Migraine is the Most Prevalent Primary Headache in Individuals with Temporomandibular Disorders

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Aims: *To assess the prevalence of primary headaches (HA) in adults with temporomandibular disorders (TMD) who were assessed in a specialty orofacial pain clinic, as well as in controls without TMD.* ***Methods:*** *The sample consisted of 158 individuals with TMD seen at a university-based specialty clinic, as well as 68 controls. The Research Diagnostic Criteria for TMD were used to diagnose the TMD patients. HAs were assessed using a structured interview and classified according to the Second Edition of the International Classification for Headache Disorders. Data were analyzed by chi-square tests with a significance level of 5% and odds ratio (OR) tests with a 95% confidence interval (CI).* ***Results:*** *HAs occurred in 45.6% of the control group (30.9% had migraine and 14.7% had tension-type headache [TTH]) and in 85.5% of individuals with TMD. Among individuals with TMD, migraine was the most prevalent primary HA (55.3%), followed by TTH (30.2%); 14.5% had no HA. In contrast to controls, the odds ratio (OR) for HA in those with TMD was 7.05 (95% confidence interval [CI] = 3.65–13.61;* P = *.000), for migraine, the OR was 2.76 (95% CI = 1.50–5.06;* P = *.001), and for TTH, the OR was 2.51 (95% CI = 1.18–5.35;* P = *.014). Myofascial pain/arthralgia was the most common TMD diagnosis (53.2%). The presence of HA or specific HAs was not associated with the time since the onset of TMD (*P = *.714). However, migraine frequency was positively associated with TMD pain severity (*P = *.000).* ***Conclusion:*** *TMD was associated with increased primary HA prevalence rates. Migraine was the most common primary HA diagnosis in individuals with TMD.* J OROFAC PAIN 2010;24:287–292

Key words: facial pain, migraine, prevalence, temporomandibular joint, tension-type headache

Temporomandibular disorders (TMD) refer to a group of conditions characterized by a constellation of symptoms and signs, including: pain in the temporomandibular joint (TMJ), periauricular area, or muscles of mastication, TMJ sounds, and deviations or restriction in mandibular range of motion.[1] TMD reflect the most common types of chronic orofacial pain referred to dentists.[2]

Headache (HA) is a frequent symptom related to TMD. Patients with TMD often have headache as one of their main symptoms; sometimes HA is the only manifestation of TMD.[3–6] According to the Second Edition of the International Classification of Headache Disorders (ICHD-II),[7] HA syndromes are divided into *(1)* primary headache syndromes; *(2)* secondary headaches, and *(3)* cranial neuralgias, primary or central facial pain, and others. In primary

headaches, causal events are not identified, whereas in "secondary" headaches, a cause (eg, intracranial tumors, infections) can be recognized and temporarily related to headache onset.

Epidemiological studies have also suggested an association between TMD and specific primary HAs, such as migraine and tension-type headache (TTH).[8–21] Indeed, individuals with TMD are 1.8 to 2 times more likely to have other primary HAs as compared to controls.[9,15] However, there is large variability among results, and the nature of the association is not clear.[12] Clarifying this topic is of clinical importance. If comorbidity between TMD and a specific primary HA is established, diagnosing one condition raises the suspicion that the other may exist. Also, one (eg, TMD) may make the treatment of the second (eg, migraine) more refractory. Alternatively, if both conditions are not comorbid, the findings are of interest as well, since migraine and TTH sufferers are often diagnosed as having "TMD" or receive dental treatments in an attempt to improve their HAs.[20–22] Accordingly, the aim of this case-control study was to assess the prevalence of primary HAs in adults with TMD who were assessed in a specialty orofacial pain clinic, as well as in controls without TMD. It was hypothesized that TMD patients would present higher frequency of primary HAs.

Materials and Methods

This was a case-control study where the presence and frequency of HAs were compared in individuals with TMD (cases) and without TMD (controls). The study was approved by the Research Ethic Committee, Araraquara Dental School (UNESP—Univ Estadual Paulista, Brazil) (proc: 33/06). Patient consent forms were obtained.

Inclusion/Exclusion Criteria

Cases were identified among individuals seeking treatment because of facial pain at the TMD and Orofacial Pain Clinic at Araraquara Dental School (UNESP—Univ Estadual Paulista, Brazil). They included individuals consecutively diagnosed as having chronic TMD with pain according to the Research Diagnostic Criteria for TMD (RDC/TMD) Axis I.[23] Following the criteria established by the International Association for the Study of Pain (IASP),[24] the presence of TMD for more than 6 months was considered chronic. Exclusion criteria included: acute pain; isolated Axis I group II (disc displacements); osteoarthrosis without other TMD manifestations; odontalgia; neuropathy, intraoral lesions; any chronic pain syndrome (other than headaches), including fibromyalgia or arthritis; and impairments in cognition or language. Individuals of the control group had no TMD and were selected among patients seeking routine dental care treatment at the same university. Among the 235 examined individuals (cases and controls), nine were excluded because of exclusion criteria. Overall, no individuals refused to participate in the study. Since the questionnaires applied are part of the routine examination in the Orofacial Pain Clinic, all patients agreed that data collected would be used for this research. While the control group was composed of non-TMD patients recruited from other clinics, the authors attribute their high response rate to the noninvasive procedures that were applied in a single session.

Assessments

All individuals were evaluated by one trained experimenter, according to standardized methods of assessment, as noted below:

Orofacial Pain Clinic Protocol. All participants were interviewed and systematically examined for cervical, cranial, facial, dental, and other oral structures. Objectives were to obtain: *(1)* the chief complaint; *(2)* TMD diagnosis and TMJ pain characteristics (location, intensity, quality, duration, exacerbating factors); *(3)* HA and extracephalic pain; and *(4)* the medical history. The American Academy of Orofacial Pain (AAOP) diagnostic criteria[1] were applied for the differential diagnoses with other conditions that may mimic TMD. In those with a confirmed TMD diagnoses, the classification was made according to the RDC/TMD criteria, Portuguese version.[23,25–27]

RDC/TMD. TMD were classified using the RDC/TMD. The RDC/TMD questionnaires were filled in by the patients; a single investigator (dentist with experience in diagnosing TMD) was present to help in case of any doubts, but did not help with the patients' responses. The clinical examination was made by one trained experimenter who had graduated in dentistry and had experience in the diagnosis of TMD. Based on the Axis I of the RDC/TMD, patients were stratified as a function of their TMD status into: no TMD (controls); TMD with myofascial pain; TMD with TMJ arthralgia (without osteoarthrosis); and TMD with myofascial pain and TMJ arthralgia.

The Axis II of the RDC/TMD assesses, among other domains, severity of TMD chronic pain (Grade 0 to IV), psychological symptoms and

depression, and other nonspecific physical symptoms. It also asks about limitations related to mandibular functioning. Based on responses, severity of TMD chronic pain was graded as: Grade 0 (no chronic TMD pain in the prior 6 months); Grade I (Low Disability-Low Intensity Pain); Grade II (Low Disability-High Intensity Pain); Grade III (High Disability-Moderately Limiting); and Grade IV (High Disability-Severely Limiting).[23]

ICHD-II Based Questionnaire. The HA questionnaire consisted of 26 questions assessing the distinguishing features required for the diagnosis of the most common primary HAs. Questions focused on characteristics of pain (eg, throbbing, in pressure), severity of pain (mild, moderate, or severe), location (strictly unilateral, bilateral, etc), frequency of HA attacks, duration of HA attacks, associated symptoms (nausea, photophobia, and phonophobia), and precipitating factors. The questions were based on the International Classification for Headache Disorders (ICHD-II)[7] and applied by one investigator with specific training in HA medicine.[20,21] Based on this questionnaire, all primary HA diagnoses could be screened: *(1)* migraine, *(2)* TTHs, *(3)* cluster HAs and other trigeminal autonomic cephalalgias, and *(4)* other primary HA.

Data Analysis

According to prior reports, power analyses suggested a sample of 68 controls and 136 TMD patients (randomized at a 1:2 ratio). Descriptive statistics were used to characterize the sample. Participants were stratified by gender and HA status, and contrasts were performed in cases versus controls. Specific HA subtypes (migraine and TTH) were also contrasted. Finally, the sample was stratified according to the grade of TMD chronic pain in order to study a possible association with HA diagnoses. For comparison of proportions, the chi-square test was performed and the significance level adopted was 5%. Also, odds ratio (OR) tests, with 95% confidence interval (CI), were used to study associations between TMD and primary HA overall, as well as specific primary HAs. Statistical analyses were performed using Stata (version 10) and SPSS (version 15.0 for Windows).

Results

The sample consisted of 158 individuals with TMD and 68 without TMD (controls). In those with TMD, 133 (84.2%) were women and 25 (15.8%) were men; mean age was 40.1 (18 to 76) and 41.7 (18 to 62) years old, respectively. The control group comprised 52 (76.5%) women and 16 (23.5%) men, with mean age of 38.4 (18 to 69) and 36.8 (18 to 62) years, respectively. There were no significant differences between groups for gender (P = .233) or age (P = .213).

From the total sample, 167 (73.9%) individuals presented HA. HA was more common in the TMD group than in the control group (85.5% versus 45.6%, respectively). Migraine was the most common HA subtype in individuals with TMD and in controls (55.3% versus 30.9%), followed by TTH (30.2% versus 14.7%). Other types of HAs were seen in six individuals with TMD, but not in controls. One had HA attributed to rhinosinusitis, four presented HA attributed to neck disorders, and one was classified as presenting HA attributed to other facial or cranial structures.[7] Those individuals were excluded from the analyses since they presented secondary HAs.

After stratifying the subjects by gender, primary HAs were found to be more common in women compared to men (77.7% versus 56.1%, respectively, P = .008), while TMD was not (71.0% versus 61.0%, respectively, P = .280).

Overall, HAs were more common in TMD relative to controls. Any HA was seen in 85.5% (n = 130) of those with TMD versus 45.6% (n = 31) of controls. For migraine, rates were 55.3% versus 30.9%; for TTH, rates were 30.2% versus 14.7% (P = .000). Also, as compared to controls, individuals with TMD presented higher risk for any HA (OR = 7.05; 95% CI = 3.65–13.61), for migraine (OR = 2.76; 95% CI=1.50-5.06), and for TTH (OR = 2.51; 95% CI = 1.18–5.35) (Table 1).

In individuals with TMD, myofascial pain/arthralgia was the most common diagnosis, presented in 53.2% (n = 117) of the sample, followed by myofascial pain (n = 29; 13.2%) and arthralgia only (n = 6; 2.7%).

Among individuals with myofascial pain, 51.7% had migraine, 34.5% had TTH, and 13.8% had no HAs. In those with arthralgia, 50% were also diagnosed with migraine, 33.3% with TTH, and 16.7% with no HA. Among the myofascial pain/arthralgia group, migraine was found in 56.4%, TTH in 29.1%, and 14.5% had no HA (Table 2).

Onset of TMD pain was compared among individuals with no HA, migraine, or TTH. Individuals with migraine presented a longer but nonsignificant duration of TMD (mean of 89.3 months) than individuals with TTH (78.8 months) or no HA (72.8 months) (ANOVA, P = .714).

Table 3 displays HA diagnoses as a function of the grade of TMD. Frequency of migraine attacks

Table 1 Frequency of Primary HA According to the Presence of TMD and Associations Between TMD and Primary HA Overall, Migraine, and TTH

	HA						
TMD	No HA* n (%)	OR 95% CI	Migraine** n (%)	OR 95% CI	TTH*** n (%)	OR 95% CI	Total n (%)
No	37 (54.4)	Reference	21 (30.9)	Reference	10 (14.7)	Reference	68 (100.0)
Yes	22 (14.5)	7.05 (3.65–13.61)	84 (55.3)	2.76 (1.50–5.06)	46 (30.2)	2.51 (1.18–5.35)	152 (100.0)
Total	59 (26.8)		105 (47.7)		56 (25.5)		220 (100.0)

χ^2 *P = .000; **P = .001; ***P = .014

Table 2 Distribution of HA Diagnosis as a Function of TMD Subtype

TMD diagnoses	No HA n (%)	Migraine n (%)	TTH n (%)	Total n (%)
No TMD	37 (54.4)	21 (30.9)	10 (14.7)	68 (100)
Myofascial pain	4 (13.8)	15 (51.7)	10 (34.5)	29 (100)
Arthralgia	1 (16.7)	3 (50.0)	2 (33.3)	6 (100)
Myofascial pain/arthralgia	17 (14.5)	66 (56.4)	34 (29.1)	117 (100)
Total	59 (26.8)	105 (47.7)	56 (25.5)	220 (100)

Table 3 Frequencies of HA Diagnoses According to Chronic TMD Pain Severity

TMD diagnoses	No HA n (%)	Migraine n (%)	TTH n (%)	Total n (%)
No TMD	34 (54.0)	20 (31.7)	9 (14.3)	63 (100.0)
I and II	19 (17.1)	54 (48.6)	38 (34.3)	111 (100.0)
III and IV	4 (11.1)	23 (63.9)	9 (25.0)	36 (100.0)
Total	57 (27.1)	97 (46.2)	56 (26.7)	210 (100.0)

χ^2 test: P = .000.

increased as a function of TMD chronic pain (P < .0001). Migraine was diagnosed in 31.7% of the controls, in 48.6% of individuals with grade I or II of TMD pain, and in 63.9% of individuals with more severe chronic pain. For TTH, the figures were 14.3%, 34.3%, and 25%, respectively. Ten individuals were excluded from this analysis because of incomplete responses.

Discussion

This study was carried out because of the relative paucity of information about the potential comorbidity between TMD and primary HAs. The most important findings were: *(1)* most patients who sought specialty care for TMD treatment had HA; *(2)* migraine was the most common HA in individuals with TMD; and *(3)* there was an association between TMD severity and primary HA frequency.

As expected, most of the sample was composed of middle-aged women since TMD is two to five times more common in women than in men, mostly in the third to fifth decades of life.[2,13] HAs, especially migraine, are also more common in women aged between 20 to 50 years.[28–30] Current evidence supports the existence of gender differences in pain response. Most pain conditions are more common in women (eg, TMD, fibromyalgia, migraine). Furthermore, women tend to characterize their pain as more intense, frequent, and continuous than men.[13,31,32] This could explain the higher frequency of women than men seeking care for TMD treatment observed in the present sample (85% women and 15% men).

The prevalence of HA in individuals with TMD (85.5%) in the present study was aligned with the reported overall prevalence of HA (70 to 85%).[13,17] However, ORs were increased for any HA, and the prevalences of migraine and TTH were higher in TMD individuals versus controls. The data corroborate previous finds that primary HA and TMD are comorbid.[9,12,15,17] The ORs were higher than previously reported,[9,15] probably because the sample was composed of patients presenting chronic TMD from a tertiary clinic.

The present results are in accordance with the literature. HA is almost two times more frequent in TMD patients than in the general population,[9,15] especially if the sample is selected from specialty centers.[12] Similarly, there are commonly reported signs and symptoms of TMD in HA populations, strongly correlated with the frequency and intensity of HA crises.[19] Also, it was verified that patients with primary HA experience worsening and/or first onset of pain when TMD is present, with different degrees of severity and frequency of involvement.[5,9,10,15,17,19]

The association noted between TMD and primary HAs could be explained by two different but nonmutually-exclusive hypotheses. First, continuous stimulation of the trigeminal subnucleus caudalis (which might occur in individuals with TMD) could influence the frequency and intensity of primary HAs.[33–36] Accordingly, in migraineurs, TMD may act as a perpetuating, aggravating, and/or triggering factor.[37–39] Similarly, in TTH patients, TMD may predispose patients to changes in nociceptive pathways in the brain, especially in those related to the pericranial and masticatory muscles.[40–43]

Alternatively, the very areas involved with HA onset (in the brain) may explain peripheral symptoms.[37,42–46] After activation of areas responsible for HA onset, central sensitization of the trigeminal subnucleus caudalis may happen.[40] A hallmark of trigeminal central sensitization is cutaneus allodynia in the trigeminal nerve distribution. Accordingly, TMJ pain may reflect a lower peripheral threshold as a result of such central changes,[33–36] and central sensitization associated with primary HAs could predispose patients to generalized pain (head, face, and neck),[47] which might explain the high frequency of myofascial pain/arthralgia combined diagnoses evident in the present sample.

In the present study, the frequency of migraine attacks increased as a function of TMD chronic pain, and these data are also consistent with the literature. A previous study demonstrated that 78.5% of TMD patients present low disability Studies have shown that 78.5% of TMD patients present low disability.[48] However, when HAs are considered, more than 31% of patients with migraine may report high disability.[49] Additionally, it was verified that TMD patients presented more HA and more related disability, suggesting that coexisting HA and TMD may lead to higher incapacitation.[17,50]

When interpreting the present results, some aspects deserve caution. First, this study used a case-control design, which can assess causality. However, since the chronology of symptoms was not assessed, only the association of symptoms, not causal effects, was reported. Also, the TMD sample was composed of patients seeking treatment for facial pain, so the results are not representative of the general population. Nonetheless, similar findings were observed in a recent population-based study.[20,21] Finally, the sample size limited multivariate analyses.

In conclusion, TMD were found to be associated with increased primary HA prevalence rates. Migraine was the most common primary HA in individuals with TMD. Populational and longitudinal studies need to investigate putative mechanisms for this comorbidity.

Acknowledgments

The authors thank the State of São Paulo Research Foundation–FAPESP (06/02848-3) and the National Council for Scientific and Technological Development–CNPq (133352/2007-2) for the financial assistance provided.

References

1. Okeson JP. Orofacial Pain: Guidelines for Assessment, Diagnosis and Management. Chicago: Quintessence, 1996.
2. Dworkin SF, Huggins KH, LeResche L, et al. Epidemiology of signs and symptoms in temporomandibular disorders: Clinical signs in cases and controls. J Am Dent Assoc 1990;120:273–281.
3. Kreisberg MK. Headache as a symptom of craniomandibular disorders I: Pathophysiology. Cranio 1986;4:135–142.
4. Kreisberg MK. Headache as a symptom of craniomandibular disorders II: Management. Cranio 1986;4:219–228.
5. Graff-Radford SB. Temporomandibular disorders and headache. Dent Clin N Am 2007;51:129–144.
6. Lupoli TA, Lockey RF. Temporomandibular dysfunction: An often overlooked cause of chronic headaches. Ann Allergy Asthma Immunol 2007;99:314–318.
7. Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders, ed 2. Cephalalgia 2004; 24:8–160.
8. Lous I, Olesen J. Evaluation of pericranial tenderness and oral function in patients with common migraine, muscle contraction, headache and "combination headache." Pain 1982;12:385–393.
9. Kemper JT, Okeson JP. Craniomandibular disorders and headaches. J Prosthet Dent 1983;49:702–705.
10. Schokker RP, Hansson TL, Ansink BJJ. Craniomandibular disorders in patients with different types of headache. J Craniomandib Disord 1990;4:47–51.
11. Haley D, Schiffman E, Baker C, Belgrade M. The comparison of patients suffering from temporomandibular disorders and a general headache population. Headache 1993; 33:210–213.
12. Molina OF, Santos J Jr, Nelson SJ, Grossman E. Prevalence of modalities of headaches and bruxism among patients with craniomandibular disorder. Cranio 1997; 15:314–325.

13. Drangsholt M, LeResche L. Temporomandibular disorder pain. In: Crombie IK, Croft PR, Linton SJ, LeResche L, Von Korff M (eds). Epidemiology of Pain. Seattle: IASP, 1999:203–233.
14. Pettengill C. A comparison of headache symptoms between two groups: A TMD group and a general dental practice group. Cranio 1999;17:64–69.
15. Ciancaglini R, Radaelli G. The relationship between headache and symptoms of temporomandibular disorder in the general population. J Dent 2001;29:93–98.
16. Bernhardt O, Gesch D, Schwahn C, et al. Risk factors for headache including TMD signs and symptoms, and their impact in quality of life. Results of the study of health in Pomerania (SHIP). Quintessence Int 2005;36:55–64.
17. Mitrirattanakul S, Merrill RL. Headache impact in patients with orofacial pain. J Am Dent Assoc 2006; 137:1267–1274.
18. Glaros AG, Urban D, Locke J. Headache and temporomandibular disorders: Evidence for diagnostic and behavioral overlap. Cephalalgia 2007;27:542–549.
19. Ballegaard V, Thede-Schimidt-Hansen P, Svensson P, Jensen R. Are headaches and temporomandibular disorders related? A blinded study. Cephalalgia 2008;28:832–841.
20. Gonçalves DAG, Bigal ME, Jales LCF, Camparis CM, Speciali JG. Headache and symptoms of temporomandibular disorder: An epidemiological study. Headache 2010;50:231–241.
21. Gonçalves DAG, Speciali JG, Jales LCF, Camparis CM, Bigal ME. Temporomandibular symptoms, migraine and chronic daily headaches in the population. Neurology 2009;73:645–646.
22. Okeson JP. Management of Temporomandibular Disorders and Occlusion, ed 4. St Louis: Mosby, 1998.
23. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: Review, criteria, examinations and specifications, critique. J Craniomand Disord 1992;6:301–355.
24. Merskey H, Bogduk N. Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms. Seattle: IASP, 1994.
25. Kosminsky M, Lucena LBS, Siqueira JTT, Pereira FJ Jr, Góes PSA. Cultural adaptation of the "Research Diagnostic Criteria for Temporomandibular Disorders: Axis II" questionnaire. J Bras Clin Odontol Int 2004;8: 51–61.
26. Lucena LBS, Kosminsky M, Costa LJ, Góes PSA. Validation of the Portuguese version of the RDC/TMD Axis II questionnaire. Braz Oral Res 2006;20:312–317.
27. Pereira FJ, Favilla EE, Dworkin SF, Huggins KH. Criterios de diagnostico para pesquisa das disfuncoes temporomandibulares (RDC/TMD) traducao official para a lingua portuguesa. J Bras Clin Odontol Integr 2004;8:384–395.
28. Scher AI, Stewart WF, Lipton RB. Migraine and headache: A meta-analytic approach. In: Crombie IK, Croft PR, Linton SJ, LeResche L, Von Korff M (eds). Epidemiology of Pain: A Report of the Task Force on Epidemiology. Seattle: IASP, 1999:159–170.
29. Rasmussen BK. Epidemiology of headache. Cephalalgia 2001;21:774–777.
30. Stovner JL, Scher AI. Epidemiology of headache. In: Olesen J, Goadsby PJ, Ramadan NM, Tfelt-Hansen P, Welch KMA (eds). The Headaches, ed 3. Philadelphia: Lippincott Williams & Wilkins, 2006:393–398.
31. Dao TT, LeResche L. Gender differences in pain. J Orofac Pain 2000;14:169–184.
32. Cairns BE. The influence of gender and sex steroids on craniofacial nociception. Headache 2007;47:319–324.
33. 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.
34. Jensen TS. Recent advances in pain research: Implications for chronic headache. Cephalalgia 2001;21:765–769.
35. Merrill RL. Central mechanisms of orofacial pain. Dent Clin N Am 2007;51:45–59.
36. Woolf CJ. Central sensitization. Uncovering the relation between pain and plasticity. Anesthesiology 2007;106: 184–187.
37. Olesen J. Clinical and pathophysiological observations in migraine and tension-type headache explained by integration of vascular, supraspinal and myofascial inputs. Pain 1991;46:125–132.
38. Shevel E, Spierings EH. Cervical muscles in the pathogenesis of migraine headache. J Headache Pain 2004;5:12–14.
39. Okeson JP. Bell's Orofacial Pains: The Clinical Management of Orofacial Pain, ed 6. Chicago: Quintessence, 2005.
40. Fernández-de-las-Peñas C, Schoenen J. Chronic tension-type headache: What is new? Curr Opin Neurol 2009;22: 254–261.
41. Jensen R, Bendtsen L, Olesen J. Muscular factors are of importance in tension-type headache. Headache 1998;21: 765–769.
42. Bendtsen L. Central sensitization in tension-type headache: Possible pathophysiological mechanisms. Cephalalgia 2000;20:486–508.
43. Bendtsen L, Schoenen J. Synthesis of tension type headache. In: Olesen J, Goadsby PJ, Ramadan NM, Tfelt-Hansen P, Welch KMA (eds). The Headaches, ed 3. Philadelphia: Lippincott Williams & Wilkins, 2006: 679–683.
44. Goadsby PJ. Migraine pathophysiology. Headache 2005;45:S14–S21.
45. Olesen J, Goadsby PJ. Synthesis of migraine mechanisms. In: Olesen J, Goadsby PJ, Ramadan NM, Tfelt-Hansen P, Welch KMA (eds). The Headaches, ed 3. Philadelphia: Lippincott Williams & Wilkins, 2006:393–398.
46. Buchgreitz L, Lyngberg AC, Bendtsen L, Jensen R. Frequency of headache is related to sensitization: A population study. Pain 2006;123:19–27.
47. Mense S, Simons DG, Russel IJ. Muscle Pain: Understanding its Nature, Diagnosis, and Treatment. Baltimore: Lippincott Williams & Wilkins, 2001.
48. Yap AU, Chua EK, Hoe JK. Clinical TMD, pain-related disability and psychological status of TMD patients. J Oral Rehabil 2002;29:374–380.
49. Patel NV, Bigal ME, Kolodner KB, Leotta C, Lafata JE, Lipton RB. Prevalence and impact of migraine and probable migraine in a health plan. Neurology 2004;63: 1432–1438.
50. Dando WE, Branch MA, Maye JP. Headache disability in orofacial pain patients. Headache. 2006;46:322–326.