

Sleep Bruxism and Pain

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KEYWORDS

• Sleep bruxism • Orofacial pain • Headache

KEY POINTS

- To help the dental community understand the relationship between bruxism and pain, based on the available scientific literature.
- Highlight the evidence that supports different types of treatment management of these type of patients.
- Emphasize the need for further research on this topic with a higher methodological rigor.

INTRODUCTION

Grinding the teeth has been described as an oral behavior that humans engage in related to the perception of pain and anger; it is even reported in the Old Testament of the Bible. This oral behavior was later defined as “bruxism” and this word has its origins in the Greek word *brygmos* (βρυγμός) that means gnashing of the teeth.¹ This behavior was scientifically described for the first time in the early 1900s.²

Today bruxism may be defined as “a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible.”³ Bruxism has 2 distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism [SB]) or during wakefulness (indicated as awake bruxism [AB]).³ These 2 distinct manifestations of bruxism may not be just 2 manifestations of the same entity that occurs in different circadian phases (sleep or awake) but more likely are 2 distinct entities that, even though they may share common risk factors and lead to similar consequences on the masticatory system, may have different etiology and pathophysiology.⁴ Moreover, it has been proposed that bruxism should be diagnosed using a grading system that categorizes the diagnoses based on the methods used into possible (self-report and questionnaires), probable (clinical signs and symptoms), and definitive (polysomnography) bruxism.³

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The scientific knowledge in the field of bruxism has increased largely in recent years with more information being obtained about SB than AB. Despite the progress achieved in the knowledge of bruxism, many questions remain unanswered. With this available knowledge it has been suggested that bruxism can only be defined as an “oral behavior that may lead to harm” and it cannot be labeled as a harmful dysfunction itself (ie, disorder) or even a risk factor for harmful oral health outcomes.⁵ Moreover, the scientific evidence has indicated that the focus of bruxism etiology should be changed from peripheral to central mechanisms⁶ and occlusion can be categorically disregarded as an etiology of bruxism.⁷

In clinical practice, it is common to base conclusions and treatments plans on paradigms supported by evidence that suggests that many different harmful oral health outcomes are reported to be associated with bruxism, such as tooth wear, masticatory muscle tenderness, headaches, and painful temporomandibular disorders (TMDs).^{8–10} This evidence leads to a simplistic cause-effect model based on the assumption that bruxism causes pain due to an overload of the masticatory system and also that higher bruxism muscle activity leads to more pain.¹¹ This article critically examines the current evidence that links pain (TMD and headache) and SB.

BRUXISM AND PAIN: COMPLICATIONS IN DRAWING CONNECTIONS

The challenge in accepting a simplistic cause-effect model is that a large part of the available scientific information is based mainly on studies where self-report (possible bruxism) is the diagnostic method used.^{9,12} At this time, the reliability of the self-report method to determine the real existence of SB has been demonstrated as low,¹³ with a reported sensitivity of 50% and specificity of 73%.⁴ Nevertheless, it is still one of the most used methods (clinically and for research) to diagnose bruxism due to difficult access to gold standard diagnostic methods (polysomnography) that can aid in establishing a definitive bruxism diagnosis (sensitivity of 74% and specificity of 90%).⁴ Despite all the limitations of the self-report method, patients suffering from TMDs often self-report (probable bruxism) more bruxism activity than non-TMD patients.¹⁴ This difference disappears when a diagnosis of SB is confirmed by means of polysomnography (definitive bruxism).¹⁵ As discussed previously, it is currently accepted that bruxism has 2 distinct circadian manifestations—SB and AB³—that may have different etiology and pathophysiology,⁴ therefore making it difficult to validate and interpret the results of studies in which this variable was not taken into account, as evidenced by a published systematic review of headaches and bruxism.¹⁶

Another complication in interpreting the relationship between bruxism and orofacial pain is that changes in classifications of TMDs^{17,18} and headaches¹⁹ make recent results difficult to compare with findings reported using earlier classifications. For example, in cases of a diagnosis of “headache attributed to TMD” and tension-type headache (TTH), it is not clear if a reported headache could actually be “myalgia of the temporalis muscle” and as such should not be given a headache diagnosis but a TMD diagnosis. Furthermore, most of the studies looking at the association between bruxism and headache do not take into account the diagnosis of medication-overuse headache that may cause morning headaches and, according to the literature, makes up 25% to 50% of the chronic headache population.²⁰

Additionally, there are studies that suggest that bruxism may lead to the development of headaches⁸ and also to the development of TMDs.^{12,21} This makes the relationship and limits between bruxism/headache and bruxism/TMD difficult to understand due to their overlap. Furthermore, obstructive sleep apnea (OSA) has been associated with SB²² and also may be related to TMD²³; approximately 50%

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