Alterations in Masticatory Muscle Activation in People with Persistent Neck Pain Despite the Absence of Orofacial Pain or Temporomandibular Disorders

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Aim: To assess whether patients with persistent neck pain display evidence of altered masticatory muscle behavior during a jaw-clenching task, despite the absence of orofacial pain or temporomandibular disorders. **Methods:** Ten subjects with persistent, nonspecific neck pain and 10 age- and sex-matched healthy controls participated. Maximal voluntary contractions (MVCs) of unilateral jaw clenching followed by 5-second submaximal contractions at 10%, 30%, 50%, and 70% MVC were recorded by two flexible force transducers positioned between the first molar teeth. Task performance was quantified by mean distance and offset error from the reference target force as error indices, and standard deviation of force was used as an index of force steadiness. Electromyographic (EMG) activity was recorded bilaterally from the masseter muscle with 13 × 5 grids of electrodes and from the anterior temporalis with bipolar electrodes. Normalized EMG root mean square (RMS) was computed for each location of the grid to form a map of the EMG amplitude distribution, and the average normalized RMS was determined for the bipolar acquisition. Between-group differences were analyzed with the Kruskal Wallis analysis of variance. **Results:** Task performance was similar in patients and controls. However, patients displayed greater masseter EMG activity bilaterally at higher force levels ($P < .05$). **Conclusion:** This study has provided novel evidence of altered motor control of the jaw in people with neck pain despite the absence of orofacial pain or temporomandibular disorders.
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Keywords: *bite force, electromyography, masticatory muscles, motor control, neck pain*

Neck pain is a disabling musculoskeletal disorder, with the majority of people experiencing recurrence of symptoms following their first episode.[1] Maladaptive motor behavior during the initial pain episode may partially explain poor recovery.[2]

It is well documented that people with neck pain display a number of changes in neck muscle behavior, including increased superficial neck muscle activity during isometric contractions[3–6] and functional upper limb activities,[7–9] reduced specificity of neck muscle activity,[10,11] and reduced activity of the deep cervical flexors[12] and deep extensors[13–15] during isometric tasks.

Studies indicate that people with temporomandibular disorders (TMD) and/or orofacial pain may also display changes in activation of their neck flexor and extensor muscles.[16,17] This is not surprising considering the neurophysiologic, biomechanical, and functional associations between the cervical and orofacial regions[18–21] as well as the clinical association often observed between neck pain and TMD.[22] Based on these considerations, it may be expected that patients with neck pain would display altered motor control of the jaw, especially since animal studies have shown that cervical nociceptive inputs excite trigeminal brainstem nociceptive neurons and evoke an increase in jaw muscle activity.[18–21] However, the effect of neck pain, in the absence of TMD or orofacial pain, on the neural control of the jaw in humans has not been investigated.

The aim of this study was to assess whether patients with persistent neck pain display evidence of altered masticatory muscle behavior during a jaw-clenching task, despite the absence of orofacial pain or TMD. To achieve this aim, the study compared bite force control, measured by indices of precision and steadiness, and masticatory muscle activity between people with persistent neck pain and healthy age- and sex-matched controls. It was hypothesized that people with persistent neck pain would show indications of altered motor control of jaw clenching.

Materials and Methods

Subjects

Ten volunteers with persistent nonspecific neck pain were recruited from the Pain Clinic of the University Hospital in Göttingen, Germany. These patients were included if they were between 18 and 45 years of age and had a history of neck pain ≥ 3 months over a continuous period during the last year, with an average pain intensity of ≥ 3/10 on a numeric rating scale (NRS).[23]

Ten age- and sex-matched healthy individuals were recruited to act as the control group. Pain-free participants were included if they had no relevant history of neck or shoulder pain or injury that limited their function and/or required treatment from a health care professional. Patients and control subjects had to have the capacity to give written informed consent at their own will. Ethical approval for the study was granted by the local Ethics Committee (14/11/14) and the procedures were conducted according to the Declaration of Helsinki.

Participants were excluded from both groups if they had any major circulatory, neurologic, or respiratory disorders; recent or current pregnancies; previous spinal surgery; current treatment for neck pain from health care providers; participation in neck muscle exercise in the past 12 months; presence of orofacial pain or any Axis I TMD assessed with the new Diagnostic Criteria for TMD[24]; or absence of molar or premolar teeth (the absence of wisdom teeth was not considered). Participants were also excluded from either group if they were taking medications such as opioids, anticonvulsants, or antidepressants, or regularly taking high-dose nonsteroidal anti-inflammatory drugs (NSAIDs); however, taking NSAIDs as needed was allowed. Initial screening was accomplished by telephone, and eligible persons attended a baseline evaluation appointment where they were screened by a physiotherapist and a medical doctor. Both groups were asked not to take NSAIDs on the day of the experiment.

Questionnaires

A questionnaire was administered to document subject demographics, history, average pain intensity, and duration of pain. Patients completed the State-Trait Anxiety Inventory (STAI), a 40-item questionnaire that has been shown to be a reliable and sensitive measure of anxiety.[25] The Neck Disability Index (NDI) was used to assess pain-related disability specifically related to neck pain (10 items).[26] Patients also completed the Short Form (SF-36) Health Survey,[27] a measure of the general health status of the patient, as well as the Tampa Scale for Kinesiophobia (17 items),[28] a measure of fear-avoidance behavior and fear-avoidance beliefs. Finally, the patients were asked to verbally rate their current level of perceived pain intensity at the beginning of the session on an 11-point NRS anchored with "no pain" (0) and "the worst possible pain imaginable" (10).

Procedures

The subjects sat upright with their back supported, their arms resting on their lap, hips and knees in 90 degrees of flexion, and their feet flat on the floor. Subjects wore a lightweight helmet that housed two laser pointers. They were asked to assume their natural neutral head position, at which point the positions of the projected laser beams were marked on the wall in front of the subject to ensure a comparable head and neck posture throughout the experiment.

A force sensor (see below), housed in a 7-mm-thick soft envelope, was positioned over the first mandibular molar. The bite force was displayed as a red cursor in real time on a PC monitor, positioned 100 cm in front of the subject. Subjects performed twice a maximum voluntary contraction (MVC) of unilateral jaw clenching on both the right and left sides with a 1-minute rest in between. During each MVC, the subjects were encouraged to reach the maximal force over 5 seconds. The maximum value achieved during right-sided and left-sided jaw clenching was retained as the MVC.

A training session was provided before data acquisition. It consisted of targeting 35% and 65% of MVC for 5 seconds, separated by 5 seconds of rest. This was performed on both the right and left sides. The experimental procedure then involved matching four force targets, representing 10%, 30%, 50%, and 70% of MVC, which were displayed randomly. Subjects performed these four contractions on both the right and left sides (order randomized with 2 minutes of rest in between). The targets were displayed as rectangular steps with heights corresponding to the target percentage of MVC with a standard length of 5 seconds separated by 5 seconds of rest.

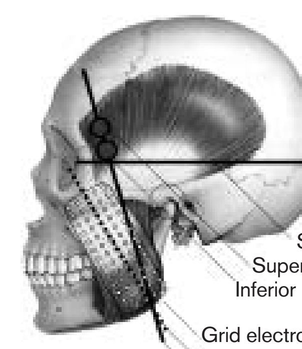


Fig 1 Schematic representation of electrode locations. The electrode grid was placed over the superficial bundle of the masseter muscle, and the mandibular angle-cantus line *(dotted line)* was used as a reference for the central column of the grid. The bipolar electrodes *(grey circles)* were positioned over the anterior temporalis, with the intersection between the superior horizontal line and the line passing through the mandibular angle and the condylar head, rotated forward 20 degrees, as a reference line.

Force

Bite force was registered with piezoresistive force transducers (Flexiforce A201, Tekscan) with a maximum load of 784.5 N. The force signal was amplified (2-channel force amplifier, OT Bioelettronica), sampled at 15 Hz and converted to digital form by a 16-bit analog-to-digital converter.

The following indices were calculated for each force target to characterize the task performance obtained at the different force levels:

- The mean distance (MD) indicates the overall goodness of task execution and is represented by the average value of the difference between the absolute values of the force delivered by the subject and the force target.
- The offset error (OE) characterizes the degree of over/undershooting of the force delivered by the subject with respect to the force target. It is calculated as the difference between the mean of the force values obtained by the subject and the force target.
- The standard deviation (SD) characterizes the precision of the performance and measures the steadiness of force irrespective of the reference target (ie, a subject may show a very high MD and OE combined with a low SD).

The three indices were computed over the three central seconds (seconds 2 to 4) of each reference target and reported as percentage of the MVC. These indices have been shown to be a valid and reliable method to characterize jaw-clenching performance.[29]

Electromyography

Electromyographic activity was detected with two semidisposable adhesive grids of electrodes (OT Bioelettronica) placed on the skin over the masseter muscle bilaterally. Each grid consisted of 13 rows and 5 columns of electrodes (1-mm diameter, 8-mm interelectrode distance in both directions) with one electrode absent from the corner. The position corresponding to the missing electrode was used as the origin of the coordinate system to define the electrode location (the origin and orientation of the coordinate system was adjusted according to the laterality). The subject's skin was prepared by gentle local abrasion with abrasive paste (Medic-Every) and cleaned with water. Each grid was located with the third column aligned with the mandibular angle–cantus straight line[30] (Fig 1). Conductive gel (30 µL) was inserted into each cavity of the grid to provide electrode-skin contact.

EMG activity was recorded from the anterior temporalis muscle bilaterally with Ag/AgCl electrodes (Ambu Neuroline; conductive area: 28 mm^2) following skin preparation. Two reference lines were considered to standardize electrode placement. The first was a straight line passing through the mandibular angle and condylar head, rotated forward with an inclination of 20 degrees. The second was the superior horizontal line, parallel to the Frankfurt plane passing through the cantus. The inferior electrode was placed at the intersection of the two reference lines, and the superior electrode was positioned superiorly.[30] A reference electrode was placed over the spinous process of the seventh cervical vertebra. The bipolar EMG activity was amplified (USB-EMG2, OT Bioelettronica; –3 dB bandwidth 10 to 500 Hz) by a factor of 2,000, sampled at 2,048 Hz, and converted to digital form by a 12-bit analog-to-digital converter.

A total of 59 bipolar EMG signals were obtained from each grid (12 longitudinal bipolar recordings in each column except the far right, which had 11 electrode pairs) (Fig 2). Root mean square (RMS) values were computed from each bipolar recording from adjacent, non-overlapping signal epochs of 1-second duration, as described previously.[31] For graphic representation, the 59 values were interpolated by a factor of 8, but only the original values were used for data processing and statistical analysis. To characterize the spatial distribution of muscle activity, the following variables were extracted from the 59 bipolar signals: RMS averaged over the 59 signals and the two coordinates of the centroid of the root mean square map (x- and y-axis coordinates for the ventrodorsal and craniocaudal directions, respectively).[32–34]

The coordinates of the centroid of the RMS map were computed as follows:

$$x_c = \frac{\sum_{i=1}^{5}\sum_{j=1}^{12} rms\ (i,j) \cdot i}{\sum_{i=1}^{5}\sum_{j=1}^{12} rms\ (i,j)}$$

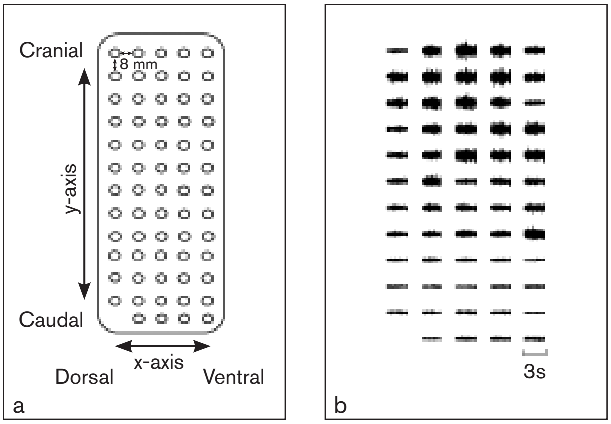
$$y_c = \frac{\sum_{i=1}^{5}\sum_{j=1}^{12} rms\ (i,j) \cdot j}{\sum_{i=1}^{5}\sum_{j=1}^{12} rms\ (i,j)}$$

where *rms (i,j)* is the RMS value at column i and row j, and x_c and y_c are computed in interelectrode distance units. The two coordinates are reported in millimeters in the results.

Values of RMS (for the electrode grid and bipolar electrodes) and x- and y-axis coordinates of the centroid (for the electrode grid only) were obtained by averaging the results from three adjacent windows, the second of which was aligned to the central sample of the contraction trial. Therefore, the EMG variables were calculated over the 3 central seconds for each target. The actual duration of the trial was determined as the interval in which the force signal exceeded a threshold of three times the SD of its resting value. For the computation of the resting value, the subject was instructed to relax prior to each trial while the signal was continuously recorded. The RMS values during the submaximal contractions were normalized relative to the maximum RMS detected during the MVC and expressed as a percentage.

Statistical Analyses

Nonparametric statistical analyses were performed to account for the relatively small sample of the groups. The Kruskal Wallis analysis of variance (ANOVA) tests were applied to evaluate differences between groups (neck pain, controls) for all masseter EMG variables (RMS, y- and x-coordinate of the centroid) recorded bilaterally at each force level. Furthermore, Kruskal Wallis ANOVAs were used to evaluate the difference in anterior temporalis RMS between groups (neck pain, controls) recorded bilaterally at each force level. Kruskal Wallis ANOVAs were also applied to the force variables (MVC, MD, OE, and SD) to compare between groups, and MD, OE, and SD were evaluated for each force level. Results are reported as mean and SD in the text. Statistical significance was set at $P < .05$.



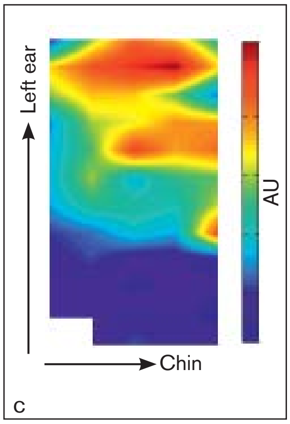


Fig 2 **(a)** Schematic representation of the electrode grid with indication of the coordinate axes. **(b)** A total of 59 bipolar EMG signals were obtained from each grid (12 longitudinal bipolar recordings in each column except the far left, which had 11 electrode pairs). **(c)** For graphic representation, the 59 values were interpolated by a factor of 8 to generate a topographic map of the EMG amplitude. Only the original values were used for data processing and statistical analysis.

Table 1 Baseline Characteristics of the Neck Pain and Control Groups

Characteristic	Neck pain (n = 10)	Control (n = 10)
Age (y)	28.9 ± 6.0	27.2 ± 5.8
Sex (% female)	70	70
Height (cm)	175.2 ± 11.4	170.0 ± 7.0
Weight (kg)	64.8 ± 7.9	63.9 ± 11.6
Duration of pain (mo)	67.0 ± 64.5	
Current pain intensity (NRS)	5.2 ± 1.5	
Neck disability index (%)	22.5 ± 7.1	
SF-36		
Physical	46.5 ± 5.3	
Mental	47.6 ± 11.0	
TSK	29.2 ± 5.0	
STAI	48.7 ± 5.8	

Values are presented as mean ± SD. NRS = numeric rating scale for intensity of neck pain; TSK = Tampa Scale for Kinesiophobia; STAI = Spielberger State-Trait Anxiety Inventory; SF-36 = Short Form Health Survey.

Results

Baseline characteristics of the patient and control groups are presented in Table 1. No significant differences in age, weight, or height were detected between groups (all $P > .05$).

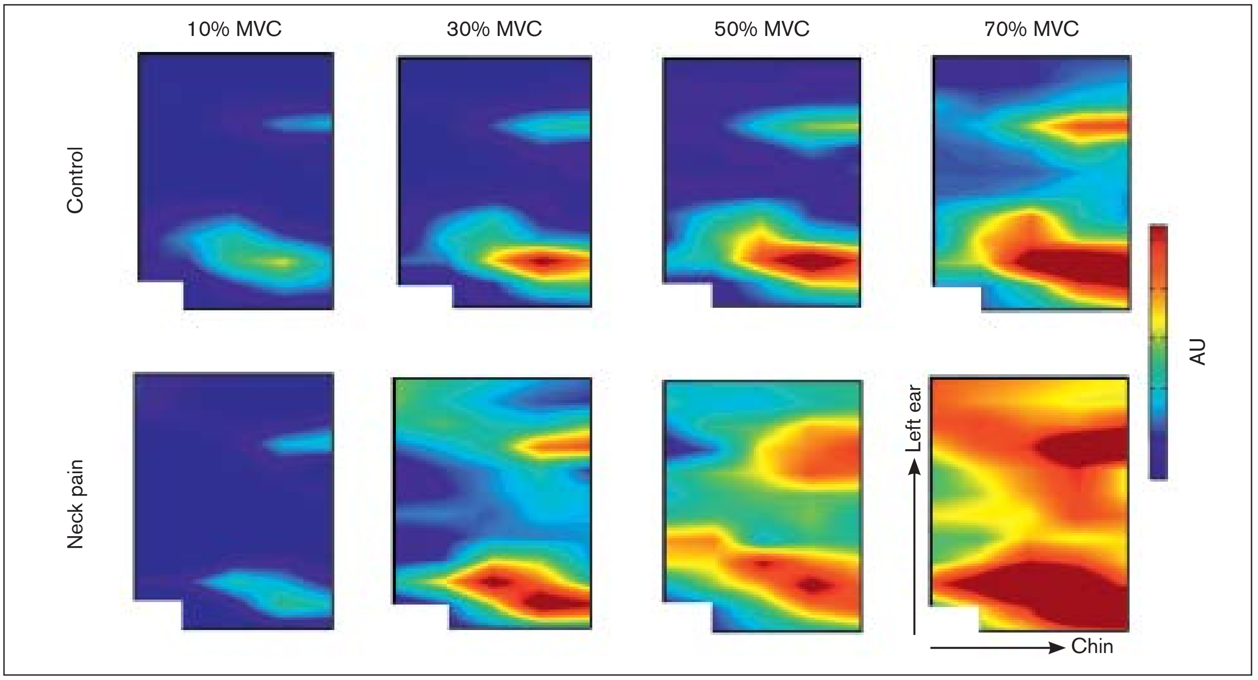


Fig 3 Representative topographic maps (interpolation by a factor 8) of the electromyographic root mean square (EMG RMS) values recorded from the ipsilateral masseter for *(top row)* a control subject and *(bottom row)* a patient with neck pain performing right-sided unilateral jaw clenching at 10%, 30%, 50%, and 70% of MVC. The bottom left section of the map corresponds to the location of the absent electrode. The arrows indicate the orientation of the electrode grid over the masseter muscle with respect to the ventrodorsal and craniocaudal directions. Areas of dark red correspond to high EMG amplitude and areas of blue to low EMG amplitude. Values are expressed in arbitrary units (AU). Note the larger increase in EMG amplitude for the person with neck pain, especially at the higher force levels. Overall, the patient group showed significantly greater average ipsilateral masseter RMS values (averaged across the entire grid of electrodes) at the force target of 70% MVC ($P < .05$).

Table 2 Force Exerted During the Maximal Voluntary Contraction (MVC) of Jaw Clenching on the Right and Left Sides and Force Variables (MD, OE, SD) Determined During the Submaximal Jaw-Clenching Tasks at 10%, 30%, 50%, and 70% MVC

Force variable	Neck pain (n = 10)	Control (n = 10)
MVC (N)		
Left	356.1 ± 189.3	352.2 ± 253.1
Right	306.1 ± 170.7	334.5 ± 167.8
MD (%)		
10%	1.3 ± 0.4	1.7 ± 1.4
30%	3.3 ± 1.3	3.9 ± 2.5
50%	6.0 ± 5.4	6.9 ± 7.4
70%	7.1 ± 3.7	8.5 ± 7.5
OE (%)		
10%	−0.4 ± 0.7	−0.8 ± 1.7
30%	−1.8 ± 2.0	−3.0 ± 2.0
50%	−4.1 ± 5.7	−5.6 ± 7.4
70%	−5.6 ± 4.1	−7.1 ± 8.0
SD (%)		
10%	1.2 ± 0.5	1.2 ± 0.7
30%	3.1 ± 1.2	3.4 ± 1.9
50%	5.2 ± 3.6	5.2 ± 3.5
70%	6.6 ± 3.9	6.9 ± 3.1

Values of the neck pain and control groups are presented as mean ± SD.
MD = mean distance; OE = offset error; SD = standard deviation.

Force

The force variables for the neck pain and control groups are presented in Table 2. No significant group differences were evident for right MVC, left MVC, MD, OE, or SD (all $P > .05$).

Electromyography

Representative topographic maps of the masseter EMG RMS values recorded for a control subject and a patient are presented in Fig 3. The patient group showed significantly greater average ipsilateral masseter RMS values (averaged across the entire grid of electrodes) at the force target of 70% MVC ($P < .05$), and greater contralateral masseter RMS values ($P < .05$) at the force targets of 30%, 50%, and 70% MVC (Fig 4). Despite differences in the amplitude of masseter EMG activity between groups, the distribution of activity across the muscle was the same for the patients and controls for both the y-coordinate and x-coordinate (both $P > .05$) of the centroid of the RMS map (Fig 5). Moreover, no differences in anterior temporalis RMS were identified between groups ($P > .05$; Fig 6).

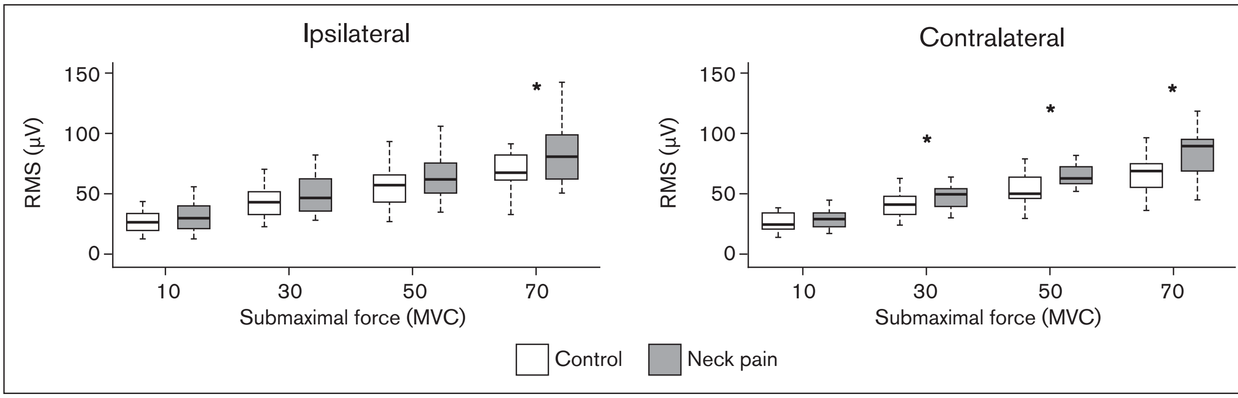


Fig 4 Boxplots of the average normalized root mean square (RMS) values recorded from the ipsilateral and contralateral masseter muscles during unilateral jaw clenching at 10%, 30%, 50%, and 70% of the maximal voluntary contraction (MVC) in the control and patient groups. *$P < .05$

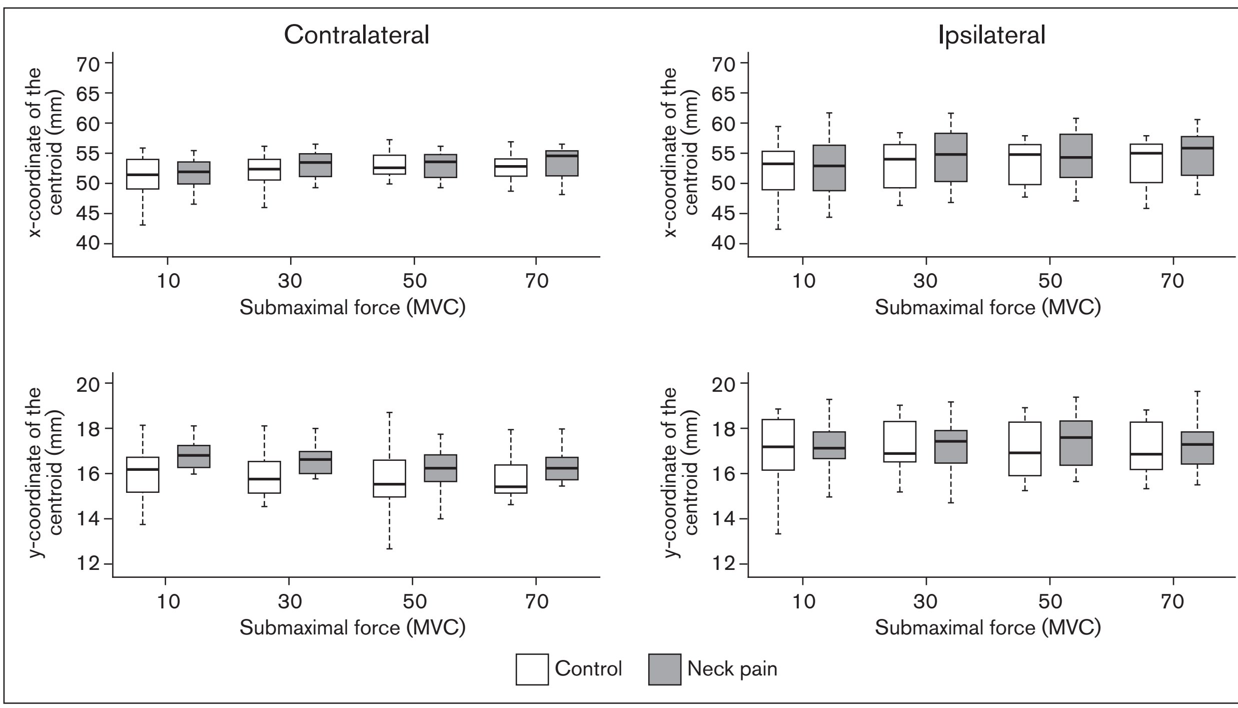


Fig 5 Boxplots of the *(top row)* x- and *(bottom row)* y-coordinates of the root mean square (RMS) map recorded from the ipsilateral and contralateral masseter muscles during unilateral jaw clenching at 10%, 30%, 50%, and 70% of the maximal voluntary contraction (MVC) in the control and patient groups.

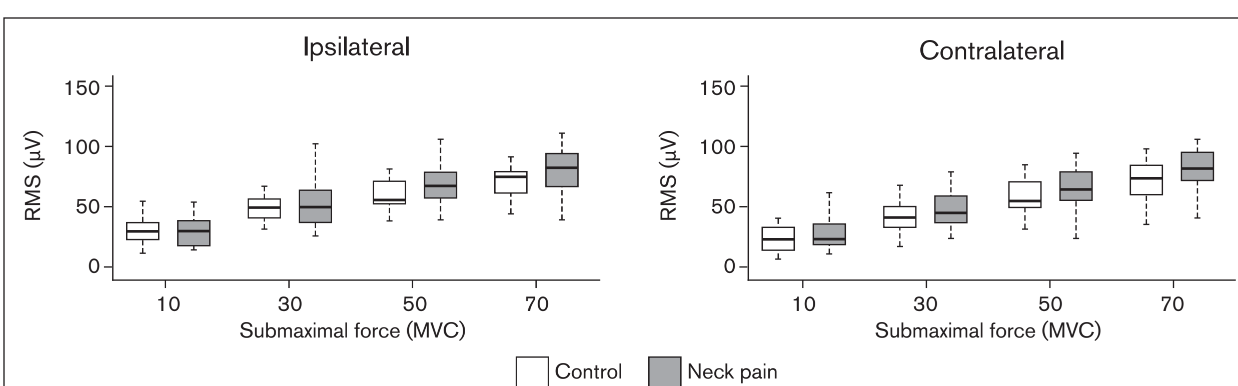


Fig 6 Boxplots of the average normalized root mean square (RMS) values recorded from the ipsilateral and contralateral anterior temporalis muscles during unilateral jaw clenching at 10%, 30%, 50%, and 70% of the maximal voluntary contraction (MVC) in the control and patient groups.

Discussion

Despite a comparable motor performance to asymptomatic individuals, patients with persistent neck pain displayed elevated EMG activity of their masseter muscles during a unilateral jaw-clenching task. Differences with controls were present at higher force levels, as indicated by higher RMS of both the ipsilateral and contralateral masseter muscles at 70% of MVC and of the contralateral masseter also at 30% and 50% MVC.

Bilateral Masseter Muscle Activity During Unilateral Clenching

The pattern of masseter muscle activation resembled the expected physiologic activations observed previously in healthy subjects.[35] The comparable activation of the contralateral masseter muscle during unilateral jaw clenching is likely attributed to the bilateral and symmetric anatomical corticobulbar projections[36] and to the presence of shared presynaptic inputs at the level of the trigeminal motor nucleus.[37] These would contribute to the synchronization of motor units during tonic voluntary contraction,[38] although functional studies show contradictory results, as reported in Ortu et al.[39]

Force

The MVC force values of ~350 N were similar between groups and comparable with previous studies.[40] Although some studies have reported higher values,[35,41] this difference is most likely attributable to different methods of measurement[42] and to the sex composition of the groups in the current study, in which women, who usually have a lower MVC, accounted for 70% of the subjects. The lack of difference in MVC between groups may not be surprising considering that the patients did not report orofacial pain. Differences would more likely be observed in MVC between groups if the patient group suffered from orofacial pain or TMD, which could either directly or indirectly (eg, through fear) inhibit their maximal performance.

The values of the error indices were consistent with those found in the validation study of the device[29] used at the same target force levels. No significant differences in task performance were observed between the asymptomatic and neck pain subjects as assessed with the indices of MD, OE, and SD. However, previous studies have shown that the motor output can remain unaltered so that the task is executed in the same manner in the presence of pain.[43,44] This has been attributed to reorganization of activity among synergistic muscles.

Elevated Masseter Muscle Activity in Neck Pain

Previous research has documented the presence of sensorimotor disturbances of the neck muscles in subjects with TMD or orofacial pain.[45] In addition, animal studies have shown that cervical nociceptive inputs excite trigeminal brainstem nociceptive neurons and can evoke an increase in jaw muscle EMG activity.[18–21] The current results support the possibility of a bidirectional relationship between neck/jaw pain and motor disturbances; that is, subjects with persistent neck pain displayed changes in the activation of their masticatory muscles.

The current data add to the body of knowledge on the alterations of sensorimotor control observed in people with persistent neck pain disorders. Moreover, the results demonstrate that altered neuromuscular control can be observed also in regions apparently unrelated to the source of pain. However, it is well known that the neck and jaw share common neurophysiologic pathways, as represented by the trigeminal brainstem sensory nuclei that receive afferents from both the jaw and a considerable portion of the cervical spine,[18–21] which could explain these findings. A functional relationship between the jaw and neck has also been demonstrated previously in humans.[46] For example, normally coordinated head extension–mouth-opening and head flexion–mouth-closing movements occur, but in people with whiplash-induced neck pain there is a reduction in the amplitude of movement and a delay in head extension in relation to mouth opening.[47,48]

The main observation in the current study was elevated masseter EMG activity in people with neck pain during jaw clenching at submaximal loads equal to or greater than 30% MVC. Elevated superficial neck muscle activity has also been observed in people with neck pain during isometric neck contractions[3–6] and functional upper limb activities,[7–9] and a coactivation of neck muscles during submaximal biting has been demonstrated in healthy subjects.[49] The range of forces between 10% and 30% MVC are commonly experienced during chewing.[50] Although speculative, it may be plausible that the weight of the head and inherent neck stiffness were sufficient to stabilize the head movement against the torque created at the mandible during these low-load contractions. However, when higher forces were required, neck muscle control may have been inadequate in the patient group and compensatory elevated activity of the masseter muscles was required to accomplish the bite task. A further possible explanation of the elevated masticatory muscle activity in neck pain may be attributed to direct changes in the neural control of the masticatory muscles. Afferents from the cervical region converge on the trigeminal brainstem sensory nuclei[18–21] and potentially their action at this level and/or at supraspinal levels may modify the motor response of the masticatory muscles in the presence of ongoing persistent pain. However, the specific mechanisms underlying the observation of elevated masseter EMG activity in neck pain cannot be determined from the current results.

Although altered masseter EMG activity was noted during the submaximal contractions, this was not reflected in the performance of the task or the ability to produce a maximal voluntary contraction. It should be noted that the tasks performed (ie, maximal effort versus force accuracy task) differ substantially, and the submaximal tasks were more challenging and demanding since the subject had to correct the force on the basis of visual feedback, whilst during the MVC the precision of force delivery was irrelevant.

Distribution of Masseter Muscle Activity

The centroid of the RMS indicates the spatial distribution of the activity within the studied muscle.[33] Unlike previous observations in experimental[51,52] and clinical pain,[53,54] no significant differences in the spatial distribution of muscle activity were observed between groups. However, these previous studies have observed changes in the spatial distribution of muscle activity in the muscle at the site of pain, unlike the current study, in which the muscle monitored was remote to the site of the patient's pain.

Methodologic Considerations

Although trends suggested elevated anterior temporalis EMG activity in the neck pain group, no significant differences were observed. This lack of significant difference could be attributed to the small sample size, which is a limitation of the current study. However, it should also be noted that the EMG activity of the anterior temporalis was measured with standard bipolar electrodes, which, unlike high-density EMG electrode grids, provide a limited evaluation of muscle activation. Sampling over a larger area or different region may have revealed larger and significant differences between groups. That said, the use of the electrode grid to record masseter EMG activity may have been prone to crosstalk from facial muscles.

The force transducer used in this study measures force in bidimensional space. Force transducers that detect forces over a three-dimensional space may have yielded differences in the force variables. Further, the small sample size may diminish the generalizability of the findings, although both the range of forces and the RMS values were comparable to other studies,[33,35] making it plausible that the current study sample was representative. Furthermore, the multiple comparison may have led to type I error, especially since nonparametric tests were used.

Conclusions

This study has provided novel evidence of altered masticatory muscle activity in people with neck pain, despite the absence of orofacial pain or signs of TMD. Further studies are needed to better understand the mechanisms underlying the occurrence of increased EMG activity of the masticatory muscles and the clinical relevance of this finding.

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