

Orthodontic treatment does not cause pulpal necrosis

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Introduction

The dental pulp has an arborized vascular system and its only blood source is represented by a delicate artery that penetrates the apical foramen. Seldom there is a vascular communication of the pulp with the periodontal ligament through the lateral canals and accessories from the lateral and apical foramina.

Connective tissue has many functions such as filling in the spaces between the organs, ducts and other structures. Another important function of connective tissue is the support of specialized cells in organs such as liver, kidney, pancreas and glands. In these organs, the connective tissue support is called stroma and the specialized cells parenchyma. The connective tissue can, besides filling and supporting, assume very specialized roles as in the dental pulp, which provides sensitivity and form dentin.

There are different kinds of connective tissues such as fibrous, bone, cartilage, adipose and others. They are the only vascularized tissues and they get blood to nourish and keep the cells alive and functional. Just as the vessels, there is a conjunctive plot of neural threads.

During compression and massage of soft tissues of the body, major shifts in centimeters can occur without breakdown of connective tissue structures, especially of vessels and nerves. This elasticity of the connective tissue is caused by the presence of collagen and elastic fibers in the extracellular matrix, especially when subjected to forces applied gradually and slowly. When there are sudden movements

of conjunctive structures, there may be disruptions of vessels and nerves. When this occurs bruises are formed clinically characterized by reddish-purple spots. These hematomas can occur after pinching, biting and hitting, i.e., sudden traumatic and intense actions on the fibrous connective tissues.

Dental traumatism, occlusal trauma and orthodontic movement: are different in their effect the in periodontal tissue

Although the terms traumatism and trauma account for the deleterious action of physical agents, as forces on the tissues, their characteristics are not always equal in intensity and frequency. The dental traumatism can break vessels and promote ruptures, characterized by the application of sudden and intense forces on the teeth. Differently from the occlusal trauma, in which the forces are intense, small extension, short duration, but constantly repeated. On the other hand, the forces of tooth movement are very light, even the most intense and prolonged ones slowly applied on the teeth, so that they gradually disappear within hours or days. In short, although they are caused by forces, dental traumatism, occlusal trauma and orthodontic movement are not alike regarding the characteristics of the forces applied and their effects on connective tissues.

In dental traumatism the forces act abruptly with rupture of connective structures, including vessels and nerves. When a force apparently light acts on the tooth, depending on its angle of incidence and

location in which it acted, there may be a resultant of forces in the apical third of the tooth root with rupture of the vascular and nerve bundle that enters the pulp. An example of dental traumatism is the concussion, with no clinically detected mobility and pain, if any, is easily controlled with common analgesics, lasting several hours or even 2 to 3 days.^{2,3} Apparently, the tooth gets back to normal, but within time the pulp may show its damage with the presence of calcium metamorphosis of the pulp or pulp aseptic necrosis, both clinically revealed by coronary darkening in an apparently healthy tooth.

In occlusal trauma the death of cell and the structural rupture are minimized by the quick length and repetitiveness of the process, although it is for a long time. In this case, there is no structural damage to vascular and nervous bundle of pulp, nor fast aging of the pulp. The periodontal lesions are light and subtle. The periodontal structure must be acknowledged as an example of an organization to receive the strong forces of chewing. The periodontal fibers are organized in a space with an average thickness of 0.25 mm, but even so during chewing the teeth do not touch the bone.

In the orthodontic movement the forces applied to the tooth structure, even the most intense, gradually disappear in the surrounding tissues. The plasticity of the connective tissue of the periodontal ligament, plus the deflection capacity of the bone crest and the rotation that happens in the tooth socket promote a slow and gradual adaptation of the surrounding tissues. The orthodontic movement is limited to a maximum of 0.9 mm at the crown during the first hours¹ providing no conditions for the structural rupture of vessels and nerves to happen.

There should not be a comparison among the tissue effects induced by dental traumatism, occlusal and orthodontic movement, as they are different situations. In the apical third of root the induced orthodontic tooth movement is confined practically to the compression of the periodontal ligament, because the bone deflection in the periapical bone is much smaller and the tooth hinge axis is near the apex. The forces are absorbed and dissipated slowly, without rupturing vessels. Small movements are naturally absorbed by fibrous and elastic connective tissue.

Another important information concerns the duration in which the orthodontic forces are active: 2 to 4 days. After this time these forces are dissipated and the reorganization of the periodontal structures begins with resorption of the periodontal bone surface, cell migration for reorganization with the production of new collagen fibers.¹ After 15 to 21 days the periodontal ligament and other structures are ready for a new cycle of events by the reactivation of the orthodontic appliance. In other words, the induced tooth movement is achieved in cycles of 15 to 21 days, the tooth does not move all the time. In the orthodontic movement forces are mitigated by the collagen and elastic fibers, without damaging the structures that carry blood and sensitivity to the pulp.

With each activation period of orthodontic appliances — from 15 to 21 days — the periodontal tissues reorganize themselves and return to normal. The ultimate effects of orthodontic treatment on the structures and position of teeth are the sum of all cycles from 15 to 21 days. The forces and the effects were not continuous and unceasing. Sometimes the question is: when there is rotation of the tooth around its long axis, as in giroversion, vascular and nerve bundles get twisted around themselves, does it not compromise the blood supply to the pulp? No, they are not twisted, because the tissues reorganize themselves in each period of 15 to 21 days, they return to their normal position and relationship. When the new cycle of movement is established by a new activation, the vessels and nerves are in normal relationship with no change in their shape. Tissues constantly renew its structures, remodel and adapt themselves well to new positions and structural relationships.

Consolaro,⁴ in his investigation of Masters in 2005, and Massaro et al⁹ in 2009, examined microscopically the pulp of 49 first molars of rats under induced tooth movement after 1, 2, 3, 4, 5, 6 and 7 days. Resorption was detected in the external surfaces of the root, indicating the efficiency of the applied forces. However, no morphological changes was detected in the pulp tissues (Figs 1-6).

Synopsis for endodontists of the induced tooth movement, or does intense force increase the chance of pulpal necrosis by orthodontic movement?



Figure 1. Rat's molar 7 days after been moved. P = pulp, D = dentin, C = cementum; PL = periodontal ligament, AB = alveolar bone, G = gum. (HE; 4X).

The orthodontic forces compress a certain segment of the periodontal ligament, because the teeth are bent on the alveolar bone crest or on the apical third on the opposite side (Figs 1 and 2). The compressed blood vessels reduce the amount of blood to the cells of that local: they momentarily stop the production and renewal of the extracellular matrix, including collagen; and get disorganized (Figs 3, 4 and 5).

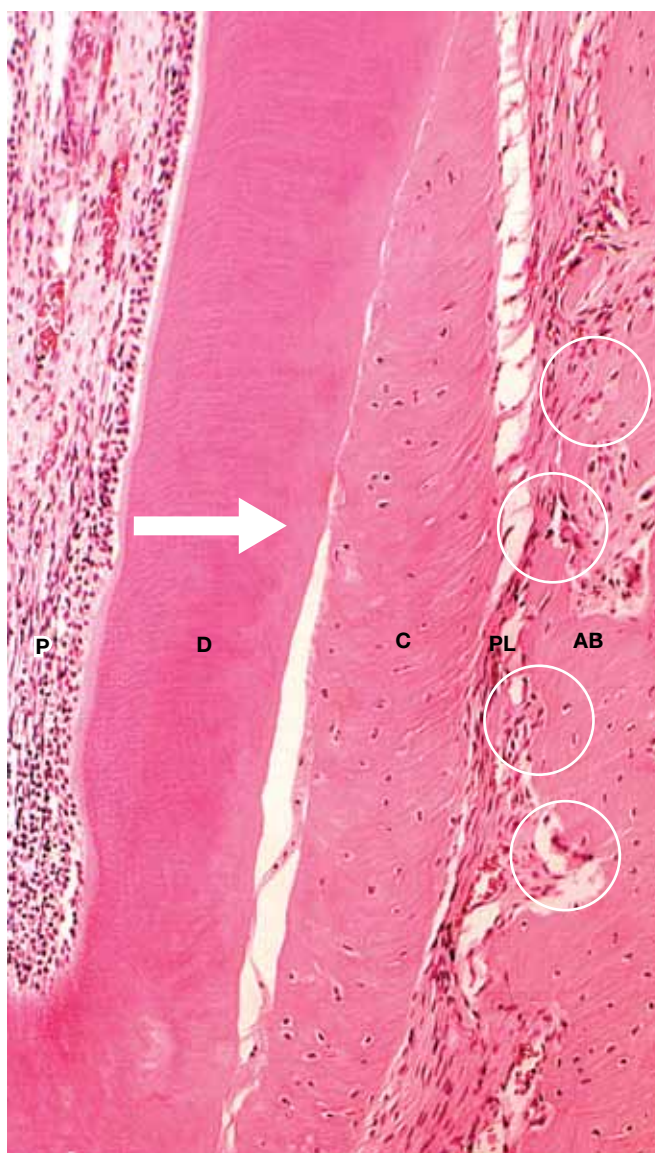


Figure 2. Area of compression of the periodontal ligament (PL) of the rat's molar 7 days after been moved. The arrow indicates the direction of the applied force and the narrowing of the periodontal space. Despite the compression of periodontal ligament, cells and fibers are present in the area, as well as cementoblasts, osteoblasts and also the clasts (circles). The morphological pattern of normal dental pulp is highlighted (P). D = dentin, C = cementum; AB = alveolar bone. (HE, 25X).

In some cases the cells migrate to surrounding areas still vascularized. Only the extracellular matrix in some areas that have been strongly affected by hypoxia remains in the local. These areas turn into a glassy aspect to the optical microscope and are, therefore, called hyaline areas (Fig 6).

In this segment of the compressed periodontal ligament and with reduction of blood support, there will

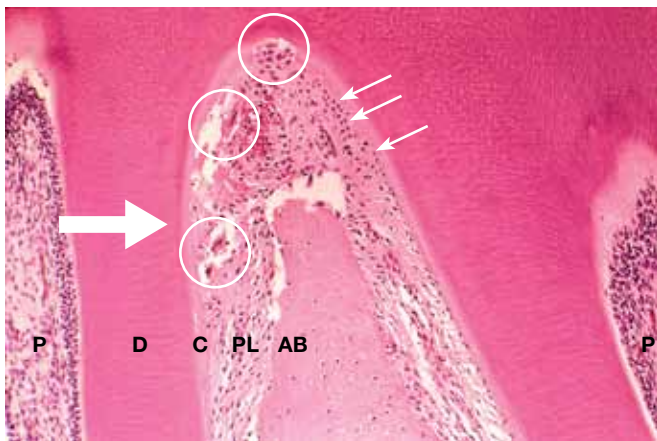


Figure 3. Area of compression of the periodontal ligament (PL) of the rat's molar 7 days after been moved. The larger arrow indicates the direction of the applied force. The small arrows indicate the cementoblasts, which are absent in the area of pressure, indicating efficiency of the applied force. Despite the compression of periodontal ligament, cells and fibers are present in the area, as well as cementoblasts, osteoblasts and also the clasts (circles). It is important to notice the morphological pattern of normal dental pulp (P). D = dentin, C = cementum; AB = alveolar bone. (HE, 25X).

be an increased local production of cellular mediators produced by metabolic stress and by the mild induced inflammation. The periodontal ligament is alive, metabolically viable, with blood supply and with clasts sufficiently activated to resorb the periodontal bone surface of the tooth socket (Fig 5). The periodontal bone resorption occurs in front of the compressed periodontal ligament and therefore it is nominated frontal bone resorption (Fig 5). Gradually, over few days, the tooth will be displaced to one side of the tooth socket, reoccupying its new place, and ligament cells restore the average thickness of 0.25 mm. In the process, especially in the apical region, vascular rupture does not occur in tissues that enter into the root canal. From this normal restored relationship, the periodontal ligament and surrounding tissues will be reorganized in a few days. After 15 to 21 days it is ready to reactivate the appliance as the tissues return to normal.

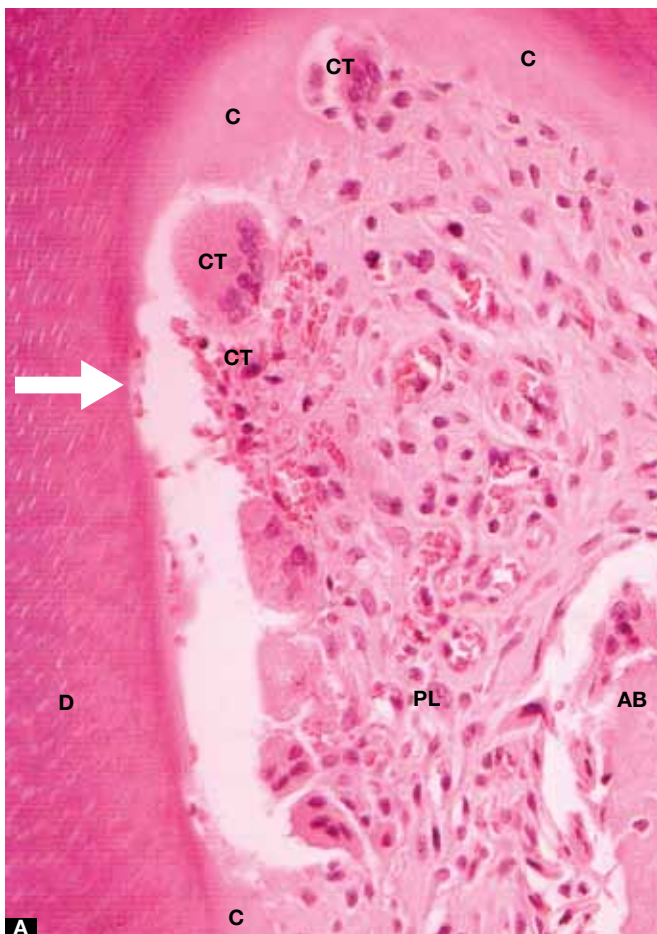


Figure 4. A) Area of compression of the periodontal ligament (PL) of rat's molar 7 days after been moved. The arrow indicates the direction of the applied force. The clasts (CT) in the root surface indicate efficiency of the applied force. In **B**, there is the morphological pattern of normal dental pulp with odontoblastic layer (small arrows). D = dentin, C = cementum; AB = alveolar bone. (HE, 40X).

The displacement of the root apex is very small and slow, the connective tissue is elastic enough to withstand much larger displacements. Besides having elastic fibers, the extracellular matrix of connective tissue display a gel between the cells and fibers, damping forces and applied displacements, without cell death and vascular rupture (Figs 1 and 2).

When a very intense force, as the one applied to the teeth that act as support for jaw expander appliance, acts on the tooth there will not be an effective movement of the tooth in the tooth socket. A very intense force collapses the blood vessels, interrupts

normal vascularization in that periodontal segment. The local cells die, or, more often, flee to surrounding areas, including inflammatory and clast cells (Fig 6). Without blood supply there will be no cell activity in the periodontal surface of the alveolar bone. That is, the compressed periodontal segment gets hyaline in these conditions and without any cell activity (Fig 6).

When the vascularization is restored due to the gradual dissipation of excessive force applied, the neighboring cells will change from center to the periphery, resorbing and remodeling the hyalinized area of the periodontal ligament. Therefore, the tooth will

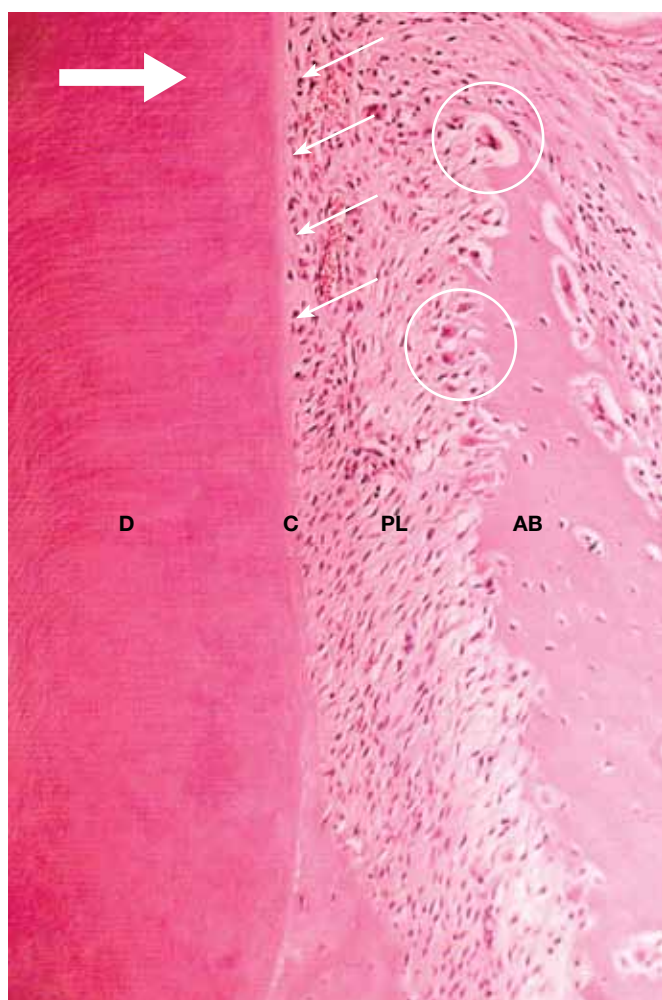


Figure 5. Area of compression of the periodontal ligament (PL) of the rat's molar 6 days after been moved with typical *frontal bone resorption*. The larger arrow indicates the direction of the applied force. The small arrows indicate the cementoblasts. Despite the compression of periodontal ligament, cells and fibers are present in the area, as well as cementoblasts, osteoblasts and clasts (circles). D = dentin, C = cementum; AB = alveolar bone. (HE, 25X).

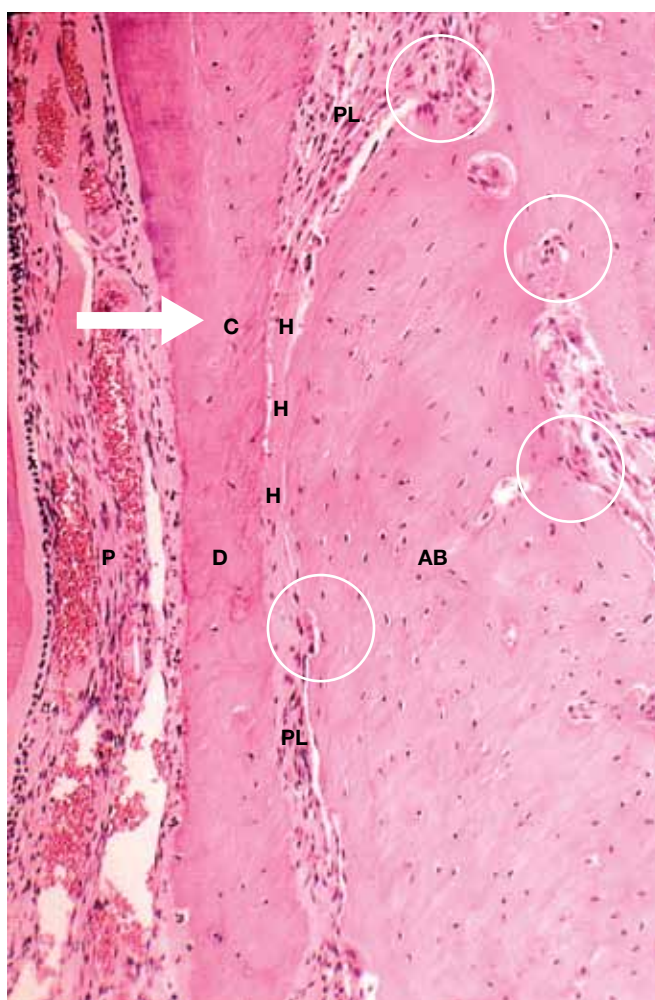


Figure 6. Classic *bone resorption at distance* in the area of compression of the periodontal ligament (PL) of the rat's molar 3 days after been moved. The arrow indicates the direction of applied force and the narrowing of the periodontal space. The area of compression of the periodontal ligament was hyalinized (H) without osteoblasts and cementoblasts. The clasts (circles) act at a distance from the compression area of the periodontal ligament (PL). The morphological pattern of normal dental pulp is highlighted (P). D = dentin, C = cementum; AB = alveolar bone. (HE, 25X).

not move because the clasts are not in metabolic conditions of nutrition and with no metabolism to act in the periodontal surface of the alveolar bone. The remodeling process of bone and hyaline area will be done from the periphery to the center, including the underlying part of the alveolar bone plate (Fig 6).

The bone resorption process and reorganization that should take place in front of the compression of the periodontal ligament, will take place at distance: *bone resorption at distance*, but it is undesirable (Fig 6). In this case, the tooth did not move neither displaced minimally, thus can not have broken the vascular and nerve bundle.

This explanation helps to understand why the teeth that act as anchoring for palatal expansion appliances, the strongest possible force to be applied to a tooth, do not suffer necrosis neither pulp aging. In short, *the more intense the orthodontic force applied is, the smaller the chance of the tooth to move in its socket; and as consequence, there is no way to infer associated pulp necrosis*. Valadares Neto,¹³ in his master research in 2000 with me as advisor, analyzed the effects of rapid maxillary expansion in the dentin-pulp complex in 12 adolescents. Using devices like Hass, he examined microscopically the entire length of the pulp and dentin of 12 premolars right after the removal of the appliance with the expansion of the jaw established and other 12 premolars after 120 days from the removal of appliances. Other 6 premolars of adolescents that did not undergo any orthodontic and/or orthopedic procedure were used as control group. In every analyzed teeth the pulp-dentin complex was fully normal, without any microscopically detectable change.

And when the pulp necrosis is diagnosed in sound teeth during the orthodontic treatment?

Based on the above explanations, it is perfectly possible to understand why orthodontic treatment does not induce pulp necrosis nor accelerates its aging. In all the cases in which pulp necrosis is detected during orthodontic treatment, the history of dental traumatism must be recalled. Patients do not

remember those concussions and small dental injuries in children, but they occur daily. Small strokes, bump and home accidents can be seemingly innocent, but by concentrating forces at the apex they may cause sudden and small displacements with rupture in the pulp vascular bundle. In many cases of dental traumatism, no coronary nor gingival damage or bleeding occur, but there may be aseptic pulp necrosis. In some dental traumatism, there may be severe gingival damage and heavy bleeding, but without breaking the pulp vascular bundle.

Dental concussion can also occur in the following situations: teeth that act as levers to support the extraction of adjacent teeth, small forceps beats in the extraction of third molars, unerupted and pulled canines luxation, laryngoscope trans-operative beats during general anesthesia, or even accidental bites in cutlery, seeds or strange materials during feeding.

There is no clinical, laboratorial or experimental evidence to assign, although theoretically, the pulpal necrosis as a result of orthodontic movement.^{5-8,10,11,12} When facing a situation like this, try to recall the history of dental traumatism and do not assign pulpal necrosis to the orthodontic movement.

Final Considerations

1. The aseptic pulp necrosis cannot be attributed clinically and experimentally to orthodontic movement.
2. In cases of pulpal necrosis during orthodontic treatment, the history of dental traumatism should be researched, especially the lighter types, such as concussion.
3. In cases of very strong forces used in orthodontic and orthopedic treatment, tooth movement does not occur and displacement with rupture of the pulp vascular bundle has even less chance of happening.
4. Dental traumatism, orthodontic tooth movement and occlusal trauma situations are totally different from each other, although they are physical events on the tissues. The biological effects in each of these three situations are different and specific and therefore not comparable.

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