Assessment of Proprioceptive Allodynia After Tooth-Clenching Exercises

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Aims: *To (A) evaluate test-retest reliability of vibrotactile sensitivity in the masseter muscle and (B) test if* (1) *the vibration threshold is decreased after experimental tooth clenching,* (2) *intense vibrations exacerbate pain after tooth clenching,* (3) *pain and fatigue are increased after tooth clenching, and* (4) *pressure pain thresholds are decreased after tooth clenching.* ***Methods:*** *In part A, 25 healthy female volunteers (mean age: 42 ± 12 years) participated, and 16 healthy females (mean age 32 ± 10 years) participated in three 60-minute sessions, each with 24- and 48-hour follow-ups in part B. Participants were randomly assigned tooth-clenching exercises with clenching levels of 10%, 20%, or 40% of maximal voluntary clenching. A Vibrameter applied to the right masseter muscle measured perceived intensity of vibration and perceived discomfort, which were assessed on 0–50–100 numeric rating scales. An electronic algometer measured pressure pain threshold (PPT). Two 0- to 100-mm visual analog scales measured pain intensity (VAS_{pain}) and fatigue ($VAS_{fatigue}$). Measurements were made on the right masseter muscle. Interclass correlation coefficient (ICC) was used to calculate test-retest reliability of VT measurements. Outcome variables were tested with two-way ANOVAs for repeated measures and Dunnett's post-hoc test.* ***Results:*** *Moderate long-term (ICC 0.59) and good short-term (ICC 0.92) reliability was found for VT on the masseter muscle. Clenching level had no main effect on perceived intensity of vibration; time effects (*$P < .05$*) were only observed at 40 minutes (Dunnett's test:* $P < .01$*). Clenching level and time had no effect on perceived discomfort. Only time effects were significant for PPT (*$P < .01$*), with reductions at 50 and 60 minutes compared to baseline (Dunnett's test:* $P < .05$*). Clenching level and time had main effects for VAS_{pain} and $VAS_{fatigue}$ (*$P < .001$*).* ***Conclusion:*** *Experimental tooth clenching appears to evoke moderate levels of pain and fatigue and short-lasting hyperalgesia to mechanical stimulation, but not proprioceptive allodynia. The absence of proprioceptive allodynia does not necessarily exclude delayed onset muscle soreness (DOMS) but warrants further studies on the clinical manifestations of DOMS in jaw muscles.* J OROFAC PAIN 2012;26:39–48

Key words: experimental pain, muscle pain, pain measurement, proprioceptive allodynia, tooth clenching

Myofascial temporomandibular disorders (TMD)—persistent muscle pain in the orofacial region—has a prevalence of 10% in the general population and occurs more frequently in females than males.[1] Symptoms manifested in this condition are pain and soreness in the masticatory muscles, limited jaw function, and restricted mouth opening.[2,3] Tooth clenching has been reported

to be an etiologic factor in persistent muscle pain in the orofacial region[4] and is a feature of bruxism, which is a parafunctional activity involving clenching, gnashing, bracing, and grinding that can occur during either sleep or waking hours.[5] Lund[6] suggested that bruxism is a contributing factor to delayed onset muscle soreness (DOMS). Thus, DOMS may be involved in persistent muscle pain in the orofacial region.

The phenomenon of DOMS was first described in 1902 by Hough and has since been a focus for research.[7] Despite this, no operationalized criteria for DOMS have been developed or tested; however, it is well known that intense muscle exercise can provoke pain on movement, stiffness, fatigue, and soreness the following day, that is, DOMS.[7–12] Other features of DOMS that are considered to be secondary to fatigue and pain are allodynia, hyperalgesia, and edema[13–17] and the intriguing observation that vibration at 80 Hz significantly increases perceived pain in a muscle with DOMS.[12] However, it has not been established how strong the association between vibratory stimulus and increased pain in a muscle with DOMS is. Several studies have reported that eccentric muscle exercise is more associated with DOMS than other types of muscle exercise.[18–20] However, intense isometric muscle exercise may also be able to provoke DOMS.[21] DOMS normally evolves 8 to 10 hours after exercise, reaching a peak after 48 hours.[22,23] Armstrong et al[24] suggested that eccentric muscle exercises damage and break down muscle fibers. Local muscle inflammation may occur, causing sensitization of primary afferent nerve fibers.[25,26] Weerakkody et al[27] suggested that large-diameter mechanoreceptive afferents contribute to the development of DOMS, with mechanisms similar to those of secondary hyperalgesia and allodynia involved in the generation of DOMS. Vibration creates an illusion of movement[28] and is considered an effective stimulus of mechanoreceptive afferents. As noted above, 80-Hz vibrations exacerbate pain in a DOMS muscle.[12] A nonpainful stimulus that elicits pain by activating proprioceptive afferents has been termed proprioceptive allodynia.[27,29] Studies also found that TMD patients exhibit an altered sense of vibrations.[30,31] Thus, according to the literature, it seems that DOMS may be associated with proprioceptive allodynia, hyperalgesia, pain intensity, and fatigue profiles.

To gain a better understanding of clinical pain, pain models that mimic pain conditions are essential. Several human experimental pain models for investigating jaw muscle pain after eccentric or concentric exercise have been developed.[32–38] Concentric contraction can be dynamic or static; in jaw-closing muscles, such contractions represent a pain model that resembles tooth clenching and can cause intense muscle pain in the jaws.[33–38] Concentric contraction without sufficient relaxation is reported to cause pain, perhaps by the same mechanisms observed in ischemic pain.[39,40] Both eccentric muscle exercise and intense isometric exercise can provoke DOMS.[21] Most jaw motor functions consist of either isometric or isotonic contractions, so it is interesting to explore whether experimental tooth clenching provokes DOMS.[41] So far, experimental tooth-clenching models have not been used to investigate proprioceptive allodynia in jaw muscles. Thus, the aims of this study were to (A) evaluate test-retest reliability of vibrotactile sensitivity in the masseter muscle, and (B) test if *(1)* the vibration threshold is decreased after experimental tooth clenching, *(2)* intense vibrations exacerbate pain after tooth clenching, *(3)* pain and fatigue are increased after tooth clenching, and *(4)* pressure pain thresholds are decreased after tooth clenching.

Materials and Methods

Participants

Twenty-five healthy female volunteers (mean age: 42 ± 12 years) participated in part A, and 16 healthy female volunteers (mean age 32 ± 10 years) in part B. Three subjects participated in both parts. The study sample was based on similar experimental studies with a crossover design.[42,43] All subjects were recruited from the staff at Malmö University. All participants were screened per the Research Diagnostic Criteria (RDC) for TMD.[3]

Exclusion criteria were *(1)* younger than 18 years of age; *(2)* male gender; *(3)* TMD or other orofacial pain complaints; *(4)* systemic inflammatory connective tissue diseases (eg, rheumatoid arthritis); *(5)* whiplash-associated disorder; *(6)* fibromyalgia; *(7)* neuropathic pain; *(8)* analgesics, eg, paracetamol, nonsteroidal anti-inflammatory drugs, salicylate drugs, and opioids, or other medication that would influence pain perception, eg, antidepressants, and antiepileptic drugs; *(9)* pregnancy; *(10)* severe skeletal malocclusions; and *(11)* extensive restorations, such as fixed partial dentures.

The Declaration of Helsinki guidelines were followed, and the Regional Ethics Review Board at Lund University approved the methods and selection of participants (2009/264). Participants signed informed-consent forms before entering the study, and they were informed that they could withdraw at any time with no consequences. Subjects received

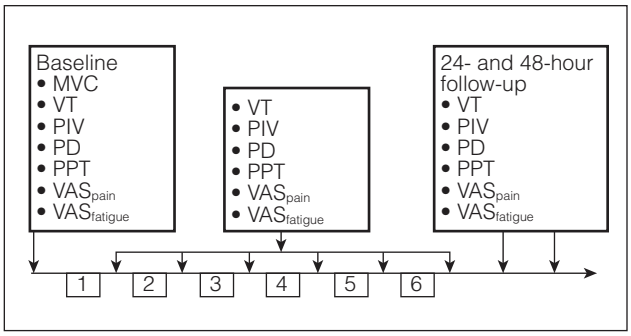

Fig 1 Schematic illustrations of the experimental protocol for one 60-minute session with 24- and 48-hour follow-ups. Six bouts of tooth clenching (1 to 6) over 1 hour. Each bout lasted 5 minutes. The level of contraction was randomized between sessions (10%, 20%, or 40%). PIV = perceived intensity of vibration; PD = perceived discomfort.

no financial compensation for participation. Part A comprised two sessions, and part B three 60-minute sessions, each with 24- and 48-hour follow-ups. Each follow-up lasted 5 minutes. Initially, 17 subjects were included in part B, but 1 subject dropped out due to medical reasons. The remaining 16 subjects took part in all three sessions and six follow-ups.

Study Design

Part A comprised test-retest measurements of the vibration threshold (VT) at baseline, after 10 minutes, and after 1 week; no tooth-clenching exercises were conducted. Part B was a randomized crossover trial comprising three 60-minute sessions with a 24- and 48-hour follow-up after each session. Time intervals between the 60-minute sessions were a minimum of 1 week to avoid carryover effects. Tooth-clenching exercises were done in the 60-minute sessions, with follow-ups at 24 and 48 hours. Maximal voluntary clenching (MVC) was assessed in each participant at the beginning of each 60-minute session.

To assess MVC, a bite-force transducer (Aalborg University) was placed between the first or second molars on the right side, and subjects were encouraged to clench as intensely as possible for 2 to 3 seconds. Three MVC measurements were made at the beginning of each 60-minute session, and a mean was calculated. The transducer displayed the MVC being measured. The mean MVC was used to define clenching levels of 10%, 20%, and 40% of MVC. Participants were instructed to observe the display to ensure that clenching force was constant during the longer sessions. The operator continually encouraged the subjects to maintain clenching force.

In each 60-minute session, subjects were randomly assigned a clenching level of 10%, 20%, or 40% of MVC. The subjects underwent six 5-minute bouts of tooth clenching with intervals of about 5 minutes between bouts. Perceived intensity of pain (VAS_{pain}) and fatigue ($VAS_{fatigue}$) were each rated by the subjects on a visual analog scale (VAS). These values, plus VT, perceived intensity of vibration, perceived discomfort, and pressure pain threshold (PPT), were measured at baseline, between bouts (every 10 minutes), and at the 24- and 48-hour follow-ups. Subjects drew the pain distribution on a two-dimensional, anatomical representation of the head at baseline after 60 minutes and at the 24- and 48-hour follow-ups. Subjects were instructed to keep their teeth slightly apart during measurements to avoid contraction of the jaw-closing muscles.

During the experiment, the participant sat upright in a dental chair with a support at the back of the head. The same examiner conducted all measurements, which were made on the most prominent, central part of the right masseter muscle. Figure 1 is a schematic diagram for one 60-minute session and its follow-ups.

Measures

The Vibrameter (SOMEDIC Sales) delivered 100-Hz vibratory stimuli to the right masseter muscle with a constant application pressure of 650 g. The stimulating probe was a plastic cylinder with a diameter of 13 mm. Ascending vibratory stimuli were used to make three assessments of the VT, defined as the amplitude (μm) at which the participant first perceived vibration. Descending vibratory stimuli were used to make three assessments of the vibration disappearance threshold, ie, the amplitude at which vibration was no longer perceived. Means of the three measurements determined the vibration perception and disappearance thresholds. VT was then calculated as the mean of these two thresholds.[44]

Perceived intensity of vibration and perceived discomfort were assessed with 15-second fixed vibratory stimuli (Vibrameter, 100 Hz, 399.99-μm amplitude) applied to the right masseter muscle with a constant application pressure of 650 g. Subjects were instructed to rate perceived intensity of vibration and perceived discomfort on 0–50–100 numeric rating scales (perceived intensity of vibration: 0 = no

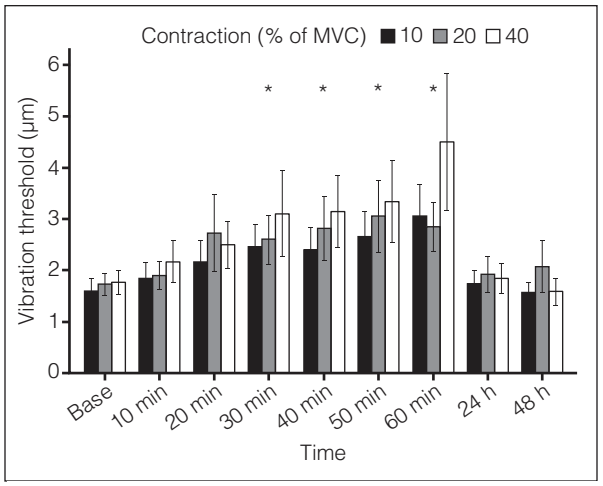

Fig 2 VT on the right masseter muscle at baseline (base), and in response to clenching tasks at 10%, 20%, and 40% of MVC. Clenching tasks were done in six 5-minute bouts, at intervals of 5 minutes, during 1 hour. The VT was measured in the 5-minute resting period after clenching: mean ± SEM and *P* values. *Significant difference from baseline values (Dunnett's test: $P < .05$).

sensation, 50 = pain threshold, 100 = most imaginable pain; perceived discomfort: 0 = no sensation, 50 = discomfort, 100 = most imaginable discomfort).

An Algometer (SOMEDIC Sales) applied to the right masseter muscle assessed PPT, defined as the amount of pressure needed to produce a sensation of pain. Upon reaching the PPT, subjects pressed a button to stop stimulation. A constant pressure of 30 kPa/s was applied with a 1.0-cm^2 probe. The mean of three measurements, made at 60-second intervals, was calculated.[45] The PPT, when measured in this way, was found to have acceptable reliability.[46]

Two 100-mm VASs were used to determine VAS_{pain} (anchor definitions "no pain" and "most imaginable pain") and $VAS_{fatigue}$ (anchor definitions "no fatigue" and "most imaginable fatigue") when the jaw muscles were in a relaxed state.

Statistical Analyses

In part A, means and standard deviations (SDs) were calculated for VT, and the interclass correlation coefficient (ICC) calculated test-retest reliability of the VT measurements. An ICC > 0.75 was considered good reliability.[47] In part B, means and SDs were calculated for the outcome variables VT, perceived intensity of vibration, perceived discomfort, PPT, VAS_{pain}, and $VAS_{fatigue}$ at the various time points and clenching levels (10%, 20%, and 40% of MVC). Two factor-dependent analyses of variance (ANOVAs) tested for significant alterations in the outcome variables' mean values at different levels of contraction and time, and for interaction effects between time and clenching level. Dunnett's post-hoc test identified at what point in time and/or what level of contraction the difference was significant.

Sample size was based on 5% risk of type I and 20% risk of type II errors, an estimated intraindividual variation of 20%, and the possibility to detect a minimal relevant difference of 20%. Thus, 16 subjects were included.

Statistical tests were performed two-tailed and at the 5% significance level. The Statistical Package for the Social Sciences for Windows, version 17 (SPSS, IBM) was used for all statistical calculations. Unless stated otherwise, *P* values were determined with ANOVA.

Results

In part A, the ICC for test-retest reliability of VT measurements on the right masseter muscle between baseline and 10 minutes was good (0.92; 95% confidence interval [CI]: 0.81–0.96) and between baseline and 7 days, moderate (0.59; 95% CI: 0.08–0.82). No significant time effects were seen for VT between baseline, 10 minutes, and 7 days ($F = 0.141$; $P = .869$).

In part B, there were no significant between-session differences in MVC measurements at baseline ($F = 0.523$; $P = .598$).

Clenching level had no significant main effects on VT ($F = 1.79$; $P = .184$), but main effects of time were observed: VT increased significantly ($F = 7.23$; $P < .001$) compared with baseline at 30, 40, 50, and 60 minutes (Dunnett's test: $P < .05$). No significant changes in VT were observed at the 24- and 48-hour follow-ups (Fig 2).

Clenching level had no significant main effects on perceived intensity of vibration ($F = 0.472$; $P = .628$), but there were significant time effects ($F = 2.54$; $P = .014$) with significant increases at 40 minutes compared with baseline (Dunnett's test: $P < .05$, Fig 3). There were no significant effects of clenching level or time for perceived discomfort ($F = 0.66$; $P = .524$; $F = 0.289$; $P = .969$, respectively, Fig 3).

Mean PPT did not change significantly with contraction level ($F = 2.69$; $P = .084$), but a significant time effect ($F = 3.17$; $P = .003$) with decreases in mean PPT was observed at 50 minutes and 60 minutes compared with baseline (Dunnett's test: $P < .05$). PPT was not significantly changed at the follow-ups (Fig 4).

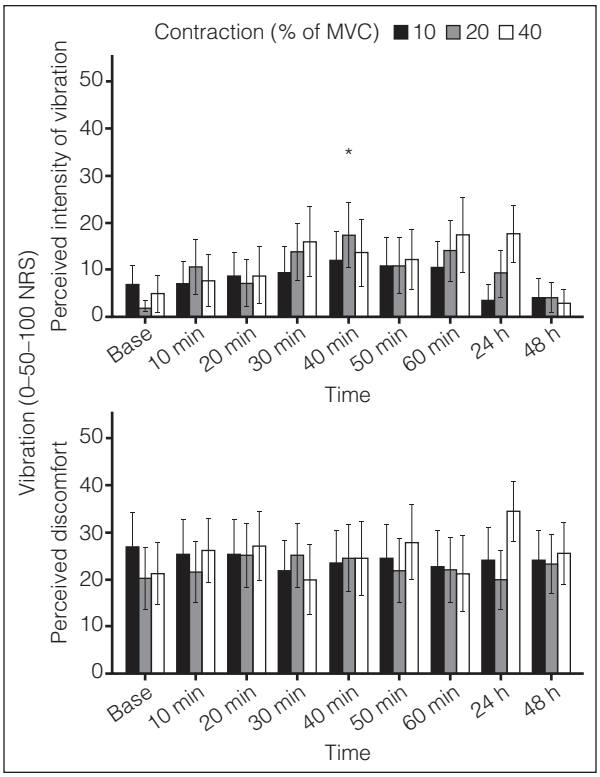

Fig 3 Perceived intensity of vibration and perceived discomfort measured on the right masseter muscle at baseline (base) and in response to clenching tasks at 10%, 20%, and 40% of MVC. Clenching tasks were done in six 5-minute bouts, at intervals of 5 minutes, during 1 hour. Perceived intensity of vibration and perceived discomfort were measured in the 5-minute resting period after each clenching bout: mean ± SEM and *P* values. *Significant difference from baseline values (Dunnett's test: $P < .05$). NRS = numerical rating scale.

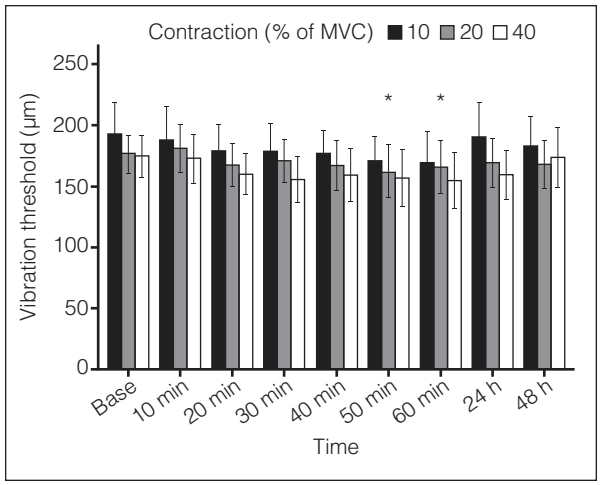

Fig 4 PPT on the right masseter muscle at baseline (base) and in response to clenching tasks at 10%, 20%, and 40% of MVC. Clenching tasks were done in six 5-minute bouts, at intervals of 5 minutes, during 1 hour. The PPT was measured in the 5-minute resting period after clenching: mean ± SEM and *P* values. *Significant difference from baseline values (Dunnett's test: $P < .05$).

There were significant effects of clenching level and time on mean VAS_{pain} ($F = 19.4$; $P < .001$; $F = 12.4$; $P < .001$, respectively) and mean $VAS_{fatigue}$ ($F = 18.2$; $P < .001$; $F = 41.8$; $P < .001$, respectively). Thus, significant increases from baseline occurred for VAS_{pain} and $VAS_{fatigue}$ at all time points between 10 and 60 minutes and at the 24-hour follow-up (Dunnett's test: $P < .05$). Clenching level at 40% of MVC increased VAS_{pain} (mean 31.4 ± 31.6) and $VAS_{fatigue}$ (mean 49.9 ± 33.2) significantly (Dunnett's test: $P < .05$) compared to 10% of MVC (mean VAS_{pain} 15.1 ± 23.1; mean $VAS_{fatigue}$ 34.1 ± 31.2). A significant interaction between contraction level and time was only found for VAS_{pain} ($P < .001$, Fig 5).

Discussion

The main findings of the present study were that VT has a moderate long-term and a good short-term reliability, and that tooth clenching *(1)* causes moderate levels of pain and fatigue in a relaxed state, *(2)* increases sensitivity to suprathreshold mechanical stimuli (ie, decrease in PPT), and *(3)* has no major effects on vibrotactile function, ie, robust indications of proprioceptive allodynia could not be detected.

Clarkson et al found that both eccentric muscle exercise and intense isometric concentric exercise can provoke DOMS in limb muscles.[21] A link between bruxism and DOMS has also been suggested.[6] Studies have reported that experimental tooth grinding for 30 and 45 minutes results in muscle pain that persists for several days.[48,49] Tooth clenching is characterized by isometric concentric muscle exercises, either static or dynamic. Because tooth clenching may be a contributing factor to the etiology of myofascial TMD,[4] this study used a human experimental pain model that induces muscle pain through tooth clenching.

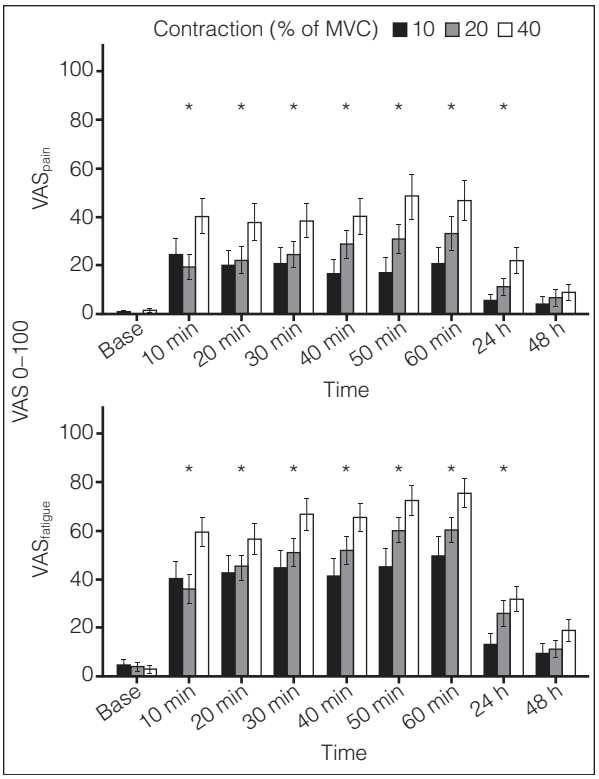

Fig 5 VAS$_{pain}$ and VAS$_{fatigue}$ in the right masseter muscle at baseline (base) and in response to clenching tasks at 10%, 20%, and 40% of MVC. Clenching tasks were done in six 5-minute bouts, at intervals of 5 minutes, during 1 hour. VAS$_{pain}$ and VAS$_{fatigue}$ were measured in the 5-minute resting period after clenching: mean ± SEM and *P* values. *Significant difference from baseline values (Dunnett's test: $P < .05$).

Proprioceptive allodynia has been defined as a nonpainful stimulus that elicits pain by activating proprioceptive afferents, a phenomenon that can be found in muscles with DOMS.[27,29] However, the relationship between vibrotactile stimulus and DOMS has not been fully established. Thus, if an isometric exercise provokes DOMS, it cannot be excluded that proprioceptive allodynia could be an indicator of DOMS, together with the previously mentioned features.[8–17]

The mechanism that underlies DOMS is not yet fully understood, but one possible explanation is that eccentric exercise causes inflammatory responses in the muscles[50,51]; nociceptive sensitization and increased excitability in presynaptic inhibitory interneurons might then occur. Following mechanoreceptive input, activated inhibitory interneurons could generate a dorsal root reflex in the nociceptive afferents, with a subsequent perception of pain. Weerakkody et al[27] suggested another mechanism by which the primary endings of muscle spindles could be involved in the generation of DOMS. Wide dynamic range (WDR) neurons are localized in the dorsal horn of the spinal cord and in the trigeminal brainstem nuclei. WDR neurons are able to respond to input from nociceptors *and* non-nociceptors, thus allowing proprioceptive input entrance to the nociceptive pathway.[12]

Generation of DOMS has also been discussed in terms of the gate control theory of pain. It has been suggested that vibration reduces pressure-induced pain due to increased excitability in presynaptic inhibitory interneurons between large-diameter and nociceptive afferents.[12] Nociceptive input is thus inhibited in the normal state—the gate control theory of pain[52]—but in DOMS the opposite may occur, with pain becoming more intense due to vibrations.[12]

In this study, time and contraction level (%MVC) had no main effects on perceived intensity of vibration and perceived discomfort, except for a significant increase in perceived intensity of vibration at 40 minutes. This increase, however, did not exceed the pain threshold, indicating that tooth clenching is not directly related to proprioceptive allodynia, ie, intense vibrations did not provoke pain. Lack of eccentric muscle exercise in the model used could explain these results. It has been demonstrated that vibratory stimulation applied before eccentric exercise might prevent DOMS.[53] In the present study, vibratory stimulation was applied every 10 minutes to measure perceived intensity of vibration and perceived discomfort, and this could potentially have biased the results, thus preventing DOMS with its previously described features such as proprioceptive allodynia. Türker et al[54] demonstrated that intense eccentric contractions in the jaw muscles provoked DOMS.[12,21] Thus, DOMS is mainly associated with eccentric muscle exercise,[18] and microinjuries occurring after eccentric exercise are greater and more severe than after other types of muscle exercise.[19] Another explanation for the absence of proprioceptive allodynia might be that the microinjuries due to concentric tooth-clenching exercise were not severe enough to induce sufficient intramuscular inflammation to generate DOMS. This also suggests that the consequences of bruxism depend on the specific type of jaw muscle contraction being performed and that better discrimination and classification of bruxism subtypes are needed.[55]

Hollins et al[30,31] found higher VTs in TMD pain patients than healthy controls. The present results suggest a significant increase in VT over time that is unrelated to clenching level. The most likely interpretation of these observations is that vibrotactile adaptation impairs the sense of vibration.

Another study[56] demonstrated that motor activity alters the electrical detection threshold and decreased the ability to discriminate thermal cutaneous input. Hollins et al[30] discussed whether the impaired vibration sense in TMD pain patients is an effect of increased muscle tension due to muscular hyperactivity. They concluded it was not since an unlikely amount of physiologic vibration would have been required to induce vibrotactile adaptation. Supporting this, the present results suggest that clenching level is unlikely to account for the significant increase in VT since no significant VT differences occurred between various levels of clenching. Gallasch and Kenner[57] showed that physiologic vibrations increase with higher levels of muscular contraction. In each session of the present study, tooth-clenching force was fixed, which implies fixed muscle tonus amplitude; VT would most likely be unaffected.

The study by Okayasu et al[58] investigated the effect of tooth clenching on orofacial tactile detection thresholds in healthy participants. Tactile detection thresholds were significantly higher after the clenching exercise. The same study confirmed these findings in a repetition of the trial without tooth clenching. It was concluded that the modulated tactile detection threshold was a result of habituation. Other research groups have shown that vibratory stimulation can desensitize cutaneous mechanoreceptive afferents, causing higher VTs.[59–61] This agrees with the present results, which indicate that increased VTs are due to an adaptation effect.

The present study also revealed significant reductions in PPT over time. One likely interpretation of this is that tooth clenching alone is responsible.[46] List et al[46] demonstrated acceptable reliability and validity for the algometer and, among others,[62–65] showed that repeated algometer measurements in healthy participants with no intervention did not alter the PPT. Farella et al[43] observed significant time effects for PPT in healthy participants after tooth clenching—which agrees with the present results—and a reduced PPT that persisted 1 day after the tooth-clenching exercise—which was not observed in the current study. They also found that low-intensity tooth clenching (7.5% and 10% of MVC), but not higher levels (15%, 25%, and 40% of MVC), was related to the reduction in PPT.[43] In contrast, no significant effects of contraction level on PPT, only significant time effects, were observed in the present study. The use of different experimental tooth-clenching models might explain why the two sets of results diverge. The pain model used here comprised six bouts of clenching during 1 hour, each bout lasting 5 minutes, while Farella et al[43] used a model with clenching levels of 7.5%, 10%, 15%, 25%, and 40% of MVC and instructed their study participants to clench their teeth at these contraction levels until exhaustion.

The experimental tooth-clenching model produced pain and fatigue, which agrees with the studies of others.[38,66–69] Pain and fatigue were observed up to 24 hours postexercise. Normally, DOMS peaks after 48 hours,[22,23] which was not corroborated by the present results since pain and fatigue were not observed at 48 hours. It does not seem that experimental tooth-clenching is directly related to DOMS as traditionally described for limb muscles, since the current results do not follow the time course characteristics of DOMS as previously described. A possible explanation for these results could be a change in the intramuscular metabolism. It has been demonstrated that isometric contractions lead to a significant pH-increase in the masseter muscle[70] and a change of intracellular levels of Ca++. These alterations are considered to be pain-related factors.[71] Furthermore, it cannot be excluded that the perceived pain is caused by a peripheral sensitization caused by an ischemia-induced release of algesic substances such as serotonin, glutamate, bradykanin, and PGE_2, which could be responsible for the development of pain.[72–77] Low-intensity muscle exercise might not affect the release of inflammatory substances to the same extent as clenching at 40% of MVC. This might explain the significant contraction effects for pain and fatigue that were observed when clenching at 40% compared to clenching at 10%. Intramuscular biochemical events due to tooth clenching, however, need to be further investigated.

The overall findings of the present study indicate that the type of tooth clenching used in the study is not directly related to DOMS since *(1)* pain was perceived not only in a resting state after tooth-clenching exercises but also at follow-ups, *(2)* no hyperalgesia was seen at follow-ups, and *(3)* proprioceptive allodynia was not observed. It cannot be ruled out that other types (intensity, duration, force directions) of tooth clenching could evoke more robust characteristics of DOMS.

Some methodologic issues of the current study that should be addressed are the exclusion of males and the use of the Vibrameter to assess VT. Females only were included because chronic muscle pain in the orofacial region is more frequently reported in females than males and gender differences would bias the results. Another limitation is the lack of control for sex hormones since pain intensity varies during the menstrual cycle.[78] This might have influenced the results. Most likely, the menstrual phase of the participants differed during the sessions. However, this aspect probably has no major

importance for the results since it has been shown that the intraindividual variability in pain response is greater than the influence of estrogen.[78,79] Hence, this aspect should only have a very limited effect on the results. The Vibrameter was used to assess vibrotactile sensitivity before and after experimental tooth clenching. This instrument had not previously been used in the orofacial region. Instead, the Rydel tuning fork is commonly used. The present results suggest acceptable reliability for VT on the masseter muscle; however, validity has not yet been assessed. Also, it is uncertain whether the VT or hearing ability was tested. Some subjects remarked that it was difficult to assess whether they were feeling or hearing the vibrations. Possibly, the vibrations were transmitted through the bone to the ear, which might have influenced findings.

The strengths of this study are that different levels of clenching were used and that there were 24- and 48-hour follow-ups after each 60-minute session to assess DOMS. Other strengths are the introduction of the Vibrameter in orofacial pain research, which might be an alternative to the Rydel-Seiffer graded tuning fork. The validity of the Vibrameter is unknown and must be determined before the Vibrameter can be used as a valid instrument in orofacial pain research.

To the authors' knowledge, this is the first study to assess proprioceptive allodynia after experimental tooth clenching. Further research would improve the understanding of the relation between bruxism and the clinical manifestations of DOMS.

Conclusions

This study found moderate long-term reliability and good short-term reliability for the vibrotactile sensitivity in the masseter muscle. This study also found that tooth clenching at various contraction levels is not directly related to DOMS but to *(1)* development of moderate levels of pain and fatigue and *(2)* short-lasting reductions in the PPT in the masseter muscle. Proprioceptive allodynia did not appear to be a prominent feature of the type of tooth clenching used in the present investigation, but further studies on the clinical manifestations of DOMS in jaw muscles are needed.

Acknowledgments

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References

1. Drangsholt M. Temporomandibular disorder pain. In: Crombie IK, Croft PR, Linton SJ, LeResche L, Von Korff M (eds). Epidemiology of Pain. Seattle: IASP, 1999:203–233.
2. Dworkin SF, Huggins KH, LeResche L, et al. Epidemiology of signs and symptoms in temporomandibular disorders: Clinical signs in cases and controls. J Am Dent Assoc 1990;120:273–281.
3. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: Review, criteria, examinations and specifications, critique. J Craniomandib Disord 1992;6:301–355.
4. Velly AM, Gornitsky M, Philippe P. Contributing factors to chronic myofascial pain: A case-control study. Pain 2003;104:491–499.
5. McNeill C (ed). Temporomandibular Disorders: Guidelines for Classification, Assessment, and Management, ed 2. Chicago: Quintessence, 1993.
6. Lund JP. Effects of pain on muscular activity in temporomandibular disorders and related conditions. In: Stohler CS, Carlson DS (eds). Biological and Psychological Aspects of Orofacial Pain, Craniofacial Growth Series, vol 29. Ann Arbor, MI: The University of Michigan, 1994:75–91.
7. Hough T. Ergographic studies in muscular soreness. Am J Physiol 1902;7:76–92.
8. Francis KT, Hoobler T. Effects of aspirin on delayed muscle soreness. J Sports Med Phys Fitness 1987;27:333–337.
9. Gulick DT KI. Delayed onset muscle soreness: What is it and how do we treat it? J Sport Rehab 1996;5:234–243.
10. Safran MR, Seaber AV, Garrett WE Jr. Warm-up and muscular injury prevention: An update. Sports Med 1989;8:239–249.
11. Gulick DT, Kimura IF, Sitler M, Paolone A, Kelly JD. Various treatment techniques on signs and symptoms of delayed onset muscle soreness. J Athl Train 1996;31:145–152.
12. Weerakkody NS, Whitehead NP, Canny BJ, Gregory JE, Proske U. Large-fiber mechanoreceptors contribute to muscle soreness after eccentric exercise. J Pain 2001;2:209–219.
13. Nosaka K, Clarkson PM. Muscle damage following repeated bouts of high force eccentric exercise. Med Sci Sports Exerc 1995;27:1263–1269.
14. Bajaj P, Graven-Nielsen T, Wright A, Davies IaI, Arendt-Nielsen L. Muscle hyperalgesia in postexercise muscle soreness assessed by single and repetitive ultrasound stimuli. J Pain 2000;1:111–121.
15. Clarkson PM. Exercise-induced muscle damage: Animal and human models. Med Sci Sports Exerc 1992;24:510–511.
16. Clarkson PM. Eccentric exercise and muscle damage. Int J Sports Med 1997;18(suppl 4):S314–S317.
17. Clarkson PM, Hubal MJ. Exercise-induced muscle damage in humans. Am J Phys Med Rehabil 2002;81:S52–S69.
18. Armstrong RB, Warren GL III. Strain-induced skeletal muscle fibre injury. In: Macleod D (ed). Intermittent High Intensity Exercise: Preparation, Stresses and Damage Limitation. London: Routledge, 1993:275–285.
19. Cheung K, Hume P, Maxwell L. Delayed onset muscle soreness: Treatment strategies and performance factors. Sports Med 2003;33:145–164.
20. Proske U, Morgan DL. Muscle damage from eccentric exercise: Mechanism, mechanical signs, adaptation and clinical applications. J Physiol 2001;537:333–345.
21. Clarkson PM, Byrnes WC, McCormick KM, Turcotte LP, White JS. Muscle soreness and serum creatine kinase activity following isometric, eccentric, and concentric exercise. Int J Sports Med 1986;7:152–155.

22. Brockett CL, Morgan DL, Proske U. Human hamstring muscles adapt to eccentric exercise by changing optimum length. Med Sci Sports Exerc 2001;33:783–790.
23. Newham DJ, Mills KR, Quigley BM, Edwards RH. Pain and fatigue after concentric and eccentric muscle contractions. Clin Sci (Lond) 1983;64:55–62.
24. Armstrong RB, Warren GL, Warren JA. Mechanisms of exercise-induced muscle fibre injury. Sports Med 1991;12:184–207.
25. Smith LL. Acute inflammation: The underlying mechanism in delayed onset muscle soreness? Med Sci Sports Exerc 1991;23:542–551.
26. MacIntyre DL, Reid WD, McKenzie DC. Delayed muscle soreness: The inflammatory response to muscle injury and its clinical implications. Sports Med 1995;20:24–40.
27. Weerakkody NS, Percival P, Hickey MW, et al. Effects of local pressure and vibration on muscle pain from eccentric exercise and hypertonic saline. Pain 2003;105:425–435.
28. Goodwin GM, McCloskey DI, Matthews PB. The contribution of muscle afferents to kinaesthesia shown by vibration induced illusions of movement and by the effects of paralysing joint afferents. Brain 1972;95:705–748.
29. Treede R-D, Klein T, Magerl W. Pain memory and central sensitization in humans. In: Flor H, Kalso E, Dostrovsky JO (eds). Proceedings of the 11th World Congress on Pain. Seattle: IASP, 2006:251–267.
30. Hollins M, Sigurdsson A, Fillingim L, Goble AK. Vibrotactile threshold is elevated in temporomandibular disorders. Pain 1996;67:89–96.
31. Hollins M, Sigurdsson A. Vibrotactile amplitude and frequency discrimination in temporomandibular disorders. Pain 1998;75:59–67.
32. Scott DS, Lundeen TF. Myofascial pain involving the masticatory muscles: An experimental model. Pain 1980;8:207–215.
33. Christensen LV. Jaw muscle fatigue and pains induced by experimental tooth clenching: A review. J Oral Rehabil 1981;8:27–36.
34. Bowley JF, Gale EN. Experimental masticatory muscle pain. J Dent Res 1987;66:1765–1769.
35. Clark GT, Adler RC, Lee JJ. Jaw pain and tenderness levels during and after repeated sustained maximum voluntary protrusion. Pain 1991;45:17–22.
36. Glaros AG, Baharloo L, Glass EG. Effect of parafunctional clenching and estrogen on temporomandibular disorder pain. Cranio 1998;16:78–83.
37. Glaros AG, Tabacchi KN, Glass EG. Effect of parafunctional clenching on TMD pain. J Orofac Pain 1998;12:145–152.
38. Plesh O, Curtis DA, Hall LJ, Miller A. Gender difference in jaw pain induced by clenching. J Oral Rehabil 1998;25:258–263.
39. Larsson B, Bjork J, Kadi F, Lindman R, Gerdle B. Blood supply and oxidative metabolism in muscle biopsies of female cleaners with and without myalgia. Clin J Pain 2004;20:440–446.
40. Larsson R, Oberg PA, Larsson SE. Changes of trapezius muscle blood flow and electromyography in chronic neck pain due to trapezius myalgia. Pain 1999;79:45–50.
41. Miles TS. Masticatory muscles. In: Miles TS, Nauntofte B, Svensson P (eds). Clinical Oral Physiology. Copenhagen: Quintessence, 2004:199–217.
42. Torisu T, Wang K, Svensson P, et al. Effects of eccentric jaw exercise on temporal summation in jaw-closing muscles of healthy subjects. Eur J Pain 2010;14:719–724.
43. Farella M, Soneda K, Vilmann A, Thomsen CE, Bakke M. Jaw muscle soreness after tooth-clenching depends on force level. J Dent Res 2010;89:717–721.
44. Goldberg JM, Lindblom U. Standardised method of determining vibratory perception thresholds for diagnosis and screening in neurological investigation. J Neurol Neurosurg Psychiatry 1979;42:793–803.
45. List T, Leijon G, Svensson P. Somatosensory abnormalities in atypical odontalgia: A case-control study. Pain 2008;139:333–341.
46. List T, Helkimo M, Falk G. Reliability and validity of a pressure threshold meter in recording tenderness in the masseter muscle and the anterior temporalis muscle. Cranio 1989;7:223–229.
47. Portney LG, Watkins MP. Foundations of clinical research: Applications to practice. Upper Saddle River, NJ: Pearson/Prentice Hall, 2009.
48. Christensen LV. Facial pain and internal pressure of masseter muscle in experimental bruxism in man. Arch Oral Biol 1971;16:1021–1031.
49. Arima T, Svensson P, Arendt-Nielsen L. Experimental grinding in healthy subjects: A model for postexercise jaw muscle soreness? J Orofac Pain 1999;13:104–114.
50. Friden J. Delayed onset muscle soreness. Scand J Med Sci Sports 2002;12:327–328.
51. Proske U. Muscle tenderness from exercise: Mechanisms [comment]? J Physiol 2005;564:1.
52. Cervero F, Laird JM. Mechanisms of touch-evoked pain (allodynia): A new model. Pain 1996;68:13–23.
53. Bakhtiary AH, Safavi-Farokhi Z, Aminian-Far A. Influence of vibration on delayed onset of muscle soreness following eccentric exercise. Br J Sports Med 2007;41:145–148.
54. Türker KS, Koutris M, Sumer NC, et al. Provocation of delayed-onset muscle soreness in the human jaw-closing muscles. Arch Oral Biol 2010;55:621–626.
55. Svensson P, Jadidi F, Arima T, Baad-Hansen L, Sessle BJ. Relationships between craniofacial pain and bruxism. J Oral Rehabil 2008;35:524–547.
56. Feine JS, Chapman CE, Lund JP, Duncan GH, Bushnell MC. The perception of painful and nonpainful stimuli during voluntary motor activity in man. Somatosens Mot Res 1990;7:113–124.
57. Gallasch E, Kenner T. Characterisation of arm microvibration recorded on an accelerometer. Eur J Appl Physiol Occup Physiol 1997;75:226–232.
58. Okayasu I, Oi K, De Laat A. The effect of tooth clenching on the sensory and pain perception in the oro-facial region of symptom-free men and women. J Oral Rehabil 2009;36:476–482.
59. Leung YY, Bensmaia SJ, Hsiao SS, Johnson KO. Time-course of vibratory adaptation and recovery in cutaneous mechanoreceptive afferents. J Neurophysiol 2005;94:3037–3045.
60. Berglund U, Berglund B. Adaption and recovery in vibrotactile perception. Percept Mot Skills 1970;30:843–853.
61. Hollins M, Goble AK, Whitsel BL, Tommerdahl M. Time course and action spectrum of vibrotactile adaptation. Somatosens Mot Res 1990;7:205–221.
62. Brennum J, Kjeldsen M, Jensen K, Jensen TS. Measurements of human pressure-pain thresholds on fingers and toes. Pain 1989;38:211–217.
63. Isselee H, De Laat A, Lesaffre E, Lysens R. Short-term reproducibility of pressure pain thresholds in masseter and temporalis muscles of symptom-free subjects. Eur J Oral Sci 1997;105:583–587.
64. Kosek E, Ekholm J, Nordemar R. A comparison of pressure pain thresholds in different tissues and body regions. Long-term reliability of pressure algometry in healthy volunteers. Scand J Rehabil Med 1993;25:117–124.

65. Nussbaum EL, Downes L. Reliability of clinical pressure-pain algometric measurements obtained on consecutive days. Phys Ther 1998;78:160–169.
66. Hedenberg-Magnusson B, Brodda Jansen G, Ernberg M, Kopp S. Effects of isometric contraction on intramuscular level of neuropeptide Y and local pain perception. Acta Odontol Scand 2006;64:360–367.
67. Glaros AG, Forbes M, Shanker J, Glass EG. Effect of parafunctional clenching on temporomandibular disorder pain and proprioceptive awareness. Cranio 2000;18:198–204.
68. Clark GT, Carter MC. Electromyographic study of human jaw-closing muscle endurance, fatigue and recovery at various isometric force levels. Arch Oral Biol 1985;30:563–569.
69. Clark GT, Jow RW, Lee JJ. Jaw pain and stiffness levels after repeated maximum voluntary clenching. J Dent Res 1989;68:69–71.
70. Lam EW, Hannam AG. Regional 31P magnetic resonance spectroscopy of exercising human masseter muscle. Arch Oral Biol 1992;37:49–56.
71. Graven-Nielsen T, Mense S. The peripheral apparatus of muscle pain: Evidence from animal and human studies. Clin J Pain 2001;17:2–10.
72. Rosendal L, Larsson B, Kristiansen J, et al. Increase in muscle nociceptive substances and anaerobic metabolism in patients with trapezius myalgia: Microdialysis in rest and during exercise. Pain 2004;112:324–334.
73. Ernberg M, Hedenberg-Magnusson B, Alstergren P, Kopp S. The level of serotonin in the superficial masseter muscle in relation to local pain and allodynia. Life Sci 1999;65:313–325.
74. Hedenberg-Magnusson B, Ernberg M, Alstergren P, Kopp S. Pain mediation by prostaglandin E2 and leukotriene B4 in the human masseter muscle. Acta Odontol Scand 2001;59:348–355.
75. Babenko V, Graven-Nielsen T, Svensson P, Drewes AM, Jensen TS, Arendt-Nielsen L. Experimental human muscle pain and muscular hyperalgesia induced by combinations of serotonin and bradykinin. Pain 1999;82:1–8.
76. Ernberg M, Lundeberg T, Kopp S. Pain and allodynia/hyperalgesia induced by intramuscular injection of serotonin in patients with fibromyalgia and healthy individuals. Pain 2000;85:31–39.
77. Svensson P, Cairns BE, Wang K, et al. Glutamate-evoked pain and mechanical allodynia in the human masseter muscle. Pain 2003;101:221–227.
78. LeResche L, Mancl L, Sherman JJ, Gandara B, Dworkin SF. Changes in temporomandibular pain and other symptoms across the menstrual cycle. Pain 2003;106:253–261.
79. Sherman JJ, LeResche L. Does experimental pain response vary across the menstrual cycle? A methodological review. Am J Physiol Regul Integr Comp Physiol 2006;291:R245–R256.