Can Experimentally Evoked Pain in the Jaw Muscles or Temporomandibular Joint Affect Anterior Bite Force in Humans?

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Aims: To test the hypothesis that experimental pain in the masseter muscle or temporomandibular joint (TMJ) will decrease the anterior maximum voluntary bite force (MVBF) and jaw muscle activity in relation to the perceived effort. **Methods:** Sixteen volunteers participated in two experimental sessions. Participants were injected with 0.2 mL of monosodium glutamate (1.0 M) into either the masseter muscle or TMJ. The MVBF and corresponding electromyographic (EMG) activity of the masseter, anterior temporalis, and digastric muscles were recorded 10 times at an interval of 2 minutes before and after injection. Pain was measured using a visual analog scale and McGill Pain Questionnaire. In addition, participants were asked how they perceived the interference of pain on their biting performance. The data analysis included a two-way analysis of variance model and *t* test. **Results:** There was no significant difference in peak pain intensity ($P = .066$) and duration of pain ($P = .608$) between painful muscle and TMJ injections, but TMJ injection produced a significantly larger area under the curve ($P = .005$) and a significantly higher pain rating index ($P = .030$). Pain in the muscle ($P = .421$) and TMJ ($P = .057$) did not significantly change the MVBF from baseline levels. The EMG activity also did not differ significantly from baseline levels during muscle pain. However, there was a significant increase ($P = .028$) in the EMG activity of the anterior temporalis and a significant decrease ($P = .010$) in the EMG activity of the anterior digastric muscle compared to baseline during TMJ pain. Subject-based reports also revealed that in the majority of cases (62.5%), pain did not interfere with the MVBF task. **Conclusion:** Experimental pain from either masseter muscle or TMJ did not affect the MVBF, in accordance with the subject-based reports. Jaw muscle activity, except for EMG activity of the anterior temporalis and anterior digastric muscles during TMJ pain, also remained unaffected by pain. The findings suggest that it is not pain in itself but rather how pain is perceived that may lead to adaptation of motor function, supporting an integrated pain adaptation model. *J Oral Facial Pain Headache 2015;29:31–40. doi: 10.11607/ofph.1268*

Key words: *electromyography, integrated pain adaptation model, monosodium glutamate*

The term temporomandibular disorders (TMD) encompasses a wide variety of clinical conditions and pathologic states that involve the masticatory muscles and/or the temporomandibular joints (TMJs) along with the associated structures.[1–3] Pain in the masticatory muscles and /or TMJs, as well as disordered jaw function such as limitation in mandibular movement and joint noises, are often considered to be the cardinal signs of TMD.[1,2] Clinically, the majority of TMD patients can be classified into those with problems associated with the masticatory musculature or with problems within the TMJ itself. Distinguishing these two categories is often considered a clinical challenge.[4,5]

It has been reported that pain in the musculoskeletal system (masticatory muscles and TMJ) causes significant changes in the masticatory movement pattern, maximum occlusal force, and electromyographic (EMG) activity as compared to individuals without such pain.[6,7] EMG data from animal studies have also indicated that noxious stimulation of the TMJ might have a different motor effect compared to noxious

stimulation of jaw muscles.[8] The association between pain and motor function has been explained mainly on the basis of two conflicting theories, the Vicious Cycle Theory (VCT) and the Pain Adaptation Model (PAM). According to the VCT, pain arises as a result of stress, structural abnormality, abnormal posture or movements leading to "hyperactivity" of the musculature, spasms, and thereby pain and dysfunction, thus perpetuating the cycle.[6] Most of the management strategies for pain still attempt to break this purported vicious cycle. However, several EMG studies on the effect of clinical and experimental muscle pain have questioned the validity of the VCT.[7,9,10]

According to the PAM, nociceptive activity via segmental brainstem or spinal cord motor circuits leads to alterations in muscle activity (reduced agonist and increased antagonist muscle activity), which result in slower and smaller movements in order to minimize further injury and therefore promote healing.[11] Even though this theory gives an appropriate explanation of the effect of muscle pain on motor function, it does not explain the origin of pain. Moreover, experimental and clinical orofacial pain studies have also shown evidence of no change in the jaw movement amplitude[12,13] and a decrease in agonist or an increase in antagonist EMG activity during mastication in pain and pain-free individuals.[14,15] Also the long-term consequences of altered jaw function due to sustained painful stimulation are unknown.[1] Hence, even though the two theories of pain and motor function are simple and consistent with a range of clinical and experimental evidence, several authors have cited their inadequacies.[2,16,17]

Recently it has been claimed that because pain is multidimensional in nature, the relationship between pain and motor functions may be more complex and may depend on the interaction of biopsychosocial variables that make up the complexity of the individual's pain experience.[2] Further, anatomical and functional complexities contribute effectively to the individual's sensory-motor interaction.[2] Pain catastrophizing and fear-avoidance responses to pain have also shown to be a potential modifier of self-reported pain.[18–21] It has also been suggested that sensory-motor interactions related to pain involve higher central influences.[2] These concepts have led to the formulation of the Integrated Pain Adaptation Model (IPAM).[2]

Experimental pain models have demonstrated the potential to bridge animal models and clinical findings in patients.[1,7] Therefore, in light of the current theories on pain, the present study was designed to test the hypothesis that experimental pain in the masseter muscle or TMJ will decrease the anterior maximum voluntary bite force (MVBF) and jaw muscle activity in relation to the perceived effort.

Materials and Methods

Sixteen healthy volunteers (11 male and 5 female) in the age range of 18 to 33 years (mean age ± SEM 26.1 ± 1.3 years) participated in the study. All the participants included in the study neither suffered from any serious/chronic disease nor were on any kind of medications. A clinical examination as per the Research Diagnostic Criteria for TMD (RDC/TMD)[22] confirmed the absence of TMD in the participants. The participants were also clinically screened for evidence of tooth wear by using a five-point ordinal scale.[23] Only participants with scores of 0 (no wear) and 1 (visible wear within the enamel) were included in the study. The study was conducted in accordance with the Helsinki Declaration II and was approved by the local ethics committee. The participants were informed about their right to withdraw from the study at any time, and informed consent was obtained.

Experimental Protocol

The volunteers participated in two experimental sessions separated by 3 to 4 days. In the first session (day 1), the participants were injected with a sterile solution of monosodium glutamate (MSG) to induce experimental pain in the left masseter muscle. Further, the participants returned after 3 to 4 days for the second session (day 2) and were injected with the same solution in the left TMJ. The order of the site of injection (ie, masseter muscle/TMJ) was randomized. The participants were seated upright in a comfortable office chair with the EMG electrodes attached on the left and right masseter muscles (MAL and MAR), left anterior temporalis muscle (TAL), and anterior digastric muscle (DIG). The participants were asked to clench 10 times on a bite force transducer inserted between the maxillary and mandibular incisors at an interval of 2 minutes between the clenches, in the presence of glutamate-evoked masseter muscle pain or TMJ pain (randomized sequence). Bite force and the corresponding EMG activity were recorded during each of the repeated clenches. Baseline bite force and EMG activity (without pain) were also recorded 10 times in the same manner in both sessions prior to the injection. The site of injections and the schematic illustration of the experimental protocol are shown in Fig 1.

Experimental Pain

Experimental pain was induced in the participants by injecting 0.2 mL sterile solution of MSG (1.0 M, pH 7.2). The point of injection for the masseter muscle was midway between the upper and lower borders of the masseter muscle and 1 cm posterior to its anterior border.[24] The experimental TMJ pain was induced in an open-mouth position by injecting the

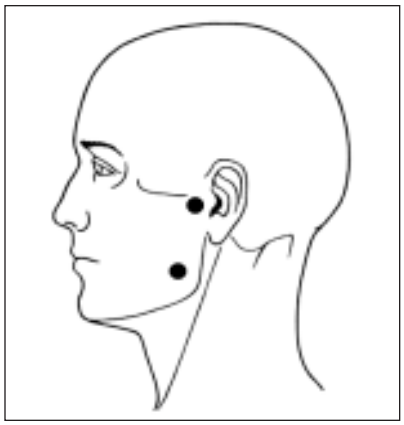

Fig 1a Sites of injection for experimental muscle and TMJ pain.

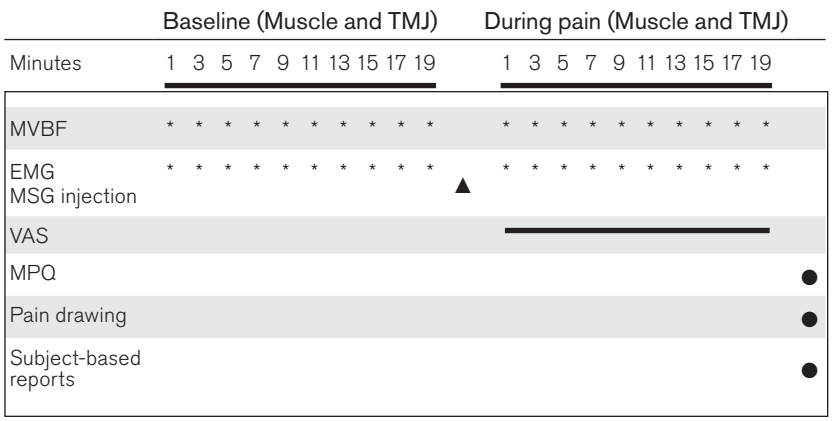

Fig 1b Schematic illustration of the experimental schedule. MVBF = maximum voluntary bite force; EMG = electromyographic recording; MSG = monosodium glutamate; VAS = visual analog scale; MPQ = McGill Pain Questionnaire.

same amount of MSG solution in the upper joint compartment of the TMJ at a point 10 mm anterior to the tragus and 2 mm inferior to the tragal canthal line.[25,26] Both injections were performed over a period of 10 seconds with a 27-gauge hypodermic needle and disposable syringe. All participants received both injections on the left side (painful side), and no short-term or long-term complications of the injections were reported with either injection. The participants were also asked to continuously rate the pain (experimental pain) on an electronic visual analog scale (VAS), with the lower extreme of the VAS marked as "no pain" and the upper extreme marked as "most pain imaginable."

At the end of each session, participants completed the McGill Pain Questionnaire (MPQ), and drew the perceived area of pain on a figure of the lateral aspect of the face, in order to describe the overall experience after the injection. The participants were also asked to provide subject-based reports on perceived pain and referred pain experienced during the experiment and how the pain or referred pain was perceived to interfere with the MVBF task (Table 1).

Table 1 Subject-Based Reports on Interference of Pain and Referred Pain with Bite Force

Question	Muscle pain	TMJ pain
1. Do you feel pain in the cheek muscle (masseter muscle)/ Joint (TMJ)?	a) Yes (100%) b) No (0%)	a) Yes (100%) b) No (0%)
2. Did you feel pain outside muscle/ TMJ?	a) Yes (25%) b) No (75%)	a) Yes (56.3%) b) No (43.8%)
3. If yes where?	a) Ear (0%) b) Temporal (12.5%) c) Chin (0%) d) Neck (0%) e) Teeth (12.5%) f) TMJ (0%) g) Others (12.5%) h) NA (75%)	a) Ear (18.8%) b) Temporal (18.8%) c) Chin (12.5%) d) Neck (0%) e) Teeth (0%) f) TMJ (0%) g) Others (6.3%) h) NA (43.8%)
4. Did the pain start at the same time as the muscle/TMJ pain or after the muscle/TMJ pain?	a) Same time (6.3%) b) After (18.8%) c) NA (75%)	a) Same time (18.8%) b) After (37.5%) c) NA (43.8%)
5. If after, how long after the muscle/ TMJ pain (min)	a) Half min (6.3%) b) Two min (6.3%) c) Four min (6.3%) d) NA (81.3%)	a) Half min (31.3%) b) Two min (12.5%) c) Four min (0%) d) NA (56.3%)
6. Was the muscle/ TMJ pain influenced by biting	a) Yes (37.5%) b) No change (62.5%)	a) Yes (37.5%) b) No change (62.5%)
7. If yes, did the pain	a) Increase (6.3%) b) Decrease (31.3%) c) No change (62.5%)	a) Increase (12.5%) b) Decrease (25.0%) c) No change (62.5%)
8. Was the pain outside masseter muscle/TMJ (referred pain) influenced by biting?	a) Yes (0%) b) No (25%) c) NA (75%)	a) Yes (0%) b) No (56.3%) c) NA (43.7%)
9. If yes, did the pain	a) Increase (0%) b) Decrease (0%) c) NA (100%)	a) Increase (0%) b) Decrease (0%) c) NA (100%)

NA = not applicable.

EMG Activity and Bite Force

The EMG activity of the MAL, MAR, TAL, and DIG was measured using disposable, bipolar surface electrodes (30 × 21-mm recording area, 720-01-K, Neuroline, Ambu). The electrodes were placed 10 mm apart on the central part of the muscle, midway between the anterior and posterior borders and the superior and inferior borders of the MAL

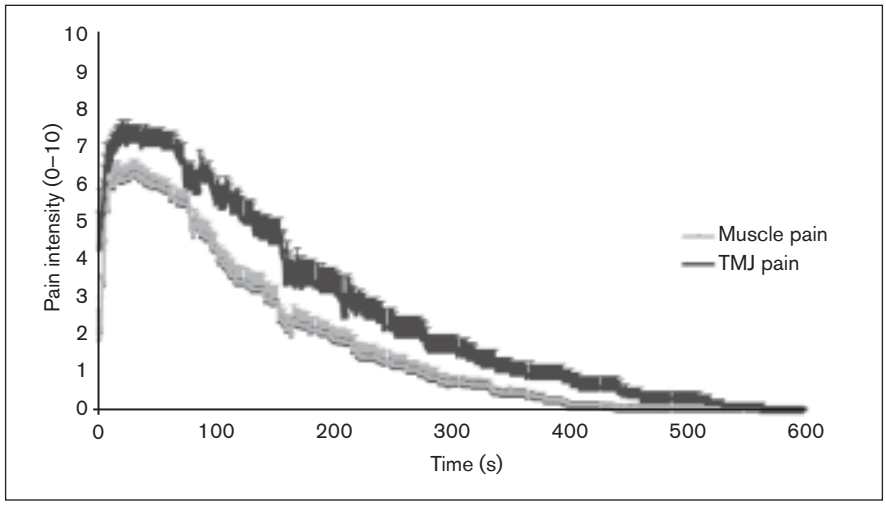

Fig 2a Mean and standard error of mean (SEM) of visual analog scale (VAS) scores (0–10) of glutamate-evoked pain intensity in masseter muscle (6.7 ± 0.3) and TMJ (7.3 ± 0.4).

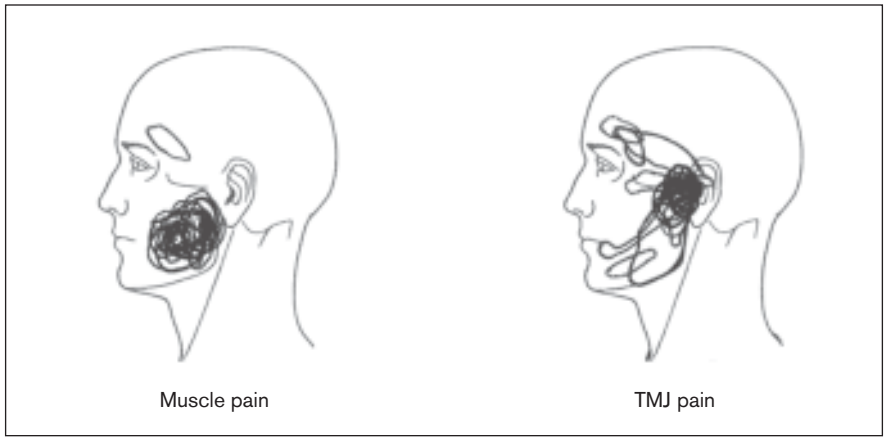

Fig 2b Pain drawings of perceived areas of pain and referred pain after injection of glutamate in the muscle and TMJ. Individual drawings from each participant (n = 16) have been superimposed.

and MAR, and the anterior part of the TAL lateral to the eyebrow. A set of electrodes was also placed on the DIG, one on either side 1 cm medial to the base of the mandible at the level of the first and second molar.[27] The left side was the painful side, where the painful injections of MSG were made either on the masseter muscle or TMJ. Hence it was decided to record the EMG activity of only the left TAL. The electrodes were positioned on the skin parallel to the direction of the muscle fibers. The skin over the recording positions was thoroughly cleaned with sterile wipes. A common reference electrode soaked in saline was attached to the left wrist of the participant. The EMG activity was recorded in 10-second epochs. The EMG signals were amplified 10,000 times (Disa 15C01), filtered in the bandwidth 20 Hz to 1 kHz, sampled at 2 kHz, and stored for off-line analysis. The participants held a customized strain gauge–based bite force transducer with their right hand, and placed it between the maxillary and mandibular anterior teeth. A piece of soft Plexiglas was glued to the surface of the bite force transducer to protect the teeth and to identify the same biting position. All the participants performed 10 repetitions of MVBF with their anterior teeth, and verbal encouragement was given to them to obtain the MVBF. The participants were asked to clench for about 3 to 4 seconds to record the bite force and EMG activity. Each repetition of the biting task was separated by 2 minutes of rest.

Statistical Analyses

A two-way analysis of variance (ANOVA) model with repeated measures was used to analyze the different outcome parameters. The factors in the ANOVA were condition (2 levels, ie, baseline and after the painful

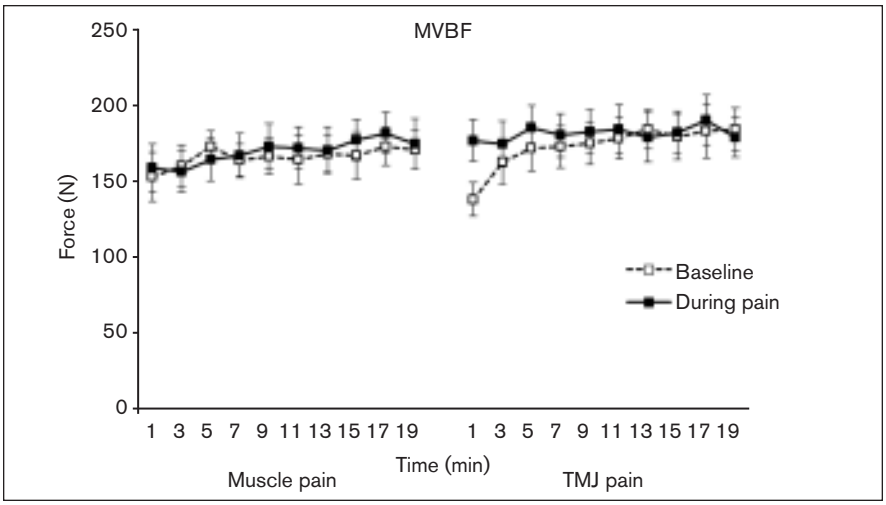

Fig 3 Mean (n = 16) and standard error of mean (SEM) of anterior maximum voluntary bite force (MVBF) at baseline and during muscle and TMJ injections. There was no significant difference in the MVBF at baseline (without pain) and during pain with respect to painful injection in the muscle (P = .421) and TMJ (P = .057).

injections) and time (10 levels, ie, 1, 3, 5, 7, 9, 11, 13, 15, 17, and 19 minutes) (see Fig 1b). Post-hoc tests were performed with the Tukey Honestly Significant Difference (HSD) test. Logarithmic conversions were done whenever necessary. The VAS pain scores were compared with a *t* test. The relative change in MVBF was calculated as [(MVBF during pain – MVBF baseline) / MVBF baseline] × 100. The level of statistical significance was set at .05.

Results

Experimental Pain

The perceived pain intensity (mean ± SEM) on the VAS in both sessions is shown in Fig 2a. There was no significant difference in peak pain intensity (*t* test, P = .066) and total duration of pain (*t* test, P = .608) between the injections (ie, muscle and TMJ). However, the area under the VAS time curve was significantly larger for TMJ pain (2,931.9 ± 1,343.9 arbitrary units) than for muscle pain (1996.5 ± 825.9 arbitrary units) (*t* test, P = .005).

MPQ

The overall pain rating index of the MPQ for muscle pain (11.0 ± 1.6) was significantly lower (*t* test; P = .030) than for TMJ pain (15.1 ± 2.5). The most commonly used adjectives for describing the pain sensation were hot (31%), boring (25%), sharp (25%), pressing (25%), taut (25%), squeezing (25%), and nagging (25%) for muscle pain, and pulsing (31%), boring (37.5%), pressing (37.5%), hot (25%), penetrating (37.5%), and nauseating (25%) for TMJ pain.

Subject-Based Reports

All participants (100%) confirmed that they experienced pain in the muscle or TMJ when they were injected with 0.2 mL of MSG on the respective site (Table 1). However, only 25% of them experienced referred pain (pain outside the site of injection) when injected into the muscle, and 56.3% of the participants experienced referred pain when injected into the TMJ (Fig 2b). Although the majority (62.5% for both muscle and TMJ pain) of the participants reported no changes in the intensity of pain during biting, pain was perceived to decrease in 31.3% of the participants during muscle pain and in 25% during TMJ pain on biting (see Table 1). Only 6.3% and 12.5% of the participants with muscle or TMJ pain, respectively, reported an increase in the intensity of pain on biting. Furthermore, the average relative change in MVBF was categorized into increase, decrease, or no change (ie, relative change < 10%) and compared with the subject-based reports. The no changes in pain according to the subject-based reports were reflected in no changes in MVBF in 70% of the participants during muscle pain and in 50% of the participants during TMJ pain.

MVBF

The results of ANOVAs revealed that there was no significant difference in the anterior MVBF at baseline (without pain) and during muscle pain (ANOVA; F = 0.685, P = .421) (Fig 3). However, there was a significant effect of time (F = 0.326, P = .001) but no significant interaction for muscle pain (F = 0.580, P = .812). Similarly, there was no significant difference in the anterior MVBF at baseline and during

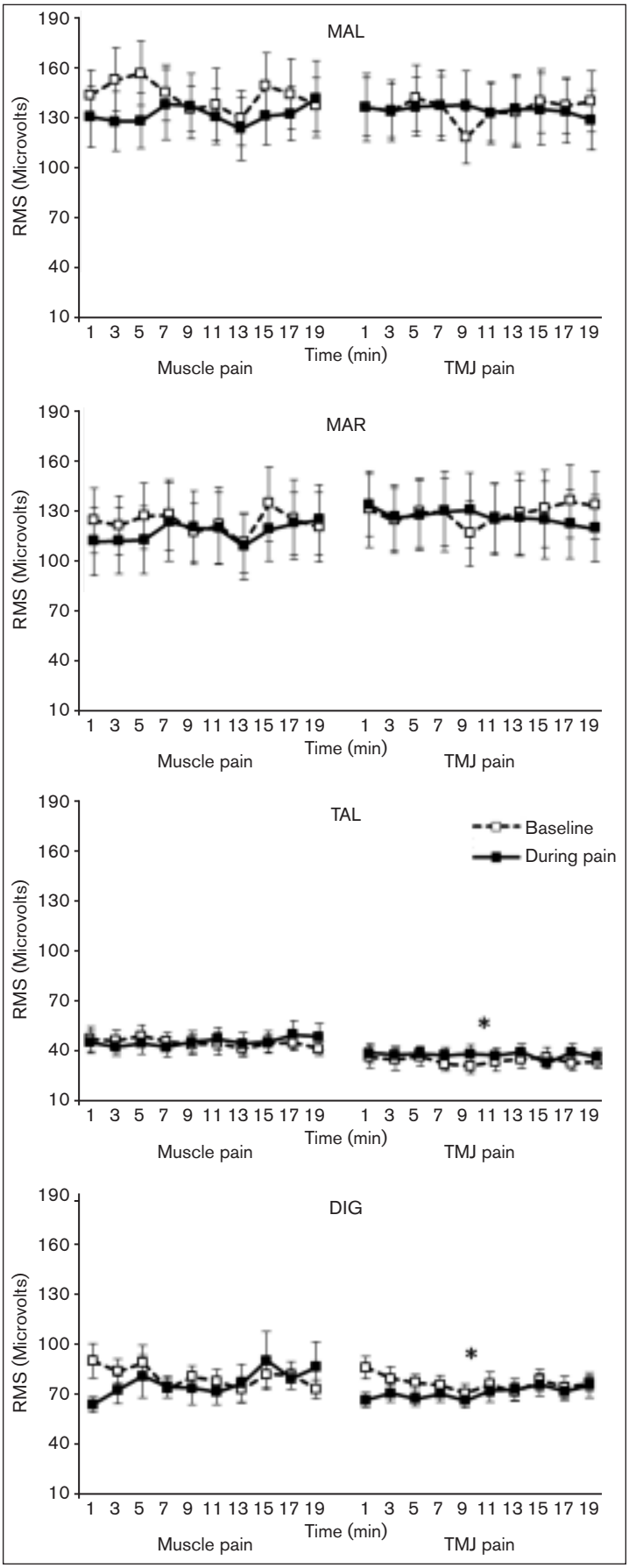

Fig 4 Mean (n = 16) and standard error of mean (SEM) of EMG activity of left and right masseter (MAL, MAR), anterior temporalis (TAL), and anterior digastric (DIG) muscles at baseline and during pain for muscle and TMJ injections. Asterisks indicate significant difference between baseline and during pain ($P < .05$).

TMJ pain (ANOVA; F = 4.248, $P = .057$) (Fig 3). However, there was a significant effect of time (F = 4.978; $P = .001$) and a significant interaction (F = 3.443; $P = .001$) for TMJ pain. Post-hoc analysis of interaction for TMJ pain showed significantly lower MVBF at 1 minute during baseline than 3, 5, 7, 9, 11, 13, 15, 17, and 19 minutes ($P < .033$) during baseline and during pain.

EMG Characteristics

There was no significant difference in the EMG activity of the MAL (ANOVA; F = 4.131, $P = .060$), MAR (ANOVA; F = 1.019, $P = .329$), TAL (ANOVA; F = 0.517, $P = .483$), and DIG (ANOVA; F = 4.466, $P = .052$) at baseline and during muscle pain (Fig 4). There was also no significant effect of time on the EMG activity of the MAL (F = 1.395, $P = .196$), MAR (F = 1.307, $P = .239$), TAL (F = 0.364, $P = .950$), and DIG (F = 1.521, $P = .146$) during muscle pain. However, the interaction of time and condition was significant for the DIG (F = 2.433, $P = .014$) but not significant for the MAL (F = 0.747, $P = .665$), MAR (F = 1.178, $P = .314$), and TAL (F = 0. 639, $P = .762$). Post-hoc analysis of the interaction for the DIG showed a significantly lower EMG activity at 1 minute during muscle pain than 1, 3, and 5 minutes during baseline ($P < .036$). Similarly, there was no significant difference in the EMG activity of the MAL (ANOVA; F = 0.027, $P = .871$) and MAR (ANOVA; F = 0.054, $P = .818$) before and during TMJ pain (Fig 4). However, there was a significant difference in the EMG activity of the TAL (ANOVA; F = 5.913, $P = .028$) and DIG (ANOVA; F = 8.662, $P = .010$) before and during TMJ pain. There was no significant effect of time on the EMG activity of the MAL (F = 0.631, $P = .769$), MAR (F = 0.507, $P = .867$), TAL (F = 0.960, $P = .476$), and DIG (F = 0.964, $P = .473$). The interaction of condition and time was also not significant for the MAL (F = 0.861, $P = .561$), MAR (F = 1.277, $P = .255$), TAL (F = 1.125, $P = .350$), and DIG (F = 1.150, $P = .332$) during TMJ pain.

Discussion

The results of this study have demonstrated the effect of two different types of experimental pain on the anterior MVBF and jaw muscle activity. The results suggested that the peak intensity and duration of pain did not differ significantly between experimental pain injections into the masseter muscle and TMJ. However, a larger area under the VAS time curve and a higher pain rating index were found for TMJ pain than for muscle pain. It can also be inferred from the results that regardless of moderate to intense levels of pain in the muscle or TMJ, there were no changes in MVBF, in accordance with subject-based reports on pain interference with function. Except for the EMG activity of the TAL and DIG during TMJ pain, jaw muscle EMG activity also remained unaffected by pain. Further, the majority of the participants reported no change in the intensity of pain during biting, indicating low interference of experimental pain with the biting task. The findings of this study may enhance the understanding of jaw muscle reorganization in painful conditions and how nociceptive activity and perceived pain may or may not impact motor function.

Experimental Pain and Sensory Manifestations

The available literature about human experimental pain suggests that injections or infusions of painful substances (eg, glutamate, hypertonic saline, and capsaicin) in healthy subjects produce similar somatosensory changes, intensity, quality, location, spread, and referral of pain as in TMD patients.[1,6,7] Clinical studies on human tendons, muscles, and TMD patients have also demonstrated elevated glutamate concentration associated with chronic non-inflammatory pain conditions.[28–30] Hence, in the present study experimental pain was produced in otherwise healthy individuals by a standard protocol of injections of 0.2 mL of 1 M sterile solution of MSG in the muscle or TMJ.[31]

Previous studies have suggested that experimental pain in healthy volunteers mimics many characteristics of persistent pain in clinical TMD pain patients.[1,21] The results of the present study are contrary to the previous clinical observations by McCreary et al[32] and Rudy et al,[33] who reported higher pain intensities for muscle pain than for TMJ pain in a group of TMD patients. The present study's results did not show any major differences in the intensity and duration of pain, although the area under the VAS time curve was different for muscle and TMJ pain. Further, the results of the present study are in accordance with Lindroth et al, who showed no significant difference in the perceived pain intensity and pain duration in a group of patients with masseter muscle pain and intracapsular pain.[4] However, the participants in the present study reported a higher pain rating index on the MPQ, which may indicate worse pain in the TMJ as compared to muscle pain. The participants described the area of perceived pain as exclusively on the same side as the injection and local to the site of injection. The participants also experienced referred pain, which was found more often during TMJ pain than during muscle pain. Similar patterns of referred pain as shown in the pain drawings (see Fig 2b) are also commonly associated with TMD patients.[34]

MVBF and EMG Activity

The MSG-induced pain in the present study produced similar pain intensity as in other human experimental pain[23,35] or clinical TMD studies.[36,37] However, the MVBF and EMG activity of the masticatory muscles, except for the TAL and DIG during TMJ pain, remained unaffected irrespective of the intensity of pain, which is unlike clinical TMD in which bite force values are characteristically decreased.[38–40] Further, previous studies on experimental pain have also reported a decrease in MVBF and EMG activity of the jaw muscles.[35,41] However, the present finding can be supported by the outcome of the subject-based reports, which suggested that pain did not influence the biting performance in 62.5% of the participants (irrespective of the site), while 31.3% of the participants during muscle pain and 25% of the participants during TMJ pain reported a decrease in pain. Only 6.3% (for muscle pain) and 12.5% (for TMJ pain) of the participants reported an increase in the intensity of pain when they were biting on the bite force transducer. One possibility is that this decrease in perceived pain could have been due to distribution of the injected MSG in the muscle or TMJ during the muscle contraction and TMJ loading, thereby leading to an enhanced washout.[1] The difference in observation between experimental pain and clinical TMD pain could also be due to longer-lasting central sensitization in persistent TMD pain conditions; this is not observed in glutamate-induced acute pain, which may mainly involve peripheral mechanisms.[42,43]

The inconsistencies in the findings between experimental pain and clinical TMD pain as to effects on motor functions can further be explained on the basis of several studies which have indicated that TMD pain patients have more psychosocial distress than matched control subjects.[24,44] Alternatively, these findings are also supported on the basis of the IPAM, according to which an individual's pain experiences interact in a unique way so as to maintain homeostasis and minimize the effect of pain and/or the metabolic cost.[2,44] Also, due to the multidimensional nature of pain, which includes sensory-discriminative, cognitive-evaluative, and

motivational-affective dimensions, each of these dimensions may have a significant and highly individual influence on its effect on motor activity.[2] Further, the anatomical and functional complexities of the sensorimotor integration results in a new optimized motor recruitment strategy, leading to unique motor responses that may aim to minimize pain. Also the redundancy of the masticatory system means that many muscle-recruitment strategies are possible to perform a specific task; therefore, individuals may develop unique recruitment strategies during pain.[45] Hence, the changes in motor control during pain cannot always be stereotypical and predictable, since they depend on the interaction between the multidimensional nature and anatomical and functional complexity of the sensorimotor system.[2,16] Therefore, it is also proposed that each pain experience, in terms of its quality, location, intensity, and/or duration in an individual, may be associated with a particular pattern of change in EMG activity.[46] Fear-avoidance responses (ie, avoidance of pain-associated activities) or endurance responses (ie, thoughts of suppression, distraction from pain, and potentially positive mood despite pain) may also affect the response to pain.[21,47] Hence, factors such as pain perception and ability to cope with pain may vary from person to person.[19]

Although the results of the present study to some extent contradict the PAM, they are consistent with clinical and experimental pain studies by Lyons and Baxendale, who did not find any difference in bite force values in a group of patients with myogenous craniomandibular disorder and healthy controls, and by Ikebe et al, who reported no difference in bite force values of TMD patients when compared to healthy controls.[48,49] Clark and Carter have also reported a lack of change in the EMG activity and bite force levels following repeated brief maximum contractions.[50] It is also suggested that pain may not have an effect on the incisal bite force and EMG activity, since the superficial and deep muscles act differently during painful clenches and the EMG activity of masticatory muscles is much lower during incisal clenching than the molar clenching.[51,52] It is also proposed that the pattern of pain-induced changes in EMG activity can vary with the task being performed, jaw displacement magnitude, and the participant being studied.[53] Further, it was also observed in the present study that pain in the TMJ affected the EMG activity of the TAL and DIG, which is consistent with the findings of previous animal studies by Cairns et al[54] and Yu et al,[55] who demonstrated that noxious stimulation of the TMJ enhanced the EMG activity of the jaw muscles in adult rats. Broton and Sessle[56] also found that noxious stimulation of TMJ afferents resulted in a sustained reflex excitation of anterior digastric and temporalis muscle in cats. These studies in animals have also suggested that noxious stimulation in the TMJ or masseter muscle evokes both peripheral and central sensitization through the activation of peripheral receptors.[55–57] Further, Okamoto et al[58] demonstrated that intra-articular TMJ injections of adenosine triphosphate (a noxious stimulus) increased the EMG activity of the masseter muscle in rats. However, the present study results did not show any significant effect of TMJ pain on the EMG activity of the masseter muscle in healthy human participants.

Even though the TMJ injections in the present study followed previously described techniques,[25,26] methodological concerns do apply to human pain experiments. One such issue may be the accuracy of the TMJ injection technique. The site of the TMJ injection is only approximately located; thus, in spite of the participants confirming subjective symptoms, the failure to identify the bone/cartilage of the upper joint compartment during the TMJ injection due to anatomical variation cannot be completely ruled out in some participants. The inter-individual variability is also one of the main methodological concerns in human pain studies, but this concern can be ruled out in the present study due to the paired design in which each participant served as his or her own control.

The present experimental study was an attempt to provide insight into how the complex jaw motor system reacts to pain and its association with the task performed both in the presence and absence of acute pain. Despite the differences between experimental pain and clinical TMD pain, the findings suggest that an individual's motor response to pain may be dependent to a large extent on the ability of the individual to cope with the painful stimulus and the ability to maintain behavioral activities in spite of the painful stimulus. Overall, the present findings are in agreement with the IPAM.

Acknowledgments

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