Experimental Jaw Muscle Pain Increases Pain Scores and Jaw Movement Variability in Higher Pain Catastrophizers

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Aims: To investigate differences between higher and lower pain catastrophizers in the effects of hypertonic saline–evoked jaw muscle pain on pain perception and jaw movement. **Methods:** Repetitive open/close jaw movements were recorded in 28 asymptomatic participants (20 men, 8 women; ages 25 to 62 years) during continuous infusion of 5% hypertonic saline or isotonic saline into the right masseter muscle. All participants completed the McGill Pain Questionnaire; the Depression, Anxiety and Stress Scales; and the Jaw Function Limitation Scale. They were divided into two groups depending on the median Pain Catastrophizing Scale score. Statistical analyses involved multivariate analysis of variance, independent samples or paired *t* tests, and Pearson correlations (statistical α: $P < .05$). **Results:** Pain intensity, unpleasantness, perceived area, and pain rating indices were significantly ($P < .05$) elevated in higher pain catastrophizers during hypertonic saline–evoked pain in comparison with lower catastrophizers. The higher catastrophizers exhibited significantly ($P < .05$) slower jaw velocity than the lower catastrophizers during hypertonic saline infusion in comparison with IS infusion. In comparison with lower catastrophizers, there was a significantly greater change in the percentage of coefficient of variation between hypertonic saline and isotonic saline infusions in higher catastrophizers for closing velocity and opening and closing amplitude. **Conclusion:** The increased reported pain intensity, pain areas, and pain rating indices are consistent with enhanced central sensitization processes in high-catastrophizing individuals. The slower velocity and greater variability of repetitive jaw movements in higher pain catastrophizing individuals in acute experimental pain may reflect changes in motor coordination as an example of avoidance behavior for the jaw motor system. *J Oral Facial Pain Headache 2014;28:191–204. doi: 10.11607/ofph.1211*

Key words: *jaw movement, orofacial pain, Pain Adaptation Model, pain catastrophizing, pain intensity*

Temporomandibular muscle and joint disorders (TMD) are musculoskeletal disorders affecting the jaw motor system and are the most common nondental orofacial pains.[1] Psychological factors play a critical role in TMD and other musculoskeletal pains.[2,3] Catastrophizing has received considerable recent attention in relation to chronic musculoskeletal body pain[4–10] and experimental pain.[11] High-catastrophizing individuals tend to focus excessively on a pain sensation (rumination), exaggerate its threat (magnification), and experience a sense of helplessness.[9,12] Less attention has been given to catastrophizing in TMD patients, although there is emerging evidence for its role in pain persistence, disability, and treatment failure in TMD.[13–17]

Catastrophizing is thought to play a critical role in the transition from acute to chronic pain.[9,12] The mechanisms whereby catastrophizing interacts with somatosensory and motor systems are unclear, however, and an experimental paradigm may clarify details of this transition. Explanatory models implicate enhanced central sensitization processes in nociceptive pathways within the central nervous system in high-catastrophizing individuals[9,18–21] as well as changes to

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motor-unit recruitment patterns due to central effects of catastrophizing on motor pathways.[12,22]

Central sensitization processes in nociceptive pathways result in changes in the response properties of central neurons, including enlargement of receptive field sizes, increased firing rates, and lowered thresholds of second-order neurons in both the spinal cord dorsal horn[23] and in the trigeminal brainstem sensory nuclear complex.[24] If catastrophizing enhances central sensitization mechanisms, then lowered thresholds for pain perception, alterations to the quality of the subjective pain experience, enlarged pain areas, and enhanced pain intensity could be expected clinically. With the exception of enlargement of perceptual areas of pain, many of these features have been demonstrated in high-catastrophizing individuals in the spinal system[4–9] but not in the orofacial motor system. The first aim of the present study was to investigate differences between higher and lower pain catastrophizers in effects of hypertonic saline–evoked jaw muscle pain on pain perception. The first hypothesis was that, during experimental jaw muscle pain, individuals scoring higher on pain catastrophizing exhibit significantly greater pain intensity and unpleasantness scores, perceived areas of pain, and pain rating indices in comparison with lower catastrophizers.

In terms of effects of pain catastrophizing on motor pathways, the Fear-Avoidance Model of Musculoskeletal Pain[12,25,26] implicates catastrophic interpretations of pain as giving rise to pain-related fear and safety-seeking behaviors, such as avoidance and escape, that in the short term may be adaptive but in the long term can lead to disuse, disability, depression, and more pain.[12] These behaviors may manifest as reductions in amplitudes and/or velocities of movement,[22,27–29] or as altered or disordered motor coordination[12,22] that might involve a different motor-unit recruitment pattern. Pain-catastrophizing scores have been shown to predict peak torques in patients with low-back pain,[28] and pain-related fear is significantly associated with electromyographic (EMG) activity and velocity in patients with low-back pain.[22,29] In terms of central mechanisms, pain-catastrophizing scores correlate with pain-related activations within cerebral regions involved in motor response/planning, including thalamus, putamen, and premotor cortex,[30] and this may be a factor influencing the motor effects of catastrophizing.

In the orofacial motor system, it has been shown that experimental or clinical orofacial pain may not always be associated with reductions in the amplitude or velocity of jaw movements during chewing[31–34] but may be associated with increased jaw movement variability.[34,35] Increased variability suggests changes to motor-unit recruitment patterns that may be of relevance in the acute to chronic pain transition. The orofacial motor system may therefore be an appropriate model system to study the possible effects of catastrophizing on changes in motor coordination occurring in pain. The second aim of this study, therefore, was to determine differences between higher and lower pain catastrophizers in the effects of hypertonic saline–induced jaw muscle pain on jaw movement. The second hypothesis was that safety-seeking or avoidance behaviors in the jaw motor system in individuals with higher catastrophizing scores may not manifest as smaller and slower jaw movements, but rather as changes to motor coordination. Knowledge of these and other factors would be of value for future studies exploring the role of these behaviors in the transition from acute to chronic pain.

Materials and Methods

Twenty-eight subjects without signs and symptoms of TMD were recruited for this study (20 men, 8 women; ages 25 to 62 years) through flyers and personal approach by one of the coauthors who was not in any relationship with the prospective volunteer. All volunteers were given a brief outline of the experimental methods but were not informed about the sequence of saline infusion (see below) nor that associations were being studied between catastrophizing and other variables. Individuals were not financially remunerated for their participation. The Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD)[36] were used to verify the absence of signs and symptoms of TMD. All subjects gave informed consent. Experimental procedures were approved by the Western Sydney Local Health District Human Ethics Committee of Westmead Hospital and the Human Ethics Committee of the University of Sydney. Some of the procedures have been described in detail previously.[32,37–39]

Inclusion criteria were Angle's Class 1 occlusion and a complete permanent dentition except for the third molar teeth. General exclusion criteria were pregnancy, high blood pressure, presence of systemic musculoskeletal pain disorders (eg, fibromyalgia, inflammatory joint disease), serious systemic disease (eg, current malignancies), medications for chronic diseases (eg, psychiatric conditions, chronic pain conditions), or other medications that might influence response to pain.

Measures

The Pain Catastrophizing Scale (PCS). The PCS[40] assesses negative cognitive and affective reactions to pain. Thirteen questions covering three subscales (magnification, rumination, and helplessness) capture

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a person's orientation toward noxious stimuli and/or previous memories of pain. Each of the 13 questions is rated on a 5-point Likert scale ranging from 0 (not at all) to 4 (all the time), and the total score ranges from 0 to 52.

Many investigations have supported the reliability (eg, Chronbach's alpha ≥ 0.75) and validity (eg, PCS scores are highly correlated with pain severity and interference measures) of the PCS as a measure of pain-related catastrophic thinking in various disorders.[40–42] The PCS was completed after the experiment before pain declined to zero, because many of the participants were young, healthy adults with limited pain experience, and therefore it was considered best to use in the analysis a PCS score based on the pain experienced at the experimental pain session. PCS scores were also collected before the experiment, and the two sets of scores were not significantly different ($P > .05$) but were highly correlated (Spearman's rank correlation coefficient $r = 0.984$; $P = .00001$).

The investigators chose to dichotomize the PCS score because of the extensive evidence suggesting higher scores (eg, ≥ 16) on the PCS are associated with an increased risk of poorer outcomes with pain-management strategies as well as evidence for enhanced temporal summation.[7,42,43] For example, in a cohort study[42] of 140 patients undergoing knee arthroplasty, the dichotomized PCS score was the only consistent psychological predictor of poor pain and function outcomes. In another study,[7] greater temporal summation of thermal pain was observed in a high-catastrophizing group in comparison with a low-catastrophizing group divided by median split. In the present study, PCS scores ranged from 2 to 31, and those subjects scoring a PCS at or above the median PCS score for the sample (ie, 15) were assigned to a higher pain-catastrophizing group (10 males, 3 females, 33.0 ± 9.9 years), whereas those who scored < 15 were assigned to the lower pain-catastrophizing group (10 males, 5 females, 35.1 ± 9.3 years).

Depression, Anxiety, and Stress Scales (DASS). The DASS is a reliable and well-validated scale[44] that measures the cognitive and affective dimensions of psychological distress. It has three scales (depression, anxiety, and stress) and 42 items. Symptoms in the past week are rated from 0 ("not at all") to 3 ("most of the time"). The total scores for each scale consist of the sum of the items, and this instrument was scored before the experiment.

The Jaw Function Limitation Scale (JFLS). The JFLS is a set of 20 questions developed with the use of Rasch analysis, which measures the constructs of limitations in mastication, jaw mobility, and verbal and emotional expression and a global function limitation score.[45] This instrument was scored before the experiment.

The McGill Pain Questionnaire (MPQ). The MPQ[46] consists primarily of three major classes of word descriptors—sensory, affective, and evaluative—that are used by patients to specify subjective pain experience. The questionnaire was designed to provide quantitative measures of clinical pain that can be treated statistically. The three major measures are: *(1)* the pain rating index (PRI), based on two types of numerical values that can be assigned to each word descriptor; *(2)* the number of words chosen; and *(3)* the present pain intensity based on an intensity scale of 1 to 5. Immediately after the pain had declined to zero following cessation of the hypertonic or isotonic saline infusion, volunteers completed the MPQ by recalling their pain experience.

Area of Pain Sensation. The areas of pain spread were evaluated using a 2 x 2-mm box grid. This grid was superimposed on the pain maps drawn by each subject, and the number of boxes covered partly or completely by the pain region outlined in each subject was counted and then multiplied by 4 to obtain the data in square millimeters. As the map used by each subject was approximately one-third the size of the head and neck, this number was scaled to the approximate size of the face by multiplying by 3.

Jaw Movement Recording and Visual Feedback

An optoelectronic jaw-tracking system (JAWS-3D) recorded the movement of the mandible in 6 degrees of freedom (sampling rate: 67 Hz/s).[47] This system displayed the mid-incisor point, ie, the point between the incisal edges of the mandibular central incisors, as a marker on a video screen in front of the subject. When the subject opened his/her jaw, this marker moved downward on the video screen. A linear bank of 10 light-emitting diodes (LEDs) was also placed on the video screen about 5 cm away from the mid-incisor point marker. Each LED was successively illuminated in sequence from the first to the last LED and back again; each LED illumination sequence was repeated at a rate of a repetition per 900 ms. The purpose of the LEDs was to assist in standardizing the rate of the jaw movement within subjects (see below). Custom-made metal clutches, temporarily attached to two or three maxillary and mandibular anterior teeth with cyanoacrylate adhesive, supported the target frames of the tracking system.

Repetitive Open-Close Jaw Movements

Recordings of jaw movement were made during a jaw task that consisted of repetitive open-close jaw movements. Each cycle of this task required subjects to lower their mandible until their lips were about to separate and then to raise their mandible to light tooth

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contact. This movement was repeated in time with the target LEDs, which oscillated at a rate of 900 ms/cycle (see above)[48] and for a duration of 30 seconds. Because the clutches prevented complete lip competence, opening displacement magnitude was standardized within each subject by measuring, before the clutches were secured to the teeth, the displacement between two arbitrary points on the face at the end of the opening phase and just before the lips were about to separate. After attaching the clutches, this amount of opening displacement was replicated by the subject monitoring the opening movement of their mid-incisor point dot to a marker at the required amount of opening displacement. Therefore, each opening movement amplitude and speed was standardized within a subject, and although speed was standardized between subjects, amplitude was not. Each 30-second jaw task was preceded by a rest period of 30 seconds during which the jaw was kept in the postural (rest) position; the combined 30-second rest period and 30-second jaw-task period was called a jaw-task sequence (Fig 1). Each jaw-task sequence was repeated six times under three conditions: baseline pretrial recordings that were done to familiarize participants with the jaw task (see below), hypertonic or isotonic saline infusion, and then isotonic or hypertonic saline infusion (Fig 1). The same participants performed the same jaw tasks during magnetic resonance imaging (MRI) scanning for the purpose of comparing the MRI, jaw movement, and EMG data of masseter, temporalis, digastric, and sternocleidomastoid muscles (to be reported). The six sequences were designed to facilitate the recording of brain activity changes in association with the tasks. Subjects were instructed not to swallow or talk during the recording throughout the experiment.

Induction and Assessment of Jaw Muscle Pain

Subjects sat in a relaxed, alert manner in a chair without any head support. An intravenous catheter (JELCO, 22 gauge × 1 inch, Smiths Medical ASD) was placed via a needle in the right masseter muscle midway between its upper and lower border.[39,49] The needle was retracted at bone contact or at 2-cm depth if there was no bone contact, and the catheter remained in the muscle. The catheter was connected via a tube (extension set with polyethylene inner line, 75 cm, 0.7 mL) to an infusion pump (IVAC Model P2000) with a 10-mL syringe. Subjects were given standardized instructions and were unaware of which solution was about to be injected (single blind). To avoid sequence effects, the order of injection of hypertonic saline and isotonic saline into the masseter muscle was alternated from subject to subject.[39] Experimental jaw muscle pain was induced by tonic infusion of 5% hypertonic saline (Pharmalab, Lane Cove) into the right masseter muscle. A bolus infusion of 0.2 mL hypertonic saline (or isotonic saline, 0.9% NaCl) was infused over 20 seconds to rapidly achieve the target pain intensity of 4 to 6 as measured on a 0- to 10-point numeric rating scale (NRS). The lower end of the scale was marked "no pain" and the upper end was marked "the worst imaginable pain." Continuous infusion was maintained by the infusion pump with a steady infusion rate of 4 mL/h for 3 minutes and then increased to 6 mL/h for the last 3 minutes to maintain moderate pain intensity. A NRS score was taken after every jaw-task sequence, and the score was used to increase or decrease the infusion rate (by 1 to 4 mL/h) during the subsequent rest period to maintain pain intensity of 4–6/10. Target NRS scores of 4–6/10 were chosen because these are comparable to pain-intensity levels reported in previous studies of experimental pain.[32,50–52] With this standard protocol, it was possible to keep pain-intensity levels relatively constant throughout the 6-minute recording period. The volume of hypertonic saline infused depended on the volume needed to achieve the target NRS score of 4–6/10. The infusion of isotonic saline was used as a control for possible EMG and/or jaw movement effects from volumetric change within the muscle. The rate for the isotonic saline infusion was set at the rate of 4 mL/h for 3 minutes and then increased to 6 mL/h for the last 3 minutes when isotonic saline was infused first. When isotonic saline was infused second, the rate was modified to match the rate of the previous hypertonic saline infusion in that subject. The subjects were also asked to rate the unpleasantness of the hypertonic/isotonic-evoked pain sensation after every jaw-task sequence on a 0- to 10-point NRS, where 0 represented "no unpleasantness at all" and 10 "the most unpleasantness imaginable."

After completion of each saline infusion, subjects were asked to draw their maximum distribution of perceived pain on right and left lateral-profile outline pictures of the head and neck.[32] Pain referral sites on the pain maps were noted. A pain referral site was defined as a region outlined on the lateral-profile outline picture of the head and neck that was separate from and did not overlap with the area of pain spread outlined by the subject and surrounding the injection site. No referral sites were noted outside of the head and neck region. Pain effect was quantified with the MPQ[46] after each infusion was terminated. The pain rating indices for the sensory-discriminative, affective, evaluative, miscellaneous, and total dimension of pain were calculated according to previous criteria.[46] Subjects also indicated whether they noticed any changes in pain intensity between the first and last jaw-task sequence in each condition (isotonic and hypertonic saline) and if they indicated yes, whether the pain increased or decreased.

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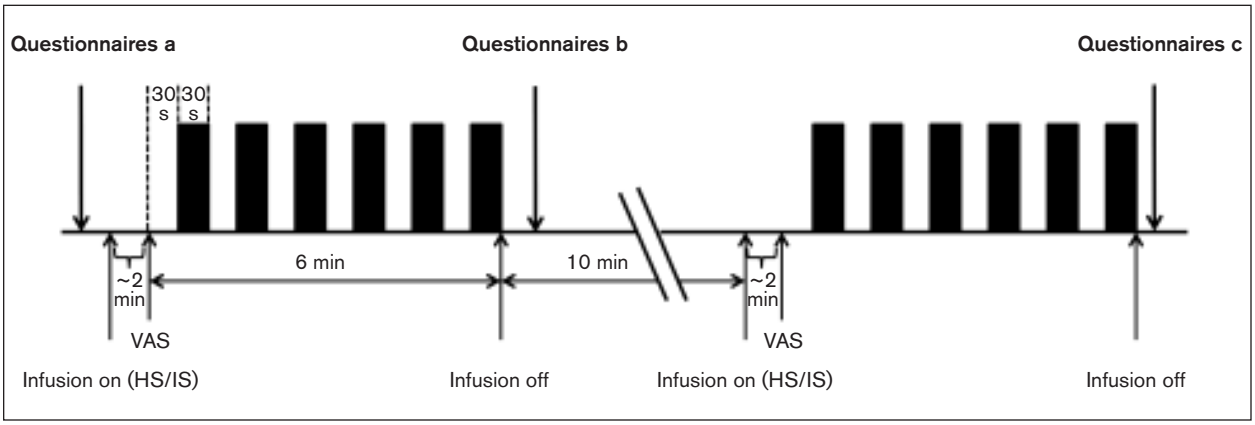

Fig 1 Schematic of the experimental protocol for isotonic saline infusion (IS) and hypertonic saline infusion (HS) conditions. Order of infusion was alternated between participants. Filled rectangles indicate 30-second periods of standardized repetitive open-close jaw movements. Each jaw-task sequence (Seq) consisted of 30 seconds of rest plus 30 seconds of repetitive open-close jaw movements. **Questionnaires a:** Research Diagnostic Criteria for Temporomandibular Disorders; Depression, Anxiety, and Stress Scales; Jaw Function Limitation Scale. **Questionnaires b:** Numeric rating scale (NRS) for pain intensity; NRS for unpleasantness; pain drawing/mapping; McGill Pain Questionnaire. **Questionnaires c:** All questionnaires from b and Pain Catastrophizing Scale. VAS, visual analog scale.

Jaw movement trajectories were recorded during the six jaw-task sequences in the following repeated-measures design (Fig 1):

1. Baseline pretrial (no infusion and prior to catheter insertion): This trial was done to familiarize subjects with the experimental setup and also to minimize any subsequent learning effects.
2. Test 1 (during hypertonic or isotonic saline infusion).
3. Test 2 (during isotonic or hypertonic saline infusion).

The sequence, hypertonic saline infusion first/isotonic saline infusion second, was alternated between subjects. All jaw-task sequences were performed on a single experimental day in a single sitting. Recordings commenced after the beginning of hypertonic saline infusion when pain intensity achieved the target of 4–6/10 on the NRS. A washout period of 10 minutes followed the end of each 6-minute hypertonic or isotonic saline infusion period, and NRS scores were zero before the next set of jaw-task sequences. The total recording session lasted about 2 to 3 hours. Consistent with previous reports,[32,53,54] there were no complications, and any pain subsided to 0/10 within a few minutes after the infusion was terminated or the catheter was removed.

Data Analysis

For the first aim and hypothesis, the following measures were compared between higher and lower catastrophizers by independent samples *t* tests for each of the isotonic and hypertonic saline infusions and for the differences in each measure between hypertonic saline and isotonic saline infusions: the total volume of the infusion, the NRS scores for pain and unpleasantness, the MPQ PRI scores, and perceived pain areas. Each PRI score on the MPQ was compared between the isotonic and hypertonic condition via paired *t* tests. The number of pain referral sites during hypertonic saline infusion was compared between higher and lower catastrophizers by an independent samples *t* test.

For the second aim and hypothesis, the following data processing was performed. For each jaw task in each subject, the mid-incisor point trajectories in the z-axis (ie, superoinferior axis) were plotted as time-displacement plots. A customized computer program divided all cycles into opening and closing phases by defining the times and amplitudes at maximum closing and maximum opening. The amplitude and velocity of each opening and closing phase of the repetitive open-close jaw movement were calculated for each 30-second period of repetitive jaw movements over the entire 6-minute period (~180 empty chewing cycles) during infusion of hypertonic saline or isotonic saline. The amplitude of movement for each opening and closing phase was calculated as the maximum displacement of the jaw between maximum closure and maximum opening for that phase along the z-axis (superior-inferior). The velocity was calculated for each opening and closing phase by dividing the amplitude by the duration of that phase. The mean opening and closing velocities and amplitudes across all trials in a subject were log transformed to

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Table 1 Total Infused Volume of Hypertonic Saline (HS) and Isotonic Saline (IS) in Lower and Higher Catastrophizers

Total volume (mL)	All subjects n = 28 (Mean ± SD)	Low catastrophizers n = 15 (Mean ± SD)	High catastrophizers n = 13 (Mean ± SD)	Mean difference (LC–HC)	95% CI		*P**
					Lower	Upper	
IS	0.9 ± 0.3	0.9 ± 0.3	0.9 ± 0.2	–0.0	–0.2	0.2	.9
HS	0.9 ± 0.2	0.9 ± 0.2	0.9 ± 0.3	0	–0.2	0.2	.8
Change (HS–IS)	–0.0 ± 0.1	–0.0 ± 0.1	–0.1 ± 0.2	0	–0.1	0.1	.5

*Significance tested by *t* test.
n = number of subjects; SD = standard deviation; LC = lower catastrophizer; HC = higher catastrophizer; CI = confidence interval.

Table 2 Pain and Unpleasantness Intensity Ratings in Lower and Higher Catastrophizers

Variables	All subjects n = 28 (Mean ± SD)	Low catastrophizers n = 15 (Mean ± SD)	High catastrophizers n = 13 (Mean ± SD)	Mean difference (LC–HC)	95% CI		*P**
					Lower	Upper	
Pain intensity							
IS	0.9 ± 0.8	0.8 ± 0.6	1.1 ± 0.9	–0.3	–0.9	0.3	.3
HS	5.1 ± 1.1	4.5 ± 0.7	5.8 ± 1.1	–1.3	–2.0	–0.6	.001
Change (HS–IS)	4.2 ± 1.3	3.7 ± 0.8	4.7 ± 1.5	–1.0	–1.9	–0.1	.04
Unpleasantness intensity							
IS	1.0 ±1.1	0.7 ± 0.9	1.3 ± 1.2	–0.6	–1.4	0.2	.1
HS	4.9 ± 1.8	3.9 ± 1.5	6.0 ± 1.4	–2.1	–3.3	–1.0	.001
Change (HS–IS)	3.9 ± 1.8	3.2 ± 1.6	4.7 ± 1.8	–1.5	–2.8	–0.2	.02

*Significance tested by independent samples *t* test.
n = number of subjects; SD = standard deviation; CI = confidence interval; LC = lower catastrophizer; HC = higher catastrophizer; IS = isotonic saline; HS = hypertonic saline.

approximate normality and to stabilize the variance and tested with a multivariate analysis of variance (MANOVA). The effect of hypertonic saline or isotonic saline infusion on the mean amplitude and velocity of each jaw movement was compared across all subjects with paired *t* tests. To assess the influence of catastrophizing on jaw movements, changes (hypertonic saline–isotonic saline; HS–IS) in opening and closing mean velocity and amplitude between hypertonic saline and isotonic saline infusion were compared between higher and lower catastrophizers by independent-samples *t* tests. The dispersion of the data within subjects for each of velocity and amplitude during opening and closing movements was expressed as the coefficient of variation (CV). Independent-samples *t* tests were used to test for differences between higher and lower catastrophizers in the percentages of CV for both opening and closing velocity and amplitude for each of isotonic saline, hypertonic saline, and differences between infusions (ie, HS–IS).

Pearson's correlations were performed between psychological measures and changes in outcome parameters between hypertonic saline and isotonic saline infusions. Chi-square tests compared proportions of words chosen between catastrophizing groups. The data were analyzed with statistical software SPSS (version 18.0; SPSS). Significance was accepted at $P < .05$ throughout all analyses.

Results

A total of 37 participants agreed to participate; 4 withdrew during the experiment because of excessive discomfort, and for 5 participants equipment failure or other technical issues prevented the completion of recordings. The data from the remaining 28 participants are included in all the analyses below. There were no significant differences in total infusion volumes between higher and lower catastrophizers (Table 1).

Pain Intensity and Psychological Variables

At baseline there were no significant differences in the DASS and JFLS scores between higher and lower catastrophizers ($P > .05$, independent samples *t* test). Tables 2, 4, and 5 summarize the effects of pain catastrophizing on pain variables. During HS infusion, higher catastrophizers exhibited significantly greater values than lower catastrophizers for pain intensity and unpleasantness intensity ratings (Table 2), for all the PRI scores on the MPQ (Table 4), for the perceived area of pain spread (Table 5, eg, Fig 2), and for the number of referral sites (higher catastrophizers, 2.8 ± 1.3 sites vs lower catastrophizers, 1.5 ± 0.5 sites, $P = .01$; eg, Fig 2). Each PRI score on the MPQ was significantly greater during hypertonic saline than isotonic saline conditions (Table 3). During hypertonic saline infusion, the most common MPQ pain descriptors were "sharp," "aching," "taut,"

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Table 3 Mean Pain Rating Indices for Sensory, Affective, Evaluative, and Miscellaneous Categories From the McGill Pain Questionnaire (MPQ) During Infusion of Isotonic Saline (IS) and Hypertonic Saline (HS)

MPQ descriptors	IS (Mean ± SD)	HS (Mean ± SD)	95% CI Lower	95% CI Upper	*P**
PRI-Sensory	2.9 ± 2.8	9.0 ± 4.3	–7.7	–4.4	.001
PRI-Affective	0.3 ± 0.8	1.3 ± 1.4	–1.5	–0.4	.001
PRI-Evaluative	0.9 ± 1.4	3.4 ± 2.4	–3.5	–1.4	.001
PRI-Miscellaneous	0.1 ± 0.4	0.9 ± 1.5	–1.4	–0.1	.018
PRI-Total	4.2 ± 3.6	14.5 ± 7.0	–12.7	–7.8	.001

*Significance tested by *t* test.
PRI = pain rating index; SD = standard deviation; LC = lower catastrophizer; HC = higher catastrophizer; CI = confidence interval.

Table 4 Analysis of Number of Words Chosen for the McGill Pain Questionnaire Pain Rating Indices in Lower Catastrophizers (LC) and Higher Catastrophizers (HC)

Pain Rating Index	All subjects n = 28 (Mean ± SD)	Low catastrophizers n = 15 (Mean ± SD)	High catastrophizers n = 13 (Mean ± SD)	Mean difference (LC–HC)	95% CI Lower	95% CI Upper	*P**
PRI-Sensory							
IS	2.9 ± 2.8	2.4 ± 2.1	3.4 ± 3.5	–1.0	–3.2	1.2	.4
HS	8.9 ± 4.3	7.3 ± 4.6	10.8 ± 3.2	–3.6	–6.7	0.5	.03
Change (HS–IS)	6.1 ± 4.3	4.9 ± 4.0	7.5 ± 4.4	–2.6	–5.9	0.7	.1
PRI-Affective							
IS	0.3 ± 0.8	0.1 ± 0.4	0.5 ± 1.1	–0.3	–1.0	0.3	.3
HS	1.3 ± 1.4	0.6 ± 1.1	2.0 ± 1.4	–1.4	–2.3	–0.5	.01
Change (HS–IS)	1.0 ± 1.3	0.5 ± 1.1	1.5 ± 1.5	–1.1	–2.0	0.1	.03
PRI-Evaluative							
IS	0.9 ± 1.4	0.9 ± 1.3	1.0 ± 1.5	–0.1	–1.2	0.9	.8
HS	3.4 ± 2.4	2.3 ± 1.5	4.6 ± 2.7	–2.3	–4.0	–0.6	.01
Change (HS–IS)	2.5 ± 2.7	1.5 ± 2.3	3.6 ± 2.8	–2.1	–4.1	–0.2	.03
PRI-Miscellaneous							
IS	0.1 ± 0.4	0	0.3 ± 0.6	–0.3	–0.6	0	.07
HS	0.9 ± 1.5	0.1 ± 0.4	1.8 ± 1.9	–1.6	–2.6	–0.6	.01
Change (HS–IS)	0.8 ± 1.6	0.1 ± 0.4	1.5 ± 2.1	–1.3	–2.4	–0.2	.02
PRI-Total							
IS	4.2 ± 4.0	3.4 ± 2.8	5.2 ± 4.3	–1.8	–4.5	1.0	.2
HS	14.5 ± 7.0	10.3 ± 4.5	19.2 ± 6.3	–8.9	–13.1	–4.7	.001
Change (HS–IS)	10.3 ± 6.3	6.9 ± 3.3	14.1 ± 6.7	–7.1	–11.2	–3.1	.001

*Significance tested by independent samples *t* test.
n = number of subjects; SD = standard deviation; PRI = pain rating index; HS = hypertonic saline; IS = isotonic saline; CI = confidence interval.

Table 5 Perceived Areas of Pain (mm^2) Drawn by Lower Catastrophizers Compared with Higher Catastrophizers for Hypertonic Saline (HS) and Isotonic Saline (IS) Infusion Conditions

	All subjects n = 28 (Mean ± SD)	Low catastrophizers n = 15 (Mean ± SD)	High catastrophizers n = 13 (Mean ± SD)	Mean difference (LC–HC)	95% CI Lower	95% CI Upper	*P**
IS	57.0 ± 40.0	44.3 ± 41.0	71.6 ± 33.7	–27.3	–56.7	2.1	.07
HS	314.5 ± 264.4	207.1 ± 141.7	438.3 ± 320.6	–231.2	–419.2	–43.2	.02
Change (HS–IS)	257.5 ± 251.1	162.8 ± 148.7	366.7 ± 303.2	–203.9	–385.5	–22.3	.03

*Significance tested by independent samples *t* test.
n = number of subjects; SD = standard deviation; LC = low catastrophizer; HC = high catastrophizer; CI = confidence interval.

"fearful," and "intense." The words "sharp," "intense," and "dreadful" were chosen significantly ($P < .05$, $< .001$, and $< .05$, respectively, chi-square test) more commonly by the higher catastrophizers group than the lower catastrophizers group.

There were significant correlations between PCS scores and pain intensity ($r = 0.55$, $P = .002$), pain unpleasantness ($r = 0.51$, $P = .006$), the change in unpleasantness scores ($r = 0.48$, $P = .032$), the changes in affective ($r = 0.55$, $P = .003$) and total

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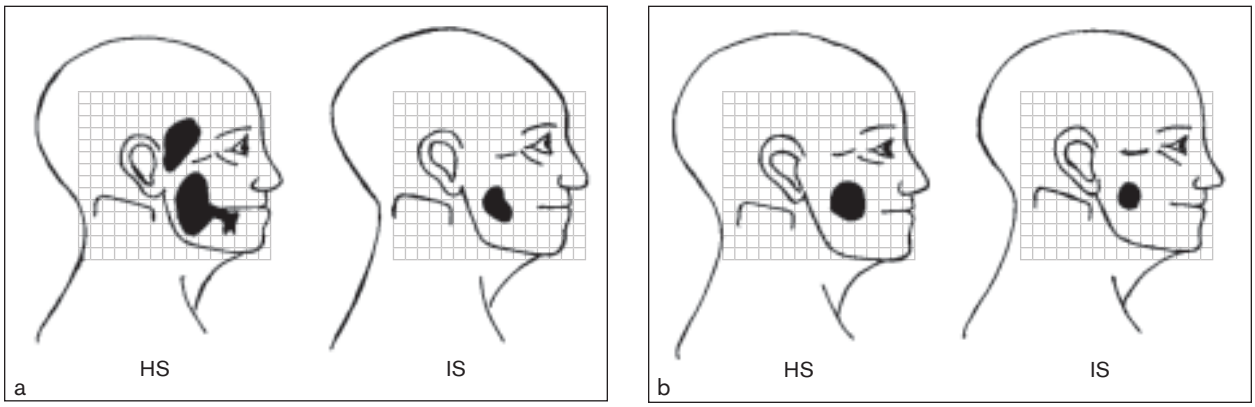

Fig 2 Representative data of pain maps from two subjects, from **(a)** high-catastrophizer group and **(b)** low-catastrophizer group, after performance of the jaw-task trial during hypertonic saline (HS) and isotonic saline (IS)–infusion conditions. The areas of pain spread were evaluated using a 2 x 2-mm^2 box grid superimposed on the subject's drawing.

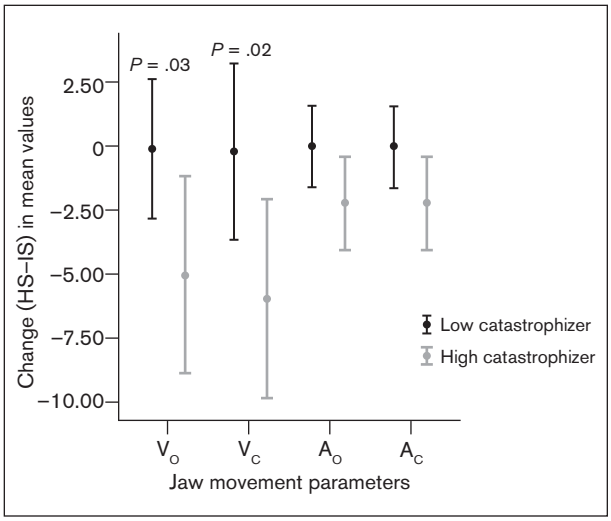

Fig 3 Mean changes (circles) and 95% confidence intervals (error bars) in opening velocity (V_O), closing velocity (V_C), opening amplitude (A_O), and closing amplitude (A_C) between hypertonic and isotonic saline infusion conditions (HS–IS) for low catastrophizers and high catastrophizers. There were significant ($P < .05$) differences between low and high catastrophizers for opening and closing velocity. Significance tested by independent samples *t* test.

Table 6 Mean Opening and Closing Velocity and Amplitude for the Experimental Conditions: Isotonic Saline (IS) and Hypertonic Saline (HS) Infusion

Kinematic parameters	IS (Mean ± SD)	HS (Mean ± SD)
Mean opening velocity (mm/s)	27.1 ± 11.1	24.7 ± 12.1*
Mean closing velocity (mm/s)	27.7 ± 12.6	24.8 ± 12.5†
Mean opening amplitude (mm)	12.3 ± 5.5	11.2 ± 5.7
Mean closing amplitude (mm)	12.3 ± 5.5	11.2 ± 5.7

*$P = .05$; †$P = .03$. Significance tested by paired samples *t* test.
SD = standard deviation.

($r = 0.49$, $P = .007$) PRI scores, the changes in perceived area of pain ($r = 0.52$, $P = .005$), and number of referral sites ($r = 0.58$, $P = .008$). There were no significant correlations between PCS scores and age ($P = 1.0$) or sex ($P = .4$).

Kinematic Variables

Across the four kinematic variables (opening and closing velocity and amplitude), the hypertonic saline outcome for a participant was on average 12.5% smaller than the isotonic saline outcome for that participant (95% confidence interval [CI] 1.5% to 24.8%, MANOVA, $P = .026$). The velocity and amplitude for both opening and closing jaw movements are shown in Table 6 for hypertonic and isotonic saline infusions. The higher catastrophizers exhibited significantly ($P < .05$, independent samples *t* tests) slower velocity than the lower catastrophizers for both opening and closing jaw movements during hypertonic saline in comparison with isotonic saline infusion (Fig 3).

During hypertonic (but not isotonic, Fig 4a) saline infusion, the percentage CV for opening amplitude ($P < .05$) was significantly greater in the higher catastrophizers than the lower catastrophizers (Fig 4b). In comparison with lower catastrophizers, there was a

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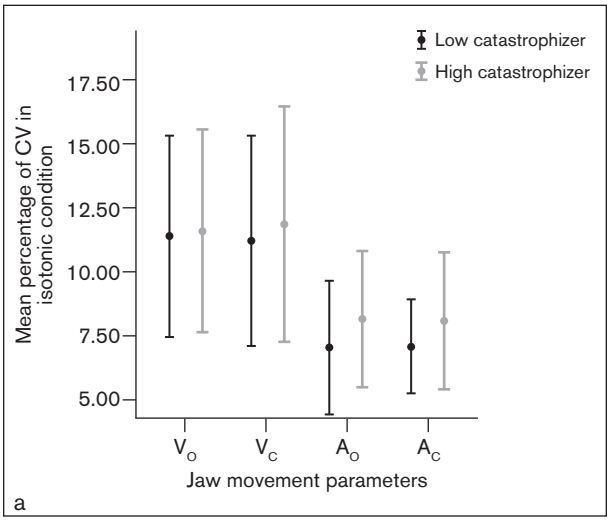

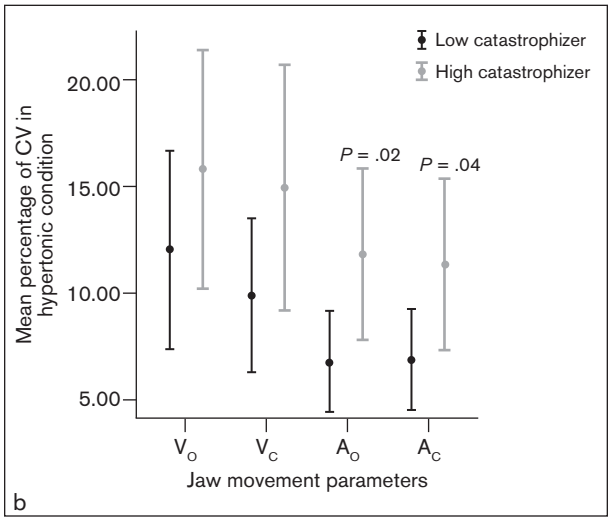

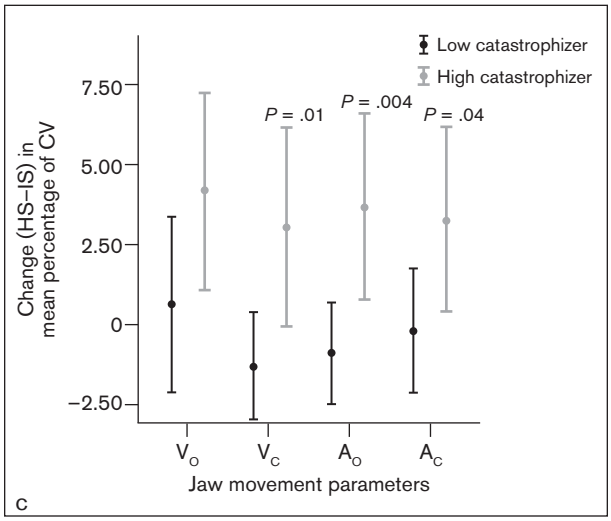

Fig 4 Percentage of coefficient of variation (CV) and 95% confidence intervals *(error bars)* for opening velocity (V_O), closing velocity (V_C), opening amplitude (A_O), and closing amplitude (A_C) in **(a)** isotonic, **(b)** hypertonic, and **(c)** hypertonic-isotonic saline infusion conditions. In each plot, all participants have been divided into the low and high catastrophizers. In Fig 4b, there are significant differences between low and high catastrophizers in terms of A_C, and in Fig 4c in terms of V_C, A_O, and A_C. Significance tested by independent samples *t* test.

significantly greater change (HS–IS) in the percentage of coefficient of variation between hypertonic and isotonic infusions in higher catastrophizers for closing velocity and opening and closing amplitude, but not opening velocity (Fig 4c). There was a significant correlation (r = 0.43, P = .02) between pain catastrophizing scores and the change (HS–IS) in CV for mean opening amplitude. There were no other significant correlations, that is, between any of the stated kinematic variables and depression, anxiety, stress or the jaw function limitation scale.

There were no significant differences (*t* test, P = .08) in the overall speed of the jaw task (number of cycles/total time) between the isotonic (1.1 ± 0.2 cycles/s) and hypertonic (0.9 ± 0.1 cycles/s) infusion conditions. There were no significant differences between higher and lower catastrophizers for the overall speed of the jaw task in isotonic, hypertonic, and the change (HS–IS) conditions.

Discussion

This study reports the association between catastrophizing, pain perception, and jaw movement during experimental jaw muscle pain. It demonstrated that, in comparison with control isotonic saline infusion, experimental jaw muscle pain resulted in a small but significant reduction in the amplitude or velocity of jaw movements during the jaw task. However, the level of catastrophic thinking was related to the variability and velocity of jaw movements during jaw muscle pain. The data are consistent with the hypothesis that safety-seeking or avoidance behaviors in the jaw motor system in individuals with higher catastrophizing scores may not manifest as smaller and/or slower jaw movements, but rather could lead to more subtle changes to motor coordination, and these changes may involve greater variability. Clarification of changes in motor coordination could be derived from EMG

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studies. In addition and in comparison with lower catastrophizers, higher catastrophizing scores were significantly associated with higher pain intensity (HS–IS: mean ± SD, 4.7 ± 1.5, $P = .02$) and unpleasantness scores, larger perceived areas of pain (mean ± SD, 366.7 ± 303.2, $P = .03$), and higher scores on all PRI values of the MPQ (except PRI-sensory). The data are consistent with the presence of enhanced central sensitization processes within the trigeminal somatosensory nervous system in higher catastrophizing individuals. These findings provide support for the view that pain catastrophizing is an important psychological contributor to the perception of orofacial pain and modulation of jaw motor function, and thereby provide support for the applicability of the Fear-Avoidance Model of Musculoskeletal Pain to the trigeminal sensorimotor system.

Experimental Masseter Muscle Pain and Standardized Jaw-Task Cycle Parameters

The Pain Adaptation Model proposes that pain leads to reduced amplitude and velocity of movement so as to promote healing,[55–57] and some previous data are consistent with this model.[33,53,55,58–60] Only mild reductions in jaw velocity and amplitude were noted in pain in the present study; other previous findings of chewing gum during experimental masseter muscle pain[32,33] and in clinical TMD pain[31,34] have observed no reductions in amplitude or velocity of jaw movement during pain. It is possible that the goal-directed nature of the jaw task in the present and some of these previous studies was able to override or reduce the inhibitory effects of nociceptive activity on motor activity, whether these inhibitory influences are acting on agonist brainstem motoneurons as proposed by the Pain Adaptation Model[55–57] and/or at supraspinal/suprabulbar levels, eg, at the level of the primary motor cortex as recently proposed.[61,62]

The data point to the emerging notion[57,63] that the association between pain and motor activity is not a hard-wired segmental or brainstem-level response as proposed by previous models (ie, Pain Adaptation Model, Vicious Cycle Theory) but is influenced by higher centers. In the present study, for example, motivation to complete the jaw task correctly may have influenced the effect of nociceptive activity on jaw amplitude and velocity, and individuals in pain can chew more quickly if necessary.[31,32,53] As previously pointed out,[31,53] this may be of significance where orofacial pain patients need to perform a demanding task, eg, clearly articulated speech in demanding work situations.

Associations Between Kinematic Variables and Catastrophizing

The PCS scores in the present study appear to be generally consistent with PCS scores reported in other datasets of asymptomatic individuals.[40] The presence of greater variability in jaw-task cycle velocity and amplitude in individuals with higher catastrophizing scores suggests changes in the pattern of muscle activation in this higher catastrophizing group. These findings are consistent in general terms with previous studies of experimental and clinical pain that have demonstrated differences between higher and lower catastrophizers in motor coordination,[22] in the total load able to be lifted,[64] and in the level of overall disability.[65–67]

The data cannot be interpreted in terms of some previous models (ie, Pain Adaptation Model, Vicious Cycle Theory). The Fear-Avoidance Model provides a better framework for the data. In terms of this model, catastrophic interpretations of orofacial pain would give rise to increased variability of jaw movement as a manifestation of safety-seeking or avoidance jaw behaviors. These behaviors may be adaptive in the short term but in the long term could lead to more pain.[12]

The neural mechanisms whereby pain catastrophizing might be exerting its effect on movement variables are unclear. Recent evidence, however, has shown that higher catastrophizing was associated with elevated cortisol profiles in acute pain, with the possibility of disruptions to the integrity of the hypothalamic-pituitary-adrenal axis.[68] These observations are interesting given the evidence that sympathetic activation can decrease the sensitivity of muscle spindle afferents and thereby decrease the fidelity with which spindle afferents monitor muscle length and velocity changes, critical aspects in proper motor control.[69] It is possible that this effect on spindle afferent fidelity is greater in higher catastrophizers in pain and that this mechanism might be mediated through the hypothalamic-pituitary-adrenal axis.

Alternatively, or possibly in combination, effects of catastrophizing on motor activity may involve changes in brain activity within regions involved in motor control and/or in top-down modulation of perception of nociceptive activity. There is evidence for impaired top-down processing of pain intensity in higher catastrophizers, who appear to have difficulty disengaging from and suppressing more intense pain.[30] This might have contributed to the impairment in the ability of the subjects in the present study to focus their attention on the task at hand, which may have contributed to the impaired movement performance. It should be pointed out, however, that the association between catastrophizing and descending pain-modulatory mechanisms is not fully established.[70]

Associations Between Pain Variables and Catastrophizing

Consistent with previous studies in non-orofacial body regions in experimental pain models[9,30,40,64,71–73]

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and in a variety of chronic pain conditions,[4,7,65,74–77] there were significant associations between catastrophizing and jaw muscle pain intensity and unpleasantness ratings. During hypertonic saline–evoked jaw muscle pain, the higher catastrophizers exhibited significantly higher MPQ pain rating indices. The weak significant relationship (P = .03, Table 4) between pain catastrophizing and the PRI sensory scores might be surprising given the association demonstrated between pain intensity and catastrophizing scores. However, it needs to be remembered that pain intensity scores represent a sum of multiple dimensions, including sensory, affective, and evaluative dimensions.[78] Importantly, the findings are consistent with previous findings that subjects with higher PCS scores selected a significantly greater number of affective words (eg, ''sickening'' and ''fearful'') to describe the pain of noxious electrical stimuli.[30] Further, pain catastrophizing has been shown to be significantly associated with increased activity in brain areas related to the attentional and emotional aspects of pain, but not brain regions associated with the sensory-discriminative aspects of pain.[30,74]

Pain spread was mainly posterior and/or cranial to the masseter injection site, and this is consistent with previous observations.[32,79] Referred pain or spread of pain around the masseter injection site reflects the organization of afferent nerves and central connections as well as neuroplastic changes.[80–83] The significant association of catastrophizing with total perceived pain areas is consistent with previous studies demonstrating that catastrophizing had significant association with tender-point counts in population studies of musculoskeletal tenderness and fibromyalgia.[84]

Taken together, the findings of the present paper provide new evidence that catastrophizing plays an important role in the perception of acute experimental orofacial pain. It is postulated that high-catastrophizing individuals may exhibit a greater central nervous system sensitization during pain[7,9] that may involve enhanced activity in higher centers (eg, prefrontal cortex), and this may influence the interpretation of information from nociceptive pathways. While central sensitization is most likely to be the mechanism underlying the increases in pain scores and jaw movement variability in the higher pain catastrophizers, possible changes in peripheral sensitization might also contribute to some of the effects. The association recently demonstrated between pain catastrophizing and exaggerated hypothalamic-pituitary-adrenocortical activity[68] might help explain changes in peripheral sensitization.

Study Limitations

The study would be strengthened with a symptomatic subject component, although well-controlled experimental pain studies are of value in analyzing the early stages that might be involved in the transition from acute to chronic pain. The participants were subjects who voluntarily agreed to participate in an experiment causing them acute pain, and this likely also limits the generalizability of the conclusions to the general population. Only associations are demonstrated, and no causal inferences can be made. All analyses were exploratory, and no adjustments were made for multiple comparisons. Given these issues, the significant findings need to be confirmed by further independent studies. Future studies could consider gender issues, manipulation of catastrophizing, and the use of other forms of experimental pain induction, eg, glutamate infusion, as well as clinical samples. Generalization of the results to the predominantly female TMD clinical samples may be premature given that this study used a sample of mainly male subjects. The pain-catastrophizing score requires an individual to recall the pain experience, and this may have caused some additional variance. In addition, the jaw task employed in the present study was a goal-oriented task, and the effects on motor coordination identified may be different with other naturally occurring jaw movements/tasks.

Conclusions

The present findings add to the growing literature confirming the importance of pain catastrophizing in the perception of acute orofacial pain, and they also demonstrate that catastrophizing may play a role in influencing how an individual's jaw motor system reacts to acute orofacial pain, that is, through increased jaw movement variability. Notwithstanding the limitations of this study as outlined above, the findings nonetheless add to our understanding that the interaction between pain and motor activity is significantly influenced by psychological factors (pain catastrophizing) and that previous models (Pain Adaptation Model, Vicious Cycle Theory) cannot adequately explain these interactions.[31,32,57] The data point to the need for future studies in a chronic orofacial pain group with a view to developing interventions focusing on psychological variables, possibly in relation to motor activity.

Acknowledgments

The authors thank Ms Terry Whittle for her assistance and advice in the study. The research was supported by the National Health and Medical Research Council of Australia (Grant #512309) and the Australian Dental Research Foundation, Inc. Prof P. Svensson was an International Visiting Research Fellow of the University of Sydney. Dr Rahena Akhter is a recipient of a University of Sydney Postdoctoral Fellowship. There are no conflicts of interest.

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