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THE BIOLOGIC EFFECTS OF BRUXISM. REVIEW

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ABSTRACT

Traumatizing forces may act on an individual tooth or on groups of teeth in premature contact relationship; they may occur in conjunction with parafunctions such as clenching and bruxism, or in conjunction with loss or migration of premolar and molar teeth with an accompanying, gradually developing spread of the anterior teeth of the maxilla etc. The local, regional and systemic effects of bruxism include tooth wear, excessive stress on periodontal ligaments and alveolar bone, temporomandibular joint effects and even effects on the nervous central system.

Keywords: bruxism, stress, occlusal trauma

INTRODUCTION

Bruxism may cause occlusal forces on teeth susceptible to periodontitis to be increased in intensity and/or frequency, magnifying potential amplification of damage. Daytime occlusal parafunction is commonly limited to clenching of the teeth during incidents requiring a person's focused effort or mental concentration.

Night-time bruxing of the teeth can take the form of grinding the teeth in various excursions and/or clenching of the teeth. Sleep bruxism is probably an extension of the rhythmic masticatory muscle activity that is also observed in nonbruxers. Why nuclei in the brainstem allow bruxing to occur with some individuals while others are spared is unclear. Bruxing is associated with greater frequency and persistence of TMJ dysfunction, orofacial pain, and possibly periodontal attachment loss.

Sensory input of teeth subject to bruxism is probably dampened, which may interfere with both diagnosis and treatment. There seems to be limited influence on bruxing tendencies from occlusal interferences. Selective serotonin reuptake inhibitors, such as Prozac, have been reported to encourage bruxism.

Since bruxism is considered a possible etiological factor for TMD and tooth wear, its clinical importance is obvious. Other effects of bruxism may include tooth movement and tooth mobility, as well as changes in oral soft tissues and jawbone.

Tooth wear

Bruxism was for long considered a major cause of tooth wear. In recent years, however, the multifactorial etiology and the importance of other factors related to tooth wear, such as erosion, have been emphasized [1]. Nevertheless, a systematic review concluded that ''attrition seems to be co-existent with self-reported bruxism''[2]. Rather than confirming a relationship, this may be indicative of a

common perception among both patients and dentists.

For example, a positive self-response to a question about bruxism may simply reflect a preconception on the part of the patient, or the dentist, about the de facto existence of a causative relationship between tooth wear, and/or TMD-related symptoms for that matter, and bruxism. Indeed, when nocturnal bruxism has been diagnosed more robustly, with polysomnography, no consistent relationship has been found between bruxism and tooth wear, or between bruxism and TMD.

In fact, there have been suggestions that an inverse relationship may apply [3]. A review concluded that a number of published observations strengthen the concept of the multifactorial etiology of tooth wear. The review went on to state that it seemed fair to conclude that the overall significance of bruxism as a causative factor for tooth wear is not fully known, but it is even fairer to say that it is probably overestimated [1]. It follows that there are significant limitations with self-reports to provide a reliable diagnosis of sleep bruxism.

Therefore, the use of the term bruxism implies an acceptance of this limitation, and that what it refers to might equally be just heavy loading through high biting/chewing forces operating as a direct factor, rather than it being categorically due to parafunctional activity. Irrespective of the aetiology, restoration of worn teeth that will frequently involve prosthetic treatment will be needed in some patients [4].

Because such treatment is typically complex and often extensive, there is a tendency to defer treatment until the tooth wear is well advanced. This complicates treatment further, and with greater mechanical vulnerability to the restoration provided. There is a scarcity of studies on the outcome of prosthetic restoration of worn dentitions, leading to widely differing opinions among prosthodontists in different countries about how these complex treatment situations should be managed [5].

Biological effects on periodontium

Periodontal lesions represent the result of multifactorial interactions between the bacterial elements and the defence system of the host [6, 7]. In these interactions systemic factors play an important role [f8, 9, 10]. Nevertheless, local factors can act in a very destructive manner. Over the years, the role of occlusion and its dynamic interactive impact on the periodontium has been an issue of controversy and extensive debate.

The central focus has been on occlusal trauma resulting from excessive forces applied to the periodontium. In the animal studies, the majority of these early studies agreed that occlusal trauma, in and of itself, failed to result in pocket formation or loss of connective tissue attachment. One particular investigation identified the bacterial ‘plaque front’ as the agent responsible for the severity and sites of attachment loss and associated bony defects. In the absence of existing inflammation, it was noted that bone changes accompanying occlusal trauma might be reversed by discontinuing offending occlusal forces [11].

A co-destructive theory was proposed based on a zone of irritation (marginal and interdental gingiva and gingival and transeptal fibres) and zone of co-destruction (PDL, alveolar bone, cementum, transeptal and alveolar crest fibres). This theory suggested that plaque-induced gingival inflammation was confined to the zone of irritation. Occlusal forces or traumatogenic occlusion affected the zone of co-destruction but did not cause gingival inflammation.

However, occlusal trauma together with plaque-induced inflammation acted as co-destructive forces resulting in an alteration of the normal pathway of inflammation and the formation of angular bony defects and infrabony pockets. Some researchers agreed with this theory of co-destruction and indicated that local inflammation of the PDL might be induced mainly by periodontal bacteria, and mechanical stress might promote local inflammation [12].

Many cytokines are believed to participate in periodontal injury [13, 14]. Receptor activator of nuclear factor kappa B ligand (RANKL) is an important factor in osteoclast differentiation, activation and survival. Some researchers investigated the distribution of RANKL expressing cells in rat periodontium during lipopolysaccharide-induced inflammation, with or without occlusal trauma. The results demonstrated that RANKL expression on endothelial cells, inflammatory cells and PDL cells was involved in inflammatory bone resorption and the expression was enhanced by traumatic occlusion. It suggested that RANKL expression on these cells was closely involved in the increase in osteoclasts induced by occlusal trauma [15].

Osteopontin is known to be produced upon mechanical loading and is considered to induce the migration of osteoclasts to the resorption site. RANKL is one of the essential factors for osteoclast maturation and induces the constitutive induction of intra-cellular osteopontin in vitro. However, no correlation between RANKL distribution and osteopontin production in osteoclasts could be found [16].

Biological effects on alveolar bone

Traumatic occlusal forces have been considered as one of the local factors that accelerate the inflammatory alveolar bone resorption when periodontitis is present. The alveolar bone loss as a result of occlusal trauma on periodontitis could represent an irreversible codestructive effect or could merely be a functional adaptation of the periodontium. Some researchers identified sequential changes of bone resorption and formation after the application of experimental traumatic occlusion.

The bone resorptive activity produced by occlusal trauma was enhanced by oestrogen deficiency and nicotine that is manifested as a generalised skeletal osteopenia [17]. Other authors evaluated the influence of diabetes mellitus on bone response in the furcation area of teeth subjected to occlusal trauma in the presence or absence of experimental periodontitis in the rat model. They came to the conclusion that diabetes mellitus enhanced bone loss in the presence of occlusal trauma associated with experimental periodontitis [18].

Frost et al. [19] reported the cellular reaction of bone to different microstrain levels. Excessive bone strain may cause bone cellular resorption. Therefore, the hypothesis that occlusal stresses beyond the physiologic limits of bone may result in bone strain that is significant enough to cause bone resorption is plausible from a cellular biomechanics standpoint [20]. Increased tooth mobility may be the adaptation of the periodontium to occlusal forces and may not be pathologic in the absence of inflammation.

Connective tissue attachments may be maintained with mobile teeth. Increasing tooth mobility is a sign of pathology. Occlusal forces producing tooth hypermobility may accelerate attachment loss in progressive periodontitis. Some researchers identified sequential changes of bone resorption and formation after the application of

experimental traumatic occlusion. They found that 3D tensile- and compressive-stress distributions are specifically determined within the internal and external aspects of the PDL and influenced by the tooth surface in relation to the load condition.

Moreover, the stress magnitude, nature, direction and distribution generated in the PDL due to different occlusal loading conditions might enhance the understanding of the biological reaction of the PDL in health and disease [21].

Biological effects on masticatory muscles

To explore the relationship between improper occlusion (occlusal trauma) and masticatory muscle pain, some researchers created an occlusal trauma animal model by directly bonding a crown to a maxillary molar to raise the masticating surface of the tooth in rats. They raised the occlusal surface to three different heights (0.2, 0.4 and 0.6 mm), and for one month, they quantitatively measured mechanical nociceptive thresholds of the temporal and masseter muscles on both sides. Results showed a stimulus–response relationship between the height of occlusal interference and muscle hyperalgesia. Removal of the crown 6 days after occlusal interference showed that the removal at this time could not terminate the 1-month duration of mechanical hyperalgesia in the masticatory muscles [22].

Nishide et al. [23] investigated the histological alterations of rat masseter muscles following experimental occlusal alteration with unilateral bite raising. A total of eight male adult Wistar rats were equally divided into control and experimental groups. The experimental rats wore bite-raising splints on the unilateral upper molar. Four weeks after the operation, the anterior deep masseter muscles were removed and then stained with succinic acid dehydrogenase, haematoxylin–eosin and myofibrillar ATP-ase.

Most of the muscle fibres in experimental rats remained intact, although partial histological changes were observed, such as extended connective tissue, appearance of inflammatory cells in the muscle fibres and existence of muscle fibres with central nuclei and central cores.

Moreover, the fibre area–fibre frequency histograms of experimental muscle indicated a broad pattern than that of controls. These results indicated that occlusal interference caused histological changes in masseter muscles and that this might be related to the fact that the masseter energy level was reduced during masticatory movements in unilateral bite-raised rats.

Some researchers also found that the central neuronal sensitisation in the brainstem might play an important role in the masticatory muscle pain induced by occlusal trauma [24]. To assess mechanical hyperalgesia of the bilateral masseter muscles following the induction of experimental unilateral malocclusion, acute and chronic traumatic occlusion was induced in rats. In the gradually induced malocclusion group, hyperalgesia was induced in bilateral masseter muscles from 3 to 9 days, and the peak time was the 7th day.

However, in the acute traumatic occlusion group, the ipsilateral masseter muscle was demonstrated hyperalgesia for 5–7 days, and the peak time was the 2nd day. The results suggested that traumatic occlusion might lead to masseter muscle hyperalgesia, and there were some differences for pain behaviour between the acute and the chronic occlusion trauma [25]. Occlusal features can affect the electrical signal recordings of masticatory muscles [26]. Further research in this area is strongly requested to determine whether this altered

muscular activity can turn in the occurrence of temporomandibular disorders (TMDs).

Biological effects on temporomandibular joint (TMJ)

There is no consensus on the association between occlusion and TMDs. Several studies have failed on proving that occlusion has nothing to do with TMD. Some have argued that little or no relationship exists between faulty occlusion and TMDs. Some authors still consider occlusal alterations necessary for the onset of TMD symptoms [27].

It was reported that degenerative changes of TMJ tissues were found following occlusal trauma in rabbits [28]. The articular surface of the condyle was damaged, and the chondrocytes showed signs of degeneration. The synovial lining cell consisted of a number of vimentin intermediate filaments (IFs). The vermiform body in the deeper interstitium was also found in this experiment, which indicated that the occlusal trauma might really be a factor inducing degenerative changes of the TMJ.

Large amounts of IFs had been reported in the cells of the articular cartilages, in intervertebral disc and in the cells of some disease states such as osteoarthritis and chondromalacia. It was believed that the cells respond to the changes in the mechanical environment by developing integrin and IFs, which was a major component of the synovial lining cell in TMJ degeneration, and it is commonly seen on the cell membrane.

Moreover, vermiform body has been demonstrated to be collections of abnormal amounts and types of elastic tissue. The distribution suggested a stress elastosis, which might contribute to the loss of mechanical integrity of articular surfaces in arthropathy. It was also reported that a working-side interference had an immediate, significant effect on the working side condylar movement [29]. However, the acceptance of theories about the multifactorial aetiology of TMD has resulted in less emphasis being placed on occlusion as a TMD-related factor.

Some researchers investigated the effects of an experimentally induced increase in the occlusal vertical dimension (iOVD) on the functional characteristics of TMJ mechanoreceptors in rats [30]. Sixty 13-week-old male albino Wistar rats were divided into control and iOVD groups. The vertical dimension between the maxillary and mandibular molars in the iOVD group was increased by 2.0 mm with a build-up of resin on the maxillary molars.

The results suggested that TMJ mechanoreceptors in adult rats might ultimately adapt to iOVD. It has often been suggested that inputs from the TMJ mechanoreceptors are involved in the physiologic mechanism of occlusal vertical dimension (OVD) regulation. Thus, TMJ mechanoreceptors play a role in regulating mandibular position. In this study, there was no significant difference between the sensitivity of the TMJ mechanoreceptors in the normal rats and those that underwent long-term iOVD.

However, the rat TMJ has a different character compared with the human one. For example, a unilateral condylectomy in rats reduced growth of the mandible and a subsequent lateral shift to the affected side, but a functional appliance eliminated the reduced growth and the lateral shift of the mandible, and prominent regeneration of the condyle also occurred. These are not adopted in human condyle [31]. Thus, caution should be exercised when the present findings are applied to humans.

Biological effects on central nervous system

In the last few decades, the effect of occlusal trauma on the oro-facial tissue

has been reported in many studies. However, less attention has been paid to the possible effect of occlusal trauma on the nervous system. In recent years, it has been shown that traumatic occlusion has effects on the sensitisation and conduction of primary sensory neurons, which comprise nearly half of the total CNS [32]. Both the production of excitatory neurotransmitters in primary sensory neurons and the release of neurotransmitters from central nerve endings increase during occlusal trauma. These results indicated that the sensations of primary sensory neurons were enhanced and that nociceptors were sensitized during occlusal trauma. It suggested that sensitisation of primary sensory neurons was a cause of chronic oro-facial pain following occlusal trauma [33]. Similarly, other studies indicated that occlusal interference was directly related to masticatory muscle pain and that central sensitisation mechanisms were involved in the maintenance of the occlusal interference–induced mechanical hyperalgesia.

Nerve growth factor is an important mediator in the generation of inflammatory hypersensitivity. It has been proposed that peripheral up-regulation of NGF contributes to sprouting of primary afferent terminals and to increase expression and peripheral content of neuropeptides [34]. Periodontal and pulpal sensory information evoked by nociceptive mechanical stimulation is processed mainly within the trigeminal ganglion (TG).

It has been shown that NGF binds the high-affinity neurotrophic receptor TrkA through a cytokine-like function. NGF up-regulates expression, promotes the release of several genes including CGRP and pre-protachykinin-A (PPTA) in TG, which are involved in nociception, and excites primary afferent terminals in the oral-facial area. The up-regulated expression of NGF and TrkA genes and proteins suggested a possible role of NGF in the initiation of oro-facial pain induced by traumatic occlusion [33].

Several reports have suggested the involvement of astrocytes in the modulation of nociception. Over the last decade, astrocytes have also been recognised as being involved in spinal nociceptive processing and sensitisation [35]. Glial fibrillary acidic protein (GFAP), produced as an intermediate filament protein, has been widely used to monitor astrocyte reactivity in response to nociceptive stimulation. Activated astrocytes can produce and release proinflammatory cytokines (TNF, IL-1 and IL-6) and neuroactive substances (leukotrienes and excitatory amino acids).

Together, these substances regulate the conduction of noxious stimuli [36]. Some researchers examined by immunocytochemical methods and nociceptive behaviour assessment in rats whether astrocytes in the parabrachial nucleus (PBN) were involved in the regulation of traumatic occlusion [37]. The expression of GFAP in PBN of ipsilateral and contralateral sides was up-regulated 4 h after occlusal changes in molars, reached peak levels at 24 h and was then gradually down-regulated.

PBN astrocytes activated by traumatic occlusion were found to have enlarged cell bodies and thickened processes within 8 h. An inhibitor of glia metabolism (FCA and fluorocitrate) reduced astrocyte activation and significantly attenuated the development of pain hypersensitivity in this model.

The results suggested that the GFAP-immunoreactive astrocytes in PBN within the bridge of Varolius were activated by traumatic occlusion and that they were involved in the transmission and modulation of nociceptive information in the CNS. However, although astrocytes in PBN were thus probably involved in

causing post-occlusal hyperalgesia, the researchers had not been able to exclude that astrocytes at other locations also contribute to this effect [38].

Previous studies have shown that occlusal disharmony induces chronic stress, which results in learning deficits in association with the morphologic changes in the hippocampus, for example, neuronal degeneration and increased hypertrophied GFAP–positive cells [38].

Moreover, the aged bite-raised mice showed decreased acetylcholine release in the hippocampus and a reduced number of choline acetyltransferase immune-positive neurons in the medial septal nucleus compared to age-matched control mice [39]. These findings suggested that the bite-raised condition in aged SAMP8 mice enhanced the age-related decline in the septohippocampal cholinergic system, leading to impaired learning. Amyloid-b deposition and its neurotoxicity in areas of the brain, such as the hippocampus, play a causative role in Alzheimer's disease (AD) [40].

Some researchers indicated that psychological stress induced by occlusal disharmony reversibly induced amyloid-b 40 and 42 in the rat hippocampus through the glucocorticoid signal.

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.

Correction of these occlusal overloads and interferences will typically avert these signs and symptoms. The presence of occlusal interferences and overloads does not necessarily indicate that the signs and symptoms of the clinical entity called occlusal trauma are present: They may take many months before emerging in the clinical context and in different imaging methods.

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