

## **Does pulp have a role in root resorption?**

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One of the most common clinical conclusions of studies related to orthodontically induced inflammatory root resorption (RR) is that lower force levels are preferred over higher ones. This conclusion might be valid for surface resorption of the roots, but not necessarily for apical root shortening (ARS). There are at least two examples of extremely high forces, many times more than 500–1000 g, namely rapid maxillary expansion and headgear. For both, according to the aforementioned clinical conclusion, it might be supposed that complete resorption of the roots would occur; yet this does not happen. This example demonstrates that there are two different processes in root resorption: one originates in the periodontal ligament (PDL), and the other originates in the pulp. Both are outcomes of decreased blood flow. The differences between the nature of those reactions and their outcome, although the distance between them is minimal, are immense.

We believe that this concept explains why there has been no breakthrough in understanding the orthodontic ARS process so far. Data kept being added on as more studies were published, but the assumed major pathway remained almost the same: In short, orthodontic force application (OFA) activates an aseptic inflammatory process due to a local decrease in the blood supply to areas of the PDL. This inflammation results in orthodontic tooth movement (OTM) and root surface resorption, both microscopic on any root surface, or severe ARS. The specialty was not excited even when Oppenheim, many years ago, pointed out that these were two different processes.<sup>1</sup>

In 2014, at the American Association of Orthodontists (AAO) convention, and also published subsequently in *The Angle Orthodontist*, the term “orthodontitis” was presented as a new generic name to the process.<sup>2</sup> This name, we thought, better describes the aseptic inflammatory processes that orthodontists develop by OFA. We even divided

orthodontitis into two different processes: “instrumental orthodontitis” (IO) and “instrumental detrimental orthodontitis” (IDO). IO is orthodontitis on the outside surface of the tooth, mainly in the PDL. With IO, there are usually no radiographic signs of damage to the teeth. However, its traces can be detected microscopically, and rarely as surface irregularities, on all root surfaces. IDO describes orthodontitis leaving pathognomonic scars at the apices of involved teeth that are detected by radiographs during or after treatment. IDO ranges from apical surface irregularities to losing a large amount of root material. IO and IDO do not damage the blood supply or the nerve transmission signals to and from the crowns of the teeth.

IDO is the topic we would like to address. It is a problem that may affect the prognosis of damaged teeth and, with time, has also become a legal problem in which members of our own community support the prosecution. Ironically, one day, those same members may become victims of similar problems because the cause of IDO remains unknown. Indeed, none of the protocols suggested in the literature to avoid IDO can actually prevent it. Although relatively rare, IDO can be an immediate reaction after a few months in orthodontic treatment even though all precautions suggested by any protocol were taken. Additionally, the recommended 6- to 9-month progress periapical radiographs of the incisors often show no changes, thus clearing the way to continue treatment without restrictions. Even with this type of confirmation, the final radiographs of the same teeth could show them as victims of severe IDO. That might be one of the worst moments in a good practitioner's life that might cause severe anxiety and might lead to a lawsuit. Unfortunately, over the years, we did not succeed as a specialty in defending our own members by proving what needs to be shown: that there is no way to prevent ARS in patients desiring orthodontic treatment.

It is known that IDO cases are aseptic. Additionally, even in cases where most of the root is resorbed, the remainder of the tooth continues to survive, and there is definitely no need for root canal treatment. From our experience, this fact is not common knowledge among all dentists, and we often see unnecessary root canal treatment in IDO-damaged teeth.

Considering that the topic of orthodontically-induced inflammatory root resorption is a popular research area

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in orthodontics, the question is, why do so many unknowns remain?

We are not sure, but we believe this is the first time this theory is being presented. The basis of this new theory is described by the following parameters:

1. No theory or actual findings, mechanical, biological, even genetics, or others, can clearly explain the IDO process we encounter, especially in its severe form.
2. No parameter can prevent or predict IDO, as the AAO website says and, according to our theory, this likely will remain the case.
3. Most of the past and present in vivo short-term studies describe IO rather than IDO.
4. The amount of the root surface material loss measured during in vivo short-term studies of 2 months or more is minute. If this amount were used for quantifying the time it would take to resorb what is often lost during full treatment, it would have taken many years to reach severe apical root loss.
5. Reports published in the 20th century about severe IDO cases showed that most of the process happened in the last stages of treatment: the finishing stage when torque and intrusion/extrusion movements are usually executed. Thus, IDO can be associated with certain movements and their known contribution to this process, but the short period it takes for this IDO to develop is also noteworthy.
6. The foundations of most, if not all, clinical recommendations to prevent IDO are based on IO and not IDO study outcomes.
7. Several studies reported on the pulpal reaction and changes resulting from OFA but, no study, as far as we know, ever related those changes to IDO.
8. The body has many defense mechanisms (DM). Most DM are reactions to a local decrease in the blood supply to organs or even to significant parts of the body itself. For example, when a main artery in the heart is partially closed by growing fat depositions or plaques, the DM is expressed by developing collateral blood supply. In cases with severe blood loss from the body, the DM shuts down the blood supply to the kidneys and bowels to save the more essential organs: the brain, the heart, and the lungs (shock). As we know, even orthodontic root movement is the outcome of a local DM to necrosis due to a decrease in the local blood flow. IO and IDO result from two different DM to a decrease in local blood supply.
9. Femoral avascular necrosis<sup>3</sup> is an excellent example of bony tissue necrosis of a part of an organ due to interruption of its blood supply.

10. Teeth with incomplete root formation, in which the apex is open, are not shortened during orthodontic treatment.

Therefore, we believe that all those parameters lead us to the insight that we missed something since, for years, we concentrated almost exclusively on analyzing the side effect of this aseptic inflammatory process on the root surface. We probably missed that there is another organ, very close by, that might be the one that is, perhaps, more directly responsible, by itself or in conjunction with the PDL aseptic inflammation, for the extensive loss of root material. We are referring to the pulp.

The pulp is the organ that supplies all the needs to the rest of the tooth. The pulp, according to researchers, is known for "its uniqueness that can be appreciated only when provoked."<sup>4</sup> Therefore, we believe that IDO might be the outcome of the integration between two different local reactions to OFA: one is in the root surface zone and the other is in the pulp. Both reactions are related to the decrease in the local blood supply, leading to local necrosis. The difference is that the PDL is a relatively open zone. Although it is encircled by bone and cementum, its blood supply and drainage can be considered unlimited compared to that of the pulp. According to many studies, the necrotic PDL and the injured cementum are fully or almost entirely regenerated most of the time.

On the other hand, the pulp is in a nearly closed area. If there is an injury to the pulp, the recruitment of extra blood supply and inflammatory components is probably close to impossible. The blood supply to the pulp is most often through a small orifice located at the apical foramen. This foramen is positioned in different areas near the apex, but not always at the anatomical apex. The location of the apical foramen relative to the alveolar bone might be crucial in orthodontics. The force that moves the apex can cause changes in the bloodstream to the root. If the bloodstream decreases, the provoked pulp probably reacts. This reaction, as we assume, is expressed by shutting down the blood supply to parts of the pulp itself. Studies demonstrated that the blood supply to the apical zone is about five times less than the blood supply to the crown.<sup>5</sup> If there is a decrease in the blood supply, the first zone to be damaged, maybe by reactive constriction of arterioles, is the apical zone. This is probably also the most sensitive area in the root. Theoretically, the crown is the more 'important' part of the tooth. It is needed for biting and chewing food. Therefore, by sacrificing the apex, nothing or almost nothing will change for performing the tooth's role. It might even be that, by resorbing the apex or parts of it, the localized

decreasing blood supply effect of the force on the arterioles will vanish, and more blood will reach the pulp, restoring the previous order. Therefore, we believe that the damage we observe is directly related to the decrease in the blood supply to parts of the pulp nourishing the local dentin and cementum. At the same time, we have to remember that there is a “storm” next to it, in the PDL. The outcome of this PDL storm is cementum damage that causes it to lose its role to protect the inner side of the tooth, thereby allowing inflammatory substances to flow there. We think that the contour or the outline of the damage can only result from a decrease in the blood supply inside the pulp. The process always resorbs the apex fully or partially; the ghost of the nonresorbed site can be detected on many x-rays, demonstrating the resorption outcome.

In conclusion, we understand that the tooth is a unique structure with blood supply from both sides, and each side reacts differently to a decrease in that blood supply. We have presented this theory to explain extensive root material loss that occurs relatively quickly, by showing the pulp’s involvement and its control on the internal blood supply to different parts of the root. We are trying to demonstrate a new comprehensive theory explaining IDO. This theory discusses the integration between two processes: one is in the apical area of the pulp, resulting from a local decrease in its blood supply due to tooth and root movement; the second is in the PDL (IO) which, by itself cannot explain significant apical resorption. However, this PDL destruction can further increase the pulpal reaction by damaging the protective role of the cementum layer. This makes the penetration of inflammatory substances through the canaliculi into the

pulp easier. This penetration might become an additional provocation to the pulp, already damaged from decreased local blood supply.

Finally, this theory further reduces or even nullifies the importance of the force level in developing IDO. It might be that, under certain morphological or anatomical conditions, with the apical foramen located next to the bone, even very low forces might provoke the pulp by decreasing the blood supply to the root, leading to IDO.

We believe that this hypothesis might not only highlight the fact that there is a lot of information not known about IDO, but also that it will create new opportunities to study the devastating outcome of the process, and maybe it will decrease or change the orthodontic community’s attitude in the legal field of this subject.

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