Effect of Chronic and Experimental Jaw Muscle Pain on Pain-Pressure Thresholds and Stimulus-Response Curves

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Pain-pressure thresholds (PPTs) and stimulus-response (S-R) curves in masseter muscles and index fingers of 11 female patients with chronic jaw-muscle pain were compared with that of 11 matched control subjects. Experimental hyperalgesic and hypoalgesic conditions in the masseter muscles of control subjects were induced by intramuscular injection of 5% saline and of local anesthetic, respectively. The PPTs were found to be significantly lower in the masseter muscles of pain patients than in those of control subjects. The mean slopes of the S-R curves were significantly steeper for the masseter muscles of pain patients (0.481 ± 0.213) than of control subjects (0.274 ± 0.201, P < .0256). There were no statistically significant differences in PPTs or S-R curves for the index finger. The PPTs in masseter muscles of control subjects were not significantly affected by injection of 5% saline; however, the slopes of the S-R curves for the masseter muscles were significantly steeper for saline-injection values compared to baseline values (21.7% ± 29.6%, P < .037). Injection of local anesthetic into masseter muscles of control subjects increased the PPTs significantly and reduced the slopes of the S-R curves significantly as compared to baseline values (–22.9% ± 34.6%, P < .0155). The present results suggest that PPTs and S-R curves are valuable tools for quantitative description of chronic and experimental jaw muscle pain.

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The pathophysiology of chronic myofascial pain and the mechanisms responsible for the transition from acute to chronic muscle pain are not understood.[1,2] Tissue trauma, overload, myositis, and intermittent claudication can sensitize muscle nociceptors, and these factors have been implicated as possible mechanisms of chronic muscle pain.[1] In addition to peripheral mechanisms, attention has recently focused on central neural mechanisms involving hyperexcitability and spontaneous activity of wide-dynamic-range neurons (WDR) and nociceptive-specific neurons in the brain stem and spinal cord.[1,3] This neuroplasticity alters the normal processing of nociceptive information, and consequently, pain thresholds may be lowered.[3,4] The masseter and temporal muscles of subjects with chronic jaw muscle pain have significantly lower pain-pressure thresholds (PPTs) than those of control subjects.[5–9] Recently this has been suggested to be compatible with the concept of central hyperexcitability and altered processing of nociceptive input.[5] Moreover, McMillan and Blasberg[6]

found that PPTs remained unchanged after trigger point injection with local anesthetic (LA) in patients with chronic jaw muscle pain. This could point to a continued sensitivity of muscle nociceptors reinforced by mechanical trauma from the needle[10] and/or a hyperexcitability of brain stem WDR neurons.[3]

A psychologic explanation of lowered PPTs in subjects with muscle pain could be a generalized overresponsiveness to peripheral stimuli, ie, hypervigilance.[11] Malow et al[12] found that PPTs were significantly lower in the fingers of patients with chronic myofascial pain than in those of control subjects. However, the control and patient groups were not age matched. This is a point of concern, since age and gender have been shown to affect PPTs.[13] Furthermore, PPTs were not measured in painful jaw muscles. The PPTs at both tender points and control points are generally lower in patients with fibromyalgia than in control subjects.[14] Therefore, it may be useful to measure PPTs in trigeminal and extratrigeminal regions in patients with chronic jaw muscle pain but without fibromyalgia to determine the extent of segmental PPT changes.

Stimulus-response (S-R) curves have been widely used to assess hypoalgesic and hyperalgesic cutaneous reactions but have not been used in the study of muscle pain.[4,15] Recently, Lavigne et al[16] provided experimental evidence of a linear relationship between pressure intensity and pain intensity measured on visual analog scales (VAS) in healthy subjects. The construction of S-R curves includes the advantage of applying a triangulation procedure in which pain patients match the stimulus intensity to their clinical pain.[17,18] However, it needs to be shown that a relationship exists between pressure intensity and VAS in subjects with muscle pain before the triangulation procedure can be used.[16]

Experimental pain induced by intramuscular injection of hypertonic saline offers the possibility to study basic effects of standardized painful stimulus in jaw muscles.[19–22] Sensory effects of experimental trigger points may then be compared with sensory experiences of subjects with chronic muscle pain.[23]

The aim of this study was to test the hypothesis that *(1)* subjects with chronic jaw muscle pain have lower PPTs in the masseter muscles but not in the fingers, *(2)* subjects with pain have steeper S-R curves in the masseter muscles, but not in the fingers, than do matched control subjects, and *(3)* S-R curves in the masseter muscles of control subjects can be modulated experimentally by injection of hypertonic saline and LA.

Materials and Methods

Subjects

Eleven female patients (mean age ± standard deviation [SD] 25.6 ± 2.3 years) and 11 female control subjects (mean age 25.3 ± 3.1 years) participated in the study, which had been approved by the local ethics committee. Each participant signed an informed consent according to the Second Helsinki Declaration.

Control subjects had no history of temporomandibular disorders (TMD) or other orofacial pain syndromes, and clinical examination revealed no muscle or temporomandibular joint (TMJ) pathosis according to the Research Diagnostic Criteria (RDC) described by Dworkin and LeResche.[24]

Pain patients were recruited from the waiting list at the Royal Dental College, Aarhus, Denmark. The possibility that pain patients suffered from fibromyalgia or a generalized myofascial pain syndrome was ruled out during the comprehensive history if the patients had no other complaints of chronic pain in the body except in the head and face. In brief, a myofascial TMD was defined as pain of muscle origin including a complaint of pain as well as pain associated with localized areas of tenderness to palpation in muscle.[24] Thus, inclusion criteria for pain patients was chronic (greater than 6 months) myofascial TMD with bilateral masseter muscles tender to palpation (RDC group I.a[24]). Patients with pain primarily of arthrogenous origin were excluded (RDC groups II and III[24]). However, TMJ sounds (clicking) without preauricular pain or tenderness to palpation were accepted. Radiographs were not used for the majority of pain patients.

The pain patients described their mean daily intensity of jaw pain on a 100-mm VAS with the left endpoint labeled "no pain at all" and the right endpoint labeled "pain as worse as it could be." Pain patients and control subjects had not taken any medication on the day of examination, and medical histories revealed no chronic disorders or malfunctions.

Pressure Algometry

An electronic pressure algometer (Somedic AB, Farsta, Sweden) was used. This algometer has been widely used and has previously been described in detail.[5,13,15,20,25–27] During measurements of PPT, the pressure application rate was kept constant at 30 kPa/s with use of visual feedback from a dis-

play. Horizontal light bars indicated whether the applied pressure rate was greater than or less than the preset rate of 30 kPa/s. The 6-mm probe (28 mm^2) was applied perpendicular to the central part of the masseter muscles midway between the upper and lower border and 1 cm posterior to the anterior border. The center of the pulpa of the index finger was stimulated on the dominant hand.[25] The sequence of pressure application to the measurement sites was randomized in a balanced way. During pressure stimulation, the subjects kept their teeth in the intercuspal position with a minimum of voluntary contraction in their jaw-closing muscles because increasing contraction levels have been shown to increase the PPT.[28] Subjects were seated in an upright position in a dental chair in a quiet room and were asked to focus their attention on the experimental task.

Pain-Pressure Thresholds. When the threshold was reached, subjects pushed a small thumb switch that froze the display. The PPT consisted of a pain-detection threshold (PDT) and a pain-tolerance threshold (PTOL). The PDT was defined as the amount of applied pressure (kPa) necessary for a subject to report pain; the PTOL was defined as the maximal pressure (kPa) a subject was willing to accept.[27] The PDT was measured three times with 1 minute between each stimulus. The PTOL was measured only once to minimize the occurrence of high-intensity pressure stimuli. Prior to PPT measurements, all subjects had tried pressure stimulation in the brachioradial muscle.

Stimulus-Response Curve. A constant pressure was applied for 5 seconds, and the pain intensity was scored on a vertical VAS with 100 equidistant steps that were displayed on a computer screen. The lower endpoint was labeled "no pain at all," and the upper endpoint labeled "pain as worse as it could be." The mean and the SD of the pressure during the 5 seconds was calculated by the computer. Five different pressure intensities in random order were applied with 2 minutes between successive stimuli. The pressure intensities varied from 39.5% ± 14.2% to 156.2% ± 40.3% for the individual PDTs.

Experimental Study in Control Subjects

The PPTs and S-R curves in control subjects were determined as described above and were compared with those of pain patients. In addition, jaw muscle pain was introduced in the control subjects by injection of 0.15 mL sterile 5% (hypertonic) saline into the deep masseter muscle by means of a 27-G hypodermic needle and disposable syringe. For comparison of muscles within a subject, the other masseter muscle was injected with 0.15 mL sterile 0.9% (isotonic) saline. Subjects were blinded to the type of saline being injected, and the order and side of saline injections were distributed equally among subjects. Before saline injection, the skin surface was anesthetized with 0.1 mL 1% mepivacaine, (Carbocaine, Astra, Södertälje, Sweden). Injection of saline was performed in the central part of the deep masseter muscle midway between the upper and lower borders and 1 cm posterior to the anterior border.[21] The needle was inserted to a depth where bony contact was made and then retracted about 2 mm before aspiration and injection. The bolus was injected over 10 seconds, and subjects rated the evoked pain intensity on an electronic 100-mm VAS for the next 5 minutes with their jaws at rest. A computer sampled the VAS signals every 3 seconds. The lower endpoint of the VAS was labeled "no pain at all," and the upper endpoint was labeled "pain as worse as it could be." The peak VAS score, the area under the VAS curve, the onset, the peak time, and the offset were determined on the VAS profiles. After pain had subsided, the subjects completed a Danish version of the McGill Pain Questionnaire (MPQ).[29] Pain-rating indexes of sensory, affective, evaluative, miscellaneous, and total dimension of the experienced pain, as well as the number of words chosen, were calculated.[30] Patterns of pain referral were drawn by the subjects on anatomic maps. The PPTs and S-R curves were measured 5 minutes after saline injection. Following these measurements, 0.5 mL sterile 1% mepivacaine was injected into the left and right deep masseter muscles in the same area that the saline had been injected (0.9% and 5%), and PPTs and S-R curves were measured. The session lasted 1.5 hours.

Statistics

Nonparametric statistics were used because Komolgorov-Smirnov tests failed to show normality for several data samples. Between-group values were analyzed with Mann-Whitney *U* tests, and within-group values were analyzed with Friedman's analysis of variance and Wilcoxon's rank sum tests. Spearman's rank correlation coefficient was used to test the relationship between VAS, duration of pain, and PPT in pain patients. Simple linear regressions were performed on the S-R data. For each subject, the individual regression line was described with respect to the slope, y-axis intercept, and coefficient of determination (R^2). The x-axis intercept was estimated from the linear

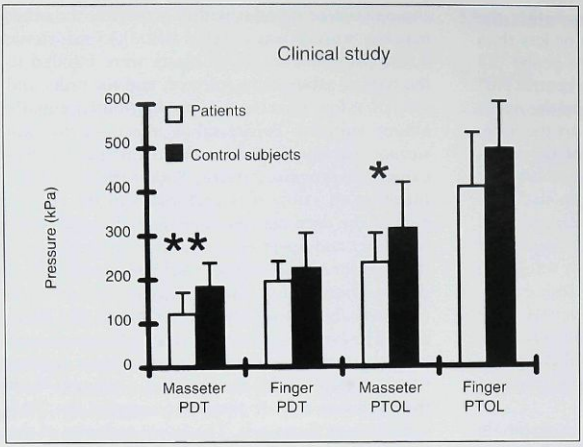

Fig 1 Mean values and SD of pain-detection thresholds (PDT) and pain-tolerance thresholds (PTOL) in masseter muscles and index fingers of patients with chronic jaw muscle pain (n = 11) and of control subjects (n = 11). Statistically significant differences between groups (Mann-Whitney: $*P < .05$; $**P < .01$).

relation. In addition, regressions were performed on mean pressure and mean VAS values from the groups. Statistical significance was accepted at $P < .05$ (two tailed).

Results

Clinical Study

The mean duration of jaw muscle pain in pain patients was 5.1 ± 4.9 years, and the mean daily pain intensity was 40.8 ± 19.7 mm measured on a 100-mm VAS. There were no statistically significant correlations between pain duration and PDT or PTOL (–.558, $P < .071$; –.489, $P < .116$) or between VAS and PDT or PTOL (–.252, $P < .545$; –.108, $P < .781$).

Pain-Pressure Threshold. Since no statistically significant side differences in PDT or PTOL were observed in the masseter muscles of the two groups, the data from the left and right masseter muscles were averaged. The PDT and PTOL in masseter muscles of pain patients were statistically lower than in control subjects ($P < .00862$; $P < .0356$) (Fig 1). However, PDTS and PTOLs in the finger were not statistically different between groups ($P > .341$; $P > .101$) (Fig 1).

Stimulus-Response Curves. Linear correlations between pressure intensity and VAS scores were found both in the masseter muscle and in the finger of pain patients and control subjects (Fig 2). Since no statistically significant side differences were observed in the two groups, mean values of the data from the left and right masseter muscles were calculated. The slopes of the S-R curves in the masseter muscles of pain patients (0.481 ± 0.213) were significantly steeper than in control subjects (0.274 ± 0.201, $P < .0256$), and the estimated intercept on the x-axis was shifted significantly to the left for masseter muscles of pain patients (Table 1, Fig 2). There were no statistically significant differences in the finger.

Experimental Study in Control Subjects

Pain-Pressure Threshold. Both PDTs and PTOLs were statistically different in the three conditions (baseline, after saline, after LA) in control subjects (Friedman: $P < .0022$) (Fig 3). The PDTs and PTOLs were not affected significantly by injection of 5% saline or by 0.9% saline, except PTOL, which was significantly increased by injection of 0.9% saline ($P < .045$) (Fig 3). However, the change in PDT and PTOL (after saline, baseline) was significantly lower for the 5% saline injection (–6.2 ± 37.7 kPa; –19.1 ± 63.8 kPa) than for 0.9% saline injection (25.3 ± 38.4 kPa; 48.5 ± 59.8 kPa) ($P < .029$; $P < .036$). Intramuscular injection of LA after 0.9% and 5% saline injections increased both PDTS and PTOLs significantly (Fig 3).

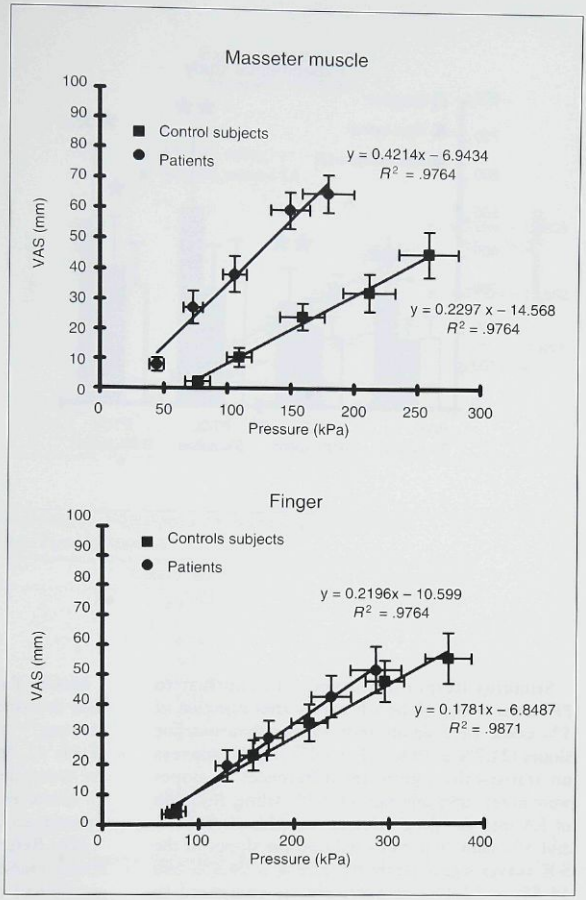

Fig 2 Linear regressions of mean pressure (± standard errors of mean [SEM]) and mean VAS scores (± SEM) from masseter muscles and index fingers of patients with chronic jaw muscle pain (n = 11) and of control subjects (n = 11).

Table 1 Analysis of Individual Regression Lines in Patients With Chronic Jaw Muscle Pain (n = 11) and Control Subjects (n = 11)

	Pain patients	Control subjects	P^*
Masseter			
Slope	0.481 ± 0.213	0.274 ± 0.201	.0256
Y-intercept	–6.8 ± 13.1	–14.7 ± 14.2	NS
X-intercept	12.6 ± 45.9	62.1 ± 40.0	.0181
R^2	.867 ± .081	.864 ± .103	NS
Finger			
Slope	0.227 ± 0.195	0.172 ± 0.085	NS
Y-intercept	–11.1 ± 13.6	–6.6 ± 9.2	NS
X-intercept	47.0 ± 55.9	37.5 ± 57.5	NS
R^2	.887 ± .085	.941 ± .045	NS

*Mann-Whitney *U* test; NS = not significant.

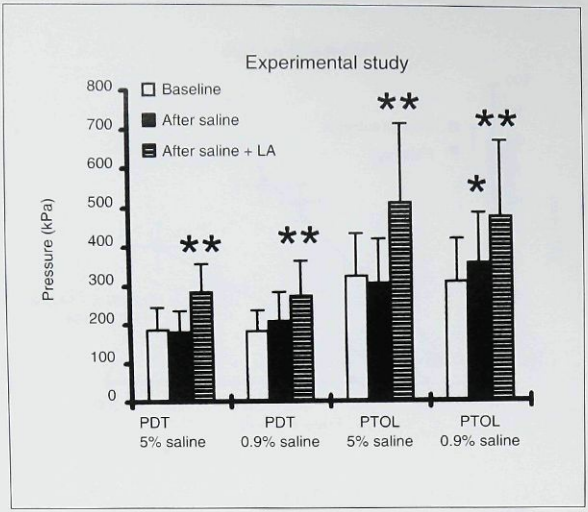

Fig 3 Mean values and SD of pain-detection thresholds (PDT) and pain-tolerance thresholds (PTOL) in masseter muscles of control subjects (n = 11) after injection of 5% or 0.9% saline and after injection of local anesthetic (LA). Statistically significant different from baseline values (Wilcoxon: $^{*}P < .05$, $^{**}P < .01$).

Stimulus-Response Curves. In contrast to PPTs, the slopes of the S-R curves after injection of 5% saline were significantly steeper than baseline slopes (21.7% ± 29.6%, $P < .037$) (Fig 4), whereas no statistically significant differences in slopes were noted after injection of 0.9% saline. Injection of LA into the deep masseter muscles after 0.9% and 5% saline injections reduced the slopes of the S-R curves significantly by 22.9% ± 34.6% and 34.5% ± 32.4%, respectively, as compared to baseline slopes ($P < .0155$; $P < .045$) (Fig 4).

Sensory Characteristics

Visual Analog Scale Profile. In control subjects, the mean peak VAS after 5% saline injection was 53.4 ± 18.2 mm with a mean peak time at 71.5 ± 27.4 seconds. The mean onset time was at 11.7 ± 5.8 seconds, and offset at 223.3 ± 81.3 seconds. The mean area under the VAS curve was 719 ± 302 arbitrary units (cm × s). Seven of 11 control subjects found the injection of 0.9% saline to be completely pain free. The mean peak VAS after 0.9% saline injection was 3.0 ± 6.1 mm, and the mean area under the VAS curve was 23 ± 60 units.

McGill Pain Questionnaire. The control subjects described the experimental muscle pain as a "boring," "shooting," "taut," and "intense" pain (Table 2). Muscle-pain patients more often chose the word descriptors "taut" and "tiring" (Table 2). However, no statistically significant differences between groups could be detected (Table 2).

Pain Referral Patterns. In addition to bilateral tender masseter muscles, tender areas (latent trigger points) on palpation were found in the anterior temporal (5 of 11), posterior temporal (5 of 11), medial pterygoid (4 of 11), and sternocleidomastoid (6 of 11) muscles in pain patients. Only one pain patient experienced referred pain when the sternocleidomastoid muscle (active trigger point) was palpated. The pain was referred to the ipsilateral masseter and temporal muscles. In contrast to the few active trigger points found in the patient group, the control subjects (10 of 11) consistently experienced referred pain and spreading of pain after 5% saline injection. The pain was always felt locally (11 of 11) but also was radiating anteriorly to the site of injection (seven of 11). In addition, the pain was referred to the posterior maxillary teeth (4 of 11) and spread toward the TMJ (2 of 11) and toward the eye (1 of 11).

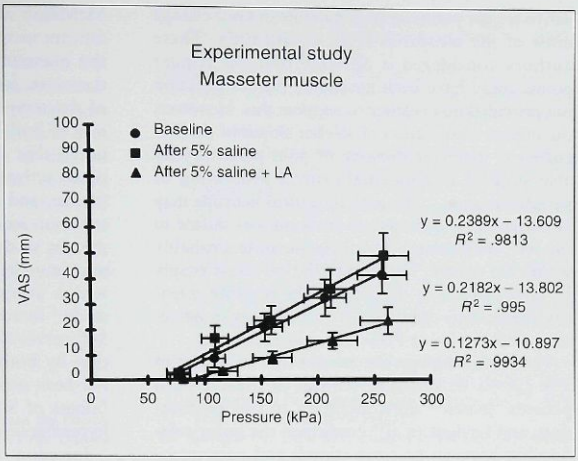

Fig 4 Experimental modulation of linear regressions of mean pressure (± SEM) and mean VAS scores (± SEM) from masseter muscles of control subjects (n = 11).

Table 2 McGill Pain Questionnaire Description of Saline-Induced Pain in Control Subjects and Clinical Pain in Patients*

	Control subjects	Pain patients
Sensory pain-rating index	9.0 (4.8)	5.8 (4.6)
Boring	4/11	2/11
Shooting	4/11	1/11
Taut	4/11	6/11
Affective pain-rating index	1.0 (1.4)	1.7 (1.7)
Tiring	1/11	5/11
Evaluative pain-rating index	1.3 (1.4)	0.9 (1.2)
Intense	4/11	1/11
Miscellaneous pain-rating index	2.4 (3.2)	2.6 (3.3)
Total pain		
Pain-rating index	12.8 (9.6)	10.9 (9.7)
No. of words chosen†	5.6 (3.3)	5.1 (4.3)

*Mean values with SD in parentheses.
†Frequency of words chosen by at least 30% of patients or control subjects.

Discussion

Clinical Study

The present study population was completely matched with respect to age and gender. This is important because pain-pressure thresholds (PPTs) are affected by these variables [13,25,27,31] Although the effect of the ovulatory cycle on PPT is not known, it has been shown that the sensitivity to other painful stimuli is greatest during the ovulatory phase.[32] Furthermore, birth control pills and dysmenorrhea also influence the pain sensitivity.[33] However, it is very unlikely that such factors could account for the observed differences in the masseter muscles of pain patients and control subjects because there were no differences for the fingers.

There were no statistically significant side differences between masseter muscles, which was in agreement with Reid et al.[5] The lower PPTs in masseter muscles of jaw muscle-pain patients than in control subjects has also been demonstrated in several other studies with use of different pressure algometers.[5–9] Thus, the present results supported the validity of the Somedic pressure algometer. Reid et al[5] suggested that lower PPTs indicated a hyperexcitability of central neurons in the brain stem of chronic muscle-pain patients. This concept was partly supported by the McMillan and Blasberg study,[6] in which injection of LA into

active trigger points in pain patients did not change most of the measured PPTs significantly. These authors considered it unlikely that the trigger points could have been missed by the LA injection but provided no evidence to support this. However, the present observation of steeper slopes of the S-R curves in masseter muscles of pain patients may also suggest an abnormal central processing of peripheral input.[4] The gain of central neurons may be increased, and the x-axis intercept was shifted to the left, indicating a hyperalgesic state probably within the muscle. The shift to the left could simply be interpreted as a decrease in the pressure intensity required to elicit pain,[4,15] which was in agreement with the lower PDTs in pain patients.

To our knowledge, we are the first to report of S-R curves of pressure stimuli in muscle-pain patients. Jensen[15] drew attention to this relationship, and Lavigne et al[16] confirmed the linear relationship between pressure stimuli and pain intensity in healthy subjects. We found similar high correlations in pain patients and control subjects, which indicated that both groups reliably can estimate the intensity of pressure stimuli. Thus, the S-R curves may provide the basis for a triangulation procedure.[17,13] It has been shown that orofacial pain patients scale experimental pain and their clinical pain in an internally consistent manner.[34,35] Thus, triangulation may also be a useful technique in the study of chronic muscle pain patients.

The present results did not support the hypervigilance theory[11] because PPTs and S-R curves in the index finger were not significantly different in jaw muscle pain patients and control subjects. It would have required at least 100 subjects in each group to detect a significant PDT difference in the finger (see Fig 1), provided a type I and type II error of 0.05 and 0.20, respectively. Thus, central neuroplasticity seems to have occurred at a distinct segmental level (brain stem) in jaw muscle pain patients in contrast to patients with fibromyalgia. Myofascial pain and fibromyalgia are clearly interrelated,[36] but in fibromyalgia, PPTs are lowered both at tender points and at control points,[14] which suggests a widespread neuroplasticity and/or a generalized overresponsiveness to peripheral stimuli.

Experimental Muscle Pain in Control Subjects

Jensen and Norup[20] have previously shown that baseline PPTs in the temporal muscle were not significantly different from PPTs measured 4 to 5 minutes after injection of hypertonic saline, which was in agreement with our present PPT results. McMillan and Blasberg[6] noted a statistically significant increase in PPTs after injection of LA into the masseter muscles of control subjects. Furthermore, Jensen et al[26] found an increase in PPTs of the temporal muscle after subcutaneous injection of both lidocaine and 0.9% saline. It must be noted that PPTs are normally results of a combined activation of afferents from the skin, muscle fasciae, and periosteum and that the specific contribution from different tissues is unknown. In the present study, injection of LA into the deep masseter muscle also increased the PPTs significantly, which supported the McMillan and Blasberg study.[6] In addition, we found that the slopes of the S-R curves were sensitive to experimental modulation by both hypertonic saline and LA. This has not been shown before and may illustrate the usefulness of S-R curves to describe hypoalgesic and hyperalgesic conditions within the jaw muscles.

Injection of hypertonic saline has been used to study sensory aspects of jaw muscle pain.[19,22] In the present study, experimental jaw muscle pain was found to be similar to clinical jaw muscle pain, in agreement with the findings of Stohler and Lund.[22] Both types of muscle pain were primarily described as "taut," and there were no statistically significant differences in MPQ scores. However, the lack of statistical differences should be interpreted cautiously because of the relatively small sample size. Pain patients tended to describe their clinical pain as more tiring but less intense than did control subjects. This may be related to the different durations of pain symptoms.[22]

The pain intensity caused by 5% saline injection (peak VAS 53.4 ± 18.2 mm) in the control subjects was higher than the patients' daily pain level (40.8 ± 19.7 mm). The latter level was within the limits of previous reports of muscle-pain patients.[37] The greater experimental pain intensity may be the reason why 10 of 11 control subjects experienced referred pain and spreading of pain to characteristic areas in the orofacial region.[10]

The importance of anesthetizing the skin prior to intramuscular saline injection has been pointed out by Wall.[38] As the needle penetrates the skin and the subcutaneous layers, saline may leak up the needle track and activate cutaneous nociceptors. Stohler et al[21] and Stohler and Lund[22] have described saline-induced jaw muscle pain in detail, but they have not anesthetized the skin prior to injection. However, their VAS profiles and our VAS profiles were similar with respect to onset, offset, and peak VAS, which may suggest little or no contribution from cutaneous nociceptors to the perceived pain intensity.

Conclusions

The present study has shown that PPTs and S-R curves were statistically different in masseter muscles but not in the index fingers of chronic jaw muscle pain patients as compared to matched control subjects. Furthermore, the slopes of the S-R curves in masseter muscles of control subjects could be increased by 5% saline injection and decreased by injection of local anesthetic. The combined use of PPTs and S-R curves may provide complementary information of hypoalgesic and hyperalgesic conditions in jaw muscles.

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Resumen

El efecto del dolor muscular mandibular crónico y experimental sobre los umbrales de la presión-dolor y las curvas de la respuesta al estímulo

Se compararon los umbrales de presión-dolor (UPD) y las curvas de la respuesta al estímulo (R-E) en los maseteros y dedos índices de 11 pacientes del sexo femenino que padecían de dolor muscular mandibular crónico, con aquellos de 11 sujetos de control correspondientes. Se indujeron condiciones experimentales de hiperalgesia e hipoalgesia en los maseteros de los sujetos de control, por medio de inyecciones intramusculares de solución salina al 5% y de anestésicos locales, respectivamente. Los UPD registrados fueron significativamente menores en los maseteros de las personas del grupo experimental, en comparación al grupo de control. Las inclinaciones medias de las curvas R-E fueron significativamente mas empinadas en los músculos maseteros del grupo experimental (0,481 ± 0,213), en comparación al grupo de control (0,274 ± 0,201, $P <$ 0,0256). No se observaron diferencias estadísticamente significativas en los UPD o las curvas de R-E en relación al dedo índice. Los UPD de los maseteros del grupo de control no estuvieron afectados significativamente por la inyección de solución salina al 5%; sin embargo, las inclinaciones de las curvas R-E en los maseteros estuvieron significativamente mas empinadas con la inyección de solución salina, en comparación a los valores iniciales (21,7% ± 29,6%, $P <$ 0,037). La inyección de anestésicos locales en los músculos maseteros de los sujetos de control aumentó los UPD significativamente y redujo las inclinaciones de las curvas S-R significativamente, en comparación a los valores iniciales (–22,9% ± 34,6%, $P <$ 0,0155). Los resultados presentes indican que los UPD y las curvas de R-E son instrumentos útiles para la descripción cuantitativa del dolor muscular mandibular crónico y experimental.

Zusammenfassung

Der Einfluss von chronischem und experimentellem Kaumuskelschmerz auf Druckschmerzschwellen und Reiz-Antwort Kurven

Die Druckschmerzschwellen und die Reiz-Antwort Kurven von Massetermuskeln und Zeigfingern von 11 Patientinnen mit chronischen Schmerzen in den Kaumuskeln wurden verglichen mit denjenigen von 11 entsprechenden Kontrollsubjekten. Durch intramuskuläre Injektion von 5% Kochsalzlösung resp. Lokalanaesthetikum wurden bei den Kontrollsubjekten experimentell hyper- resp. hypalgetische Bedingungen geschaffen. Die Druckschmerzschwellen waren signifikant tiefer in den Masseteren der Schmerzpatienten als in denjenigen der Kontrollsubjekte. Die Abhänge der Reiz-Antwort Kurven waren für die Masseteren der Schmerzpatienten (0,481 ± 0,213) signifikant steiler als für diejenigen der Kontrollsubjekte (0,274 ± 0,201, $P <$ 0,256). Es gab keine signifikanten Unterschiede der Druckschmerzschwellen und Reiz-Antwort Kurven der Zeigfinger. Die Druckschmerzschwellen der Masseteren der Kontrollsubjekte wurden nicht signifikant beeinflusst durch die Injektion von 5% Kochsalzlösung, die Abhänge der Reiz-Antwort Kurven waren jedoch signifikant steiler für Werte nach Injektion von Kochsalzlösung als für Grundwerte (21,7% ± 29,6%, $P <$ 0.037). Die Injektion von Lokalanaesthetikum in die Masseteren der Kontrollsubjekte liess die Druckschmerzschwellen signifikant ansteigen und reduzierte die Steilheit der Abhänge der Reiz-Antwort Kurven im Vergleich zu den Grundwerten signifikant (–22,9% ± 34,6%, $P <$ 0,0155). Aufgrund der vorliegenden Resultate sind Druckschmerzschwellen und Reiz-Antwort Kurven valable Instrumente zur quantitativen Beschreibung von chronischen und experimentellen Kaumuskelschmerzen.