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# The two main theories on dental bruxism

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SUMMARY

Bruxism is characterized by non-functional contact of mandibular and maxillary teeth resulting in clenching or grating of teeth. Theories on factors causing bruxism are a matter of controversy in current literature. The dental profession has predominantly viewed peripheral local morphological disorders, such as malocclusion, as the cause of clenching and gnashing. This etiological model is based on the theory that occlusal maladjustment results in reduced masticatory muscle tone. In the absence of occlusal equilibration, motor neuron activity of masticatory muscles is triggered by periodontal receptors.

The second theory assumes that central disturbances in the area of the basal ganglia are the main cause of bruxism. An imbalance in the circuit processing of the basal ganglia is supposed to be responsible for muscle hyperactivity during nocturnal dyskinesia such as bruxism. Some authors assume that bruxism constitutes sleep-related parafunctional activity (parasomnia).

A recent model, which may explain the potential imbalance of the basal ganglia, is neuroplasticity. Neural plasticity is based on the ability of synapses to change the way they work. Activation of neural plasticity can change the relationship between inhibitory and excitatory neurons.

It seems obvious that bruxism is not a symptom specific to just one disease. Many forms (and causes) of bruxism may exist simultaneously, as, for example, peripheral or central forms.



## 1. Introduction

The term bruxism refers to a non-functional contact of mandibular and maxillary teeth often resulting in the clenching or grinding of teeth (Graf, 1969; Glaros and Rao, 1977). This dyskinesia most often occurs during sleep although it also may occur while awake (Nadler, 1972; Bader et al., 1997). Typical symptoms are abrasion of the dental hard substance, chipping or even fractures of teeth and prostheses, pain in the affected muscles and joints, and teeth which are sensitive to biting (Rugh and Orbach, 1988; Greene et al., 1998) (Fig. 1).

Bruxism can be divided into idiopathic and iatrogenic types. The idiopathic form, which includes clenching and grating as well as nocturnal bruxism, is not linked to neurologic or psychiatric disorders (Glaros, 2006). Nocturnal bruxism often starts after the cutting of the first teeth (Widmalm et al., 1999). The prevalence of bruxism during infancy is 14–20%. Orofacial dyskinesia affects about 8% of adolescents (Wänman and Agerberg, 1986; Egermark et al., 2003) and 8% to 9% of adults (Lavigne and Montplaisir, 1994; Ohayon et al., 2001; Egermark et al., 2003), a percentage that decreases to less than 3 in the age group of 60 years and older (Alexander and Crutcher, 1990; Ohayon et al., 2001).

## 2. The two different theories of bruxism

In the current literature, the following theories of the factors causing bruxism are controversially discussed (Glaros, 2006; Lobbezoo and Naeije, 2001):

### 2.1. Theory 1 (peripheral causes)

Up to now, the dental profession has predominantly viewed local morphological disorders in the periphery, such as malocclusion, as the cause of clenching and gnashing. This etiological model is based on the theory that malocclusion results in reduced masticatory muscle tone. In the absence of occlusal equilibration, motor neuron activity of masticatory muscles is triggered by periodontal receptors. Proponents of this theory refer to their long-term clinical experience and success (Kerstein and Farell, 1990; Dawson, 2007). However, successful reports including the typical features of non-blinded treatment concepts, and controlled clinical studies are rare. For example, when investigating the effects of artificial occlusal

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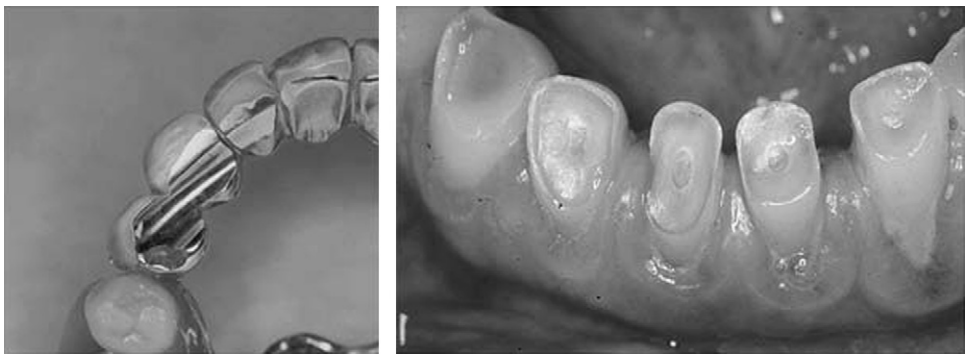

**Fig. 1.** Left: Clenching pathway of a lower canine in the precious alloy framework of an upper removable denture. Right: Tooth substance lost of lower premolars caused by nocturnally clenching.

interference in patients with bruxism and in healthy people, Shiau and Syu (1995) could only show that occlusal disturbances were well-tolerated by both groups. Proponents of the occlusion theory often refer to the 1961 study by Ramfjord, who was probably the first to carry out electromyographical investigations in patients with bruxism (Ramfjord, 1961). Ramfjord proposed that bruxism is caused by discrepancies between retruded and habitual contact positions as well as by balanced contacts. According to Ramfjord, occlusal corrections always result in the disappearance of bruxism symptoms, a claim which he thought to be able to prove by means of electromyographical records of 45–60 min duration. His critics rightly state that such a short period of time could hardly be significant; furthermore, Ramfjord's study failed to include randomized, blinded or control groups (Lobbezoo and Naeije, 2001).

A further problem of the occlusion theory is, that, so far, nobody has been able to show how 'perfect' occlusion should be achieved (Payne, 1961; Lundeen, 1969; Suckert, 1992). No controlled clinical study has yet been able to show that bruxism symptoms can be significantly abated, either by removal of occlusal interferences or by equilibration methods (Kardachi et al., 1978; Clark and Adler, 1985; Greene et al., 1998; Rugh et al., 1984; Türp et al., 2004; Garcia et al., 2005; Macedo et al., 2007). However, it would be premature to disregard the influence of occlusion on the development of bruxism. Occlusion determines the localization of biomechanical transmission. The intramuscular functional patterns of the masticatory muscles are regulated via the receptors of the periodontal apparatus. These functional patterns are modified by different motor tasks as well as by dislocation of the mandible in relation to the maxilla (Türp and Schindler, 2003). The receptors of the periodontal apparatus relay information on the location of the mandible in relation to the maxilla in the state of equilibrium. For equilibration, the body requires propioreceptors providing the present location of body parts, such as arms, legs, or the mandible. Roccabado pointed out, that, during involuntary deglutition (occlusion), mandibular and maxillary teeth briefly make contact so that the receptors of both tooth rows become activated for a short

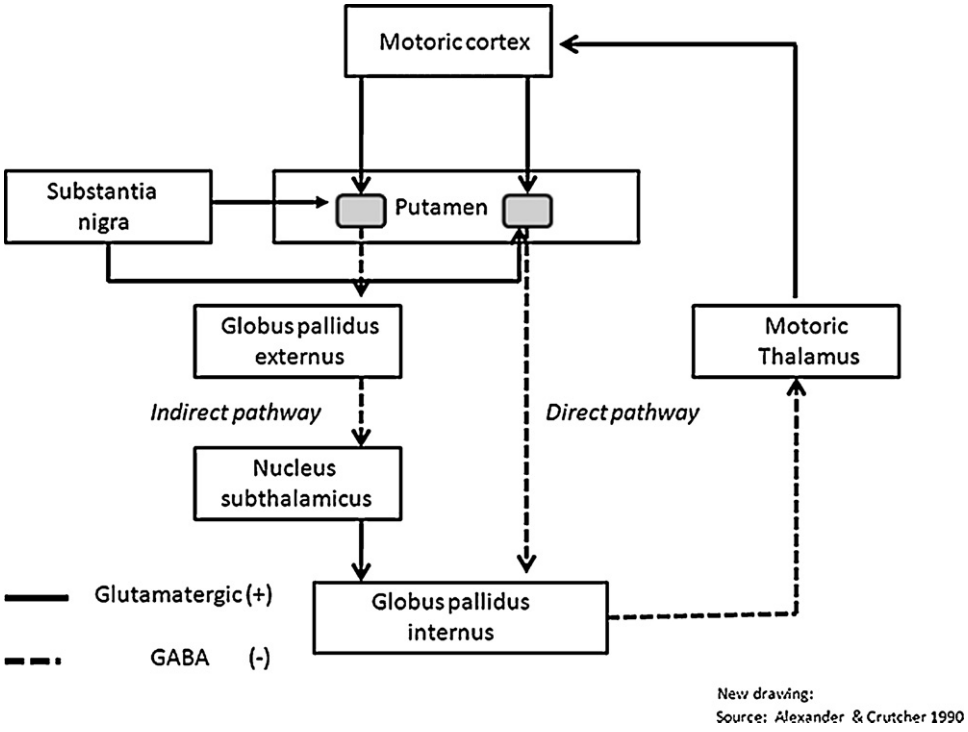


**Fig. 2.** Diagram of the circuitry and neurotransmitters of the basal ganglia – thalamus circuitry. An indirect and direct pathway exists which may modulate motor programs from the motor cortex. Inhibitory pathways are GABA (GABA: γ-aminobutyric acid) i and excitatory pathways are glutamate initiated. New drawing.
Source: Alexander and Crutcher, 1990.

time (Roccabado et al., 1982; Roccabado and Iglarsh, 1991). If load is evenly distributed on all teeth during the final clamping position, receptors send information that the mandible is in the best physiological position for the body and thus also for the sense of equilibrium. During malocclusion, premature and one-sided contact is registered. Receptors may interpret this contact in such a way that the mandible needs to be retracted to the resting position by muscular activity. If assuming a final clamping position is not possible because of malocclusion, movement patterns in the motor cortex are constantly triggered in attempt to achieve the resting position (Roccabado and Iglarsh, 1991).

### 2.2. Theory 2 (central causes)

In the second theory, central disturbances in the area of the basal ganglia, for example sleep-related dysfunctions, are assumed to cause bruxism. Nocturnal parafunctional activity occurs in different stages of sleep (Bader et al., 1997; Lavigne et al., 1996, 2001). Some authors assume that bruxism constitutes a sleep-related dysfunction (parasomnia), occurring in association with sleepwalking, talking, or enuresis (Lavigne et al., 1996, 2001). Polysomnography is considered the gold standard for diagnosis of parasomnia, recording electrophalometric, electromyographic, and electrooculographic activities as well as respiration, pulse, blood pressure, and cardiac output by means of ECG (Lavigne et al., 1996; Glaros, 2006).

Basal ganglia (caudate nucleus, putamen, and globus pallidus) are components of functional loops arranged in parallel that include the thalamus and the cortex (Alexander and Crutcher, 1990). The information flow in these compartments controls the organisation of motor preparation and the execution of muscular movements (Fig. 2). In each case, specific cortical areas send excitatory projections to the striatum. The striatum represents the input stage of the basal ganglia. The basal ganglia output nuclei, i.e. the internal segment of the globus pallidus, the pars reticularis of the substantia nigra, and the ventral part of the pallidum, exert GABA-mediated inhibition to the target nuclei in the thalamus (Cools, 1984; Chevalier et al., 1985; Joel and Weiner, 1994). This inhibitory outflow is differentially modulated by two opposing but parallel pathways. One arises directly from the inhibitory striatal efferents and tends to disinhibit the thalamic stage of the circuit (Chevalier et al., 1985). The indirect pathway first leads to the external segment of the globus pallidus, then passes to the subthalamic nucleus, and, finally, to the output nuclei via an excitatory projection from the subthalamic nucleus (Cools, 1984; Joel and Weiner, 1994). Both pathways may be activated selectively and concurrently in association with cortically initiated movements. Then, the inputs from the indirect pathway, which are reinforced by the direct pathway, may smooth or break the cortically initiated motor pattern. Thus, both pathways contribute to the motor pattern of initiated muscles allowing controlled purposeful movements. Striatal operations are furthermore influenced by neurotransmitters such as dopamine. The overall influence of dopamine on the striatum may reinforce any cortically initiated activation and facilitate conduction via the circuit's direct pathway, which has an excitatory effect on the thalamus (Cools et al., 1983; Cools, 1984; Alexander and Crutcher, 1990). The antagonist to dopamine is acetylcholine, the clinical effects of which are well-known, for example, in Parkinson's disease (Herrera-Marschitz et al., 1986; Crossmann, 2000). Imbalance in the circuit processing of the basal ganglia is thought to be responsible for muscle hyperactivity during nocturnal dyskinesia such as bruxism (Lobbezoo et al., 1997a,b; Lobbezoo and Naeije, 2001).

The question arises as to how an imbalanced function of the basal ganglia can be explained. One answer may be plasticity related diseases. Neural plasticity is based on the ability of synapses to change the way they work. Activation of neural plasticity can change the relationship between inhibition and excitation. Activation of neural plasticity can also change synaptic efficacy and create or eliminate contacts between nerve fibers and nerve cells and their dendrites. According to Møller (2009), the two main causes of plasticity disease are faulty expression of neural plasticity and lack of expression of neural plasticity. Faulty expression may cause hyperactive diseases, such as central neuropathic pain, tinnitus, and hyperactive movement disease, whereas lack of expression is suspected to result in the development of diseases such as some forms of autism.

## 3. Conclusions

The literature contains conflicting reports on the cause of dental bruxism. It seems to be obvious that bruxism is not a specific entity of just one disease. Many forms (and causes) of bruxism may exist, for example peripheral or central forms. So far, no clear diagnostic tools are available for allocation of a patient to any respective group and many incorrect treatment procedures may be carried out because bruxism is regarded from a single point of view only.

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