THERE IS NO PULP NECROSIS AND NO CALCIUM METAMORPHOSIS OF PULP INDUCED BY ORTHODONTIC TREATMENT

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ABSTRACT:

The orthodontic movement does not induce pulp necrosis or calcium metamorphosis of the pulp, as revealed by clinical research in humans and laboratory animals. When pulp necrosis or calcium metamorphosis of the pulp is diagnosed during or shortly after orthodontic treatment, its etiology should be attributed to concussion-type dental trauma, and not to orthodontic treatment. The two pulp diseases that lead to coronary dimming in apparently healthy teeth are aseptic pulp necrosis and calcium metamorphosis of the pulp, both of which are induced exclusively by dental trauma.

KEYWORDS:

Tooth movement techniques. Orthodontic. Dental pulp necrosis.

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INTRODUCTION

Innumerable baseless claims are frequently made about orthodontic treatments that may induce pulp necrosis. Such allegations clearly ignore the following:

1. The mechanisms of tooth movement induced in periodontal tissues – forces act only on the periodontal ligament, alveolar bone and tooth surface, and orthodontic forces are never sudden, not even when intensely activated, and do not affect pulp tissues.

   Orthodontic forces are dissipating, not sudden or abrupt, and poor knowledge about it comes from the fact that graduate courses and specialty training in Endodontics and in other specialties do not offer lectures or training classes about how orthodontic movements occur in orthodontic practice. Such knowledge ends up being restricted to orthodontists.

2. The effects of tooth concussion, the most common and mild form of trauma to tooth tissues – even specialists in dental trauma find it difficult to interpret its insidious results along time. Most studies and authors focus on more serious dental trauma, such as luxation, avulsion or reimplantation. However, although milder and subclinical, concussion is the most frequent dental trauma and may have consequences as important as those of the more serious types.

3. Concussion forces are sudden and intense and may affect:

   a) The apex, with partial or complete injuries to the vascular bundle that crosses the pulp; in this case, concussion leads to calcific metamorphosis of the pulp and aseptic pulpal necrosis.

(b) The pulp, when concussion results in the focal displacement of the odontoblastic layer and induces internal resorption.
(c) Cementoenamel junction, which induces external cervical resorption.
(d) Lateral periodontal tissue, which may induce lateral periodontal resorptions.

THE SEARCH FOR FUNDAMENTAL INFORMATION SHOULD BE CONDUCTED CAREFULLY

When we search for biological and clinical explanations of the effects of pulp changes induced by orthodontic forces, we often search for information, studies and books using keywords such as "pulp", "pulp changes", "pulp diseases", "pulp biology", "endodontics" and other keywords associated with the pulp.

Similarly, if we want to ask a specialist about "possible changes, opinions, dogmas and beliefs" about the effects of orthodontic treatments on pulp tissues, it is common to ask those that supposedly study and research about biology and pulp diseases very intensively, usually endodontists. Directly and simply, their involvement with the tooth pulp seems logic.

However, if we expect to find studies and research to give us fundamental knowledge, or ask relevant specialists, we should search for those that focus on the periodontal ligament specifically, because orthodontic movement is a phenomenon exclusively associated with the periodontal ligament, and not the tooth pulp.

We hardly ever find Endodontics or Dental Trauma books or studies that discuss the biology of orthodontic movement, which is often found in, for example, the Periodontology literature. The true understanding of why the tooth pulp is not affected by orthodontic movement comes from the comprehensive study of periodontal biology and changes, because the pulp does not participate in tooth movement.

In addition, books, texts and studies about dental trauma hardly ever discuss and describe the effects of tooth concussion on pulpal, gingival and periodontal tissues thoroughly. Specialists in Dental Trauma are usually concerned with those clinical aspects of dental trauma that are symptomatic and require immediate interventions. Concussion requires observation management, because it is clinically silent.

Tooth concussion leads to tissue lesions because of the sudden, short and intense forces applied to the tooth. This is in contrast with the weak, mild, dissipating and decreasing forces that move teeth orthodontically.

With the purpose of sharing this knowledge and comparative understanding of the effects of tooth concussion and the mechanisms of orthodontic movement, we set ourselves to disseminating findings of

studies and discussions that may give support to dentists of several specialties to no longer claim, equivocally, that induced tooth movements may lead to pulpal necrosis.[1] In this paper, we address our arguments and reasoning, including texts and figures, to all specialists who face these questions in their daily practice.

ACTIVE ORTHODONTIC FORCES MUST BE WEAK OR MODERATE

Orthodontic movements are achieved by the application of forces to teeth, which lead to a biological disruption, known as cell stress, that affects the cells of the periodontal ligament and may evolve into an initial mild inflammation lasting a few hours or days,[2] with the production of discrete inflammatory exudate and an initial infiltrate (Fig. 1 to 5).

Orthodontic forces are very weak in any situation because they have to induce cell stress and periodontal ligament inflammation. This structure is membranous and only 0.25 mm thick, the thickness of a bond paper sheet, and is made up of specialized fibrous connective tissue. [2,3]

In addition to weak and moderate, the forces applied to the periodontal ligament are dissipating, that is, in 2 to 7 days their intensity decreases gradually and they disappear, replaced with periodontal reorganization, and the ligament returns to normal in 10 to 15 days after activation of orthodontic appliances.[2]

IMMEDIATE: immediately after its application, orthodontic forces reduce or dissipate partially due to two mechanisms:

» Displacement of liquids and gel from the extracellular matrix of the periodontal ligament into the medullary and perivascular spaces, thus partially buffering the effect of the orthodontic forces applied. This is a physiological mechanism used by the periodontal ligament to buffer the heavy masticatory loads.[2,3] If it works for the more intense and incomparably extreme and heavy occlusal loads, we may conclude, by extension, that it works even more efficiently for the extremely weak and moderate orthodontic forces.

» Most applied orthodontic forces decrease about 20% to 30% almost immediately after their application, because the alveolar bone crest, which supports the tooth to be moved, undergoes deflections or deformations due to its elastic or plastic capability.[2] Alveolar bone is thin and has a high organic component, liquids and cells, as all bone does.

FROM 10 TO 12 HOURS: innumerable osteoclasts, are juxtaposed on the periodontal bone surface, which initiates bone resorption and the widening of the periodontal space narrowed by the compression of the tooth on periodontal structures.[2] This is stimulated by chemical mediators released by the compressed, hypoxic cells of the periodontal ligament.

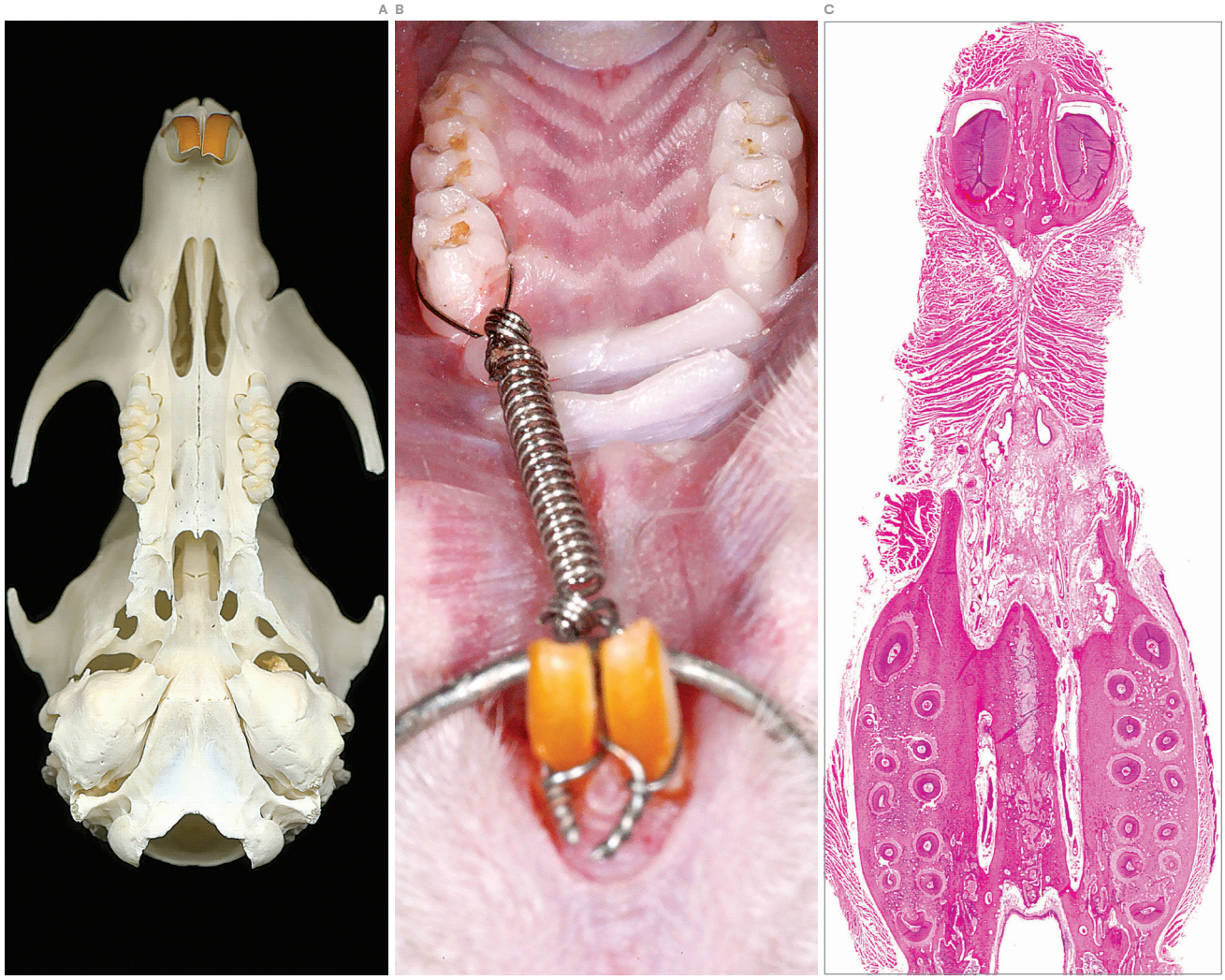


Figure 1:

(**A**) Rat maxillary molars; first rat molar used in experimental model of induced tooth movement, developed by Heller and Nanda,[10] in (**B**). In (**C**), axial plane of maxillary tissue to evaluate periodontal, osseous and pulpal changes induced by tooth movement (**C** = HE, original magnification 2.5X).

AFTER 7 TO 10 DAYS: there are practically no active forces to move the teeth to which the orthodontic forces were initially applied. From this point on, periodontal phenomena are predominantly reparative and reorganize the tissue back to normality, preparing it to receive a new cycle of forces.

From the first moment, the vascular and neural bundles that go through the periodontal ligament and enter the apical foramen to supply the pulpal tissue with blood cannot be ruptured.[4] There are no abrupt, sudden orthodontic movements that may result in partial or total lesions of the vessels that supply blood to the pulp. SEVERE or heavy orthodontic forces do no move teeth

Since the beginning of the treatment, orthodontic forces dissipate gradually and, after 5 to 7 days, tend to disappear and decrease their intensity as the tooth moves.

To move a tooth orthodontically, the periodontal ligament has to be live and biologically viable to receive and nourish the osteoclasts that will produce bone resorption on the periodontal surface of the alveolus (Fig. 2 to 5). Without vessels for blood circulation, without an extracellular matrix or cells and mediators, there are no tools to move the tooth in the bone (Fig. 6 and 7).

When forces are so strong or heavy, due to accidental or intentional compression of the periodontal ligament, that the periodontal vessels are occluded, the cells migrate to a site neighboring the one where anoxia occurred (Fig. 6 and 7). At the original site, only the extracellular matrix remains, with no cells or with only some sporadic cell rests, and the matrix acquires a hyaline appearance under microscopy and hematoxylin-eosin staining. This is known as the hyalinization of the periodontal ligament.

Orthodontic movement is never sudden or unexpected! Sudden movements are characteristic of dental trauma [5], and not of orthodontic movement. Orthodontic movement and dental trauma lead to completely different tissue changes or lesions, and the forces that cause each one have completely different intensity, duration and area of action.

In the area where heavy forces act on the periodontal ligament, only the extracellular matrix remains, without cells and with only the now occluded blood vessels. There, osteoclasts will not find the conditions for resorption of the alveolar bone, and it will not be possible to enlarge the periodontal space or to move the tooth. Microscopically, these areas, called hyaline, have only the extracellular matrix and no cells, and this process is called periodontal hyalinization (Fig. 6 and 7).

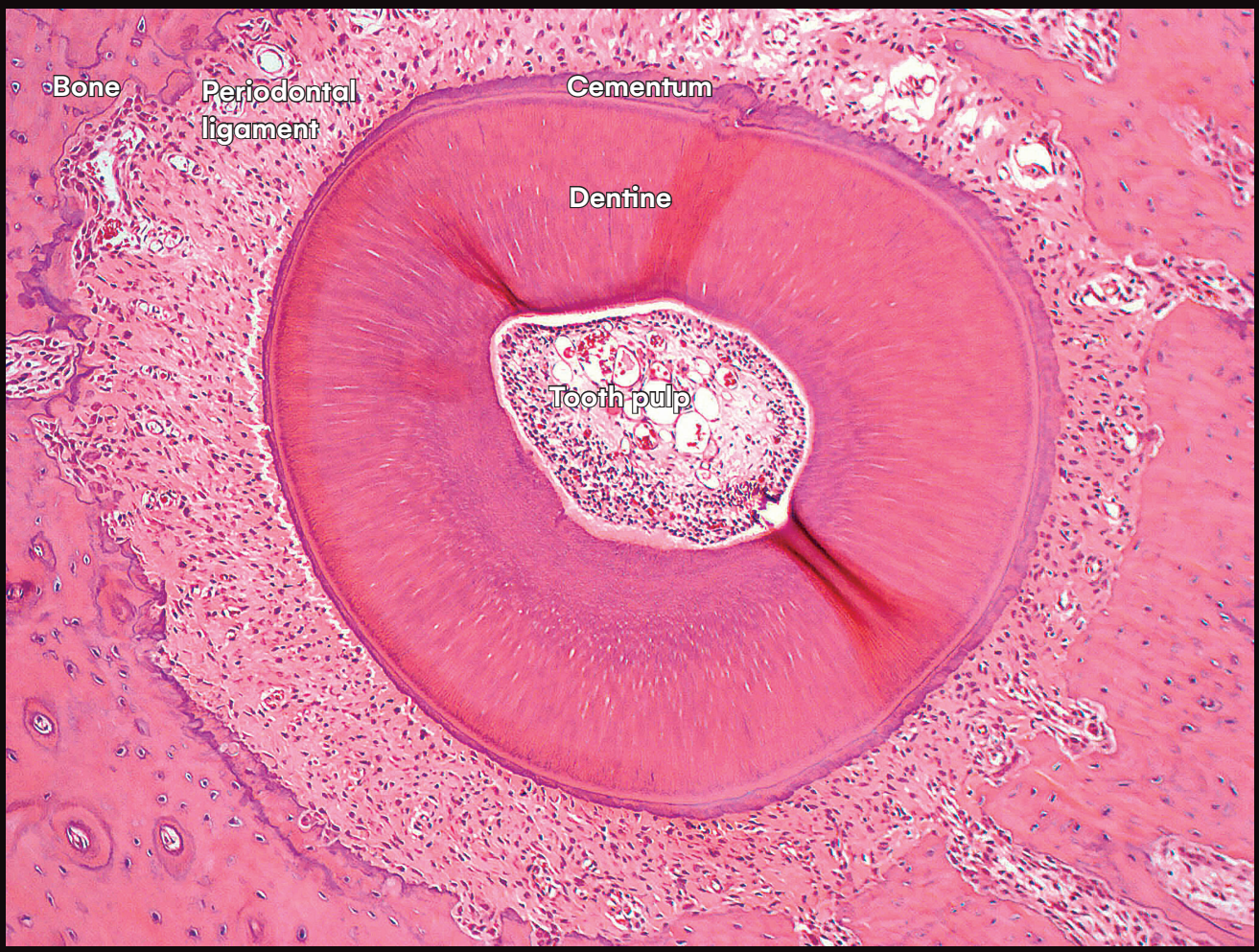


Figure 2:

Microscopic appearance of rat molar root on axial plane, or cross-section, reveals normal root structures, including pulp, alveolar bone and periodontal ligament (HE, original magnification 10X).

Figure 3:

Microscopic appearance at greater magnification of same rat molar root on axial plane, or cross-section, seen in Figure 2 reveals normal root structures, including pulp, alveolar bone and periodontal ligament (HE, original magnification 40X).

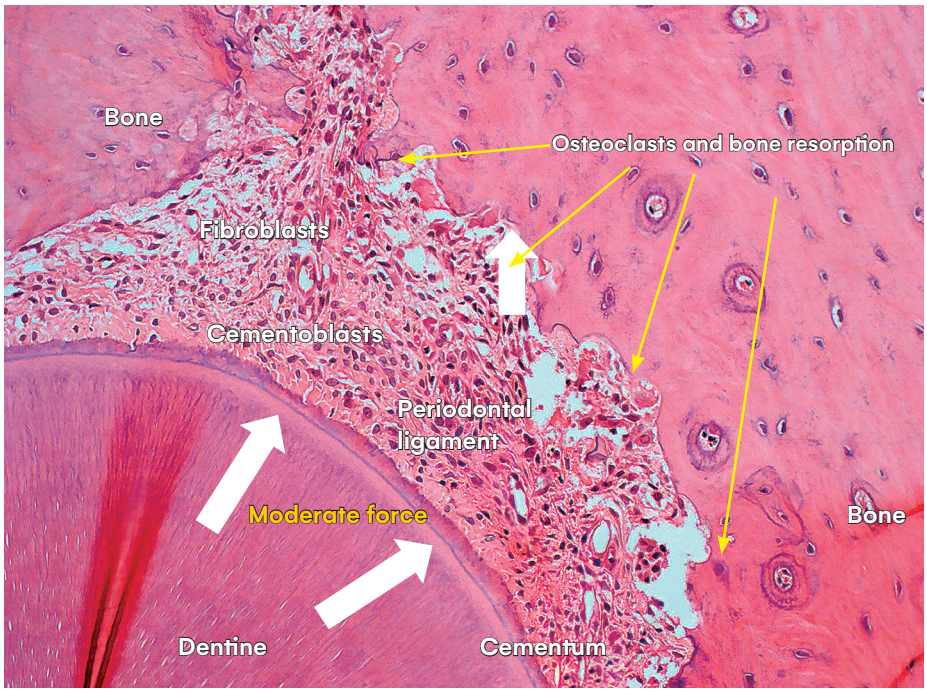


Figure 4:
Microscopic appearance of rat molar root on axial plane four days after application of moderate orthodontic forces that induced release of mediators to stimulate osteoclastic activity on surface. Tooth movement is slow, and forces dissipate without leading to changes in vascular or neural bundles that cross apical foramen (HE, original magnification 25X)

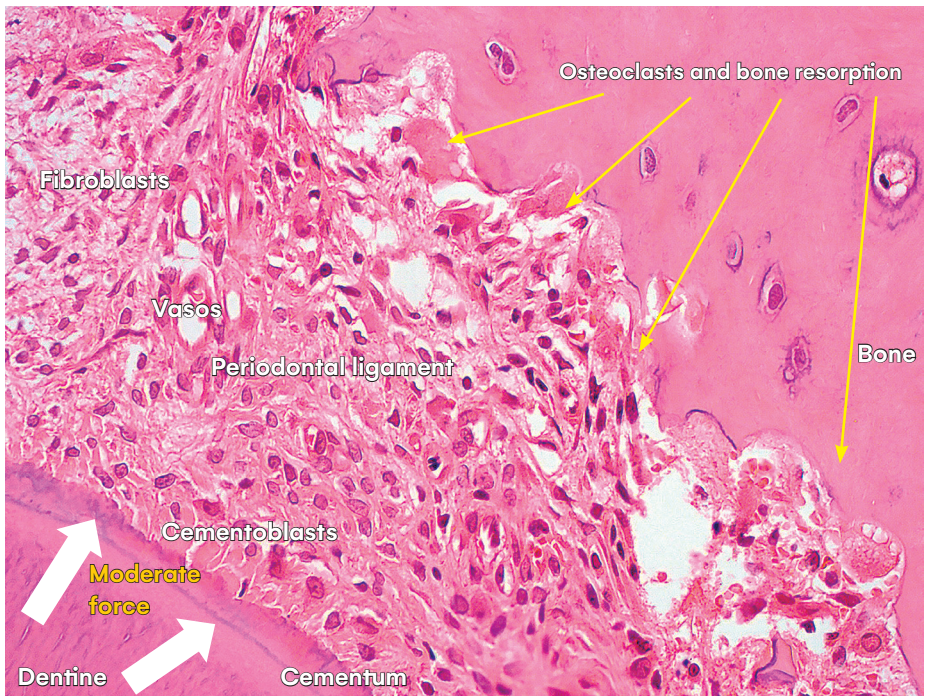


Figure 5:
Microscopic view at a greater magnification of same rat molar root seen in Figure 4 on axial plane four days after constant application of moderate orthodontic forces. Activity of osteoclast and other cells ensured by preservation of periodontal structures without hyalinization (HE, original magnification 40X).

Figure 6:
Microscopic appearance of rat molar root seen on axial plane four days after application of strong orthodontic forces that induced hyalinization of a periodontal ligament segment. Tooth movement here does not occur, because osteoclasts cannot produce bone resorption on periodontal surface (HE, original magnification 10X)

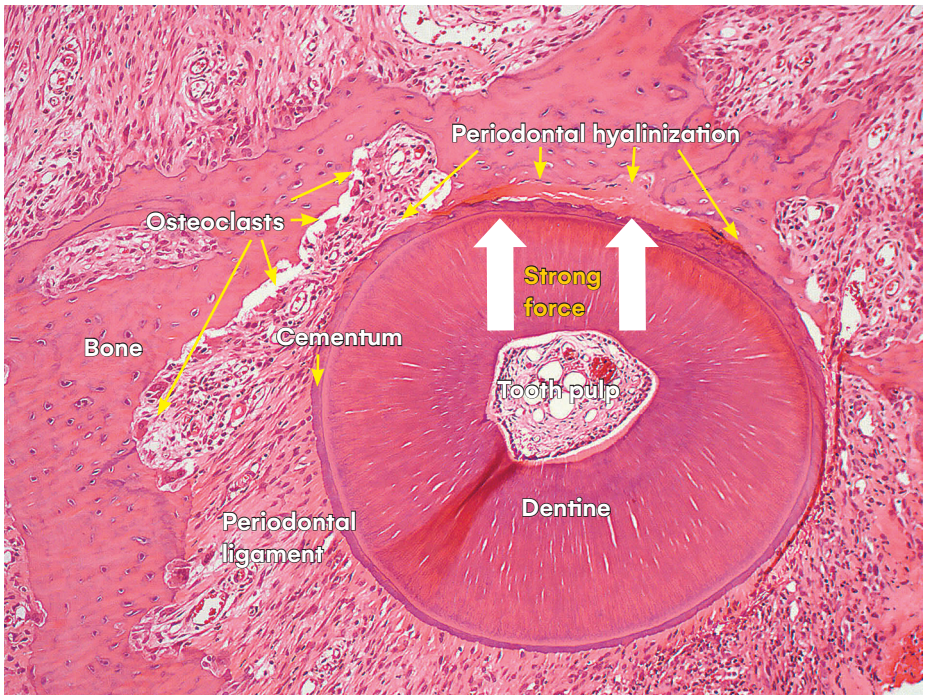
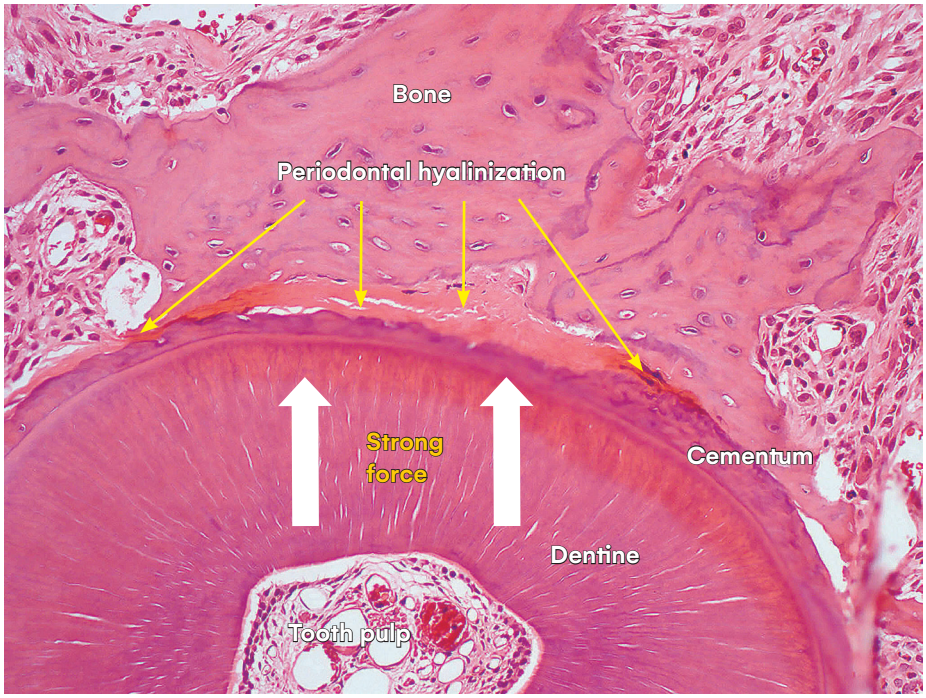


Figure 7:
Microscopic appearance at greater magnification of rat molar root seen in Figure 6 on axial plane four days after application of strong orthodontic forces that induced hyalinization of a periodontal ligament segment. Tooth movement here does not occur, because osteoclasts cannot produce bone resorption on periodontal surface (HE, original magnification 40X)



In other words, when the forces are not weak or moderate, there will be no tooth movement.[2-4] That is, severe or heavy forces do not move teeth orthodontically. Therefore, if heavy forces are applied by professionals that are not appropriately trained in orthodontics, the risk of pulpal necrosis and calcific metamorphosis of the pulp is reduced

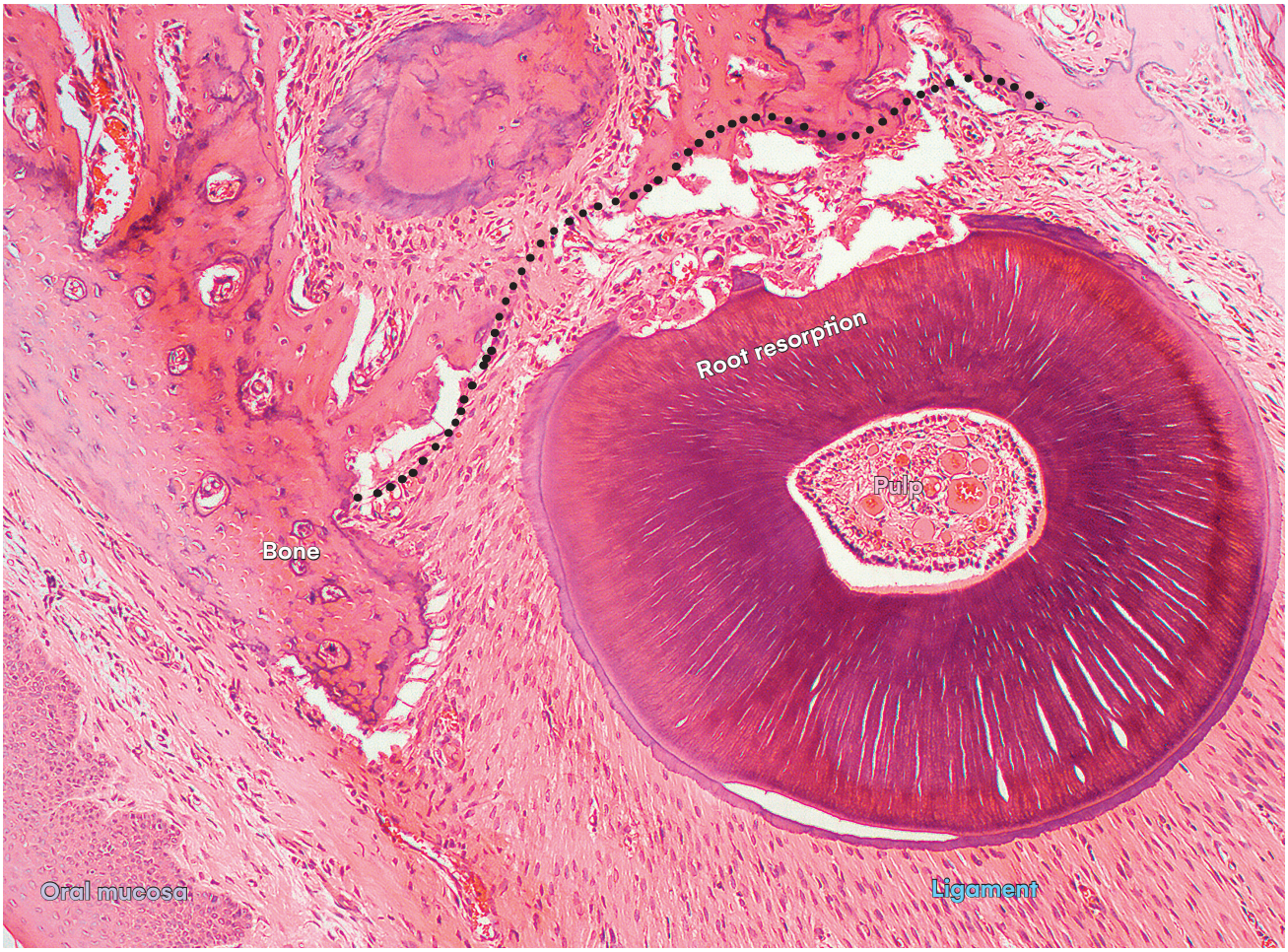


Figure 8:

Microscopic appearance of rat molar tooth on axial plane nine days after application of strong orthodontic forces that induce extensive root resorption. Pulp looks normal (HE, original magnification 10X).

to zero. However, cementoblast death breaks the protection to tooth roots because, when uncovered or exposed, they become the site for the action of osteoclasts, thus promoting severe root resorption. The pulp retains its full normal structure and organization (Fig. 8 and 9), and we may say that, in orthodontic movement, less is more!

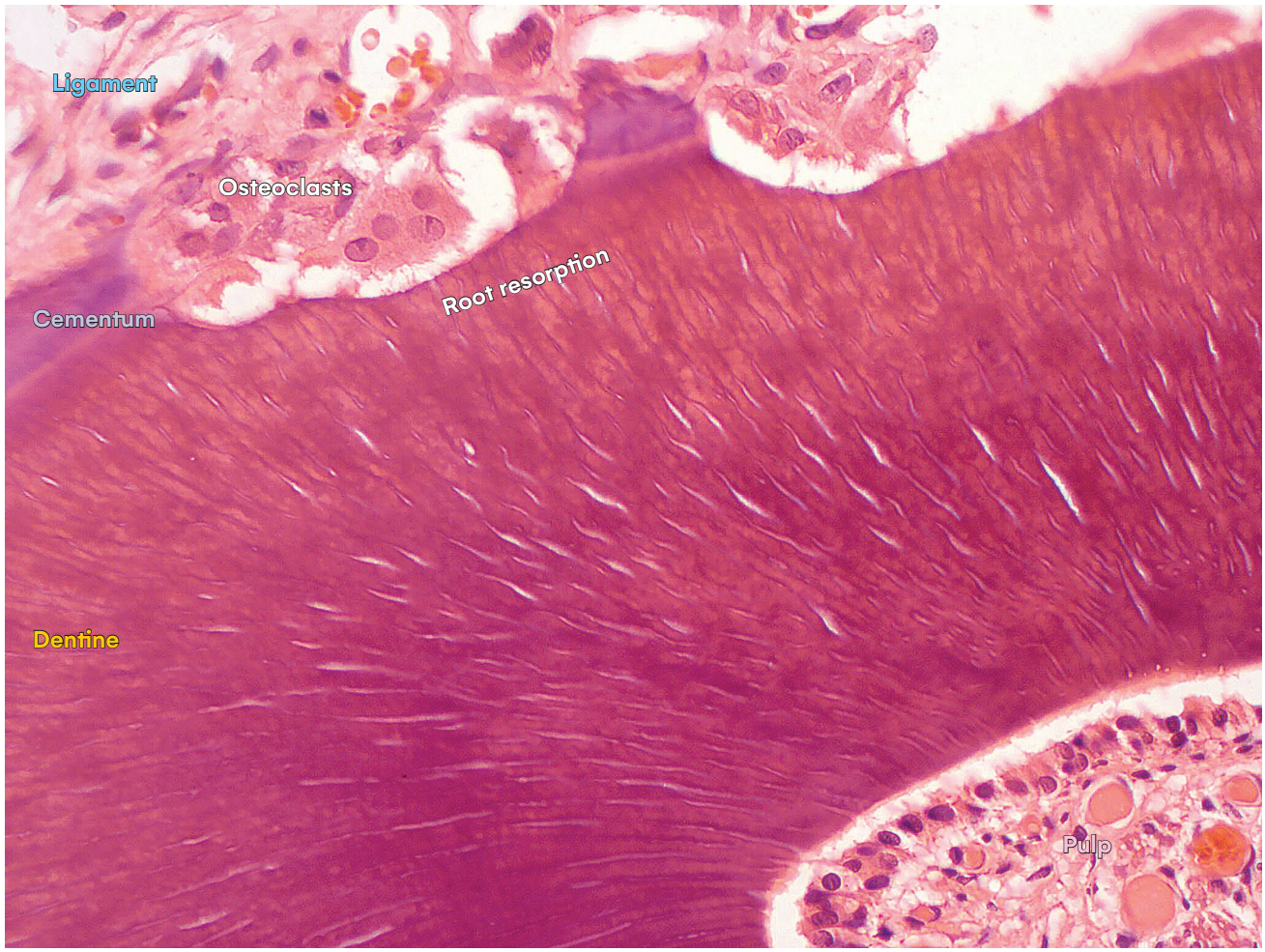


Figure 9:
Microscopic appearance at greater magnification of rat molar tooth on axial plane nine days after application of strong orthodontic forces that induce extensive root resorption. Pulp looks normal (HE, original magnification 40X).

ORTHODONTIC MOVEMENT AND DENTAL TRAUMA: HOW TO INTERPRET THEM CLINICALLY

Pulpal changes supposedly assigned to orthodontic treatment are, in fact, associated with dental trauma, particularly concussion. Dental trauma in the form of concussion may lead to silent aseptic pulpal necrosis and calcific metamorphosis of the pulp.[2,4,5] During orthodontic treatment, when there is pulpal necrosis or calcific metamorphosis of the pulp, we may confidently assert that the cause was tooth concussion.

Dental trauma induces sudden and abrupt movements of a tooth in its alveolus when the trauma forces act, which may damage the vascular and neural bundles that cross the apical foramen to supply pulpal tissues.[5]

As described above, orthodontic forces are dissipating, and they have to be weak or moderate to produce effective tooth movement. Intense forces hyalinize the periodontal ligament and impede tissue and cell phenomena that characterize tooth movement.

When dental trauma specialists and researchers are asked about the periodontal and pulpal effects of tooth concussion, their answers are often evasive. The literature about tooth concussion and its effects on tissues remains undervalued in the field of Dental Trauma, which still has experimental models focused on fractures, luxation, avulsions and reimplantation.

One of the reasons for that is the clinical relevance of these more severe types of dental trauma. Another reason in the difficulty to develop experimental laboratory and clinical human models to reproduce tooth concussion, because of its incipiency and subtlety.[5]

Tooth concussion is a type of trauma that does not induce immediate clinical changes after the shock to the affected teeth, and only discrete symptoms of pain or discomfort may, in some cases, occur for a few hours and then naturally disappear.

Patients that suffer concussions are not aware of the fact that it is a type of dental trauma, and hardly ever will report it when describing their history some months or years after the accident The main complaint of patients that had a concussion will emerge only some months or years later, when the tooth crown becomes discolored.[4]

Crown discoloration of apparently healthy teeth is the result of only one of two pulpal diseases, both induced by the same cause – dental trauma, especially concussion: aseptic pulpal necrosis and calcific metamorphosis of the pulp.[4,6,7] In other words, aseptic necrosis and calcific metamorphosis of the pulp are, obligatorily, associated with dental trauma. When patients do not report any occurrence of dental trauma, concussion should be treated, because it often had no symptoms important enough to make patients recall the trauma that, in fact, occurred!

TWO EXAMPLES OF SCIENTIFIC EVIDENCE IN HUMANS AND ANIMALS

In 2000, Valadares Neto,[8] in a study for his Master's degree, microscopically analyzed the dentinal-pulpal complex and the external root surfaces of teeth of 12 human adolescents extracted after rapid maxillary expansion and compared them with the teeth of other three adolescents that underwent no tooth movements.

The analysis of the dentinal-pulpal complex revealed, according to Valadares Neto,[8] that:

» No dentinal or pulpal changes were found in the evaluation of immediate response and response at 120 days after retention.

» No dentinal or pulpal changes were found in cases of two (0.45 mm) and four (0.9 mm) daily activations of the expansion screw.

» Rapid maxillary expansion using a modified Haas expander was a biologically safe procedure for the dentinal-pulpal complex.

The forces applied in the rapid expansion of the maxillary are very intense because they have to hyalinize the buccal periodontal ligament and to ensure that there is no tooth movement. Although the orthodontic forces are applied to the teeth, no pulpal changes were seen under microscopy.

In 2005, Consolaro[9] described tooth movements in 39 rats during one to seven days using the model initially developed by Heller and Nanda,[10] recognized worldwide as the most frequently used in studies in this area. The pulpal tissues were analyzed[9,11] and compared with teeth that were not moved in other nine animals. They concluded that induced tooth movement did not result in morphological changes of the tooth pulp, whether degenerative or inflammatory, detectable under microscopy.

We often try to detect molecular, biochemical and enzyme differences in pulp that underwent orthodontic movements, but results are not visible or associated with morphological changes, and neither detectable under microscopy.[12]

In several studies[13-18] conducted to investigate periodontal variables and changes induced by tooth movement using the same standardized experimental model and hundreds of specimens, pulpal tissue was equally normal, regardless of whether movements were weak or strong (Fig. 4 and 5), in teeth with periodontal hyalinization (Fig. 6 and 7) and in teeth with severe root resorption (Fig. 8 and 9). The pulp of teeth that underwent orthodontic movements were morphologically normal in practically all the studies that used photomicroscopy. The same was found in all the studies that allegedly detected pulpal effects of orthodontic procedures, but the photomicroscopic analyses revealed incredible interpretation errors, such as:

» Classification as abscesses of the negative or white spaces that were generated on the slides due to the sectioning effect and that were surrounded by odontoblasts. Pus forms only in the presence of bacteria interacting with neutrophils. In healthy teeth moved orthodontically, there is no possible explanation for such fact.

» Assigning congested pulpal blood vessels to lesions induced by orthodontic forces. Extractions leave congested vessels in any pulpal tissue, and these phenomena should not be classified as degenerative changes.

Description of inflammatory cells, that is, lymphocytes, in pulp with a preserved odontoblastic layer, when they were, in fact, cells of the subodontoblastic layer that, due to the histological sectioning for analysis, were more diffusely distributed.

FINAL CONSIDERATIONS

Orthodontic movement does not induce pulpal necrosis or calcific metamorphosis of the pulp, as revealed by clinical studies with humans and laboratory animals.

No human clinical or experimental models in the literature confirm or show minimum evidence of pulpal changes induced by orthodontic movements.

When pulpal necrosis or calcific metamorphosis of the pulp is diagnosed during orthodontic treatment or immediately after the removal of orthodontic appliances, its etiology should be assigned to the concussion, a type of dental trauma, and not to orthodontic treatment.

The two pulpal diseases that lead to crown discoloration in apparently healthy teeth are aseptic pulpal necrosis and calcific metamorphosis of the pulp, both induced by dental trauma exclusively.

Finally, concussions are a type of dental trauma that should be the focus of several clinical and laboratory studies using pertinent experimental models to further explain their effects on periodontal and pulpal tissues.

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