on LPT in TMDs, which used wavelengths of 780, 790 and 830 nm, combined or not to a 660-nm wavelength, have shown that combined red-infrared wavelengths were the most efficient in alleviating myogenous pain; arthrogenous pain was equally relieved by combined and not combined infrared wavelengths [72]. Studies on LPT ability to accelerate skin wound healing in rats have found that a 632-nm wavelength was the most efficient (among six wavelengths between 442 and 830 nm), despite its lesser skin transmission, compared to that of an 830-nm wavelength [73]. This suggests that even diminished by a low skin transmission, an irradiating wavelength well-matched to the absorption spectrum of a cellular photoacceptor induces cellular effects. Now the most important photoacceptor is considered to be the terminal enzyme of the respiratory chain, cytochrome c oxidase, whose absorption specter has four peaks in red and near infrared domains: 620, 680, 760, and 820 nm [74]. The simultaneous use of two wavelengths of 635 and 810 nm could induce a higher mitochondrial activation, increasing the electron transport, cell respiration, oxygen consumption and ATP production. Thus, the muscular function, inducing electrolytic and metabolic changes (ATP and glycogen depletion, oxidative stress, tissue hypoxia and acidification), can be optimized in hypoxic conditions, such as mechanical stress, fatigue and neurogenic inflammation.

The LPT-induced muscle relaxation and analgesia may be the result of anti-inflammatory effects [50], neural blockade of nociceptors and motor nerves inhibition [75]. Indeed, an enhanced mitochondrial activity initiates signaling pathways (via reactive oxygen species, nitric oxide, and cyclic AMP), leading to the activation of several transcription factors; by regulating the expression of genes, they modulate the levels of cytokines, growth factors and inflammatory mediators [65]. Our outcomes at 15 minutes are consistent with the time frame reported for laser-induced inhibition of noxious transduction/transmission and motor nerves blockade [75]. Thus, direct effects on somatosensory and/or motor nerves could be responsible, at least partly, of resolving the painful trismus.

The distribution of the energy applied on muscles, so as to cover the largest area, appears to be another important parameter, as demonstrated by studies on muscular pre-conditioning with LPT [76]. On the other side, the best LPT effectiveness on nerves appears to be due to an additive effect caused by the irradiation at several points rather than to a single point [77]. Such an effect is the most likely obtained by scanning a large area, if not the whole muscle.

In the abundant literature dedicated to TMDs and LPT, very few studies dealt with acute TMDs. We could not find any study on LPT in acute TMDs performed within hours after their onset. Thus, it is challenging to compare our results to that of the literature. However, it should be noted that in the only study which compared the effectiveness of LPT versus NSAIDs in TMDs (arthrogenous chronic TMDs), the magnetic resonance imaging following a 10-days treatment revealed that the TMJ intra-articular effusion completely disappeared after daily LPT, but not after the NSAID conventional treatment [33]. In line with the recommended dose for LPT in TMJ acute arthritis [78], the combination of parameters in this protocol may induce anti-inflammatory effects in myogenous complaints, as well.

Current primary therapy of acute musculoskeletal traumas, including masticatory system [58], is based on a largely empirical pharmacotherapy, despite their potentially severe adverse effects [79] and interferences with tissue recovery processes, such as delayed oedema resorption induced by NSAIDs [51]. In posttraumatic acute conditions, the effectiveness of paracetamol compared to NSAID's is not yet well-established [79]. Also, it has been shown that prototypic NSAIDs or muscle relaxants administered alone were not more efficient than placebo in short-term management of myogenous TMDs [80]. Therefore, one could say that the control group in this study experienced an evolution which was likely not much different of the natural one in posttraumatic acute TMDs.

Previous studies have reported a laser-induced placebo effect quickly decreasing after LPT, regardless of the arthrogenic/myogenic origin or the acute/chronic nature of TMDs [33, 38, 49]. This study has shown that the pain relief was stable at 15 days, which was longer then the reported durations of the LPT placebo effect.

Conclusion

Acute posttraumatic painful trismus with a history of less than 48 hours was resolved immediately by LPT alone, by scanning the whole area of the involved structures (TMJ, masticatory muscles and their respective innervation), bilaterally if necessary, with a combined red-infrared laser. The benefit of one-session LPT over pharmacotherapy was highly significant and the LPT outcomes were stable at day 15. Further randomized double-blinded clinical trial and longer follow-up are needed to confirm these very encouraging outcomes and their stability.

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Table 1 – The pain-related TMD diagnoses established according to DC/TMD in patients with painful posttraumatic acute trismus always associated acute arthralgia to one of the myalgia types. Most often, the masseter and temporalis muscles were both affected, either unilaterally (on the side of the arthralgia or on the opposite side) or bilaterally. For symptoms' evolutions longer than 48 hours (control group, n = 32), the headache attributed to TMD was more than twice as frequent as for shorter evolutions (LPT group, n = 54).

Volume : 6 | Issue : 10 | October 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

Acute Pain-Related TMD Diagnoses LPT		One/same side		Opposite side		Both sides	
		Control	LPT	Control	LPT	Control	
Arthralgia	without myalgia	0	0	0	0	0	0
	associated to myalgia	27 (50%)	15 (47 %)	8 (15 %)	3 (9 %)	19 (35 %)	14 (44 %)
Myalgia	masseter muscle only	0	0	1 (2 %)	1 (3 %)	2 (4 %)	2 (6%)
	temporalis muscle only	0	0	0	0	0	0
	masseter & temporalis muscles	27 (50 %)	15 (47 %)	7 (13 %)	2 (6 %)	17 (31 %)	12 (38 %)
Headache attributed to TMD		5 (9 %)	8 (25 %)	2 (4 %)	1 (3 %)	1 (2 %)	2 (6 %)

Table 2 – The myalgia type was noted for each painful muscle, regardless of the association to arthralgia or their unilateral/bilateral character. Globally, the masseters were more affected than the temporalis muscles and most often, the myalgia was spread in the whole muscle (myofascial pain). Further pain spreading (myofascial pain with referral) has been noted in longer symptoms' evolutions (≥ 49 hours in the control group, n = 32) than in shorter ones (≤ 48 hours in the LPT group, n = 54).

Myalgia type	Masseter		Temporalis	
	LPT	Control	LPT	Control
Local myalgia	5 (7 %)	5 (11 %)	4 (6 %)	3 (7 %)
Myofascial pain	59 (81 %)	28 (61 %)	56 (82 %)	26 (63 %)
Myofascial pain with referral	9 (12 %)	13 (28 %)	8 (12 %)	12 (29 %)

Figure 1 – The laser scanned a surface of about 25 cm², composed of the following areas:

1 – the TMJ lateral aspect and the auriculotemporal nerve, running between the condyle neck and the external auditory canal, at minimum 8 mm in front of the posterior aspect of the tragus ;

2 – the whole surface of the masseter muscle, underneath the zygomatic arch, including the mandibular notch, from which the masseteric nerve emerges;

3 – the lower part of the temporalis muscle, about 1.5 cm above the zygomatic arch, between the auricle and the orbital ridge, by avoiding the haired skin. The deep temporalis nerves travel upwards at the temporalis bone contact, just above the superior ridge of the zygomatic arch.

The zygomatic arch has not been irradiated. Arthralgia was always accompanied by myalgia, and sometimes by headache. If on one side only arthralgia was present, area 1 alone was irradiated.

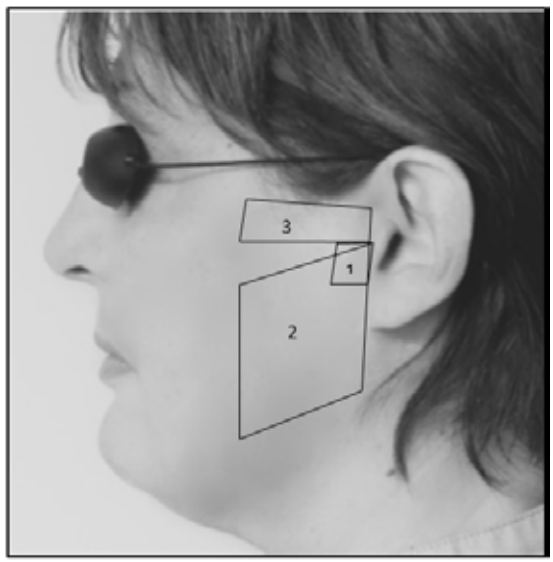


Table 3 – The irradiation parameters and the dosimetry are indicated for each of both wavelengths composing the laser beam. The whole surface (areas 1 + 2 + 3 = 25 cm²) received during 60 s a total radiant energy of 57 J (i.e. 2.3 J/cm²). The values in the brackets correspond to the TMJ (area 1 = 2.3 cm²), when irradiated alone: it received during 10 s a total radiant energy of 9.4 J (i.e. 4 J/cm²).

Parameter	Value	
Central wavelength (nm)	635	810
Spectral bandwidth (FWHM), nm	1.6	1.6
Emitter Type	InGaAlP	GaAlAs
Operating mode	pulsed	pulsed
Frequency (Hz)	5	5
Pulse on duration (sec)	0.133	0.133
Pulse off duration (sec)	0.07	0.07
Peak radiant power (mW)	125	1300
Average radiant power (mW)	83	864.5
Aperture diameter (cm)	$6 \cdot 10^{-2}$	$6 \cdot 10^{-2}$
Beam divergence (degrees)	10	10
Beam spot size at target (cm²)	$28 \cdot 10^{-4}$	$28 \cdot 10^{-4}$
Irradiance at target (W/cm²)	29.6	308.8
Exposure duration (s)	60 (10)	60 (10)
Radiant exposure (kJ/cm²)	1.8 (0.3)	18.5 (3)

Volume : 6 | Issue : 10 | October 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

Radiant energy (J)	5 (0.8)	52 (8.6)
Irradiated area (cm²)	25 (2.3)	25 (2.3)
Total radiant energy (kJ)	45 (0.7)	463 (7)
Polarization	no	

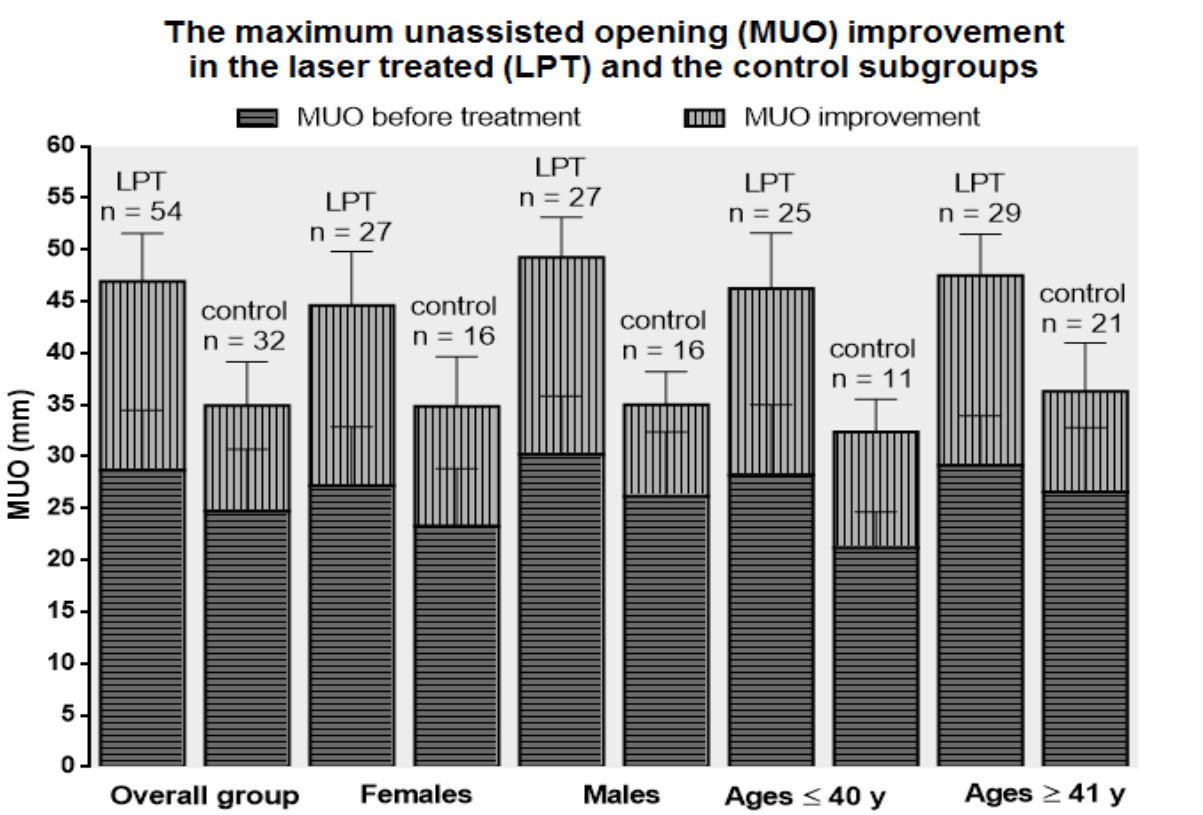
Beam shape	circular
Beam profile	top hat
Beam Delivery System	silica optical fibre
Application technique	scanning in quasi contact mode

No. of treatment sessions	1

Table 4 – Maximum unassisted opening (MUO) mean, standard error of measurement (SEM) and standard deviation (SD), before and after treatment in the subgroups of control and LPT patients. The clinical improvement (MUO gain) was significantly poorer in all the control subgroups.

MUO (mm) LPT n = 54		Overall		Females		Males		Ages ≤ 40 y		Ages ≥ 41 y	
		Control n = 32	LPT n = 27	Control n = 16	LPT n = 27	control n = 16	LPT n = 25	Control n = 11	LPT n = 29	Control n = 21	
before treatment	Mean	28.70	24.72	27.19	23.25	30.22	26.19	28.20	21.18	29.14	26.57
	SEM	0.79	1.06	1.09	5.57	1.08	6.19	1.36	3.46	0.90	6.24
	SD	5.79	5.98	5.66	1.39	5.60	1.55	6.81	1.04	4.82	1.36
after treatment	Mean	46.94	34.94	44.63	34.88	49.26	35.00	46.28	32.36	47.52	36.29
	SEM	0.69	0.73	0.86	3.32	0.90	4.90	1.17	3.85	0.81	3.65
	SD	5.10	4.12	4.47	0.83	4.68	1.22	5.85	1.16	4.38	0.80
gain	Mean	18.24	10.22	17.44	11.63	19.04	8.81	18.08	11.18	18.38	9.71
	SEM	0.63	0.75	1.01	4.79	0.75	3.19	1.07	3.16	0.74	4.71
	SD	4.63	4.25	5.23	1.20	3.89	0.80	5.35	0.95	4.00	1.03

Figure 2 – In LPT patients, the MUO gain was significantly higher than in the corresponding control subgroups. No significant differences have been observed between the subgroups of each group concerning the MUO before and after treatment or the MUO gain, except for the posttreatment MUO between the gender subgroups in LPT patients.



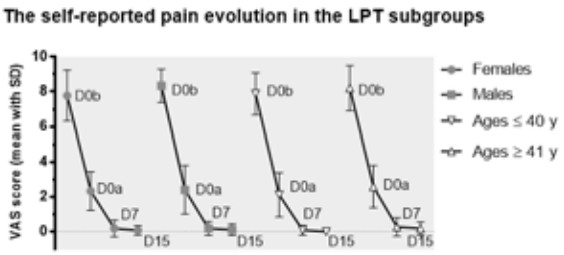
Volume : 6 | Issue : 10 | October 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

Table 5 – In the LPT group, the VAS score was assessed at four time points, at day 0 before and after LPT, at day 7 and 15; in the control patients it has been assessed at day 0 and 15. The between-subgroups analysis performed in the same group for each time point has not revealed any significant difference. Significant differences were observed between all the corresponding LPT and control subgroups, for the posttreatment VAS score and its reduction between the first and last assessment.

VAS score LPT n = 54		Overall		Females		Males		Ages ≤ 40 y		Ages ≥ 41 y	
		Control n = 32	LPT n = 27	Control n = 16	LPT n = 27	Control n = 16	LPT n = 25	Control n = 11	LPT n = 29	Control n = 21	
D0 before treatment	Mean	8.06	7.78	7.78	7.69	8.33	7.88	7.88	7.82	8.21	7.76
	SEM	0.17	0.21	0.28	1.01	0.18	1.36	0.24	1.08	0.24	1.26
	SD	1.25	1.18	1.45	0.25	0.96	0.34	1.20	0.33	1.29	0.28
D0 after treatment	Mean	2.37		2.33		2.41		2.12		2.59	
	SEM	0.17	–	0.21	–	0.27	–	0.25	–	0.22	–
	SD	1.25		1.11		1.39		1.27		1.21	
at D7	Mean	0.19		0.19		0.19		0.08		0.28	
	SEM	0.06	–	0.09	–	0.08	–	0.06	–	0.10	–
	SD	0.44		0.48		0.40		0.28		0.53	
at D15	Mean	0.09	2.13	0.07	2.44	0.11	1.81	0.00	1.82	0.17	2.29
	SEM	0.04	0.18	0.05	1.15	0.06	0.75	0.00	1.17	0.07	0.90
	SD	0.29	1.01	0.27	0.29	0.32	0.19	0.00	0.35	0.38	0.20
reduction at D0	Mean	5.69		5.44		5.93		5.76		5.62	
	SEM	0.21	–	0.31	–	0.28	–	0.36	–	0.24	–
	SD	1.54		1.60		1.47		1.81		1.29	
reduction D0 ○ D7	Mean	2.19		2.15		2.22		2.04		2.31	
	SEM	0.17	–	0.22	–	0.26	–	0.25	–	0.23	–
	SD	1.24		1.13		1.37		1.24		1.26	
reduction D7 ○ D15	Mean	0.09		0.11		0.12		0.08		0.10	
	SEM	0.05	–	0.06	–	0.06	–	0.06	–	0.08	–
	SD	0.35		0.32		0.33		0.28		0.41	
reduction D0 ○ D15	Mean	7.96	5.66	7.67	5.25	8.22	6.06	7.88	6.00	8.03	5.48
	SEM	0.18	0.26	0.27	1.39	0.19	1.48	0.24	1.55	0.26	1.44
	SD	1.89	1.47	1.41	0.35	0.97	0.37	1.20	0.47	1.38	0.31

Volume : 6 | Issue : 10 | October 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

Figure 3 – The self-reported pain at day 0, before (D0b) and after (D0a) the laser phototherapy (LPT), at day 7 (D7) and 15 (D15). Within a given subgroup, the VAS score means at successive time points was compared by RM ANOVA. This within-subgroup analysis revealed significant reductions between the first three time points, while no difference was observed between the last two time points.



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Volume : 6 | Issue : 10 | October 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

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