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# Bruxism - Association to jaw-muscle pain -

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**ABSTRACT** : Jaw-muscle pain associated with temporomandibular disorders (TMD) has traditionally been linked to hyperactivity or abnormal contraction of masticatory muscles such as “bruxism”. A frequent clinical observation has been that many patients with TMD exhibit a tendency to clench or grind their teeth during sleep. It has been considered for a long time that unaccustomed or abnormal contractions of the muscles causes this pain, and once the pain had developed, it causes more muscle hyperactivity, setting up a vicious cycle. However, this theory has not been clarified scientifically and the relationship between various types of orofacial motor activity and TMD is still unclear. This review will discuss the association between bruxism and jaw-muscle pain. All data is based on narrative evidence. The results showed that jaw-muscle activity would evoke significant levels of pain but it is not as prolonged as other temporomandibular disorders.

**Key Words** : bruxism, jaw-muscle pain, temporomandibular disorders, myalgia, orofacial pain

## Introduction

Jaw-muscle pain associated with temporomandibular disorders (TMD) has traditionally been linked to hyperactivity or abnormal contraction of masticatory muscles such as “bruxism”. A frequent clinical observation has been that many patients with TMD exhibit a tendency to clench or grind their teeth. It has been considered for a long time that unaccustomed or abnormal contractions of muscles caused pain, and once pain had developed, it caused more muscle hyperactivity, setting up a vicious cycle. However, this theory has not been clarified scientifically and the relationship between various types of orofacial motor activity and TMD is still unclear.

The aim of this review was to discuss the association of bruxism with jaw-muscle pain. To avoid the confusion, the terms, bruxism and jaw-muscle activity, are carefully distinguished especially in this article.

## 1. Definition of bruxism

The term “bruxism” is often used clinically but with no stringent criteria and may, therefore, have caused some confusion. The following paragraphs will deal with an overview of definitions.

The term “bruxism” is originally derived from the French “la bruxomanie” first used by Marie and Pietkiewicz (1907).<sup>1)</sup> “Bruxism” was regarded as a form of “bruxomania”, which is a purely psychic state, and it was suggested that “bruxism is not necessarily audible”.<sup>2)</sup> Lately, Miller used “bruxomania” to denote habitual grinding of the teeth in the daytime and “bruxism” for nocturnal grinding.<sup>3)</sup> Other terms have been suggested for habitual grinding of the teeth, such as “neuralgia traumatica”,<sup>4)</sup> “Karolyi-effect”,<sup>5)</sup> and “occlusal habit neurosis”.<sup>6)</sup> The term “bruxism” became well established, and has widely been used without a critical definition. “Bruxism” was commonly used to indicate a nonfunctional gnashing, grinding or pressing of the teeth day or night,<sup>7)</sup> and was defined as the nonfunctional contact of teeth, including clenching, grinding and tapping of the teeth as

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well as the chewing of pencils, cheeks and lips.<sup>8)</sup> In 1993, the term of “bruxism” was re-defined by the American Academy of Orofacial Pain as “diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing, and grinding of the teeth”.<sup>9)</sup> It contained all types of activity that are not related to speech, swallowing, and functional mastication. Lately, the definition of bruxism was arranged as “a form of orofacial motor activity that should not be confused with other facial movements such as swallowing, coughing, sleep talking, grunting, or alternating jaw opening-closing”, since the original definition lacked specific criteria to discriminate normal from pathological oromandibular activities occurring during both sleep and wakefulness.<sup>10-12)</sup> Furthermore, the original definition included “bracing” which does not necessarily involve “tooth contact”. This was in disagreement with other bruxism definitions in which “tooth contact” apparently is a necessary condition.<sup>13-15)</sup> The patterns of bruxism might be considered as “tooth grinding (rhythmic, chewing-like movements)” and “tooth clenching (prolonged, strong isotonic contractions)”,<sup>13)</sup> which are clearly different from normal daily orofacial motor activities, such as chewing, drinking, exercising, eating, smiling/laughing, sexual activity, cleaning teeth or face, yawning, swallowing, talking, or having usual facial appearance. Most recently, bruxism was defined as “repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible” by the current authorities.<sup>16)</sup>

Sleep bruxism is the forcible grinding or gnashing of the teeth during sleep, produced by rhythmic contraction of the masseter and other muscles, and was defined as “stereotyped movement disorder characterized by grinding or clenching of teeth during sleep”.<sup>15)</sup> To avoid confusion, the use of “nocturnal” bruxism was replaced by “sleep” bruxism as suggested by the American Sleep Disorders Association<sup>15)</sup> to include the activities that can occur with daytime sleep. Sleep bruxism is a common condition, and is normally not recognized by the subjects. Lately, Lavigne *et al.* (1995)<sup>17)</sup> reported that there is a atypical repetitive rhythmic oromandibular activity during sleep, which is different from “sleep bruxism”, called “rhythmic masticatory muscle activity (RMMA)”. Sleep bruxism includes rhythmic (or phasic) and sustained (or tonic) masticatory muscle activity with grinding, tooth wear, and occasional pain in jaw muscles, while RMMA is characterized by repetitive bursts in jaw-closer muscles in the absence of signs and symptoms of bruxism.

## 2. Prevalence of bruxism

Epidemiological studies have shown that the prevalence of self-reported clenching while awake is in the 20% range, a figure derived from studies in college or university students,<sup>18, 19)</sup> general dental practices,<sup>20)</sup> and general populations.<sup>21)</sup> Prevalence of tooth clenching when asleep is estimated to be 9.6%, and sleep tooth grinding is reported by 6% to 12% of the population.<sup>20)</sup> The prevalence of sleep bruxism is 6% of the general Canadian population, and the prevalence of rhythmic masticatory muscle activity is 55.7% of the general sleep population in the laboratory from Lavigne’s study (1995).<sup>17)</sup> Female patients report more tooth clenching, but no gender differences have been noted for tooth grinding.<sup>19, 20)</sup> The highest prevalence of bruxism is noted for the 20 to 50 year old age group,<sup>19, 20, 22, 23)</sup> and it decreases with age.<sup>24)</sup> However, there is a problem with reliability of the estimation of sleep bruxism without electromyographic (EMG) recording since it depends on how the question was asked. Many persons with sleep bruxism are unaware of the condition themselves, low estimates are, thus, obtained when individuals are simply asked whether they brux their teeth. The validity might be increased if there is a bed partner who is also asked about the patient’s condition. Although it is still circumstantial evidence, dental examination is the most frequently used estimation in clinic.

## 3. Etiology of bruxism

The etiology of bruxism might be classified into three major factors. First, morphological factor is considered a main factor of bruxism. It has been found that the prevalence of bruxism is much higher in population groups with malocclusion than in comparable groups with normal occlusion.<sup>25)</sup> Occlusal discrepancies were also suggested as the most common cause for bruxism.<sup>7)</sup> The suggested mechanism of bruxism was that occlusal disharmony evokes mental stress and then increase bruxism activity. Correction of occlusal disharmony would, therefore, result in immediate disappearance of the habitual grinding of the teeth since the adjustment would remove the exciting factor.<sup>6)</sup> However, the importance of disharmony of occlusion has still being discussed,<sup>14, 26, 27)</sup> because it was demonstrated that experimental disharmony of occlusion did not effect bruxism.<sup>28)</sup> It has also been reported that the craniofacial

morphology (notably the bizygomatic and cranial widths) is significantly different between bruxers and non-bruxers.<sup>29)</sup> Second, in addition to the mental stress from occlusal disharmony, psychological problems are possibly considered as a factor of bruxism. A tendency to gnash and grind the teeth in association with feeling of anger or aggression has been recognized since Biblical times, and it has been observed in animals as well as in humans.<sup>30)</sup> Furthermore, some authors have noted a higher incidence of bruxism in patients with mental retardation.<sup>31)</sup> Third, there is a perception that sleep bruxism is a symptom of sleep disorders. Sleep bruxism is often observed in conjunction with conditions such as sleep apnea, periodic leg movements in sleep (PLMS), epilepsy, tardive dyskinesia, Gilles de la Tourette syndrome, schizophrenia, mental retardation, tics, and posttraumatic stress disorder.<sup>32)</sup>

Polysomnographic studies indicated that sleep bruxism is a part of a sleep arousal shifting the sleep to lighter sleep stages.<sup>12, 33-42)</sup> K-complex in the electroencephalography (EEG), neck and legs movements were followed by jaw-muscle activation.<sup>42, 43)</sup> Pathophysiological factors, like cigarette smoking<sup>44, 45)</sup> are also showing an association with sleep bruxism, and alcohol, illicit drugs, trauma, disease, and medication also might be suggested as factors for disorders.

#### 4. Bruxism and jaw-muscle pain

Many older epidemiological surveys have still indicated a strong-positive correlation between bruxism and TMD.<sup>24, 46-52)</sup> A frequent clinical observation has been that 43% to 50% of myofascial TMD patients<sup>53, 54)</sup> and 26.6% to 66% of TMD patients<sup>22, 55, 56)</sup> had a tendency to clench or grind their teeth. Grieder (1973)<sup>57)</sup> reported that ninety-seven of 100 masticatory dysfunction patients he studied showed evidence of bruxism. In a study of thirty-seven TMD patients, Lupton (1966)<sup>58)</sup> noted 77% as having chronic oral habits. Ramfjord (1961)<sup>7)</sup> found that all thirty-two TMJ pain patients in his study had clinical indications of bruxism. Solberg *et al.* (1979)<sup>59)</sup> examined 585 subjects and found that bruxers showed a statistically higher incidence of superficial masseter pain on palpation than non-bruxers. These studies although suggestive are limited in that they did not measure actual levels of muscle activity by EMG. Muscle hyperactivity was inferred from verbal reports, tooth wear, or muscle symptoms. Furthermore, some studies

claim that continuous muscle activity (muscle spasm) is always present in TMD.<sup>60, 61)</sup> Laskin (1969)<sup>62)</sup> proposed a new concept of etiology -the psychophysiologic theory of TMD, which was based on Schwartz's (1959)<sup>63)</sup> study. He suggested that jaw-muscle spasm is the primary factor responsible for the signs and symptoms of TMD. The jaw-muscle spasm is thought to be initiated in one of three ways: muscular overextension, muscular overcontraction, or muscle fatigue resulting from tooth grinding or clenching as post-exercise muscle soreness (PEMS). The spasm not only leads to pain and limitation, but also may leads to change in the jaw position. Travell (1960)<sup>64)</sup> suggested that an injury produces a "trigger area" within a muscle, and this results in muscle pain and referred pain to other areas. Laskin (1969)<sup>62)</sup> also suggested that a functional problem causes an organic disease. The "condition" then would become self-perpetuating with abnormal patterns of muscle activity reinforcing the original myospasm and pain, thus setting up a chronic vicious cycle, like the one proposed by Travell *et al.* (1942).<sup>65)</sup> Some investigators suggested that stress induced muscle activity is a primary etiological factor in masticatory musculoskeletal symptoms.<sup>62, 66)</sup> This vicious cycle theory was widely accepted even though there is no experimental evidence that explains the entire chain of events constituting the vicious cycle.<sup>67)</sup>

However, there are some questions related to the simple concept of a vicious cycle because there is evidence that only some bruxers have facial pain<sup>22, 26, 48, 49, 52, 68)</sup> and non-painful bruxers have much higher jaw-muscle activity during sleep than painful bruxers.<sup>69, 70)</sup> It is difficult to understand that lower-muscle activity during sleep should be linked to a higher-pain intensity. These results are in disagreement with the first part of the concept that jaw-muscle hyperactivity should lead to jaw-muscle pain and soreness. The second part of the statement that jaw-muscle pain induces more jaw-muscle hyperactivity has also been challenged. There is evidence that experimental muscle pain in human does not cause significant increases in EMG activity<sup>71-73)</sup> although animal studies have suggested a robust increase in both jaw opening and closing muscles.<sup>74)</sup>

Experimental pain models on human subjects is useful since the results can more directly referred to clinical patients, psychological measurements can be involved, pain intensity can be maintained stable, and pain distribution is reproducible and the stimulation area is often well localized. Furthermore, these pain experiments

with healthy subjects are available to measure the absolute levels of pain intensity and soreness evoked by standardized noxious stimuli, and is also available for investigating cause-effect relationships by excluding the confounding factors (Fig. 1). If the patients with complex chronic pain are recruited for experimental pain study, the estimation of pain may become more difficult because the type of clinical pain may vary, and the difficulty to identify the source of the original pain.

**[Hypothesis]**  
**Bruxism is the cause of jaw-muscle pain**

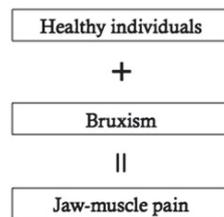


Fig. 1

For the exercise induced pain model in humans it is possible to control the levels of jaw-muscle activity (*e.g.* voltage, duration, and repetition) with visual feedback.<sup>70, 75-78)</sup> This might be the reason for the different results from the models in animals because muscle activity was not always voluntary movements but was directly evoked by electrical stimulation,<sup>79, 80)</sup> and pain adaptation was not allowed to occur. Although experimental jaw-muscle exercise has been tested in several ways since 1970's, there are not so many studies of PEMS models in jaw muscle compared to other muscles (*e.g.* limb muscles). In the early stage, tooth-clenching models were often used, but these were more focused on the onset of pain and fatigue sensation by brief isometric tooth contraction.<sup>81-83)</sup> Christensen reviewed that the onset of muscle fatigue and pain by maximal voluntary tooth clenching appeared after about 0.5 minutes and 1 minute respectively, and the tolerance of clenching endurance was about 2 minutes. He suggested that the psychological parameter of muscle endurance is large, and individual psychological factors seem to play a role. There is an interesting finding that the right masseter muscle was studied because it was particularly susceptible to the effects of tooth clenching.<sup>82, 84)</sup> It means that the human jaw muscles have a predominant side and this result is in accordance with Arima *et al.* (2000)<sup>70)</sup> that right side jaw muscle showed significantly higher number of jaw-muscle activity during sleep.

Standardized experimental tooth-clenching methods have started to be used from the 1980's. Significant levels of pain intensity and soreness were often observed immediately after experimental tooth clenching. However, there is a lack of information on pain intensity provided for the following days, which is the most important for investigation of PEMS. Furthermore, not all subjects followed this time course. Clark *et al.* (1991)<sup>85)</sup> examined the effect of 4 repeated sustained voluntary contraction of 25, 50, 75 and 100% of maximum effort at a protrusive position in eight subjects, and it was the first time to follow the subjects for several days after the experimental tooth clenching (for tooth grinding: Christensen 1971<sup>86)</sup>). Although maximum intensity of the immediate pain was quite high (about 75 mm on VAS of pain intensity), the pain resolved quickly within a few minutes, and there was no significant masticatory muscle pain in the volunteers during the days after concentric and eccentric (protrusive) exercises. Svensson *et al.* (1996)<sup>78)</sup> described submaximal (25% of subject's maximal bite force) tooth clenching for 15 min each day for 5 consecutive days. This study was the first to report the effects of 5 days of repeated episodes of submaximal tooth clenching in healthy subjects. Furthermore, Takeuchi *et al.* (2015)<sup>77)</sup> reported that three-consecutive days of 2-hours tooth clenching at 10% of subject's maximal bite force caused perceived jaw-muscle pain during the tasks, however, the pain did not last long time (within a day). These results provided no evidence to suggest that simple isometric muscle activity may cause a progressive increase in pain and soreness of masticatory muscles and that a vicious cycle could be initiated by this level of muscle activity.

Tooth-grinding models also have been used for exercise-induced jaw-muscle pain due to the suggestion of tooth grinding as a typical phenomena in patients with TMD. It was hypothesized that sleep jaw-muscle activity might cause PEMS in the masticatory muscles.<sup>87)</sup> Christensen originally made an experimentally jaw-muscle exercise induced pain in human, and he suggested that tooth grinding, or true bruxism, and other dynamic and strenuous work by the jaw muscle may give rise to facial pains.<sup>86, 88, 89)</sup> It was the first report that tooth-grinding involving movement of the mandible (isotonic contractions) causes a development of post-exercise masticatory muscle pain lasting for several days.<sup>86)</sup> Bowley and Gale (1987)<sup>90)</sup> reproduced Christensen's short-term findings but unfortunately did not follow their subjects the days after the exercise. Arima *et al.* (1999)<sup>75)</sup>

made the first attempt to induce jaw-muscle pain by a standardized tooth-grinding exercise and reported that, as the same as the tooth-clenching studies, significant levels of perceived pain and soreness were observed but did not last for a day.

It may be true that bruxism plays a role in the etiology of jaw-muscle pain, but it does not seem appropriate to suggest that bruxism is the main and necessary factor of jaw-muscle pain. Because there is no scientific evidence showing a strong relation between bruxism and jaw-muscle pain, and there is, even, evidence to suggest the opposite that frequent bruxers have less pain than occasionally bruxers. It may be explained by the finding that muscle pain in itself does not increase muscle hyperactivity but rather inhibits muscle activity, which has been postulated in the pain adaptation model.<sup>91)</sup>

### Conclusions

In conclusion, it is suggestive that bruxism may cause jaw-muscle pain in human but the effect is not high or long enough like TMD. Pain adaptation model might be the best fit to the jaw-muscle pain system.

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