The Influence of Time, Facial Side, and Location on Pain-Pressure Thresholds in Chronic Myogenous Temporomandibular Disorder

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This study examined masseter and temporalis pain-pressure thresholds in 29 patients with chronic bilateral myogenous temporomandibular disorder and in 11 controls. Patients with evidence of temporomandibular joint pathosis were omitted. The influence of time, facial side, muscle site, and side of greatest spontaneous pain on pain-pressure thresholds was measured. No significant pain-pressure threshold differences were found between the more and less painful sides, as indicated by the patients, which lends support to theories of centrally mediated pain. Mean pain-pressure thresholds in patients differed over the four sessions, which is consistent with recent reports of fluctuating levels of pain in patients with temporomandibular disorders. Additional findings included significant pain-pressure threshold differences among muscle sites in patients and controls, and lower patient pain-pressure thresholds relative to controls. Within- and between-session reliability was adequate for patients (r = .85 and r = .75, respectively) and controls (r = .90 and r = .75, respectively).
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Manual palpation is the most widely employed clinical method to assess muscle pain. The many variations in technique,[1] however, share common problems.[2] They are subjective and difficult to quantify or standardize. For example, the degree of finger pressure during palpation undoubtedly varies within and between investigators despite efforts to exert a specified amount of pressure.[2] Evidence of these problems was highlighted in a recent study evaluating the inter-rater reliability of manual detection of "trigger points" in the back.[3] Kappa values ranged from .29 to .38, which prompted these authors to conclude that reliable manual detection of trigger points among different therapists could not be achieved with acceptable accuracy and that this clinical practice should be re-evaluated. In addition, Dworkin et al[4] examined the reliability of clinical measurements in temporomandibular disorders (TMD) and concluded that manual palpation of extraoral and intraoral muscles of mastication could be improved with extensive examiner training, but only to "marginal, not high levels of reliability."

In contrast, the reliability and validity of pain-pressure thresholds (PPTs) have been reported in patients with a variety of musculoskeletal pain syndromes as well as in asymptomatic subjects.[1] Pain-pressure thresholds are obtained with the aid of a pressure algometer and are defined as the amount of applied pressure necessary for a subject to report pain.

Pressure algometry has been employed in a variety of anatomic areas in patients[2,5-8] and in asymptomatic subjects.[9-12] Jensen et al[13] recently examined PPTs in bilateral anterior temporalis muscles in a general population of 740 adults and reported that PPTs were higher in men and were elevated with increasing age. In another investigation, the same authors found no significant differences in pain-pressure thresholds of anterior temporalis among subjects with migraine, episodic, and chronic tension-type headache, thus challenging the theoretical validity of generalized increased pain sensitivity in tension-type headache sufferers.[14] In contrast, Bovim[8] examined 22 pericranial and cervical sites and found that PPTs differed significantly among migraineurs, tension-type, and cervicogenic headache sufferers, although the differences were attributed to disproportionately low scores in subjects with cervicogenic headache.

Two controlled PPT investigations examined multiple muscles of mastication in patients and controls.[2,7] In a comparison of 45 patients with "myofascial pain" and 45 age- and sex-matched asymptomatic controls, Schiffman et al[7] concluded that pressure algometry was reliable and valid. These conclusions were repeated by Ohrbach and Gale[2] in a series of studies designed to test the validity and reliability of PPTs. Investigations have examined masticatory sites in conjunction with other body areas,[5,6] and some have studied PPTs in masticatory sites alone.[2,15] All of these studies, whether in patients or asymptomatic controls, support the reliability and validity of pressure algometry.

Most PPT studies of masticatory muscles in patients with temporomandibular disorders (TMD) are based on data obtained from algometers that lacked the capacity to regulate the rate of application, a factor known to significantly alter PPTs.[15,16] Additionally, there are little available data comparing PPTs in well-defined TMD patient groups with asymptomatic controls.

This study compared PPTs in the masseter and anterior temporalis muscles in patients with well-delineated myogenous TMD and asymptomatic controls. Patients with temporomandibular joint (TMJ) pain were specifically excluded. The lateral and medial pterygoid muscles as well as the digastric muscles were excluded from study due to poor accessibility to palpation. Using an electric pressure algometer, test-retest reliability in the temporalis and masseter muscles was evaluated within and between sessions. Further, the relationship of PPTs to the side and site of greatest pain as indicated by patient report was assessed. Finally, the influence of muscle site and facial side on the PPT was examined.

Materials and Methods

A group of 29 patients (28 women, 1 man; average age 28.5 ± 5 years) and 11 controls (11 women; average age 39.0 ± 12 years) were studied. Patients were diagnosed with bilateral, chronic (≥ 3 months) myogenous TMD on the basis of history and physical exam.[17] Bilaterality was defined as a minimum of one manually palpable site at the temporalis and masseter muscles on both sides of the face in addition to a subjective verbal report of bilateral jaw muscle pain. The presence of subclinical temporomandibular joint "noise" was not an exclusion criterion, although every attempt was made to omit patients with intra-articular (TMJ) pathosis such as disc displacements and any of the arthritides. Subjects with focal preauricular pain and clinical evidence of TMJ pathosis underwent TMJ imaging (magnetic resonance, arthrographic, and/or tomographic) and were excluded if intra-articular pathosis was detected. Clinical determination of normal and pathologic TMJs in both patients and controls was based on the clinical criteria of Anderson et al[18] (Table 1). Supporting evidence of intra-articular TMJ pathosis was based on previously published criteria for MRI,[19] arthrography,[20] and tomography.[21] Control subjects had no history of a TMD or orofacial pain syndrome and were free of masticatory muscle or TMJ pain on examination. Subjects taking potentially confounding medications or having immediate dental needs were excluded. A history of trigeminal neuralgia, fibromyalgia, connective tissue disease, or systemic disease known to cause generalized musculoskeletal pain resulted in exclusion from participation. Subjects being treated for a TMD were required to discontinue all treatment for a minimum of 2 weeks.

Each subject participated in two sessions separated by 1 week. The location of tender muscle sites was detected manually and recorded on a transparent template that was used to re-locate these areas for each session. Patients were asked to identify their "most painful side" before obtaining PPTs. Pain-pressure thresholds were measured at four bilateral sites in the following order: anterior temporalis (T); posterior deep masseter (Md); anterior masseter (Ma); and inferior masseter (Mi). Each PPT was defined by the mean of three trials in which the pressure of a 0.5 cm^2 probe was increased at a rate of 50 kPa/s using a Somidec

Table 1 Temporomandibular Joint Diagnostic Criteria Adapted From Anderson et al[18]

Diagnosis	History	Exam	Tomography
Normal	No history of locking	Vertical range of motion ≥40 mm Lateral movements ≥7 mm	No osseous changes
Disc displacement with reduction	Positive history of joint noise Recriprocal click	Vertical range of motion ≥40 mm Lateral movements ≥7 mm	No osseous changes
Disc displacement without reduction			
Acute	History of locking Reduced range of motion Reduced translation	Vertical range of motion ≤35 mm Lateral movements ≤7 mm	Maximal condylar movement is posterior to crest of articular eminence on tomogram
Chronic	History of locking	Vertical range of motion ≥35 mm Lateral movements ≥7 mm	
Osteoarthritis/ Osteoarthrosis	Clinically evident crepitus	–	Tomographic evidence of osseous involvement – reduced joint space – sclerosis – osteophyte formation – subchondral cysts

pressure algometer (Farsta, Sweden). Measures were repeated after 30 minutes to assess intrasession repeat reliability. Data were analyzed by ANOVA to assess overall effect of time, location, and side on PPTs for both patients and controls. Finally, test, retest, and side-to-side reliability were evaluated by means of correlation coefficients.

Results

Figure 1 shows mean PPTs in patients and controls. Mean PPTs of patients, but not controls, differed significantly over the four sessions ($F[3,81] = 3.12$, $P < .05$). The mean PPTs differed significantly between the groups as well ($F[1,36] = 6.08$, $P < .02$). Figure 2 shows comparisons of PPTs obtained from patients' right and left facial sides at each tested site. Pain pressure thresholds differed significantly between all locations ($F[3,81] = 14.72$, $P < .0001$] but did not differ between left and right sides. Comparison of PPT values obtained from the patient-designated more and less painful sides revealed no significant differences ($F[1,27] = 0.44$, $P = .51$) (Fig 2). Pain pressure thresholds in controls differed significantly among the various sites examined ($F[3,27] = 9.63$, $P < .0002$) but, as in patients, did not differ between left and right sides (Figs 3 and 4). A significant location/side interaction ($F[3,27] = 5.91$, $P < .005$) was observed in controls, which suggested that the relative sensitivity at the four sites was not the same for each side.

The consistency of PPTs over time and from side to side is illustrated by the correlations shown in Fig 5. Each panel shows repeat reliability between sessions (1 week) and within sessions (30 minutes) as well as the reliability between left and right sides for all four sessions. Within-session reliability was similar in both patients ($r = .85$) and controls ($r = .90$). Between-session reliability was identical in patients and controls ($r = .75$). Conversely, side-to-side PPT reliability in patients ($r = .68$) was lower than in controls ($r = .86$), although this difference was not significant.

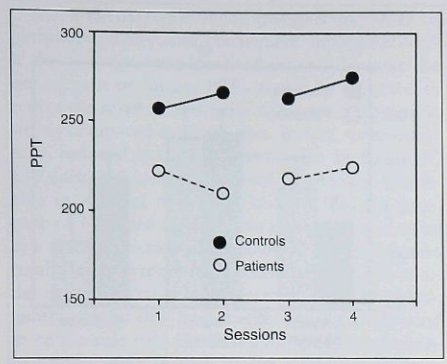

Fig 1 Mean PPTs of all sites combined over the four study sessions for control subjects (filled circles) and patients (open circles). Sessions 2 and 3 were separated by 1 week; sessions 1 and 2 as well as 3 and 4 were separated by 30 minutes. Mean PPTs of patients, but not controls, differed significantly over the four sessions ($F[3,81] = 3.12$, $P < .05$). PPTs differed significantly between controls and patients ($F[1,36] = 6.08$, $P < .02$).

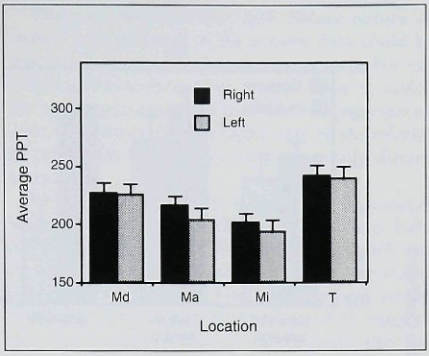

Fig 2 PPTs in patients obtained from right and left sides for the four muscle sites evaluated: Md (deep masseter); Ma (anterior masseter); Mi (inferior masseter); and T (anterior temporalis). PPTs differed significantly over location ($F[3,81] = 14.72$, $P < .0001$), but did not differ between right and left sides.

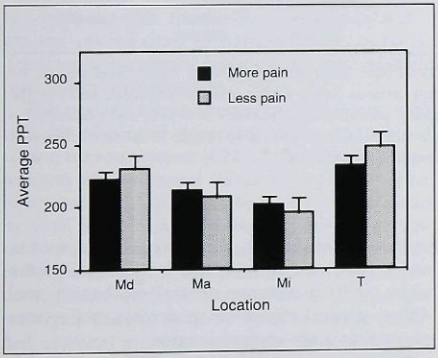

Fig 3 PPTs in patients organized by patient report of most (black) and least (shaded) painful sides. PPTs did not differ between most and least painful sides.

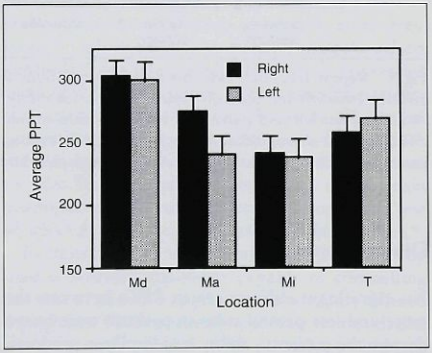

Fig 4 PPTs in controls. PPTs obtained from right (black) and left (shaded) sides for the four muscle sites evaluated. PPTs differed significantly over location ($F[3,27] = 9.63$, $P < .0002$). PPTs did not differ between left and right sides, although the relative sensitivity at the four sites differed between the two sides (location × side interaction) ($F[3,27] = 5.91$, $P < .005$.)

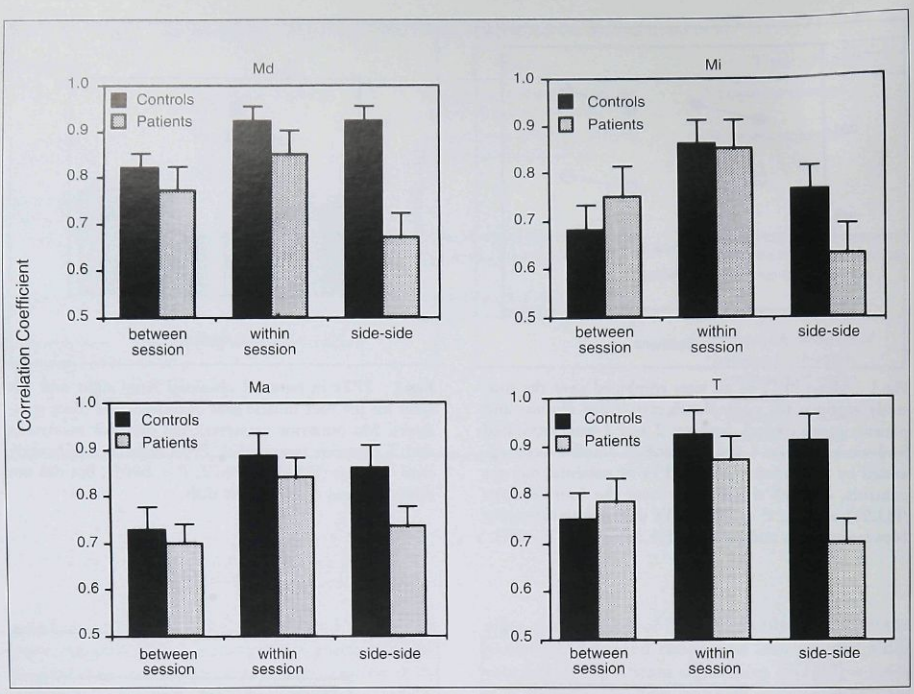

Fig 5 Repeat reliability and side-to-side correlations for PPTs at four different muscle sites. Each panel shows, for patients (shaded) and controls (black), repeat reliability between sessions (left), within sessions (middle), and correlations between left and right sides (right). Within-session reliability was similar in patients (r = .85) and controls (r = .90). Between-session reliability was identical in both groups (r = .75). Conversely, side-to-side reliability differed in controls (r = .86) and patients (r = .68) although this difference was not significant.

Discussion

No significant differences in PPTs between the more and less painful sides in patients were found despite the patients' ability to identify a predominantly painful side prior to commencement of the study. This is similar to findings of other authors who found similar occlusal force levels[22] and electromyographic activity[23] on the left and right facial sides in patients with unilateral facial pain. Relatively poor correlation of PPTs was observed between right and left sides in patients as compared to controls. Thus, there was considerable variability of tenderness between sides that was unrelated to the side patients identified as being more painful. Together, these findings suggest that evoked tenderness, which in patients is less symmetric than in controls, is independent of patient-reported spontaneous pain. Evoked and spontaneous pain may be mediated by separate mechanisms or by a common central mechanism with diffuse general effects on tenderness and spontaneous pain in the orofacial region.

In the present study, patient PPTs were highest at the anterior temporalis, and control PPTs were highest at the deep masseter. The former finding is in contrast to another investigation in which temporalis PPTs were the highest in healthy subjects.[2] It is unclear why certain areas of skeletal muscle would show differential sensitivity to mechanical stimulation, although there is experimental evidence confirming the electromyographic[24] and metabolic[25] heterogeneity of masseter and temporalis muscles. These factors may be operative in the pathophysiology of chronic masticatory muscle pain.

Some theories attribute myogenous TMD to peripheral injury and consequent inflammation.[26] If the etiology were localized inflammation at the site of pain or injury, PPTs would be expected to reflect the relative sensitivity of a particular muscle site as compared to other sites. In fact, experimentally induced muscle inflammation in humans[27] and animals[28] causes decreased mechanical thresholds in affected muscles. Theoretically, the presence of localized inflammatory infiltrates released as a result of muscle damage could produce similar results in nonexperimental conditions. If this were the case, a facial side designated as the predominantly painful one by patients would be expected to be the side that contained the greatest numbers of painful muscle sites or the site(s) of greatest pain intensity. The summed PPTs on this side would then be expected to be lower than for the contralateral side (ie, pain would be greater). However, this is in contrast to findings in the present study. Although PPTs did vary significantly among the four masticatory sites tested (which would tend to support an inflammatory etiology), similar findings were observed in the control subjects, as well as in those of another report.[2] There is currently little direct evidence indicating that inflammation is causal in myogenous TMD, which is consistent with the finding of PPT variability among tested sites in patients and controls. Together, these results implicate alternate etiologic mechanisms in myogenous TMD.

The present observations are consistent with theories of central hyperexcitability and altered central nervous system (CNS) processing as consequences of peripheral tissue injury.[29] An acute injury within myofascial tissues may be followed by CNS hyperexcitability that results in generalized spontaneous and evoked pain commonly observed in myofascial pain and related conditions. Recent evidence has confirmed that peripheral tissue damage or nerve injury results in amplified neuronal activity both at the injured site and within the spinal cord.[30] If similar pathophysiology is operative in myogenous TMD, the overall clinical picture may become one in which innocuous stimuli, such as those during normal mastication, become painful and sensitivity to painful stimulation is increased. Localization of the primary source of pain may be clinically difficult. The present observations of lower patient PPTs, poor side-to-side correlation, and absence of side differences in PPTs may be similar to the findings in animal studies of CNS hyperexcitability and increased spread of pain after peripheral injury.

The poor localizability and diffuse nature of muscle pain reflected in the present data could be due to a number of other factors as well. For example, afferent drive from muscle tissue to multiple spinal segments may contribute to widespread pain.[31] Another potential factor may be the referral of pain from deep tissues to anatomically distant areas.[32,33]

Finally, a recent report highlighted the cyclical fluctuation in levels of TMD pain that may have influenced the present observation of significant patient variations in mean pressure threshold between study sessions. However, the high repeat reliability between experimental sessions suggests that this fluctuation did not substantially alter the present results.

Compelling evidence regarding centrally mediated pain after tissue injury, as well as recent data regarding autonomic involvement in TMDs,[35] suggests systemic involvement in either the onset or perpetuation of myogenous TMD. Thus, pain as a result of peripheral muscle injury and inflammation is likely mediated at the site of injury and within the CNS. Accordingly, effective therapies may require both peripheral interventions and therapeutic attention to CNS changes. The need for central intervention is supported by the demonstrated therapeutic efficacy of tricyclic antidepressants,[36,37] which are known to have analgesic effects centrally[38] but not peripherally.[39] In a controlled trial of a heterogeneous population of patients with chronic orofacial pain, Sharav et al[39] showed an analgesic effect of amitriptyline. Others have recommended the use of tricyclic antidepressants to treat fibromyalgia,[40] a generalized muscle pain syndrome with possible systemic etiology[41] and one of which myogenous TMD pain may be a subset.[42]

In contrast to previous reports, the present study used a pressure algometer capable of controlling the rate of pressure application in a rigidly defined myogenous TMD patient population that excluded patients with painful TMJ pathology. Indeed, every effort was made to exclude patients with any evidence of TMJ disc displacements or of the arthritides, as well as those with less common orofacial pain syndromes such as atypical odontalgia or burning mouth syndrome. Consistent with earlier reports,[1,6,7] it was found that PPTs were highly reproducible within and between sessions, and significantly lower in patients as compared to controls. Lower patient PPTs relative to controls were also observed, which lends support to the conclusions of others that pressure algometry is a valid measurement of evoked muscle pain.[15]

References

1. Jensen K. Quantification of tenderness by palpation and use of pressure algometers. In: Fricton JR, Awad E (eds). Advances in Pain Research and Therapy, vol 17. New York: Raven Press, 1990:165–181.
2. Ohrbach R, Gale EN. Pressure pain thresholds, clinical assessment, and differential diagnosis: Reliability and validity in patients with myogenic facial pain. Pain 1989;39:157–169.
3. Nice D, Riddle D, Lamb R, Mayhew T, Rucker K. Intertester reliability of judgments of the presence of trigger points in patients with low back pain. Arch Phys Med Rehabil 1992;73:893–898.
4. Dworkin SF, LeResche L, DeRouen T. Reliability of clinical measurement in temporomandibular disorders. Clin J Pain 1988;4:89–99.
5. Tunks E, Crook J, Geoffrey N, Kalaher S. Tender points in fibromyalgia. Pain 1988;34:11–19.
6. Reeves J, Jaeger B, Graff-Radford S. Reliability of the pressure algometer as a measure. Pain 1986;24:313–321.
7. Schiffman E, Fricton JR, Haley D, Tylka D. A pressure algometer for myofascial pain syndrome: reliability and validity testing. In: Dubner R, Gebhart GF, Bond MR(eds). Proceedings of the Vth World Congress on Pain. New York: Elsevier, 1988:407–413.
8. Bovim G. Cervicogenic headache, migraine, and tension type headache. Pressure-threshold measurements. Pain 1992;52:169–173.
9. Ohrbach R, Gale E. Pressure pain thresholds in normal muscles: Reliability, measurement effects, and topographic differences. Pain 1989;37:257–263.
10. Davenport J. Pressure-pain thresholds in the oral cavity in man. Arch Oral Biol 1969;14:1267–1274.
11. Petersen K, Brennum J, Olesen J. Evaluation of pericranial myofascial nociception by pressure algometry. Reproducibility and factors of variation. Cephalgia 1992; 12:33–37.
12. Brennum J, Kjeldsen M, Jensen K, Jensen TS. Measurement of human pressure-thresholds on fingers and toes. Pain 1989;38:211–217.
13. Jensen R, Rasmussen B, Pedersen B, Lous I, Olesen J. Cephalic muscle tenderness and pressure pain threshold in a general population. Pain 1992;48:197–203.
14. Jensen R, Rasmussen B, Pedersen B, Olesen J. Muscle tenderness and pressure pain thresholds in headache. A population study. Pain 1993;52:193–200.
15. Jensen K, Andersen H, Oleson J, Lindblom. Pressure-pain threshold in human temporal region. Evaluation of a new pressure algometer. Pain 1986;25:313–323.
16. List T, Helkimo M, Karlsson R. Influence of pressure rates on the reliability of a pressure algometer. J Craniomandib Disord Facial Oral Pain 1991;5:173–178.
17. Fricton JR, Schiffman E. The craniomandibular index: Validity. J Prosthet Dent 1987;58:222–228.
18. Anderson G, Schiffman E, Schellhas K, Fricton JR. Clinical vs. arthrographic diagnosis of TMJ internal derangement. J Dent Res 1989;68:826–829.
19. Westesson P. Temporomandibular joint: Comparison of magnetic resonance images with cryosectional anatomy. Radiology 1987;164:59–64.
20. Ross JB. Diagnostic criteria and nomenclature for TMJ arthrography in sagittal section. Part 1. Derangements. J Craniomandib Disord Facial Pain 1987;3:185–201.
21. Rohlin A, Ackerman S, Kopp S. Tomography as an aid to detect macroscopic changes of the temporomandibular joint: An autopsy study of the aged. Acta Odontol Scand 1986;44:131–140.
22. Marklund G, Molin C. Horizontal isometric muscle forces of the mandible. A comparative study of subjects with and without manifest mandibular pain dysfunction syndrome. Acta Odontol Scand 1972;30:97–115.
23. Dolan EA, Keefe FJ. Muscle activity in myofascial pain dysfunction syndrome patients: A structured clinical evaluation. J Craniomandib Disord Facial Oral Pain 1988; 2:101–105.
24. Blanksma NG, Van Eijers TMGJ, Weijs WA. Electromyographic heterogeneity in the human masseter muscle. J Dent Res 1992;71:47–52.
25. Lam EW, Hannam AG. Regional magnetic resonance spectroscopy of exercising human masseter muscle. Arch Oral Biol 1992;37:49–56
26. Fricton J. Myofascial pain syndrome. Neurologic Clinics 1989;7:413–427.
27. Jensen K, Tuxen C, Pedersen-Bjergaard V, Jansen I, Edvinsson L, Olesen J. Pain and tenderness in human temporal muscle induced by bradykinin and 5-hydroxytryptamine. Peptides 1990;11:1127–1132.
28. Berberich P, Hoheisel V, Mense S. Effects of a carrageenan-induces myositis on the discharge properties of Group III and IV muscle receptors in the cat. J Neurophys 1988;59:1395–1409.
29. Dubner R. Spinal cord neuroplasticity. In: Vecchiet L, Lindbloom V (eds). Mechanisms of Persistent Pain Following Tissue Damage and Nerve Injury. Proceedings of International Congress on New Trends in Referred Pain and Hyperalgesia. Chieti, Italy, 1993, In press.
30. Dubner R. Neuronal plasticity and pain following peripheral tissue inflammation or nerve injury. In: Dubner, Gebhart, Bond (eds). Proceedings of the Vth World Congress on Pain. New York: Elsevier, 1991:263–276.
31. Tattersall JE, Cervero F, Lumb BM. Effects of reversible spinalization on the visceral input to visceromatic neurons in the lower thoracic spinal cord of the cat. J Neurophys 1986;56:785–796.
32. Fields H (ed). Pain. New York: McGraw Hill, 1987:79–97.
33. Kojima Y. Convergence patterns of afferent information from the temporomandibular joint and masseter muscle in the trigeminal subnucleus caudalis. Brain Res Bull 1990;24:609–616.
34. Whitney C, Von Korff M. Regression to the mean in treated versus untreated chronic pain. Pain 1992;50:281–285.
35. Carlson C, Okeson J, Falace D, Nitz A, Curran S, Anderson D. Comparison of psychologic and physiologic functioning between patients and matched controls. J Orofacial Pain 1993;7:15–22.
36. Max M, Culnane M, Schafer S, et al. Amitriptyline relieves diabetic neuropathy pain in patients with normal or depressed mood. Neurology 1987;37:589–596.
37. Max M, Schafer S, Culnane M, Smoller B, Dubner R, Gracely R. Amitriptyline but not lorazepam relieves postherpetic neuralgia. Neurology 1988;38:11427–11432.
38. Yaksh TL, Aimone LD. In: Wall PD, Melzack R (eds). Textbook of Pain. Edinburgh: Churchill Livingstone, 1990:181–205.
39. Sharav Y, Singer E, Schmidt E, Dionne R, Dubner R. The analgesic effect of amitriptyline on chronic facial pain. Pain 1987;31:199–209.
40. Goldenberg DL. A review of the role of tricyclic medications in the treatment of fibromyalgia syndromes. J Rheumatol 1990;16(suppl 19):137–139.

41. Wolfe F, Smythe HA, Yunnus MB, et al. The American College of Rheumatology criteria for the classification of fibromyalgia. Arthritis Rheum 1990;33:160–172.

42. Simons DG. Myofascial pain syndromes: Where are we? Where are we going? Arch Phys Med Rehabil 1988; 69:207–212.

Resumen

Influencia del tiempo, lado facial y localización del los umbrales del dolor a la presión en los desórdenes temporomandibulares miógenos crónicos

Este estudio examinó los umbrales del dolor a la presión en los músculos temporal y masetero de 29 pacientes con desórdenes temporomandibulares (DTM) miógenos bilaterales crónicos, y de 11 pacientes de control. Los pacientes con evidencia de patología de la articulación temporomandibular fueron excluidos de estudio. Se midió la influncia del tiempo, lado facial, sitio muscular y lado con el mayor dolor a la precisión. No se encontraron deferencias segnificatives en los umbrales del dolor a la presión entre los lados que pesentaban mas o menos dolor, tal y como fue indicado por los pacientes, lo cual soporta la teoría del dolor mediado centralamente. Los umbrales del dolor a lo presión en los pacientes variaron en las cuatro sesiones, lo cual es consistente con los reportes recientes de niveles fluctuantes de dolor en pacientes con DTM. Otros hallazgos incluyen las diferencias significativas en los umbrales del dolor a la presión entre los sitios musculares en los pacientes experimentales y los umbrales del dolor a la presión mas bajos experimental en relación al de control. La fiabilidad intra-sesiones e inter-sesiones fue adecuada para los pacientes experimentales (r = .85 y r = .75 respectivamente), y para los pacientes de control fue de r = .90 y r = .75 respectivamente.

Zusammenfassung

Einfluss von Zeit, Gesichtsseite und Gebiet auf die Druckschmerzschwelle in der chronisch myogenen Myoarthropathie des Kausystems (MAP)

Diese Studie untersuchte die Druckschmerzschwelle am Masseter und Temporalis von 29 Patienten mit chronisch myogener MAP und 11 Kontrollpatienten. Patienten mit nachgewiesener Kiefergelenkpathologie wurden weggelassen. Der Einfluss von Zeit, Gesichtsseite, Muskelstelle und der Seite des grössten spontanen Schmerzes auf die Druckschmerzschwelle wurde gemessen. Man konnte keine signifikanten Unterschiede in der Druckschmerzschwelle zwischen der mehr und der weniger spontan-schmerzhaften Seite finden, was die Theorie des zentral gesteuerten Schmerzes unterstützt. Die mittleren Schmerzschwellen der Patienten differierten über 4 Sitzungen, was mit aktuellen Berichten über den fluktuierenden Charakter der Schmerzintensität bei MAP-Patienten übereinstimmt. Weitere Resultate zeigten signifikante Unterschiede in der Druckschmerzschwelle zwischen einzelnen Muskelstellen in der Patienten-und Kontrollgruppe; die Patienten hatten tiefere Druckschmerzschwellen. Der Korrelationskoeffizient innerhalb und zwischen den Sitzungen war für Patienten r = 0.85 und r = 0.75, in der Kontrollgruppe r = 0.9 und r = 0.75.