Tactile and Pain Thresholds in Patients with Myofascial Pain of the Jaw Muscles: A Case-Control Study

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Aims: To compare the tactile detection threshold, the filament-prick pain detection threshold, the pressure pain threshold, and the pressure pain tolerance detection threshold at multiple measuring points in the orofacial region and at the thenar muscle of symptom-free subjects and patients with myofascial pain of the masticatory muscles. ***Methods:*** *Twenty patients (age range: 25 to 55 years) and 20 healthy subjects (age range: 25 to 55 years) were recruited. The tactile detection threshold and the filament prick-pain detection threshold were measured at the cheek skin overlying the central part of the left and right masseter muscles, at the right thenar muscle and at the tip of the tongue, using Semmes-Weinstein monofilaments. The pressure pain threshold and the pressure pain tolerance threshold were measured at the central part of the masseter muscle and on the thenar muscle, using a pressure algometer. The intensity of pain perceived during the assessment of filament prick-pain detection threshold, pressure pain threshold, and the pressure pain tolerance threshold was scored on visual analog scales.* ***Results:*** *The tongue tip had the lowest filament prick-pain detection thresholds as compared to the other sites. Filament prick-pain detection thresholds of the tongue and thumb sites were significantly lower in myofascial pain patients than in controls. Pressure pain thresholds of the masseter and thenar muscles were significantly lower in patients with myofascial pain than in control subjects whereas pressure pain tolerance thresholds did not differ significantly between patients and controls.* ***Conclusions:*** *The findings of the present study show topographic variations in the pain responses to different stimulus modalities. Different pain responses were also found between patients with myofascial pain and control subjects and were interpreted to support theories of centrally mediated pain for temporomandibular disorders.* J OROFAC PAIN 2008;22:139–145

Key words: jaw muscles, pain thresholds, temporomandibular disorders, trigeminal sensory testing, Von Frey hairs

Temporomandibular disorders (TMD) represent a heterogeneous group of pathologies affecting the temporomandibular joint, the masticatory muscles, or both[1] and show a peculiar distribution in the general population, with a predominance in females, especially during their reproductive years.[2]

Usually, nociceptive pain is not localized, but it can be regional or even generalized.[3] Referred pain can be explained by the convergent projection of multiple nociceptive afferents on fewer neurons in the central nervous system,[4,5] especially in the trigeminal area.[6] Another process that helps explain referred pain mecha-

nisms is central sensitization,[7,8] whereby a continuous barrage of painful input can lead to a hyperexcitability and spontaneous activity of wide-dynamic-range neurons and nociceptive-specific neurons in the brainstem and spinal cord,[9,10] expanding the receptive field area and causing nonpainful information to be reported as painful. This neuroplastic process alters the normal processing of nociceptive information, and consequently, pain threshold may be lowered.[6,11]

In a previous study[12] the tactile detection threshold (TDT), the filament prick-pain detection threshold (FPT), the pressure pain threshold (PPT), and the pressure pain tolerance threshold (PTOL) at multiple measuring points in the orofacial region and at the thenar muscle have been evaluated. The findings of this study suggested that these thresholds differ significantly between sites and genders. Even though several PPT investigations in cases and controls have been published,[13–17] to the authors' knowledge little is known about TDTs and FPTs in myofascial pain patients as compared to control subjects. Consequently, the aim of the present study was to compare the TDT, FPT, PPT, and PTOL at multiple measuring points in the orofacial region and at the thenar muscle of symptom-free subjects and patients with myofascial pain of the masticatory muscles.

Materials and Methods

Patients and Control Subjects

Twenty consecutive female patients (mean age ± SD, 45 ± 7.8 years), all of whom were referred to the Department of Oral and Maxillofacial Surgery of the Catholic University of Leuven, were included in this study. Inclusion criteria for the study were female patients aged between 25 and 55 years diagnosed with myofascial pain of the masticatory muscles (Axis I – Group I) according to the Research Diagnostic Criteria for TMD.[18]

Exclusion criteria were the presence of TMD of arthrogenous origin, fibromyalgia, migraine, neuralgia, and other musculoskeletal diseases; history of drug abuse; use of medication; recent facial or cervical trauma; and the presence of general health problems or periodontal disease.

Twenty age-matched female subjects were selected as controls from the university staff; all were healthy Caucasians asymptomatic for pain in the head or neck region. The mean age of these subjects was 37.8 ± 11 years. The age matching procedure was not completely successful, as the control subjects were significantly younger than the TMD patients (unpaired t test: $t = 2.39$; $P = .022$). However, the subjects were similar with respect to weight and height. The patient group had a mean weight of 65.3 ± 10.1 kg, while the control group had a mean weight of 62.9 ± 9.1 (t test; $P = .424$). The mean height was 164.0 ± 5.4 cm for the patient group and 166.7 ± 5.7 cm for the control group (t test; $P = .133$).

Since some authors have reported a systematic variation of sensory perception during the menstrual phase,[19,20] the participating subjects were not tested in that phase.

In addition, previous studies have shown that subjects tolerate pain longer when they are tested by an examiner of the opposite sex.[21,22] In the present study, all subjects were examined by the same female examiner (AS).

All the participants were fully informed about the experimental procedure in a standard way, and all gave their written consent prior to participation in the study.

TDT and FPT

TDT and FPT were determined using Semmes-Weinstein monofilaments (Premier Product). The instrument and the procedure have been described in detail elsewhere.[12] Briefly, the 20 monofilaments used each had a different diameter, and the filament number (1.65–6.65) corresponded to a logarithmic function of the equivalent forces of 0.0045 to 447 g.

The sensory threshold and sensory pain threshold were measured:

- On the cheek skin overlying the central part of the left and right masseter muscles midway between the upper and lower borders and 1 cm posterior to the anterior border
- At a point 5 mm proximal to the anterior tip of the tongue
- On the skin overlying the palm side of the right thenar muscle

During the first test (TDT), subjects were instructed to close their eyes and to raise their hand as soon as they felt the stimulus. The filament was applied vertically on the site, and pressure was applied slowly until the filament bowed. Quick applications of filaments against the skin were avoided. If the subject raised her hand with the first filament used, this was considered a positive response, and the following filament applied was one increment smaller. This procedure was repeated

with decreased filament diameters until the subject did not feel pressure anymore. This was considered a negative response. Once a negative response had been reported, the filament with a higher diameter was applied, and this procedure was continued until 8 positive and 8 negative answers were obtained. Finally, the sensory threshold was calculated as the average of these values (filament numbers: 1.65 to 6.65). If the subject still had a positive response when the lowest (most narrow) filament was applied, this was considered the threshold. During the test, 2 placebo trials were performed after peaks 5 and 11, and if the subject reported a negative response, the test was continued. Otherwise it was stopped, the whole procedure was explained again, and the test was restarted.[23]

During the second test (FPT) stimuli were applied in the same way as for the TDT test, but subjects could open their eyes and were instructed to raise their hand when they felt pain, not only pressure. No placebo stimuli were applied. There was a time lag of 3 minutes between the measurements on a similar site in order to avoid sensitization. After the examination, subjects were asked to indicate the perceived pain intensity on a visual analog scale (VAS) where 0 cm indicated "no pain" and 10 cm indicated "the most pain imaginable."

PPT and PTOL

PPT was determined with an electronic algometer (Somedic). The instrument and the procedure have been described in detail elsewhere.[12] Briefly, the tip of the algometer had a surface of 1 cm^2, and a rate of pressure increase of approximately 30 kPa/s was chosen. These measurements were made at least 5 minutes after the FPT measurements. The PPT was determined as the point at which the pressure stimulus applied to the skin changed from a pressure sensation into a sensation of pain.[14] PPTs were assessed by a single examiner at the left and right masseter. An additional measurement was performed at the right thenar muscle, which was selected as a control muscular site. The PTOL was defined as the maximum pain that subjects were able to accept. The subjects indicated the PPT and the PTOL by pressing a button, which froze the current pressure value on the digital display. The measurements were made at least 5 minutes after the sensory pain threshold was determined. The subjects sat in a dental chair and were asked to relax in the mandibular rest position during the recordings to avoid contraction of jaw-closing muscles during the stimulation. While the PPT and the PTOL were being assessed, the subject's head was supported by counter-pressure from the opposite hand of the examiner. During the measurements, the algometer was held perpendicular to the skin. Algometric measurements were performed on the right and left masseter muscles and on the right thenar muscle. Three PPT measurements were made at each recording site with a 2-minute rest interval between trials, and the mean value of the 3 measurements was used for further statistical analysis. The PTOL measurement was performed just once at the end of the whole experimental session. After each examination, the average pain intensity during the PPT and the PTOL measurements was assessed on a VAS where 0 cm indicated "no pain" and 10 cm indicated "the most pain imaginable."

Statistical Analysis

Data were first analyzed by means of conventional descriptive methods (ie, means, range, standard deviations). The influence of anatomical site, facial side, and of case-control status was tested by means of repeated measurements analysis of variance, which included age and body mass index as covariates into the regression model. When statistical significance was obtained, post-hoc analysis (Bonferroni corrected) was used.

Results

Preliminary analysis of TDT, FPT, PPT, and PTOL at the recording sites was carried out. Comparison of the left and right masseter muscle sites did not reveal significant differences (TDT: $F = 3.89$; $P = .056$; FPT: $F = 0.86$; $P = .36$; PPT: $F = 1.21$; $P = .28$; PTOL: $F = 0.18$; $P = .67$); consequently, the data from the 2 sides were averaged in order to obtain a single value.

Results for the TDT test are illustrated in Fig 1. The case-control status did not influence the TDT ($F = 0.05$; $P = .83$), and no significant differences between anatomic sites (masseter, tongue tip, thenar muscle) were observed ($F = 1.81$; $P = .18$). There was no age effect ($F = 1.41$; $P = .24$), but body mass index was found to have a positive influence on TDT values ($F = 4.3$; $P = .04$; ie, higher body mass indices corresponded to higher TDT values). None of the first-order interactions was statistically significant ($F \leq 1.81$; $P \geq .20$). A post-hoc power analysis was carried out using the estimates of variability of TDT (main outcome variables) obtained from control subjects, setting type I error at 0.05, and considering a clinically

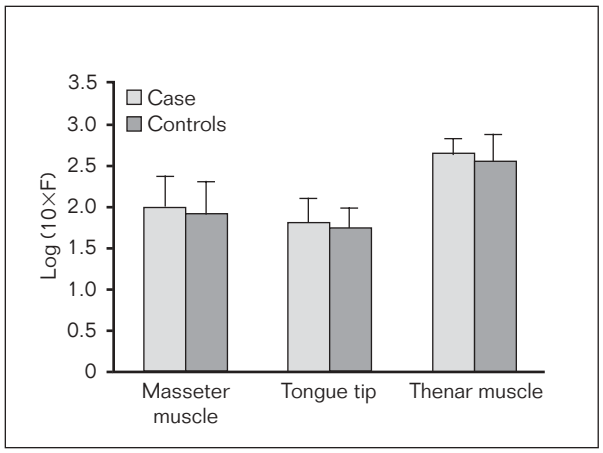

Fig 1 Means and standard deviations for TDT at the masseter and thenar muscles and tongue tip of cases and controls. Data from the left and right masseter were averaged.

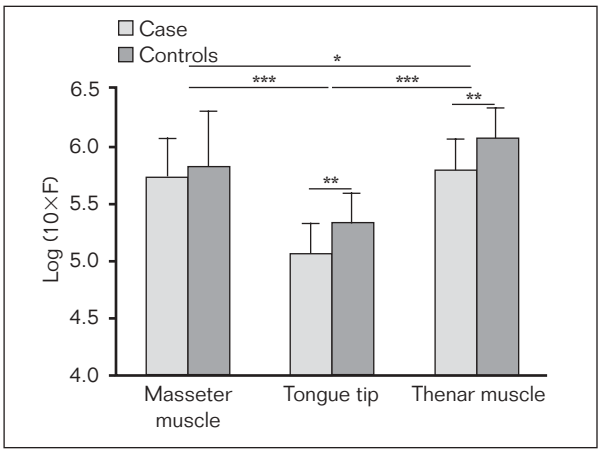

Fig 2 Means and standard deviations of FPT at the masseter muscles, thenar muscles, and tongue tip of cases and controls. Data from the left and right masseter were averaged. $*P < .05$, $**P < .01$, and $***P < .001$.

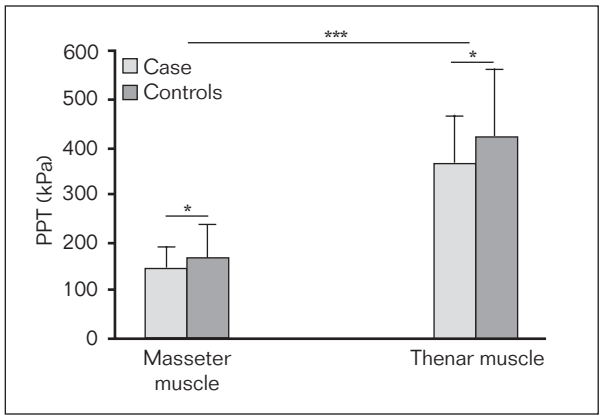

Fig 3 Means and standard deviations of PPT at the masseter and thenar muscles of cases and controls. Data from the left and right masseter were averaged. $*P < .05$, $***P < .001$.

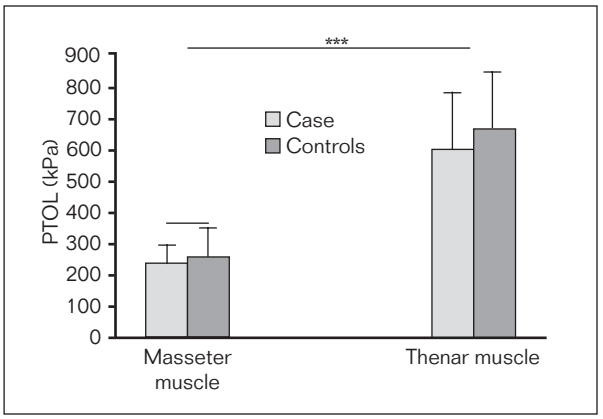

Fig 4 Means and standard deviations of PTOL at the masseter and thenar muscles of cases and controls. Data from the left and right masseter were averaged. $***P < .001$.

relevant difference in TDT between population means of 20% or greater. This analysis revealed that the power of the statistical tests performed for TDT of the masseter, tongue, and thenar was always greater than 90%.

The results of the FPT test are shown in Fig 2. FPT values of myofascial pain patients were significantly lower than those of controls at the tongue ($P = .006$) and thumb ($P = .002$) sites, and there was a significant difference between each pair of anatomic sites ($P < .05$). There was no age ($F = 0.03$; $P = .87$) or body mass index ($F = 0.00$; $P = .99$) effect.

PPTs were significantly lower ($F \geq 4.19$; $P \leq .048$) in patients than in controls for all the muscles investigated; there was an apparent difference between the masseter muscle and the thenar muscle ($F = 183$; $P < .001$; Fig 3) that disappeared after introducing body mass index and age in the analysis ($F = 3.53$; $P = .07$). The analysis revealed no significant age ($F = 1.99$; $P = .17$) or body mass index ($F = 0.02$; $P = .88$) effect.

Results of the PTOL test are illustrated in Fig 4. PTOL did not differ significantly between patients and control subjects ($F = 2.63$; $P = .11$). Also in this case, the apparent difference between the masseter muscle and thenar muscle ($F = 269.3$; $P < .001$; Fig 4) disappeared after introducing body mass index and age in the analysis ($F = 3.03$; $P = .09$). Statistical analysis revealed no age ($F = 1.5$; $P = .23$) or body mass index ($F = 0.33$; $P = .57$) effect.

Due to the small sample size, Pearson's correlations coefficients between the different stimulus modalities were calculated for the entire sample (n = 40). Significant correlations were found between

FPT and PPT (r = 0.360; P = .023), between FPT and PTOL (r = 0.414; P = .008), and between PPT and PTOL (r = 0.636; P < .001).

Discussion

Some authors have suggested that clinical and experimental pain could modify the sensory threshold.[24,25] In the present study, mechanical tests were used to investigate possible differences of sensitivity and pain between myofascial pain patients and healthy controls. There were no significant differences in TDT measurements either among sites or between case-control statuses. Previous studies investigating changes in mechanosensitivity during several pain conditions have led to inconsistent findings, as both skin hyperesthesia and hypoesthesia have been reported using experimental and clinical pain models.[26–28] This apparent contradiction can probably be explained by the different equipment (ie, the number and the bending force of monofilaments) used for sensory evaluations.[26] Interestingly, a previous study[26] in which Semmes-Weinstein monofilaments with a procedure similar to that of the present study were used showed that the tonic experimental muscle pain inhibited touch perception in a gender-related manner, with the inhibition being much more pronounced in males than in females. The fact that only female patients were investigated in the present study can help explain the lack of difference of TDT between patients and controls investigated.

An interesting finding of the study was that FPT values of myofascial pain patients at the tongue and thenar muscle sites were significantly lower than those of control subjects. FPT values of myofascial patients at the cheek, however, did not differ significantly from those of control subjects. The lack of difference at the latter site might be ascribed to the fact that the skin layer overlying the masseter muscle is much thicker than that of the tongue and thenar sites.[29,30] It needs also to be emphasized that both the tongue and the finger are more widely represented in the primary somatosensory cortex than the cheek.[31] Finally, the nociceptive epidermal nerve fiber density of the cheek area is probably lower that that of the tongue and thumb sites. This latter suggestion, however, is mostly speculative because, to the authors' knowledge, there are no data comparing the density of nociceptive endings between the tongue, the cheek, and the thumb.

FPT values of myofascial pain patients have never been assessed in other studies; therefore, the present findings cannot be compared with previous ones. The reduced FPT values at the tongue and thenar muscle sites in patients affected with myofascial pain of the jaw muscle, however, point to a widespread neuroplasticity and/or generalized overresponsiveness to peripheral stimuli, and this supports the hypothesis of a diffuse disruption of central pain-modulating systems in this subgroup of chronic pain patients.[32]

The observed differences of FPT between various skin sites are, to some extent, in agreement with a previous study[12] and underline the variability of the tested afferents with respect to their density and/or of variations in the processing within the central nervous system of tactile/pain information.[33] The pressure applied with the filaments to obtain the FPT is not necessarily confined to skin tissues, as the monofilament might have also stimulated the underlying muscle. Hence, the lowered FPT found in these patients may reflect the combined tenderness of both skin and muscle.

In agreement with previous studies,[13–17] a reduction of PPTs occurred in patients with myofascial pain compared to the control group. The reduced PPT found at the masseter muscles of the myofascial pain patients might be explained by both peripheral and central mechanisms. In the case of peripheral mechanisms, an increase in the excitability of the nociceptor terminal membrane will reduce the amount of depolarization required to initiate an action potential discharge. This modulation occurs on exposure of the terminal to sensitizing agents such as prostaglandin E2 (PGE_2) and 5-hydroxytryptamine (5-HT).[34] In the case of central mechanisms, previous experimental animal studies have shown that repetitive nociceptive stimulation in the orofacial area produces prolonged hyperexcitability in trigeminal brainstem wide-dynamic-range and nociceptive-specific neurons.[7,35] This phenomenon has been defined as central sensitization.[36] Trigeminal wide-dynamic-range and nociceptive-specific neurons receive extensive convergent inputs from both superficial and deep tissues and can be modulated by nerve injuries as well as by inflammatory conditions affecting orofacial tissues.[7,37–39] Thus, also the central sensitization along with the convergence of noxious inputs to trigeminal WDR and NS neurons can account for the decrease of PPT found in the present study.[40]

The comparison of PPT between the most and the least painful side in the patient sample revealed no differences; this is consistent with another investigation[16] and can be interpreted in support of theories of centrally mediated pain for TMD.[15] In a previous study,[16] PPTs at the index finger were

compared between female patients and matched control subjects, and no statistically significant differences were reported; in contrast, the present study documented a significant difference in PPTs at the thenar muscle between patients and control subjects. The lowered PPTs found at both the painful masseter and at an extracephalic site (ie, thenar muscle) give further support to the hypothesis that patients suffering with myofascial pain of the jaw muscles exhibit a generalized hypersensitivity of the central nervous system, which also amplifies nonsegmental nociceptive inputs.

In the present study, there was no significant difference in PTOLs between patients and control subjects at either the masseter or the thenar muscles, in contrast to another study[16] that showed a lower PTOL at the masseter muscle in patients compared to controls. However, in the present study, statistically significant differences in PPT and PTOL between masseter and thenar muscles disappeared after introducing body mass index and age as covariates in the regression model. These findings could be partly explained by the effect of adipose tissue thickness on both pain thresholds, thus underlining the importance of taking into account the body mass index when using pressure algometers. In contrast to this hypothesis, however, a previous study reported higher PPT values in obese patients with eating disorders but not in obese patients without eating disorders. These observations may suggest that the "body mass index effect" on pain threshold can be explained by more complex mechanisms involving interactions between vagal afferents and nociceptive response. In this respect, it is interesting to note that patients affected with eating disorders have an abnormal functioning of the vagal nerve, and may also suffer from abnormal nociceptive processing.[41]

The positive correlations found between FPT and PPT, between FPT and PTOL, and between PPT and PTOL are consistent with the findings of a recent study[42] obtained in a group of symptom-free subjects and suggest that, even if specific stimuli should predominantly test for specific sensory inputs, the perception of pain after these stimuli is correlated, regardless of the stimulus modality used.

In conclusion, the findings of the present study showed topographic variations in the pain responses to different stimulus modalities. Different pain responses were also found between patients with myofascial pain and control subjects. These latter differences are probably centrally mediated and support the occurrence of diffuse disruption of central pain-modulating systems in patients affected with myofascial pain of the jaw muscles.

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