The Effects of Experimental Temporalis Muscle Pain on Jaw Muscle Electromyographic Activity During Jaw Movements and Relationships with Some Psychological Variables

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Aims: To determine if the effects of experimental temporalis muscle pain on jaw muscle activity vary with the jaw task performed, jaw displacement magnitude, participant being studied, and with psychological measures. **Methods:** Jaw movement was tracked, and electromyographic (EMG) activity was recorded from the masseter and anterior temporalis and digastric muscles in 14 asymptomatic participants during standardized opening/closing jaw movement, free chewing, and standardized chewing tasks. Tasks were repeated in three blocks: Block 1 (baseline), Block 2 (during 5% hypertonic or 0.9% isotonic saline infusion into the anterior temporalis), and Block 3 (during infusion of the opposite solution). Participants also completed the Depression, Anxiety, and Stress Scales 21 (DASS 21), the Fear of Pain Questionnaire (FPQ III), the Pain Self-Efficacy Questionnaire (PSEQ), and the Pain Catastrophizing Scale (PCS). Analyses involved linear mixed-model analysis and Pearson correlations. $P < .05$ was considered statistically significant. **Results:** The presence of a significant difference in jaw muscle EMG activity between hypertonic and isotonic saline infusions varied between tasks and between jaw muscle agonists and antagonists, but not in displacement magnitude. There were qualitative differences between participants in the effects of infusion on EMG activity. During hypertonic saline infusion, significant positive correlations were noted between jaw-closing EMG activity and anxiety, fear of medical pain, and PCS scores. **Conclusion:** Noxious stimulation of the temporalis muscle results in changes in jaw muscle activity, which can vary with the task, the muscle, the participant, and some psychological variables. *J Oral Facial Pain Headache 2018;32:29–39. doi: 10.11607/ofph.1821*

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The most prevalent nondental orofacial pain is musculoskeletal pain involving the masticatory muscles and the temporomandibular joint (TMJ), collectively termed temporomandibular disorders (TMD). Although pain and limitation of jaw movement are common symptoms of TMD, the precise relationship between the two is unclear.[1–4] Two of the most widely cited theories of the relationship between pain and motor activity are the Vicious Cycle Theory and the Pain Adaptation Model. The Vicious Cycle Theory hypothesizes that an initiating factor—such as stress, a structural abnormality, or an unusual posture or movement—results in pain that progresses to an increase in muscle activity and then further pain.[3–6] While the Vicious Cycle Theory implicates muscle hyperactivity in the generation of pain, the Pain Adaptation Model proposes that existing pain (whose origin is not explained by the model) results in decreased agonist muscle activity and increased antagonist muscle activity to reduce the amplitude and speed of movement, thereby promoting healing by protecting the skeletomotor system from further injury.[7–9] Some current management strategies are based on these earlier models.

There is little support in the spinal and trigeminal motor systems for the Vicious Cycle Theory,[10] and there are some data sets consistent with the Pain Adaptation Model.[3,410–13] For example, some clinical and/or experimental pain studies have demonstrated that, in comparison with asymptomatic controls, pain is associated with smaller and slower

movements, reduced agonist muscle activity, or increased antagonist muscle activity.[4,7,14,15] However, other data sets show that, in comparison with pain-free controls, pain does not always result in significant reductions in jaw movement amplitude during jaw tasks or mastication, in jaw-closing force, or in masseter activity.[1,2,4,14,16–21] There is also evidence that experimental noxious stimulation of the masseter can result in changes in jaw muscle activity that can vary with the task performed, the jaw displacement magnitude, and the participant being studied.[22] Therefore, while some of the data appear consistent with some features of the Vicious Cycle Theory or the Pain Adaptation Model, other data sets are not.[22]

Given this range of effects of pain on motor activity described in previous studies, newer models[3,4,8,23] have been proposed. One of these newer models introduces two proposals: *(1)* pain results in a redistribution of activity within and between muscles, and *(2)* this redistribution is influenced by the functional complexity of the sensorimotor system, as well as by the multidimensional nature of pain.[3,4] The former proposal includes the possibility of pain effects differing depending on the motor task being performed, and the latter consists of sensory-discriminative, cognitive-evaluative, and motivational-affective components, where factors such as pain location, intensity, and characteristics—as well as pain-related cognitions and mood—may modify the effects of pain on motor activity.

It is well known that TMD patients frequently experience pain not only in the masseter muscle, but also in the temporalis muscle.[24] While there have been studies of the effects of experimental pain evoked from algesic chemical injections into the temporalis, these studies have mostly focused on the sensory features of pain and/or reflex effects.[25,26] A recent study has shown that experimental noxious stimulation of the anterior temporalis had no significant effects on the amplitude or velocity of jaw movement during standardized opening/closing jaw movements and free and standardized chewing.[21] However, the absence of these effects does not preclude possible EMG effects, which might not become manifest in the kinematic variables examined in this recent study. Therefore, the first aim of the present study was to determine if the effects of experimental temporalis pain on jaw muscle activity vary with the jaw task performed, the jaw displacement magnitude, and the participant being studied.

Another problem with the earlier theories mentioned is that they implicate only brainstem or spinal cord circuits and do not include a possible role for psychological factors (eg, catastrophizing, depression) operating at the suprabulbar/supraspinal level in any pain-induced motor effects. However, there is emerging evidence that psychological factors appear to play a significant role in the development of chronicity in TMD[9] and to be capable of influencing the relationship between pain and motor activity in both the spinal and trigeminal systems; therefore, these factors may play a significant role in the progression of musculoskeletal pain conditions. In the spinal motor system, for example, some psychological constructs (eg, catastrophizing, fear avoidance, and depression) are correlated with motor performance.[27–31] There is also recent evidence for significant correlations between psychological variables (eg, catastrophizing, depression, and orofacial pain intensity and quality) and jaw muscle and movement features in experimentally induced masseter or clinical pain or under pain-free conditions.[2–4,32,33] Therefore, the second aim of the present study was to determine if the effects of experimental temporalis pain on jaw muscle activity vary with psychological measures. The overall hypothesis was that the effect of experimental temporalis muscle pain on jaw muscle activity varies with the jaw task performed, the jaw displacement magnitude, and the participant being studied, and that psychological measures are correlated with jaw muscle activity.

Materials and Methods

A total of 14 asymptomatic participants (mean ± standard deviation [SD] age: 33.8 ± 7.8 years; range: 26 to 54 years; 9 males and 5 females) volunteered. The absence of a TMD diagnosis was verified by the application of the Research Diagnostic Criteria for TMD (RDC/TMD).[34] Ethical approval was gained from the Western Sydney Local Health District Human Ethics Committee of Westmead Hospital, and all participants affirmed their informed consent prior to their inclusion in the study. Many of the details have been previously documented.[2,21,22] The recordings of EMG activity analyzed in the present study were made during the previously published recordings of jaw movement.[21]

Figure 1 is an overview of the experimental procedure. Informed consent and all questionnaires (except for the McGill Pain Questionnaire [MPQ]) were completed first. The tasks consisted of standardized opening/closing jaw movements, free chewing, and standardized chewing. These tasks were performed in a repeated-measures design over three blocks of about 10-minute duration, separated by 10-minute rest periods. The total duration of the experiment for each participant, including set-up time, was about 80 minutes.

Psychological Questionnaires

Prior to the experiment, all participants completed the Depression, Anxiety, and Stress Scales 21 (DASS 21), the Pain Catastrophizing Scale (PCS), the Fear

Item	Pre	Block 1 (baseline)	Rest	Block 2	Rest	Block 3	Rest
Rest			×		×		×
Questionnaires	×						
Tasks		×		×		×	
Infusions				×		×	
Pain VAS and maps				×		×	
MPQ					×		×
Duration (min)		10	10	10	10	10	

Fig 1 Outline of the experimental procedure. Informed consent and all questionnaires (except the McGill Pain Questionnaire [MPQ]) were completed initially. The tasks were performed in a repeated measures design over three blocks of ~10-minute duration and separated by 10-minute rest periods. Tasks were three trials of standardized opening/closing jaw movements and two trials each of free chewing and standardized chewing. × indicates the procedure was completed. VAS = visual analog scale.

of Pain Questionnaire (FPQ III), and the Pain Self-Efficacy Questionnaire (PSEQ). The DASS 21[35,36] assesses depression, anxiety, and stress. The participants endorsed statements over the past week on a 4-point severity scale, and the scores were summed for each scale. The PCS comprises 13 items and has 3 subscales: rumination, magnification, and helplessness. Each item is rated on a 5-point temporal scale, from 0 (not at all) to 4 (all the time).[37,38] The FPQ III is a 30-item measure that explores fear across three dimensions; namely, fear of severe pain (eg, breaking your leg), fear of minor pain (eg, getting a papercut on your finger), and fear of medical pain (eg, receiving an injection in your hip/buttock). Each item is scored on a 5-point severity scale ranging from 1 (not at all) to 5 (extreme).[39] The PSEQ is a 10-item inventory that scores both the strength and generality of a patient's beliefs about his/her ability to perform their daily activities or to function despite their pain. The scores for the PSEQ range from 0 to 60, with higher scores being associated with stronger self-efficacy beliefs.[40–45]

Jaw Muscle and Movement Recordings

Recordings of electromyographic (EMG) activity were made during the assessed tasks (see below). These recordings were taken from the right anterior temporalis (RAT), left anterior temporalis (LAT), right masseter (RMAS), left masseter (LMAS), and right digastric (RDIG) muscles with bipolar surface electrodes (Duo-Trode, Myotronics). The EMG activity was amplified (×2,000 to ×10,000; World Precision Instruments), filtered (bandwidth 100 Hz to 10 kHz), and digitized (sampling rate 5,000 samples/second; Cambridge Electronic Design, model micro1401).

Mandibular movement was also recorded during the tasks with an optoelectronic jaw-tracking system (JAWS3D or JAWS2000). The movement of the mid-incisor point (ie, the point between the incisal edges of the mandibular central incisors) was displayed as a moving dot on a video screen in front of the participant for tracking a computer-controlled target positioned on the screen.[22] The target was used for the standardized tasks. Custom-made clutches were attached to 2 to 4 maxillary and mandibular anterior teeth on the right side of the participant with cyanoacrylate adhesive, and these clutches supported the target frames of the jaw-tracking system. The target frames were tracked by cameras that allowed the display of the mid-incisor point along three orthogonal axes.

Tasks and Sequencing of Blocks

There were three blocks of tasks: Block 1 (baseline prior to infusion), Block 2 (during infusion of 5% hypertonic saline or 0.9% isotonic saline), and Block 3 (during infusion of the opposite solution) (Fig 1). The order the solutions were infused in each participant (ie, hypertonic saline first or isotonic saline first) was reversed from that of the immediately preceding participant; the first participant received hypertonic saline first. The tasks performed in each block were three trials of standardized opening/closing jaw movements, two trials of 15 seconds of free gum chewing, and two trials of 15 seconds of standardized gum chewing. A single repetition of a task was termed a trial, and there was a 20-second rest period between each trial. Each trial commenced with the jaw at the postural position.

Prior to the chewing tasks, participants were initially asked to chew a piece of chewing gum (0.14 g) on the left side until it was soft and then move it to the right side immediately prior to commencement of the jaw movement trials. During free chewing, the participants were asked to chew at their natural speed, while during standardized chewing, the participants were asked to chew at the speed dictated by a linear bank of light-emitting diode (LED) lights placed in front of the participant, which oscillated at 900 milliseconds/cycle. Participants matched their chewing speed to this linear bank by viewing their mid-incisor point movement on a computer monitor.

At the beginning of the standardized opening/closing jaw movements, the jaw remained for 2 seconds at the postural jaw position, and then the participant was instructed to match as closely as possible the displacement of the mid-incisor point of their jaw to a linear bank of sequentially illuminated LEDs placed to the side of the mid-incisor point trajectory in the z axis (ie, vertical axis). Participants initially opened as wide as comfortable to ensure that they could open sufficiently to track the LEDs to an opening displacement of 20 mm from the postural position. The Spike2 software (Cambridge Electronic Design) illuminated the LEDs in sequence so that ideal tracking of these LEDs resulted in a jaw movement speed of 2.2 mm/second. Following a 2- to 3-second holding phase, the participant moved the jaw back to the postural position by again following the illuminated LED sequence.

Experimental Temporalis Pain

Experimental pain was induced by the infusion of 5% hypertonic saline into the RAT via an intravenous catheter (JELCO, 22 G × 1", Smiths Medical ASD). The catheter was connected to a tube (extension set with polyethylene inner line, 75 cm, 0.7 mL) leading from an infusion pump (IVAC Model P2000, UK) holding a 10-mL syringe filled with 5% hypertonic saline or 0.9% isotonic saline. Hypertonic saline (0.2 mL) was initially infused rapidly to achieve a moderate pain rating of 40 to 60 mm on a visual analog scale (VAS). The VAS had verbal anchors of no pain at all (0 mm) and the worst pain imaginable (100 mm). Infusion was continuous at a rate between 2 and 9 mL/hour for about 15 minutes. A VAS for pain intensity and a pain map (on an outline of a head) to indicate location of the pain were completed after the insertion of the needle but prior to the commencement of the infusion, during the bolus infusion but prior to the jaw movement task, and after each trial of each jaw task (Fig 1). Each VAS score was used by one operator to adjust the infusion rate so as to maintain pain as closely as possible in the range of 40 to 60 mm. The isotonic saline infusion was a control for any possible effects arising from volumetric change within the muscle of the solution. For trials when isotonic saline was initially infused, the rate was set at 2 mL/hour for 5 minutes and then increased to 4 mL/hour until all tasks were completed. When hypertonic saline was infused first, the rate of isotonic saline infusion followed the rates and changes in rates of the previous hypertonic saline infusion for that participant. Participants were given standardized instructions and were unaware which solution was injected first.

The MPQ[46] was completed at the end of Blocks 2 and 3 (Fig 1). It contains three classes of words (78 words in total) that can be used to evaluate perceived pain, and scores can range from 0 to 78. Participants described the quality of their pain on the MPQ after the termination of each infusion. Of the 14 participants, only 1 did not receive the isotonic saline infusion for technical reasons. The entire experiment was performed in each participant on one experimental day.

Data Analysis

The first part of the analysis was to divide the displacement data along the superior-inferior (z) axis into 0.5-mm increments to allow a comparison of the associated EMG activity at the same amount of displacement across the three blocks. Each trial of the standardized opening/closing jaw movements and each chewing cycle was divided into an opening phase and a closing phase. For the standardized opening/closing jaw movements, the onset of the opening phase was defined as the first 0.5 mm of displacement along the z axis from the postural jaw position, and the end was defined as the last 0.5 mm of displacement prior to the 2- to 3-second holding phase. The beginning of the closing phase was defined as the first 0.5 mm of displacement along the z axis back to the postural position, and the end was defined as when the jaw had returned to a stable position near the postural position (participants did not always return exactly to the same position as at the beginning of the trial). For the chewing cycle analysis, aberrant or incomplete cycles were initially identified manually and were not analyzed (eg, chewing cycles that were < 50% or > 150% of the mean amplitude for that individual, as well as cycles with sudden jerky movements). Then, for each chewing trial, the opening phase was defined as the point at which the mid-incisor point had displaced 0.5 mm from maximum closure of the previous chewing cycle (or from postural jaw position if the first cycle was being considered) to the point of maximum opening displacement, and the closing phase was defined from the point at maximum opening to 0.5 mm from maximum closure of that chewing cycle. Maximum closure was slightly open from intercuspal position by the gum bolus.

After this, calculations were made of the EMG root mean square (RMS) values at each 0.5-mm increment of mid-incisor point displacement during the opening and closing phases for each trial of the standardized jaw movements and each cycle of the free chewing and standardized chewing. These RMS values were then averaged across all trials and cycles within a participant. This allowed displays of RMS values across blocks within participants. After the data were processed in this way, all the EMG data were log transformed to stabilize the variance. A linear mixed-effects model was then used to assess the difference in the log-transformed EMG activity between the blocks (baseline, isotonic saline infusion, hypertonic saline infusion) across the displacement

for each jaw muscle and task. The analysis takes into account the dependence between repeated observations in the same participant.[22,47]

The difference in EMG activity for a particular muscle between baseline and hypertonic saline or isotonic saline at each 0.5 mm of displacement was expressed as:

Difference log RMS
(isotonic or hypertonic saline and baseline) at X mm =
EMG-intercept difference (infusion and baseline) +
slope difference (infusion and baseline) ×
X mm of displacement

The EMG-intercept difference is the ratio of difference in EMG activity at 0-mm displacement between hypertonic or isotonic saline conditions and baseline, while the slope difference is the ratio of difference in EMG slope; that is, the difference between the y coordinates (log RMS) divided by the difference in the x coordinates (mm displacement) of hypertonic or isotonic saline infusion and baseline. For example, the ratio of difference between baseline and hypertonic saline blocks for the RDIG during the opening phase of the standardized chewing task at 10-mm displacement would be calculated as:

0.163 (intercept difference) +
[0.025 (slope difference) × 10] = 0.413

The ratio is < 1, which means that the EMG activity during hypertonic saline infusion is less than that of baseline.

The effect of pain on EMG activity was considered in relation to the agonist/antagonist function of each recorded muscle so as to interpret any EMG effects observed in terms of the proposals of the Vicious Cycle Theory and the Pain Adaptation Model. For the purposes of this analysis, the following muscles were classified as agonists: RAT, LAT, RMAS, and LMAS during the closing phases of standardized opening/closing jaw movements and during free and standardized chewing, and RDIG during the opening phases of these tasks. The following muscles were classified as antagonists: RAT, LAT, RMAS, LMAS during the opening phases of the standardized opening/closing jaw movements and during free and standardized chewing, and the RDIG during the closing phases of these tasks.

Multiple regression analyses were used to explore the association between the EMG activity of each jaw muscle and the participants' psychological scores. A single EMG change score was calculated for each participant during the hypertonic saline infusion for each jaw muscle and in each of the opening and closing phases of each jaw movement task. As every participant was able to displace their jaw 7 mm across all movement tasks, the change score at 7 mm of displacement (log RMS at 7 mm – log RMS at 0 mm) was used. The statistical software used was SPSS version 19 (IBM Statistics). $P < .05$ was considered to reflect statistical significance.

Results

As previously reported for the same participants who were infused with both solutions,[21] 5% hypertonic saline infusion into the RAT resulted in significantly more pain (mean [SD] VAS = 47.1 [6.6] mm) than during infusion of isotonic saline (9.0 [0.7] mm) (paired samples *t* test, $P = .0002$). For the isotonic saline infusion, 5 of the 14 participants reported VAS scores of 0, and only 2 reported scores > 20 out of 100 mm (both < 30/100 mm). Post hoc power analyses based on a repeated-measures F test with one group, an alpha of .05, a moderate effect size, and three measures determined the study achieved 0.64 power.

Qualitative Observations on Individual Participants' EMG Data

There was interparticipant variation in the effects of hypertonic or isotonic saline infusion on EMG activity during a particular jaw movement. For example, during the closing phase of free and standardized chewing, the EMG activity of the LAT muscle during hypertonic saline infusion could be greater than (Fig 2a), less than (Fig 2b), or approximately equal to (Fig 2c) the average RMS activity during isotonic saline infusion. During the opening phase of the standardized opening/closing jaw movements, the average RMS activity of the RDIG during hypertonic saline infusion could be greater than (Fig 2d), equal to, or mostly less than (Fig 2e) the average RMS activity during isotonic saline infusion.

Quantitative Analysis of Grouped Jaw Muscle EMG Activity

There were significant differences in EMG activity for most of the recorded jaw muscles for hypertonic saline infusion compared to baseline (83% of comparisons) and for isotonic saline infusion compared to baseline (73%) (Table 1). The remainder of the outline of results will focus on the difference between hypertonic saline infusion and isotonic saline infusion as representing the net effect of pain on EMG activity (see Discussion).

Effect of the Jaw Tasks

The presence of a significant difference in the EMG activity of a jaw muscle between hypertonic and isotonic saline infusions could vary with the task in which the muscle participated (Table 2). The effect of pain observed and its direction were independent of whether the muscle was an agonist or antagonist in the task. For example, there were no significant

Table 1 Differences in EMG Activity for All Muscles in Different Jaw Tasks Between Hypertonic Saline and Isotonic Saline Infusion Blocks Compared to Baseline Block 1

Jaw tasks	Muscles	Hypertonic saline—baseline (*P* value)	Isotonic saline—baseline (*P* value)
Free chewing			
Opening phase	RAT	.928	.156
	LAT	**.003**	.128
	RMAS	**.041**	.210
	LMAS	.944	**.000**
	RDIG	.828	.395
Closing phase	RAT	**.002**	**.000**
	LAT	**.000**	**.000**
	RMAS	**.000**	**.000**
	LMAS	**.000**	**.000**
	RDIG	**.000**	**.000**
Standardized chewing			
Opening phase	RAT	**.000**	**.002**
	LAT	**.000**	**.000**
	RMAS	**.000**	**.000**
	LMAS	**.000**	**.000**
	RDIG	**.000**	**.000**
Closing phase	RAT	**.026**	**.014**
	LAT	**.029**	.787
	RMAS	**.001**	.990
	LMAS	**.000**	**.009**
	RDIG	**.000**	**.001**
Standardized opening/closing jaw movements			
Opening phase	RAT	**.000**	**.000**
	LAT	**.000**	**.000**
	RMAS	**.000**	**.000**
	LMAS	**.000**	**.000**
	RDIG	.065	**.001**
Closing phase	RAT	**.003**	.349
	LAT	**.029**	.629
	RMAS	**.000**	**.000**
	LMAS	**.000**	**.000**
	RDIG	.286	**.000**

Significant values are bolded ($P < .05$). RAT = right anterior temporalis; LAT = left anterior temporalis; RMAS = right masseter; LMAS = left masseter; RDIG = right digastric.

Table 2 Differences in EMG Activity for All Muscles in Different Jaw Tasks Between Hypertonic Saline (h) and Isotonic Saline (i) and Consistency of Changes with Pain Adaptation Model (PAM) and Vicious Cycle Theory (VCT)

Jaw tasks	Muscles	Hypertonic saline—isotonic saline (*P* value)	PAM	VCT
Free chewing				
Opening phase	RAT	.173	Ø	Ø
	LAT	**.000, h > i**	✓	✓
	RMAS	.482	Ø	Ø
	LMAS	**.000, i > h**	Ø	Ø
	RDIG	.522	Ø	Ø
Closing phase	RAT	.370	Ø	Ø
	LAT	.292	Ø	Ø
	RMAS	.131	Ø	Ø
	LMAS	.947	Ø	Ø
	RDIG	**.000, i > h**	Ø	Ø
Standardized chewing				
Opening phase	RAT	.979	Ø	Ø
	LAT	.273	Ø	Ø
	RMAS	.200	Ø	Ø
	LMAS	.306	Ø	Ø
	RDIG	.052	Ø	Ø
Closing phase	RAT	.706	Ø	Ø
	LAT	**.002, h ≥ i***	Ø	✓
	RMAS	**.001, h > i**	Ø	✓
	LMAS	**.024, h > i**	Ø	✓
	RDIG	.308	Ø	Ø
Standardized opening/closing jaw movements				
Opening phase	RAT	.178	Ø	Ø
	LAT	.249	Ø	Ø
	RMAS	.104	Ø	Ø
	LMAS	.455	Ø	Ø
	RDIG	.104	Ø	Ø
Closing phase	RAT	**.045, i > h**	✓	Ø
	LAT	.102	Ø	Ø
	RMAS	**.007, i > h**	✓	Ø
	LMAS	.393	Ø	Ø
	RDIG	**.000, h > i**	✓	✓

Significant values are in bold ($P < .05$). ✓ = consistent with expected pain-related EMG effects as proposed by the Vicious Cycle Theory or the Pain Adaptation Model; Ø = not consistent with previous theories; RAT = right anterior temporalis; LAT = left anterior temporalis; RMAS = right masseter; LMAS = left masseter; RDIG = right digastric. h > i or h < i indicates that EMG activity during hypertonic saline was greater than (or less than) EMG activity during isotonic saline infusion. *LAT EMG activity during hypertonic saline infusion was the same as during isotonic saline infusion at 20-mm displacement (see Methods).

differences between hypertonic and isotonic saline infusions in EMG activity during the opening phase of standardized chewing or of the standardized jaw movements for any recorded jaw muscle, but there were significant differences in EMG during the opening phase of free chewing for the LAT and LMAS: the muscle activity was greater during hypertonic infusion than isotonic infusion for LAT, but was less for LMAS, and both muscles were antagonists in this movement. Also, there was significantly greater muscle activity during hypertonic than isotonic saline infusion in the closing phase of standardized chewing for the LAT, RMAS, and LMAS (Fig 3), although there were no significant differences in free chewing for these same muscles, which were all agonists in these phases of both tasks (Table 2). In the closing phase of the standardized jaw movements, significant differences were noted for the RAT, RMAS, and RDIG. The EMG activity of the RAT and RMAS (both agonists) was significantly lower during hypertonic saline infusion than during isotonic saline infusion. While EMG activity of the RDIG was unaffected by pain for the opening

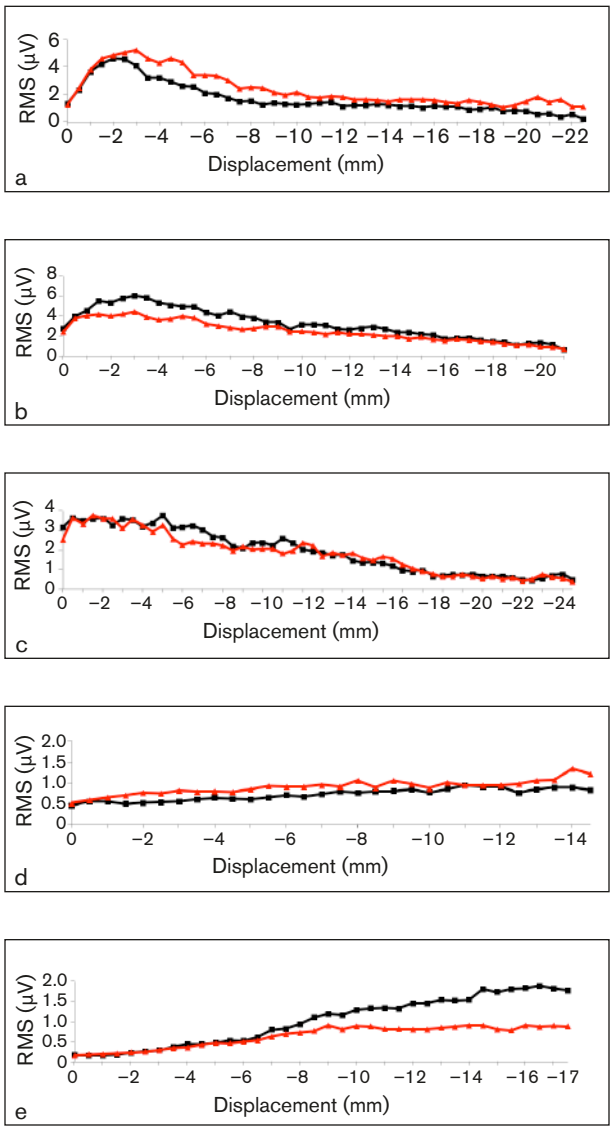

Fig 2 The mean EMG root mean square (RMS) values for the left anterior temporalis muscle at each 0.5-mm increment of mid-incisor point displacement in the z axis (superior-inferior) is shown during the closing phase of free chewing for **(a)** participant 2 and **(b)** participant 6, and during the closing phase of standardized chewing for **(c)** participant 6. The mean EMG RMS values for the right digastric muscle during the opening phase of the standardized opening/closing jaw movements are shown for **(d)** participant 5 and **(e)** participant 10. Black squares: during isotonic saline infusion; red triangles: during hypertonic saline infusion.

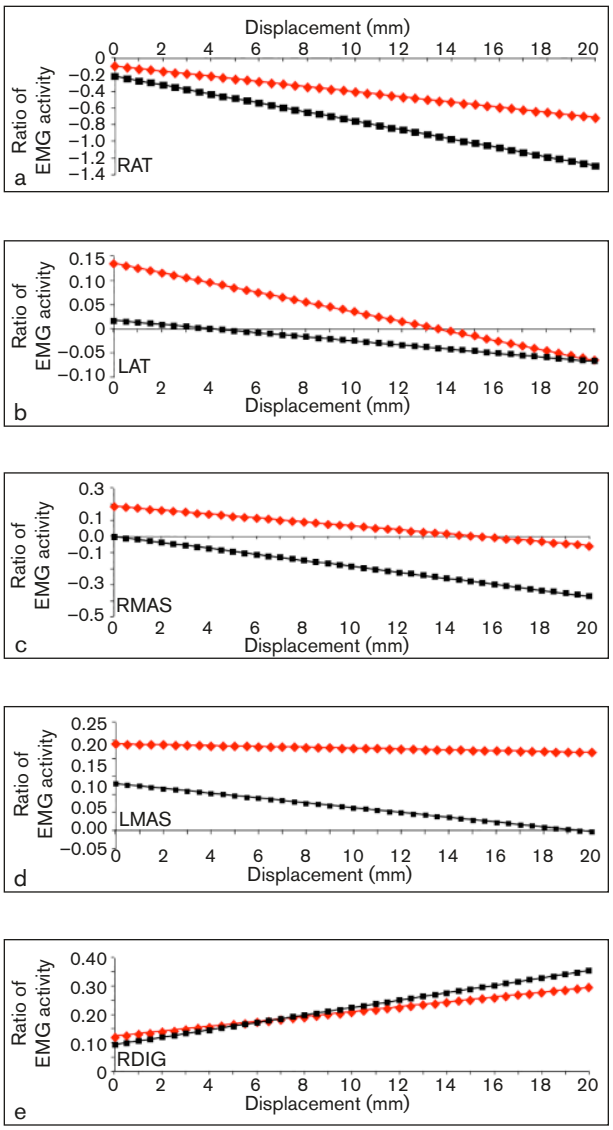

Fig 3 The ratio of EMG activity between hypertonic saline infusion and baseline (red diamonds) and between isotonic saline infusion and baseline (black squares) for the right anterior temporalis (RAT), left anterior temporalis (LAT), right masseter (RMAS), left masseter (LMAS), and right digastric muscle (RDIG) during the closing phase of standardized chewing. The x axis is the displacement (mm), and the y axis is the root mean square (RMS) ratio. There were significant differences for LAT, RMAS, and LMAS between hypertonic saline and isotonic saline infusions (see Table 2).

phase of any task, it was significantly lower during the closing phase of free chewing but significantly greater during the closing phase of the standardized jaw movements (Table 2).

Effect of Jaw Displacement

The direction of the difference in EMG activity during hypertonic saline–induced pain did not vary with jaw displacement. Figure 3 shows grouped data for the ratio of EMG activity between hypertonic saline infusion and baseline and between isotonic saline infusion and baseline throughout the closing phase of standardized chewing. For the muscles where there was a significant difference between the hypertonic and isotonic saline infusions—the LAT, RMAS, and LMAS (Table 2)—the hypertonic saline/baseline ratio was always greater than the isotonic saline/baseline ratio.

Relationships with Psychological Measures

Multiple regression analyses of each participant's psychological scores across the changes in the EMG activity from postural position to 7-mm jaw

displacement and during hypertonic saline infusion found three relationships. The change in activity of the LAT muscle during the opening phase of the jaw movement task was a significant predictor (r^2 = 0.48, P = .012) of the participant's PCS score, while during the closing phase, the change was a significant predictor (r^2 = 0.44, P = .019) of the DASS 21 anxiety subscale. The change in RMAS activity during the closing phase of the opening and closing task was a significant predictor (r^2 = 0.38, P = .034) of the FPQ III fear of medical pain subscale. No other significant associations were found.

Discussion

The first main finding of this study was that the presence of a significant difference in the EMG activity of a jaw muscle between hypertonic saline infusion and isotonic saline infusion into the RAT could vary with the task in which the muscle participated, irrespective of whether the muscle was an agonist or an antagonist. Second, where significant effects were observed, the direction of the difference in muscle activity during hypertonic saline–induced pain did not vary with jaw displacement. Third, the effects on muscle activity of an infusion could vary from participant to participant. Finally, some associations were observed between masseter and anterior temporalis muscle EMG activity during pain and the psychological construct scores. These findings support the overall hypothesis, except that an association with jaw displacement magnitude was not observed. These findings are generally consistent with some of the findings from an earlier study of experimental masseter muscle pain on jaw tasks[21] and therefore do not support earlier models of pain-motor interaction (namely, the Vicious Cycle Theory and Pain Adaptation Model), but do lend support for more recent models.[3–6]

Present Findings in Relation to Previous Findings

The significant isotonic saline/baseline EMG differences found in the present study can be attributed, as previously argued,[1,22] to a volume effect of the injected solution reflexively influencing motor activity through activation of low-threshold afferents or the possible effect on EMG activity of a low pain level. Low levels of pain have been previously reported with isotonic saline infusions.[11,32]

A previous related study[21] from the same individuals showed that there were no significant differences in jaw movement kinematic variables for any of the three tasks between hypertonic saline and isotonic saline infusions and no significant correlations between psychological scores and kinematic variables during hypertonic saline infusion. In the present study, there are two possible explanations for the presence of changes in jaw muscle activity during experimental RAT pain in the absence of the kinematic changes described in the previous study. First, while there may have been changes in kinematic parameters, the methodology used in the previous publication[21] was not sufficiently refined to identify significant differences; for example, there may have been differences in the shapes of the chewing cycles, and these were not quantified in the present nor the previous study. Alternatively, if indeed there are minimal or no differences in kinematic features, then the changes in muscle activity during hypertonic saline in comparison with isotonic saline infusion suggest that there is a reorganization of muscle activity within and/or across the jaw muscles during pain so that the movements can still be carried out with normal kinematic parameters. The compensatory changes in muscle activity throughout the jaw motor system may allow kinematic performance at the level of or approaching the level apparent in the absence of pain. If this latter interpretation of the findings in relation to the previous study[21] is indeed correct, then the present data tentatively extend the earlier evidence for reorganization within the masseter during experimental noxious stimulation[48] to reorganization occurring across muscles. These findings are consistent with a recent model of pain-motor interaction.[4]

Consistent with some previous studies,[1,22,49,50] there was evidence for task dependency in the effect of pain on the activity of some jaw muscles. Any effect appeared to be independent of whether the muscle acted as an agonist or an antagonist. A possible reason for the differences in pain effects within the same agonist (or antagonist) muscle across different tasks and between different agonist (or antagonist) muscles in the same task may reflect the differing mix of somatosensory afferent and descending motor inputs to the motoneurons of different muscles during the different tasks, which are likely to differentially modulate the effects that nociceptive inputs have on the neural networks driving these motoneurons. For example, opening and closing jaw movements are dominated by descending inputs from the primary motor cortex driving the movement, while free chewing is largely driven by the brainstem central pattern generator.[51]

It is difficult to compare the effects of pain on jaw muscles noted in the present study with a previous study that employed experimental masseter pain,[1] as some of the tasks were different, some different jaw muscles were recorded, and there was a different group of participants. Nonetheless, the previous study[1] showed that experimental masseter pain, in

comparison with isotonic saline infusion, resulted in significantly different muscle activity in the RMAS, LMAS, and RDIG muscles during the opening phase of the standardized jaw movement task. There were no significant effects of pain noted in the same muscles for the same task during RAT experimental pain in the present study. Further studies are needed to confirm these possible differences between experimental anterior temporalis or masseter noxious stimulation, but the data comparison provides cautious support for more recent models of pain-motor interaction that propose that the site of the noxious stimulation may influence the motor effects observed.[4]

Present Findings in Relation to Pain Adaptation Model and Vicious Cycle Theory

An assessment was made of whether changes in muscle activity during hypertonic saline infusion vs isotonic saline infusion were consistent with the Pain Adaptation Model or the Vicious Cycle Theory (Table 2). This analysis showed that most of the comparisons were consistent with neither the Model nor the Theory. Most of the nonconsistent comparisons occurred for nonsignificant differences between hypertonic and isotonic saline. Given the possibility of a Type II error for the nonsignificant comparisons, the remaining nine significant differences were analyzed. Of these nine, there were four significant effects of pain that were consistent with the Pain Adaptation Model, five that were consistent with the Vicious Cycle Theory, and two that were consistent with both.

For example, the activity of the LAT muscle (P = .000), an antagonist for the opening phase of free chewing, was higher during hypertonic saline infusion than isotonic saline infusion. This increase is in agreement with the Pain Adaptation Model,[7] as well as with other studies.[8,14,16,18,52] The decrease in RAT and RMAS muscle activity during the closing phase of standardized opening and closing movements during hypertonic saline infusion vs isotonic infusion is also consistent with the Pain Adaptation Model, which proposes decreased agonist activity during pain.[7,11,17,52,53] It should be pointed out that while these changes in muscle activity appear consistent with the Pain Adaptation Model, there were no clear changes in kinematic features during the pain.[21]

Associations with Psychological Variables

A significant relationship was noted between anxiety and changes in jaw-closing muscle EMG activity. This relationship is in line with recent findings in individuals with low back pain of significant positive correlations between paraspinal muscle activity and pain, disability, depression, anxiety, and catastrophizing.[54] A significant relationship was also noted for fear of medical pain and changes in RMAS muscle activity. An earlier study of low back pain demonstrated a significant positive association between pain-related fear and lumbar muscle activity, and this association was implicated as a factor in the development and maintenance of low back pain.[55] Another study of experimentally induced pain and fear of pain was associated with a decrease in erector spinal muscle activity during gait.[56] Finally, the significant associations between PCS scores and jaw-closing muscle activity during hypertonic saline infusion may help explain the slower jaw velocity and greater variability of repetitive jaw movements in experimental pain in higher-pain–catastrophizing individuals that have been previously reported.[32]

These associations in this experimental pain model between psychological scores and muscle activity are important given that psychological factors may underlie or contribute to the development of TMD chronicity.[57–59] Given these demonstrated associations between muscle activity and some psychological scores, one interpretation of the present findings of significant differences in the EMG activity of jaw muscles between hypertonic and isotonic saline infusions in some jaw tasks is that reorganization of muscle activity during pain is occurring, and this reorganization is being influenced or modulated by psychological factors. This is an avenue for future investigation.

Study Limitations

Caution is needed when extrapolating the present findings to clinical ongoing TMD pain, and the effects of pain observed might not reflect muscle activity in clinical orofacial pain. Indeed, the findings serve to inform how the jaw motor system of a group of highly selected individuals reacts to an acute episode of expected, short-lived pain. This is quite different than an acute unexpected episode of clinical pain or chronic pain. It is likely that in this experimental pain model, the participants were highly motivated and determined to complete the tasks despite the pain. Future studies could explore more clinical samples. Another limitation is that there is the possibility of some carry-over effects of a previous infusion of hypertonic saline on subsequent task recordings. However, these possible effects are unlikely to be a problem in the present study given the alternating sequence of infusions between participants. In addition, a recent study[60] has provided no evidence for persistent postpain effects on jaw movement and jaw muscle activity, at least during chewing. Nonetheless, future studies could address this potential problem by having a postexperiment control.

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