Bruxism: An Unsolved Problem in Dental Medicine

Summary

Bruxism exists as long as humanity; since barbarian times, through the Bible and christianity until the modern stressful age. According to American Sleep Disorders Association, the prevalence of bruxism varies from 5 to 20 per cent. This difference can be related to the lack of awareness in eighty per cent of the population that has some form of teeth clenching and/or grinding. Many studies have been published, but the results are still contradictory. There is a large scale of different etiological and therapeutical approaches presented in the scientific literature, but the main problem - bruxism in everyday practice often remains undetected and badly treated.

Key words: bruxism, etiology, therapeutical problem.

History, definition and prevalence

Gnashing and clenching of teeth has probably been around as long as mankind. It was mentioned several times in the Bible. In the Old Testament (The Book of Job, “His anger has torn me and hunted me down; he has gnashed at me with His teeth”, Psalm 16:9), and in the New Testament (Matthew 8:12, “But the children of the kingdom shall be cast out into outer darkness: there shall be weeping and gnashing of teeth”, the parable of the healing of the poor in spirit from Luke...). Also, in many cultures the gnashing of teeth has been associated with suffering, physical pain, madness and possession. However, we have only recently realized that there are no evil spirits involved. The term comes from the Old Greek word brychein- the grinding of teeth. In scientific and scholarly writings, the term bruxism was first used by Marie and Pietkiewitz in 1907. Not long before that, Black had described abnormal enamel wear connected with non-functional activity and Karoly had referred to the state as neuralgia traumatica. Other researchers spoke about Karoly’s effect (Weski) and the neurosis of occlusal habits (Tishler). Miller makes a distinction between nocturnal gnashing- bruxism, and diurnal gnashing, which he calls bruxomania. There is much confusion about the very definition of bruxism because the researchers are still not unanimous on the question of whether the term includes only nocturnal, or it also includes diurnal parafunctional movements. The American Academy of Orofacial Pain (3) defines bruxism as “diurnal or nocturnal parafunctional activity which includes clenching, gnashing, gritting and grinding of teeth. It can be clinically diagnosed based on the presence of excessive tooth wear which could not have been caused by mastication”. American Sleep Disorders Association (1) talks about “gnashing or clenching of teeth during sleep”, but the diagnosis is made only when at least one of the symptoms exists (tooth wear, noises and sensitivity of the masticatory muscles which cannot be attributed to any other disorder).
According to Suvin (4) bruxism is gnashing and gritting of teeth due to unconscious, and thus several times stronger, contraction of masticatory muscles. Nadler (5) writes about antagonistic contacts which sometimes produce the noises of grating or clicking. The contacts are the result of strong rhythmical contractions of masticatory muscles (m. masseter, m. temporalis, and m. pterygoideus medialis). In fact, normal physiological state of inactivity of the mandible during sleep is disrupted.

Data on the prevalence of bruxism vary because of the use of different research methods, working definitions, clinical criteria and population samples. An estimated 20% of the population gnash their teeth during the day while there are 10% of those who have the same symptoms during the night; when it comes to movement of the mandible, which result in gritting, occurrence varies from 6% to 13% (6-9). Bruxism occurs most often in adolescence and in the fifth decade of life and its prevalence seems to decline with age (10). The data regarding the distribution between the sexes vary. Some research studies show higher prevalence of parafunctions in women, while other studies deny any differences in the prevalence of the symptoms of bruxism between the sexes.

**Etiology**

The cause of bruxism is still controversial. Etiology is probably made up of many factors which overlap. This in reality creates great difficulties in the preparation of an effective treatment plan (1).

At the beginning of the last century it was thought (Karoly) that the gnashing of teeth is the co-effect of occlusive interferences and psychological factors. Since that time, all studies put emphasis on one of two etiological factors. An investigation carried out in the late 70s proved that there was no direct connection between occlusive interferences and bruxism. Consequently, instead of talking about the existence of occlusive interferences, we speak of the patient’s reactions to the changes in occlusion.

Rugh, Barghi and Drago (16) concluded in their research study that “nocturnal bruxism is not caused by inserting artificial occlusal interferences even in patients who have bruxism in their case history”. Yap (17) confirmed that eliminating the interference by increasing the vertical dimension of the bite will not stop bruxism. Another investigation had similar conclusions, adding that long-lasting use of splints which increase the vertical dimension alleviates the cranio-mandibular symptoms (18).

Stress and other emotional factors are another important factor mentioned in scientific literature. The above cited examples indicate that the gnashing of teeth and increased contraction of masticatory muscles have always been associated with some traumatic experiences. Even Freud (19) noticed that the mouth cavity is very important in the psycho-sexual development and behaviour of a person. It can be a source of pleasure and/or a valve for releasing aggression. In the latter case the patient will have the habit of biting his/her teeth, lips or cheek, or clenching and or gnashing the teeth, etc. Recent research studies have shown that there is a connection between oral habits and using the oral cavity for giving vent to frustration. It has been shown that patients with bruxism have an increased tendency to self-punishment when reacting to frustrating situations, and that they are more anxiety-ridden (20). A study on the effects of psycho-emotional tension has shown that the group with a higher level of tension will consist of twice as much ‘bruxers’ as the group without tension (21). Jorgić-Srdjak et al. (22) associate bruxism with certain types of personality, while Pintigore, Chorback and Petrie established a link between bruxism and some physical abnormalities and behavioural type A (intense yearning for success, competition, acknowledgment, involvement with many physical and mental activities) (23). The connection between bruxism and some sleep-disorders (e.g. disturbance of breathing during sleep) is also very significant (24).

When considering the causes, we have to take into consideration the effects of some medications, fenfluramine, levodopa (25, 26) and amphetamine (27); malnutrition (28), the provoking effect of alcohol (29); genetic factors (30, 31); CNS disorders; tobacco and Ploceniak’s theory of atypical tetany due to the shortage of magnesium (34).

**Case record and diagnostics**

The diagnostics of bruxism is based on its case record, the symptoms and changes it causes in the
masticatory system and surrounding structures. The forces that influence these structures are usually up to three times stronger than masticatory forces and there are records of cases in which the forces of gnashing and gritting were even one thousand times stronger than the normal forces. Their persistence in chronic bruxism leads to the typical case record (36):

I. Grinding facets, attrition and other changes on the teeth

Rapid abrasion of one or more teeth and non-physiological attrition occur. Grinding facets appear on the incisal ridge and knots of the upper teeth as a result of the cracking of enamel prisms at the place of contact. Damage to the enamel is followed by dentin damage. Pain may occur, depending on the reactive hyperaemic of the pulp. In cases with long-standing bruxism facets appear more often on the front teeth, while prostodontic patients have facets on the back teeth. Fractures of teeth and fillings may occur because during eccentric bruxism the mandible may move laterally just enough for the canines to come into tete-a-tete position (18). Fractures are common on the back teeth as well, usually appearing on the lower front molars (38).

II. Effects on the periodontal ligament and the mobility of teeth

Teeth clenching causes more damage to the periodont than any other parafunctional activity. Ligaments and the ligament space (radiologically discernible) spread and the mobility of the teeth increases (1).

III. Increased tonus and hypertrophy of the masticatory muscles

M. masseter, m. temporalis and m. pterygoideus medialis are continually stimulated by bruxism. Unilateral and/or bilateral hypertrophy of m. masseter are clinically discernable in the case of long-standing bruxism, while electromyographic studies show abnormally high tonus in all three muscles.

IV. Headache and pain in masticatory muscles

Nadler talks about the existence of a closed circle between bruxism and headaches. Kampe et al. (40) refer to pain in the face and jaws as one of the most frequent symptoms of the group of symptoms termed CMD (cranio-mandibular disorder). Pain is located in the area of the front upper border of the masseter and medial pterigoid muscle and in the temporal muscles, and it is connected to jaw fatigue and limited movements of the mandible.

V. Changes in the temporomandibular joints

In 1961 Ramfjord (41) proved that patients with changes and/or pains in the temporomandibular joints usually gnash their teeth. The intensity of the changes in the joint will depend on whether the person has centric (clenching) or eccentric (gnashing, gritting) and, naturally, on the strength of the parafunctionally used forces (36). The changes in the temporomandibular joint can be manifested by noises (clicking, popping, and crepitating), pain, limited movement in the joint itself and the spasm of the muscles.

Some types of bruxism lead to the spread of the ligaments of the capsule, fibrosis, etc. The head of the condile can have different positions, and therefore after some time the configuration of the joint changes and it results with the movement and the above mentioned TMJ symptoms.

Other consequences of bruxism are: changed appearance of the face due to the changes of the teeth, decrease in the vertical dimension (42) and clinically discernible hypertrophy of the masticatory muscles (43), damage to the salivary glands due to the halt caused by masseteric hypertrophy (43) and social discomfort because of the noises that the patients make.

Treatment

The aim of every practitioner in the treatment of bruxism must include reduction of psychological stress, treatment of the symptoms and signals of mandibular parafunctions, reduction of occlusive irritation and the change of neuromuscular habits (44). Before deciding on treatment, one should take into consideration the patient’s complete condition because the causes and solutions often consist of many factors. This way treatment will be adjusted to the prevailing etiological factor.

Two basic points in the therapy are stress control (changes in lifestyle, professional counselling,
relaxation exercises, even hypnosis) and direct methods of treatment applied to the stomatognathic system, which include the adjustment of occlusion, intraoral devices, pharmacotherapeutics and physical therapy (2).

The most widely known and commonly used therapeutic means is an occlusal splint. The types and variants of splints are as varied as the research done on their therapeutical effect. Some research studies speak in favour of the use of splints while others note only partial success in the reduction of nocturnal bruxism and even aggravation in 20% of the cases (45). One study concluded that 80-90% of the patients examined in the research experienced an alleviation of symptoms after the therapy, although masticatory forces were reduced in only 50% of the cases (46). This all speaks in favour of splints and the role of splints in the redistribution of forces and the easing of their effect on the temporomandibular joint (47). On the other hand, a splint does not stop nocturnal bruxism, but only relieves the symptoms by modifying parafunctional activities and/or changing the distribution of the trauma in the masticatory system. However, when the therapy ends the symptoms exacerbate (18).

Messing (48) warns that the splint creates discomfort for patients, that it is impractical and can cause changes in occlusion, it can cause open bite, caries, and periodontithesis and can lead to degenerative disorders of the joint. Perl (49) concludes that during periods of depression there “is no way of preventing the clencher or bruxer from engaging in such parafunctional habits. However the clinician may be able to decrease the potential for destruction by adding a splint to the treatment protocol”.

Other attempts of therapy have been neglected by scientific testing. Many are completely abandoned today, for example, most types of psychotherapy, equilibrium therapy, pharmacotherapy, sound-alarm based on EMG activity of the muscles. Ploconic’s research study in which he reports almost incredible success in the treatment of bruxism with an increased dosage of magnesium, is also well known.

There are also some new approaches, such as mini-splint and the ‘taste-oriented approach’ which stops the parafunction by producing unpleasant taste sensations in the patient (50). These methods still have to undergo serious clinical testing for their therapeutical value to be confirmed.