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Reduced thermal threshold in patients with Temporomandibular Disorders

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SUMMARY Background Many studies have demonstrated the presence of somatosensory modulation changes at different sites in patients with temporomandibular disorders (TMDs) using different modalities. However, the neck area, a well-know condition related to TMD, remains unexplored. Objective To assess the thermal pain threshold in patients with TMD and controls at cephalic and extra-cephalic areas, including the neck. Methods Twenty female patients with TMDs diagnosed by the Research Diagnostic Criteria for TMD (RDC/TMD) and twenty age-matched controls underwent a first interview about neck pain and disability (NDI questionnaire). A blinded evaluator assessed the thermal pain threshold for cold (CPT) and heat (HPT) stimuli in accordance with an ascending method of limits of the Quantitative Sensory Testing at the following sites: periorbital, masseter, cervical posterior and ventral forearm. The groups were compared using a *t*-test with $\alpha = 5\%$. Results Patients with TMDs reported pain at higher temperature for cold stimuli in all sites ($P < 0{\cdot}05$) and at lower temperature for heat stimuli in the right periorbital site ($P < 0{\cdot}05$) than controls. Pain and disability due tothis symptom were reported more often in the TMD group ($P < 0{\cdot}05$). Conclusion Patients with TMD have pain modulation changes in the neck area as well, especially for cold stimuli, associated with higher disability and a higher report of neck pain than controls. These findings reinforce the evidence regarding the relationship between TMDs and neck pain.

KEYWORDS: central nervous system sensitisation, orofacial pain, pain threshold, somatosensory disorders, temporomandibular disorders, thermal allodynia

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Background

Temporomandibular disorders (TMDs) are a broad group of conditions that affect 12% of the population (1) where pain can be located in one or more the following areas: temporomandibular joint (TMJ), masticatory muscles and related tissues (2, 3). The signs and symptoms of TMDs include facial pain, tenderness to palpation of the masticatory structures, decreased mandibular range of motion, joint noise (clicking, popping or crepitus), myofascial pain and functional limitations (4, 5).

According to the Research Criteria for temporomandibular disorders, TMDs can be divided into three general groups according of the aetiology of the pain: arthrogenic, myogenic and mixed (6). These disorders are frequently associated with other chronic conditions such as fibromyalgia (7), headache (8–10), sleep disorders (11), myofascial syndrome (12) and neck pain (13).

All these chronic conditions could be related to central sensitisation, process occurring when the pain sensory is amplified (14). In fact, patients with these disorders exhibit long-lasting increases in the

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excitability of spinal cord neurons, which lead to changes in the response of the somatosensory system (15). This central facilitation could then manifest itself as a reduction in the pain threshold (cutaneous allodynia) or as an increase in responsiveness and prolonged after-effects to noxious stimuli (hyperalgesia) (14, 16). Both of these conditions are related to the chronicity process of the disease that might lead to pain despite the absence of pathologies or peripheral pain stimuli. This can reduce the patient response to standard treatments and predispose to the co-occurrence or comorbidity of different disorders (17, 18).

Many studies have verified the presence of somatosensory modulation changes in patients with TMDs using different sensory modalities and conditions (14, 15, 19–23). Vierk *et al.* showed lower thermal pain thresholds in patients with TMD for heat stimuli in the calf, arm and face (14). Also, it has been demonstrated differences in pain threshold for both cold and heat stimuli on cephalic and extra-cephalic areas in patients with TMD (22) and with arthrogenic TMD (23). However, some studies did not find any thermal pain threshold changes in TMD population (19, 21).

Neck pain is a condition with a strict and well-known relationship to TMDs. It has been previously demonstrated that patients with TMDs have a lower pressure pain threshold (24, 25), frequent reports of pain, disability (13) and hypersensitivity in the neck area (15). Also, patients with TMD exhibit movement limitations of high cervical segments and more trigger points in the neck muscles compared to controls (26).

Previous studies demonstrated that cervical spine treatment could improve facial pain symptoms (25) and also TMD treatment through occlusal splints can lead to less cervical pain and increased cervical mobility (27). The relationship between those disorders is justified by anatomic–biomechanical connections and by the convergence of both TMD and neck afferences to the trigeminocervical nucleus (14, 28).

However, there is a lack of information concerning somatosensory modulation of different dermatomes such as the cervical, that could be altered due to the high comorbidity between temporomandibular and neck disorders. Moreover, there is much variability in the pain threshold results depending on the methodology adopted and population characteristics.

The aim of this study was to investigate the thermal pain threshold of patients with TMD compared to healthy controls, in cephalic and extra-cephalic areas, including the cervical site. We hypothesised that the thermal pain threshold would be altered also in the neck region in patients with TMD compared with healthy controls.

Methods

From January 2010 to November 2011, 83 subjects were screened from two Public-based Healthcare Centers of Ribeirão Preto city – Brazil (patients with TMDs) and from the community (healthy controls). Women between 18 and 65 years of age were included if they had artrogenic, myogenic or mixed TMD diagnosis according to the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) (6).

Patients with the following conditions were excluded: chronic diseases such as fibromyalgia, diabetes mellitus, neurologic pain, any type of headache including migraine, report of primary pain conditions or other medical disorders other than TMDs. Also, patients using any medication/substance that could interfere with the pain threshold were not included. Controls with report of any facial pain were not included and the same patients' exclusion criteria were applied for controls recruitment.

After the screening, 20 patients with chronic TMDs and 20 age-matched controls were recruited. The remaining forty-three subjects were excluded and this high exclusion rate is attributed mainly to the presence of any report of headaches or other chronic pain conditions, which are highly related to TMDs.

The study was approved by the Local Ethics Committee (process number: 2010/1950), and all the subjects signed the Consent Form before the assessment.

All the subjects were assessed by a trained evaluator that applied the RDC/TMD – Axis 1, that includes palpation of the orofacial joint and muscles, TMJ range of motion and joint noises assessment during the movements. Subjects also completed a structured questionnaire that enabled the identification of the frequency and intensity of neck pain based on the number of days within a month with pain and on a Numeric Rating Scale (NRS), respectively. Furthermore, if the neck pain was reported, patients were asked to describe their perception of its influence on TMDs by answering the following question: 'Do you think that the neck pain, if present, triggers your

TMD pain or makes the TMD symptoms worse?'. After answering those questions, patients were instructed to complete the Neck Disability Index Questionnaire (29).

A second and blinded evaluator assessed the subjects' thermal pain threshold, for cold (CPT) and heat (HPT) by the Quantitative Sensory Testing (QST) using a Medoc Pathway Neurosensory Analyzer* with a 30 × 30 mm thermode in accordance with an ascending method of limits (30). Thermal QST has demonstrated good to excellent reliability in healthy adults for HPT (ICC for intra-rater = 0·43 and for inter-rater = 0·86) and for CPT (ICC for intra-rater = 0·89 and for inter-rater = 0·94) (31).

The periorbital region, masseter, cervical posterior region and ventral forearm were evaluated bilaterally in a random order and with a constant pressure of the thermode over the skin (Fig. 1). Those sites were chosen to assess the V1, V3, C3-C4 and T1 dermatomes, respectively.

From a baseline of 32 °C, the temperature of the thermode surface increased or decreased at a rate of 1·0 °C/s until participants responded by pressing a mouse button to indicate when they first felt pain. The equipment had built-in controls for the temperature at extremes to avoid tissue injury, and stimulation was terminated once it reached 0 °C or 50 °C, for CPT and HPT, respectively. This test is considered the gold standard for thermal threshold assessment has an excellent reliability (32, 33). The test was repeated three times to obtain the average pain threshold of each site.

All tests were performed in a quiet room, after a familiarisation trial, with no visual access to the operator's screen. Subjects were lying comfortably on their back for all sites assessments except for the cervical posterior site. For this latter site, patients were instructed to sit on a chair and have their head resting on a stretcher with the neck exposed.

Data analysis

Based on a pilot study, we powered our study to detect seven units of difference in the QST with a standard deviation of 10 and the sample required for 80% power. This yielded a sample size of 20 participants per group. Data from both sides were averaged as there was no evidence of difference among left and right side over the groups tested ($P > 0·05$). The two-tailed *t*-test was used to detect the differences between patients with TMDs and controls in the QST test. The Qui-square Test and Fisher Exact Test were used to compare parameters related to the frequency of pain and percentage comparisons between groups. The results are summarised using descriptive analysis with a significant level of 0·05 and confidence interval of 95%.

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Results

There were no differences between the groups regarding age, weight and height (Table 1). Patients with TMD reported neck pain more frequently than controls, and 40% of them identified neck pain as a trigger for TMD pain (Table 1). Patients with TMD also exhibited mild disability according to the NDI questionnaire compared to controls ($P < 0·05$) (Table 1). The range of motion of the temporomandibular joint was smaller in patients with TMD than in controls only for the conditions of maximal opening and mouth opening without pain ($P < 0·05$) (Table 1).

The sample was composed 55% of patients with Mixed TMD, 35% of Myogenic TMD and 10% with Arthrogenic TMD according to the RDC/TMD (Table 2).

In the periorbital region (cephalic area), patients with TMD exhibited pain at lower temperature than controls for heat stimuli (42·37, 95% CI 41·82–44·64 *versus* 47·04, 95% CI 45·1–47·65) ($P < 0·01$) (Table 3). For cold stimuli, they felt pain at higher temperature than controls (18·93, 95% CI 16·92–22·94 *versus* 11·87, 95% CI 10·11–16·15) ($P < 0·01$) (Table 3).

At all the other sites evaluated, patients with TMDs exhibited pain at higher temperature than controls just for cold stimuli. In the masseter site, a cephalic area as well, patients with TMD reported pain at the temperature of 18·93 degrees (95% CI 16·92–22·94) while at controls it was 11·87 (95% CI 10·11–16·15) ($P < 0·01$).

In the ventral forearm region, an extra-cephalic area, patients with TMD exhibited pain at lower temperature than controls for cold stimuli (18·3 (95% CI 13·47–22·29) *versus* 7·66 (95% CI 5·92–14·23), respectively – $P < 0·01$). In the posterior cervical site, patients with TMD exhibited cold pain at 18·93

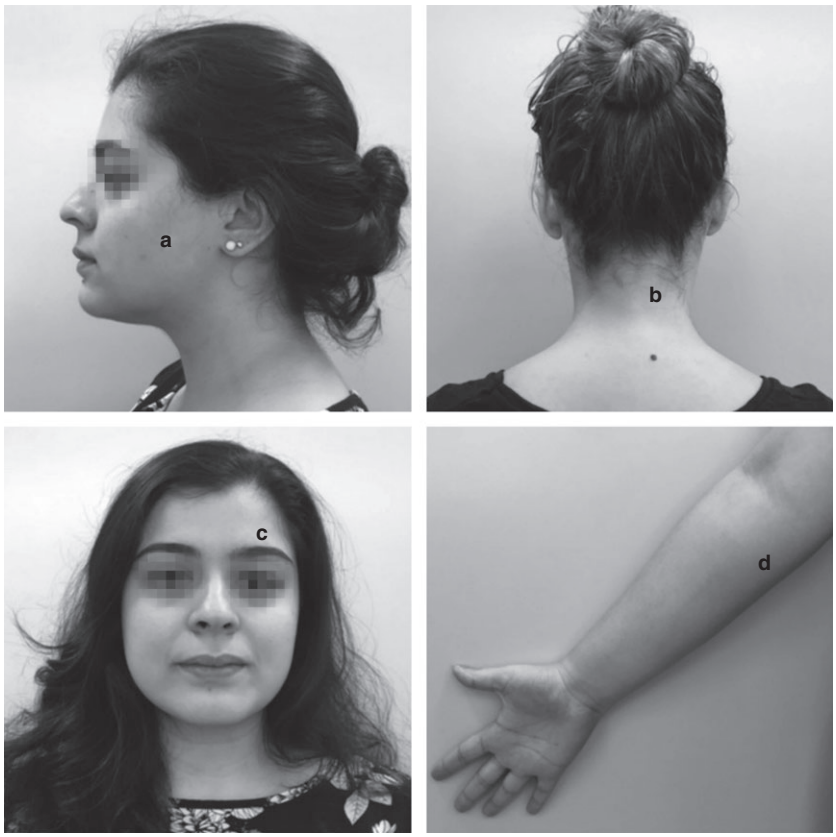

Fig. 1. Thermal pain threshold sites assessed. The thermode was applied bilaterally in a random order of the sites: a. Masseter; b. Posterior Cervical; c. Periorbital; and d. Ventral Forearm.

degrees (95% CI 16·92–22·94) while controls exhibit pain at 11·87 (95% CI 10·11–16·15) ($P < 0·01$). All the data regarding the thermal threshold of both groups are summarised at Table 3.

Discussion

Our results showed changes in the thermal pain threshold in patients with TMDs, especially for cold stimuli, at all sites assessed, including the cervical. Furthermore, patients with TMDs reported the presence of neck pain more often than controls, which led to disability.

Previous studies in this research field have shown mixed findings. Specifically, they observed sensitisation just for heat (14, 15, 20), for both cold and heat stimuli (22, 23), or no abnormal thermal threshold (19, 21). Our results are partially in accordance with Park *et al.* (2010) and Fernandez de las Penas *et al.* (2010), which have found differences compared to controls at cold and heat pain threshold in both artrogenic, mixed (23) and myogenic (22) TMDs. Maixner *et al.* (1995, 1998) just found differences at heat pain threshold on masseter and forearm in patients with myofascial and mixed TMD (14, 15, 20) while Raphael *et al.* (2009) and Ribeiro da Silva (2012) have not shown any differences over face, hand (21) and ventral forearm (19) regions in patients with TMD compared to controls.

Our study was the first to reveal differences in somatosensory thresholds between patients with TMD and controls in the neck region. Our results demonstrated a different pattern of sensitisation – at almost all sites evaluated, just the cold stimuli had a higher threshold.

The differences between the results of cold and heat pain threshold could be related to distinct mechanisms in the processing of painful stimuli. Although heat and cold pain stimuli are both mediated by Aδ fibres (myelinated fibres) and C-fibres (unmyelinated fibres), heat pain stimuli are predominantly mediated by C-fibres, which have slow conduction sensitivity and velocity (34).

We could speculate that the greater sensitivity to cold stimuli in our sample, comparing to previous studies (14, 15, 19–21), could be related to lower

Table 1. Patients' characteristics (Mean and 95% Confidence Interval)

	Control Group (n = 20)	TMD group (n = 20)
Age (years)	26·2 (23·6–28·7)	31·6 (25·5–37·8)
Weight (kg)	59 (54·9–63·1)	58·6 (55·5–61·7)
Height (m)	1·65 (1·69–1·64)	1·63 (1·61–1·66)
Time of TMD (years)	–	3·88 (2·63–5·12)
Mean TMD pain intensity (0–10)	–	4·39 (3·66–5·14)
Report of Neck Pain	15% (n = 3)	75% (n = 15)*
Frequency of Neck Pain (days/month)	0·5 (0·1–1·1)	12·9 (7·4–18·5)*
Neck pain triggers the TMD pain	–	40% (n = 8)*
Neck Disability Index (0–50 points)	1 (0·1–2·2)	7·7 (5·3–10·2)*
Mouth opening without pain (mm)	47 (45·5–48·6)	37·2 (33·5–40·9)*
Maximal active mouth opening (mm)	47 (45·5–48·6)	41·3 (37·6–45)*
Maximal passive mouth opening (mm)	47·4 (45·9–48·9)	44·7 (42·4–46·9)
Right mouth lateral movement (mm)	8·9 (8–9·8)	8·2 (7·2–9·2)
Left mouth lateral movement (mm)	8·6 (7·6–9·6)	8·6 (7·2–9·2)
Protrusion (mm)	7·4 (6·8–8)	8 (7·1–8·8)

*$P < 0{\cdot}05$.

Table 2. TMD diagnosis of the sample based on the Research Diagnostic Criteria (RDC/TMD)

Myofascial pain	35% (n = 7)
Myofascial pain with limited opening + Disc Displacement with Reduction	5% (n = 1)
Myofascial pain with limited opening + Arthralgia	20% (n = 4)
Myofascial pain+ Disc Displacement with Reduction	5% (n = 1)
Myofascial pain+ Disc Displacement without Reduction and limited opening + Arthralgia	5% (n = 1)
Myofascial pain+ Arthralgia	10% (n = 2)
Myofascial pain+Osteoarthritis	10% (n = 2)
Disc Displacement with Reduction	5% (n = 1)
Arthralgia	5% (n = 1)
Myogenic TMD	**35% (n = 7)**
Arthrogenic TMD	**10% (n = 2)**
Mixed TMD	**55% (n = 11)**

Bold values because this is the grouping of the above diagnosis. The conditions cited above are the detailed diagnosis and this is the reference group in the RDC/TMD that they belong.

Table 3. Mean and confidence interval (95% CI) of pooled left and right sides of thermal threshold (cold and warm) in controls and patients with TMD

Threshold (°Degrees)	Control group (n = 20)	TMD group (n = 20)
Periorbital		
Heat	46·38 (45·1 to 47·65)	43·23 (41·82 to 44·64)*
Cold	13·13 (10·11 to 16·15)	19·93 (16·92 to 22·94)*
Masseter		
Heat	45·54 (43·91 to 47·17)	43·34 (41·70 to 44·97)
Cold	9·52 (4·74 to 14·3)	18·6 (14·08 to 23·12)*
Ventral Forearm		
Heat	44·79 (43·36 to 46·22)	42·85 (41·41 to 44·28)
Cold	10·08 (5·92 to 14·23)	17·88 (13·47 to 22·29)*
Posterior Cervical		
Heat	46·04 (44·34 to 47·74)	43·92 (42·12 to 45·72)
Cold	9·54 (5·14 to 13·93)	18·07 (14·14 to 21·99)*

*$P < 0{\cdot}01$.

adaptation to cold due to the subjects' living experience in tropical areas. The opposite could also be true; the absence of heat sensitisation may be related to the predominance of the hot stimuli, due to the weather that these subjects are used to, leading to an adaptation process of the sensorial pathways.

In addition to thermal thresholds, it was also verified by other authors that patients with TMDs present lower pressure pain thresholds (14, 20), pain due to the vibrotactile stimulation (35), mechanical and electrical intra-articular stimuli (35) and alterations on the temporal summation of the pain (20, 21), in cephalic and extra-cephalic areas. Along with the thermal threshold results, it highlights the evidence of a generalised hyperexcitability in the central processing of nociceptive inputs, process that has important clinical implications such as pain chronification (14, 36, 37).

The increased activity of peripheral nociceptors can then lead to the development of central sensitisation and expansion of the receptive fields of second order nociceptive neurons, amplifying the nociceptive

signals and evoking pain even outside the area of clinical pain (38). This process plays a remarkable role in the presence and persistence of other chronic pain conditions such as migraine, tension-type headache (9, 39), fibromyalgia (7, 40) and neck pain (13, 41, 42).

The quantitative thermal threshold assessment has been used in clinical practice for the evaluation of pain disorders without an established neuropathic aspect (43, 44) and has been shown to be more appropriate than detection thresholds for assessing thermal nociceptive mechanisms in chronic pain conditions (45). It has been used in the examination of patients who exhibit symptoms of both peripheral and central origin (46) such as fibromyalgia (43), irritable bowel syndrome (44), neuropathic pain (47), headache (8) and TMDs (16), among others.

In patients with myofascial TMDs, the referred neuropathic pain is elicited from the more frequent trigger points that are found in these patients than in controls (42). However, in patient with arthrogenic TMD, the widespread central sensitisation could be due to the higher level of peripheral afferent nerve afferences caused by the joint inflammatory environment (48), which enhances pain sensitivity and facilitate pain transmission (49). Patients with mixed TMDs may have both subgroups mechanisms involved, although they do not present additional level of pain intensity or thermal threshold differences compared with those with pain originating from only one source (23).

Similar to our results regarding the report and disability due to neck pain in patients with TMDs, Da Costa *et al.*(13) also showed mild disability according to the Neck Disability Index Questionnaire. This was correlated with lower pressure pain thresholds even in facial sites. This relationship between neck and TMDs is explained by the convergence of nociceptive trigeminal and cervical afferent fibres in the trigeminocervical nucleus caudalis (50).

Accordingly, the presence of abnormal pain processing in the neck area as well as the report of disability due to this pain highlights the importance of taking into account the management of the cervical area in the clinical treatment of TMDs.

Our study has some limitations. First, we did not control for the presence of sleep problems, physiological disorders and hormonal phases: factors that are likely to interact with the nociceptive system sensitisation (23, 33). Second, we cannot make inferences regarding the influence of the TMD subtypes or presence of neck pain on the thermal threshold estimations, due to the small sample size of each subtype. Third, generalisability of the results is limited to the same gender and population. However, due to the exclusion of comorbidities and wide thermal threshold characterisation, this study can contribute to a better understanding of pain processing and care for TMD sufferers.

Conclusion

Patients with TMDs have pain modulation changes, especially for cold stimuli, at trigeminal and extra-trigeminal region, including the neck area. Moreover, they exhibited higher disability and a higher report of neck pain than controls. These findings reinforce the evidence regarding the relationship between TMDs and neck pain.

Ethical approval and disclosure

The present study was approved by the Local Ethics Committee (process number: 1950/2010), and the authors declare no conflict of interest.

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References

1. Slade GD, Fillingim RB, Sanders AE, Bair E, Greenspan JD, Ohrbach R *et al.* Signs and symptoms of first-onset TMD and sociodemographic predictors of its development: the OPPERA prospective cohort study. J Pain. 2013;12(Suppl): T20–T32.
2. Wadhwa S, Kapila S. TMJ disorders: future innovations in diagnostics and therapeutics. J Dent Educ. 2008;72:930–947.
3. Greene CS. Managing the care of patients with temporomandibular disorders: a new guideline for care. J Am Dent Assoc. 2010;141:1086–1088.
4. Schiffman E, Ohrbach R, List T, Anderson G, Jensen R, John MT *et al.* Diagnostic criteria for headache attributed to temporomandibular disorders. Cephalalgia. 2012;32:683–692.
5. Durham J, Newton-John TR, Zakrzewska JM. Temporomandibular disorders. BMJ. 2015;350:h1154.
6. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations

and specifications, critique. J Craniomandib Disord. 1992;6:301–355.
7. Pimentel MJ, Gui MS, Martins de Aquino LM, Rizzatti-Barbosa CM. Features of temporomandibular disorders in fibromyalgia syndrome. Cranio. 2013;31:40–45.
8. Bevilaqua-Grossi D, Lipton RB, Napchan U, Grosberg B, Ashina S, Bigal ME. Temporomandibular disorders and cutaneous allodynia are associated in individuals with migraine. Cephalalgia. 2010;30:425–432.
9. Speciali JG, Dach F. Temporomandibular dysfunction and headache disorder. Headache. 2015;55(Suppl 1):72–83.
10. Graff-Radford SB, Bassiur JP. Temporomandibular disorders and headaches. Neurol Clin. 2014;32:525–537.
11. Smith MT, Wickwire EM, Grace EG, Edwards RR, Buenaver LF, Peterson S *et al.* Sleep disorders and their association with laboratory pain sensitivity in temporomandibular joint disorder. Sleep. 2009;32:779–790.
12. Velly AM, Look JO, Schiffman E, Lenton PA, Kang W, Messner RP. The effect of fibromyalgia and widespread pain on the clinically significant temporomandibular muscle and joint pain disorders–a prospective 18-month cohort study. J Pain. 2010;11:1155–1164.
13. da Costa DR, de Lima Ferreira AP, Pereira TA, Porporatti AL, Conti PC, Costa YM *et al.* Neck disability is associated with masticatory myofascial pain and regional muscle sensitivity. Arch Oral Biol. 2015;60:745–752.
14. Vierck CJ, Wong F, King CD, Mauderli AP, Schmidt S, Riley JL III. Characteristics of sensitization associated with chronic pain conditions. Clin J Pain. 2014;30:119–128.
15. Maixner W, Fillingim R, Booker D, Sigurdsson A. Sensitivity of patients with painful temporomandibular disorders to experimentally evoked pain. Pain. 1995;63:341–351.
16. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain. 2011;152(Suppl 3):S2–S15.
17. Latremoliere A, Woolf CJ. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. J Pain. 2009;10:895–926.
18. Furquim BD, Flamengui LM, Conti PC. TMD and chronic pain: a current view. Dental Press J Orthod. 2015;20:127–133.
19. Ribeiro-Dasilva MC, Goodin BR, Fillingim RB. Differences in suprathreshold heat pain responses and self-reported sleep quality between patients with temporomandibular joint disorder and healthy controls. Eur J Pain. 2012;16:983–993.
20. Maixner W, Fillingim R, Sigurdsson A, Kincaid S, Silva S. Sensitivity of patients with painful temporomandibular disorders to experimentally evoked pain: evidence for altered temporal summation of pain. Pain. 1998;76:71–81.
21. Raphael KG, Janal MN, Anathan S, Cook DB, Staud R. Temporal summation of heat pain in temporomandibular disorder patients. J Orofac Pain. 2009;23:54–64.
22. Fernandez-de-las-Penas C, Galan-del-Rio F, Ortega-Santiago R, Jimenez-Garcia R, Arendt-Nielsen L, Svensson P. Bilateral thermal hyperalgesia in trigeminal and extra-trigeminal regions in patients with myofascial temporomandibular disorders. Exp Brain Res. 2010;202:171–179.
23. Park JW, Clark GT, Kim YK, Chung JW. Analysis of thermal pain sensitivity and psychological profiles in different subgroups of TMD patients. Int J Oral Maxillofac Surg. 2010;39:968–974.
24. Silveira A, Armijo-Olivo S, Gadotti IC, Magee D. Masticatory and cervical muscle tenderness and pain sensitivity in a remote area in subjects with a temporomandibular disorder and neck disability. J Oral Facial Pain Headache. 2014;28:138–146.
25. Wright EF, Domenech MA, Fischer JR Jr. Usefulness of posture training for patients with temporomandibular disorders. J Am Dent Assoc. 2000;131:202–210.
26. De Laat A, Meuleman H, Stevens A, Verbeke G. Correlation between cervical spine and temporomandibular disorders. Clin Oral Invest. 1998;2:54–57.
27. Walczynska-Dragon K, Baron S, Nitecka-Buchta A, Tkacz E. Correlation between TMD and cervical spine pain and mobility: is the whole body balance TMJ related? Biomed Res Int. 2014;2014:582414.
28. Armijo Olivo SMD, Parfitt M, Major P, Thie NM. The association between the cervical spine, the stomatognathic system, and craniofacial pain: a critical review. J Orofac Pain. 2006;20:16.
29. Vernon H, Mior S. The Neck Disability Index: a study of reliability and validity. J Manipulative Physiol Ther. 1991;14:409–415.
30. Lowenstein L, Jesse K, Kenton K. Comparison of perception threshold testing and thermal-vibratory testing. Muscle Nerve. 2008;37:514–517.
31. Geber C, Klein T, Azad S, Birklein F, Gierthmühlen J, Huge V *et al.* Test-retest and interobserver reliability of quantitative sensory testing according to the protocol of the German Research Network on Neuropathic Pain (DFNS): a multicentre study. Pain. 2011;152:548–556.
32. Heldestad V, Linder J, Sellersjo L, Nordh E. Reproducibility and influence of test modality order on thermal perception and thermal pain thresholds in quantitative sensory testing. Clin Neurophysiol. 2010;121:1878–1885.
33. Bigal ME, Ashina S, Burstein R, Reed ML, Buse D, Serrano D *et al.* Prevalence and characteristics of allodynia in headache sufferers: a population study. Neurology. 2008;70:1525–1533.
34. Hansson P, Backonja M, Bouhassira D. Usefulness and limitations of quantitative sensory testing: clinical and research application in neuropathic pain states. Pain. 2007;129:256–259.
35. Fillingim RB, Fillingim LA, Hollins M, Sigurdsson A, Maixner W. Generalized vibrotactile allodynia in a patient with temporomandibular disorder. Pain. 1998;78: 75–78.
36. Ayesh EE, Jensen TS, Svensson P. Hypersensitivity to mechanical and intra-articular electrical stimuli in persons with painful temporomandibular joints. J Dent Res. 2007;86:1187–1192.
37. Sarlani E, Greenspan JD. Evidence for generalized hyperalgesia in temporomandibular disorders patients. Pain. 2003;102:221–226.
38. Sessle BJ. Acute and chronic craniofacial pain: brainstem mechanisms of nociceptive transmission and neuroplasticity,

and their clinical correlates. Crit Rev Oral Biol Med. 2000;11:57–91.
39. Zakrzewska JM. Temporomandibular disorders, headaches and chronic pain. J Pain Palliat Care Pharmacother. 2015;29:61–63.; discussion 3.
40. Fraga BP, Santos EB, Farias Neto JP, Macieira JC, Quintans LJ Jr, Onofre AS *et al.* Signs and symptoms of temporomandibular dysfunction in fibromyalgic patients. J Craniofac Surg. 2012;23:615–618.
41. Olivo SA, Fuentes J, Major PW, Warren S, Thie NM, Magee DJ. The association between neck disability and jaw disability. J Oral Rehabil. 2010;37:670–679.
42. Fernandez-de-Las-Penas C, Galan-Del-Rio F, Alonso-Blanco C, Jimenez-Garcia R, Arendt-Nielsen L, Svensson P. Referred pain from muscle trigger points in the masticatory and neck-shoulder musculature in women with temporomandibular disoders. J Pain. 2010;11:1295–1304.
43. Geisser ME, Casey KL, Brucksch CB, Ribbens CM, Appleton BB, Crofford LJ. Perception of noxious and innocuous heat stimulation among healthy women and women with fibromyalgia: association with mood, somatic focus, and catastrophizing. Pain. 2003;102:243–250.
44. Zhou Q, Fillingim RB, Riley JL III, Malarkey WB, Verne GN. Central and peripheral hypersensitivity in the irritable bowel syndrome. Pain. 2010;148:454–461.
45. Woolf CJ, Bennett GJ, Doherty M, Dubner R, Kidd B, Koltzenburg M *et al.* Towards a mechanism-based classification of pain? Pain. 1998;77:227–229.
46. Olney RK. Clinical trials for polyneuropathy: the role of nerve conduction studies, quantitative sensory testing, and autonomic function testing. J Clin Neurophysiol. 1998;15:129–137.
47. Koltzenburg M, Torebjork HE, Wahren LK. Nociceptor modulated central sensitization causes mechanical hyperalgesia in acute chemogenic and chronic neuropathic pain. Brain. 1994;117(Pt 3):579–591.
48. Eliav E, Benoliel R, Tal M. Inflammation with no axonal damage of the rat saphenous nerve trunk induces ectopic discharge and mechanosensitivity in myelinated axons. Neurosci Lett. 2001;311:49–52.
49. Takahashi T, Kondoh T, Fukuda M, Yamazaki Y, Toyosaki T, Suzuki R. Proinflammatory cytokines detectable in synovial fluids from patients with temporomandibular disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1998;85:135–141.
50. de Wijer A, Steenks MH, Bosman F, Helders PJ, Faber J. Symptoms of the stomatognathic system in temporomandibular and cervical spine disorders. J Oral Rehabil. 1996;23:733–741.

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