Headache
© 2014 American Headache Society

ISSN 0017-8748
doi: 10.1111/head.12446
Published by Wiley Periodicals, Inc.

Review Article

Association Between Tension-Type Headache and Migraine With Sleep Bruxism: A Systematic Review

Graziela De Luca Canto, DDS, MSc, PhD; Vandana Singh, DDS, MSc; Marcelo E. Bigal, MD, PhD; Paul W. Major, DDS, MSc, FRCD(C); Carlos Flores-Mir, DDS, DSc, FRCD(C)

Aim.—To evaluate the association between tension-type headache and migraine with sleep bruxism (SB).

Background.—The association between SB and headaches has been discussed in both children and adults. Although several studies suggested a possible association, no systematic analysis of the available published studies exists to evaluate the quantity, quality, and risk of bias among those studies.

Methods.—A systematic review was undertaken, including articles that classified the headaches according to the International Classification of Headache Disorders and SB according to the criteria of the American Association of Sleep Medicine. Only articles in which the objective was to investigate the association between primary headaches (tension-type and migraine) and SB were selected. Detailed individual search strategies for The Cochrane Library, MEDLINE, EMBASE, PubMed, and LILACS were developed. The reference lists from selected articles were also checked. A partial grey literature search was taken by using Google Scholar. The methodology of selected studies was evaluated using the quality in prognosis studies tool.

Results.—Of 449 identified citations, only 2 studies, both studying adults, fulfilled the inclusion criteria. The presence of SB significantly increased the odds (study 1: odds ratio [OR] 3.12 [1.25-7.7] and study 2: OR 3.8; 1.83-7.84) for headaches, although studies reported different headache type.

Conclusion.—There is not enough scientific evidence to either support or refute the association between tension-type headache and migraine with SB in children. Adults with SB appear to be more likely to have headache.

Key words: headache, migraine, tension-type headache, sleep bruxism, review

Abbreviations: AASM American Association of Sleep Medicine, CM chronic migraine, CTTH chronic tension-type headache, EM episodic migraine, ETTH episodic tension-type headache, ICHD International Classification of Headache Disorders, OR odds ratio, PSG polysomnography, QUIPS quality in prognostic studies, RR risk ratio, SB sleep bruxism, TMD temporomandibular disorder, TrPs trigger points, TTH tension-type headache

(*Headache* 2014;••:••-••)

From the Department of Dentistry, Federal University of Santa Catarina, Florianópolis, Brazil (G. De Luca Canto); School of Dentistry, Faculty of Medicine and Dentistry, University of Alberta, Edmonton, AB, Canada (G. De Luca Canto, V. Singh, P.W. Major, and C. Flores-Mir); Therapeutic Area Head, Migraine and Headache Clinical Development, Teva Pharmaceuticals, Frazer, PA, USA (M.E. Bigal).

Address all correspondence to G. De Luca Canto, Departamento de Odontologia – CCS, Universitário Federal de Santa Catarina – UFSC, Campus Universitario Trindade, Florianópolis, Santa Catarina 88040-900, Brasil.

Accepted for publication May 12, 2014.

Headache is a nearly universal human experience.[1] For example, in Europe, slightly more than 50% of the population complains of recurrent headaches.[2] Headache significantly burdens lifestyle and health: almost all people with migraine and 60% of those with tension-type headache (TTH) experience reductions in work capacity and social activities.[3]

Conflict of Interest: None.

Funding: This research was conducted without funding support.

Although the pathophysiology of TTH and migraine is complex and multifactorial, past studies suggest that dysfunctions of the masticatory and cervical muscles are associated with an increased prevalence for these disorders.[4]

It has been hypothesized that sleep bruxism (SB) may cause headaches during the day.[5] Furthermore, both TTH and migraine have been associated with SB.[6,7] As a result of periodic mechanical grinding, besides headaches,[8] SB can lead to disruption of the bed partner's sleep,[9,10] tooth wear,[8] tooth mobility,[8] tongue/cheek indentation, and masticatory muscle hypertrophy.[8] Complaints of tooth grinding occurring during sleep decline over time, from 14% in children[11] to 12.8% in adults[12] and 3% in patients over 60 years of age.[11]

The association between SB and headaches has been discussed in both children[13-18] and adults.[6,7,19-22] Although several studies suggested a possible association, no systematic analysis of the available published studies exists to evaluate the quantity, quality, and risk of bias among those studies. Therefore, the main goal of this systematic review is to evaluate and synthesize the possible association between the most common primary headaches disorders (TTH and migraine) with SB.

METHODS

Protocol and Registration.—The Preferred Reporting Items for Systematic Reviews and Meta-Analysis Checklist[23] was followed in this systematic review. We did not register the systematic review protocol.

Eligibility Criteria.—We selected only articles in which the objective was to investigate the association between primary headaches (TTH and migraine) and SB. Retained articles were only those that diagnosed primary headaches according to the International Classification of Headache Disorders (ICHD)[24-26] and SB by those that diagnosed it based on the international diagnostic criteria proposed by the American Association of Sleep Medicine (AASM)[27] (Box 1). Any publication language was considered, and published reviews, letters, and personal opinions were excluded from the data synthesis.

Box 1.—AASM Criteria for Sleep Related Bruxism Diagnosis.[27]

A. The patient reports or is aware of tooth-grinding sounds or tooth clenching during sleep.
B. One or more of the following is present:
   a. Abnormal wear of the teeth
   b. Jaw muscle discomfort, fatigue, or pain and jaw lock upon awakening
   c. Masseter muscle hypertrophy upon voluntary forceful clenching
C. The jaw muscle activity is not better explained by another current sleep disorder, medical or neurological disorder, medication use, or substance use disorder.

Information Sources.—We developed detailed individual strategies for each of the following bibliographic databases: The Cochrane Library, MEDLINE, EMBASE, PubMed, and LILACS (Appendix S1). We also hand searched the reference lists from the selected articles for any additional references that might have been missed in the electronic database searches. We undertook a partial grey literature search by using Google Scholar.

Search.—Appropriate truncation and word combinations were selected and adapted for each database search (Appendix S1). We managed the references and removed the duplicates by using appropriate software (RefWorks/COS, ProQuest, Bethesda, MD, USA). All the electronic database searches were conducted from their starting coverage date through January 13, 2014.

Study Selection.—We selected the final articles through a 2-phase process. In phase 1, 2 reviewers (G.D.L.C and V.S.) independently reviewed the titles and abstracts for all the identified references. They excluded any articles that did not appear to meet the inclusion criteria. In phase 2, the same reviewers (G.D.L.C. and V.S.) applied the inclusion criteria to the full text of the articles, as some abstracts may have presented study details incorrectly or only partially. Any disagreement in first or second phase was

resolved by discussion until a mutual agreement between the 2 authors was attained. A third author (C.F.-M.) became involved when required to make a final decision and when the articles were published in German, Portuguese, and Spanish.

Data Collection Process.—One author (G.D.L.C.) collected the required information (author[s], year of publication, setting, sample size, age of participants, objectives, methods, findings, and main conclusion) from the selected articles. A second author (V.S.) cross-checked all the collected information. Again, any disagreement was resolved by discussion and mutual agreement between the 2 reviewers (G.D.L.C. and V.S.). A third author (C.F.-M.) was involved, when required, to make a final decision.

Data Items.—For each of the included studies, we recorded: author, year of publication, sample origin, size and demographic features, results, and conclusions pertaining to the association between primary headache and SB. If the required data were not complete, attempts were made to contact the authors to retrieve any pertinent missing information.

Risk of Bias in Individual Studies.—We evaluated the methodology of selected studies by using the quality in prognosis studies (QUIPS) tool.[28] Two reviewers categorized each 6 bias domains (study participation, study attrition, prognostic factors measurement, outcome measurement, study confounding, and statistical analysis and reporting) in high, moderate, or low risk of bias. Any disagreement was resolved by discussion until a mutual agreement between the 2 authors was attained. A third author became involved when required to make a final decision.

Summary Measures.—Any outcome measurement was considered (risk ratio [RR], odds ratio [OR], or risk difference for dichotomous outcomes; mean difference or standardized mean difference for continuous outcomes) that evaluated the association of primary headaches and SB in studies with and without a control group.

Synthesis of Results.—A meta-analysis was planned because the data from the included studies were considered relatively homogeneous. A fixed-effect meta-analysis was planned following a 2-stage process. In the first stage, a summary statistic measurement was calculated for each study, if not reported already, to describe the observed intervention effect. The summary statistic considered RRs or ORs, if data were dichotomous, or a difference between means, if data were continuous. In the second stage, the summary intervention effect estimate was calculated as a weighted average of the intervention effects estimated in the individual studies. The standard error of the summary intervention effect was calculated from the confidence interval and associated the *P* value.

Risk of Bias Across Studies.—We assessed the clinical heterogeneity (by comparing variability among the participant's characteristics, type of interventions, and outcomes studied), methodological heterogeneity (by comparing the variability in study design and risk of bias), and statistical heterogeneity (by comparing variability in the intervention effects in the different included studies).

RESULTS

Study Selection.—In phase 1, we found 878 citations across the 5 electronic databases. After we removed the duplicate articles, we had 449 remaining different citations. Eighteen of those citations were found through Google Scholar. Then, we conducted a comprehensive evaluation of the abstracts and excluded 427, resulting in a final number of 40 articles after phase 1. We did not identify any additional studies from the reference lists of these studies. Therefore, we retrieved 40 articles to conduct a full-text review and later excluded 38 studies (Appendix S2). At the end, we have only 2 selected articles. A flow chart of the process of identification, inclusion, and exclusion of studies is shown in the Figure.

Study Characteristics.—Both selected articles were published in dental journals.[6,7] Sample sizes ranged from 286[7] to 1031[6] participants. The studies were conducted in 2 different countries: Brazil[7] and Germany.[6] Both studies[6,7] were published in English. A summary of the study descriptive characteristics can be found in Table 1.

Risk of Bias Within Studies.—The reported methodological quality of the 2 included studies[6,7] ranged between low and high depending on the domain.

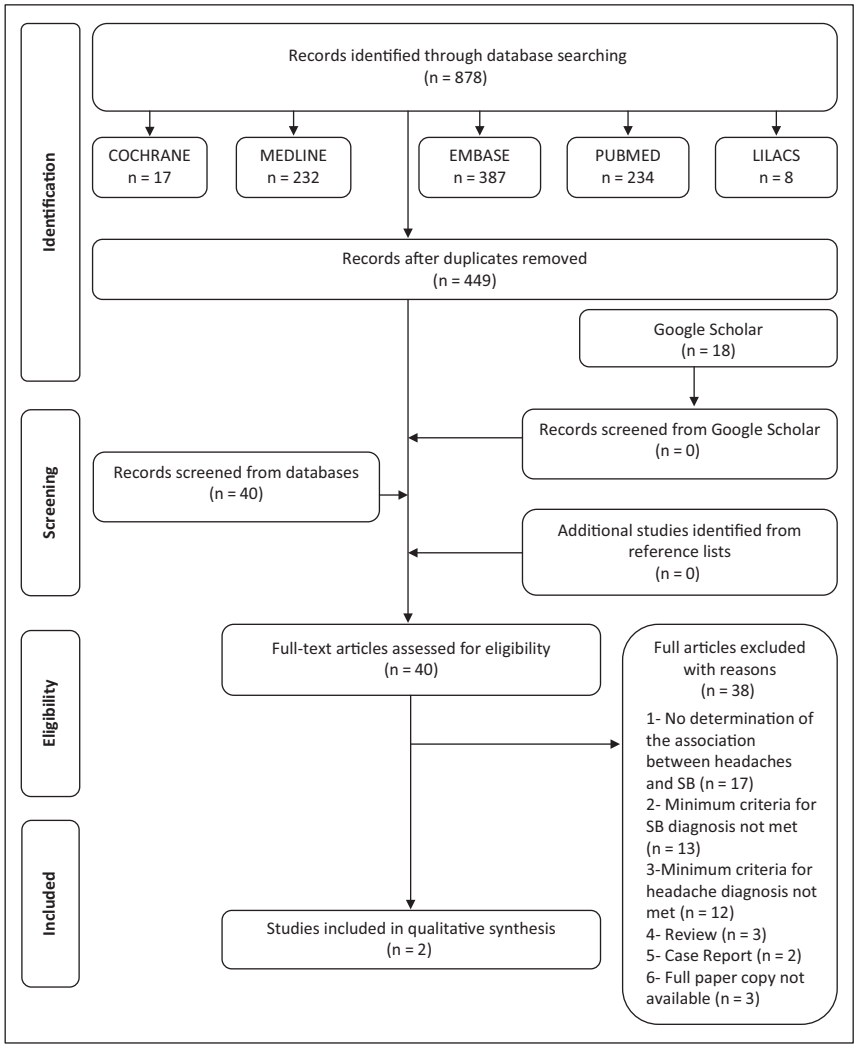

Figure.—Flow diagram of literature search and selection criteria (adapted from Preferred Reporting Items for Systematic Reviews and Meta-Analysis [PRISMA][23]).

Table 2 shows the potential risk of bias per domain while Appendix S3 presents a complete analyzed item list.

Synthesis of Results.—Troeltzsch et al[6] classified the individuals with headache in 4 groups: (1) TTH; (2) migraine; (3) TTH and migraine; and (4) other. The authors[6] reported that headaches affected women more frequently than men (1.7:1). When the authors[6] classified the subjects (mean age 49.6 years old) according to the primary headache diagnosis (presence or absence), frequent headaches were present in 67.4% of the patients. SB significantly increased the OR of the presence of headache by 3.12 times (confidence intervals = 1.25-7.7/*P* = .001). Headache intensity and frequency decreased with age. TTH was most frequently diagnosed (48%), followed by migraine (1%).

Fernandes et al[7] classified the experimental sample in 3 groups: (1) episodic migraine (EM); (2) episodic TTH (ETTH); and (3) chronic migraine (CM). The authors[7] reported a significant association between SB and CM in individuals aged from 18 to 76 years old. SB was present in 58.7% of the patients. This prevalence of SB was higher in individuals with CM (74.6%) and smaller in those with EM (58.2%) and ETTH (56.1%). The presence of SB significantly increased the OR for CM by 3.8 times (confidence intervals = 1.83-7.84/*P* = .0005). The association

Table 1.—Summary of Descriptive Characteristics of Included Articles

Author	Setting	Sample	Age	Objectives	Headache Criteria	Statistical Analysis	Findings	Main Conclusion
Troeltzsch et al[6]	Oral and Maxillofacial Surgery Private Practice, Germany	1031 (595 women and 436 men) consecutive patients in 11 months period	Mean 49.6 257 patients younger than 30 years, 474 between 30 and 60 years, and 288 older than 60 years	To identify the possible association of occlusal interferences, parafunction, TMD, physiologic, muscular, or prosthodontic factors with the occurrence of headache.	ICHD	Chi-square Mann–Whitney U Kruskal–Wallis Multinomial logistic regression analysis	67.4% were diagnosed with some type of headache. TTH was the most frequent diagnosed (48%). 8.2% of the patients showed both TTH and migraine. Headache decreased with age. SB significantly influenced the presence of primary headache (TTH and migraine) (OR = 3.12/P = .001).	SB is associated with primary headache (TTH and migraine).
Fernandes et al[7]	TMD and Orofacial Pain Clinic at Araraquara School of Dentistry, Brazil	286 consecutive subjects (mainly women)	18-76 (mean 37.3)	To investigate the association among TMD, SB, and primary headaches (episodic and chronic migraine and episodic TTH).	ICHD-II	Chi-square Odds ratio tests 95% confidence interval, significance level .05	Prevalence of SB was higher in individuals with headaches (74.6% in CM, 58.2% in EM, 56.1% in ETTH) than individuals without headaches (43.7%). The association between headaches and SB was significant only for CM (OR = 3.8/P = .0005)	The presence of SB significantly increased the risk for CM.

CM = chronic migraine; EM = episodic migraine; ETTH = episodic tension-type headache; ICHD = International Classification of Headache Disorders; ICHD-II = International Classification of Headache Disorders, 2nd Edition; OR = odds ratio; SB = sleep bruxism; TMD = temporomandibular disorders; TTH = tension-type headache.

Table 2.—Quality in Prognostic Studies (QUIPS) Risk of Bias Assessment

Studies Biases	Troeltzch et al[6] Rating of "Risk of Bias"	Fernandes et al[7] Rating of "Risk of Bias"
1. Study participation	Low	Moderate
2. Study attrition	Moderate	High
3. Prognostic factor measurement	Low	Low
4. Outcome measurement	Low	Low
5. Study confounding	High	Low
6. Statistical analysis and reporting	Moderate	Low

between SB and EM and ETTH was not statistically significant.

A meta-analysis was not conducted because the available data were not adequate. One author[6] was contacted to clarify some of the reported data. However, even with the additional data provided, it was not justifiable to conduct a meta-analysis.

Risk of Bias Across Studies.—The selected studies[6,7] used similar diagnostic methods, which reduced the possibility of misdiagnosis. They were considered homogeneous, but they did not present adequate data for assessment of risk of bias across the included studies.

DISCUSSION

Summary of Evidence.—In this systematic review, we investigated the potential association between TTH and migraine with SB. Several studies have suggested this association, but most them did not classify the headaches..[13,14,18-20,22,29-34] This predicated association between headaches and SB is probably one of the most debated issues concerning SB effects. Although the first studies supporting this argument date back to more than 50 years ago,[35-37] conclusive evidence has yet to be found. Several methodological limitations in the published studies may have contributed significantly to the current inconclusiveness in this regard.

In this systematic review, the 2 finally selected studies[6,7] reported a significant association between SB and TTH and migraine. Troeltzsch et al[6] reported that the presence of SB significantly increased the OR for primary headaches (TTH and migraine). Fernandes et al[7] found that the presence of SB significantly increased the OR for CM. Although both studies had similar OR values (3.8 and 3.12), the apparent consistency of findings needs to be analyzed with caution. The study of Troeltzsch et al[6] showed that SB influenced the presence of primary headaches (migraine and TTH). The study of Fernandes et al[7] showed that SB only increased the risk for CM, not of primary headaches overall. In other words, it was associated with increased headache frequency among individuals with migraine. Because the study of Troeltzsch et al[6] was done following the ICHD-1, which did not provide diagnostic criteria for CM, and because the study did not report headache frequencies, it is possible that some of the individuals with TTH and migraine would now be classified as having CM. This is only speculation. In summary, although both studies agree that SB increased by 3-fold the odds of headaches, they report association with different type of headache. It is important to emphasize that both studies did not include children younger than 14 years old.

The findings reported by Troeltzsch et al[6] and Fernandes et al[7] agree with previous studies that tried to demonstrate the association between headaches and SB.[13-15,17-22,29,32,38,39] Also, the reported OR in both studies was closer OR values from Carra et al[18] (4.3). However, this study[18] was excluded because it did not meet the minimum criteria for SB and for headache diagnosis.

Unfortunately, most of the studies preselected were later excluded because they did not meet the minimum criteria for SB diagnosis,[13-15,17-22,29-32] and/or headache diagnosis.[13,14,18-20,22,29-34] The most striking limitations of these studies are represented by the subjectivity of the self-report diagnosis of headaches and/or bruxism, and by the lack of information on whether they are studying sleep or awake bruxism. In this regard, it is frustrating that a consensus is still missing regarding a definition and a diagnostic grading system for bruxism, bearing in mind that this is a prerequisite for evidence-based practice management.[40]

The SB diagnosis based on polysomnographic recordings is very well documented and accepted by most clinicians and researchers as the gold standard.[41] Unfortunately, overnight polysomnography (PSG) sleep studies are onerous, labor intensive, and expensive. Access is also significantly limited in some places. Waiting times between referral for evaluation to diagnosis commonly take 5-6 months across the United States and around the world.[42] In fact, in clinical dentistry, polysomnographic recordings are rarely made.[43] SB diagnosis is usually clinical (although the gold standard remains a full-night PSG).[5] In our study, we used a tool to SB diagnosis proposed by the AASM.[27] The AASM is a well-recognized professional organization that developed this consensus-based set of criteria. In fact, we only selected studies that met this classification criterion regardless of whether the authors actually mentioned specifically the use of this classification system. In the second phase of the selection process, 13 articles were excluded because they failed to meet the AASM diagnosis criteria.[27]

Furthermore, across studies that were excluded, the instruments employed to measure TTH and migraine had questionable properties because almost all were not previously validated. Prior to 1988, there were no systematic or internationally recognized criteria for the evaluation and diagnosis of headaches.[44] The IHS published the first ICHD in 1988,[24] the second edition (ICHD-II) in early 2004,[25] and the third edition (ICHD-3 beta) was published in 2013.[26] The diagnostic criteria and headache classifications are based on clinical and epidemiologic research as well as expert consensus. Although all classification systems have limitations when applied to clinical practice, the ICHD provides a tool by which headaches can be diagnosed, studied, and discussed in a consistent manner throughout the international research and academic community.[44]

Some mechanisms have been described to justify the association between SB and TTH and migraine. Past pain models for TTH suggest that nociceptive inputs from peripheral tender muscles can lead to central sensitization and chronic TTH (CTTH) conditions. Such models support that possible peripheral mechanisms leading to pericranial tenderness include activation or sensitization of nociceptive nerve endings by liberation of chemical mediators.[45] Based on available data, an updated pain model for CTTH was proposed by Fernandez-de-las-Penas et al,[45] in which headache could be at least partly explained by referred pain from trigger points (TrPs) in the posterior cervical, head, and shoulder muscles. In this updated pain model, TrPs would be the primary hyperalgesic zones responsible for the development of central sensitization in CTTH.[45] Bruxism may also be an important contributing factor for the development of TrPs in the head and neck, which in turn generate and/or contribute to TTH and myofascial headache.[22] Related to this hypothesis, Glaros et al[46] found that patients with headaches reported significantly more frequent and intense tooth contact, more masticatory muscle tension, more stress, and more pain in their face/head than controls without headaches.[46] These hypotheses appear to be solid, especially to explain the association between SB and TTH.

Regarding migraine, the most plausible hypothesis for the association between SB and migraine is that nociceptive inputs from the masticatory muscle enhance the excitability of the trigeminal subnucleus caudalis nociceptive neurons, increasing the risk for migraine attacks. Bruxism-induced muscular changes could also lead to central sensitization of trigeminal subnucleus caudalis nociceptive neuron, which is associated with increased headache frequency among individuals with migraine.[47,48] Free nerve endings in peripheral tissues provide the peripheral basis for pain. Many of these free nerve endings act as nociceptors, and their activation may result in the production of nerve impulses. The masticatory muscles and the temporomandibular joint contain numerous free nerve endings. It is clear that the endings may respond to a wide range of peripheral stimuli that cause pain in humans.[47]

In summary, this systematic review showed that in adults, there is a significant association (more than three times) between TTH and migraine with SB. Because of this, a patient at risk should be referred to a dentist in order to assess the presence of SB and, if present, identify the best therapeutic approach for symptom management. Considering that SB is a

disorder of significant relevance to the practicing neurologist, because SB patients frequently complaint about headaches,[18,22] a close interaction between neurologists and dentists is strongly recommended when evaluating and managing patients suffering from facial pain and primary headaches.[7] Ultimately, SB is a dynamic behavior that concerns medical, dental, and psychological health care domains and may be an important behavior to consider within a multidisciplinary health care context.[49] Therefore, multidisciplinary teams should be constituted to develop research and clinical strategies to diagnose and treat these patients.

Limitations.—Some methodological limitations of this review should be considered. First, only 2 articles met the inclusion criteria. Second, we did not find articles in children younger than 14 years old.

Regarding the available evidence, in one study[7] the sample consisted of patients who sought treatment for temporomandibular disorder (TMD), and results may not be representative of the general population. The reduced numbers of individuals free of primary headaches, SB, and TMD probably tended to increase the overall OR. Similarly, in the other study[6] no systematic process of randomization was conducted; thus, there was a risk of selection bias. Both were cross-sectional studies, determining the state of the patient at only one point in time; thus, a cause-and-effect relationship could not be made.

Methodologically stronger research in these populations is warranted and strongly needed, especially in children.

CONCLUSION

There is not enough scientific evidence to either support or refute the association between TTH and migraine with SB in children. Adults with SB appear to be more likely to have headache.

STATEMENT OF AUTHORSHIP

Category 1

(a) Conception and Design
Graziela De Luca Canto; Marcelo E. Bigal; Paul W. Major; Carlos Flores-Mir

(b) Acquisition of Data
Graziela De Luca Canto; Vandana Singh; Marcelo E. Bigal; Carlos Flores-Mir

(c) Analysis and Interpretation of Data
Graziela De Luca Canto; Vandana Singh; Marcelo E. Bigal; Paul W. Major; Carlos Flores-Mir

Category 2

(a) Drafting the Manuscript
Graziela De Luca Canto; Vandana Singh; Paul W. Major; Marcelo E. Bigal; Carlos Flores-Mir

(b) Revising It for Intellectual Content
Marcelo E. Bigal; Paul W. Major; Carlos Flores-Mir

Category 3

(a) Final Approval of the Completed Manuscript
Marcelo E. Bigal; Paul W. Major; Carlos Flores-Mir

REFERENCES

1. Cady R, Schreiber C, Farmer K, Sheftell F. Primary headaches: A convergence hypothesis. *Headache*. 2002;42:204-216.
2. Stovner LJ, Andree C. Prevalence of headache in Europe: A review for the Eurolight project. *J Headache Pain*. 2010;11:289-299.
3. Matilde Leonardi M, Steiner TJ, Scher AT, Lipton RB. The global burden of migraine: Measuring disability in headache disorders with WHO's Classification of Functioning, Disability and Health (ICF). *J Headache Pain*. 2005;6:429-440.
4. Gonçalves DA, Camparis CM, Speciali JG, Franco AL, Castanharo SM, Bigal ME. Temporomandibular disorders are differentially associated with headache diagnoses: A controlled study. *Clin J Pain*. 2011;27:611-615.
5. Carra MC, Huynh N, Lavigne G. Sleep bruxism: A comprehensive overview for the dental clinician interested in sleep medicine. *Dent Clin North Am*. 2012;387-413.
6. Troeltzsch M, Troeltzsch M, Cronin RJ, Brodine AH, Frankenberger R, Messlinger K. Prevalence and association of headaches, temporomandibular joint disorders, and occlusal interferences. *J Prosthet Dent*. 2011;105:410-417.

7. Fernandes G, Franco AL, Goncalves DA, Specialli JG, Bigal ME, Camparis CM. Temporomandibular disorders, sleep bruxism, and primary headaches are mutually associated. *J Orofac Pain*. 2013;27:14-20.
8. Koyano K, Tsukiyama Y, Ichiki R, Kuwata T. Assessment of bruxism in the clinic. *J Oral Rehabil*. 2008;35:495-508.
9. Bader G, Lavigne GJ. Sleep bruxism: Overview of an oromandibular sleep movement disorder. *Sleep Med Rev*. 2000;4:27-43.
10. Lavigne G, Manzini C, Huynh NT. Sleep bruxism. In: Kryger MH, Roth T, Dement WC, eds. *Principles and Practice of Sleep Medicine*, 5th edn. St. Louis, MO: Elsevier Saunders; 2011:1129-1139.
11. Lavigne GJ, Montplaisir JY. Restless legs syndrome and sleep bruxism: Prevalence and association among Canadians. *Sleep*. 1994;17:739-743.
12. Manfredini D, Winocur E, Guarda-Nardini L, Lobbezoo F. Epidemiology of bruxism in adults: A systematic review of the literature. *J Orofac Pain*. 2013;27:99-110.
13. Aromaa M, Sillanpaa ML, Rautava P, Helenius H. Childhood headache at school entry: A controlled clinical study. *Neurology*. 1998;50:1729-1736.
14. Wanman A, Agerberg G. Headache and dysfunction of the masticatory system in adolescents. *Cephalalgia*. 1986;6:247-255.
15. Zarowski M, Mlodzikowska-Albrecht J, Steinborn B. The sleep habits and sleep disorders in children. *Adv Med Sci*. 2007;52(Suppl. 1):194-196.
16. Vendrame M, Kaleyias JK, Valencia I, Legido A, Kothare SV. Polysomnographic findings in children with headaches. *Pediatr Neurol*. 2008;39:6-11.
17. Yaghini O, Amir Shahkarami SM, Mahmoodian T, Haj Hashemi E. Comprehensive assessment of the relative frequency of sleep disorders in migraine and non-migraine in 6- to 14-year-old children. *Iranian J Child Neurol*. 2010;4:33-36.
18. Carra MC, Huynh N, Morton P, et al. Prevalence and risk factors of sleep bruxism and wake-time tooth clenching in a 7- to 17-yr-old population. *Eur J Oral Sci*. 2011;119:386-394.
19. Godoy ME, Rossi E, Garcia JP. The role of bruxism in chronic headache. *Odontol Chil*. 1982;30:33-36.
20. Villarosa GA, Moss RA. Oral behavioral patterns as factors contributing to the development of head and facial pain. *J Prosthet Dent*. 1985;54:427-430.
21. Costa AL, D'Abreu A, Cendes F. Temporomandibular joint internal derangement: Association with headache, joint effusion, bruxism, and joint pain. *J Contemp Dent Pract*. 2008;9:9-16.
22. Molina OF, Peixoto MG, Eid NLM, Aquilino RN, Rank RCIC. Headache and bruxing behavior types in craniomandibular disorders (CMDs) patients. *Rev Neurocien*. 2011;19:449-457.
23. Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: The PRISMA Statement. *PLoS Med*. 2009;151:264-269.
24. Headache Classification of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. *Cephalalgia*. 1988;8(Suppl. 7):1-96.
25. Headache Classification of the International Headache Society. The International Classification of Headache Disorders, 2nd ed. *Cephalalgia*. 2004; 24(Suppl. 1):1-160.
26. Headache Classification of the International Headache Society. The International Classification of Headache Disorders, 3rd ed. *Cephalalgia*. 2013; 33:629-808.
27. American Academy of Sleep Medicine (AASM) ed. *International Classification of Sleep Disorders. Diagnosis and Coding Manual. Section on Sleep Related Bruxism*, 2nd edn. Darien, IL: American Academy of Sleep Medicine; 2005.
28. Hayden J, Windt DA, van der Cartwright JL, Cote P, Bombardier C. Assessing bias in studies of prognostic factors. *Ann Intern Med*. 2013;158:280-286.
29. Magnusson T, Carlsson GE. Recurrent headaches in relation to temporomandibular joint pain-dysfunction. *Acta Odontol Scand*. 1978;36:333-338.
30. Wieselmann G, Grabmair W, Logar C, Perman R, Flooh E, Lechner H. EEG studies in bruxism with headache patients. *EEG EMG Z Elektroenzephalographie Elektromyogrraphie U Verwandte Gebbiete*. 1987;18:135-137.
31. Sponholz H, Dalisda D. About the correlation between bruxism and chronic headache. *Dtsch Stomatol*. 1991;41:226-22833.
32. Steele JG, Lamey PJ, Sharkey SW, Smith GM. Occlusal abnormalities, pericranial muscle and joint tenderness and tooth wear in a group of migraine patients. *J Oral Rehabil*. 1991;18:453-458.
33. Molina OF, dos Santos J, Nelson SJ, Nowlin T. A clinical study of specific signs and symptoms of CMD in bruxers classified by the degree of severity. *Cranio*. 1999;17:268-279.

34. Molina OF, dos Santos Junior J, Nelson SJ, Nowlin T. Profile of TMD and bruxer compared to TMD and nonbruxer patients regarding chief complaint, previous consultations, modes of therapy, and chronicity. *Cranio*. 2000;18:205-219.
35. Monica WS. Preauricular and head pain caused by bruxism. *Ann Otol Rhinol Laryngol*. 1964;73:170-172.
36. Monica WS. Bruxism as a cause of headaches. *Med Times*. 1960;88:336-337.
37. Berlin R, Dessner L. Bruxism and chronic headache. *Lancet*. 1960;6:289-291.
38. Wanman A, Agerberg G. Two-year longitudinal study of symptoms of mandibular dysfunction in adolescents. *Acta Odontol Scand*. 1986;44:321-331.
39. Widmalm SE, Gunn SM, Christiansen RL, Hawley LM. Association between CMD signs and symptoms, oral parafunctions, race and sex, in 4-6-year-old African-American and Caucasian children. *J Oral Rehabil*. 1995;22:95-100.
40. Lobezzo F, Ahlberg J, Glaros AG, et al. Bruxism defined and graded: An international consensus. *J Oral Rehabil*. 2013;40:2-4.
41. Lavigne G, Rompre PH, Montplaisir JY. Sleep bruxism: Validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J Dent Res*. 1996;75:546-552.
42. Gozal D. Serum, urine and breath–related biomarkers in the diagnosis of obstructive sleep apnea in children: Is it for real? *Sleep Resp Neurob*. 2012;18:561-567.
43. Sjoholm TT, Lowe AA, Miyamoto K, Fleetham JA, Ryan CF. Sleep bruxism in patients with sleep-disordered breathing. *Arch Oral Biol*. 2000;45:889-896.
44. Biondi DM. Classification and diagnosis of headache disorders. *Semin Pain Med*. 2004;2:54-61.
45. Fernandez-de-las-Penas C, Cuadrado ML, Arendt-Nielsen L, Simons DG, Pareja JA. Myofascial trigger point and sensitization: An update pain model for tension-type headache. *Cephalalgia*. 2007;27:383-393.
46. Glaros AG, Urban D, Locke J. Headache and temporomandibular disorders: Evidence for diagnostic and behavioural overlap. *Cephalalgia*. 2007;27:542-549.
47. Sessle B. The neural basis of temporomandibular joint and masticatory muscle pain. *J Orofac Pain*. 1999;13:238-245.
48. Goncalves D, Bigal ME, Jales LCF, Camparis C, Speciali JG. Headache and symptoms of temporomandibular disorder: An epidemiological study. *Headache*. 2009;50:231-241.
49. Insana SP, Gozal D, McNeil DW, Montgomery-Downs HE. Community based study of sleep bruxism during early childhood. *Sleep Med*. 2013;14:183-188.

SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Appendix S1.—Search.

Appendix S2.—Articles excluded and the reasons for exclusion.

Appendix S3.—Quality in prognostic studies (QUIPS) risk of bias assessment instrument for prognostic factor studies.