The Efficacy of Dry Needling and Procaine in the Treatment of Myofascial Pain in the Jaw Muscles

Anne S. McMillan, BDS, PhD, FDSRCPS
Senior Lecturer/Consultant
Head of the Prosthodontic Unit
Department of Restorative Dentistry

Anita Nolan, MD, BCh, FFDRCSI
Senior Lecturer/Consultant
Department of Oral Medicine

University of Newcastle
Newcastle upon Tyne
United Kingdom

Peter J. Kelly, BSc, PhD, C Stat
Director
Centre for Health and Medical Research
University of Teesside
Middlesborough, Teesside
United Kingdom

Correspondence to:
Dr Anne S. McMillan
Department of Restorative Dentistry
University of Newcastle
Framlington Place
Newcastle upon Tyne
United Kingdom NE2 4BW

In patients with myofascial pain, painful trigger points are often treated using dry needling and local anesthetic injections. However, the therapeutic effect of these treatments has been poorly quantified, and the mechanism underlying the effect is poorly understood. In a randomized, double-blind, double-placebo clinical trial, a pressure algometer was used to measure pain-pressure thresholds in the masseter and temporalis muscles of 30 subjects aged 23 to 53 years with myofascial pain in the jaws, before and after a series of dry needling treatments, local anesthetic injections, and simulated dry needling and local anesthetic treatments (treatment group A: Procaine + simulated dry needling; treatment group B: dry needling + simulated local anesthetic; control group C: simulated local anesthetic + simulated dry needling). Subjects rated pain intensity and unpleasantness using visual analogue scales, and the data were analyzed using analysis of variance. Pain pressure thresholds increased slightly after treatment, irrespective of the treatment modality. Pain intensity and unpleasantness scores decreased significantly at the end of treatment in all groups. There were no statistically significant between-group differences in pain pressure thresholds and visual analogue scale scores at the end of treatment. The findings suggest that the general improvement in pain symptoms was the result of nonspecific, placebo-related factors rather than a true treatment effect. Thus, the therapeutic value of dry needling and Procaine in the management of myofascial pain in the jaw muscles is questionable.
J OROFACIAL PAIN 1997;11:307–314.

key words: myofascial pain, dry needling, simulated dry needling, local anesthetic, Procaine, trigger points

Myofascial pain is a major cause of nondental pain in the orofacial region, accounting for approximately 30% of patients who seek treatment for temporomandibular disorders (TMD).[1] It is characterized by a regionalized, dull, aching pain and the presence of trigger points (TPs) in jaw muscles, tendons, or fascia that produce pain on palpation, leading to regional referred pain.[1,2] There are, however, no apparent histopathologic, biochemical, or electrophysiologic causes.[3]

Reduction and, in some cases, elimination of pain and tenderness at TPs and referral sites have resulted from acupuncture-like "dry needling" and injection of Procaine local anesthetic (LA) into active TPs, although the decrease in pain symptoms has been poorly quantified.[4,5] A common weakness in clinical studies of acupunc-

ture and related treatment has been inappropriate experimental design, notably the lack of a control group.[4,6,7] Results have often been difficult to interpret, giving no indication of outcome in the absence of active treatment (ie, nontreatment or placebo). Simulated acupuncture, in which the needle is inserted just under the skin at a nonpainful site, has been mooted as an appropriate control.[6,8]

It is also unclear whether pain relief is more profound and sustained if treatment is given on more than one occasion.[5] The present authors found that a single injection of Procaine into TPs in the masseter muscle did not result in significant changes in jaw muscle pain-pressure thresholds (PPTs).[9] Repeated intervention appears to be necessary for long-term relief of symptoms.[4]

The location of TPs in the jaw muscles is correlated with known acupuncture points in the jaws.[10] Stimulation of these areas has been shown to produce an analgesic effect locally and at more distant sites. Thus, the stimulation of the needle itself seems to have a general effect on somatosensory thresholds in the orofacial region. In contrast, when Procaine is given diagnostically, the blocking effect is presumed to occur locally at the injection site, but LA injected peripherally may reach the central nervous system (CNS), particularly when no vasoconstrictor is used to impede systemic absorption.[11] Thus, changes in tissue pain thresholds may be an effect of the LA on the CNS, or the tissue irritation effect of the needle, or both.[12,13] However, Baldry[4] observed that concurrent administration of LA may diminish the pain-relieving effect of the needle itself.

Dry needling and Procaine injection of TPs in painful jaw muscles appear to reduce pain symptoms, both locally and at more distant sites in the jaws, although the relative therapeutic efficacy of these treatment modalities is presently uncertain. Therefore, the purpose of this randomized, double-blind, double-placebo clinical trial was to determine the relative efficacy of dry needling and Procaine in subjects with myofascial pain in the orofacial region by quantifying immediate and long-term relief of pain after a series of dry needling, LA injection treatments, and simulated dry needling and LA treatments.

Materials and Methods

Subjects

Subjects were recruited from patients attending Newcastle Dental Hospital Admissions Department and Temporomandibular Joint Clinic for the treatment of temporomandibular disorders and orofacial pain. Members of the study population were deemed to have craniofacial pain of myogenous origin. Diagnoses were made on the basis of the International Headache Society's classification of myofascial pain.[1] The essential criteria for inclusion in the study were as follows: *(1)* women in the age range of 20 to 50 years (because significantly more women than men seek treatment for TMD[1]); *(2)* a primary complaint of frequent pain (at least four times per week) in the jaw muscles, of at least 12 weeks' duration; *(3)* tenderness to palpation at a minimum of three sites in the jaw muscles, including at least one in the masseter; and *(4)* palpation of a tender area in the masseter which led to changes in patterns of referred pain. Subjects were excluded from the study if they had any of the following: clinical and/or radiographic signs of pathology in the TMJ; metabolic disease; neurologic disorders such as dyskinesia; vascular disorders such as migraine; bleeding diatheses; neoplasia; a history of psychiatric illness; a history of drug abuse; recent facial or neck trauma; medication or adjunctive treatment (eg, physiotherapy) that could not be stopped during the study; or allergy to local anesthetic solutions.

Thirty patients, aged 23 to 53 years, participated in the study. To determine the sample size, a power analysis was performed on data described previously by the present authors.[9] The main outcome variable used was patients' pain reports. With 10 subjects in each group, it was estimated that there was 90% power to detect that a difference of 60% between the groups reached statistical significance ($\alpha = 0.05$).

Trigger Point Location

After the initial screening, subjects were examined on three occasions. At each experimental session, the distribution of tender areas in the masseter and temporalis muscles was noted by one clinician (AN). The presence of an active TP was determined on the basis of local and referred symptoms intensifying on firm palpation. The distribution of tender spots was noted, and the skin overlying the most tender area in the masseter was marked with a felt-tip pen. The other clinician (ASM), who conducted all pain measurements, was not present during the trigger point location process and was blinded to patient symptoms and treatment.

Experiment Design

In this prospective, randomized, double-blind, double-placebo clinical trial with parallel groups,

subjects were randomly assigned to one of the experimental treatment groups, A, B, or C, which were stratified by age (above and below 35 years). Clinician AN administered all treatments. The first group (A) received a percutaneous injection of 0.5 mL Procaine (1%) local anesthetic with no vasoconstrictor into the active TP in the right or left masseter by means of a 27-gauge hypodermic needle and disposable syringe. An acupuncture needle (Seirin Kasei, Shimizu City, Japan) was also placed just into the skin over a nontender part of the muscle, then removed immediately (simulated dry needling). The second group (B) received an acupuncture needle percutaneously into an active TP in the masseter. The needle was left in situ for 1 to 2 minutes. A drop of isotonic saline was also introduced just below the skin using a 27-gauge needle over a nontender part of the muscle (simulated LA). In the third (control) group (C), an acupuncture needle was inserted just into the skin over a nontender part of the muscle, then removed immediately. A drop of isotonic saline was also introduced percutaneously in the same area. The treatment in group C was designed to simulate dry needling and LA. Treatment was given on three occasions (by AN) 1 week apart. The experiment design is shown in Fig 1.

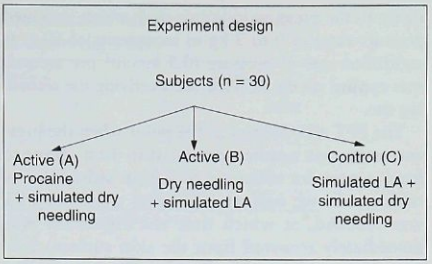

Fig 1 The study population (n = 30) and the three experimental groups: A: Procaine + simulated dry needling; B: dry needling + simulated local anaesthesia; C: simulated local anaesthesia + simulated dry needling.

Muscle Tenderness

The pain pressure threshold (PPT) was used as a measure of muscle tenderness.[5,14] The PPT recording technique has been shown to be sensitive and reliable.[5,14]

Subjects sat upright in a dental chair with their head supported by a head restraint. The masseter muscle was located, and the anterior and posterior borders of its superficial belly determined by palpation. The central point of site M1 was located 10 mm posterior to the anterior border of the muscle and 10 mm superior to the inferior border of the mandible. The central point of site M2 was located 10 mm posterior to the muscle's anterior border and 10 mm inferior to the lowest point on the zygomatic buttress. The central point of site M3 was 10 mm anterior to the muscle's posterior border, equidistant from M1 and M2 (see Fig 2).

The anterior border of the anterior temporalis muscle was located by palpation. Site T1 was 10 mm posterior to the muscles's anterior border and 10 mm above the highest point on the zygomatic buttress. Site T2 was located 20 mm above the cental point of T1. Site T3 was located 20 mm posterior to the central point of T1 (see Fig 2).

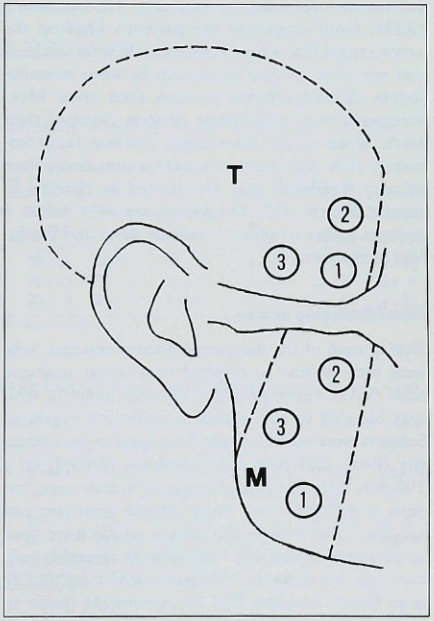

Fig 2 The location of PPT measurement sites in the right masseter (M1–M3) and temporalis (T1–T3) muscles.

Pain pressure thresholds were measured using an algometer with a recording tip of 10 mm diameter (Model PTH-AF2, Pain Diagnostics and Thermography, Great Neck, NY). The instrument was modified to incorporate the attachment of a rate

meter to the pressure indicator dial, which indicated pressure rates of 0 to 3 kg in increments of 50 g. A controlled rate of pressure (0.5 kg/cm^2 per second) was applied to the skin surface overlying the recording site.

The PPT was defined as the point when the pressure stimulus applied to the skin first changed from a pressure sensation to a pain sensation. By raising a hand, subjects indicated when the PPT was reached, at which time the algometer was immediately removed from the skin surface. Subjects were encouraged to focus on the test stimulus (algometer) in order to minimize changes in cutaneous sensitivity.[15]

Pain pressure thresholds were measured in the masseter and temporalis muscles on the side associated with the active TP. The order of measurement of PPT recording sites was randomized.[16] Two measurements were made at each site. The operator's (ASM) hand supported the patient's head on the contralateral side when measurements were made. A rest period of at least 30 seconds between measurements allowed subjects to relax their jaws. Measurements were made while subjects clenched their teeth lightly in the intercuspal position (approximately 10% maximum voluntary contraction), since sensory thresholds may be affected by changes in muscle activity.[14,17] Measurements were taken 5 minutes before treatment (baseline data) and 5 minutes after treatment.

Pain-Measuring Scales

During each of the three experimental sessions, subjects were invited to respond to a visual analogue scale (VAS), supervised by a research assistant who was blinded to the patient's treatment regimen. Subjects were asked to rate their current pain intensity (P-int) and pain unpleasantness (P-unpl) on a 100-mm VAS. For P-int, the anchor words were "no pain at all" and "the most intense pain you can imagine." For P-unpl, the anchor words were "not at all disagreeable" and "the most disagreeable pain that you can imagine." Subjects rated P-int and P-unpl before baseline PPT measurements (prior to treatment), 5 minutes after treatment, immediately before the second series of PPT measurements, 1 hour later, and 24 hours later.

A grey board (2 cm^2) on a white background was shown to subjects at each experimental session. Subjects were asked to rate the darkness of the board on a 100-mm VAS. The anchor words on the scale were "extremely pale" and "extremely dark." These data were used as a means of estimating the subjects' general sensory state over the period of the study.[18]

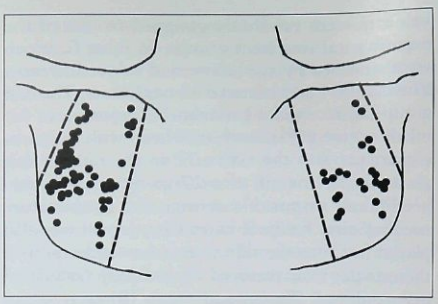

Fig 3 The location of active trigger points in the masseter muscles of myofascial pain subjects taking part in the study (n = 56).

Data Analysis

Pain pressure threshold data obtained from the two stimulus trials at each recording site, before and after treatment, were used for data analysis. Analysis of variance (balanced design) was used to compare PPTs in masseter and temporalis muscles before and after treatment, and to test for time effects and time-treatment interactions.

Analysis of the VAS data was done using ANOVA to determine the time and treatment effects on P-int, P-unpl, and visual stimulus variables.

Results

Pain Thresholds

Mean PPT data measured in the masseter and temporalis muscles of each group, before and after each treatment, are shown in Tables 1a and 1b. The treatment effect was not statistically significant for the masseter (P = .06) or temporalis muscle (P = .40). There were no specific differences in threshold increases according to treatment type. There were no time effects on the magnitude of the PPT increase after treatment, nor were there any treatment-visit interactions. There were no site differences in PPT measurements in either muscle before or after treatment. Thresholds in the anterior temporal region were consistently higher than in the masseter before and after treatment (P = .01).

The distribution of active TPs in the masseter muscles of all subjects at the three experimental sessions are shown in Fig 3. In most instances, the location of the active TP changed between experimental sessions, in the majority of occasions within the same muscle and occasionally between muscles.

Table 1a Mean Pain Pressure Threshold in the Masseter Muscle in Groups A, B, and C at Three Experimental Sessions, 1 Week Apart (kg ± SD)

	Week 1		Week 2		Week 3	
Group*	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
A	0.8 ± 0.3	0.9 ± 0.3	0.8 ± 0.2	1.0 ± 0.2	0.8 ± 0.2	1.0 ± 0.3
B	0.8 ± 0.4	0.8 ± 0.5	0.8 ± 0.4	0.9 ± 0.5	0.9 ± 0.5	1.0 ± 0.6
C	0.8 ± 0.3	0.8 ± 0.3	0.9 ± 0.4	1.0 ± 0.6	0.7 ± 0.2	0.7 ± 0.2

*Group A: Procaine + placebo; group B: dry needling + placebo; group C: double placebo control.

Table 1b Mean Pain Pressure Threshold in the Temporalis Muscle in Groups A, B, and C at Three Experimental Sessions, 1 Week Apart (kg ± SD)

	Week 1		Week 2		Week 3	
Group*	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
A	1.2 ± 0.3	1.2 ± 0.2	1.3 ± 0.3	1.4 ± 0.3	1.2 ± 0.3	1.3 ± 0.3
B	1.3 ± 0.7	1.3 ± 0.8	1.1 ± 0.6	1.3 ± 0.7	1.4 ± 0.8	1.4 ± 1.0
C	1.2 ± 0.6	1.2 ± 0.7	1.3 ± 0.7	1.4 ± 0.8	1.2 ± 0.6	1.3 ± 0.7

*Group A: Procaine + placebo; group B: dry needling + placebo; group C: double placebo control.

Table 2a Mean Visual Analogue Scale Ratings for Pain Intensity (P-int) in Groups A, B, and C at Three Experimental Sessions, 1 Week Apart (mm ± SD)

	Week 1				Week 2				Week 3			
Group*	Pretreatment	5 minutes after	1 hour after	24 hours after	Pretreatment	5 minutes after	1 hour after	24 hours after	Pretreatment	5 minutes after	1 hour after	24 hours after
A	39 ± 24	32 ± 20	33 ± 23	35 ± 32	34 ± 28	29 ± 26	29 ± 26	28 ± 27	28 ± 28	24 ± 27	19 ± 19	28 ± 32
B	37 ± 18	34 ± 21	30 ± 26	30 ± 26	37 ± 27	31 ± 26	32 ± 25	35 ± 25	32 ± 29	31 ± 30	29 ± 31	25 ± 25
C	34 ± 25	26 ± 24	28 ± 24	30 ± 26	30 ± 27	26 ± 25	20 ± 19	24 ± 19	17 ± 22	17 ± 20	16 ± 21	19 ± 20

*Group A: Procaine + placebo; group B: dry needling + placebo; group C: double placebo control.

Table 2b Mean Visual Analogue Scale Ratings for Pain Unpleasantness (P-unpl) in Groups A, B, and C at Three Experimental Sessions, 1 Week Apart (mm ± SD)

	Week 1				Week 2				Week 3			
Group*	Pretreatment	5 minutes after	1 hour after	24 hours after	Pretreatment	5 minutes after	1 hour after	24 hours after	Pretreatment	5 minutes after	1 hour after	24 hours after
A	49 ± 30	34 ± 24	41 ± 31	39 ± 36	33 ± 30	30 ± 24	31 ± 30	28 ± 31	29 ± 28	30 ± 24	22 ± 20	26 ± 31
B	52 ± 23	32 ± 19	32 ± 32	36 ± 31	40 ± 27	32 ± 20	39 ± 27	42 ± 27	33 ± 30	32 ± 22	31 ± 32	28 ± 24
C	39 ± 29	22 ± 24	28 ± 25	31 ± 27	37 ± 32	26 ± 28	23 ± 22	29 ± 26	19 ± 22	22 ± 24	19 ± 24	21 ± 24

*Group A: Procaine + placebo; group B: dry needling + placebo; group C: double placebo control.

Pain Measurements According to the Visual Analogue Scale

Pain Intensity. Pain intensity scores at key time points throughout the study are detailed in Table 2a. There were no statistical differences in P-int ratings over time ($P > .05$). The treatment modality did not affect the pretreatment P-int scores, nor were there any treatment-visit interactions. There was, however, a significant decrease in the P-int scores at the end of treatment for all groups ($P = .04$). There were no differences in the magnitude of the decrease between groups ($P > .05$). There were no group-by-time interactions.

Pain Unpleasantness. Scores for pain unpleasantness are detailed in Table 2b. Changes in the magnitude of the P-unpl scores were similar to those for P-int. The pretreatment P-unpl scores in the three groups decreased over time ($P = .03$).

Table 3 Mean Value of the Visual Stimulus for Groups A, B, and C Taken From VAS Ratings at Three Experimental Sessions, 1 Week Apart (mm ± SD)

Treatment group	Week 1	Week 2	Week 3
A: Procaine + placebo	38 ± 27	30 ± 26	33 ± 25
B: Dry needling + placebo	29 ± 15	29 ± 18	27 ± 21
C: Double placebo (control)	32 ± 20	29 ± 20	26 ± 18

There were no statistical differences between groups or over time.

However, the time difference between groups was not statistically significant ($P > .05$). The type of treatment did not affect the pretreatment P-unpl scores, nor were there any treatment-visit interactions. There was, however, a significant decrease in the P-unpl scores at the end of treatment for all groups ($P = .02$). There was no difference in the magnitude of the decrease between groups ($P > .05$). There were no group-by-time interactions.

Visual Stimulus

The mean rating of the visual (grey) stimulus (VAS) was within the range of approximately 25 to 35 mm throughout the study (Table 3). There were no between-group differences ($P = .60$), nor did the ratings change significantly over time.

Discussion

A key feature of this study was the incorporation of a control group, which allowed any changes in pain symptoms measured during the trial to be more reliably attributed to a particular treatment. A "placebo" control group was incorporated because of the potential for nonspecific factors to influence treatment outcome in addition to treatment-specific components.[7] Also, in order to minimize patient perception that actual and placebo treatments were different, a double-placebo approach involving simulated dry needling/LA (saline) injections was employed. Thus there were three experimental groups: procaine + simulated dry needling (treatment group A); dry needling + simulated LA (treatment group B); and double placebo (simulated LA and dry needling) (control group C).

Visual analogue scale ratings showed a reduction in the intensity and unpleasantness of myofascial pain over the period of the trial. The trend towards a reduction in pain symptoms was similar in all groups. The mean VAS ratings for pain intensity and unpleasantness in patients before treatment (approximately 35 to 40 mm) were similar to those reported in other studies of myofascial pain in the jaws (reviewed in Dao et al[18]).

Pain thresholds in masseter and temporalis muscles increased slightly in the three groups over the period of the trial. The reduction in muscle tenderness was of similar magnitude in the three groups and did not appear to be treatment specific. Mean PPTs in the masseter and temporalis muscles before treatment were comparable to those recorded previously under similar conditions.[2,9,19] There was no apparent regional variation in the PPT in either muscle, which concurred with previous findings at similar locations in the jaws.[9] The distribution of active TPs in the muscles corresponded relatively closely to the location of known acupuncture points in the masseter.[10,20]

There was no change in the mean VAS score for intensity of the visual stimulus throughout the trial, and there were no differences between experimental groups. These findings are in agreement with those of Dao et al[18] involving a similar population of myofascial pain patients, an argument for the validity of the pain ratings and PPT scores in this study.

An obvious interpretation of the uniform decrease in pain symptoms across experimental groups is the role of nonspecific, placebo-related factors and the absence of a true treatment effect. A concern often cited when simulated or "sham" acupuncture/dry needling treatment is performed in control groups is that the psychological impact is not equivalent to the true treatment.[6] It would appear from our findings that there was equivalent placebo power. However, the lack of difference between active and control groups cannot be attributed entirely to a placebo effect. Le Bars et al[12,21] have shown that a stimulus applied anywhere in the body, not necessarily at a painful site, may modify pain perception as a consequence of diffuse noxious inhibitory controls (DNICs). The use of simulated dry needling/saline injections at nonpainful sites in the face is likely to have produced an analgesic effect, as has been shown previously by Lewith and Machin.[22] Thus,

although simulated acupuncture/ dry needling is considered an acceptable placebo,[6,7] a specific treatment component is apparently inherent. This finding highlights one of the difficulties associated with conducting trials to assess the efficacy of acupuncture and related treatments.

The lack of a specific treatment effect must also be considered against the cyclical fluctuations in pain symptoms, which are part of the natural progression of the disease process in patients with myofascial pain.[4,18] In this study, a control group receiving no treatment was not incorporated because it was considered unethical (by the University Ethics Committee). Therefore, no direct comment could be made on any spontaneous improvement in pain symptoms, though it may have occurred in some instances.

A power calculation, based on data from a previous study by our group, was performed to determine the sample size in this study. The sample size was relatively small, and given the magnitude of the placebo effect inherent in most clinical trials, even when a double-blind approach is used, this was a potential cause for concern.[18,22–24] However, retrospectively, we used differences observed between the three groups at the end of treatment to calculate the sample size necessary to meet statistical criteria and found that 180 patients would be required using PPT data, 460 using P-int scores, and 700 using P-unpl scores. Thus, a clinical trial involving very large numbers of subjects would be necessary to discern differences between true and placebo treatments. The large sample sizes calculated were similar to those described by Dao et al[18] and suggest that the contribution of the active therapeutic component to the outcome is very small.

There was residual hyperalgesia in the masseter muscles irrespective of the treatment regimen. This is probably a result of the continued presence of active as well as "latent" TPs, which are generally symptomless unless palpated.[25] It is possible, though unlikely, that the residual muscle tenderness was a consequence of failure to locate the active TP. It is also possible that tissues bled when the needle was inserted, leading to aggravation of the TP, although such a possibility is less likely with an acupuncture needle than with a conventional hypodermic bevelled needle.[4] A standardized dry-needling procedure was also used, which may have led to overstimulation of some TPs and understimulation of others.[4]

The results of this study question the therapeutic value of dry needling and Procaine in the treatment of myofascial pain in the jaws. However, the curative value of all other treatments for myofascial pain in the orofacial region, including oral splints, do not appear to be any more effective.[18] Dry needling and Procaine should best be considered as adjunctive therapies. They may lessen the duration of myofascial pain in the jaws, which, in any event, usually spontaneously remits over time.

Acknowledgment

This study was supported by a grant from the Northern and Yorkshire Regional Health Authority (#94099926).

References

1. McNeill C (ed). Temporomandibular Disorders: Guidelines for Classification, Assessment and Management. Quintessence: Chicago, 1990:39–55.
2. Ohrbach R, Gale EN. Pressure pain thresholds, clinical assessment, and differential diagnosis: Reliabilty and validity in patients with myogenic pain. Pain 1989;39: 157–169.
3. Hu JW, Sessle BJ, Raboisson P, Dallel R, Woda A. Stimulation of craniofacial muscle afferents induces prolonged facilitatory effects in trigeminal nociceptive brain-stem neurones. Pain 1992;48:53–60.
4. Baldry PE. Acupuncture, trigger points and musculoskeletal pain. Edinburgh: Churchill Livingstone, 1993.
5. Jaeger B, Reeves JL. Quantification of changes in myofascial trigger point sensitivity with the pressure algometer following passive stretch. Pain 1986;27:203–210.
6. Vincent CA, Lewith G. Placebo control for acupuncture studies. J R Soc Med 1995;88:199–202.
7. Vincent CA, Richardson PH. The evaluation of therapeutic acupuncture: Concepts and methods. Pain 1986;24:1–13.
8. Vincent CA. Credibility assessment in trials of acupuncture. Comp Med Res 1990;4:8–11.
9. McMillan AS, Blasberg B. Pain-pressure threshold in painful jaw muscles following trigger point injection. J Orofacial Pain 1994;8:384–390.
10. Melzack R, Stillwell DM, Fox EJ. Trigger points and acupuncture points for pain: Correlations and implications. Pain 1977;3:3–23.
11. Yagiela JA. Local anesthetics. In: EA Neidle, DC Kroeger, JA Yagiela (eds). Pharmacology and Dental Therapeutics for Dentistry. St. Louis: Mosby, 1985: 248–267.
12. Le Bars D, Willer JC, De Broucker T. Morphine blocks descending pain inhibitory controls in humans. Pain 1992; 48:13–20.
13. Woolf CJ, Wiesenfeld-Hallin Z. The systematic administration of local anaesthetics produces a selective depression of C-afferent fibre evoked activity in the spinal cord. Pain 1985;23:361–374.
14. McMillan AS, Lawson E. Effect of tooth clenching and jaw opening on pain-pressure thresholds in the human jaw muscles. J Orofacial Pain 1994;8:250–257.
15. Bushnell MC, Duncan GH, Dubner R, Jones RL, Maixner W. Attentional influences on noxious and innocuous cutaneous heat detection in humans and monkeys. J Neurosci 1985;5:1103–1110.
16. Ohrbach R, Gale EN. Pressure pain thresholds in normal muscles: Reliability, measurement effects, and topographic differences. Pain 1989;37:257–263.

17. Feine JS, Chapman CE, Lund JP, Duncan G. The perception of painful and nonpainful stimuli during voluntary motor activity in man. Somatosens Mot Res 1990;7: 113–124.
18. Dao TTT, Lavigne GJ, Charbonneau A, Feine JS, Lund JP. The efficacy of oral splints in the treatment of myofascial pain of the jaw muscles: A controlled clinical trial. Pain 1994;56:85–94.
19. Reeves JL, Jaeger B, Graff-Radford SB. Reliability of the pressure algometer as measure of myofascial trigger point sensitivity. Pain 1986;24:313–321.
20. List T, Helkimo MH. Tenderness and acupuncture points in the masseter muscle: A correlation study. J Craniomandib Disord Facial Oral Pain 1988;2:133–136.
21. Le Bars D, Willer JC, de Broucker T, Villanueva L. Neurophysiological mechanisms involved in the pain-relieving effects of counterirritation and related techniques including acupuncture. In: Pomeranz B, Stux G (eds). Scientific Basis of Acupuncture. New York: Springer-Verlag, 1989:79–112.
22. Lewith GT, Machin D. On the evaluation of the clinical effects of acupuncture. Pain 1983;16:11–127.
23. Green CS, Laskin DM. Meprobamate therapy for the myofascial pain-dysfunction (MPD) syndrome: A double-blind evaluation. J Am Dent Assoc 1971;82:587–590.
24. Spilker B. Guide to Clinical Trials. New York: Raven, 1991.
25. Travell JG, Simons DG. Myofacial Pain and Dysfunction, the Trigger Point Manual: Vol 1, The Upper Extremities. Baltimore: Williams and Wilkins, 1983.

Resumen

La Eficacia del Uso de las Agujas en Seco y de la Procaína en el Tratamiento del Dolor Miofascial en los Músculos Mandibulares

Los puntos de estímulo dolorosos en los pacientes que sufren de dolor miofascial, son tratados frecuentemente utilizando agujas en seco e inyecciones con anestésicos locales. Sin embargo, el efecto terapéutico de estos tratamientos ha sido cuantificado pobremente, y tampoco se ha entendido el mecanismo fundamental del efecto. Se organizó un estudio clínico al azar, doble ciego, y doble placebo, utilizando un algómetro de presión para medir los umbrales del dolor a la presión, en los músculos masetero y temporal de 30 personas (23-53 años de edad) que padecían de dolor en las mandíbulas, antes y después de una serie de tratamientos con agujas en seco, inyecciones con anestésicos locales, y tratamientos simulados con agujas en seco y anestésicos locales (tratamiento del grupo A: Procaína + uso simulado de agujas en seco; tratamiento del grupo B: uso de agujas en seco + anestésico local simulado; grupo de control C: uso simulado de anestésico local + uso simulado de agujas en seco). Las personas evaluaron la intensidad del dolor y la molestia utilizando escalas análogas visuales. La información fue evaluada utilizando el análisis de varianza. Los umbrales del dolor a la presión aumentaron ligeramente después del tratamiento, sin importar la modalidad de tratamiento. Las puntuaciones adjudicadas a la intensidad del dolor y a la molestia disminuyeron significativamente al final del tratamiento en todos los grupos. No se observaron diferencias estadísticamente significativas entre los grupos en cuanto a las puntuaciones adjudicadas a los umbrales del dolor a la presión, y a las de la escala análoga visual al final del tratamiento. Los hallazgos indican que la mejoría general en relación a los síntomas de dolor fue el resultado de factores no específicos, relacionados al placebo, en lugar de un efecto real del tratamiento. Por lo tanto, el valor terapéutico del uso de agujas en seco y de la Procaína en el manejo del dolor miofascial en los músculos mandibulares es cuestionable.

Zusammenfassung

Die Wirksamkeit von Stechen ohne Injektion und Procain in der Behandlung von myofazialen Schmerzen in der Kaumuskulatur

Schmerzhafte Triggerpunkte werden bei Patienten mit myofazialen Schmerzen oft mittels Stechen ohne Injektion und Injektionen von Lokalanästhetika behandelt. Dagegen ist der therapeutische Effekt dieser Behandlung nur schlecht belegt, und der zugrundeliegende Mechanismus ist kaum verstanden. In einem zufälligen, doppel-blinden, doppel-Placebo klinischen Untersuch wurde ein Druck-Algometer verwendet um die Druckschmerzschwelle in den Mm masseteri und temporales bei 30 Personen im Alter von 23 bis 53 Jahren mit myofazialen Schmerzen in der Kaumuskulatur zu messen, dies vor und nach einer Serie von Stechen ohne Injektion, Injektionen von Lokalanästhetika und vorgetäuschtem Stechen und Lokalanästhetika-Injektionen (Therapiegruppe A: Procain + vorgetäuschtem Stechen ohne Injektion; Therapiegruppe B: Stechen ohne Injektion und vorgetäuschte Lokalanästhesie, Kontrollgruppe C: vorgetäuschte Lokalanästhesie und vorgetäuschtes Stechen ohne Injektion). Die Patienten stuften die Schmerzintensität und Unannehmlichkeit mittels Visual-Analog-Scale ein, und die Daten wurden mit Varianzanalyse ausgewertet. Die Druckschmerzschwelle erhöhte sich leicht nach Behandlung, unabhängig von der Behandlungsart. Die Schmerzintensitäts- und Unannehmlickeitswerte sanken signifikant in allen Gruppen am Ende der Therapie. Es gab keine statistisch signifikanten Unterschiede zwischen den Gruppen für die Druckschmerzschwelle und die Visual-Analog-Scale-Werte zu Behandlungsende. Die Befunde legen nahe, dass die allgemeine Verbesserung in Bezug auf die Schmerzsymptome eher das Resultat von nichtspezifischen, placeboverbundenen Faktoren als von echten Therapieauswirkungen sind. Somit ist der therapeutische Wert von Stechen ohne Injektion und Procain für die Behandlung von myofazialen Schmerzen in der Kaumuskulatur fragwürdig.