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Review

Effects of occlusal trauma on the periodontium, alveolar bone, temporomandibular joint and central nervous system

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Abstract. The studies published over the years have shown an interest in occlusal trauma, as well as a consequence of excessive forces applied to periodontal tissues. The symptoms of occlusal trauma occur only in situations when the intensity of the force is so great, that the periodontium of the tooth exposed to it cannot withstand the forces and cannot distribute the resulting forces without inducing changes in position and stability in the tooth involved.According to several authors, occlusal trauma is frequently considered as a broader spectrum of situations that include lesions of all the components of the maxillary-dental system produced by normal or dysfunctional occlusal interrelationships. Several studies have shown that occlusal trauma can have destructive pathological effects on the periodontium, alveolar bone, masticatory muscles, dental structure, temporomandibular joint (TMJ), and even on the central nervous system.To study the effects of occlusal trauma on the maxillary-dental system, animal models were used. Experiments have been carried out on various animal models exhibiting occlusal trauma. Pigs, monkeys, rats, beagle dogs and rabbits were used to replicate occlusal trauma using crowns or orthodontic wires applied to the posterior teeth.

Key Words: occlusal trauma, periodontium, TMJ, alveolar bone, central nervous system

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1. Effects of occlusal trauma on the periodontium

Most authors (Komatsu & Chiba 1993; Pini et al 2004) accept Glickman's findings according to which occlusion-induced trauma is an aggravating factor for the progression of the periodontal disease.

Rosling et al (1976) consider that the occlusal trauma is an important etiological factor in cases where angular bone defects combined with infraosseous pockets are located at the level of one or more teeth.

Burgett et al (1992) observed that selective grinding in order to reduce occlusal trauma resulted in a 0.4 mm reduction in the average probing depth compared to the situations in which patients who did not undergo occlusion adjustment over a 6-year period.

A report on selective occlusal grinding in periodontal therapy has revealed no encouraging evidence for it to be used as a routine procedure for maintaining the periodontal health. No encouraging evidence for selective occlusal grinding has been found in a report analyzing this issue to be used as a routine procedure. The authors of this study have concluded that selective does not have a preventative role, but in combination with periodontal therapy it can improve comfort and the occlusal grinding (Foz et al 2012).

In addition to the presence of angular bone defects and infraosseous pockets, increased mobility is frequently referred to as an important sign of occlusal trauma. In a clinical trial carried out by Rosling et al (1967), patients with advanced periodontal disease associated with multiple angular bone defects and mobile teeth have undergone antimicrobial therapy. Healing was evaluated by measurement of the level of the gingival-cervical periodontal insertion and by radiological monitoring. The authors reported that the infraosseous pockets localized at the level of mobile teeth showed the same healing degree as those associated with teeth without mobility.

Ismail and colleagues (1990) conducted a study in which a group of 165 subjects were reexamined after a 28-year period and evaluated the loss of the clinical attachment level. They found that age, smoking and dental mobility were the factors most closely related to the loss of periodontal attachment level.

Other authors (McGuire & Nunn 1996) reported that parafunction and mobility were correlated with a lower prognosis and dental loss, 5-8 years posttreatment.

Jin and Cao (1992) no showed differences in measurement of the clinical attachment level, or degree of bone loss in teeth with or without altered occlusal contacts. Instead, the teeth with mobility or increased desmodontal space presented more significant losses in the alveolar bone and an increase in the depth of the periodontal pockets.

It has also been found that this mobility may have a negative effect on post-surgical healing, thus affecting the periodontal reinsertion process (Fleszar et al 1980).

Following certain studies, Ericsson and Lindhe (2008) have observed that, within certain limits, a healthy periodontium that exhibits low insertion height has the ability to adapt to altered functional load exertion, similarly to a normal height periodontium.

Other authors (Wennström et al 1987) have demonstrated that orthodontic forces that produce total translation or dental tipping can lead to gingival recession and loss of periodontal attachment level.
The relationship between the periodontal disease and occlusion has long been discussed. According to a report by the American Academy of Periodontology (1999), the role of occlusal trauma in the evolution of the periodontal disease remains controversial. Glickman and Smullow (Lindhe & Thorkild 2008) have argued that the occlusal trauma is a contributing factor in the evolution of periodontitis induced by the bacterial plaque.

2. Effects of occlusal trauma on the alveolar bone

The authors (McCulloch & Predrag 2000) have found that the response of the cortical bone is related to the tension sensitivity rate. Asundi and Kishen (2000) have compared the distribution of tensions in the dental root and support bone. They have noticed higher tensions in the cervical region and lower ones in the apical region. The findings suggest that the periodontal dental ligament and the alveolar bone are closely correlated in distributing the masticatory forces.
The occlusal forces transmitted by the periodontal ligament to the alveolar bone are supported by spongy trabeculae maintained and supported by the vestibular and lingual compact bone tissue layer.
For good functionality it is necessary to keep a well-balanced relationship between the alveolar bone and the occlusal forces. The alveolar bone reaction occurs depending on the direction, intensity and duration of the forces. Under their action, the root of the tooth creates tension and compression zones on the alveolar walls, through the periodontal ligament. The vestibular and lingual walls of the alveolus are elastically deformed towards the action of the occlusal forces resultant. Occlusal forces can shape the alveolar bone by resorption and bone apposition, altering the height, number and thickness of the trabeculae. Thus, traumatic occlusal forces are considered as one of the local factors that accelerate bone resorption (Zhu et al 2004).
Loads exerted upon the bone induce stresses that generate signals that can be detected by specialized cells. Then, they release chemical mediators that induce bone remodeling by autocrine or paracrine mechanisms (Carter& Beaupre 2001; Kaku et al 2005). Bone stress reception is mediated by genetically determined thresholds that control the bone modeling and remodeling processes. These processes involve the presence of osteoblasts and osteoclasts. Osteoclasts are essential cells in the periodontal destruction process developing under the exertion of excessive occlusal forces. Osteoclasts produce osteopontin, which is responsible for resorption processes. Osteopontin is a noncollagenous abundant protein produced during mechanical stresses. In vivo studies have shown that osteocytes are the main cells responsible for the production of osteopontin during orthodontic treatments. The results of the study showed that the bone resorption rate was lower for intermittent forces than for continuous forces (Merry et al 1993; Dodds et al 1995; Zhu Suzuki 2004).
The scientific reasoning that occlusal trauma increases bone loss has been a topic of interest in the last decade. The stimulation of the ligand of the activator receptor of the kappa beta nuclear factor (RANKL) appears to be closely related to the bone resorption induced by occlusal trauma (Yoshinaga et al 2007). RANKL is considered essential for the differentiation and maturation process of osteoclasts, initiating bone resorption. It is secreted by osteoblasts and transforms osteoclast precursors into mature osteoclasts. This suggests the possible involvement of RANKL in intracellular osteopontin production in osteoclasts. In a study conducted in rats aiming to induce occlusal trauma, an association between RANKL and osteoclasts, and osteoblasts respectively was demonstrated (Miyamoto & Suda 2003; Takeshitaet al 2000).
Periodontal ligament cells exposed to mechanical action contribute to increasing the production of interleukin IL-6, which is a potential stimulant of RANKL, of periodontal inflammation and bone resorption (Kita et al 2011).
Kawamoto and Nagaoka (2000) have shown that ovariectomized rats loaded to experimentally induced occlusal trauma presented significant bone resorption surrounding by the periodontal ligament. Other authors (de Oliveira Diniz et al 2012) have confirmed that diabetes mellitus increases bone resorption when occlusal trauma is also involved.
Nicotine also contributes to the acceleration of bone loss (Campos et al 2014; Nogueira-Filho et al 2004).
Studies in rats have suggested that occlusal equilibration for some groups, like as those with estrogen deficiency, diabetes mellitus and exposure to nicotine, prove beneficial or, at least, worthwhile researching in more detail.
Under normal conditions, the intensity of the occlusal forces allows the maintenance of vitality of the periodontal ligament cells. On the bone surface of the alveolus, osteoclasts shortly appear in the pressure zone, thereafter direct bone resorption occurs.
If the applied load has a higher magnitude, ligament necrosis occurs in the pressure zone associated with indirect bone resorption until the hyalinized tissue penetrates the pressure zone. Prolonged functional stress, in conjunction with disease-induced inflammation, on increased pressure points, is believed to accelerate the degradation of soft and hard tissue. Recent studies (Jeremy et al 2014) have shown that the main features of periodontitis which generate the biomechanical instability of the tooth are: bone resorption of the residual ridge, deterioration of the proximal transseptal fibers of the adjacent tooth and deterioration of the periodontal insertion.
The same study (Jeremy et al 2014) reports that elevated matrix metalloproteinases (MMP) and RANKL. MMP are responsible for the destruction of the extracellular matrix of connective tissue from the dento-alveolar space and for the stimulation of bone resorption. These aspects are essential for withstanding fracture of the dento-alveolar articulation while the latter undergoes a greater intensity effort and a higher action speed.
With the expansion of the use of dental implants over the last few years, occlusal trauma and overload in peri-implantation bone resorption has started to be investigated. Studies on monkeys (Miyata et al 2000; Miyata et al 2002) have shown that, as with natural teeth, occlusal forces do not induce inflammation, produce bone resorption, over which the inflammatory factor can overlap. In this case, the evolution of the degenerative process becomes co-factorial.
A similar study on dogs (Reinhardt & Killeen 2015) has presented the effects of occlusal trauma on implant which suggested that occlusal force alone do not cause a peri-implant tissue.

When overload and plague are presented, appear the periodontal pockets and the loss of alveolar bone.
Recent attempts to mimic shock absorption by the dento-periodontal ligament have shown that insertion of a material around the implant can attenuate the increased forces.

3. The effects of occlusal trauma on the temporomandibular joint

When observing the masticatory cycles of a person with temporomandibular pain, a pattern with lower repetitiveness can be detected, with slow, irregular, but repeated trajectories, and associated with the altered functional movements of the condyle around which pain is concentrated.
The temporomandibular dysfunction shows symptoms such as: TMJ pain, the limitation of the mandibular movements and the presence of clicking during mandibular movements. The occlusal factors, parafunctions, and stress are considered possible causes of the temporomandibular dysfunction. The role of occlusive etiopathogenic factors, such as occlusal interferences, has sparked controversy among specialists.
Some authors (Alanen 2002, Badel et al 2012) consider occlusal disorders as a major etiological factor in initiating the symptoms of the temporomandibular dysfunction, while others (Tsukiyama et al 2001; Landi et al 2004)suggest that these disorders are only one of the many factors associated with this dysfunction. The first group of aforementioned authors have stated that the critics of this theory confuse the two notions, namely: "sufficient cause" and "causal factor". It is possible that the studies, by which artificial interference was induced into healthy patients, may have underestimated the role of occlusive factors because there are healthy individuals with a higher pain threshold who naturally tolerate interference. Hence, they have a greater adaptation capacity to pulses of artificially induced interference. The hypothesis is confirmed by a study in which patients, who are in the remission phase of dysfunctional symptomatology, showed reduced adaptability to artificially induced interference. Current studies do not exclude a possible cause- effect relationship between the occlusion and the temporomandibular dysfunction.
From a biomechanical standpoint, certain secondary malocclusions occurred after the early extraction of the first mandibular molars. They are accompanied by the pathological migration of the adjacent and antagonistic teeth. Moreover, they may also produce medial or lateralotrusive interferences. These malocclusions represent more important risk factors than certain dento-maxillary anomalies.
The presence of interferences is considered a predisposing factor by some authors (Fu et al 1998). Other authors (Minagi et al 1997), suggest that these interferences can act as a protective factor, while other studies (Dworkinet al1990; Adler 1993) report close links existing between the two factors. It is certain that occlusal alterations are potentially generating symptoms associated with the temporomandibular dysfunction.
Other studies (Fu et al 1998) have reported the existence of degenerative articular changes following the occlusive trauma induced in rabbits. The joint surface of the condyle was affected and chondrocytes presented signs of degeneration.
Some researchers (Naito et al 2011) have evaluated the effects of the increase of the vertical dimension of the occlusion upon the functional characteristics of the articular baroreceptors in rats. The results suggested that at an increase in the 2-mm dimension, the articular baroreceptors can adapt. The impulses from the articular mechanoreceptors are involved in the physiological mechanisms of OVD regulation, the periodontal baroreceptors having an important role in the regulation of the mandibular functional positions.
Disturbance of the correlative relationship between the condyle and the meniscus during mandibular movements may produce an atypical condylar displacement with pressure on the condyle and discus (Morita et al 2016, Huang et al 2006). Since dental contact should not occur on the passive side during mastication, the mandible is maintained functional in three points during the chewing cycle, namely: the two joints and the dental guidance of the active side in the lateral movements, and the two TMJs and the anterior guidance in the propulsion movement. The mandible shows stability if the resultant of the forces exerted by the elevator muscles falls within the support triangle. If this does not occur, one of the three aforementioned points will separate as the mandible undergoes a pivotal or rotation motion around the axis joining the other two points.
In conclusion, most authors believe that dental interferences are an etiological predisposing factor, which triggers the temporomandibular dysfunction.

4. Effects of occlusal trauma on the central nervous system

The occlusal trauma has effects on the sensitivity and mode of conduction of neuronal impulses from the dentoalveolar complex to the primary sensitive neurons. The study showed that both the production of excitatory neurotransmitters in primary sensory neurons and the release of neurotransmitters from central nerve endings increased during occlusal trauma. These results indicated that the sensations of primary sensory neurons were enhanced and that nociceptors were sensitized during occlusal trauma (Zhu et al 2004; Woolf 1994).
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. Pulpal and periodontal sensory information, augmented by nociceptive biomechanical stimulation, is mostly processed in the trigeminal ganglion (Dong et al 2004; Dong et al 2010). Nerve endings continually induce nerve impulses produced by occlusal pressure. Occlusal interference may activate the receptors which produce NGF synthesis. They determine changes in the immune reactivity and the primary corresponding endpoints in the maxillofacial area (Chen et al 2007; Moria et al 2016).
Immunohistochemical methods have demonstrated nociceptive behavior in rats and have assessed whether the astrocytes in the parabrachial nucleus are involved or not in regulating occlusal trauma.
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. GFAP in the parabrachial nucleus of the same and contralateral side increased

4 hours after the increasing of the occlusion, occurred peak levels at 24 h and was then gradually down-regulated. The results suggested that GFAP-immunoreactive astrocytes in the parabrachial nucleus in the pons Varolius were activated by the occlusal trauma and were involved in the transmission and modulation of nociceptive information in the central nervous system (Chen et al 2007; Liu et al 2013).

The occlusal trauma could cause a variety of harmful biological effects on stomatognathic system.

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