Effects of Muscle Pain Induced by Glutamate Injections During Sustained Clenching on the Contraction Pattern of Masticatory Muscles

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Aims: To evaluate the contraction pattern of masticatory muscles during sustained clenching tasks with or without experimental pain induced by glutamate injection into the masseter muscle. It was hypothesized that acute muscle pain could induce compensatory changes in the electromyographic (EMG) activity of the masticatory muscles. **Methods:** Fifteen volunteers (seven males, mean age ± SD = 29.7 ± 1.1 years; eight females, mean age ± SD = 23.5 ± 1.2 years) were recruited in a crossover experimental study. All subjects participated in two randomized 20-minute experimental sessions. Each subject was asked to clench at 25% of the maximum voluntary contraction (MVC). After 10 minutes, isotonic saline or glutamate was injected in random order into the right masseter. EMG activity (root mean square [RMS] and mean power frequency [MPF]) was assessed in the masseter and anterior temporalis muscles on both sides. Pain and fatigue were assessed by 0–10 numeric rating scales (NRS) every minute. Differences between conditions (isotonic saline vs glutamate) for all the outcome parameters were analyzed by using a mixed effect model. **Results:** The EMG activity of the masticatory muscles and pain and fatigue scores were not dependent on isotonic saline/glutamate injection (all $P > .05$). The RMS in the temporalis and masseter muscles increased with time (right masseter $P = 0.001$, left masseter $P = .004$, right temporalis $P = .22$, left temporalis $P = .006$), whereas the MPF decreased (right masseter $P = 0.0001$, left masseter $P < .0001$, right temporalis $P = 0.51$, left temporalis $P = .0005$). Scores for fatigue and pain increased during the experimental sessions (all $P < .05$). **Conclusion:** Intramuscular injection of glutamate caused more pain than isotonic saline but did not affect the contraction pattern of the masticatory muscles during a sustained clenching task. This finding strongly suggests the adaptive capacity of the stomatognathic system in the presence of acute nociceptive inputs. *J Oral Facial Pain Headache 2014;28: 252–260. doi: 10.11607/ofph.1239*

Key words: *experimental orofacial pain, surface electromyography, temporomandibular disorders, tooth clenching*

Muscle fatigue and pain are frequent symptoms in patients affected by temporomandibular disorders (TMD).[1–3] It has been suggested that muscle hyperactivity resulting from low-level tooth clenching and/or grinding causes masticatory muscle fatigue and pain during experimental conditions.[4–7] However, in the clinic, scientific evidence in support of this theory is still under debate.[8–10]

Fatigue is characterized by a reduction of force-producing capacity during prolonged muscle activity. The activity of the masseter and temporalis muscles has been documented in several studies on jaw-muscle fatigue.[11–17] Peripheral fatigue is characterized by a reduction in force for a given level of muscle excitation.[4,18,19] Indeed, it has been reported that self-reported fatigue of the masticatory muscles during sustained low-level clenching tasks is characterized by electromyographic (EMG) changes with an increase of EMG signal amplitude and a decrease of the mean EMG frequency.[4]

In healthy volunteers, during sustained low-level static contractions, pain and fatigue have indeed been reported.[20] Furthermore, different contraction patterns of jaw-elevator muscles (coactivation, substitution, and several intermediate situations) have been identified.[21] This alternating

pattern could contribute to maintain the maximum voluntary contraction (MVC) to certain threshold levels. A similar alternating pattern was also described in 1983 by Hellsing and Lindström, who qualitatively reported a typical switch from the masseter to the temporalis muscle, namely a "rotation," during a prolonged isometric contraction.[22]

There is some evidence that muscle fatigue and pain, at least in part, could be related to peripheral glutamate levels. Fatiguing and painful exercises can evoke a release of glutamate,[23,24] and the increase of glutamate content and glutamate receptors in peripheral regions contribute to the enhancement of pain.[25] Furthermore, an increased interstitial concentration of glutamate has been found in the masseter muscles of individuals suffering from myofascial TMD pain as compared to healthy controls.[26] Finally, it has been suggested that experimental glutamate injection induces masticatory muscle pain in healthy individuals and can serve as a model for elucidating persistent myofascial pain mechanisms.[27] Intramuscular glutamate injection causes a reduction of pressure pain thresholds of the masticatory muscles as compared to isotonic solution during rest.[28] It is also able to evoke painful responses after a 60-minute recovery from low-level sustained clenching.[29] Nonetheless, little is known about the possible effects of experimental induced pain on the activity of the masticatory muscles during prolonged clenching. More knowledge of motor functioning of the masticatory muscles, under experimental pain, could help clinicians to better understand the manifestations of chronic muscular disorders and the compensatory potential for the stomatognathic system.

Hence, the aim of this study was to evaluate the contraction pattern of masticatory muscles during sustained clenching tasks with and without experimental pain induced by glutamate injection into the masseter muscle. This model could be useful to better understand the behavior of masticatory muscles in individuals affected by TMD pain. It was hypothesized that a painful injection could induce compensatory changes in the EMG activity of the masticatory muscles.

Materials and Methods

Fifteen young adults (seven males, mean age ± SD = 29.7 ± 1.1 years; eight females, mean age ± SD = 23.5 ± 1.2 years) were recruited from the staff and graduate students of the Department of Dentistry, Aarhus University, Denmark. All the individuals were examined by a single operator (RB) and were recruited if they fulfilled the following selection criteria: complete natural dentition with the exception of third molars, absence of TMD according to the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD)[30] with the exception of joint clicking by chance, and absence of orofacial pain (including headaches). Subjects with current orofacial inflammatory conditions, under treatment with medications that might influence pain perception at the time of the study, with neurologic or metabolic disorders were excluded.

The study was approved by the local ethics committee in accordance with the Helsinki Declaration. All subjects gave informed consent to the procedure.

Experimental Design

The study used a randomized crossover design. All subjects participated in two clenching sessions, each of which was 20 minutes long. Throughout each experimental session, data were collected for EMG parameters (root mean square [RMS] and mean power frequency [MPF]) and numeric rating scale (NRS) scores for fatigue (NRSf) and pain (NRSp) for the anterior temporalis and the superficial masseter of both right and left sides (right masseter, left masseter, right temporalis, left temporalis).

Each subject was asked to perform sustained isometric jaw clenching at 25% of MVC. Each session was divided into two blocks of 10 minutes each. During the first 10 minutes, each individual was encouraged to maintain constant tooth clenching. Thereafter, at the end of minute 10, each subject was instructed to stop clenching, and glutamate (0.2 mL) or isotonic saline (0.2 mL) was injected in random order into the right masseter. Immediately after the completion of the injection, each subject was instructed to resume clenching for other 10 minutes to complete the entire experimental task (Fig 1).

All the volunteers had constant visual feedback of the bite force, and during the task they were monitored and encouraged to maintain the force as constantly as possible. The perceived intensity of pain and fatigue were scored by the participants for each of the four masticatory muscles on the NRSf and NRSp. On these scales, the 0 endpoints were marked "no fatigue/pain" and the 10 endpoints "most imaginable fatigue/pain." The records were taken every minute throughout the clenching task.

All subjects were carefully instructed about the whole procedure, and test measurements were performed before starting the recordings, which were collected by a single examiner (RB). After a 1-week washout interval, each subject underwent a second experimental task, with the other solution injected. Hence, each volunteer participated in two experimental sessions, one starting with the injection of isotonic solution followed by the injection of glutamate (session A), and the other starting with the injection of glutamate followed by the injection of isotonic solution (session B, Fig 1).

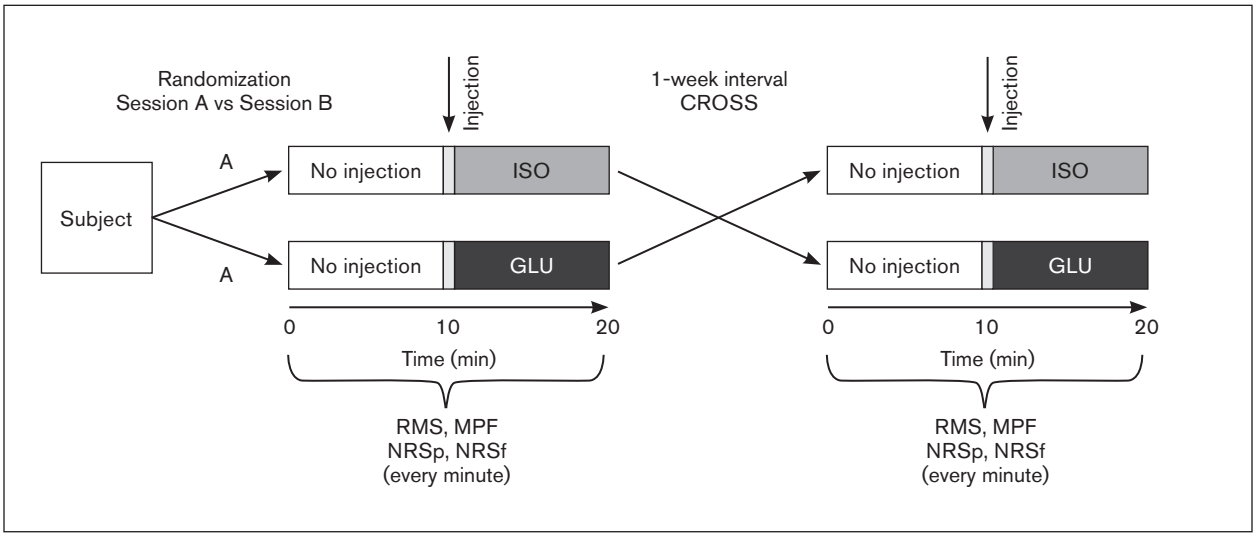

Fig 1 Overview of the experimental design. RMS = root mean square; MPF = mean power frequency; NRSp and NRSf = numerical rating scores for pain and fatigue; ISO = isotonic saline; GLU = glutamate.

Assessment of the Bite Force

A silicon-coated U-shaped force transducer (7 mm high, 11 x 11 mm area, Aalborg University) was placed on the right side between the first molars, and the subjects were asked to bite on the force transducer as hard as they could for 5 seconds in order to obtain the MVC force.[31] The MVC was recorded by the transducer as the peak value and was stored on the display.[4] The determination of the MVC was repeated three times, at 30-second intervals, and the mean was used to define the 25% MVC, which the participants had to sustain for 20 minutes.

Assessment of Muscle Activity

Bipolar EMG activity was measured at the masseter and anterior temporalis muscles on both sides by means of disposable surface electrodes (Medicotest). The skin over the muscles was cleaned with ethanol. Two electrodes for each muscle were used. For the masseter muscle, the electrode M1 was located over the most prominent part of the muscle, as determined by palpation during voluntary contraction; the electrode M2 was located 1.5 cm superiorly to M1, along the main direction of the muscle fibers. For the temporalis muscle, the electrode T1 was located between the upper orbital margin and the upper point of the outer ear, 2 cm behind the anterior border of the muscle, as determined by palpation during forceful voluntary contraction. The electrode T2 was located 2 cm above T1, along the main direction of the muscle fibers.[32] A common reference electrode was fixed to the wrist. The EMG signals were amplified (DISA 15C01), filtered (20 to 1,000 Hz) and sampled at 2,000 Hz for 30 seconds every 1 minute during the 20-minute clenching task. The RMS amplitude in the 10-second EMG epochs was calculated, and the mean frequency of the power spectrum (MPF) was determined by means of a fast Fourier transformation algorithm.

Experimental Muscle Pain

During the experiment, a solution of glutamate (1 M, 0.2 mL) or isotonic saline (0.9%, 0.2 mL) was injected into the right masseter muscle, midway between its upper and lower border and 1 cm posterior to its anterior border.[27] Glutamate was used to evoke experimental jaw-muscle pain.[27,33,34] Injections were done approximately 2 cm away from the surface EMG recording site. All injections were given manually over a 10-second period with a 27-gauge hypodermic needle and a disposable syringe. The needle was inserted until bony contact was made and then retracted about 2 mm before aspiration and injection of the solution. The pharmacy at Aarhus Hospital prepared the sterile glutamate solutions and adjusted the pH to 6.8–7.0.

The study was conducted in a single-blinded manner, since only the volunteers were unaware of the solution injected.

Statistical Analyses

Data were analyzed using SAS version 9.2 (SAS Inc) and SPSS version 20 (IBM). Continuous variables were reported as mean ± standard deviation. The RMS and the MPF parameters of each masticatory muscle were computed for each individual, every minute, during the entire experimental session. The mean outcome variables were RMS, MPF, NRSp, and NRSf for all the muscles examined.

Data retrieved from the 20-minute recordings were divided into two datasets. The first included

Table 1 Mean ± SD (n = 15) for EMG Parameters (RMS, MPF) and Numerical Rating Scale Scores (0–10) of Fatigue and Pain (NRSf, NRSp) Before (No injection) and After the Injection of Isotonic Saline (ISO) or Glutamate (GLU)

	0–9 min		11–20 min			
	Right	Left	Right		Left	
	No injection	No injection	ISO	GLU	ISO	GLU
RMS masseter (μV)	88.1 ± 47.8	100.1 ± 60	116.0 ± 50	118.9 ± 59	147.6 ± 74.2	125.4 ± 73.4
RMS temporalis (μV)	123.4 ± 106.6	66.3 ± 55.1	143.9 ± 75.7	194.4 ± 203	94.8 ± 73.3	93.7 ± 66.6
MPF masseter (Hz)	73.5 ± 26.5	73.0 ± 27.4	51.0 ± 17.6	53.8 ± 18.4	50.3 ± 19.9	48.7 ± 16.7
MPF temporalis (Hz)	72.6 ± 34.4	76.8 ± 29.6	56.0 ± 24.2	62.7 ± 30.5	53.6 ± 25.5	59.3 ± 25.1
NRSp masseter	3.7 ± 2.7	2.4 ± 2.5	5.27 ± 2.4	5.90 ± 2.6	3.91 ± 3.2	3.76 ± 3.0
NRSp temporalis	2.8 ± 2.9	1.8 ± 2.5	4.27 ± 3	4.31 ± 3.4	2.97 ± 3.1	2.37 ± 3.1
NRSf masseter	4.2 ± 3.0	2.5 ± 2.7	5.68 ± 2.9	6.74 ± 2.4	3.97 ± 3.2	3.82 ± 3.0
NRSf temporalis	3.0 ± 3.0	1.6 ± 2.5	4.23 ± 3.1	4.43 ± 3.3	2.58 ± 3.1	2.38 ± 3.0

RMS = root mean square; MPF = mean power frequency.

minutes 1 to 9 and was used to evaluate the association between the main outcome variables and time. The second dataset included the last 10 minutes (11 to 20) and was used to evaluate the influence of the injection (treatment, isotonic saline vs glutamate) on the outcomes examined. The analysis was conducted by excluding the RMS, MPF, NRSf, and NRSp recorded at minute 10 (time of injection) to avoid artifacts.

A mixed-effect model that could account for correlation between repeated measures was used by including the mean outcomes as dependent variables. For the models analyzing the first 9 minutes of the experimental sessions, time, session (A or B), and their interaction, as well as the mean bite force recorded, were included as covariates.

For the latter period (minutes 11 to 20), the covariates included in the models were: time, the interaction between time and session, the treatment (isotonic saline vs glutamate), the interaction between time and treatment, as well as the mean bite force. The statistical significance was set at $P < .05$.

The statistical power was computed a priori considering a conventional crossover design and continuous outcome variables. For a sample of 15 individuals, and assuming a significance level of .05, the study achieved 80% power, to detect a Cohen's D effect size of 1.10 (differences between treatments, ie, isotonic saline vs glutamate) for all the continuous variables considered. Nonetheless, this method has to be considered conservative while examining the 10 measurements (one for each minute) of the current experimental task. A lower effect size should be expected while considering the complexity of the data collected in the present study.

Results

All subjects completed the experiment and could sustain the required force for the entire duration. Descriptive statistics for RMS, MPF, NRSf, and NRSp recorded during the experimental sessions are reported in Table 1.

During the first 9 minutes, the RMS of temporalis and masseter muscles increased with time (right masseter $P = .001$, left masseter $P = .004$, right temporalis $P = .22$, left temporalis $P = .006$), whereas the MPF decreased (right masseter $P = .0001$, left masseter $P < .0001$, right temporalis $P = .51$, left temporalis $P = .0005$). In the same time interval, both the NRS scores for fatigue and pain increased (NRSf right masseter $P = .0008$, left masseter $P = .009$, right temporalis $P < .0001$, left temporalis $P < .0001$; NRSp right masseter $P = .003$, left masseter $P = .002$, right temporalis $P = .0009$, left temporalis $P < .0001$) (Fig 2).

Post-hoc analysis revealed significant differences in EMG parameters and NRS scores between muscles in the three experimental conditions (Table 2). In particular, the RMS of the right masseter was, on average, lower than the left masseter muscles, and the RMS of the right temporalis was significantly higher than the left temporalis muscles during the right-sided clenching. Interestingly, on the right side, the RMS of masseter muscles was significantly lower than temporalis muscles, whereas, on the left side the reverse behavior was recorded, with the RMS of masseter muscles being greater than the RMS of temporalis muscles. In addition, only after glutamate injection, the left and right temporalis muscles displayed a higher MPF than the masseters. Finally, in all

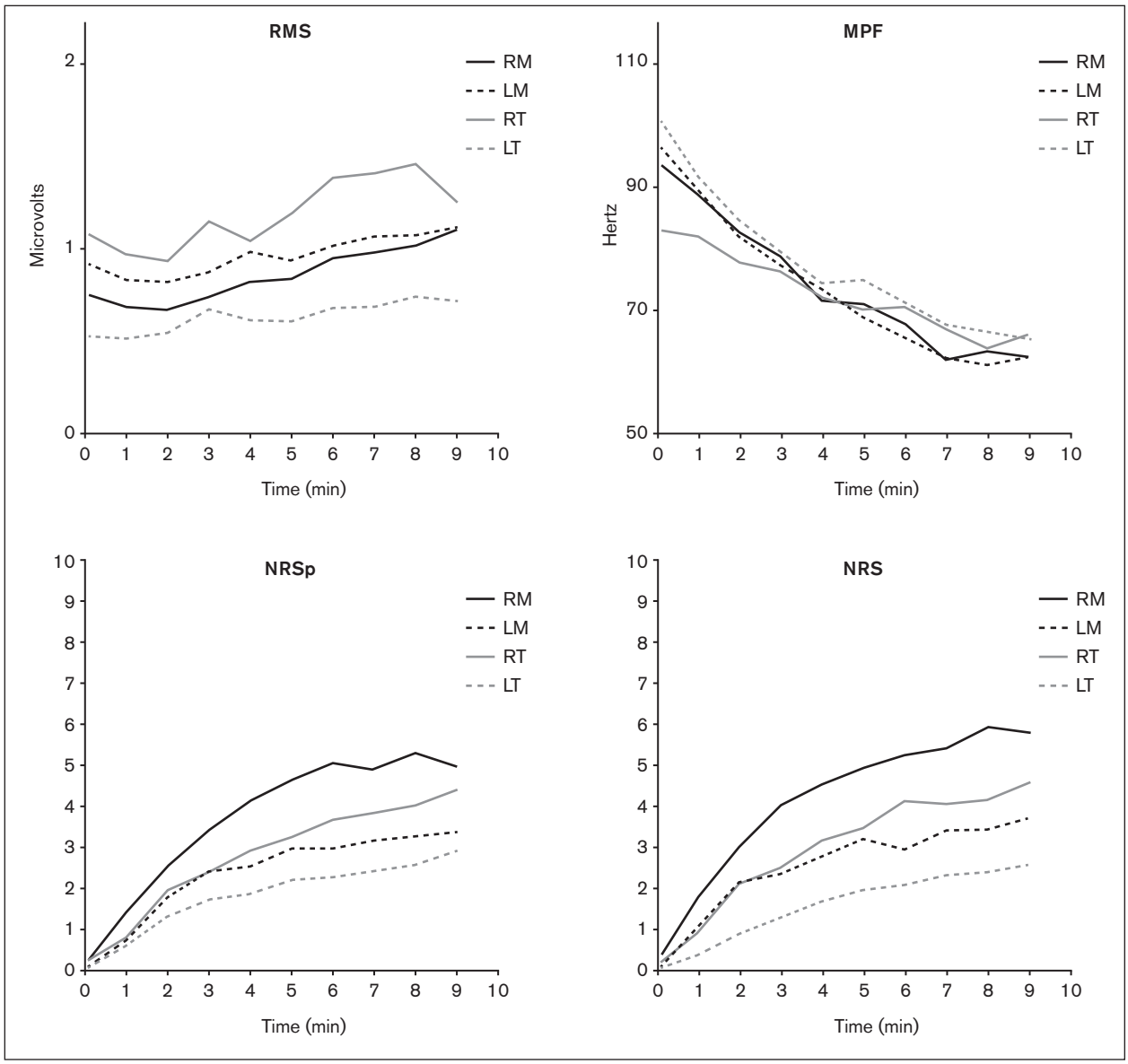

Fig 2 Mean and trend RMS (root mean square), MPF (mean power frequency), NRSp and NRSf (numerical rating scores for pain and fatigue) for all the muscles examined (RM = right masseter, LM = left masseter, RT = right temporalis, LT = left temporalis) before the injection of isotonic saline or glutamate solution during clenching (at 25% maximum voluntary contraction).

Table 2 Post-hoc Comparisons (*P* values) for EMG Parameters (RMS, MPF) and Numerical Pain Rating Scores (NRSf, NRSp) for all the Muscles Examined (n = 15) in the Three Experimental Conditions: No injection, After Injection of Isotonic Solution (ISO), After Injection of Glutamate (GLU)

		RMS			MPF			NRSp			NRSf		
Muscles		No injection	ISO	GLU	No injection	ISO	GLU	No injection	ISO	GLU	No injection	ISO	GLU
RM	RT	**< .0001**	**.001**	**< .0001**	0.80	**.05**	**.005**	.09	.13	**< .001**	**.004**	**.007**	**.001**
RM	LM	.160	**< .0001**	.95	0.99	.99	.22	**< .0001**	**< .0001**	**< .0001**	**< .0001**	**< .0001**	**< .001**
RT	LT	**< .001**	**< .0001**	**< .0001**	0.13	.85	.58	**< .0001**	**< .0001**	**< .0001**	**< .0001**	**< .0001**	**< .0001**
LM	LT	**< .0001**	**< .0001**	**.05**	0.40	.22	**.0007**	**.031**	**.009**	**< .0001**	**< .001**	**< .001**	**< .0001**

Bold type = statistically significant ($P \leq .05$). RMS = root mean square; MPF = mean power frequency; NRSp and NRSf = numerical rating scores for pain and fatigue; RM = right masseter; LM = left masseter; RT = right temporalis; LT = left temporalis.

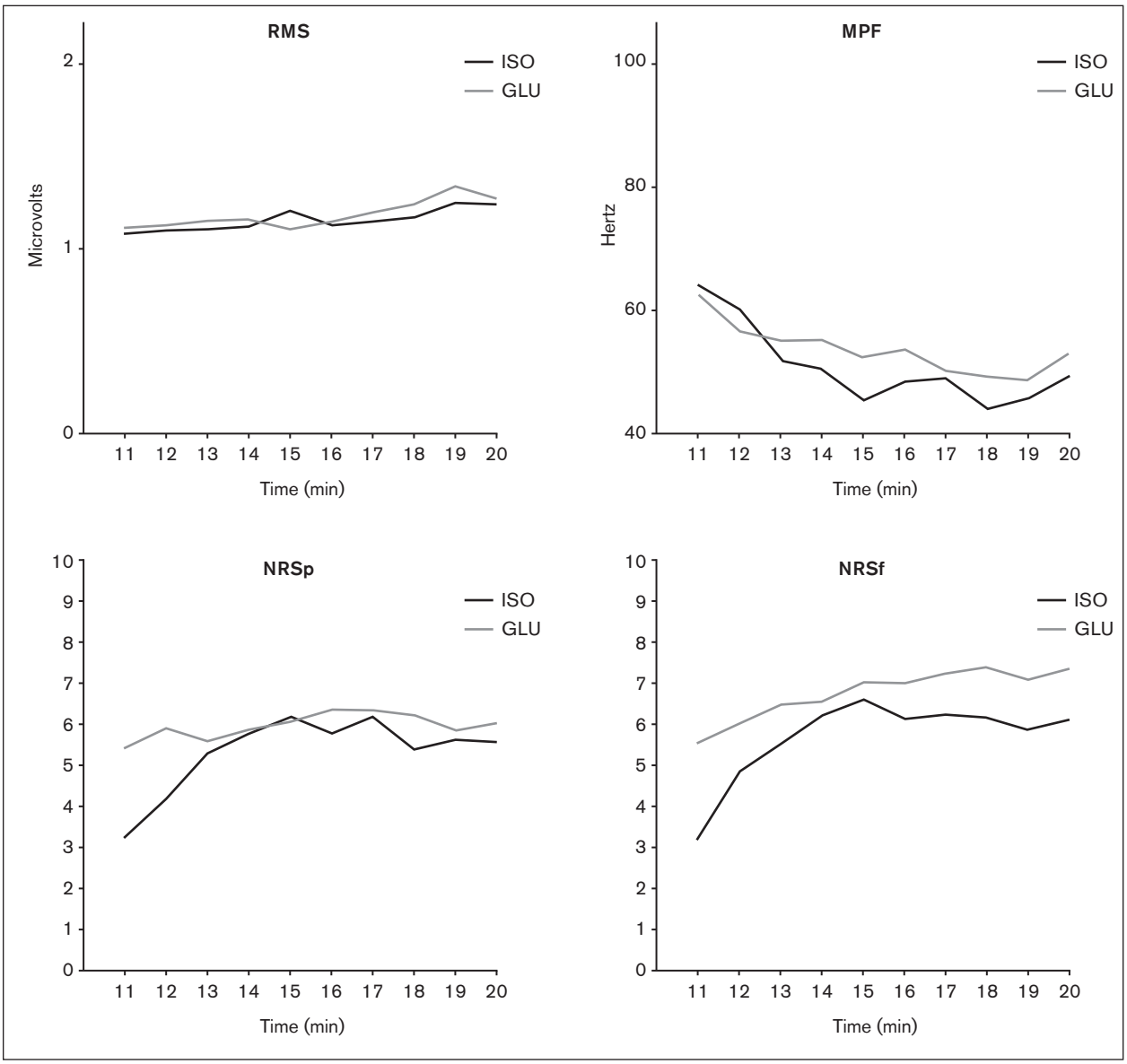

Fig 3 Mean and trend RMS, MPF, NRSp, and NRSf, of the right masseter after the injection of isotonic saline or glutamate solution during clenching (at 25% maximum voluntary contraction).

the experimental settings, the masseters were more painful and fatigued than the temporalis muscles and the left side showed significantly lower NRSp and NRSf than the right side.

The RMS and the MPF of the masticatory muscles examined, as well as NRSf and NRSp scores, were not significantly affected by isotonic/glutamate injection (Table 3). Although in the first 2 minutes following the injection, the NRSp of the right masseter was on average lower in the isotonic session as compared to the glutamate session (Fig 3), the mixed model showed no significant overall differences (P = .061). A sensitivity analysis, which was performed by adding in the mixed models gender and age as covariates, obtained similar results (data not shown).

Table 3 *P* values from Mixed Regression Model for the Interaction Between Time and Isotonic Saline/Glutamate Injection for All the Muscles Examined (n = 15) in the Latter Interval (11–20 minutes)

	Right	Left
RMS masseter	.75	.41
RMS temporalis	.44	.95
MPF masseter	.59	.49
MPF temporalis	.51	.15
NRSp masseter	.84	.67
NRSp temporalis	.38	.84
NRSf masseter	.37	.70
NRSf temporalis	.58	.54

RMS = root mean square; MPF = mean power frequency; NRSp and NRSf = numerical rating scores for pain and fatigue. See statistical analyses for the characteristics of the model.

Discussion

To the authors' knowledge, this study is the first examining the effects of experimental induced pain on the activity of the masticatory muscles during prolonged clenching. A MPF was used instead of median power frequency (MDPF) for the EMG assessments. Both reflect muscular fatigue,[35] but the MPF was preferred for the purpose of comparison to earlier findings, eg, Torisu et al, who evaluated the effect of low-level clenching on experimental muscle pain.[29] MPF is an average frequency that is calculated as the sum of product of the EMG power spectrum and the frequency divided by the total sum of the power spectrum; MDPF is a frequency at which the EMG power spectrum is divided into two regions with equal amplitude.[36] A previous study has shown that both MDPF and MPF characterize muscular fatigue, although MDPF may be more sensitive than MPF.[35] Indeed, during the sustained clenching task, the MPF decreased in all jaw-closing muscles, indicating fatigue in accordance with others, eg, Torisu et al.[29]

The present findings showed that, during sustained clenching tasks, the EMG activity of the masseter and temporalis muscles changed with time without any significant influence of glutamate-evoked pain. In accordance with previous studies,[4,21] the EMG parameters showed a high variability within and between subjects. It was found that the RMS increased as an indication of increased muscle recruitment, while the MPF decreased during sustained clenching at 25% of the MVC.

Previous reports have also shown during prolonged clenching a "rotation" of the masticatory muscles (determined by an alternate switch of activity between two synergistic muscles) that was either frequent[22] or sporadic.[21] Contrary to these reports, the present experiment found a synchronized contraction pattern of masticatory muscles and no rotation between the muscles. Hellsing and Lindström[22] found at least one episode of rotation after several trials in all the patients, suggesting that this mechanism seems to be completely out of voluntary control and shows facilitation at repeated tests. Moreover, Farella and coworkers performed an experiment at different MVC values (10%, 15%, and 20%) with a continuous EMG sampling and found sporadic episodes of rotation only at 10% of MVC, without a facilitation effect over consecutive trials.[21] However, in the present study, the sample was subjected to two trials with a 1-week interval and was invited to clench at 25% of MVC with EMG parameters collected every minute. Hence, the discrepancies between the study results could be ascribed to different experimental designs and/or to the high variability of EMG recordings.

To evoke pain, the excitatory amino acid glutamate was used because it has a clearance time suitable for this experimental design, being shorter than capsaicin[37] and hypertonic saline.[38] Furthermore, glutamate injections have been used recently to induce experimental pain of the jaw muscles in a number of reports,[26,27,39] serving as a model for experimental TMD pain. These studies showed that glutamate can evoke an immediate pain response in individuals at rest or after short-lasting experimental clenching sessions. Similarly to these reports, the present study found that the injection of glutamate during muscle exercise was followed by higher average pain scores in the first 2 minutes as compared to isotonic solution. This is consistent with the mean clearance time of the glutamate (2 minutes) reported in the literature.[40] However, the lack of a significant difference between isotonic saline and glutamate could be related to the fact that the muscle subjected to the glutamate injection was already to some extent painful because of the long-lasting clenching task. Thus, it is likely that the additional nociceptive input from the glutamate injection had been masked by the ongoing pain evoked by the sustained clenching task. Indeed, a previous report has shown that the interstitial glutamate concentration is not correlated to pain scores in subjects suffering from myofascial TMD pain.[26]

The mixed-model analysis used in the present study showed that the injection of glutamate into the masseter did not affect the average EMG activity of any of the muscles examined as compared to isotonic saline solution. Interestingly, this masked nociceptive input induced by glutamate in a muscle already subjected to sustained contraction did not affect the overall contraction pattern. As a consequence of this observation, it is possible that compensatory mechanisms (eg, rotation of jaw muscle activity) of the other jaw muscles were not evoked after the injection. This could be the consequence of a spatial reorganization of the firing motor units occurring in the right masseter in order to maintain tooth clenching during the experimental task[20,39,41] and to the adaptation of the masticatory muscles to current painful conditions.[42]

The average EMG amplitude of the masseter and temporalis muscles was different between the right and left sides; it showed a reverse behavior, being on average higher in the right temporalis and left masseter and lower in the left temporalis and right masseter. This phenomenon displayed a harder work effort of the right temporalis and left masseter during the experimental sessions. It was likely that the average RMS was influenced by the mandibular lateral displacement due to the positioning of the bite force transducer between the molars of the right side[43]; however, this did not, on average, cause any differences in MPF parameters between sides and illustrates the difference in sensitivity between the RMS and MPF measures.

This study has contributed to the characterization of the pain and fatigue profiles for each main jaw-closing muscle, as evaluated in a previous study.[4] The EMG signal characteristics strongly indicated that sustained low-intensity clenching could contribute to the development of fatigue and pain. Indeed, the pain and fatigue as assessed by NRS increased significantly in all muscles during the experimental tasks. This is consistent with previous findings showing that prolonged clenching can evoke jaw muscle pain and fatigue.[4,20]

Significantly higher pain scores in the right masseter as compared to the other muscles were found during the clenching tasks, independently, by the glutamate injections. This could be explained by a referred dental pain[44,45] related to the presence of the bite force transducer on the right side during the experimental tasks, by an attentional bias toward a predictable threatening stimulus represented by the awareness of the injection in the right masseter,[46] or by the expectation of a negative outcome (ie, pain related to the injection) that may have led to the worsening of the symptom in the right masseter.[47]

Some limitations in the present study need to be noted. The power of the study was rather limited. The level of 25% MVC was chosen because it has been shown to evoke moderate to severe pain,[20,21] can be maintained for at least 10-minute experimental sessions,[21] and is a reasonable submaximal loading to be tested because of its frequency in healthy individuals in natural environments.[48] However, other MVC levels could be tested to provide a more comprehensive understanding of the interaction between prolonged clenching and nociceptive input. If a randomization in the positioning of the bite force transducer and in the side of injection had been performed, this might have determined possible side effects in EMG signals. Another limitation was related to the EMG sampling being recorded only every minute. Continuous EMG recordings would have allowed a more detailed insight into the contraction patterns and potential rotation, but this was not feasible in the present study due to technical limitations. Although there are well-described gender differences in glutamate-evoked pain,[33,34] the study did not aim to test for gender differences in EMG or pain responses. The crossover, within-in group design of the study may nevertheless circumvent the potential impact of gender-related differences in sensory-motor interactions, and future studies will be needed to address this question in more detail. Moreover, the injection in itself may have acted as confounder, being an additional possible cause of increased pain in both isotonic saline and glutamate conditions. To limit this effect, the analysis was conducted by excluding the RMS, MPF, NRSf, and NRSp, recorded at minute 10 (time of injection). Finally, the study did not provide any information concerning the pressure pain thresholds at different muscular sites before and after the experimental pain injection, mainly due to technical reasons, but also because the inclusion of stimulus-evoked pain was beyond the scope of the present study. Despite these potential shortcomings, the authors believe that the present study has shed additional light on the contraction patterns during sustained tooth clenching.

In conclusion, the results of this study show that, during prolonged sustained clenching at 25% MCV, the EMG characteristics of masticatory muscles are dependent only on time and not sensitive to additional nociceptive inputs. This result strongly indicates the adaptive capacity of the stomatognathic system and may contribute to a better understanding of the masticatory muscle adaptation in individuals suffering from myofascial pain of the masticatory muscles. In particular, it appears that individuals suffering from acute myofascial pain can preserve their masticatory performance without significant changes in their stomatognathic function. However, further studies are clearly needed in chronic myofascial pain conditions.

Acknowledgments

The authors report no conflicts of interest related to this study.

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