Cutaneous Sensory Thresholds from Skin Overlying Masseter and Forearm in MPD Patients and Controls

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Cutaneous sensory thresholds from skin overlying both masseters and right ventral forearm were measured in a group of 12 MPD patients and an appropriately matched group of non-patients. No significant differences were found between the two groups, nor were there significant differences between symptomatic and contralateral asymptomatic sites in the patients. There does not appear to be evidence for differences in sensory characteristics in the cutaneous afferent pathway of MPD patients.

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Introduction.

Unilateral myofascial pain of the masticatory musculature is reported in 70 to 80% of patients suffering from myofascial pain dysfunction syndrome (MPD).[1-6] Whereas there is disagreement with respect to the etiology of MPD, most authorities agree that the pain symptom originates from the involved muscle groups.[7-9] The unilateral nature of the pain may be explained either by differential muscle activity levels between the pain and non-pain sides, or by differential sensory thresholds between the pain and non-pain sides.

Malmo *et al.*[10] and Malmo[11] were the first to report differential response characteristics, *i.e.,* different levels of motor activity, between pain and non-pain sides in patients complaining of "tensional discomfort" in a symptomatic muscle group. Procacci *et al.*[12] compared painful and non-painful limb muscles in patients with unilateral myofascial pain associated with reflex sympathetic dystrophies. They also reported different levels of activity of the symptomatic muscle compared to the contralateral asymptomatic muscle.

In contrast to the limb muscles, there is as yet no experimental evidence to support such a differential motor response in the masticatory musculature. When subjected to stress, MPD patients did not respond with differential muscle activity between pain *vs.* non-pain sides.[13-15] On the basis of these studies, differential responsivity has not been demonstrated in masticatory muscles and, therefore, could not have accounted for unilateral pain.

An alternative source of unilateral symptomatology could be differential sensory thresholds in the cutaneous pathways overlying the masticatory musculature.

With respect to general cutaneous sensory thresholds, Molin *et al.*[16] compared MPD patients with controls, and characterized the patient group as "less tolerant to painful stimuli than the non-patient group." Malow *et al.*[17] compared MPD patients with controls and found significant differences in sensory thresholds. For both studies, in response to experimentally induced pain, patients were more sensitive than non-patients.

While both studies reported that MPD patients had lower pain thresholds than did a comparison group of non-patients, neither study attempted to examine the pain site and sensory threshold. The present study is a replication and extension of the work of Molin *et al.* and Malow *et al.* If sensory differentials are responsible for pain, then one might expect differential sensory characteristics not only between patient and non-patient, but between the pain and non-pain sites as well. MPD patients would be expected to have lower pain thresholds compared to non-patients, and the symptomatic side would be expected to have a lower pain threshold than the contralateral (asymptomatic) side.

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Materials and methods.

Subjects. – The MPD clinic of the School of Dentistry of the State University of New York at Buffalo yielded 12 patients diagnosed as having MPD syndrome; these were included in the study. The diagnosis of MPD syndrome was based on the presence of unilateral masseter pain and the presence of at least one other cardinal sign of temporomandibular joint dysfunction, *e.g.,* limitation of jaw mobility or clicking of the jaw joint. Patients who fit these criteria, with no other known disease of the head or neck, were included in the study. Based upon previously reported characteristics, *e.g.,* sex and occupation,[1-6] the group represented typical MPD patients.

The patient group consisted of ten women and two men. The mean age of the patients was 37.5 yr, with a range from 18 to 57 yr. The duration of their symptoms ranged from six mo to over two yr. Ten of the 12 patients had had previous treatment as follows: three patients, equilibration; three patients, equilibration and splints; two patients, medication and splints; one patient, third molar extractions; and one patient, medication only.

Control subjects were screened on the basis of a negative history of TMJ/MPD-associated myofascial pain. None of the control subjects reported spontaneous masseteric pain or had tenderness to palpation of the masseter muscle. Previous research has indicated that factors such as ethnicity,[18] socio-economic status,[19] and occupation[20] may influence sensory thresholds. Control subjects were matched for age (mean age, 37.75 yr, with a range from 19 to 61 yr), sex, ethnicity, and at least one other of the following variables: socio-economic status, marital status, or occupation.

Equipment. – The stimulating apparatus consisted of a Grass Model S-4 constant voltage stimulator connected to a Grass Model SIU-5 stimulus isolation unit. A Grass EZM-3 impedance meter was used to measure electrode impedance, and a Tektronix 564-B oscilloscope was used to calibrate the stimulator.

Procedure. – After giving informed consent, each subject was interviewed and examined clinically. The interview consisted of a brief psychosocial profile and standard medical history. The clinical examination consisted of: (1) assessment of mandibular dysfunction, (2) location of muscle pain, (3) assessment of temporomandibular joint sounds, and (4) intra-oral evaluation.

For the experimental trials, each subject was seated in a comfortable chair. For the patient group, the cutaneous areas over three sites – the symptomatic masseter, the asymptomatic masseter, and the right ventral forearm – were prepared for threshold studies. The forearm site was chosen to represent a non-cephalic somatosensory control. For each non-patient, the masseter corresponding to the matched patient's symptomatic masseter, the contralateral masseter, and the right ventral forearm were prepared. Each site was scrubbed with alcohol to remove surface oils and reduce impedance. For the masseter sites, an adhesive-backed corn pad (3/8" diameter) was placed at a point one inch from the angle of the mandible on a line that bisected the angle. A second pad, tip-to-tip with the first, was placed on the same bisecting line. The pads were filled with conductive gel, and gold-plated electrodes were placed inside and secured with tape. The tip-to-tip placement of the pads assured a consistent inter-electrode distance, and, since the conduction area for each pad was the same, the current density for each electrode was presumably constant.

For the forearm site, corn-pad placement was standardized lateral to the midpoint between the wrist and cubital fossa. As with the masseter sites, the pads were filled with conductive gel, and gold-plated electrodes were placed inside and secured with tape.

Impedance for each pair of electrodes was measured with the impedance meter immediately prior to electrical stimulation. The frequency of the stimulator was set at 10 Hz, and the pulsewidth was held constant at 0.2 ms. Thresholds were determined by the method of ascending limits. For each site, the voltage was increased gradually from zero, and the subject was instructed to report the first noticeable sensation. This value, in volts, was recorded as the threshold for detection of sensation (TDS). The voltage was then increased slowly, and the subject was instructed to report when the stimulus became painful. This value, in volts, was entered as the pain detection threshold (PT). The voltage was reduced to zero, and the sequence was repeated four more times. The mean voltage for five trials for each site was taken as the final data point.

At the completion of the fifth trial, the impedance was recorded again. Impedance for each site was considered the mean of the two readings. To offset possible order effects, subjects were completely counterbalanced with respect to the sequence of site stimulation. Once the mean voltages were computed, current was calculated for each threshold.

The voltage delivered to a subject was transformed to milliamperes (ma) by means of the following procedures: For impedance levels corresponding to those of patients and controls, actual voltage was measured on an oscilloscope across a known resistance. Amperage was calculated using Ohm's law, and was plotted against dial voltage.

Results.

Results indicated no significant differences in thresholds between patients and controls, and no significant differences between symptomatic and asymptomatic masseter sites in the patient group. The sensory thresholds, TDS, ranged from 0.1 to 3.1 ma for the patients, and from 0.1 to 1.9 ma for controls. The pain thresholds, PT, ranged from 0.4 to 19.3 ma for the patients, and from 0.9 to 12.6 ma for controls. The means and standard deviations for both groups at all sites are presented in the Table.

To determine significance levels, an all-within analysis of variance was calculated for each threshold, by site and group. Interpretation of the results indicated that TDS and PT did not differ significantly between patients and controls for any site. There was a significant interaction for both thresholds between groups and sites (TDS $F = 31.5$, $df = 2,22$, $p < .01$; PT $F = 19.82$, $df = 2,22$, $p < .01$). There was also a significant difference among sites for both thresholds (TDS $F = 3.59$, $df = 2,22$, $p < .05$; PT $F = 5.2$, $df = 2,22$, $p < .05$).

To further confirm that there was a significant difference among sites in the patients specifically, a separate one-way all-within analysis of variance was calculated for each threshold. Results indicated significant differences among sites for both thresholds in this group (TDS $F = 4.8$, $df = 2,11$, $p < .025$; PT $F = 6.7$, $df = 2,11$, $p < .01$). Further analysis of these differences was carried out using the paired t tests. Comparing symptomatic and asymptomatic masseters revealed no significant differences at either threshold. When comparing the forearm to the symptomatic masseter site at TDS, we noted a significant difference ($t = 2.59$, $df = 11$, $p < .025$). When comparing the forearm to the symptomatic masseter site at PT, we noted a significant difference as well ($t = 3.12$, $df = 11$, $p < .01$).

TABLE
MEAN AND STANDARD DEVIATION IN MILLIAMPERES (MA) FOR THRESHOLD OF SENSATION AND PAIN FOR PATIENTS AND CONTROLS

	Patients	Controls
	Threshold of Sensation	
Symptomatic Masseter	1.56 ± 1.2	1.15 ± .51
Asymptomatic Masseter	1.06 ± 0.7	1.05 ± 0.5
Forearm	0.65 ± 0.4	0.51 ± 0.4
	Pain Threshold	
Symptomatic Masseter	6.25 ± 4.3	6.4 ± 2.8
Asymptomatic Masseter	5.19 ± 3.8	6.28 ± 2.6
Forearm	3.35 ± 1.9	5.00 ± 3.2

Discussion.

Lower pain thresholds in response to experimentally induced noxious stimulation of an asymptomatic, non-cephalic site have been reported in MPD patients when compared to non-patient populations.[16,17] We failed to demonstrate these differences between patients and non-patients in response to stimulation of a symptomatic cephalic, asymptomatic cephalic, and non-cephalic site. Differences were observed between the cephalic and non-cephalic sites, which may have been the result of site preparation. Most importantly, there were no differences between patients and controls at the cephalic and non-cephalic sites.

Molin *et al.*[16] attributed lower pain thresholds in their patients to either inherent sensitivity to pain, or the influence of specific personality traits characteristic of MPD patients. With respect to the former, there have been no published reports to support this hypothesis.

With respect to personality traits, recent research does not support the hypothesis that MPD patients can be reliably and consistently characterized on the basis of personality traits.[21-23] Indeed, pain threshold is reported to be fairly constant for an individual, regardless of psychological state.[24] Psychological variables are thought to exert far more influence on pain tolerance levels[24,25] than on pain threshold. Interestingly, in Molin's study, pain tolerance levels did not differ significantly between patients and controls.

Malow *et al.*, in a more complete assessment of sensory characteristics of MPD patients and controls, found significant differences between these two groups in three areas: Patients "exhibited significantly lower pain thresholds . . . lower sensitivity (discrimination) . . . and had a significantly greater tendency to report pain." Malow *et al.* conducted a more precise and more quantitative study than usually encountered. However, their comparison group, the source of normative data, may not have been appropriately matched. The "normal" group – "female psychology majors (mean age 19 yr ± 1.2)" – was significantly younger than the patient group (mean age 28 yr ± 0.1), [$t = 4$; df $= 38$, $p < .001$]. Any one of a number of variables besides age, among them ethnicity[18] or socio-economic status,[19] could have accounted for group differences. Thus, to consider their control group representative of an appropriate control may not be entirely valid.

Differences between the present study and previous work could be the result of sample size. Molin *et al.* studied 27 patients and a control group of 26 subjects, and Malow *et al.* studied 20 patients and 20 control subjects. Differences between the MPD groups could have accounted for the lack of positive findings in the present study. Our group had a mean age of 37.75 yr, higher than that of Molin *et al.* (29.0 yr) or Malow *et al.* (28.0 yr). In a recent study, however, Sharav *et al.*[26] did not observe differences in pain thresholds in response to pulpal stimulation in MPD patients and controls.

Unilateral MPD pain may stem from intrinsic differences in sensory response or differential motor activity of the symptom side compared to the non-symptom side. With respect to sensory differences, Callaghan *et al.*[27] studied patients with chronic pain in one limb, and reported differences between painful and contralateral non-painful sites in "sensory sensitivity." We were unable to demonstrate similar differences between symptomatic and asymptomatic masseters in our patient population.

With respect to motor activity levels, recent studies have reported differences in skin potential and EMG response in patients with unilateral pain associated with reflex sympathetic dystrophies.[12] In separate studies of patients suffering from chronic myofascial pain in one lower limb, differences in EMG responses between the painful and non-painful limb were reported.[28,29] Studies by Malmo *et al.*[10] and Malmo[11] reported differential findings comparing pain sites with non-pain sites in patients with unilateral muscle pain. When subjected to stress, these patients demonstrated significantly higher muscle tension, measured electromyographically, in the affected limb.

Similar lateralization has not been reported in masticatory muscles. Mercuri *et al.*[30] and Thomas *et al.*[15] reported response specificity for the masticatory system as a whole when patients were compared to controls. Consistent with others,[13,14] however, they were unable to report differential motor activity levels comparing pain and non-pain sides in the masticatory system. These results may be explained in part by the failure to use what may be considered a "relevant stimulus," *i.e.*, a stimulus shown empirically to be "related to the disorder of interest."[31] It is possible that measurable differences in responsivity depend on a relevant stimulus, whereas the general hierarchy of responsivity can be elicited with various stimuli.

Unilateral pain may be the result of differences between afferent pathways that we were unable to detect, *e.g.*, deep somatosensory receptors or fibers which we did not stimulate. It is also possible that differential motor responses as yet undiscovered are responsible for the clinical finding of unilateral pain. Interestingly, in a rather qualitative evaluation of functional habits, pain and activity levels, Franks[32] suggested that unilateral pain was associated with hyperactivity on the symptom side in MPD patients.

In light of the evidence supporting differential motor response associated with unilateral pain in limb muscles, continued consideration should be given to this hypothesis as a source of unilateral pain in masticatory muscles.

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LETTER TO THE EDITOR

Dear Sir:

RE: *Mineralization of Dentin in Regional Odontodysplasia*

The apparent disagreement between Kerebel and Kerebel[1] and ourselves[2] concerning the mineralization of dentin in regional odontodysplasia is readily explained. The case studied by the Kerebels did not exhibit the peculiar amorphous areas in the dentin that are a characteristic, although inconstant, feature of this disorder. We did examine these amorphous areas and found them to be more highly mineralized than both the surrounding dentin and the dentin of control teeth. On the other hand, we found that the dentin of regional odontodysplasia, other than these amorphous areas, was apparently slightly less mineralized than normal dentin, although the difference was not significant. This latter finding is in agreement with that of the Kerebels.

— D.G. Gardner, DDS, MSD,
Professor and Chairman,
Division of Oral Pathology,
The University of Western Ontario.
DGG:sdc

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