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Bruxism in Childhood - Etiology, Clinical Diagnosis and the Therapeutic Approach

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Abstract: *Bruxism was defined as nonfunctional movements of the mandible with or without audible sound occurring during the day or night. The clinical indicators of diagnosing this parafunction were the presence of dental wear/attrition and bruxofacets. The disorder appears more frequently in the younger population. The prevalence in children is between 14 to 20%. The present case report refers to a patient who reported to the Center For Dentistry,Research & Aesthetics, Jatt, Almothalath, Israel ,with the complain of Bruxism .A brief review is made of the literature concerning the etiology,clinical diagnosis and the therapeutic approach of the disease.*

Keywords: *Bruxism, etiology , child, Review of bruxism*

I. Introduction:

The American Academy of Orofacial Pain 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".[1]

American Sleep Disorders Association 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 .[2]

The prevalence of bruxism varies among studies, but generally the prevalence of awake bruxism is thought to be about 20%, being more common among women, while the prevalence of sleep bruxism is about 8% in the adult population being same among sexes [3,4] . However, based on self-report studies, the prevalence of bruxism is highest in children, gradually decreasing with increasing age; from 14–18% in childhood to 3% in the elderly . The importance of factors like ethnicity on the prevalence of bruxism is uncertain .[3,4,5,6]

Bruxism is a pathological activity of the stomatognathic system that involves tooth grinding and clenching during parafunctional jaw movements. Clinical signs of bruxism are mostly related to dental wear and muscular and joint discomforts, but a large number of etiological factors can be listed, as local, systemic, psychological and hereditary factors. The association between bruxism, feeding and smoking habits and digestive disorders may lead to serious consequences to dental and related structures, involving dental alterations (wear, fractures and cracks), periodontal signs (gingival recession and tooth mobility) and musclejoint sensitivity, demanding a multidisciplinary treatment plan.[6,7]

In children, bruxism may be related to growth and development of the jaws and teeth. Children may brux because their maxillary and mandibular teeth do not occlude properly and comfortably as they are erupting. Children may also grind their teeth because of tension, anger or as a response to pain from an earache or teething[8.9] .

The most important characteristic of bruxism is that there is non-functional contact of mandibular and maxillary teeth resulting in clenching or grating of teeth. There are few controversies regarding theories on bruxism[6,9].

Disorders, such as malocclusion may be the cause of clenching and gnashing. It is based on the theory that occlusal maladjustment leads to reduction in masticatory muscle tone. In the absence of occlusal equilibrium, motor neuron activity of masticatory muscles is triggered by periodontal receptors[3,4].

Second theory states that, a central disturbance in the area of basal ganglia plays an important role in causing bruxism. An imbalance caused due to the processing of basal ganglia is the main reason behind muscle hyperactivity during nocturnal dyskinesia such as bruxism. Few authors suggest that bruxism constitutes sleep-related parafunctional activity such as parasomnia. A recent study which explains the

potential imbalance of the basal ganglia is neuroplasticity. Neural plasticity is based on the ability of synapses to change or modify the way they work. Due to activation of neural plasticity, changes in the relationship between inhibitory and excitatory neurons occur[3,4].

Etiology of bruxism can be divided into three categories; they are psycho-social factors, peripheral factors and patho-physiological factors. The etiology of sleep bruxism is uncertain; the factors responsible are occlusal discrepancies and the anatomy of bony orofacial structures[10]. Other factors include smoking, trauma, alcohol, drugs, systemic disease, stress or peer pressure. Heredity appears to play an important role in the occurrence of sleep bruxism. Recent studies suggest that sleep bruxism episodes of individual are part of sleep arousal response. It is a sudden change in the depth of sleep of an individual. Besides this, sleep bruxism appears

to be a disturbance in the dopaminergic system. Previously, morphological factors like occlusal discrepancies and anatomy of the bony structures of orofacial region have been considered as pivotal factors for bruxism. However, nowadays these factors are thought to play only a minor role, as recent studies focuses more on patho-physiological factors.[7,8,9,10]

Studies on the etiology of bruxism are not concluded yet. Researchers have suggested that local factors, such as malocclusion, are loosing importance, whereas behavioral cognitive factors such as stress, anxiety and personality traits are gaining more space[11,12]. The current focus is directed to the fact that bruxism is part of a wakening reaction. This parafunctional activity seems to be modulated by several neurotransmitters in the central nervous system, however, it can not be affirmed that it has just a central control[13,14,15].

Sari and Sonmez reported a statistically significant relationship between bruxism and some occlusal factors, whereas, in other research studies, this association can not be proved.[16]

Bruxism can be associated to craniomandibular disorders including headache, temporomandibular disorder , muscular pain, early tooth loss due to excessive attrition and mobility and sleep interruption from both the subject and the person with whom he shares the room. Studies have shown the close relationship between bruxism and some pathologies such as breathing disorders and the Obstructive Sleep Apnea Syndrome .[1,2,10,12]

II. Bruxism may be classified according to several criteria;

a.According to presence:

a. Past bruxism
b. Present bruxism

b.According to etiology:

a. Primary, essential or idiopathic bruxism: No apparent cause is known.
b. Secondary bruxism: Secondary to disease (e.g. coma, ictus, cerebral palsy), medicinal products (e.g. antipsychotic medication, cardioactive medication), drugs (e.g. amphetamines, cocaine, ecstasy).

c.According to occurrence:

a. Awake bruxism
b. Sleep bruxism
c. Combined bruxism

According to motor activity type:

a. Tonic: Muscular contraction sustained for more 2 sec.
b. Phasic: Brief repeated contractions of masticatory musculature with three or more consecutive bursts of EMG activity that last 0.25 - 2 sec.
c. Combined: Alternating appearance of tonic and phasic episodes. Approximately 90% of the episodes of SB are phasic or combined, unlike in awake bruxism, where
episodes are predominantly tonic.[1,2,3,8,9,12]

Frequently, clinicians who treat children and adolescents are questioned about the etiology, the prevalence and the effect of bruxism. It is important that clinicians are well informed on recent studies and on the variables related to this parafunctional activity.[1,2,6,9,13]

The present case report refers to a patient who reported to the Center For Dentistry, Research & Aesthetics, Jatt, Almothalath, Israel, with the complain of Bruxism .A brief review is made of the literature concerning the etiology,clinical diagnosis and the therapeutic approach of the disease.

III. Case Report

A 4-year-old boy was brought to the Center For Dentistry,Research & Aesthetics, Jatt, Almothalath, Israel, by his mother. On history, his mother revealed that he gnashed his teeth at night, but she could not tell for how long this had been going on. During the case history taking, his mother revealed that he clicked his teeth at night for the last two years. No previous dental treatment was reported. Ingestion of acid drinks or medication was denied.Clinical examination revealed that the patient had late primary dentition with Flush Terminal Plane (Class I molar relationship on both sides) **(FIG 1).**

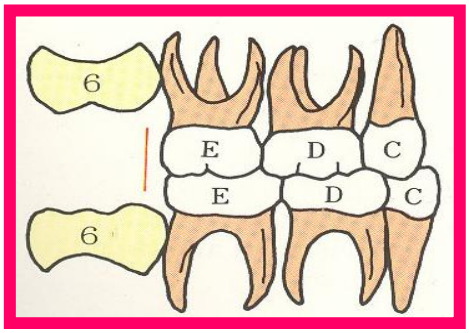

Fig. 1;In the flush terminal plane relationship, both the maxillary and mandibular planes are at the same level antero-posteriorly

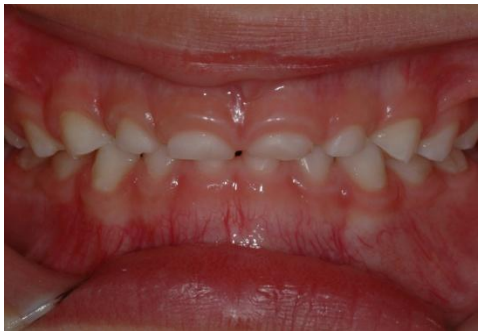

Fig. 2a

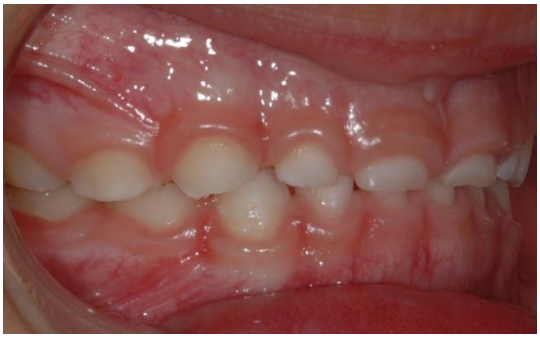

Fig. 2b

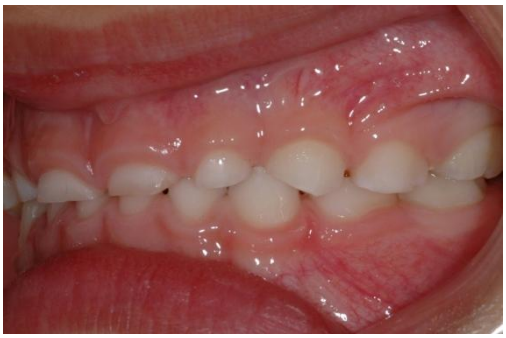

Fig. 2c

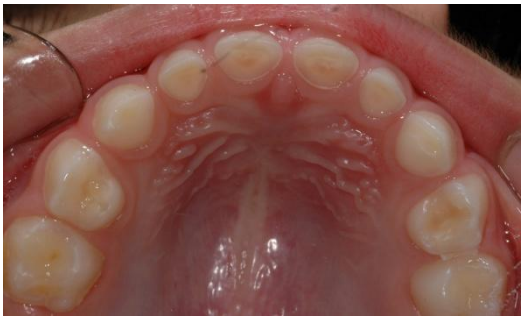

Fig. 2d

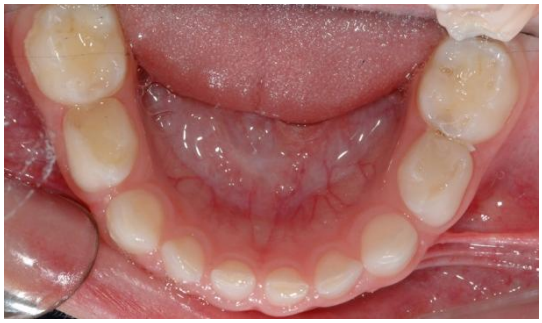

Fig. 2e

Fig. 2a-d: Clinical situation of a patient with 4 years. strong abrasions

There was no midline deviation or malocclusion. No occlusal interferences, mandibular deviation, mouth-opening limitation or any other clinical sign indicating temporo-mandibular dysfunction was noticed during intra-oral clinical examination. Good oral hygiene was observed. No carious lesions or gingival inflammation was present.

The occlusal surfaces of all primary molar teeth were worn but sensitivity was not present. A slightly increased overbite (2–3 mm) was present. The child had no history of pain, even with mechanical stimulation. Grinding of teeth, which has a characteristic sound, Pain in the masticatory and cervical muscles, Hypersensitive teeth,and Abnormal tooth wear.

The incisal faces of all anterior maxillary primary teeth were badly worn.Worn occlusal facets in teeth 55, 65, 75, 74, 73, 72, 82, 83 and 84 were also present

There were small fractures in the palatine cusps of teeth 54 and 64 and in the distovestibular cusp of tooth 85. The child had no history of pain, even with mechanical stimulation **(FIG 2a-e)**

A function orthodontic appliance has been used as prophylactic treatment procedure **(FIG 3a,b, FIG 4a-c)**. The goal of this treatment step was:

1) To stop or minimize the crunching at night

2) To control the growth in the vertical Dimention, so that it is not for severely impaired.

The control was performed every 8-10 weeks. The eruption was controlled. This leads to a bite raising, the appliance was grinded to allow the eruption of permanent teeth.

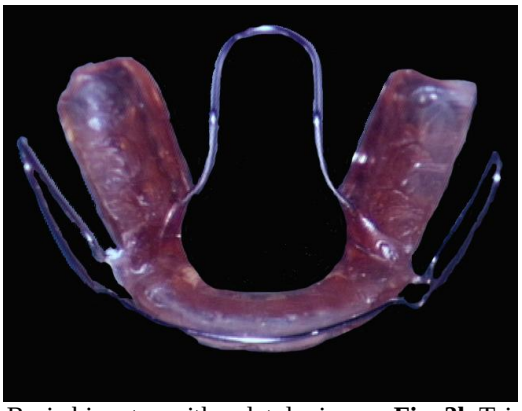

Fig. 3a: Basic bionator with palatal wire

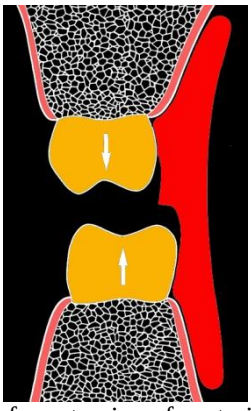

Fig. 3b Trimmed for extrusion of posterior teeth

Fig. 4a

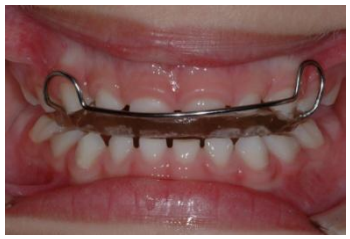

Fig. 4b

Fig. 4c

Fig. 4a-c: Bionator in mouth

Follow-up visits were scheduled every 2-3month to verify tooth wear and monitor eruption of permanent teeth and bone growth. When necessary, appliance (Bionatur Type I) were modified to allow adequate bone growth **(FIG 5a-c)**.

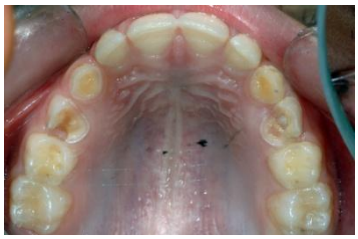
Fig. 5a

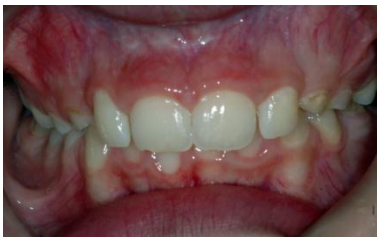
Fig. 5b

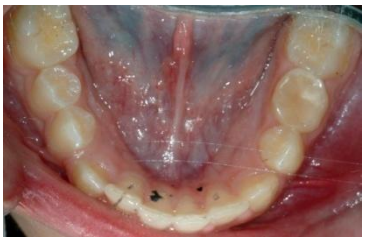
Fig. 5c

Fig. 5a-c Situation after 3 years

IV. Discussion

The prevalence of bruxism in children is estimated to range from 7% to 15.1%. A few studies confirm a higher rate in females than males. Some authors describe bruxism as a condition of multifactorial etiology, determined by an association of psychological, local and systemic factors.[3,4,7,8,9]

Craniofacial growth involves distinct structures and follows a complex chronological pattern, peaking in prepuberty. Thus, when removable prostheses are placed in young
children, osseous discrepancies may arise. The bite-plates are usually removable and quarterly revision appointments are scheduled to monitor patients' bone growth and the eruption of permanent teeth'[5,9,10,11,14]

Funch and Gale state that bruxism is correlated with psychological factors, suggesting that the kind of life the patient leads exerts great influence on the frequency, duration and severity of the condition. Thus, based on the presence of
the emotional problems reported by the parents, we believed that in all probability the condition presented by these patients was bruxism[17].

Restrepo et al demonstrated that several psychological techniques have been efficacious in reducing signs of bruxism when they were applied to 33 children aged
3–6 years.[15]

Vanderas and others have demonstrated that stress and anxiety may be directly related to bruxism, as patients suffering from bruxism show a higher catecholamine level, generally ascribed to emotional stress .[18]

Landry and Rompre compared the effect on sleep bruxism and tooth-grinding activity of a double-arch temporary custom-fit mandibular advancement device (MAD) and a
single maxillary occlusal splint (MOS). Short-term use of a temporary custom-fit MAD is associated with a remarkable reduction in sleep bruxism motor activity. To a smaller extent, the MOS also reduces sleep bruxism [19] .

Hachmann et al and McDonald et al, a bite-plate covering the occlusal surfaces of all teeth should be used by patients suffering from bruxism to prevent continuous abrasion.[20,21]

Dawson's experience has shown that signs and symptoms of eccentric bruxism seem to disappear completely with careful elimination of all occlusal interferences. According to Dawson, occlusal interferences can cause parafunctional movements which had not existed before the interference and by invasion to the envelope of function, delete mechanism is predictable[6].

Williamson's classic study and the that of **Ramfjord s**howed the causal relationship
between eccentric occlusal interferences and muscle hyperactivity and found that a marked reduction in muscle tones and action follows the elimination of occlusal disharmony.[22,23]

Solberg and others assert that the use of biteplates reduces muscular activity, thus giving more comfort to the patient. A soft-based material was chosen to protect
the primary teeth, as suggested by **Casamassimo**. Furthermore, the thickness (3 mm) was sufficient to prevent perforation and increase resistance to impact.[24,25]

Kato et al suggested a diagnostic criteria for recognizing patients with severe SB: recent history of tooth noise during sleep, occurring at least 3 to 5 nights a week for a period of 6 months; presence of tooth wear; discomfort or fatigue in the masticatory muscles in the morning; and hypertrophy of the masseter muscle in voluntary clenching. Studies assessing the prevalence of SB in children should adopt patient's complete history and a rigorous physical examination for the diagnosis of SB.[26]

Mittelman described the use of biofeedback in the management of bruxism. He described an EMG technique which provides the daytime clencher with auditory feedback from his/her muscle activity letting the individual to know the degree of muscle activity or relaxation that is taking place.[27]

Riolo et al. reported almost no relation between individual reporting of TMJ sounds and actual clinical joint noises that are audible when examining children. study found a significant association between

temporomandibular symptoms (anamnestic findings), as reported in questionnaires, and temporomandibular signs, as found in a clinical examination. This may be due to the fact that our questionnaire was designed to be filled out by the parents (in collaboration with their children) possibility indicating that initial screening of children for TMD should be done with parental assistance.[28]

Nilner and Lassing showed that functional disturbances of the masticatory system are common in children aged 7–14 years (with 64% experiencing pain upon muscle palpation and 39% experiencing pain on TMJ palpation). These values are substantially higher than the prevalence found in the present study . The difference between the two studies may be explained by the different age and/or different, ethnic/socioeconomic status of the study populations, differences between questionnaires, difference in the content and performance of the clinical examination, and other factors.[29]

Kampe and others, who also demonstrated the presence of a higher level of anxiety in agroup of people with bruxism.[30]

Lobbezoo and Naeije, level of stress and personality type have been included in the etiology of bruxism for many years. However, the exact contribution of psychological factors remains debatable. A controlled questionnaire study demonstrated that those with bruxism generally present emotional imbalance and tend to develop more psychosomatic disorders.[31,32]

Lindqvist conducted a study on 196 children aged 10-13 years to investigate existing differences in the prevalence of morphologic malocclusion between children with and without bruxism. The recorded types of morphologic malocclusion were Class I, II, and III molar relationship, overjet, and overbite. Bruxism was diagnosed
by the presence of atypical facets on the permanent teeth. The results showed no statistically significant differences in any type of malocclusion between children with and without recorded facets[33].

Egermark-Eriksson et al. followed up 238 of 402 children of the initial sample to investigate the relationship between functional malocclusion and bruxism. Their second examination, performed 4 to 5 years after the first, studied the children at 11,15, and 20 years old. The criteria to diagnose bruxism as well as to record
functional malocclusion were the same as those used in their cross-sectional study. The results showed no statistically significant correlations between any type
of occlusal interferences and dental wear or attrition.[34]

Gunn et al.studied the relationship between functional malocclusion and bruxism on 151 migrant children aged 6-18 years. Functional shift of the mandible was the only recorded type of functional malocclusion. Bruxism was identified as tooth grinding by an interview. No statistically significant correlation was reported between functional shift of the mandible and tooth grinding.[35]

Statistically significant correlations were found between different types of morphologic malocclusion such as Class II and III molar relationship, deep bite, overjet, and dental wear or grinding. The occlusal conditions are an etiologic factor of bruxism in children and adolescents. Therefore, it seems safe to assume that malocclusion cannot increase the probability of the occurrence of bruxism.[36]

Most authors suggest bruxism to have a multifactorial etiology. Basically, two groups of etiological factors can be distinguished, peripheral & morphological factors and central (pathophysiological and psychological) factors. Among the emotional features, anxiety has been the factor most often studied in children.[36]

Dental wear can be caused by digestive problems and physiological masticatory functions. Dental wear of natural teeth depends on variables such as structure and hardness of the dental enamel; charge applied to the contact surfaces, saliva, and duration of the contact.[37,38]

As this article deals with isolated clinical case, its findings cannot be generalized. Therefore, we suggest that more epidemiological investigations be made to provide a
better understanding of the etiological factors in bruxism.

V. Conclusion

Bruxism is becoming an increasingly common condition in children. In treating this parafunctional habit, clinicians play a leading role in determining possible etiological factors. In many situations it is the dentist's task to warn parents and institute multidisciplinary treatment. Recently, a multifactor model showing different etiological events had been accepted which includes genetic, neuro-physiological, psycho-emotional and pharmacological factors. Researchers will have to evaluate other aspects as well to determine the risk and consequences such as tooth damage and pain by bruxism.

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