Headache, Temporomandibular Disorders, and Bruxism: Uncovering Connections

Pain and dysfunction of the temporomandibular joints and associated structures can be a source of headache and orofacial pain.

BY STEVEN D. BENDER, DDS

A 29-year-old female presented with a six-and-a-half year history of daily, migraine-like headaches. She wanted to find out if her temporomandibular joint "problems" contributed in any way to her daily head pain. She indicated being aware of sleep bruxism since high school and reported utilizing a dental splint since 2000.

Prior to her consultation in our office, she had previously consulted with an acupuncturist, general dentist, oral surgeon, chiropractor, endocrinologist, internist, neurologist, physical therapist, pain medicine physician, psychologist and psychiatrist. Previous therapies included medication; both abortive and prophylactic, inpatient treatment at a nationally known headache center, nerve blocks, rhizotomies, cryotherapy and a dental splint. She also reported PFO closure in 2008. She indicated that the most robust relief resulted from cryotherapy but it would only last for approximately two months before the pain returned to the previous baseline level.

Of significance in her medical history was hypothyroidism and treated depression. Her current medications included; thyroid, Celexa, Xanax, fenofibrate, fish oil, a multivitamin, B-complex, Atarax, Phenergan and Zofran.

TAKE HOME TIPS

Headache and temporomandibular disorders (TMDs) are very prevalent conditions in the general population. TMDs are defined as a collection of symptoms and signs involving masticatory muscles, the temporomandibular joints (TMJs) or both. The pain reported by TMD patients is typically located in the muscles of mastication, in the preauricular area, or in the temporomandibular joint (TMJ).

Clinical and epidemiological studies have demonstrated an association between headache and TMDs, suggesting that individuals with headache and those with TMDs, often share similar signs and symptomatic presentations.

Patients with sleep bruxism (SB) patient may not always demonstrate significant pain in the TMDs and associated structures. SB may produce subclinical nociceptive signaling from the stomatagnathic structures to the trigeminal nucleus increasing central sensitization. Recognition, evaluation and effective management of the SB patient has the potential to increase headache treatment efficacy and potentially reduce the need for pharmacotherapy.

Her pain presentation was continuous and bilateral involving the temples, occiput, periocular and neck areas.

She indicated her pain would vary in presentation and she used the descriptors; throbbing, dull, stabbing, pressure, burning and shooting. Along with her pain she indicated nausea, vomiting, photophobia, phonophobia and osmophobia. She gave no history of aura. A family history of headaches included her mother, sisters and maternal grandmother.

Physical examination revealed a normal neurologic screening, BMI of 33.1, normal pulses and afebrile. She was alert and oriented in all spheres. Her ENT screening was also normal. Opthalmoscopic exam revealed grossly normal fundi.

There was mild pain to palpation of the masseter and temporalis muscles bilaterally as well as the left cervical spine. Mild pain was also reported to palpation of the right and left occipital nerve areas. Her intraoral examination was normal as was her mandibular range of motion. There was evidence of tooth wear as well as scalloping of the tongue lateral borders and ridging of the buccal mucosa noted. No pain was reported at the right or left TMJ nor was there pain reported with mandibular movement.

Based on her history of sleep bruxism as well as signs and symptoms of such, the decision was made to fabricate a custom intraoral splint for sleep. After approximately two months of nightly wear, she reported a decrease in her headache severity although she still had daily pain. At 10 months, her headaches had completely resolved and she remains headache free at 18 months.

QUESTION

When is appropriate to consider bruxism and the TMJs in the refractory headache patient?

EXPERT ANALYSIS

Headache and temporomandibular disorders (TMDs) are very prevalent conditions in the general population.[1-8] TMDs are defined as a collection of symptoms and signs involving masticatory muscles, the temporomandibular joints (TMJs) or both.[9] The pain reported by TMD patients is typically located in the muscles of mastication, in the preauricular area, or in the temporomandibular joint (TMJ).[10]

Clinical and epidemiological studies have demonstrated an association between headache and TMDs, suggesting that individuals with headache and those with TMDs, often share similar signs and symptomatic presentations.[8,11,12] Inflammatory mechanisms have been shown to be involved in temporomandibular joint pain and dysfunction.[13] Milam proposed a possible etiology for inflammatory mechanisms of the temporomandibular joint structures by what was described as a hypoxic-reperfusion injury.[14] This process occurs when the capsular pressure of the temporomandibular joint exceeds the end-capillary perfusion blood pressure of the feeding vasculature. The area then undergoes reperfusion via mouth opening or relaxation of the elevator muscles. It was speculated the reperfusion resulted in the release of free radicles and initiated inflammatory processes. Capsular nociceptive fibers triggered by pathologic loading of the highly innervated synovial tissues may also stimulate the release of calcitonin gene-related peptide and substance P, leading to further inflammatory processes. Pathologic loading is often attributed to sleep parafunctional behaviors such as sleep bruxism (SB).[15] Sleep bruxism may also be responsible for stomatagnathic muscle nociceptive signaling. Christensen reported that muscle pain was noted in subjects who voluntarily clenched for 20-30 seconds.[16] Kydd and Daly reported that nocturnal clenching events can last as long as 20-40 seconds.[17] These sustained isometric contractions observed in sleep bruxism could lead to tissue injury and subsequent nociceptive signaling from both the myogenous and arthrogenous components of the temporomandibular joint complex. Previous investigations have also described mechanisms for nociceptive referral from the temporomandibular joints (TMJs) to the cranial structures.[18,19]

A CLOSER LOOK AT BRUXISM

Bruxism may be classified as:

- Awake bruxism, usually but not always diurnal or
- Sleep bruxism (SB), usually but not always nocturnal

Both types of bruxism are either

- Primary (idiopathic); no associated medical condition, or
- Secondary (iatrogenic); associated medical condition.

Overall prevalence of awake clenching is about 20 percent in adults.

Approximately 60 percent of "normal" sleepers exhibit rhythmic masticatory muscle activity (RMMA) during sleep.

WHAT IS BRUXISM?

The American Academy of Orofacial Pain defines bruxism as diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing, and grinding of the teeth.[20] Bruxism may be classified as awake bruxism (usually but not always diurnal) or sleep bruxism (SB) (usually but not always nocturnal). Both types of bruxism are either primary (idiopathic), in which case there is no associated medical condition, or secondary (iatrogenic), in which case there is an associated medical condition. Awake bruxism mostly involves teeth clenching or tapping and jaw bracing, with or without tooth contact.[21] Grinding is rarely noted during

waking hours. The overall prevalence of awake clenching is about 20 percent in the adult population, with more women reporting clenching awareness than men.[21] As it appears that SB differs in etiopathology from awake oromandibular parafunctional activities, care should be taken to differentiate the two as different entities.[22] In 2005, the International Classification of Sleep Disorders classified sleep bruxism as a "sleep-related movement disorders."[23] Previously it had been termed a "parasomnia," or a disorder of arousal. Sleep related movement disorders are considered simple, stereotypic, repetitive, and localized movements during sleep that also includes periodic limb movement disorder and rhythmic movement disorder.[24] Although SB is comprised of rhythmic, repetitive mandibular movements, it can also involve forceful, as well as prolonged, clenching of the dentition. Nishigawa and colleagues demonstrated that bite force during SB can exceed the amplitude of maximum voluntary bite force during the daytime by as much as 111.6 percent.[25] Up to 65 percent of SB patients of all ages report frequent headaches.[26,27] Risk factors that have been shown to exacerbate SB are:

1. smoking, caffeine and heavy alcohol consumption;[28, 29]
2. type A personality and/or anxiety;[28,30-32]
3. sleep-related breathing disorders;[28,33] and
4. periodic limb movements.[28, 34, 35]

Due to the continued disagreement about the definition and diagnosis of this SB,[36] the literature on the subject is often difficult to interpret. Currently, there is no single clear pathophysiologic mechanism identified as responsible for SB. Studies have demonstrated that approximately 60 percent of "normal" sleepers exhibit what is known as rhythmic masticatory muscle activity (RMMA) during sleep.[37] RMMA is defined as three masseter muscle bursts or contractions within an episode, in the absence of teeth grinding.[37] This type of oromotor activity corresponds to the chewing automatism previously described by Halasz[38] and is not necessarily correlated with tooth grinding. The frequency of RMMA during sleep is three times greater in SB subjects.[37] RMMA can be induced seven times more frequently in SB patients, suggesting that SB is probably related to a heightened responsiveness to transient arousal during sleep.[39] Also, many of these experimentally induced arousals were accompanied by tooth-grinding. In SB subjects, episodes occur at a frequency of 5.8 times per hour of sleep as compared to 1.8 episodes for non-SB individuals.[37] Grinding noises are reported to occur in approximately 44 percent of SB/RMMA events.[40,41] SB episodes tend to occur primarily in non-rapid eye movement sleep (NREM) stages 1 and 2 (light sleep) with only 10 percent occurring during rapid eye movement sleep (REM).[37,42,43] SB episodes appear more in the second and third NREM to REM sleep cycles as compared to the first and fourth cycles.[44] Also, SB episodes will occur more frequently in the ascending period of sleep within a cycle.[44] Ascending into lighter sleep has been shown to be associated with an increase in sympathetic tone and in arousal activity.[45,46] SB has been demonstrated to occur subsequent to a sequence of physiological events that consists of an increase in sympathetic nervous system activity, cortex activation, heart rate increase, and ultimately an increase in jaw depressor muscle activity.[47] Eighty-eight of the time, SB episodes tend to occur along with cyclic alternating patterns (CAPs).[42]

CAPs consist of activation of electroencephalogram (EEG) and electrocardiogram (EKG or ECG) patterns and occur approximately every 20–60 seconds during non-REM sleep.[48] These events are thought to be physiologic events that support sleep quality. Some of the suggested causes for sleep bruxism include; stress and anxiety, occlusal factors, genetics, sleep related breathing disorders and neurochemical factors.

EVALUATION

The most widely used and accepted method for evaluating the condition of the stomatagnathic musculature is by digital palpation.[49-51] Application of about four to five pounds of pressure (the pressure necessary to blanch the finger nail bed) applied with the palmer surface of the index, middle and fore fingers across the muscle fibers can be diagnostic of muscular abnormalities.[52] The examination should identify tender areas as well as potential trigger points, which are thought to be resultant from abnormal motor end-plate activity releasing excessive amounts of acetylcholine.[53] A cursory stomatagnathic muscle examination would include the following muscle groups; temporalis, masseter, sternocleidomastoid, splenius capitis, semispinalis capitis and the anterior portion of the trapezius muscle. The lateral pterygoid muscle, involved in opening and protruding the mandible, must be functionally assessed as it is not possible to manually palpate this muscle.[54,55]

The parafunctioning patient may not necessarily present with painful masticatory symptoms. Examination of the oral structures may reveal worn dentition as well as scalloping of the oral tongue lateral borders and ridging of the buccal mucosa.[56-59]

The temporomandibular joint can also be assessed by digital palpation. The location of the mandibular condyle can be identified in the area anterior to the tragus of the ear by having the patient open and close several times and feeling for the movement of the lateral aspect. It is important to have the patient then clench their teeth in order to ensure proper positioning of the finger tips. If muscle contraction is felt, it is probable that the fingers are resting on the area of the deep portion of the masseter muscle and not the lateral

aspect of the condyle. Joint popping or crepitation can also be assessed by light digital palpation or by using the bell end of a standard stethoscope.

CONCLUSION

Temporomandibular disorders include a variety of musculoskeletal disorders that may affect mandibular function. Pain and dysfunction of the temporomandibular joints and associated structures can be a source of headache and orofacial pain. However, the SB patient may not always demonstrate significant pain in these structures. SB may produce subclinical nociceptive signaling from the stomatagnathic structures to the trigeminal nucleus increasing central sensitization. Recognition, evaluation and effective management of the SB patient has the potential to increase headache treatment efficacy and potentially reduce the need for pharmacotherapy, especially in those who present as refractory to the traditional treatment protocols. ■

Reprinted with permission from "Broca's Area," the newsletter of the Texas Neurological Society.

Steven D. Bender, DDS is a Fellow of both the American Headache Society and American Academy of Orofacial Pain. He is Director of The North Texas Center for Head, Face & TMJ Pain in Plano, TX.

1. Scher AI, Stewart WF, Liberman J, Lipton RB. Prevalence of frequent headache in a population sample. Headache 1998;38(7):497-506.
2. Stovner L, Hagen K, Jensen R, et al. The global burden of headache: a documentation of headache prevalence and disability worldwide. Cephalalgia 2007;27(3):193-210.
3. Schwartz BS, Stewart WF, Simon D, Lipton RB. Epidemiology of tension-type headache. JAMA 1998;279(5):381-3.
4. Stewart WF, Shechter A, Rasmussen BK. Migraine prevalence. A review of population-based studies. Neurology 1994;44(6 Suppl 4):S17-23.
5. Jensen R, Rasmussen BK, Pedersen B, Lous I, Olesen J. Prevalence of oromandibular dysfunction in a general population. Journal of orofacial pain 1993;7(2):175-82.
6. LeResche L. Epidemiology of temporomandibular disorders: implications for the investigation of etiologic factors. Critical reviews in oral biology and medicine : an official publication of the American Association of Oral Biologists 1997;8(3):291-305.
7. Lipton JA, Ship JA, Larach-Robinson D. Estimated prevalence and distribution of reported orofacial pain in the United States. Journal of the American Dental Association 1993;124(10):115-21.
8. Ciancaglini R, Radaelli G. The relationship between headache and symptoms of temporomandibular disorder in the general population. J Dent 2001;29(2):93-8.
9. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. J Craniomandib Disord 1992;6(4):301-55.
10. McNeill C, Mohl ND, Rugh JD, Tanaka TT. Temporomandibular disorders: diagnosis, management, education, and research. Journal of the American Dental Association 1990;120(3):253, 55, 57 passim.
11. Glaros AG, Urban D, Locke J. Headache and temporomandibular disorders: evidence for diagnostic and behavioural overlap. Cephalalgia 2007;27(6):542-9.
12. Pettengill C. A comparison of headache symptoms between two groups: a TMD group and a general dental practice group. Cranio 1999;17(1):64-9.
13. Graff-Radford SB. Temporomandibular disorders and headache. Dental clinics of North America 2007;51(1):129-44, vi-vii.
14. Milam SB, Zardeneta G, Schmitz JP. Oxidative stress and degenerative temporomandibular joint disease: a proposed hypothesis. J Oral Maxillofac Surg 1998;56(2):214-23.
15. Hirose M, Tanaka E, Tanaka M, et al. Three-dimensional finite-element model of the human temporomandibular joint disc during prolonged clenching. European journal of oral sciences 2006;114(5):441-8.
16. Christensen LV. Facial pain from experimental tooth clenching. Tandlaegebladet 1970;74(2):175-82.
17. Kydd WL, Daly C. Duration of nocturnal tooth contacts during bruxing. J Prosthet Dent 1985;53(5):717-21.
18. Cairns BE, Sessle BJ, Hu JW. Evidence that excitatory amino acid receptors within the temporomandibular joint region are involved in the reflex activation of the jaw muscles. The Journal of neuroscience : the official journal of the Society for Neuroscience 1998;18(19):8056-64.
19. Thalakoti S, Patil VV, Damodaram S, et al. Neuron-glia signaling in trigeminal ganglion: implications for migraine pathology. Headache 2007;47(7):1008-23; discussion 24-5.
20. Leeuw d, editor. Orofacial Pain: Guidlines for assesmment, diagnosis and management. Fourth ed. Hanover Park, IL: Quintessence Publishing Company; 2008.
21. Shetty S, Pitti V, Satish Babu CL, Surendra Kumar GP, Deepthi BC. Bruxism: a literature review. J Indian Prosthodont Soc 2010;10(3):141-8.
22. Okeson J. Orofacial Pain Guidelines for Assessment, Diagnosis,and Management; Chicago: Quintessence; 1996.
AASM. International Classification of Sleep Disorders. 2nd ed. Westchester: American Academy of Sleep Medicine; 2005.
24. Walters AS. Clinical identification of the simple sleep-related movement disorders. Chest 2007;131(4):1260-6.
25. Nishigawa K, Bando E, Nakano M. Quantitative study of bite force during sleep associated bruxism. Journal of oral rehabilitation 2001;28(5):485-91.
26. Bader GG, Kampe T, Tagdae T, Karlsson S, Blomqvist M. Descriptive physiological data on a sleep bruxism population. Sleep 1997;20(11):982-90.
27. Camparis CM, Siqueira JT. Sleep bruxism: clinical aspects and characteristics in patients with and without chronic orofacial pain. Oral surgery, oral medicine, oral pathology, oral radiology, and endodontics 2006;101(2):188-93.
28. Ohayon MM, Li KK, Guilleminault C. Risk factors for sleep bruxism in the general population. Chest 2001;119(1):53-61.
29. Lavigne GL, Lobbezoo F, Rompre PH, Nielsen TA, Montplaisir J. Cigarette smoking as a risk factor or an exacerbating factor for restless legs syndrome and sleep bruxism. Sleep 1997;20(4):290-3.
30. Major M, Rompre PH, Guitard F, et al. A controlled daytime challenge of motor performance and vigilance in sleep bruxers. Journal of dental research 1999;78(11):1754-62.
31. Pingitore G, Chrobak V, Petrie J. The social and psychologic factors of bruxism. J Prosthet Dent 1991;65(3):443-6.
32. Pierce CJ, Chrisman K, Bennett ME, Close JM. Stress, anticipatory stress, and psychologic measures related to sleep bruxism. Journal of orofacial pain 1995;9(1):51-6.
33. Sjoholm TT, Lowe AA, Miyamoto K, Fleetham JA, Ryan CF. Sleep bruxism in patients with sleep-disordered breathing. Archives of oral biology 2000;45(10):889-96.
34. Lavigne GJ, Montplaisir JY. Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. Sleep 1994;17(8):739-43.
35. Bader G, Lavigne G. Sleep bruxism; an overview of an oromandibular sleep movement disorder. REVIEW ARTICLE. Sleep medicine reviews 2000;4(1):27-43.
36. Lavigne GJ, Goulet JP, Zuconni M, Morrison F, Lobbezoo F. Sleep disorders and the dental patient: an overview. Oral surgery, oral medicine, oral pathology, oral radiology, and endodontics 1999;88(3):257-72.
37. Lavigne GJ, Rompre PH, Poirier G, et al. Rhythmic masticatory muscle activity during sleep in humans. Journal of dental research 2001;80(2):443-8.
38. Halasz P, Ujszaszi J, Gadoros J. Are microarousals preceded by electroencephalographic slow wave synchronization precursors of confusional awakenings? Sleep 1985;8(3):231-8.
39. Kato T, Montplaisir JY, Guitard F, et al. Evidence that experimentally induced sleep bruxism is a consequence of transient arousal. Journal of dental research 2003;82(4):284-8.
40. Reding GR, Zepelin H, Robinson JE, Jr., Zimmerman SO, Smith VH. Nocturnal teeth-grinding: all-night psychophysiologic studies. Journal of dental research 1968;47(5):786-97.
41. Miyawaki S, Lavigne GJ, Pierre M, et al. Association between sleep bruxism, swallowing-related laryngeal movement, and sleep positions. Sleep 2003;26(4):461-5.
42. Macaluso GM, Guerra P, Di Giovanni G, et al. Sleep bruxism is a disorder related to periodic arousals during sleep. Journal of dental research 1998;77(4):565-73.
43. Lavigne GJ MC, Kato T Sleep Bruxism. In: Kryger MH RT, Dement WC, editor. Principles and Practice of Sleep Medicine. Philadelphia: Saunders; 2005. p. 946-59.
44. Huynh N, Kato T, Rompre PH, et al. Sleep bruxism is associated to micro-arousals and an increase in cardiac sympathetic activity. Journal of sleep research 2006;15(3):339-46.
45. Halasz P, Terzano M, Parrino L, Bodizs R. The nature of arousal in sleep. Journal of sleep research 2004;13(1):1-23.
46. Terzano MG, Parrino L, Boselli M, Smerieri A, Spaggiari MC. CAP components and EEG synchronization in the first 3 sleep cycles. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 2000;111(2):283-90.
47. Lavigne GJ, Huynh N, Kato T, et al. Genesis of sleep bruxism: motor and autonomic-cardiac interactions. Archives of oral biology 2007;52(4):381-4.
48. Terzano MG, Parrino L, Spaggiari MC. The cyclic alternating pattern sequences in the dynamic organization of sleep. Electroencephalography and clinical neurophysiology 1988;69(5):437-47.
49. Burch. Occlusion related to craniofacial pain. In: Alling CC MP, editor. Facial Pain. Philadelphia: Lea & Febiger; 1977. p. 165-80.
50. Krogh-Poulsen O. Management of the occlusion of the teeth. In: Schwartz C, editor. Facial pain and mandibular dysfunction. Philadephia: Saunders; 1969. p. 236-80.
51. Schwartz C. The history and clinical examination. In: Schwartz C, editor. Facial pain and mandibular dysfunction. Philadelphia: Saunders; 1969. p. 159-78.
52. Fischer AA. Pressure algometry over normal muscles. Standard values, validity and reproducibility of pressure threshold. Pain 1987;30(1):115-26.
53. Meunier FA, Schiavo G, Molgo J. Botulinum neurotoxins: from paralysis to recovery of functional neuromuscular transmission. Journal of physiology, Paris 2002;96(1- 2):105-13.
54. Turp JC, Minagi S. Palpation of the lateral pterygoid region in TMD--where is the evidence? J Dent 2001;29(7):475-83.
55. Johnstone DR, Templeton M. The feasibility of palpating the lateral pterygoid muscle. J Prosthet Dent 1980;44(3):318-23.
56. Sapiro SM. Tongue indentations as an indicator of clenching. Clin Prev Dent 1992;14(2):21-4.
57. Long RG, Hlousek L, Doyle JL. Oral manifestations of systemic diseases. Mt Sinai J Med 1998;65(5-6):309-15.
58. Gray RJ, Quayle AA, Hall CA, Schofield MA. Physiotherapy in the treatment of temporomandibular joint disorders: a comparative study of four treatment methods. Br Dent J 1994;176(7):257-61.
59. Kampe T, Tagdae T, Bader G, Edman G, Karlsson S. Reported symptoms and clinical findings in a group of subjects with longstanding bruxing behaviour. Journal of oral rehabilitation 1997;24(8):581-7.