Muscle Pain Modulates Mastication: An Experimental Study in Humans

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In this study, pain was induced in the masseter muscle by tonic infusion of hypertonic saline (5%) for up to 800 seconds in 12 healthy men. Subjects continuously scored the pain intensity on a 10-cm visual analogue scale. Mastication ipsilateral and contralateral to the infusion side was quantitatively assessed with the use of jaw-tracking and electromyograph recordings of jaw-closing muscles before, during, and after periods of constant muscle pain intensity. The maximum voluntary occlusal force (MVOF) during short static contractions also was monitored. Jaw movements and electromyographic data were divided into single masticatory cycles and analyzed on a cycle-by-cycle basis to account for intercycle variability. In all subjects, tonic infusion caused a deep localized pain at a clinically relevant intensity (mean VAS ± SE, 4.6 ± .3 cm). MVOF was significantly affected by muscle pain ($P < .0005$), with significantly lower MVOF during pain compared to prepain and postpain ($P < .05$). In a significant number of masticatory cycles, the averaged electromyograph activity of all jaw-closing muscles during their agonist function was decreased for both ipsilateral and contralateral painful mastication ($P < .05$). These electromyographic changes are probably a reflection of the natural bilateral recruitment pattern of jaw-closing muscles during mastication. Significant changes in jaw movements during painful mastication could not be detected with the present jaw-tracking device, but further studies with more accurate and sensitive devices are needed.
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Studying cause-effect relationships in painful temporomandibular disorders (TMD) is not a trivial task. To explain the clinical effect of myogenous TMD pain on jaw motor performance, it was recently suggested that activity in nociceptive afferents, via local circuits in the brain stem, could cause facilitation of inhibitory pathways to the agonist motorneuron pool and of excitatory pathways to the antagonist motorneuron.[1–3] The consequences of such neural circuits would be reduction of the agonist electromyographic (EMG) burst and an increase of the antagonist EMG burst, which in turn could cause slower movements with smaller amplitudes and, presumably, avoid further damage to the system. Schwartz and Lund[4] presented experimental data from decerebrated rabbits to support this hypothesis; they showed that fictive mastication during noxious pressure stimulation of the zygoma was associated with smaller EMG bursts of the jaw-closing muscles and with smaller and slower jaw movements. Svensson et al[5]

showed that an acute bolus injection of hypertonic saline into the masseter muscle caused similar changes in the mean profiles of human mastication patterns. Thus, experimental animal and human pain models may be used to gain some insight into the basic effects of muscle pain on motor function. However, it has been argued that changes in motor performance observed during acute saline-induced pain can be difficult to interpret because the intensity of the pain is not constant.[6] Furthermore, tonic infusion of hypertonic saline may produce muscle pain that is more comparable to clinical pain than acute bolus injections.[6] The choice of experimental pain model may therefore be important.

The next problem is to provide a quantitative description of jaw motor function. Many studies have described masticatory patterns in animals and in humans (for a review, see Lund[7]). One conclusion from the reviewed studies was that mastication is characterized by a considerable cycle-to-cycle variability. This variability probably reflects a continuous adjustment of a rhythmic function by sensory feedback generated by the movement and the bolus.[7] The jaw-closing muscles are activated bilaterally during mastication, reflecting a substantial degree of functional symmetry even though side-to-side differences also are apparent in the natural masticatory patterns.[8,9] The variability of human mastication has not yet been taken into account when the effect of experimental muscle pain has been studied.

The aims of the present study were to determine the effect of constant experimental muscle pain on jaw motor function, performed as mastication both ipsilateral and contralateral to the infusion side, and to extend the analysis of the mastication patterns to a cycle-by-cycle level. The final aim was to study the influence of muscle pain on isometric jaw motor function.

Materials and Methods

Subjects

Twelve healthy, unmedicated men (mean age 24.4 ± 1.0 years [SE]; range 18 to 31 years) were recruited for the study from among the students at Aalborg University. (Only men responded to the advertisement in the university paper.) The absence of TMD in these subjects was verified by screening procedures described by the American Academy of Orofacial Pain.[10] All subjects were fully dentate, with the possible exception of third molars. Informed consent, according to the Helsinki-II Declaration, was obtained from all subjects prior to study entry. The study was approved by the local Ethics Committee. Subjects received $50 for their participation.

Jaw Movement Recordings

A Sirognathograph (Siemens, Munich, Germany) was used to track jaw movements in a vertical axis (z), a lateral axis (y), and an anteroposterior axis (x).[11] Since the output of the Sirognathograph has been shown to be distorted,[12,13] the performance of the present system was tested in three dimensions. A lightweight magnet (1 g) was centered (0, 0, 0) in a three-dimensional stereotaxic bench that allowed placement in 7 (x axis) × 7 (y axis) × 17 (z axis) standardized positions. The x, y, and z axes spanned 48 mm, 40 mm, and 52 mm, respectively. The output signal of the Sirognathograph was recorded for each of the corresponding 833 positions. The actual position of the magnet in three-dimensional space was then related to electrognathographic (EGG) signals, and the distortion could be compensated by a computer algorithm. Within a radius of 15 mm from the center of the aerial, the uncompensated distortion of the Sirognathograph is on average 5.9%.[13] Within the same range of movements and after compensation, the distortion in the present system was less than 3.5%. The EGG signals were recorded with the subject sitting upright in a chair of nonferromagnetic material with the Frankfort horizontal parallel to the floor.[12] With a conventional neck rest supporting the head, subjects were asked to fix their glance at a point on the wall and to avoid movements of the head during mastication. Before each recording, the output was zero-adjusted (0, 0, 0) in the maximum intercuspal position. The EGG signals were sampled at 1024 Hz (DT2801-A, Data Translation, Marlboro, MA).

Electromyographic Recordings

Self-fabricated bipolar surface silver electrodes, with an active area of 12 × 6 mm and with their long axes in parallel, were arranged 10 mm apart on a piece of adhesive tape and coated with conductive paste (DanTENS, Copenhagen, Denmark). The skin was cleansed with ethanol and the electrodes were placed with their long axes transverse to the main direction of the muscle fibers in the central part of the right and left masseter and anterior temporalis muscles. Electrode placement was based on palpation of the muscles during full effort, as previously described by Møller.[8] A saline-soaked ground electrode was wrapped around the neck. The EMG signals were

amplified differentially (5,000 to 20,000 times) (Disa 15C01, Copenhagen, Denmark), filtered (20 to 500 Hz), sampled at 1024 Hz, digitized, and stored. Subjects were asked to make a forced choice of their preferred chewing side. The test food consisted of one piece of chewing gum (1.5 g, Sorbits), which was softened for 3 to 4 minutes. Three orthodontic elastics (Energy Pak Elastics, 280) were then introduced into the bolus to give it a constant consistency.

Recordings of Maximum Voluntary Occlusal Force

A U-shaped occlusal force meter (Aalborg University, Aalborg, Denmark), based on a double-strain gauge principle, was used to measure the maximum voluntary occlusal force (MVOF). To protect the teeth, the occlusal area (1.1 × 1.1 cm) was covered with plastic tubes. The total height of the occlusal force meter with plastic tubes was 12 mm. The particular features of this apparatus were insensitivity to variations in temperature and placement of loading on the occlusal area. Calibration curves showed a strong linear relation between standardized loading and readings up to 1000 N (coefficient of determination, $R^2 = 0.9999$). Subjects were instructed to clench their teeth as hard as they could for 3 to 4 seconds, while MVOF was measured between the second premolars on both sides. Verbal encouragement was given to obtain the maximum effort, but no visual feedback was provided. The MVOF was determined as the peak value and stored on a display. This was repeated at least three times. About 15 to 30 seconds elapsed between repeated measurements. The average of the three peak MVOF measurements for each side was used for further calculations.

Induction of Jaw Muscle Pain

A plastic catheter inside a hypodermic needle (Venflon, 22 g/25 mm, BOC Ohmeda AB, Helsingborg, Sweden) was inserted through the anesthetized skin (0.1 mL lidocaine) into the masseter muscle on the preferred chewing side (denoted ipsilateral), 1 cm anteriorly and 1 cm caudally to the EMG electrode. Placement deep in the muscle was secured by bone contact before the needle was fully retracted and the plastic catheter left in the muscle with the position secured by tape on the skin. The catheter was connected via a tube (extension set with polyethylene inner line, Ivac G303030, San Diego, CA) to a computer-controlled syringe pump (Ivac, model 770) with a 10 mL syringe.[14] A bolus injection of 0.2 mL 5% saline was given over 30 seconds and followed by a steady infusion at a rate of 66 μL/minute. This infusion rate was semiautomatically increased or decreased, depending on the subject's scores on a 10-cm electronic visual analogue scale (VAS). The lower end of the scale was marked "no pain" and the upper end was marked "the worst imaginable pain." The analogue signal from the VAS box was A/D converted and fed to the computer at a sampling frequency of 0.2 Hz. One investigator followed the VAS scores on a monitor and responded to changes by either increasing or decreasing the infusion rate in a stepwise procedure. A VAS between 3 and 5 was designated as the target level because it is comparable to the pain levels reported by a majority of TMD patients.[15] Based on pilot experiments in the authors' laboratory, a step was determined to be an increase or decrease of the infusion rate by a factor of 2. The change in infusion rate was initiated if the VAS scores in two consecutive samples (10 seconds) indicated increased or decreased pain intensity outside the target level. The correction of the infusion rate was repeated 30 seconds later if the VAS scores did not change. With use of this standard protocol, it was possible to keep a relatively constant pain intensity during recordings of mastication and MVOF. After completion of saline infusion, subjects were asked to fill out a Danish version of the McGill Pain Questionnaire[16] and to draw the distribution of the pain on anatomic maps.

Experimental Procedure

After insertion of the catheter, EGG and EMG signals during gum chewing ipsilateral and contralateral to the preferred side were recorded for 60 seconds (prepain series). The infusion of 5% hypertonic saline was then started. When the pain intensity was at a constant level (VAS from 3 to 5), two series of 60-second ipsilateral and contralateral mastication were recorded in random order followed by measurements of MVOF (pain series). The infusion was stopped, and sufficient time was allowed for the pain to disappear. When subjects had been pain-free for at least 10 minutes, recordings of ipsilateral and contralateral mastication and MVOF were repeated (postpain series). The session lasted about 2 to 3 hours.

Data Analysis

In the present study, the analysis of the EGG signals was focused on the maximum displacements in three dimensions during mastication. These jaw movements normally have a frequency of about 1 to 2 Hz. Accordingly, the EGG signals were smoothed with a low-pass finite impulse response filter (filter order = 20; cut-off frequency = 10 Hz)

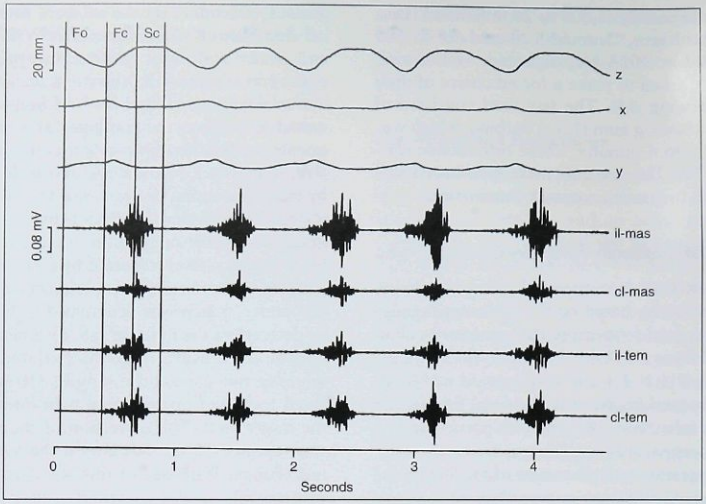

Fig 1 Recordings of jaw movements in vertical (z), anteroposterior (x), and lateral (y) directions, and EMG activity of ipsilateral and contralateral masseter (il-mas, cl-mas) and anterior temporalis (il-tem, cl-tem) muscles. Masticatory cycles were divided into fast-opening (Fo), fast-closing (Fc), and slow-closing (Sc) occlusal phases.

because no attempt was made to study physiologic tremor, which has a peak frequency of about 10 Hz.[17] The vertical position was differentiated with respect to time and provided an estimate of the vertical velocity. This was used to determine the onset of the jaw-opening phase, defined as the zero-crossing velocity where the velocity changed from positive (jaw-closing) to negative (jaw-opening). When the mean velocity in a moving 20-sample window was above a defined threshold of 10 mm/second, an onset was marked.

Attempts were made to subdivide the closing phase into fast-closing and slow-closing (occlusal) phases similar to the detailed descriptions of animal masticatory patterns provided by Schwartz et al.[18] The fast-closing phase in rabbit mastication corresponds to a swift upward movement, whereas during the slow-closing phase the mandible either stops moving vertically or is closed slowly as the food is crushed.[18] In the present authors' experience, the distinction between a fast- and a slow-closing phase was less clear in human than in animal mastication, and modified criteria therefore had to be applied. In the present study, the onset of the slow-closing phase was defined as the time point at which the mean vertical velocity in the moving 20-sample window was below 10 mm/second. Thus, the slow-closing phase was essentially the onset of an occlusal phase even though the mandible may not have entered the maximum intercuspal position as a result of the unbreakable elastics in the chewing gum. The onset of the fast-closing phase was determined as the maximum vertical value (negative) during jaw-opening. The period from one jaw-opening to the next jaw-opening was termed a masticatory cycle (Fig 1).[18] The jaw-opening phase corresponds to the antagonist phase and the jaw-closing phase to the agonist phase of the jaw-closer muscles.

The EMG and EGG signals were divided into single masticatory cycles, which were analyzed on a cycle-by-cycle basis. Since subjects were instructed to avoid swallowing, all masticatory cycles could be detected and included in the analysis. A total of 49 consecutive and unselected masticatory cycles were analyzed for all subjects. Because of constraints in the statistical software, no more than 49 cycles could be included in the repeated measurement analysis. For each masticatory cycle, the following parameters were calculated: maximum displacement in three dimensions (mm); maximum velocities during jaw-opening and fast-

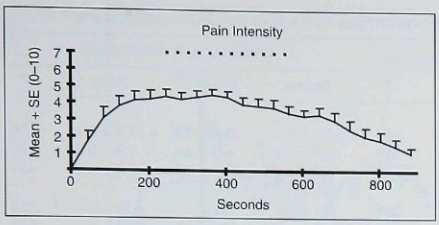

Fig 2a Pain intensity induced by tonic infusion of hypertonic saline in 12 subjects (mean ± SE). An electronic 0 to 10 cm visual analogue scale was used. The horizontal stippled line indicates the mean period with recordings of mastication.

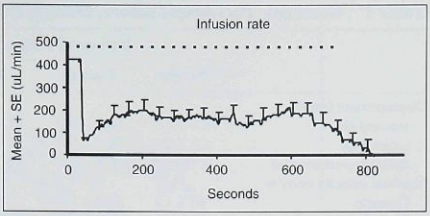

Fig 2b Infusion rate of hypertonic saline measured by the microinfusion pump during the experiment (mean ± SE). The horizontal stippled line indicates the mean infusion period.

closing phases (mm/second); duration of the opening, fast-closing, and slow-closing phases (milliseconds); and the root-mean-square (RMS) amplitude (μV) of the rectified EMG activity in the opening and closing (fast + slow) phase (Fig 1).

Statistics

The data were analyzed using the Statistica software (Statsoft 5.1, Tulsa, OK). Mean values ± SE are presented in the text and figures. Multivariate analysis of variance (MANOVA) with repeated measures was used to analyze the effect of the factors: experimental condition (prepain, pain, postpain), masticatory cycle (49 levels), and muscles (4). The kinematic parameters were analyzed with respect to experimental condition (3) and masticatory cycle (49 levels). The factors in the analysis of MVOF were experimental condition (3) and side of bite (2). Student-Neuman-Keuls (SNK) post-hoc tests were used to compensate for multiple comparisons. Significance was accepted at $P < .05$.

Results

Subjective Description of Experimental Masseter Pain

All subjects experienced deep, local pain originating from the site of infusion in the masseter muscle. The mean VAS-time curve is shown in Fig 2a. The mean pain intensity across subjects was 4.6 ± .3 cm during recording of ipsilateral mastication, and 3.9 ± .4 cm during recording of contralateral mastication. These VAS scores were not significantly different (paired t test: $P > .05$). The changes in mean infusion rate during the course of the experiment are shown in Fig 2b. The infusion rate increased from 66 μL/minute up to about 200 μL/minute, with a total infusion volume of 2.3 ± .2 mL saline (Fig 2b). The local pain was associated with a spread of pain to adjacent areas: the temple, the TMJ area, the maxillary molar teeth, and the basis of the mandible (Fig 2c).

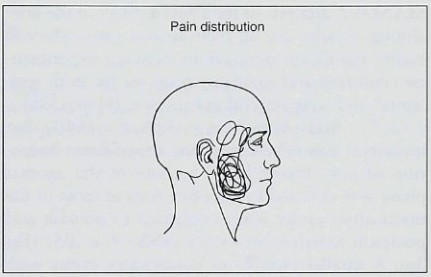

Fig 2c Distribution of self-perceived pain during tonic infusion of hypertonic saline into the masseter muscle. Tracings of original pain drawings from each subject.

The McGill Pain Questionnaire (MPQ) mean pain rating indices of sensory, affective, evaluative, and miscellaneous dimensions of pain were 10.3 ± 1.3, 2.1 ± .7, 2.5 ± .3, and 5.3 ± .9, respectively. Among the words chosen from the MPQ by at least 40% of the subjects were "taut" (67%), "drilling" (42%), "intense" (42%), and "penetrating" (42%).

Motor Consequences of Experimental Masseter Pain

According to the EMG data, there were no significant main effects of experimental condition, muscle recording site, or masticatory cycles. However, the

Table 1 Kinematic Parameters Before, During, and After Induction of Pain in the Masseter Muscle*

	Ipsilateral mastication				Contralateral mastication			
	Before	During	After	*P*	Before	During	After	*P*
Displacement (mm)								
Vertical (z)	16.4 ± 1.1	16.4 ± 0.9	17.4 ± 0.9	.076	16.5 ± 1.1	16.0 ± 0.9	17.3 ± 0.7	.166
Lateral (y)	4.6 ± 0.5	4.3 ± 0.4	4.5 ± 0.4	.410	4.8 ± 0.5	4.5 ± 0.4	4.9 ± 0.5	.440
Anterior-posterior (x)	2.1 ± 0.2	2.4 ± 0.3	2.2 ± 0.2	.195	2.3 ± 0.2	2.5 ± 0.2	2.3 ± 0.2	.431
Maximal velocity (mm/s)								
Opening	93 ± 12	92 ± 12	106 ± 13	.065	91 ± 10	93 ± 11	112 ± 13	.039
Fast-closing	99 ± 10	96 ± 9	107 ± 11	.159	98 ± 10	96 ± 9	107 ± 10	.099
Duration (ms)								
Opening	361 ± 24	369 ± 22	335 ± 21	.175	361 ± 24	342 ± 19	322 ± 23	.055
Fast-closing	349 ± 17	357 ± 21	340 ± 19	.470	340 ± 19	334 ± 18	332 ± 24	.825
Slow-closing	124 ± 12	97 ± 10	106 ± 10	.053	145 ± 19	120 ± 12	129 ± 17	.364

*The mean values ± SE of 49 masticatory cycles from 12 subjects are presented. *P* values from the ANOVA indicate the effect of experimental condition.

MANOVA analysis of the EMG activity of the jaw-closing muscles during their agonist phase showed highly significant interactions between experimental condition and masticatory cycles for both ipsilateral and contralateral mastication ($F[96,6336] = 2.12$, $P < .0001$; $F[96,6336] = 3.08$, $P < .0001$). For ipsilateral painful mastication, a significant reduction of jaw-closing EMG activity in the agonist phase was detected by post-hoc tests in most of the masticatory cycles when compared to prepain and postpain masticatory cycles (SNK: $P < .05$) (Fig 3a). A smaller number of masticatory cycles with significantly reduced EMG activity was found for contralateral painful mastication (Fig 3b).

There were no significant main effects of experimental condition on the various kinematic parameters except for the maximum opening velocity during contralateral mastication (Table 1). For this parameter, there was a significant interaction between experimental condition and masticatory cycles ($F[96,3072] = 1.71$, $P < .0001$), but post-hoc tests could not identify the masticatory cycles with significant changes.

The maximum occlusal force was significantly affected by experimental condition ($F[2,22] = 11.10$, $P < .0005$) but not by side of clench ($F[1,11] = 1.51$, $P = .24$). The MVOF was significantly lower during painful clenches (597 ± 19 N) as compared to prepain (618 ± 19 N; SNK: $P < .05$) and postpain clenches (650 + 21 N; SNK: $P < .05$).

Discussion

Subjective Description of Experimental Masseter Pain

The subjective description of jaw muscle pain induced by tonic infusion of hypertonic saline in healthy volunteers was quite similar to the description obtained from chronic myogenous TMD patients.[9] Frequently chosen words from the MPQ were the evaluative adjective "intense" and the sensory-miscellaneous adjectives "taut/tender." The Danish version of the MPQ has been validated,[16] and it supported the concept of experimental muscle pain as a multidimensional experience with sensory-discriminative, cognitive-evaluative, and affective components.[19] It may be possible to separate out an estimate of pain intensity and an estimate of unpleasantness[20]; however, ratings of these two dimensions of pain are clearly interrelated because both ratings increase with increasing stimulus intensity.[20–22] In the present study, no attempts were made to distinguish between pain intensity and unpleasantness; subjects simply used a VAS labeled with "worst pain imaginable." It has also been shown that the word descriptor "worst pain imaginable" on a VAS is the most suitable choice for measuring dental pain.[23]

The present infusion paradigm allowed periods with relatively stable pain ratings (Fig 2a), although more sophisticated infusion paradigms with feedback-control have been developed and should be preferred when possible.[24] A major advantage of the tonic infusion of hypertonic saline is that subjects avoid a very rapid change in pain intensity, and the induced pain mimics clinical pain more than after acute bolus injections of hypertonic saline.[25] Another similarity between experimental muscle pain and chronic TMD is the spread of pain to adjacent areas. Clinical and experimental pain in limb muscles is usually referred to areas distal to the region with local pain.[14,26–28] Pain in the masseter muscle is usually spread to posterior and cranial regions but rarely in an anterior direction.[5,6,26,29] The stereotyped patterns of referred

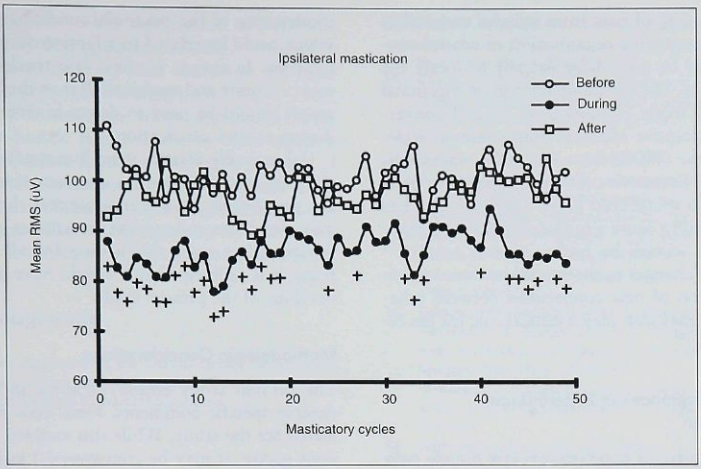

Fig 3a Effect of tonic infusion of hypertonic saline on the mean EMG activity from the masseter and anterior temporalis muscles in the jaw-closing phase during ipsilateral mastication. Recordings of 49 consecutive masticatory cycles before, during, and after infusion in 12 subjects. + indicates significant difference between the painful condition and the before and after condition (SNK: $P < .05$).

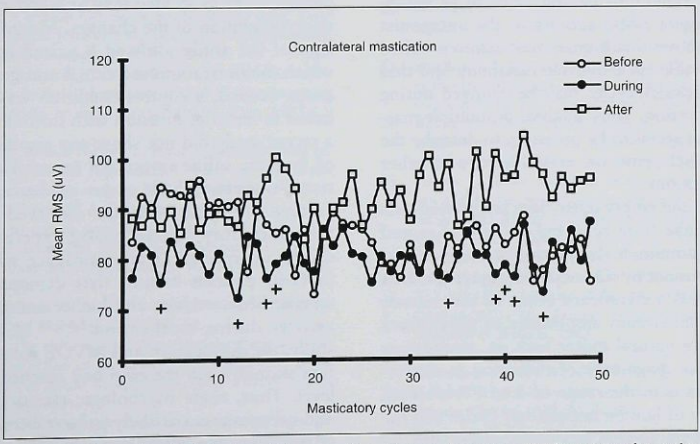

Fig 3b Effect of tonic infusion of hypertonic saline on the mean EMG activity from the masseter and anterior temporalis muscles in the jaw-closing phase during contralateral mastication. Recordings of 49 consecutive masticatory cycles before, during, and after infusion in 12 subjects. + indicates significant difference between the painful condition and the before and after condition (SNK: $P < .05$).

pain or spreading of pain from muscles may reflect a distinct topographic organization of central neurons and may be used diagnostically to locate the source of pain. The spread or referral of trigeminal pain is most likely explained by central convergence of nociceptive afferents onto common wide-dynamic-range (WDR) neurons in the subnucleus caudalis.[30–32] Extensive convergence has also been observed onto spinal cord WDR neurons[33] and has been suggested to cause a mislocalization of peripheral noxious stimuli by higher central centers.[34] Neuroplastic changes in the central nervous system with formation of new connections between adjacent neurons may also play a critical role for the referral of pain.[34]

Motor Consequences of Experimental Masseter Pain

The overall effect of experimental jaw muscle pain induced by acute bolus infusions is a reduction of agonist EMG activity.[5] In the present study, there was no statistical main effect of the experimental condition, but there were significant interactions between experimental condition and masticatory cycle, and hence a significant reduction of agonist EMG activity was seen in a majority of masticatory cycles during tonic infusion of hypertonic saline. Stohler et al[36] described masticatory cycles in TMD patients and found only painful cycles to be associated with higher EMG activity in the antagonist phase. This shows that human mastication is linked to a considerable cycle-to-cycle variability and that not all masticatory cycles may be changed during painful mastication. Thus, analysis of multiple masticatory cycles seems to be necessary to describe the effect of muscle pain on mastication and other rhythmic functions.

Symmetric and empty open-close jaw movements in humans have been reported to be slower and with smaller amplitude during experimental pain in the masseter muscle.[1] Our cycle-by-cycle analysis failed to reveal a significant effect of jaw muscle pain on the maximum amplitudes of movements during a more natural motor task, ie, mastication (Table 1). The magnitude of reduction in decerebrated rabbits is in the range of 3 to 7%[4]; taking the variability of human masticatory cycles and the limitations of the present recording system into account, this may partly explain the lack of statistical significance for kinematic parameters in the present study. It is likely that chewing on a larger bolus with larger movement amplitudes (> 20 mm) and a more intense pain would have caused significant effects. We observed a trend of faster and larger jaw movements in the postpain conditions (Table 1), which could be related to adaptive changes in mastication. In future studies, jaw-tracking devices more accurate and sensitive[37,38] than the Sirognathograph should be used to detect kinematic changes during painful mastication.

Unbreakable elastics were incorporated into the chewing gum to ensure a constant consistency of the test bolus. As a consequence, the mandible may not have entered the maximum intercuspal position, but since this is true for all the conditions, it is unlikely that it would have affected the outcome of the present study.

Methodologic Considerations

The present study employed some methods that deserve specific comments. First, only men volunteered for the study. While this enabled a homogenous group, it may be controversial in light of reported gender differences in masticatory patterns[17,39,40] and the female dominance among TMD patients.[41] However, women have lower pain thresholds to various stimuli,[42] which could suggest that tonic infusion of hypertonic saline would have been perceived as more painful in women than in men. Therefore, changes in jaw motor function during painful mastication could have been larger in a female population, and the present findings in men may at worst represent an underestimation of the changes. Moreover, the design of the study allowed a paired comparison, which minimizes interindividual and gender differences. Second, a control condition was not established in terms of infusion with isotonic saline, but a recent study did not show any significant effects of isotonic saline injections on masticatory patterns, suggesting little or no influence of infused volume.[43] Furthermore, the observed changes in the masticatory pattern during experimental pain can be interpreted as in accordance with findings in TMD patients because they demonstrate lower agonist EMG activity and higher antagonist EMG activity during mastication.[8,36,44] In the present study, the mastication and MVOF recordings were first started when the pain had reached a constant level. Thus, acute psychologic effects like anxiety and nervousness are likely to have been minimized at this time. However, all types of pain, including experimental pain, encompass psychologic as well as sensory-discriminative aspects, and it is therefore difficult in conscious humans to disregard the influence of higher brain centers on jaw motor function during pain. Nevertheless, the observed pain modulation of human mastication is in many

ways comparable to the modulation of rhythmic movements in decerebrated animals,[4] indicating the involvement of brain-stem circuits in the regulation of motor function during pain.[1]

Within the constraints of the present study, experimental jaw-muscle pain induced by tonic infusion of hypertonic saline caused a diminished capacity of the jaw-closing muscles to work against a load, which is in accordance with a functional adaptation to muscle pain. The biologic purpose of such adaptation may be to allow healing of an injured area.[1]

Acknowledgments

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Resumen

El dolor muscular modula la masticación: Estudio experimental en humanos.

En este estudio, el dolor fue inducido en el músculo masetero por medio de la infusión tónica de una solución salina hipertónica (5%) hasta de 800 en 12 hombres sanos. Los participantes registraron puntuaciones de la intensidad del dolor continuamente sobre una escala análoga visual (EAV) de 10 cm. La masticación ipsolateral y contralateral en el lado de la infusión fue evaluada cuantitativamente por medio del uso de registros electromiográficos y de rastreo mandibular de los músculos de cierre mandibular antes, durante, y después de los períodos, con una intensidad de dolor muscular constante. La fuerza oclusal voluntaria máxima (FOVM) durante las contracciones estáticas cortas también fue monitoreada. La información de los movimientos mandibulares y los registros electromiográficos fue dividida en ciclos masticatorios sencillos y analizada de ciclo a ciclo para tener en cuenta la variabilidad entre los ciclos. En todos los participantes, la infusión tónica causó dolor localizado profundo, con una intensidad relevante clínicamente (EAV media, ± desviación estándar, 4,6 ± 0,3 cm). La FOVM fue afectada significativamente por el dolor muscular ($P < 0,0005$) siendo la FOVM significativamente menor durante el dolor en comparación con antes y después del dolor ($P < 0,05$). En un número significativo de ciclos masticatorios, la actividad electromiográfica promedio de todos los músculos de cierre mandibular durante su función como agonistas disminuyó durante la masticación dolorosa, tanto ipsolateral como contralateral ($P < 0,05$). Estos cambios electromiográficos son probablemente una reflexión del patrón de reclutamiento bilateral natural de los músculos de cierre mandibular durante la masticación. No se pudieron detectar cambios significativos en cuanto a los movimientos mandibulares durante la masticación dolorosa, con el dispositivo de rastreo mandibular actual, pero son necesarios más estudios con dispositivos más precisos y sensitivos.

Zusammenfassung

Muskelschmerz steuert das Kauen: eine experimentelle Studie am Menschen

In dieser Studie wurde ein Schmerz im Musculus Masseter durch eine tonische Infusion von hypertonischer Salzlösung (5%) für 800ms bei 12 gesunden Männern ausgelöst. Die Personen bewerteten laufend die Schmerzintensität auf einer 10cm visuellen Analogskala. Das Kauen ipsilateral und kontralateral zur Infusionsseite wurde quantitativ beurteilt mittels Aufzeichnen der Unterkieferbewegungen und der Elektromyographie der schliessenden Muskeln vor, während und nach Zeitabschnitten mit konstanter Muskelschmerzintensität. Die maximale willkürliche okklusale Kraft (MVOF) während kurzen statischen Kontraktionen wurde ebenfalls überwacht. Kieferbewegungen und elektromyographische Daten wurden in einzelne Kauzyklen aufgeteilt und aufgrund einer Zyklus-auf-Zyklus-Basis analysiert, um eine Veränderlichkeit zwischen den Zyklen zu erklären. Bei allen Personen verursachte die tonische Infusion einen tiefen, lokalisierten Schmerz in einer klinisch relevanten Stärke (durchschnittliche VAS ± SE, 4.6 ± .3 cm). Die MVOF war signifikant beeinflusst durch den Muskelschmerz ($P < .0005$), mit signifikant niedrigerer MVOF während dem Schmerz verglichen mit den Abschnitten vor und nach dem Schmerz ($P < .05$). in einer signifikanten Anzahl von Kauzyklen war die durchschnittliche elektromyographische Aktivität aller schliessenden Muskeln während deren agonistischen Funktion erniedrigt, sowohl für das ipsilaterale als auch das kontralaterale schmerzhafte Kauen ($P < .05$). Diese elektromyograophischen Veränderungen sind wahrscheinlich ein Spiegelbild der natürlichen bilateralen Erholungsmuster der kieferschliessenden Muskeln während des Kauens. Signifikante Veränderungen in den Kieferbewegungen während des schmerzhaften Kauens konnten mit der aktuellen Kieferbewegungsvorrichtung nicht entdeckt werden, aber es sind weitere Studien mit genaueren und empfindlicheren Vorrichtungen notwendig.