The Influence of Myofascial Temporomandibular Disorder Pain on the Pressure Pain Threshold of Women During a Migraine Attack

Lívia Maria Sales Pinto Fiamengui, DDS, MS
PhD Student
Department of Prosthodontics
Bauru School of Dentistry
University of São Paulo
São Paulo, Brazil

João José Freitas de Carvalho, MD, MS
Neurologist
General Hospital of Fortaleza
Ceará, Brazil

Carolina Ortigosa Cunha, DDS, MS
PhD Student
Department of Prosthodontics
Bauru School of Dentistry
University of São Paulo
São Paulo, Brazil

Leonardo Rigoldi Bonjardim, DDS, PhD
Professor
Department of Biological Sciences
University of São Paulo
São Paulo, Brazil

Jorge Francisco Fiamengui Filho, DDS, MS
PhD Student
Department of Periodontology
Bauru School of Dentistry
University of São Paulo
São Paulo, Brazil

Paulo César Rodrigues Conti, DDS, PhD
Professor
Department of Prosthodontics
Bauru School of Dentistry
University of São Paulo
São Paulo, Brazil

Correspondence to:
Dr Lívia Maria Sales Pinto Fiamengui
Department of Prosthodontics
Bauru School of Dentistry
University of São Paulo
Al. Octavio Pinheiro Brisola 9-75
CEP: 17012-901
Vila Universitária, Bauru, SP, Brazil
Email: livia_holanda_@hotmail.com

Aims: *To assess the influence of myofascial temporomandibular disorder (TMD) pain on the pressure pain threshold (PPT) of masticatory muscles in women during a migraine attack.* ***Methods:*** *The sample comprised 34 women, 18 to 60 years of age, with a diagnosis of episodic migraine previously confirmed by a neurologist. All subjects were evaluated using the Research Diagnostic Criteria for TMD (RDC/TMD) to determine the presence of myofascial pain. They were divided into two groups: group 1 (n = 18) included women with migraine; group 2 (n = 16) included women with migraine and myofascial TMD pain. Participants were evaluated by measuring PPT values of the masseter and anterior temporalis muscles and Achilles tendon with a pressure algometer at two moments: pain free and during a migraine attack. A three-way analysis of variance with a 5% significance level was used for statistical purposes.* ***Results:*** *Significantly lower PPT values were found during the migraine attack, especially for women with concomitant myofascial pain, regardless of the side of the reported pain.* ***Conclusion:*** *Migraine attack is associated with a significant reduction in PPT values of masticatory muscles, which appears to be influenced by the presence of myofascial TMD pain.* J OROFAC PAIN 2013;27:343–349. doi: 10.11607/jop:1059

Key words: masticatory muscles, migraine, myofascial pain, pressure pain threshold, temporomandibular disorders

Headaches and migraines are common disorders of the nervous system. According to the International Headache Society (IHS), 46% of the adult population globally have an active general headache disorder, with 11% suffering from migraine. The impact of headache on an individual's quality of life is substantial, and according to the World Health Organization's ranking of causes of disability, headaches are one of the 10 most disabling problems for men and women.[1] Based on these numbers, new studies and treatment approaches should be developed to better understand and treat those disorders.

Migraine is a common disabling brain disorder, and its pathophysiology is now better understood as a result of studies of the anatomy and physiology of the pain-related structures of the cranium and their central nervous system (CNS) modulation.[2] The pathophysiology of a migraine involves not only the activation of meningeal perivascular nociceptive fibers, but also an increase in the responsiveness (sensitization) of central nociceptive neurons, which process information from intracranial structures and extracranial skin and muscles.[3,4] Indeed, the nociceptive neurons in the first cervical (C1) spinal segment and medullary dorsal horn show extensive convergence of afferent inputs from cutaneous, musculoskeletal, dural, and visceral tissues.[4]

The phenomena of peripheral and central sensitization have been implicated in migraines and some chronic musculoskeletal entities, such as temporomandibular disorders (TMD).[5] Acute muscle pain is the result of activation of group-III (A-fiber) and group-IV (C-fiber) polymodal muscle nociceptors. The nociceptors can be sensitized by the release of neurochemicals from the nerve endings as well as from other tissue cells. This eventually may lead to central sensitization of dorsal horn neurons, manifested as prolonged neuronal discharges, increased responses to a defined noxious stimuli, response to non-noxious stimuli, and expansion of the neuronal receptive field.[6]

TMD is defined by the American Academy of Orofacial Pain as a collective term that comprises a number of clinical problems that involve the masticatory muscles, the temporomandibular joint (TMJ), and associated structures.[7] Population studies have reported the prevalence of TMD to be from 8% to 15% for women and 3% to 10% for men.[8]

The association between migraine and TMD has been demonstrated in many studies,[9–11] suggesting that both disorders often share similar signs, symptoms, and pain mechanisms. Previous studies have shown that TMD is a risk factor for increased headache frequency and the development of chronic migraine.[10,11] Muscle tenderness is one of the most distinguishing signs of TMD of muscular origin (myofascial pain), and it is usually an expression of a complex array of local and CNS changes. Likewise, people with migraines frequently report increased soreness in the temple area.[12]

The impact of myofascial pain on migraine pain and extracranial tenderness during a migraine attack can be assessed by using a pressure algometer to measure the patient's pressure pain threshold (PPT) of the masticatory muscles. The algometer has been considered an important tool for elucidating peripheral and central nociceptive mechanisms, which are most likely involved in the pathophysiology of both conditions,[13] as noted above. To the best of the authors' knowledge, no previous studies investigating the influence of TMD (myofascial pain) on the PPT of masticatory muscles during a migraine attack have been published. Therefore, the aim of this study was to assess the influence of myofascial TMD pain on the PPT of masticatory muscles in women during a migraine attack. The null hypothesis tested was that myofascial TMD pain does not influence the PPT levels of masticatory muscles in migraine patients during a migraine attack.

Materials and Methods

Subjects

Women aged between 18 and 60 years who had been diagnosed with episodic migraines were included in this study. An experienced neurologist interviewed all patients, and to be included in the study, women had to be classified as having episodic migraines according to the IHS criteria. Subjects with only menstrual-related migraine, chronic migraine, other primary headaches, secondary headaches, or systemic conditions (eg, fibromyalgia) were excluded from this study.

Initially, 250 women were evaluated and 101 met the inclusion criteria. A single experienced specialist examined all subjects according to RDC/TMD specifications. The women were divided into two groups: group 1 (n = 56) included those with migraine, and group 2 (n = 45) included those with migraine and myofascial TMD pain. The PPT measurement during a migraine attack was completed in 18 and 16 women from groups 1 and 2, respectively (Fig 1). The local Human Research Committee approved the research project. All subjects signed an informed consent form before entering the study.

PPT Assessment

The PPT recording procedure was the same for the entire sample. A digital algometer (KRATOS, Cotia, Brazil) with a 1-cm^2 flat circular-shaped tip at one end was used to apply pressure over a masticatory muscle and measure the PPT at that muscle site. The pressure application rate was previously calibrated and set at approximately 0.5 $kgf/cm^2/s$. The masseter belly and anterior belly of the temporalis muscle were tested bilaterally in a relaxed position. To record the masseter PPT, patients were asked to clench their teeth together and then relax, which allowed for the proper identification of the site to be examined. To determine the anterior temporalis PPT, pressure was applied 30 mm posterior to the lateral end of the orbit and 15 mm above the upper end of the zygomatic arch.

The procedure was fully explained to each patient before the examination. It was emphasized that the purpose of the study was to measure the PPT, not pain tolerance.[14] The PPT was reached when the subject felt the pressure begin to turn into pain. Throughout the test, the individual's head was firmly supported by the operator's hand, and each site was tested twice in a previously defined randomized sequence. The device used in the present study had a button that the patient was asked to press at the very

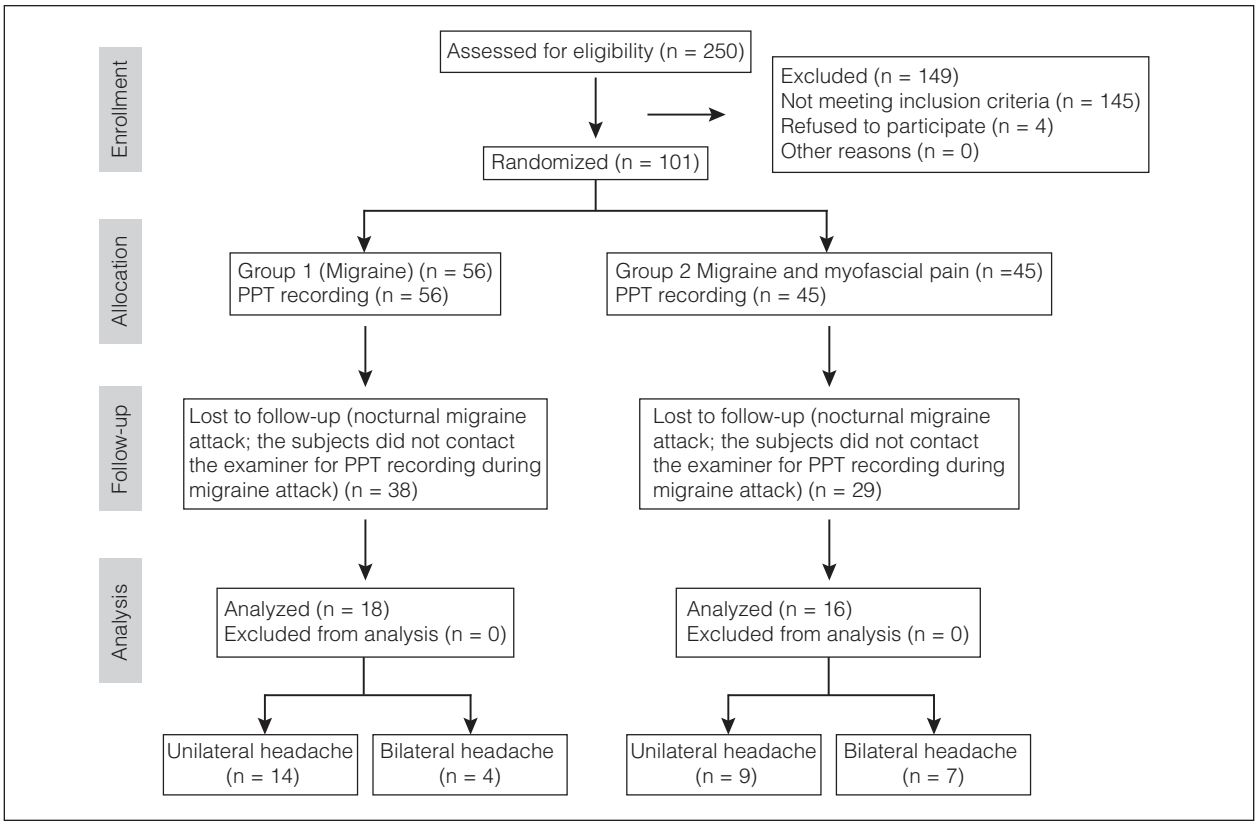

Fig 1 Flow diagram of the subjects' progress throughout the phases of the study. PPT, pressure pain threshold.

beginning of a pain sensation. Therefore, the subject had full control in determining the moment when the applied pressure became painful, with no interference of the examiner. An additional measurement was performed over the Achilles tendon, which was selected as the non-trigeminal control site.[15]

Test Procedures

The PPT was measured at two time points, always by the same calibrated examiner, and each measurement lasted 5 to 10 minutes. The first measurement (baseline) was taken when the patient was in a phase without headache complaints or in a pain-free period. To avoid possible residual migraine-related allodynia or other sensitization phenomena, which could interfere with the results, this initial evaluation was performed at least 1 week after the last migraine attack. Patients were also asked to avoid taking any analgesics or muscle relaxants 24 hours prior to the examination.

Following this initial visit, the subjects were instructed to contact the examiner at the very beginning of a moderate to severe migraine attack (pain intensity of at least 6 on a 0- to 10-cm visual analog scale [VAS], where 0 meant "no pain" and 10 "the worst pain") prior to taking any anti-migraine or analgesic medications. The examiner met subjects where they were (eg, home or work) to perform the second PPT recording. During the migraine attack, the subjects were also questioned regarding the site of the pain (unilateral [right or left] or bilateral).

Statistical Analysis

The results were expressed as the means and standard deviation. For each site tested, a three-way analysis of variance (ANOVA) was used to detect differences in PPT between groups, sides, and phases (pain free and during the migraine attack). For the Achilles tendon, a two-way ANOVA was used to detect differences between groups and phases. A 5% level of significance was considered.

Table 1 Significance *(P)* of Group, Side, and Phase and Their Interactions on the Pressure Pain Threshold Values of the Masseter Muscle, Anterior Temporalis Muscle, and Achilles Tendon

	Masseter	Anterior temporalis	Achilles tendon
Group (1/2)	.054	.046*	.135
Side (painful/nonpainful)	.285	.137	–
Phase (pain-free/migraine)	.000*	.000*	.000*
Group/Side	.480	.302	–
Group/Phase	.125	.114	.656
Side/Phase	.318	.793	–
Group/Side/Phase	.325	.418	–

*Statistically significant.

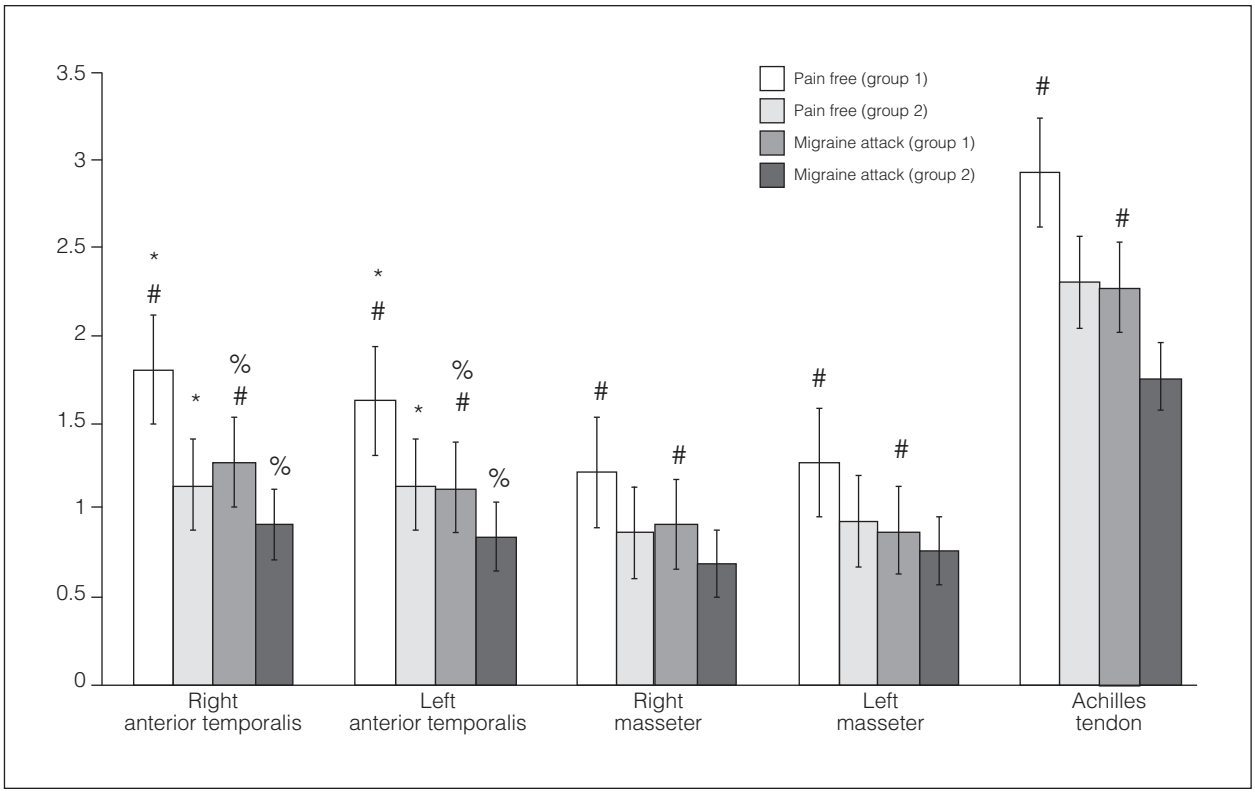

Fig 2 Pressure pain threshold (PPT) values (kgf/cm²) and standard deviations for both groups (group 1, migraine; group 2, migraine and myofascial pain) and phases (pain free and during the migraine attack). #Significant difference in PPT between phases for all sites tested ($P < .000$). *%Significant difference between groups for anterior temporalis on both phases ($P < .046$).

Results

No significant differences in age were found between groups (mean age of 37.2 years for group 1 and 38.7 years for group 2). Significantly decreased PPT values were found for both groups in the migraine-attack measurement for both masticatory muscles tested ($P < .05$). The presence of concomitant myofascial pain also influenced PPT levels of the anterior temporalis ($P < .05$) and masseter ($P = .054$). No significant differences between sides were detected (Table 1). Regarding the Achilles tendon, decreased PPT values were observed during the migraine attack ($P < .001$), but no differences between groups or interaction between variables were detected ($P > .05$), as shown in Table 1. Figure 2 shows all PPT values in both phases (pain free and during the migraine attack).

Table 2 Mean (SD) Pressure Pain Threshold Values (kgf/cm²) of Masticatory Muscles in Relation to Side of Reported Pain (Women with Unilateral Pain Only)

	Painful side		Nonpainful side	
	Anterior temporalis	Masseter	Anterior temporalis	Masseter
Group 1 (n = 14)	1.04 (0.63)	0.82 (0.45)	1.1 (0.57)	0.9 (0.46)
Group 2 (n = 9)	0.7 (0.38)	0.65 (0.33)	0.9 (0.42)	0.76 (0.43)

Side of Reported Pain and PPT Assessments

In group 1, 14 women (78%) reported unilateral headache (7 on each side), while 4 (22%) had bilateral symptoms. In group 2, 9 women (56%) had unilateral headache (4 on the right side, and 5 on the left side), and 7 (44%) reported bilateral pain. No significant decrease in the PPT values of masticatory muscles on the painful side was observed for those with unilateral pain during the migraine attack (Table 2).

Discussion

This study rejected the null hypothesis that myofascial TMD pain did not affect the PPT levels of masticatory muscles in migraine patients during a migraine attack. However, no significant difference in PPT values between the painful side and nonpainful side was detected during the migraine attack. Pericranial muscle tenderness has been found in individuals with migraine during both attack and attack-free periods,[16–18] and nociceptive inputs of myofascial origin have been postulated to play an important role in migraine pathogenesis.[19] Because migraines and myofascial TMD pain appear to encompass peripheral and central components,[13] the present study estimated these phenomena by using the masticatory PPT.[20] Most likely, the large amount of chemical mediators released during a migraine attack, which cause neuronal activation, could have a strong impact on how a patient is able to feel stimuli applied to the trigeminal area, such as the PPT. An interesting finding was that, despite the presence of myofascial TMD pain, individuals with migraines showed a significant decrease in the PPT values of masticatory muscles and the Achilles tendon during a migraine attack compared to baseline data. The significant reduction of PPT at both trigeminal and extra-trigeminal sites suggests a generalized dysfunction of the nociceptive system during migraine attacks.

The primary pathophysiologic events that account for migraines have been reported to be intracranial extra-cerebral (mostly dural) vasodilation and perivascular neurogenic inflammation resulting from the release of vasoactive peptides, such as calcitonin gene-related peptide, due to neuronal activation of the peripheral trigeminal system.[21] These changes most likely cause a peripheral sensitization of the first-order trigeminal neurons, which could explain the reduction in PPT values of the masticatory muscles regardless of whether individuals with migraines presented with myofascial pain. According to Burstein et al,[22] central sensitization of trigeminal neurons in the CNS that receive afferent inputs from facial skin, muscle, and intracranial structures could provide the neuronal substrate to explain the extracranial tenderness and cutaneous allodynia that often accompany a migraine. In the vascular-supraspinal-myogenic model proposed by Olesen (1991),[23] headache intensity is determined by the sum of nociceptive inputs from cephalic arteries and pericranial myofascial tissues converging upon the same neurons and supraspinal effects.

In the present study, the concomitant presence of myofascial pain did play a crucial role in the reduction of PPT values in individuals with migraines at baseline and during a migraine attack. Sensitization of muscle nociceptors is associated with decreased mechanical activation threshold and increased responsiveness to noxious stimuli,[24,25] which could result in more pain-related signals entering the CNS and perhaps producing central sensitization in individuals with migraine. Therefore, the simultaneous treatment of myofascial pain could benefit individuals with migraines. Some studies have suggested that TMD treatment with oral appliances and self-management therapies could be beneficial for many severe headache patients, including individuals with migraines.[26,27] This statement, however, was not tested in the present investigation and should be interpreted cautiously.

Importantly, women in group 1 (with migraine only) could conceivably have had latent trigger points in masticatory muscles that were able to influence their PPT values during migraine attacks. In 2007, García-Leiva et al[19] showed that all individuals with migraines presented at least one trigger point, most of them located on the temples, suggesting that peripheral sensitization is an important component of migraine predisposition. Furthermore, it is assumed that signals to supraspinal structures could be misinterpreted as pain in the musculature or intracranial structures distant from the site of the painful stimulus or a trigger point in a given muscle.[28] Moreover, central sensitization of the second-order neurons in the brainstem, particularly in the medullary dorsal horn, and in the cervical dorsal horn, which receive convergent afferent inputs from intracranial structures and masticatory muscles,[4] could account for masticatory muscle tenderness[29] and, consequently, the lower PPT values found in the present study. The large amount of neurotransmitters released at the second-order nociceptive neuron level and the decreased neuronal activation threshold are common features of this central sensitization process.

The PPT reduction in an extra-trigeminal site (Achilles tendon) could be interpreted as an indication of central sensitization of the third-order or higher-order trigeminal neurons in the thalamus or cerebral cortex.[22,29] Notably, as mentioned previously, all the mechanisms discussed here could be amplified by impairment of descending supraspinal pain modulatory influences.[30] This phenomenon is frequently described as contributing to the maintenance of chronic pain states, such as migraine and myofascial pain.

The transformation of a headache into whole-body allodynia and hyperalgesia is believed to occur 2 hours after the onset of the migraine attack[22] and to be mediated by the sensitization of third-order thalamic neurons that process mechanical and thermal sensory information converging from the meninges, head, body, and limbs.[31] The neural mechanisms by which trigeminovascular thalamic neurons become sensitized may involve sequential sensitization of first-order or second-order trigeminovascular neurons or, alternatively, indirect activation through pain-modulatory neurons in the brainstem.[32] This phenomenon could explain the lower levels of PPT in the Achilles tendon in both groups.

In contrast to previous studies that reported increased pericranial tenderness to be more common on the painful side,[33,34] no differences were found in the PPT values between painful sides and nonpainful sides in this investigation. These discrepancies could be due to methodological differences, such as digital palpation[33] and/or algometry palpation[34]; sites evaluated, such as cephalic and/or cervical muscles[33,34]; and the presence of trigger points.[33] The small number of subjects with unilateral migraine also may have influenced the results. The type of algometer used and the form of PPT recording could also have been factors. In the present study, a digital algometer that was controlled by the patient was used. This method has two advantages: patients feel more comfortable controlling the situation, and an examiner's bias in the PPT recording is eliminated. Furthermore, there is no consensus regarding which muscles must be evaluated. In the present study, the masseter and anterior temporalis muscles were selected for PPT recordings because they are frequently affected in TMD patients. Also, these muscles are relatively flat and overlie bone, which facilitates the PPT recording.[35,36]

This study has some limitations. First, only females were included, and the probable gender influence on the results could not be tested. The possible effect of sex hormones and the phases of the menstrual cycles were not assessed. A previous study found increased nociceptive sensitivity during the luteal or follicular phase,[37] while others have not.[38,39] In the present study, to avoid menstrual-related migraines, no PPT recording was performed during the menses. Another limitation is related to the study sample, which was reduced due to the difficulties inherent in evaluating patients during a migraine attack. Future studies with more representative samples are required to confirm the findings reported here that a migraine attack is associated with a significant reduction of PPT values of masticatory muscles, which seems to be influenced by the coexistence of masticatory myofascial TMD pain.

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The authors reported no conflicts of interest related to this study.

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