Diagnosis of occlusal trauma: Extrapolations for peri-implant bone region can be done

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Abstract

Images provide a language to describe the dynamics of bone and tissue. Bone density and space distribution vary and indicate greater or lower reaction and adaptation to functional demands, such as masticatory loads, on natural teeth or osseointegrated implants. In rehabilitation, load distributions have to be planned, and the remaining teeth and their relation with neighboring bone should be evaluated. The detection of bone responses to pre-existing occlusal trauma may provide a more accurate evaluation of masticatory conditions and parafunctional habits, that is, a true functional history of remaining teeth. Occlusal interference and overloads take months or years to induce classical signs and symptoms of occlusal trauma as a clinical entity. When a tooth has pulp necrosis and signs of occlusal trauma, the evaluation of history, as well as all tests, should be directed to the diagnosis of superposed dental trauma even when posterior teeth are affected. There is no scientific basis to confirm that occlusal interferences and overloads lead to pulp necrosis. A frequent question: Up to what point should orthodontic forces be applied to osseointegrated implants? Orthodontic forces are not greater, in any situation, than occlusal forces in terms of intensity, amplitude and variability. If an implant can bear masticatory loads, it may also receive orthodontic forces resulting from anchorage.

Keywords: Occlusal trauma. Occlusion. Gingival recession. Tooth trauma. Abfraction.

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Introduction

Bone remodels constantly, and the whole skeleton is fully renovated at a mean of 4 to 10 years, depending on the age of the person under examination.

Constant remodeling offers bone the opportunity to adapt to daily functional demands. Greater or lower trabeculae density and greater or lower cortical thickness are directly associated with the functional demands applied to each region.

Occlusal trauma is a form of rearrangement of bone and periodontal structures to respond to greater functional demands:

1) Expansion of the periodontal space to make the ligament broader so that the fibers stretch more and absorb forces better.
2) Thickening of the alveolar cortical or lamina dura for a firmer insertion of periodontal fibers.
3) Increase of bone density around the periodontal ligament to accommodate to forces that have greater frequency or intensity.

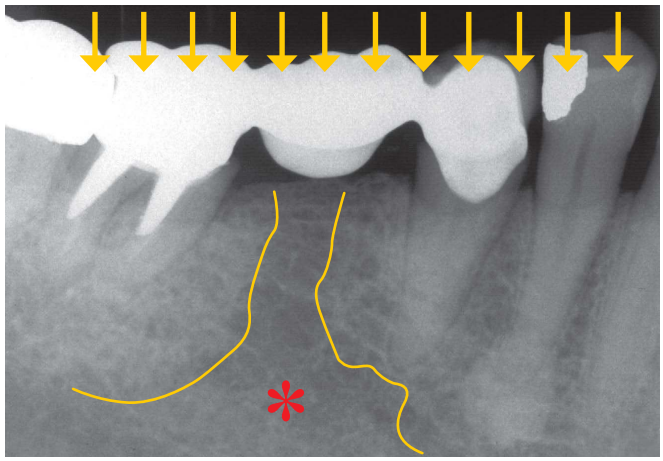

Figure 1 - The bone area which receives masticatory forces present denser trabecular bone, with thicker trabeculae and smaller marrow spaces — a process known as sclerosis or bone condensation. In some places, the spaces are not even identified in radiographic images, such as in the first premolar periapical region. In the area outside the lines, bone sclerosis appears more than in the central region defined by the two lines and the asterisk, clearly by the lack of masticatory load in the site. Bone dynamism meets the functional demands.

Figure 1 describes this bone dynamics: In areas of masticatory function, bone increases its density and becomes focally sclerotic, whereas bone trabeculae become thinner and marrow spaces larger in toothless areas.

Around an implant that receives loads (Fig 2), there are also adaptations similar to those seen in trauma or occlusal overloads. Around submersed osseointegrated implants, peri-implant bone has no trabecular density increase immediately surrounding its osseointegration interface.

Bone biology and physiology fascinates us in our attempt to understand the signs and symptoms of a known clinical event, as occlusal trauma, and to see how they occur, particularly in the areas of peri-implant bone.

Occlusal trauma: Concepts and undue comparisons

Occlusal trauma and its clinical and imaging variables are hardly seen in the training programs for undergraduates and graduates. Consequently, an accurate diagnosis and its clinical implications are often ignored when planning and following up some clinical cases.

Moreover, several specialists believe that the causes and possible progression of occlusal trauma are similar to tooth trauma and orthodontic movement, but they are, in fact, completely different entities. The tissue lesions induced by these three events are very different,[9] and their differences may be understood by analyzing the details of the function and three-dimensional aspects of periodontal structures, as shown in Figure 3, designed by Krstic.[14]

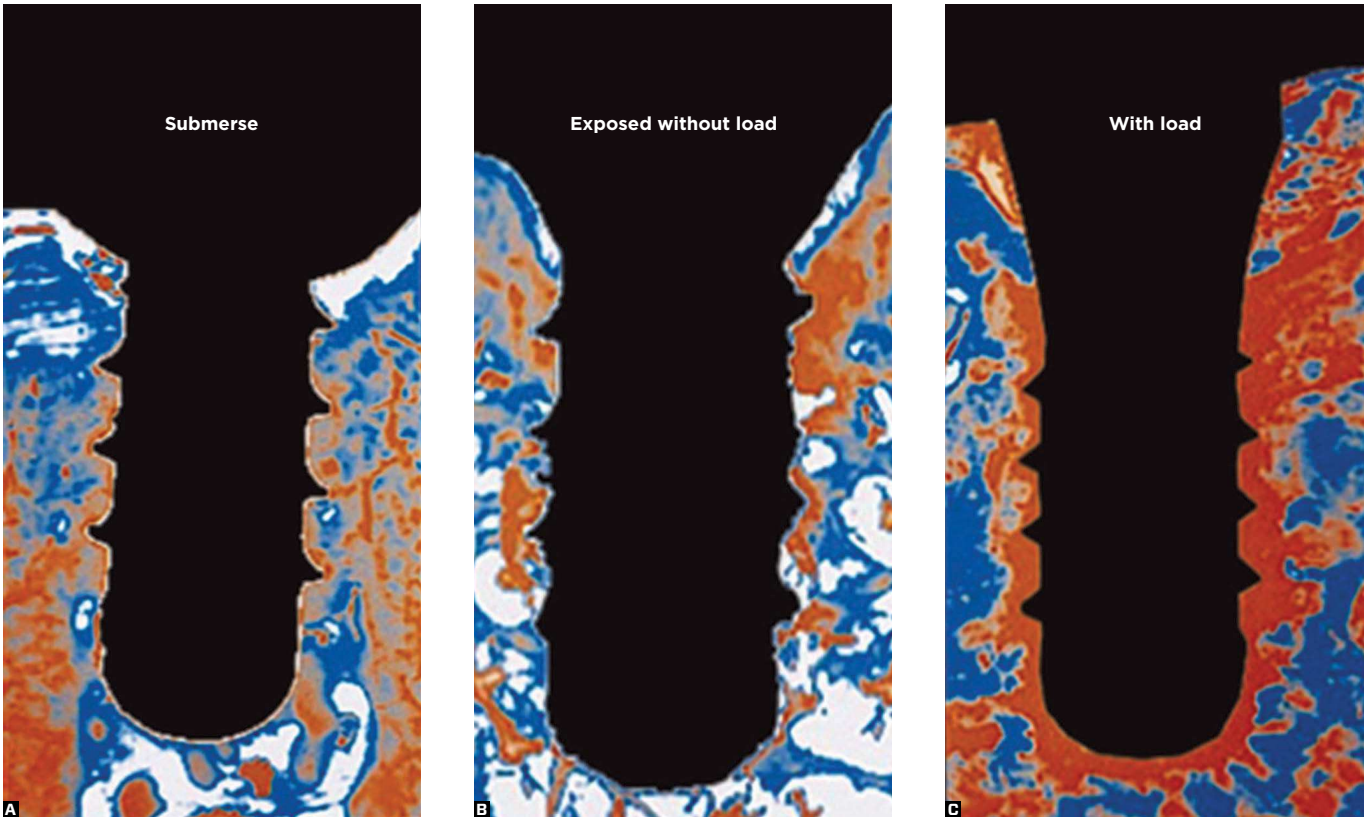

Figure 2 - In these images from Akin-Nergiz et al,[1] the radiographic densitometry in bone areas that received osseointegrated implants reveal differences when they are not subjected to load, for being submerse (**A**); when exposed, but with no masticatory load (**B**); and when exposed and subjected to occlusal loads (**C**). Bone density increases significantly (red areas) when osseointegrated implants receive masticatory load if compared to the original alveolar cancellous bone, in blue.

Orthodontic movement cannot be compared with occlusal trauma.[9] Cell and tissue changes resulting from occlusal trauma in periodontal tissues are completely different from those induced by orthodontic movement.

In dental trauma, forces are abrupt and intense, and their duration is short; the damage caused by dental trauma is the rupture of periodontal components; teeth make contact with or their roots go through the alveolar bone structures, and there is hemorrhage and necrosis of supporting tissues.

These three conditions — occlusal trauma, orthodontic movement and dental trauma — have in common only the physical nature of their causes, translated into forces, although with different characteristics, and no comparisons can be made between the lesions induced in the tissues.

We have set to publish a series of articles about occlusal trauma in journals dedicated to the various clinical specialties. Our purpose is to provide applied explanations and contribute to the specific understanding of the clinical and imaging signs of occlusal trauma in each area of expertise. Several parts of these articles are naturally repeated in their context, text and figures.[5-8] For each area, extrapolations and specific implications are discussed.

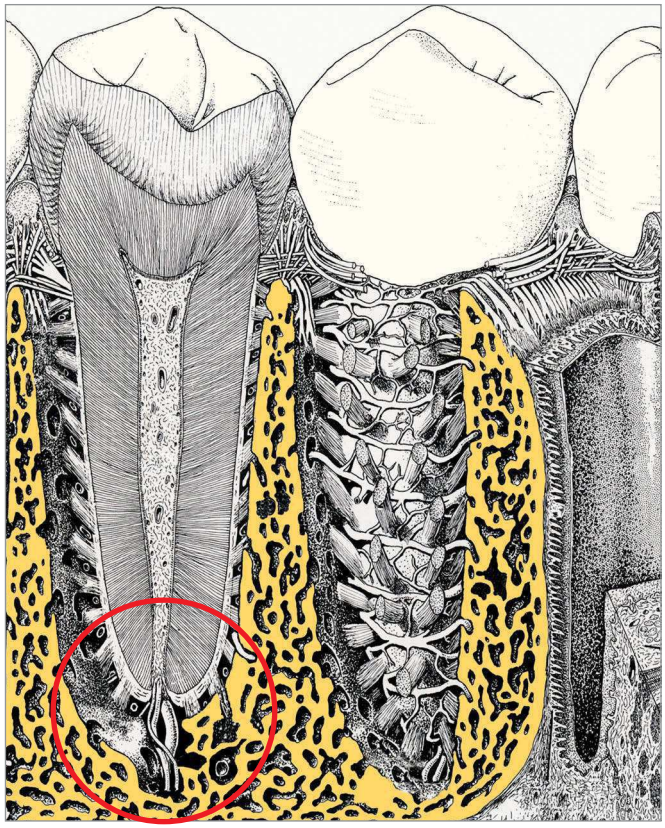

Figure 3 - Two-dimensional and tridimensional aspects of ligament and other periodontal structures. It is highlighted the conformation and extreme organization in the distribution of the collagen fiber bundles, which prevent, even in overload situations, the apex to reach the end of the alveolus (modified from Krstic,[14] 1991).

Occlusal trauma as a clinical condition or clinical entity

The clinical condition or entity known as occlusal trauma has several synonyms, such as occlusal trauma, traumatic occlusion, traumatogenic occlusion, periodontal trauma and occlusal overload.

The name of a certain clinical condition is an attempt to describe the type of lesion or set of changes that its causes produce in the tissues affected. Terminological accuracy and standardization facilitate the retrieval of information available in databases and promote communications between scholars and researchers.

The term lesion means any and all transitory or permanent structural change regardless of its nature. Lesions in periodontal tissues that characterize occlusal trauma may be induced by traumatic occlusion or an overload of occlusal forces upon a single tooth or several teeth simultaneously, depending on each clinical condition under analysis.

The induced lesion known as occlusal trauma, which is a disease or clinical entity, has been classically defined:

1. By Stillman, in 1917,[17] as the result of a situation in which the act of occluding the dental arches leads to lesions in tissue that support teeth.
2. By the World Health Organization (WHO), in 1978, as the damage induced to the periodontium by the pressure of teeth directly or indirectly produced by antagonist teeth.[15,16]
3. By the American Association of Periodontics, as a lesion to tooth-supporting structures resulting from excessive occlusal forces.[15]

The three concepts of occlusal trauma share a characteristic: The damage should result from overloads produced by occluding teeth and by antagonist teeth.

Occlusal trauma in one or more teeth may be associated with parafunctional habits, such as grinding and bruxism. In clinical practice, the causes of occlusal trauma may be associated with premature contacts due to tooth position, inadequate occlusal morphology of antagonist teeth, overloads on lateral incisors when involved in lateral canine guide, and after orthognathic surgeries.

No occlusal interference, such as premature contacts, should be confused with occlusal trauma, a clinical entity or a characteristic condition. Occlusal interference may be the cause of occlusal trauma, but the term occlusal trauma should only be used to identify the clinical condition and its signs and symptoms. Occlusion may be trau-

matic, but may not yet have induced the lesion or disease called occlusal trauma.

Occlusal trauma should not be compared to orthodontic movement

Human teeth are prepared to receive high occlusal loads, which result in movements of intrusion into the alveoli, mostly during mastication (Fig 3). A lesion to all this apparatus indicates that the forces are too strong and persistent, that is, repetitive. Even in this condition, the periodontal ligament, with a mean thickness of 0.25 mm (250 μm, Fig 3), prevents the tooth from touching the apical alveolar cortical surface, and this structural organization ensures the perfect physiological functioning of the system of tooth attachment into the alveolus.

The periodontal ligament is a delicate membrane over the root surface, attached to the alveolar bone. Fifty percent of its structure is made up of vessels (Fig 3). It is efficient for intrusive forces, but not for lateral forces, and when the plan is to move teeth orthodontically,[9] the movements planned are inclination or displacement, and the forces are often less intense, always much lower than those involved in occlusal trauma, and applied slowly in a dissipating way.

After each device activation, periodontal tissues return to normal, and new forces may be applied under similar conditions: Mild, at a single moment and dissipating.[9] Practically all aspects are different when comparing orthodontic movement and occlusal trauma, particularly in terms of induced cell and tissue reactions and their consequences.

One of the main objectives of clinical practice in orthodontics is to correct occlusal disorders, particularly those associated with the relationships between the maxilla and the mandible, as well as between dental arches. However, orthodontic education in general does not provide detailed and adequate training in detecting occlusal interferences more accurately. In the clinical specialties of dentistry in Brazil, there are dentists specialized in the analysis, diagnosis and correction of occlusion and temporomandibular disorders.

During orthodontic movement, some occlusal interferences are promoted, but they are temporary and do not usually last long enough to induce significant lesions in the supporting periodontal structures. The typical changes of occlusal trauma are produced by the prolonged action of trauma forces on the same site.

At the end of orthodontic treatment, a careful occlusal analysis should be conducted before patient discharge, and a natural "accommodation" along the subsequent months should be expected.[4] However, in several cases the patient complains of and presents with typical occlusal trauma in certain teeth during the treatment.

Occlusal trauma and orthodontic treatment do not induce pulp necrosis, but dental trauma does!

Occlusal trauma promotes cell and tissue changes that are completely different from the phenomena induced by orthodontic movement.[9] Occlusal trauma is characterized by repetitive and intense forces. In orthodontic movement, forces are extremely lighter and applied only once, slowly and progressively. In 3 to 6 days, they dissipate gradually, and in 7 to 10 days they disappear, in humans.[9] In both cases, it is not possible to induce rupture or partial lesion of the vascular bundle. Although the periodontal ligament has a mean thickness of 0.25 mm (250 μm), its organization and functioning ensure that the tooth does not touch the apical alveolar cortical surface during the high forces of mastication, which would smash the vascular bundles at the point where it enters the apical foramen (Fig 3).

The abrupt, intense and short forces of dental trauma, however, may lead to the rupture of periodontal components, as teeth touch or their roots go through the al-

veolar bone structures and produce hemorrhage and necrosis of supporting tissues. The sudden displacement by intense and short forces ruptures the vascular bundle at the entrance of the root canal, at the apical foramen. Dental trauma of the concussion type often occurs without the patient clinically presenting with clinical signs of discomfort or pain.

In orthodontic movement, forces are much less intense than in dental trauma, and are applied slowly and in a dissipating way, although the device is activated at a single time periodically. These characteristics of the forces applied in orthodontic treatment have justified the results of several studies about the absence of significant changes to the dental pulp.

There is no basis to support the suggestion that orthodontic movement may induce pulp necrosis. The higher the force applied for orthodontic movement, the less efficient and inductive of tooth displacement, with still lower chances of inducing pulp necrosis. Also, there is no basis to claim that pulp necrosis is induced by occlusal trauma: When signs suggest it, dental trauma should be defined as the cause.

In occlusal trauma, forces are repetitive and intense, but not comparable to those of dental trauma, in which the force is unique, sudden and intense. Occlusal forces, even those found in overloads, cannot bring the dental apex into contact with the bone in the bottom of the alveolus and cannot smash or injure the vascular bundle. The human periodontal ligament has been designed to absorb and dissipate intrusive forces, which are predominant in movements of mastication and deglutition.

Occlusal trauma in the mineralized structures and abfraction

In the areas of occlusal interference, occlusal trauma determines the presence of areas of wear promoted by attrition.[12,13]

At the same time, excessive pressure or force eccentricity promotes three-dimensional distortions of the mineralized dental structure. Such distortions are called temporary and repetitive distortions.[10,11]

A deflection is the act or effect of moving away from a line that had been followed and going into a different direction. Such line may be the long axis of a tooth. A tooth deflection, in which the long axis is not followed, may produce traction in one side of mineralized structures and compression in the other side.

Cement and dentin are deformable, whereas enamel is not. Dentin has a mean 60% of inorganic components and 40% of organic components, mostly proteins and water. Cement, in turn, is made up of 50% organic and inorganic components. Dentin and cement form a relatively flexible structure and do not produce any structural changes.

Enamel, which is 96% mineral components, has a minimal or irrelevant capacity of deflection. On the side of compression during tooth deflection resulting from occlusal trauma, for example, enamel resists its effect, but not on the side of traction, where enamel does not resist and has early fractures or cracks in its delicate cervical area. This process, when repeated, may lead to fragmentation and enamel structure loss, clinically known as abfraction (Figs 4 and 5). Abfraction is very common, particularly among young people, and affects mostly premolars.

Cracks may not be visible on the cervical enamel of premolars. Patients, however, may complain of intense sensitivity to temperature and food variations in these "intact" teeth. If a tooth has a wear and a V-shaped recession surface (Figs 4 and 5), abfraction, although at an initial stage and not yet detectable, may be suspected, as it may explain the increased sensitivity.

Radiographic signs of occlusal trauma in periodontal tissues

The compression of the periodontal ligament due to occlusal trauma reduces vessel caliber and disorganizes fibers and cells. Therefore, cell stress is induced, and mediators are released and accumulate at a greater rate in the periodontal ligament, particularly those that may locally define a greater or lower rate of bone remodeling.

Local bone remodeling mediators have a two-phase effect: When accumulated at very high levels, they promote bone resorption; at slightly higher levels, they induce bone formation.

The forces applied to a tooth act as a lever with intra-alveolar rotation and fulcrum between the apical and middle thirds of the tooth root. In occlusal trauma, forces tend to be well distributed along the periodontal ligament, and the overload promotes slightly increased levels of bone remodeling mediators.

The tissue dynamics of occlusal trauma is radiographically confirmed by the thickening of the lamina dura (Figs 6, 7, 8 and 9). It increases bone deposition on the alveolar cortical bone and the resistance of this structure, and elongates collagen fibers. In other words, periodontal structures adapt to better absorb the increased occlusal forces.

In primary occlusal trauma, collagen fibers should be renewed at a higher rate, and the longer and better organized their bundles are, the greater the absorption or buffering capacity of excessive forces applied repetitively. Radiographs show an irregular broadening of the periodontal space because the ligament undergoes constant structural reorganization (Figs 6, 7, 8 and 9).

The forces in occlusal trauma are excessive and eccentric, but periodontal tissues adapt by thickening of the alveolar cortical bone, increasing the density of the adjacent trabeculae and irregularly expanding the periodontal space. Such changes occur along and around all tooth roots and adjacent tissues (Figs 6, 7, 8 and 9).

At the cervical area of periodontal tissues, very intense and persistent occlusal trauma due to the lever formed by the tooth lead to stretching/traction or excessive compression of the periodontal ligament. In this cervical region,

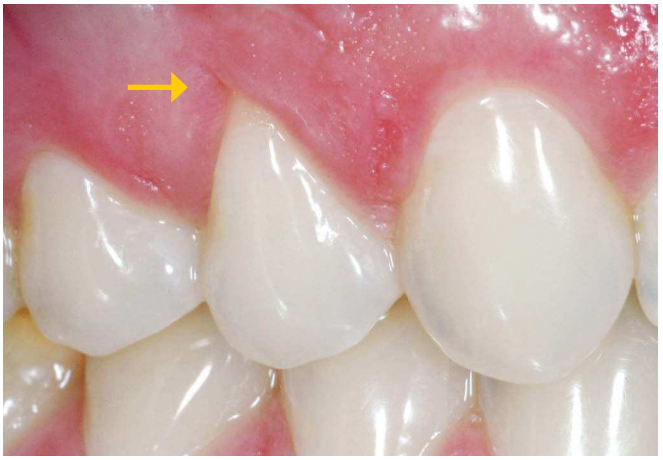

Figure 4 - V-shaped gingival recession, with a slight fissure on its end (arrow) and related to occlusal trauma.

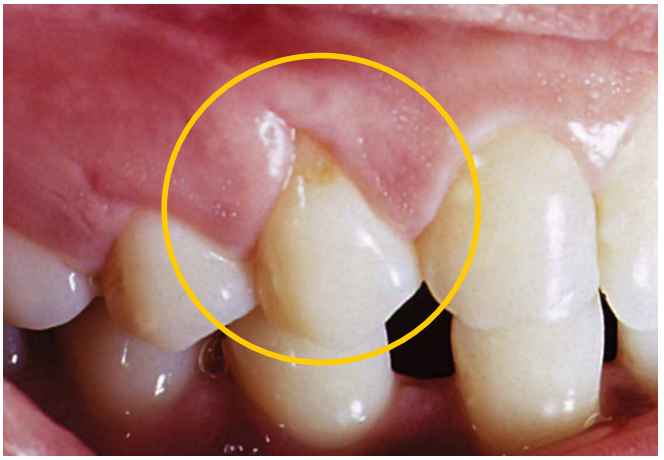

Figure 5 - V-shaped gingival recession in a tooth with abfraction: Two clinical signs of occlusal trauma.

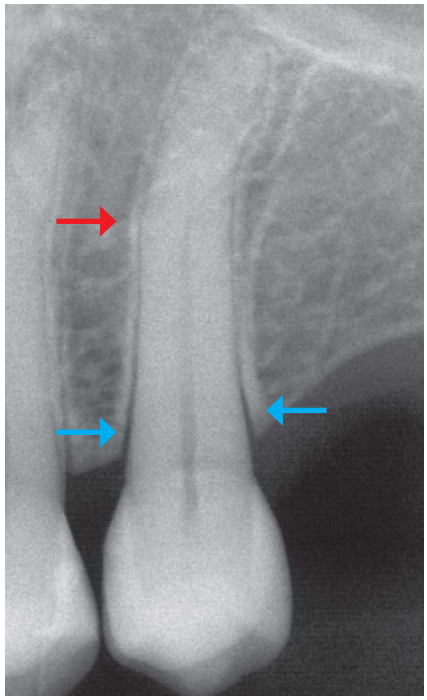

Figure 6 - Lamina dura thickening and periodontal space broadening (red arrow). It is highlighted the V-shaped bone resorption in the cervical region of alveolar bone crest (blue arrows): Initial aspects of occlusal trauma.

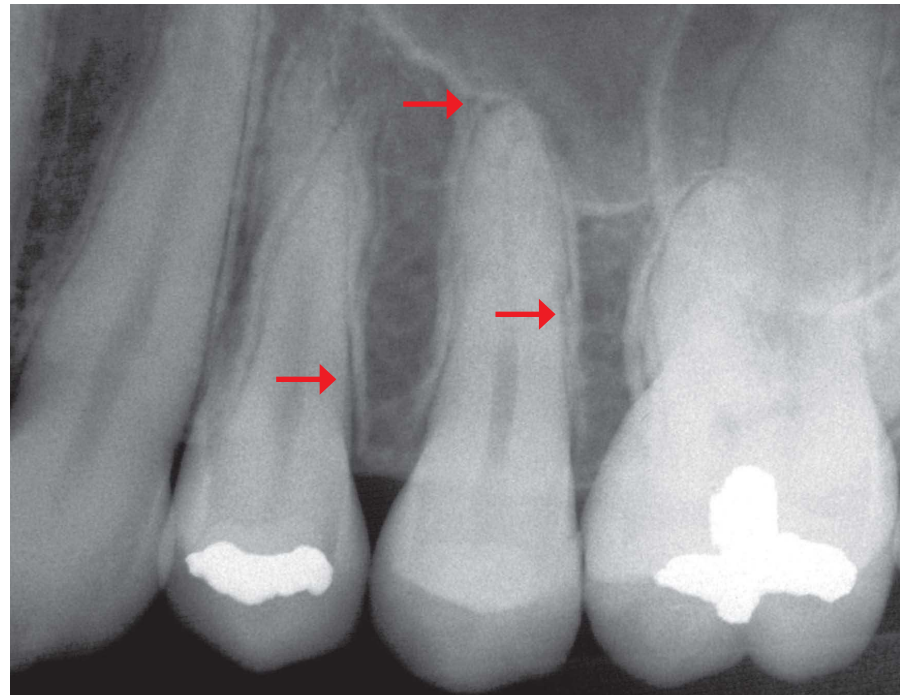

Figure 7 - Lamina dura thickening and periodontal space broadening (red arrows), associate to occlusal trauma. It is highlighted the slight increase of alveolar bone crest density.

the accumulation of mediators may increase as much as to predominantly promote bone resorption. The plane of the lamina dura surface — parallel to the tooth — may be angled in this region, indicating a V-shaped bone loss (Figs 6 and 9).

Such bone loss, seen as a "V" in imaging studies, is a sign of vertical bone loss but clinically with no periodontal pocket at careful and adequate probing. The simple elimination of the primary cause, that is, primary occlusal trauma, may restore bone to its previous level.

The first signs of occlusal trauma may, therefore, be defined as: Thickening of the lamina dura, irregular increase of periodontal space, V-shaped vertical cervical bone loss (Figs 4 to 9) and increase of apical bone density or bone crest sclerosis (Figs 10 and 11). These signs are a result of the attempt of periodontal tissues to adapt to new functional demands. Areas of inflammatory root resorption may appear at a substantially long time after that (Figs 12 and 13).

Consequences of occlusal trauma on the free buccal surface of the periodontal ligament and of the alveolar cortical bone

The same tissue and cell phenomena that occlusal trauma may induce in the alveolar bone crest surface of the periodontal ligament, under the same type of load and, consequently, of deflection, may also be induced in the free buccal surface.

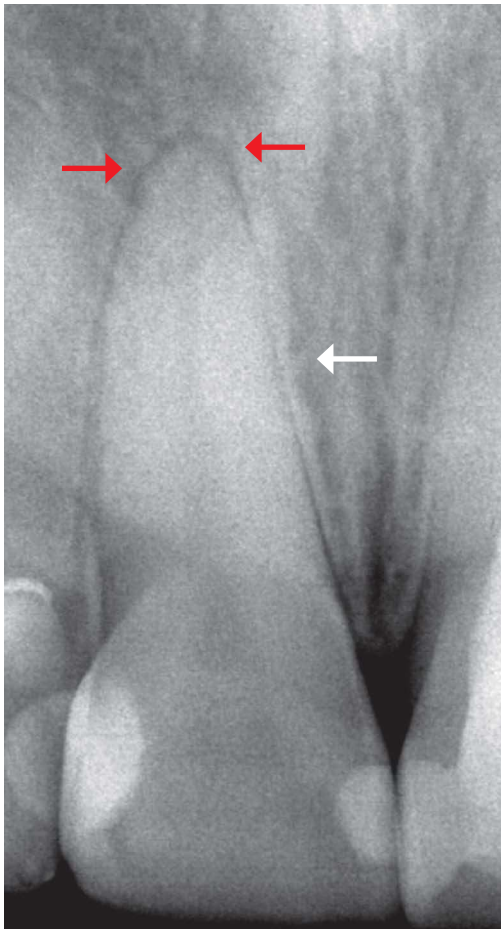

Figure 8 - Occlusal trauma with lamina dura thickening (white arrow) and broadening of periodontal space, with periodontal bone density increase (red arrows).

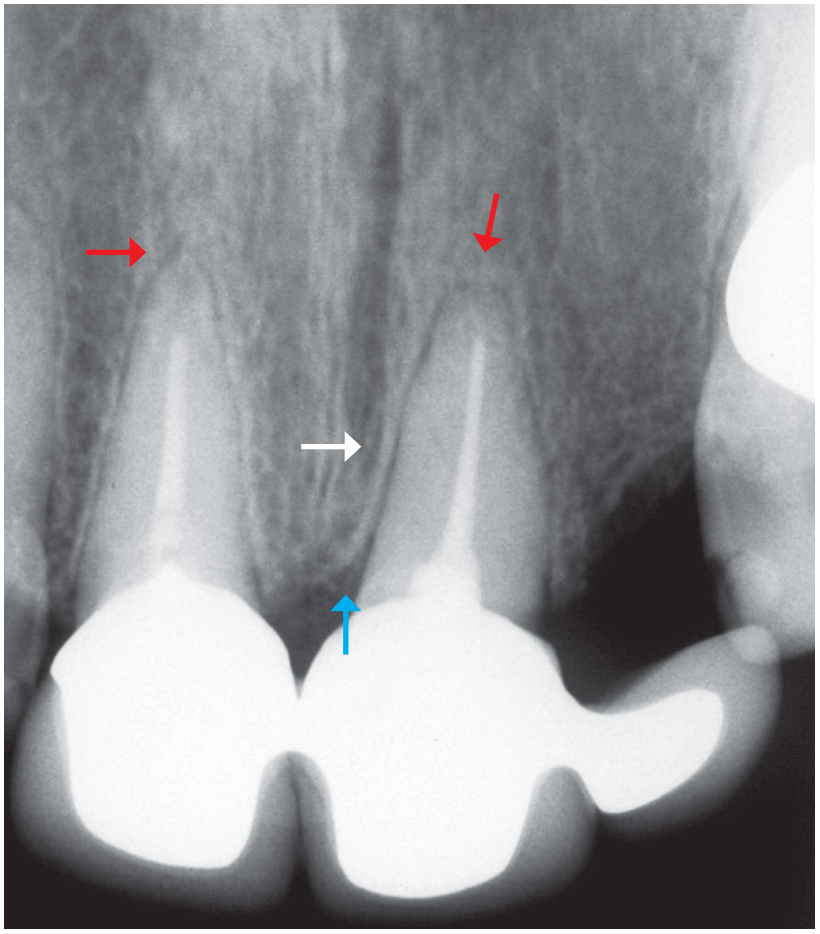

Figure 9 - Occlusal trauma with lamina dura thickening (white arrow), broadening of periodontal space with bone density increase (red arrows) and vertical bone loss (blue arrow).

However, the buccal cortical tends to be very thin, and very little resorption on its periodontal face may result in loss of cervical height and V-shaped bone dehiscence over the buccal face of the root that was affected (Fig 14).

The areas of buccal bone dehiscence are locally distributed, and the increase of their size is a gradual and slow process. Its detection using imaging studies is very difficult, although some sophisticate CT scanners bring the promise of doing it according to careful criteria. Fenestrations may also be associated with this condition (Fig 14).

At the onset of buccal bone dehiscence, first the periosteum persists locally for a indefinite period. When there is no bone to recover and protect the periosteum, and without vessels to nourish it, the periosteum tends to be fixed in the bone margins of the dehiscence area, which leaves the root surface exposed to gingival and periodontal connective tissues.

V-shaped gingival recession in occlusal trauma and how it occurs

Primarily, occlusal trauma may promote gingival recession, particular V-shaped recession (Figs 4 and 5). Some authors,

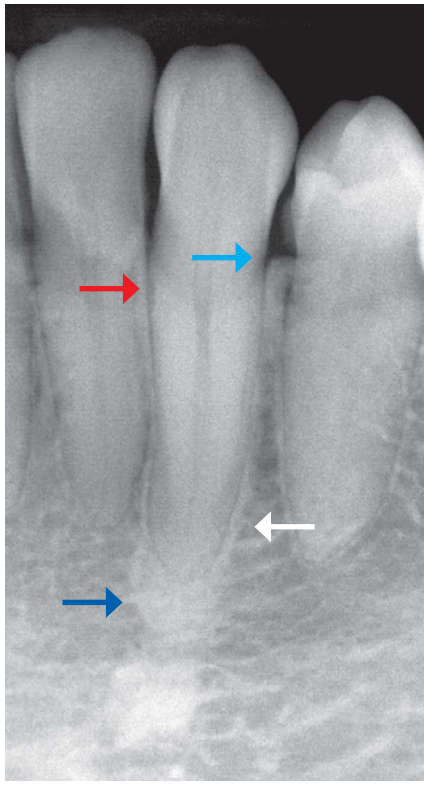

Figure 10 - Occlusal trauma with lamina dura thickening (white arrow), broadening of the periodontal space (red arrows), with periodontal and periapical bone density increase (light blue arrow) and V-shaped vertical bone loss (green arrow).

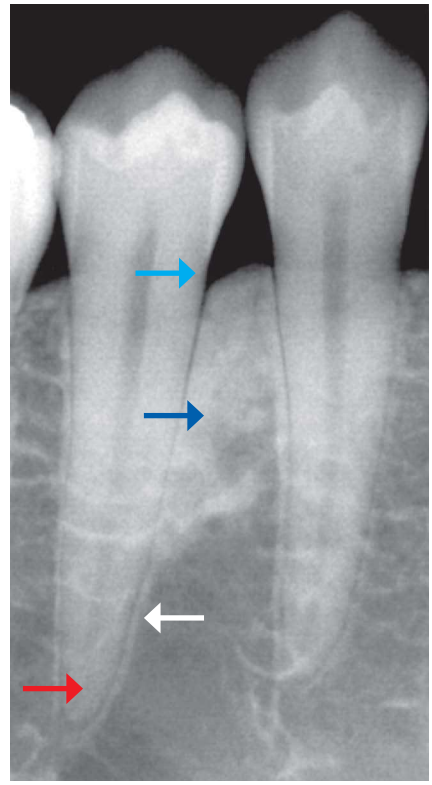

Figure 11 - Occlusal trauma with lamina dura thickening (white arrow), broadening of the periodontal space (red arrows), with periodontal bone density increase in the bone crest (light blue arrow) and V-shaped vertical bone loss (green arrow).

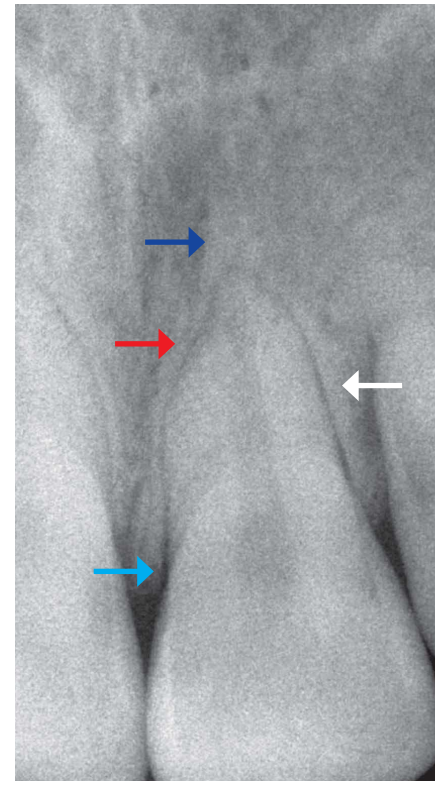

Figure 12 - Apical inflammatory radicular resorption associate to occlusal trauma, lamina dura thickening (white arrow), broadening of the periodontal space (red arrow), with periapical periodontal bone density increase (dark blue arrow) and V-shaped vertical bone loss (light blue arrow). This phase with inflammatory apical resorption occurs after a long period of overloading.

particularly the Scandinavians,[1,15] have not accepted this finding in concept and believe that for gingival recession to occur, it should always be associated with the accumulation of bacterial plaque. This position has generated controversy and polemic discussions about the topic.

One of the reasons that led Scandinavians to suggest the need of a bacterial plaque for gingival recession to occur in occlusal trauma was the focus of their studies and concepts: They compared occlusal trauma to orthodontic movement, and even called it "orthodontic trauma".[15]

Gingival recession may be generalized and affect several or almost all teeth. There may be several associated causes for its occurrence, described as atrophic changes of periodontal tissues.

U-shaped or circular recessions are closely associated with the presence of bacterial plaque and the consequent chronic inflammatory periodontal disease, frenular insertions, inadequate brushing and other less frequent events. V-shaped or angled gingival recessions have a small fissure in their most apical extremity. This type of recession is di-

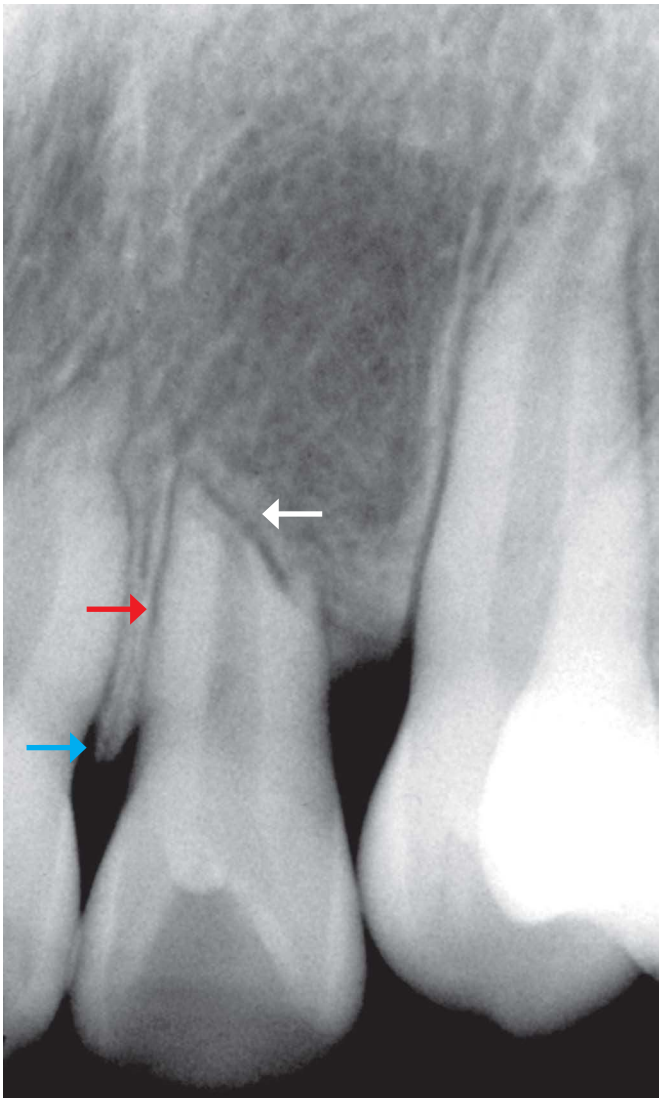

Figure 13 - Plane and more advanced inflammatory apical root resorption, associated to occlusal trauma, with lamina dura thickening (white arrow), broadening of the periodontal space (red arrow) with periapical periodontal bone density increase in this area and V-shaped vertical bone loss (blue arrow). This phase with inflammatory apical resorption occurs after a long period of overloading.

rectly associated with occlusal trauma[16] (Figs 4 and 5) and are commonly associated with abfraction.[3,10,11] At the initial stages in most cases, the elimination of occlusal trauma leads to the regression or reduction of this V-shaped recession.[16] In many of these cases, it is not possible to define a direct association with bacterial plaque accumulation.

Buccal bone dehiscence, as seen previously, temporarily brings together two very similar structures that fuse and reorganize as a single structure along time. The buccal alveolar cortical bone extends between the periosteum and the periodontal ligament, sometimes very delicately.

The periosteum is composed by two different and continuous layers of fibrous connective tissue. The very fibrous outer layer has few cells and naturally continues the inner layer, which has a richer variation of cells and more vessels. This inner layer is the direct interface between the periosteum and the cortical bone, and it is crossed by fibers that are strongly attached to the mineralized area of the cortical surface.

In human skeletons, the bone surface is not recovered by periosteum only in tendon attachments and alveolar cortical bone. The periodontal ligament acts as the periosteum on the alveolar surface. This suggests that the periodontal ligament is another form of periosteal organization.

When cortical bone is lost due to resorption and buccal dehiscence in teeth with primary occlusal trauma, the two structures should, for some time, juxtapose, but should reorganize individually in the long run. Without the presence of bone in the region and with the unification of periosteum and periodontal ligament, the two structures do not have any other active function. Because of dehiscence, the fibrous connective tissue produced under this condition gradually extends an elongated connective attachment to the attached gingiva, to a point that is very distant from the level of cervical bone.

Due to the absence of bone, the periosteum and the ligament are joined by contiguity or proximity, and the result is an elongated connective attachment and a modified biological distance between junctional epithelium and the cervical bone level. If occlusal trauma persists, it is not possible to maintain the attachment of the periodontal fibers to the cement when it has no function, because, as there is no bone, there is no counterpart to the anchorage.

The periodontal fibers without anchorage and the neighboring periosteum without bone gradually reorganize as normal gingival connective tissue. Hyperplasia and epithelial migration should be associated with this long connective attachment and the production of long junctional epithelium, which may resist and persist when the gingival level is at a normal height by a certain period of time under occlusal trauma.

Because of continuous occlusal trauma, the gingival tissue follows the level of bone dehiscence and forms a V-shaped gingival recession. Recessions are classified as periodontal diseases with atrophic changes. As it has no function, gingival connective tissue, increased due to bone loss, tends to change volume and organization in a way that is similar to that found in the gingiva of normal teeth, but this exposes the root buccally.

Tissue volume in cases of gingival recession decreases because of the adaptation of periodontal tissues to a new function and because there is no more bone in the area of dehiscence. The volume is reduced by means of constant and normal tissue remodeling, which reestablishes normal tissue proportions between bone, submucosal connective gingival tissue and mucosa, sulcus and junctional epithelia.

While the gingival level is preserved, despite vertical bone loss and as long as there is no periodontal pocket, the elimination of primary occlusal trauma may still reverse the process, even when bone loss is substantial. When the root has already been exposed in the mouth, reestablishing the gingival level usually demands surgery with or without gingiva or bone grafting.

Criteria for the diagnosis of occlusal trauma

Primary occlusal trauma, still mild and incipient, may be detected clinically (Figs 4 and 5) according to 3 factors:[4,10-13]

- Wear surfaces in the area of interference.
- Abfraction, especially in premolars.
- Discrete V-shaped recession.

These three signs practically confirm and describe occlusal trauma clinically, but only one or two of these signs may be present in some cases. Before the appearance of the V-shaped recession, when only the wear facets and abfraction are present, these signs should suggest that the clinician may conduct a careful periodontal analysis and examination (Figs 6 to 13) in periapical radiographs searching for signs such as:

- Increased thickness of the lamina dura.
- Irregular thickening of the periodontal space.
- Vertical V-shaped cervical bone loss.
- Bone sclerosis in the periapical area or in the interdental bone crest.
- Inflammatory root resorption, more common in advanced stages of occlusal trauma.

The wear surfaces due to attrition[12,13] and the abfraction[10,11] should be corrected, but only after the occlusal problems, even when gingival recessions are already present.

An early diagnosis greatly improves the prognosis of the V-shaped gingival recession, and the elimination of occlusal trauma may lead to spontaneous regression in several clinical cases.

In rare cases, pain may be detected during vertical percussion. Moreover, patients rarely report the feeling that the tooth is the first to make contact during occlusion.

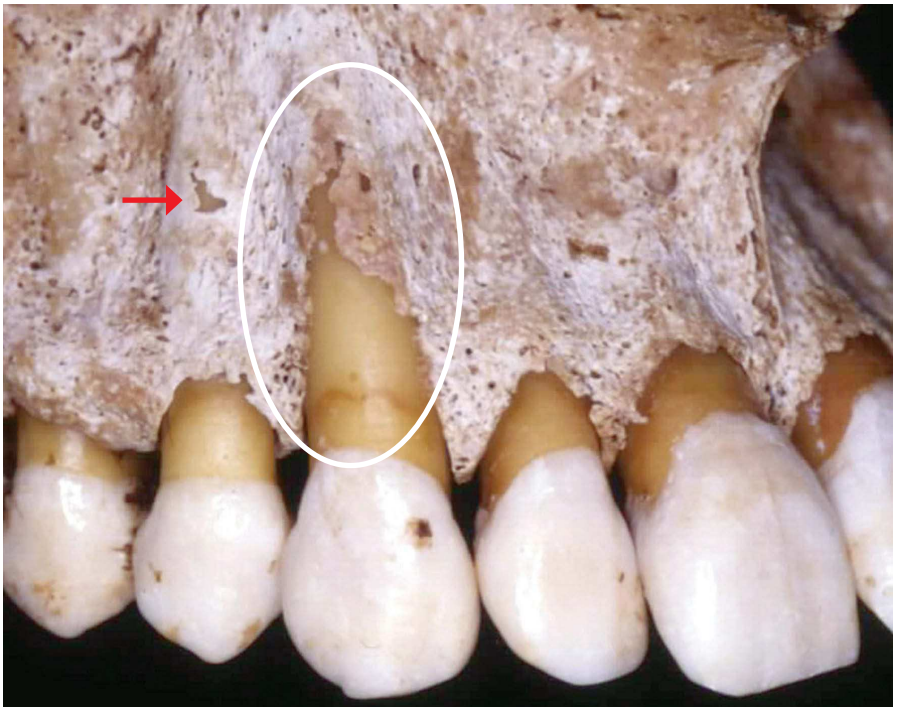

Figure 14 - Vestibular cortical bone of the upper canine with dehiscence and vestibular cortical bone of the first premolar with small fenestration (arrow). It is highlighted the reduced thickness of the buccal alveolar cortical bone.

In some of the cases described in the literature, the best outcome from surgical procedures — microscopically analyzed — was the accumulation of fibroblasts and the formation of collagen fibers parallel to the scaled and treated root surfaces, without attachment of perpendicular or functional periodontal fibers. This happens in simultaneous or alternate gingiva and bone grafts.

The very satisfactory clinical result of these surgical procedures using gingival grafts result from the formation of a long junctional epithelium and the maintenance of the postoperative gingival level for an indefinite time. Epithelial cells manage to colonize these dental surfaces that were previously exposed in the mouth and contaminated by LPS after scaling and planing.

These two signs are characteristic of apical periodontitis induced by tooth trauma and associated with pulp necrosis due to pulpitis.

Grafts in V-shaped gingival recessions associated with occlusal trauma

Severe gingival recession may indicate that the root surface was exposed in the mouth for a long time, under the action of bacterial plaque, which irreversibly contaminates the root structure with lipopolysaccharides (LPS).

Cementoblasts do not repopulate the surfaces contaminated by LPS to form new cement layers, not even after intense scaling or treatments with different acids and antimicrobial substances. In other words, it will not be possible to reattach periodontal fibers to those surfaces, not even after gingival grafting.

Unfortunately there is no solid evidence to confirm these clinical results because of the different methods when conducting clinical and experimental essays. No method is available to study the reattachment of fibers to surfaces previously exposed in the mouth for a long time and under the action of bacterial plaque.

Is there occlusal trauma in dental implants?

How can we adopt criteria to define up to what degree of trabecular density or bone sclerosis around an osseointegrated implant should be considered normal? What parameters should be used to conclude that the bone around the implant is discretely inflamed (osteitis) and, therefore, with sclerosis or condensing?

Up to 1998, little was known about the peri-implant reactions under the effect of orthodontic anchorage loads. Akin-Nergiz et al.[1] evaluated peri-implant bone densitometry under these conditions and found results that may be summarized according to two figures:

» In Figure 15, published by Akin-Nergiz et al,[1] the images in red correspond to the bone that is more densely organized, regardless of the compression or traction loads orthodontically applied to the osseointegrated implants used as anchorage.

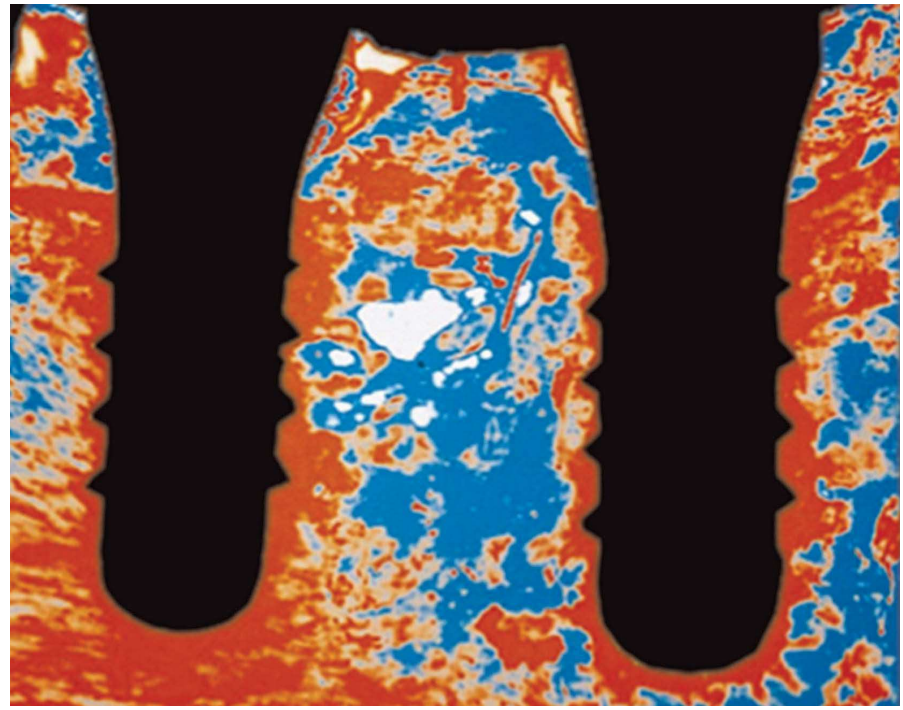

Figure 15 - In this image from Akin-Nergiz et al[1] study, radiographic bone densitometry of areas where two osseointegrated implants where subjected to orthodontic load. It is highlighted the increased bone density (in red) in the areas of compression and tension sides, when compared to the original alveolar cancellous bone, in blue.

» From the same study conducted by Akin-Nergiz et al,[1] Figure 2 shows three bone images of osseointegrated implant areas, and more dense bone trabeculae were induced in the one exposed to masticatory loads. To what point this may generate lesions similar to occlusal trauma? Unfortunately we have not advanced to that point in studies of peri-implant pathologies.

Final considerations

Images provide a language that reveals the dynamics of bone and tissue. Bone density and space distribution vary and indicate greater or lower reaction and adaptation to functional demands, such as masticatory loads on natural teeth or on osseointegrated implants.

Clinical diagnosis and procedures defined according to images should be based on signs and symptoms. Strict criteria should be followed, and specialists should use their previous knowledge. Occlusal trauma, seen as a clinical event, should be included in the differential diagnosis of apical periodontitis and dental trauma.

In rehabilitation, it is fundamental to plan loads distribution and to evaluate the remaining teeth and their relation with neighboring bone. The detection of bone response and pre-existing occlusal trauma may favor a more accurate evaluation of masticatory conditions and parafunctional habits — a true functional history of remaining teeth.

The sclerosing osteitis type of increase in periapical bone density, associated with a tooth whose pulp is vital may suggest a diagnosis of occlusal trauma even when there is inflammatory root resorption.

When a tooth has pulp necrosis and signs of occlusal trauma, the evaluation of history and tests should be directed to the diagnosis of superposed dental trauma even when posterior teeth are affected. Below are some conditions that may indicate dental trauma of the concussion type, which may lead to pulp necrosis in posterior teeth:

» Anchorage of surgical levers in neighboring teeth during extractions.
» Accidental impact of instruments, such as forceps, during surgical procedures.
» Sliding of probing instruments during gastroesophageal examinations.
» Movements of the laryngoscope during general anesthesia procedures.
» Presence of candy and other foods in the mouth in leisure and sports activities during sudden movements, such as when riding a motorcycle or roller coaster.

Occlusal interference and overloads take months or years to induce classical signs and symptoms of occlusal trauma as a clinical entity. The correction of such interference and occlusal overloads in general interrupt the onset of these signs and symptoms.

Interferences and occlusal overloads do not necessarily indicate that the signs and symptoms of the clinical entity called occlusal trauma are present: They may take many months to be identified clinically and in imaging studies.

A usual question: Up to what point can orthodontic forces be applied to osseointegrated implants? Orthodontic forces are usually not greater, in any situation, than occlusal forces in terms of intensity, amplitude and variability. If an implant may receive masticatory loads, it may also be exposed to the forces of orthodontic anchorage.

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