Received: 2 March 2021 | Revised: 19 July 2021 | Accepted: 23 July 2021

DOI: 10.1111/joor.13238

ORIGINAL ARTICLE

JOURNAL OF ORAL REHABILITATION WILEY

Stress-evoked muscle activity in women with and without chronic myofascial face pain

Malvin N. Janal[1] | Frank Lobbezoo[2,3] | Karen S. Quigley[4,5] | Karen G. Raphael[3]

[1]Department of Epidemiology and Health Promotion, New York University College of Dentistry, New York, NY, USA

[2]Department of Oral Health Sciences, Academic Centre for Dentistry Amsterdam (ACTA), University of Amsterdam and Vrije Universiteit Amsterdam, Amsterdam, The Netherlands

[3]Department of Oral & Maxillofacial Pathology, Radiology, and Medicine, New York University College of Dentistry, New York, NY, USA

[4]Department of Psychology, Northeastern University, Boston, MA, USA

[5]Edith Nourse Rogers Memorial VA Hospital, Bedford, MA, USA

Correspondence
Malvin N. Janal, Department of Epidemiology and Health Promotion, Suite 301, 380 Second Avenue, New York, NY 10010, USA.
Email: mj62@nyu.edu

Funding information
National Institutes of Health, Grant/Award Number: R01DE018569

Abstract

Background: Amplified muscle activity in reaction to daily life stressors might explain chronic pain in temporomandibular disorder (TMD).

Objectives: To assess whether patients with myofascial TMD pain (MFP) react to standardised stressors with greater masticatory muscle activity than demographically matched controls.

Methods: A total of 124 female MFP patients and 46 demographically matched and pain-free controls rated distress while performing a series of standardised stress-reactivity tasks (viz., cold pressor test, mental arithmetic test, speech stressor test and reaction time/startle response test) as well as a vanilla baseline control task. Blood pressure was measured before and after each task, and electromyographic (EMG) activity was continuously recorded over the jaw-closing muscles and several non-masticatory muscles during each task. Linear mixed model analyses were used to test the hypothesis that case status, stress-reactivity task and muscle recording site influenced EMG activity.

Results: Stress induction was successful, as evidenced by distress ratings and blood pressure measurements that were significantly elevated during performance of the stress tasks. Participants reported that some of the tasks were stressful in a way that resembled stressors experienced in their daily lives. Elevated muscle activity could be confirmed only for the reaction time/startle response task, where mean EMG activity was elevated more in cases than in controls, specifically in the jaw-closing muscles.

Conclusion: These data could not provide clear support for the theory that psychological stressors produce a differential increase in masticatory muscle activity in MFP patients than pain-free controls.

KEYWORDS

anxiety, blood pressure, bruxism, electromyography, myofascial, pain, stress, temporomandibular disorders, women

1 | INTRODUCTION

One theory of chronic myofascial pain (MFP) holds that people with chronic pain react to psychological stressors with excessive muscle activity specifically in the painful area, and this activity supports ongoing pain in those areas.[1] In support of this model, people with chronic low-back pain have been shown to exhibit high levels of muscle activity to laboratory stressors specifically in their back muscles, while those with chronic face pain showed exaggerated responses specifically in their facial muscles,[12] and other studies also support this hypothesis.[3–9] In their review of psychophysiological differences between chronic pain patients and controls in their response

to stress, however, Flor and Turk[10] suggested that a clear answer to this question required improved methodology: standardised diagnosis and test procedures, larger samples, ecologically valid stressors and multiple recording sites. Since that time, Flor et al.[11] have reported increased masseter EMG activity to imagined stress in groups of MFP patients relative to pain-free controls, Katz et al.[5] reported more prolonged stress-related elevation of masseter EMG in MFP cases than controls in a fast-paced performance task, and Glaros et al.[12] have supported a mediational model linking perceived stress to muscle tension and pain. By and large, however, few studies have addressed all of the challenges presented by Flor and Turk as necessary to answer the question of enhanced facial muscle responding as an explanation for MFP.

Further, while this question has a venerable scientific history, it remains most relevant because several generations of dentists and oro-facial pain specialist have, at least since the time of Schwartz[13] and Laskin[14] half a century ago, believed that environmental stressors will increase masticatory muscle activity, leading to increased pain in vulnerable patients. LeResche and colleagues[15] first showed this belief was prevalent among dentists, and recent reports confirm that this belief continues among dentists[16] and even patients.[17]

It was this study's aim to compare a large group of female patients with MFP (i.e., cases) to demographically matched controls without MFP in their EMG and subjective responses to a series of standardised stressors. These stressors are predicted to increase EMG activity specifically in chronically painful muscles of the cases, and to have less effect on other muscles in the cases, or in individuals without facial pain. We strive to answer the challenge posed by Flor and Turk[10] to evaluate the ecological validity and site-specific effects of each stressor, to use a strict case and control definition in large sample and to compare affected and unaffected facial and non-facial muscle groups.

2 | MATERIAL AND METHODS

2.1 | Participants

The study sample has been described by Raphael et al.[18–20] and Dubrovsky et al.[21] Details about the recruitment procedure, inclusion and exclusion criteria, and case definitions can also be found there. In brief, 124 women (mean age ± SD =40.3 ± 14.8 years) with MFP were recruited from among 169 potential case participants at the oro-facial pain clinic of the New York University College of Dentistry (NYUCD). The main reason for not enrolling cases was the pain secondary to physical trauma, and secondarily, smoking and not regularly sleeping 4+ h. MFP diagnosis was based on the Research Diagnostic Criteria for TMD (RDC/TMD), groups Ia and Ib (i.e., MFP with or without limited mouth opening respectively).[22] Diagnostic reliability was confirmed by 'calibration days' that occurred several times during the study ($\kappa = .65$). Cases were enrolled without explicitly taking into consideration their own awareness of the presence or absence of awake (or sleep) bruxism.

Based solely on the absence of MFP (i.e., neither TMD groups 1a nor 1b), 46 of 63 potential control participants (mean age ± SD =36.1 ± 13.5 years) were recruited from other clinics of NYUCD and the remainder from among acquaintances of the case participants. The main reason for not enrolling controls was the presence of a facial tender point; the remainder failed to continue participation.

For both groups and for study purposes unrelated to the results reported here, the following exclusion criteria were used: being pregnant; being a habitual smoker; insomnia, sleep apnoea, systemic diseases such as diabetes or peripheral neuropathy; having a neuropathic facial pain condition; having sustained a major and identifiable trauma to the face; prior jaw surgery; and having undergone dental treatment within two days prior to the RDC/TMD examination. Controls were excluded if they had reported having one or more weeks of facial pain in the last two years or more than one painful site on masticatory muscle palpation. Data were collected between May 2008 and June 2011.

Prior to the study, all participants completed a consent process and signed an informed consent document. The Institutional Review Board at the New York University (NYU) School of Medicine approved the research protocol and procedures (protocol 07-303).

2.2 | Procedure

All testing was completed at the Bluestone Center for Clinical Research at the New York University College of Dentistry, a clinical research centre with full dental examination facilities. On average, the total duration of the experimental session was 120 min, including the experimental set-up and the testing itself – the latter lasting a median of 108 min. After set-up, the test series always started with a 'vanilla baseline' monitoring period[23–25] of 5 min, during which time the participant completed a cognitively engaging but non-demanding task, viz., counting (silently) and keying in those counts of the number of coloured squares that appeared on the computer screen. This task provided estimates of the effects of non-stressful attention, to be used as a basis for interpreting data from the other tasks, which required attention and typing but were also designed to be stressful.

Following the vanilla baseline, participants completed four stress-reactivity tasks that have been in common use for decades: a cold pressor test; a mental arithmetic test; a speech stressor test; and a reaction time/startle response test. Different presentation orders of these tests were determined at random (by the computer algorithm controlling the experiment) for each individual and distributed evenly over the participants. During the cold pressor test, the participant was asked to immerse her preferred foot in a tub of water cooled to 10°C (to avoid early withdrawals) for 2 min. Stress-related cardiovascular changes produced by the task were maximised by providing instructions prior to the test that emphasised the difficulty of the task.[26] The mental arithmetic test versions of which are used commonly in the health psychology literature (e.g., [27,28]) comprised 5 min of serial subtractions. A computer screen displayed a 2–4 digit number (seed number) along with a smaller number (subtrahend)

that was to be subtracted from the larger one. The participant was then requested to type her answer in a box, and she was told that the answer should be both correct and given as quickly as possible. The test was designed to maintain high difficulty levels across all participants, so that additional digits were added to the seed and subtrahend for participants who quickly and correctly completed easier subtractions. The speech stressor test, also commonly used,[27–29] began with instructions telling the participant that she would have 4 min to prepare a 3-min speech on her views on illegal immigration into the United States, and that the delivery of her speech would be videotaped and scored by a panel of judges who would compare her speech to that of others. Only data from the 4-min preparation phase were analysed. Finally, during the reaction time/startle response test, the participant was first shown a 4-letter word on the computer screen, followed by another screen with a single letter. She was then instructed to indicate whether that letter did or did not appear in the prior word. The participant was told that reaction times faster than previous participants would prevent the occurrence of a white noise blast (95 dB of sound pressure level; 500 ms duration; instantaneous rise time), although 8 blasts were presented to all subjects at predefined time intervals, regardless of performance. This schedule of white noise blasts has been shown to be an effective stressor,[27] with only a minimal habituation occurring over the 8 stimuli.[30] Data from the entire task, as well as various times surrounding each white noise blast, were analysed. Finally, there was a 5-min recovery period between each task. BP and EMG recordings made during those periods are defined in Results as 'recovery' activity.

The participants were instructed not to talk during any of the tests unless specifically instructed to do so (e.g., during the second part of the speech stressor test), to avoid making large body movements and to keep the typing hand near the keyboard during the computer tests (viz., the vanilla baseline, the mental arithmetic test and the reaction time/startle response test). Participants were debriefed about how each task was related to study goals after all data had been collected.

2.3 | Data acquisition

The objective of the stress-reactivity tasks was to evoke elevated levels of task-related psychological distress. To check the adequacy of the stressor manipulation, subjective and objective (i.e., physiological) measures were used. Following each of the tasks, participants were asked to indicate how stressful the preceding task was on a 0–10 numeric rating scale, where 0 means 'not stressful at all' and 10, 'as stressful as you could ever imagine'. Participants were also asked to indicate whether the experienced stress was familiar to them, that is whether their mood state was comparable to that which occurs during stressful events outside the laboratory setting. To that end, they completed an 11-point 'similarity of experience' scale,[8] with 0 indicating 'not at all similar' and 10, 'very similar, just like I feel in my real life when I am stressed'. In addition, prior to the first stress-reactivity task, participants completed Form Y-1 of the State-Trait Anxiety Inventory (STAI), which is a 20-item STAI subscale for the assessment of state anxiety (i.e., current level of anxiety). The STAI Form Y-2, which assesses trait anxiety (i.e., general anxiety-proneness) in 20 items, was already administered as part of an earlier assessment of these women.[31]

To characterise the physiological activation produced by the stress-reactivity tasks and to assess physiological concordance with the subjective stressfulness ratings, systolic/diastolic blood pressure measurements were obtained every minute during the tests as well as during the 5-min recovery epochs. Blood pressure recordings were obtained in a seated position using a Vasotrac APM 205A system (Medwave, Inc.), which displayed systolic and diastolic blood pressure.

To quantify stress-reactivity task-related changes in masticatory muscle activity, surface electromyographic (EMG) recordings were obtained from the masseter and temporalis muscles on both sides of the face. In addition, to enable testing whether certain changes would be specific for the jaw musculature or reflected a more general increase in muscular activation, surface EMG recordings were also obtained from the mentalis, deltoid and sternocleidomastoid muscles. EMG recordings were obtained using Ag/AgCl electrodes attached to sites that were first abraded and cleaned with an alcohol wipe. The experiment was controlled by a LabView program that selected the correct stress-reactivity task sequence, controlled its presentation, and acquired and stored EMG signal data developed on a Bionex 3711-08 amplifier system (MindWare Technologies, Ltd.). Sampling occurred at a rate of 1,000 Hz, with a bandpass of 1–100 Hz and a gain of 5,000. EMG data were processed, displayed and exported for statistical analysis using EMG Analysis Software (v 3.0; MindWare Technologies, Ltd.,). Prior to export, data were filtered to include signals between 15 and 50 Hz,[32] and exclude periods containing artefacts, defined as signals at least 3× the baseline level that were associated with talking, laughing, coughing, yawning, smiling, rubbing the nose or face (and miscellaneous others). EMG amplitude (root mean square voltage or Vrms) could be computed for the entire period of the task, or averaged for segments, as needed.

2.4 | Data analysis

The following outcome measures were statistically analysed: self-reported distress ratings (experienced; familiar), trait and state anxiety, blood pressure (systolic; diastolic) and EMG amplitude (Vrms) for the total duration of each task. As a first step, the total task EMG amplitudes were rank-transformed to correct the skewness in the distribution of that measure. While a log transformation is often used to correct skew in EMG amplitudes, rank transformation was preferred because it normalised residuals and produced less residual variability in these data. Second, linear mixed model analyses were used to test the effects of Group (cases; controls), Task (recovery, vanilla baseline; cold pressor; mental arithmetic; speech stressor; reaction time/startle response) and site (masseter, temporalis, mentalis, deltoid and sternocleidomastoid muscle) on the outcome measures.

When there were significant omnibus test results, means were then compared using the Sidak test. The sample size allows for the detection of a. 45 SD difference between diagnostic groups, assuming a 2:1 ratio of cases to controls and type 1 and 2 error rates of 5% and 20% respectively. The 2:1 ratio was needed for study purposes unrelated to this report.

All statistical analyses were performed with IBM SPSS version 24 (IBM Corp). Probability levels of $p < .05$ were considered statistically significant.

3 | RESULTS

Case and control groups did not differ on any measured demographic characteristic: most indicated that their race was white (62.6%), black (14.4%) or 'other' (14.4%). Hispanic ethnicity was indicated by 22.5%. Mean age was 39.2 yr (SD = 14.6, range 19–78), with a mean of 15 year of education (SD = 2.2, range 11–20). TMD patients reported moderate intensities of characteristic pain (Mean = 5.2, SD = 1.7) and relatively low levels of pain disability (Mean = 1.8, SD = 2.2). Pain onset occurred more than 10 years before study entry (Mean = 126.1 months, SD = 127.1; Median = 84). Child-bearing potential was reported by 118 participants, with similar proportions of cases and controls (72.0% vs. 83.7%, respectively, $p = .16$). Among these 118, cases and controls were well-matched with respect to phase of the menstrual cycle ($p > .50$).

3.1 | Subjective distress ratings

Mean levels of self-reported distress experienced during the various experimental tasks are shown in Figure 1. As expected, little distress was reported during the vanilla baseline period, with a mean of less than 2 (of 10 points). By contrast, each of the tasks intended to elicit distress received average ratings at 2–3× the vanilla baseline level, and analysis indicated a Task effect ($\eta_p^2 = 0.39$; $F(4,511) = 82.49$; $p < .001$). Post hoc tests showed that significantly more distress was experienced during the mental arithmetic task than any other task. Lower and similar levels of distress were reported in the cold pressor and speech stressor tasks, and these levels were significantly greater than those reported during the reaction time/startle response task or vanilla baseline. Finally, significantly more distress was reported during the reaction time/startle response task than during the vanilla baseline. Distress ratings were similar in cases and controls on each of the tasks ($\eta_p^2 = 0.001$; $F(1,161) = 0.42$; $p = .52$), and there was no evidence of a Group X Task interaction ($\eta_p^2 = 0.006$; $F(4,511) = 0.93$; $p = .44$). Thus, each of these challenges achieved their intended effect, albeit to varying degrees, of increasing self-reported levels of distress, and these effects were comparable in cases and controls.

Some tasks produced more familiar distress than others ($\eta_p^2 = 0.26$; $F(4,509) = 43.87$; $p < .001$). The least familiar was the distress provoked by the cold pressor task (mean ± SE = 2.13 ± 0.34), which was significantly less familiar than that evoked by the reaction time/startle response task (mean ± SE=3.74 ± 0.33), which in turn was less than that evoked by the mental arithmetic task (mean ± SE= 4.47 ± 0.33). Most familiar was the distress evoked by the speech stressor task, which was significantly more familiar than any other task (mean ± SE=5.69 ± 0.32). The familiarity of task-related distress was similar between cases and controls [$\eta_p^2 < 0.001$; ($F(1,166) = 0.15$; $p = .70$) and across all tasks [Group x Task $\eta_p^2 = 0.005$; $F(4,509) = 0.63$, $p = .64$]. Thus, distress ratings were most familiar during the speech stressor task, and least familiar during the cold pressor task, and these effects were similar in cases and controls.

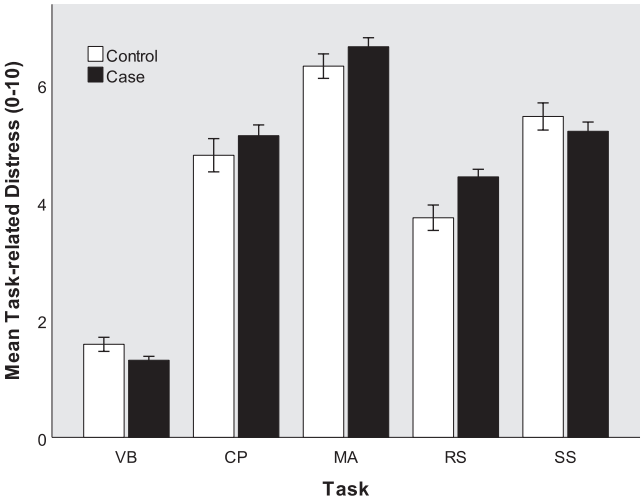

FIGURE 1 Mean ± standard error (SE) of experienced stress ratings (self-reported on a 0–10 numeric rating scale) by experimental task (vanilla baseline, VB; cold pressor, CP; mental arithmetic, MA; reaction time/startle response, RS; speech stressor, SS) and by study group (controls; cases)

3.2 | Trait and state anxiety

Myofascial pain patients reported more trait anxiety than control participants upon entrance to the study (MFP M(SD) = 42.4(10.2) vs. Control M(SD) = 36.1(8.9); $\eta_p^2 = 0.08$; $F(1,169) = 13.83$, $p < .001$), but no difference in state anxiety was reported by cases and controls immediately preceding the stress-reactivity testing (MFP M(SD) = 33.8(9.4) vs. Control M(SD) = 31.4(9.5); $\eta_p^2 = 0.013$; $F(1,169) = 2.23$, $p = .13$). Thus, while patients reported more anxiety than controls in general, they reported similar current levels on the day of testing.

3.3 | Blood pressure

As illustrated in Figure 2A,B, mean systolic blood pressure (SBP) and mean diastolic blood pressure (DBP) increased during the performance of the stress-reactivity tasks. Analysis supported these observations, showing a Task × Period effect for

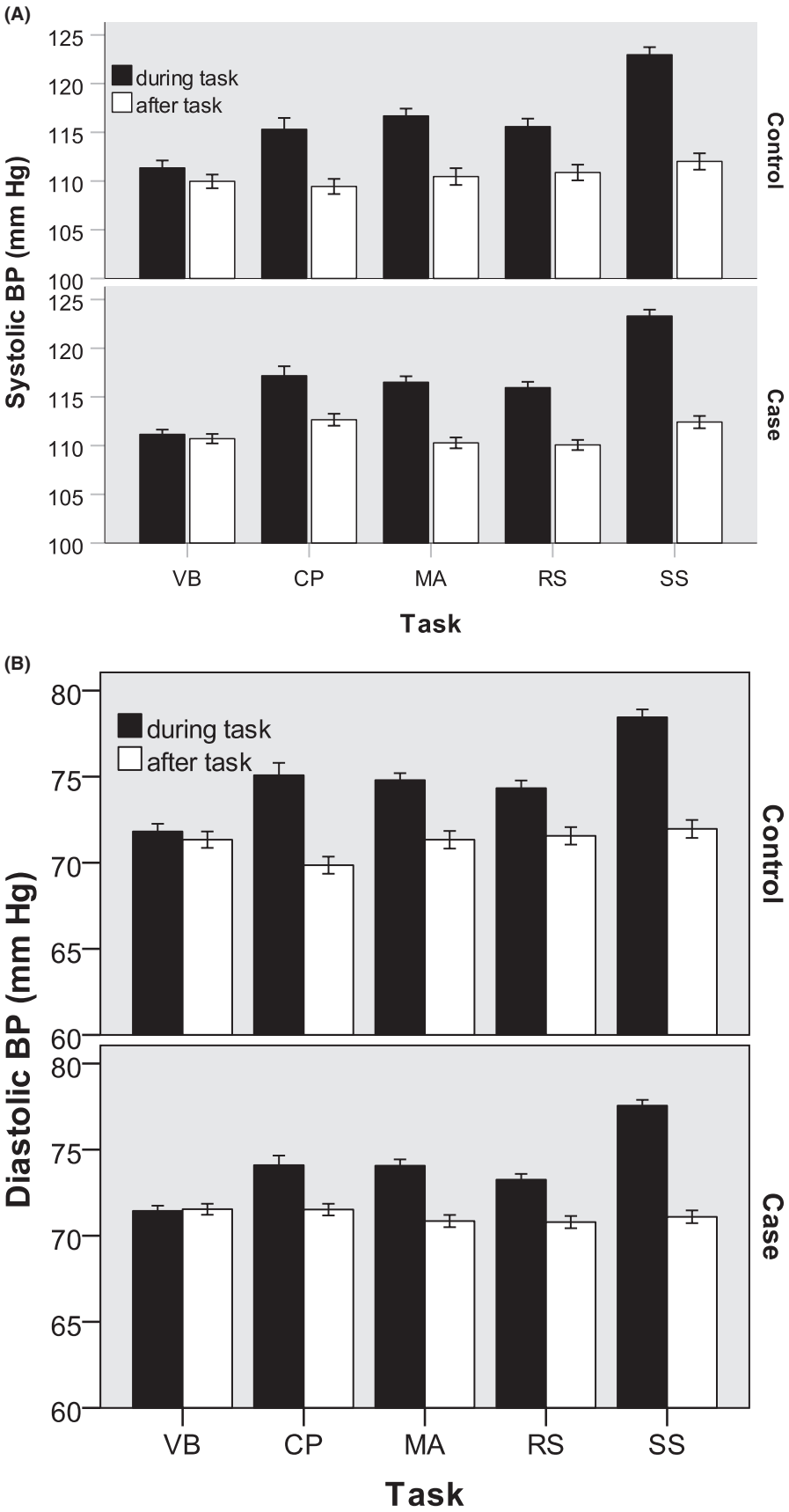

FIGURE 2 Mean ± SE of systolic blood pressure (SBP) (A) and diastolic blood pressure (DBP) (B). Measures (mmHg) are shown by study group (controls; cases), by experimental task (vanilla baseline, VB; cold pressor, CP; mental arithmetic, MA; reaction time/startle response, RS; speech stressor, SS) and by period (during task; after task)

SBP [$\eta_p^2 = 0.33$; $F(4,522) = 65.5$; $p < 0.001$] and DBP [$\eta_p^2 = 0.35$; $F(4,532) = 70.7$; $p < .001$]) that was similar for cases and controls (Group × Task × Period for SBP [$\eta_p^2 = 0.002$; $F(4,522] = 0.2$; $p = .95$] and DBP [$\eta_p^2 = 0.008$; $F(4,532] = 0.9$; $p = 0.46$]). Except for the vanilla baseline task, SBP and DBP were significantly higher during each of the other tasks relative to their associated post-task

recovery periods. Averaged over task, case and control subjects showed 5.6 and 5.8 mmHg increases in SBP, respectively, relative to the post-task recovery period ($p = .92$), and 3.0 and 3.7 mmHg increases in DBP ($p = .08$) relative to the post-task recovery period, suggesting that cases were not more physiologically reactive to the tasks, as measured by changes in SBP and DBP. Thus, the BP data also suggest that the stressor manipulations were successful. Each stressor task produced the expected increase in BP and no change in the control (vanilla baseline) task. Further, these manipulations produced similar effects on BP in cases and controls.

3.4 | Electromyography amplitude

Figure 3A summarises mean EMG amplitude (uVrms) as a function of case status, task and masticatory muscle site. Case/control differences are apparent in all four masticatory muscle regions only during performance of the reaction time/startle response task. An analysis that compared EMG responses during and after the reaction time/startle response task showed a Group x Task interaction [$\eta_p^2 = 0.05$; $F(1,169) = 7.9$, $p = .005$], indicating higher levels when on-task in cases than in controls (Figure 3B). This finding was similar for all four jaw-closing muscles (Group × Task × Site, $\eta_p^2 < 0.001$; $F(3,2493) = 0.2$, $p = 0.87$). Interestingly, although the reaction time/startle response task was rated as less distressing and provoked only small increases in BP and a moderate increase in facial EMG, this task was associated with a greater increase in masticatory muscle EMG among cases than controls, whereas other, more stressful tasks, were not.

Theory would predict that the increased EMG activity seen in cases would be specific to the masticatory muscles. Figure 4 shows, and analysis confirmed [Group × Task × Site interaction; $\eta_p^2 = 0.007$; $F(6,4443) = 4.96$, $p < .001$)], increased EMG activity during the reaction time/startle response task in the jaw-closing muscles of cases but not in those of controls. In addition, Figure 4 shows increased activity for the mentalis muscle region in both groups ($p < .05$), no change in either group for the sternocleidomastoid muscle ($p > .05$), and increased EMG activity in the deltoid muscle only of control subjects ($p < .05$). These observations show that the case-control difference in EMG activation was specific to the masticatory muscles and suggests that distress induced by this task may have a mechanistic (i.e., pain maintenance) relationship to MFP pain.

There are two features of the reaction time/startle response task that might moderate EMG responses. First, if the unpredictable white noise blasts provoke a muscle reflex, EMG should be maximised in the interval immediately following the blast. Second, if the continuous performance aspect of the task is stressful, EMG should increase with task duration. To evaluate the first hypothesis, we compared EMG activity in the 5-sec periods preceding and following each white noise blast. Results failed to show a difference in activity before and after the noise blasts ($\eta_p^2 < .001$; $F(1,654) = 0.7$, $p = .39$), or in any higher order interaction. A similar

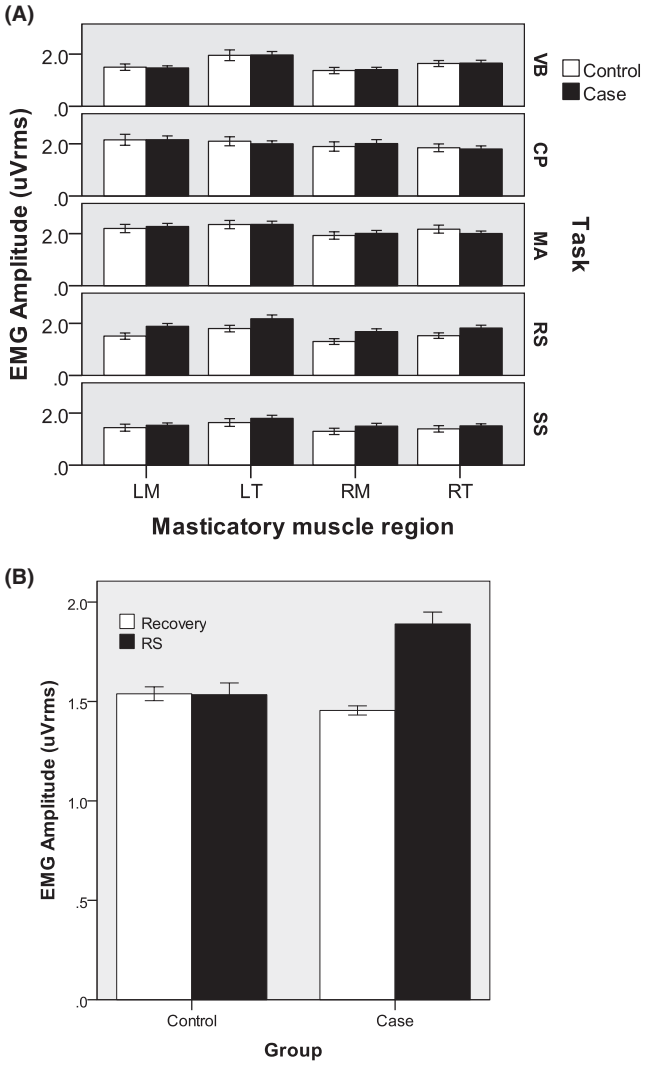

FIGURE 3 Mean ± SE of overall mean EMG amplitude (uVrms, root mean square microvoltage) values by experimental task (vanilla baseline, VB; cold pressor, CP; mental arithmetic, MA; reaction time/startle response, RS; speech stressor, SS), by site (left masseter, LM; left temporalis, LT; right masseter, RM; right temporalis, RT), and by study group (cases; controls) (A). Mean ± SE of overall mean EMG amplitude during and after RS by study group (cases; controls) (B)

absence of differences between cases and controls was obtained when the duration of the post-noise period was shortened to 0.5 sec ($0.4 < p < .7$, depending on muscle). To evaluate the second hypothesis, that the continuous performance aspect of the task was especially stressful, we compared EMG activity during each successive minute of this task. Analysis showed a small effect of time [$\eta_p^2 = 0.001$; $F(1,5620) = 7.7$, $p = .006$] but no difference in mean EMG activity between cases and controls [$\eta_p^2 < .001$; $F(4,4763) = 0.2$, $p = .92$]. Thus, the case difference in masticatory muscle EMG to this task cannot be clearly attributed to stressfulness associated with either startle or increased masticatory activity over continued performance.

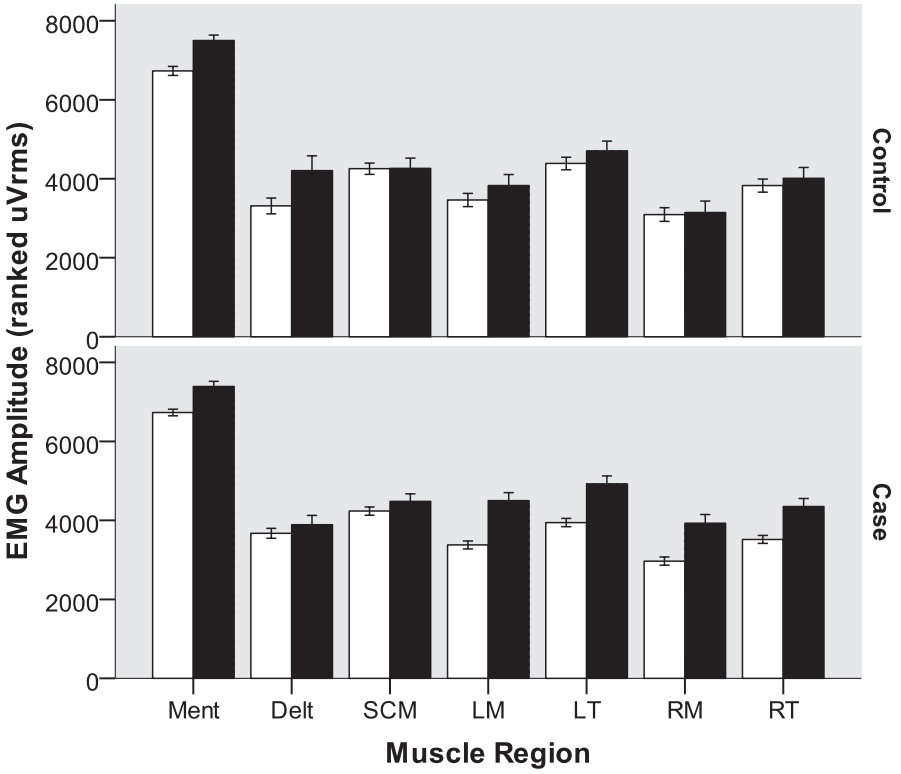

FIGURE 4 Mean ± SE of ranks of EMG uVrms values per study group (cases; controls), by muscle site (Mentalis, Ment; Deltoid, Delt; Sternocleidomastoid, SCM; left masseter, LM; left temporalis, LT; right masseter, RM; right temporalis, RT) and task period (recovery, open bars; reaction time/startle response, RS, filled bars)

4 | DISCUSSION

Theory predicts, and oro-facial pain specialists and MFP patients generally believe,[15–17] that stressors lead to increased muscle activity, which leads to muscle-specific pain in vulnerable individuals.[1,9,10,33] In this study, we induced distress through several common stressor tasks and evaluated changes in EMG amplitude. The study was designed to meet the methodological challenge proposed by Flor and Turk[10]; it employed a large sample, multiple common validated stressors, a valid case definition, and assessed activity in both jaw-closing and control muscle groups. MFP was diagnosed using the RDC/TMD criteria.[22] Good levels of reliability were shown between our clinical coordinator and a 'gold standard' instructor in RDC diagnosis ($\kappa = .65$). Results showed that participants not only reported two to three times more distress after performing each of these tasks than during recovery, they also reported that some of those tasks produced distress that was similar to that they experienced in their daily lives. Concordant with the verbal ratings of distress, blood pressure (both systolic and diastolic) increased when performing each stressor task. Cases and controls were similarly affected by these stressors, both in terms of subjective ratings and blood pressure.

The main hypothesis was confirmed for one of the four tasks, where mean EMG amplitude was elevated more in cases than controls, and this elevation was specifically seen in the jaw-closing muscles. In general, however, this suite of stress manipulations failed to provoke greater stress-related increases in EMG activity within the masticatory muscles of the case group relative to the controls. These results contrast with several studies that have shown greater increases in jaw-muscle activity in response to standardised stressors in MFP patients than controls or higher levels of resting EMG in the facial muscles of TMD cases than controls.[1,3–7,11,34] Other studies have shown increases in tooth contact, a behaviour associated with increased facial EMG activity.[35] Here, instead, EMG responses were similar in the two groups during the vanilla baseline and during all of the stressor tasks except stress reactivity.

Larger increases in EMG activity over the jaw-closing muscles were seen in MFP patients than pain-free controls specifically in response to the reaction time/startle response task. Further, these increases were observed for the jaw-closing muscles but not for other nearby (e.g., mentalis) or more distant (e.g., deltoid and sternocleidomastoid) muscles. For this finding, several possible explanations can be advanced. First, given the number of statistical tests conducted and our inability to isolate the increase as occurring specifically during the white noise blasts or over the course of task time, these findings may represent a Type I error, even though the *p*-value for the effect ($p < .001$) renders this unlikely, as does the consistency of the finding over all four masticatory muscles. Alternatively, the reaction time/startle response task generated only moderate levels of distress, less than the other tasks. This raises the possibility of a ceiling effect such that no, or only small increases in jaw-muscle activity could occur in response to the more stressful tasks, whereas differential increases were possible in response to the less stressful task. That is, cases and controls both show strong responses to highly stressful tasks, but cases were more distressed than controls by this moderately stressful task. Another explanation we considered was whether the white noise blasts themselves or the continuous performance aspect of the reaction time/startle response task

were responsible. To answer that question, muscle activity before a noise blast was compared with that during/after a blast, and we also compared activity in the early and late segments of the overall response record. Neither comparison yielded differences, again failing to explain how this task was associated with a case-specific increase in stress reactivity. More research on this question is suggested.

Discussion to this point has considered the relative effects of case status, task and muscle locus on EMG activity. It is also interesting to consider the absolute level of EMG activity, and associated changes in jaw position, associated with these effects. As shown in Figure 3A,B, the absolute EMG amplitude averaged between 1 and 2 uV, and the largest changes in amplitude were on the order of 0.5 uV. While such voltages are sufficient to move the jaw toward a closed position, they reflect only a fraction of the effort necessary to actually cause the posterior teeth to meet, estimated as a temporalis EMG of at least 6 uV and masseter EMG of at least 4 uV.[35,36] Simple tooth contact has been associated with facial pain.[37] Changes of this magnitude were seen for the mental arithmetic and cold pressor test in both subject groups, in neither group for speech stressor and only in the MFP group for the reaction time/startle task. Thus, while these stressors were rated as distressing and like those experienced in daily life, and adequate to stimulate a robust autonomic response, they were not adequate to create the larger, and potentially most relevant movements of these muscles, such as those experienced in life.

Using another approach to address the distress/muscle activity question, Glaros and his collaborators have provided partial support for the hypothesis that muscle tension mediates the relationship between distress and pain.[12] However, in contrast to the laboratory-evoked challenges used in this study, those studies focused on ratings of distress experienced in everyday life which were related to perceived levels of muscle tension.[38] It remains an interesting question as to which model of the stress/distress-muscle tension relationship may be more realistic.

Current data fit a mixed model, that is, one with a random intercept. This had the effect of removing consistent differences in individual responding from residual variability, increasing the precision of the model, based on each participant's average response across tasks and muscles. This modelling approach assumes that individual differences are consistent across tasks and muscles. One could also assess idiographic or person-specific response patterns. For example, recent machine learning approaches often utilise a large number of measures (or features) and examine whether there are clusters of individuals with similar patterns. In this example, one could search for clusters of individuals with similar patterns of EMG activity across tasks or even to specific tasks. Hypothetically, one might discover a subgroup of participants who responded with strong temporalis and masseter activity across a number of tasks, while another subgroup of individuals might be discovered that had task-specific responses or minimal responses across all tasks. Such data-driven approaches have been recently used to assess patterns of change in cardiovascular and autonomic nervous system activity during emotional episodes in real life (e.g.,[39]), or to identify previously unidentified response patterns in those engaging in motivated performance tasks such as those used here.[40] This approach is beyond the scope of our original goal to study group differences, however, but should be considered in future work.

Current results are limited to women. This was done because the rates of MFP are strikingly higher for women than for men, especially amongst those seeking treatment.[41,42] Additionally, there is evidence that pain-processing mechanisms may be different in men and women.[43,44] Hence, using both genders in a single study would have yielded difficult-to-interpret findings. Another limitation of the present study is the fact that we studied EMG activity in an experimental setting. It is not immediately clear if, and to what extent, the present findings can be extrapolated to daily life situations. Conclusions are also limited to the effects of relatively short duration stressors; the effects of longer duration, or more intense, stressors on the masticatory muscles require further study. Pain sensitivity varies across the menstrual cycle,[45] but was not controlled in this study; it is possible that the failure to find case-control differences resulted from a washing out of effects that may be present during one particular phase. We treated the masseter and temporal muscles of all participants as painful sites and compared them to the chin and shoulder. In fact, 12 cases reported unilateral pain and so some of their masseter/temporal EMG responses were not, in fact, from painful sites. While this sample was not large enough for separate analysis, future work might control this factor.

5 | CONCLUSION

This large and well-validated comparison of MFP patients and demographically matched controls hypothesised a case-specific and masticatory muscle-specific increase in jaw-closing muscle activity to four standard stressors. Only one stressor showed such effects. Within the limits of this study, there does not seem to be clear support for the theory that these stressors produce a differential increase in masticatory muscle activity in MFP patients. It remains to be shown that a consistent increase in jaw-closing muscle activity to stress-provoking tasks supports this critical step in the hypothesised link between distress and chronic face pain.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge the support of Dr Lena Nemelivsky in her role as clinical research coordinator. This study was supported in part by Dr. Raphael's grant R01DE018569 from the National Institutes of Health, Bethesda, MD, USA. Dr. Lobbezoo received funding from the University of Amsterdam/New York University Visiting Guest Faculty Program 2014-2015, for his Visiting Professorship at New York University College of Dentistry.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

AUTHOR CONTRIBUTION

MNJ, KGR and KSQ performed conception and design. LN, MNJ and KGR were involved in acquisition of data. MNJ, KGR, KSQ

and FL performed analysis and interpretation of data. MNJ, KGR, FL and KSQ drafted and revised the manuscript. KGR contributed to funding. KGR and MNJ were involved in project administration. All authors have read and agreed to the published version of the manuscript.

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1111/joor.13238.

DATA AVAILABILITY STATEMENT

Data are available from the first author upon request.

ORCID

Malvin N. Janal https://orcid.org/0000-0003-1098-9687
Frank Lobbezoo https://orcid.org/0000-0001-9877-7640
Karen G. Raphael https://orcid.org/0000-0002-2804-8124

REFERENCES

1. Mercuri LG, Olson RE, Laskin DM. The specificity of response to experimental stress in patients with myofascial pain dysfunction syndrome. *J Dent Res*. 1979;58(9):1866-1871.
2. Flor H, Birbaumer N, Schugens MM, Lutzenberger W. Symptom-specific psychophysiological responses in chronic pain patients. *Psychophysiology*. 1992;29(4):452-460.
3. Cioffi I, Landino D, Donnarumma V, Castroflorio T, Lobbezoo F, Michelotti A. Frequency of daytime tooth clenching episodes in individuals affected by masticatory muscle pain and pain-free controls during standardized ability tasks. *Clin Oral Investig*. 2017;21(4):1139-1148. https://doi.org/10.1007/s00784-016-1870-8. Online First: Epub Date]|.
4. Kapel L, Glaros AG, McGlynn FD. Psychophysiological responses to stress in patients with myofascial pain-dysfunction syndrome. *J Behav Med*. 1989;12(4):397-406.
5. Katz JO, Rugh JD, Hatch JP, Langlais RP, Terezhalmy GT, Borcherding SH. Effect of experimental stress on masseter and temporalis muscle activity in human subjects with temporomandibular disorders. *Arch Oral Biol*. 1989;34(6):393-398.
6. Moss RA, Adams HE. Physiological reactions to stress in subjects with and without myofascial pain dysfunction symptoms. *J Oral Rehabil*. 1984;11(3):219-232.
7. Nicholson RA, Townsend DR, Gramling SE. Influence of a scheduled-waiting task on EMG reactivity and oral habits among facial pain patients and no-pain controls. *Appl Psychophysiol Biofeedback*. 2000;25(4):203-219.
8. Ohrbach R, Blascovich J, Gale EN, McCall WD Jr, Dworkin SF. Psychophysiological assessment of stress in chronic pain: comparisons of stressful stimuli and of response systems. *J Dent Res*. 1998;77(10):1840-1850.
9. Glaros AG. The role of parafunctions, emotions and stress in predicting facial pain. *J Am Dent Assoc*. 2005;136(4):451-458.
10. Flor H, Turk DC. Psychophysiology of chronic pain: do chronic pain patients exhibit symptom-specific psychophysiological responses? *Psychol Bull*. 1989;105(2):215-259.
11. Flor H, Birbaumer N, Schulte W, Roos R. Stress-related electromyographic responses in patients with chronic temporomandibular pain. *Pain*. 1991;46(2):145-152.
12. Glaros AG, Marszalek JM, Williams KB. Longitudinal multilevel modeling of facial pain, muscle tension, and stress. *J Dent Res*. 2016;95(4):416-422. https://doi.org/10.1177/0022034515625216. [published Online First: Epub Date]|.
13. Schwartz LL. A temporomandibular joint pain-dysfunction syndrome. *J Chronic Dis*. 1956;3:284-293.
14. Laskin DM. Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc*. 1969;79:147-153.
15. Le Resche L, Truelove EL, Dworkin SF. Temporomandibular disorders: a survey of dentists' knowledge and beliefs. *J Am Dent Assoc*. 1993;124:90-94.
16. Porto FB, Litt M, Jennings ME, Rifaey H, Reisine S. Knowledge and beliefs regarding TMD: has anything changed after 20 years? *Health Scope*. 2016;5(2): https://doi.org/10.17795/jhealthscope-31328. [published Online First: Epub Date]|.
17. van Selms MK, Visscher CM, Knibbe W, Thymi M, Lobbezoo F. The association between self-reported wake oral behaviors and orofacial pain depends on the belief of patients that these behaviors are harmful to the jaw. *J Oral Facial Pain Headache*. 2020;343:273-280. https://doi.org/10.11607/ofph.2629. [published Online First: Epub Date]|.
18. Raphael KG, Sirois DA, Janal MN, et al. Sleep bruxism and myofascial temporomandibular disorders: a laboratory-based polysomnographic investigation. *J Am Dent Assoc*. 2012;143(11):1223-1231.
19. Raphael KG, Janal MN, Sirois DA, Svensson P. Effect of contingent electrical stimulation on masticatory muscle activity and pain in patients with a myofascial temporomandibular disorder and sleep bruxism. *J Orofac Pain*. 2013;27(1):21-31. https://doi.org/10.11607/jop.1029. [published Online First: Epub Date]|.
20. Raphael KG, Janal MN, Sirois DA, et al. Validity of self-reported sleep bruxism among myofascial temporomandibular disorder patients and controls. *J Oral Rehabil*. 2015;42(10):751-758. https://doi.org/10.1111/joor.12310. Online First: Epub Date]|.
21. Dubrovsky B, Raphael KG, Lavigne GJ, et al. Polysomnographic investigation of sleep and respiratory parameters in women with temporomandibular pain disorders. *J Clin Sleep Med*. 2014;10(02):195-201. https://doi.org/10.5664/jcsm.3452. [published Online First: Epub Date]|.
22. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord*. 1992;6:301-355.
23. Jennings JR, Kamarck T, Stewart C, Eddy M, Johnson P. Alternate cardiovascular baseline assessment techniques: vanilla or resting baseline. *Psychophysiology*. 1992;29(6):742-750.
24. Karavidas MK, Lehrer PM, Lu SE, Vaschillo E, Vaschillo B, Cheng A. The effects of workload on respiratory variables in simulated flight: A preliminary study. *Biol Psychol*. 2010;84(1):157-160. https://doi.org/10.1016/j.biopsycho.2009.12.009. Epub 2010 Jan 12.[published Online First: Epub Date]|.
25. Lesnewich LM, Conway FN, Buckman JF, et al. Associations of depression severity with heart rate and heart rate variability in young adults across normative and clinical populations. *Int J Psychophysiol*. 2019;142:57-65. https://doi.org/10.1016/j.ijpsycho.2019.06.005. [published Online First: Epub Date]|.
26. Dembroski TM, MacDougall JM, Herd J, Shields JL. Effect of level of challenge on pressor and heart rate responses in Type A and B subjects. *J Appl Soc Psychol*. 1979;9(3):209-228.
27. Berntson GG, Cacioppo JT, Binkley PF, Uchino BN, Quigley KS, Fieldstone A. Autonomic cardiac control. III. Psychological stress and cardiac response in autonomic space as revealed by pharmacological blockades. *Psychophysiology*. 1994;31:599-608.
28. Kirschbaum C, Pirke KM, Hellhammer DH. The 'Trier Social Stress Test'–a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*. 1993;28:76-81.
29. Uchino BN, Holt-Lunstad J, Bloor LE, Campo RA. Aging and cardiovascular reactivity to stress: longitudinal evidence for changes in stress reactivity. *Psychol Aging*. 2005;20(1):134-143.

30. Carlsen AN, Chua R, Inglis JT, Sanderson DJ, Franks IM. Startle response is dishabituated during a reaction time task. *Exp Brain Res*. 2003;152(4):510-518.
31. Spielberger C, Gorsuch R, Lushene R. *Manual for the State-Trait Anxiety Inventory*. Consulting Psychologists Press; 1970.
32. Cacioppo JT, Berntson GG, Tassinary LG. *Handbook of Psychophysiology*, 4th ed. Cambridge University Press; 2016.
33. Manfredini D, Lombardo L, Siciliani G. Temporomandibular disorders and dental occlusion. A systematic review of association studies: end of an era? *J Oral Rehabil*. 2017;44(11):908-923. https://doi.org/10.1111/joor.12531. [published Online First: Epub Date]|.
34. Yemm R. Comparison of the activity of left and right masseter muscles of normal individuals and patients with mandibular dysfunction during experimental stress. *J Dent Res*. 1971;50(5):1320-1323.
35. Glaros AG, Forbes M, Shanker J, Glass EG. Effect of parafunctional clenching on temporomandibular disorder pain and proprioceptive awareness. *Cranio*. 2000;18(3):198-204.
36. Glaros AG, Waghela R. Psychophysiological definitions of clenching. *Cranio*. 2006;24(4):252-257.
37. Glaros AG, Williams K. Tooth contact versus clenching: oral parafunctions and facial pain. *J Orofac Pain*. 2012;26(3):176-180.
38. Glaros AG, Williams K, Lausten L. The role of parafunctions, emotions and stress in predicting facial pain. *J Am Dent Assoc*. 2005;136(4):451-458.
39. Hoemann K, Khan Z, Feldman MJ, et al. Context-aware experience sampling reveals the scale of variation in affective experience. *Sci Rep*. 2020;10:12459.
40. Wormwood JB, Khan Z, Siegel E, et al. Physiological indices of challenge and threat: a data-driven investigation of autonomic nervous system reactivity during an active coping stressor task. *Psychophysiology*. 2019;56:e13454.
41. LeResche L. Epidemiology of temporomandibular disorders: implications for the investigation of etiologic factors. *Crit Rev Oral Biol Med*. 1997;8(3):291-305.
42. Rollman A, Visscher CM, Gorter RC, Naeije M. Care seeking for orofacial pain. *Journal of orofacial pain*. 2012;26(3):206-214.
43. Fillingim RB. Sex, gender, and pain: women and men really are different. *Curr Rev Pain*. 2000;4(1):24-30.
44. Fillingim RB, Maixner W. Sex-related factors in temporomandibular disorders. In: Fillingim RB, ed. *Sex, Gender and Pain*. IASP Press; 2000:309-325.
45. LeResche L, Mancl L, Sherman JJ, Gandara B, Dworkin SF. Changes in temporomandibular pain and other symptoms across the menstrual cycle. *Pain*. 2003;106(3):253-261. https://doi.org/10.1016/j.pain.2003.06.001. [published Online First: Epub Date]|.

How to cite this article: Janal MN, Lobbezoo F, Quigley KS, Raphael KG. Stress-evoked muscle activity in women with and without chronic myofascial face pain. *J Oral Rehabil*. 2021;48:1089–1098. https://doi.org/10.1111/joor.13238