The concept of Tooth Resorption and why it does not induce pain or necrotic pulp

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The concept of tooth resorption does not appear to be uniform in different scholarly studies, from a simple monograph to research texts published in the literature to dissertations. This article aims to contribute to the conceptual standardization of this important pathological process, which involves virtually all dental specialties, especially endodontics.

A concept can be defined as a mental representation of an object or phenomenon described by human reason based on the object's overall features. A concept can also be defined as the formulation of an idea in words. Concept can also be synonymous with conception, definition and characterization. In short, to conceptualize means to identify, describe and classify the different elements and aspects of reality.

In studies of tooth resorption, more often than not, the first sentence or paragraph is reserved for conceptualizing the very notion of tooth resorption. The concept is limited to a particular type or restricted to the context of a clinical case and does not take into account all issues involved in tooth resorption. Concepts should be of a general nature so as not to hinder understanding of the phenomenon as a whole. In some published studies[1-4] efforts were expended by the author(s), sometimes repeatedly and in different journals, to discuss the concept of tooth resorption candidly in an attempt to contribute to the formulation of future texts on the subject.

Tooth resorption: Two discrete mechanisms devoid of complexity, controversy or dispute

Two basic mechanisms have been well established in the occurrence of root resorption: Inflammatory and replacement.

Inflammatory Resorption mechanism

Cementoblasts "line" or "hide" the root surface while Sharpey's (collagen) fibers get attached in between them. The teeth are very close to the bone and separated by the periodontal ligament whose average thickness is 0.25 mm and ranges from 0.2 to 0.4 mm.

Bone is constantly remodeling through stimulation of local and systemic factors. This dynamism of the bone contributes to stabilizing the levels of minerals in the blood and imparts significant adaptive capacity to the functional demands on a daily basis. Bone remodeling depends on receptors located in the membrane of osteoblasts and macrophages, allowing local and systemic mediators to manage osteoclast activity. Osteoclasts have no receptors for mediators of bone remodeling and are functionally dependent on osteoblasts and osteoclasts.

On the other periodontal side, on the root surface, cementoblasts have no receptors for mediators of bone remodeling even though they are positioned very close to the bone. They do not respond to or "hear" the biochemical messages that induce resorption or

neoformation of mineralized tissue on the root surface: They are "deaf" to the mediators of bone remodeling, even though they have receptors for other mediators essential to cell life such as growth hormone and insulin, for example.

Any causative factor acting on the site where the cementoblasts are, removing them from the surface, is bound to expose the mineralized root surface. As a result, bone cells, given their proximity, will promote root resorption (Fig 1), even if only temporarily. Tooth resorption has local causative factors that eliminate cementoblasts from the root surface and as yet no systemic causative factor has been shown to produce this sort of effect in teeth.

Replacement Resorption mechanism

Bone remodeling involves constant resorption of mineralized structures, but concurrently, continuous bone modeling takes place, even on the periodontal surface of the tooth socket. Naturally, each new layer of bone deposited on the periodontal surface of the tooth socket would increase proximity to the tooth and, with an average thickness of 0.25 mm alveolodental ankylosis would soon develop. Cementoblasts and osteoblasts would intermingle and create areas where cementum and bone would merge, alternating randomly distributed areas of resorption and bone formation. But this does not normally occur due to the presence of epithelial rests of Malassez, a network of with long and 4-8 cells wide, which produce what resembles a basketball hoop on the periodontal ligament around the tooth root.

The epithelial rests of Malassez constantly release epidermal growth factor (EGF) - like all other epithelia of the body - to self-stimulate and proliferate, maintaining their structure. But at the same time, this mediator in the ligament stimulates bone resorption in the periodontal surface of the alveolus. Thus the periodontal space is maintained and alveolodental ankylosis prevented.

Alveolodental ankylosis occurs almost exclusively when the epithelial rests of Malassez are eliminated - usually by dental trauma – be it a mild concussion or the most severe avulsion. With alveolodental ankylosis bone remodeling also involves the mineralized dental tissues, which will gradually and inevitably be resorbed and replaced by bone (Fig. 2), hence the term tooth resorption by replacement. In long delayed unerupted teeth, severe atrophy of the periodontal ligament due to disuse may facilitate the development of alveolodental ankylosis.

Based on the description of these two potential mechanisms, it does not seem reasonable to state that tooth resorption is a complex phenomenon with unknown mechanisms. It also does not seem reasonable to assert that its causes are debatable or controversial.

The etiopathogenesis of Tooth Resorption is not multifactorial

The expression *multifactorial etiology* suggests that for a certain disease or phenomenon to occur a wide range of causative factors must act in concert, although strictly speaking this connotation is not explicitly apparent in the meaning of the word *multifactorial* as it is found in dictionary entries.

Dental caries is a classic example of a disease with a multifactorial etiology. The emergence of dental caries requires the presence of dentobacterial plaque due to lack of oral hygiene, a diet based on carbohydrates, the presence of caries-prone tooth enamel and enough time for these factors to interact and generate the disease. In other words, occurrence of the disease depends on interaction between these causes.

Diabetes mellitus etiology is also multifactorial as it requires inheritance of the gene responsible for autoimmunity against insulin-producing cells in the pancreas and interaction with environmental factors such as obesity, poor nutrition, sedentary lifestyle, stress and many others for the disease to emerge.

Tooth resorption has several causes that act independently of one another. In some special cases a number of causes might combine to cause tooth resorption, but this is not usual. From a conceptual point of view one should avoid stating that tooth resorption is multifactorial, although it would be accurate to assert that it has multiple or many causes. The term *multifactorial* may convey a mistaken connotation of simultaneity of causes for tooth resorption to occur.

The causes of tooth resorption are well known

In inflammatory tooth resorption causative factors remove the cementoblasts from the surface in the same manner as:

1) Chronic periapical lesions: Toxic bacterial products such as lipopolysaccharides (LPS), as well as other noxious microbial agents resulting from metabolism are

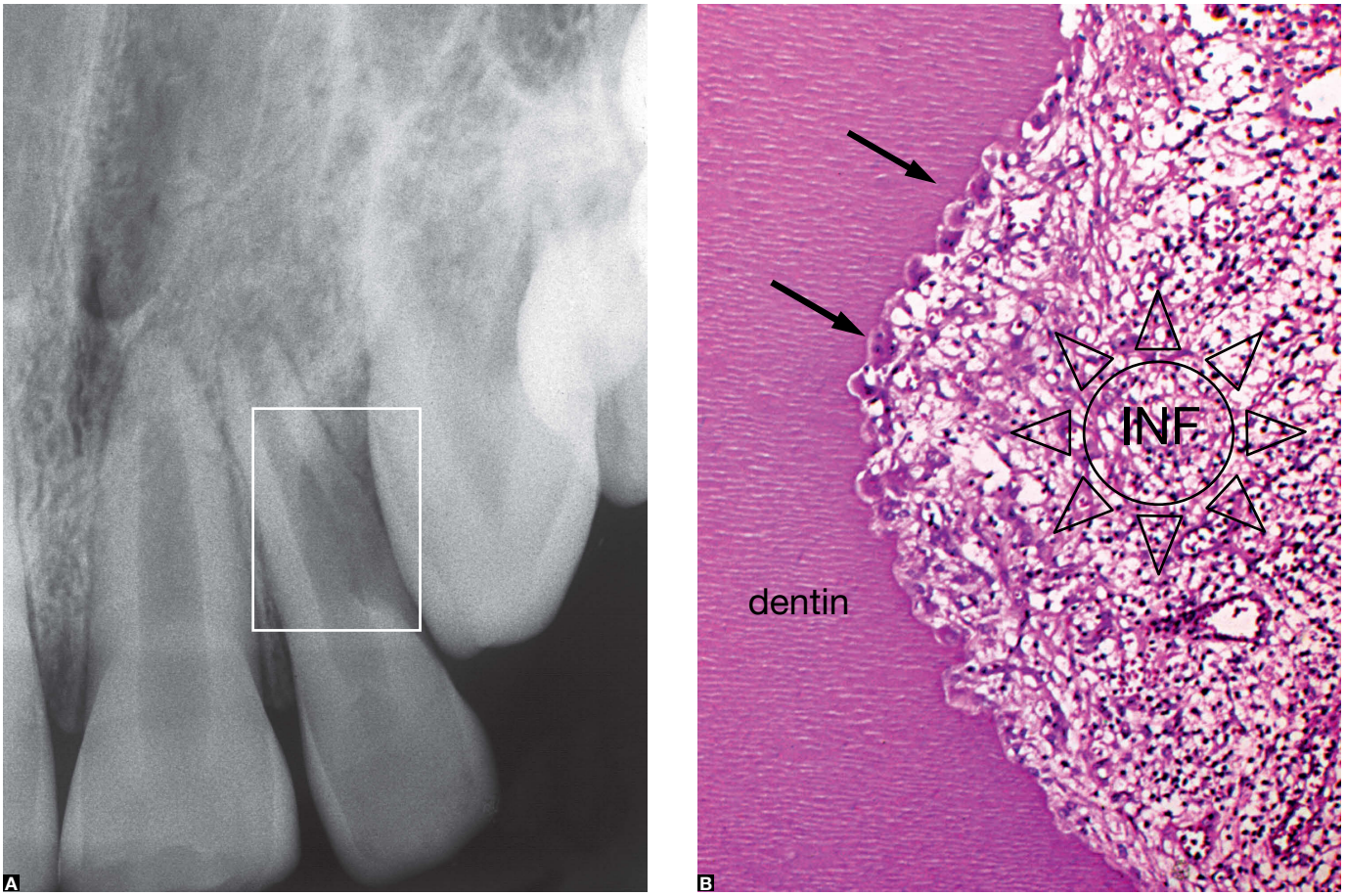

Figure 1. Inflammatory resorption by dental trauma and proximity to partially erupted maxillary canine. In B, it is emphasized that on the surface of teeth sharing the same condition, the osteoclasts (arrows) and other cells of the bone remodeling units are organized by mediators originating from the inflammatory process (INF) induced by the same causative factor responsible for the death of cementoblasts. The process is asymptomatic and of itself has no etiopathogenic relationship with the dental pulp, nor any symptoms.

either released into the periapical medium or reach the apical root surface via dentinal tubules. LPS are very toxic to human cells and, while some are killed by the cells, leukocytes release more inflammatory mediators when interacting with these molecules. In other words, LPS boost or amplify inflammatory phenomena, including any associated tooth or bone resorption.

2) Orthodontic forces can fully close the lumen of blood vessels and impair nutrition. On rare occasions the tooth-bone contact that results from excessive force can physically remove cementoblasts from the root surface by compression. The death of cementoblasts due to orthodontic movement is mainly caused by a lack of blood supply.

3) Unerupted teeth can compress the blood vessels of neighboring teeth when they are brought near to these teeth through the agency of eruptive forces, as is often the case with upper canines and third molars.

4) Accidental dental injuries can rupture blood vessels and put the tooth in contact with the alveolar bone surface (Fig 1). Dental trauma can be caused by surgical, operative and anesthetic factors.

5) Long periods of occlusal trauma can lead to death of cementoblasts and, in severe cases, induce inflammatory root resorption.

In replacement tooth resorption the causative factors eliminate the epithelial rests of Malassez in the periodontal ligament. The main and almost exclusive causative factor responsible for elimination of this ligament component is dental trauma (Fig 2), which can range

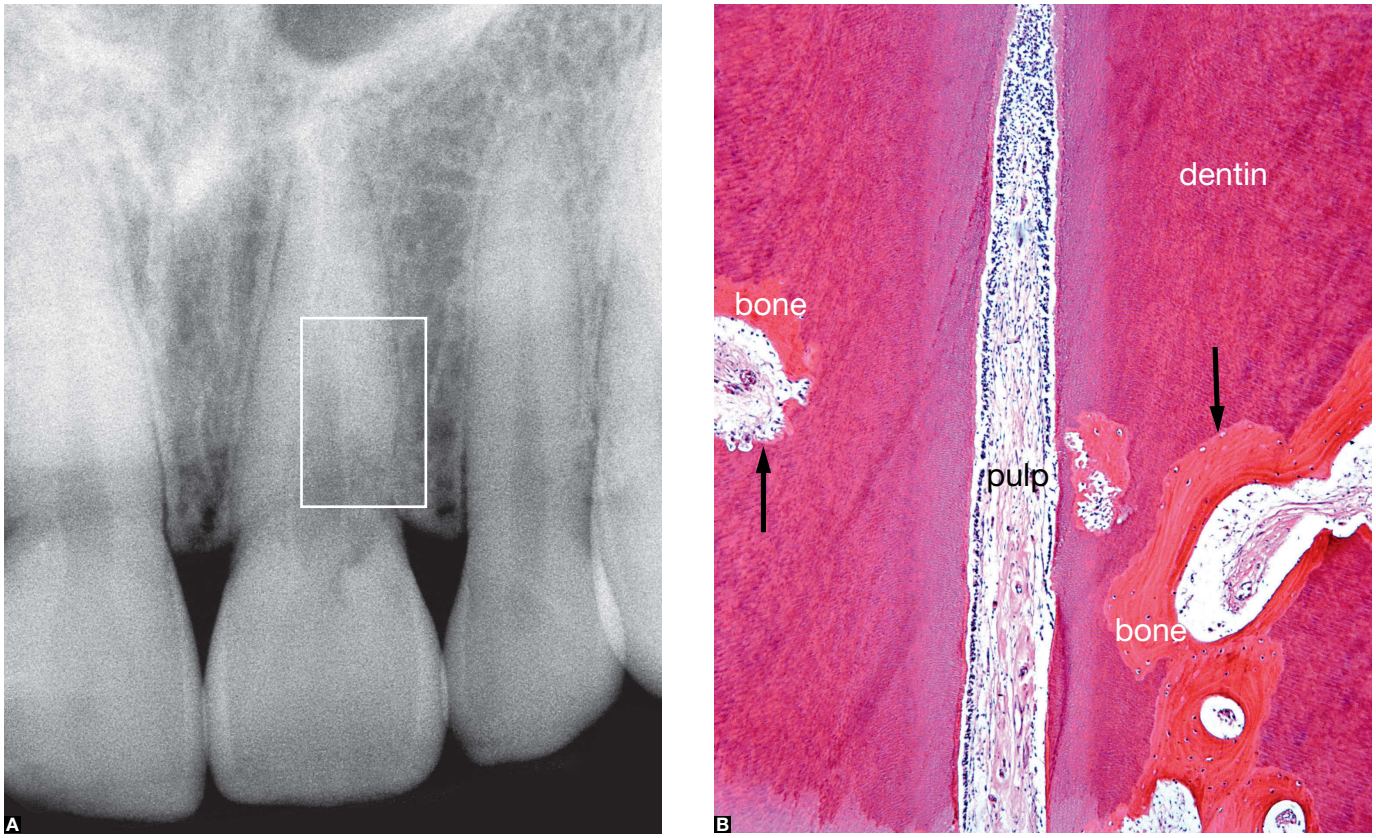

Figure 2. Replacement tooth resorption due to trauma. After ankylosis, dentin is gradually replaced by bone tissue, without any associated inflammatory process, as part of the remodeling process (arrows) that involved the tooth due to the death of the epithelial rests of Malassez. The process is asymptomatic and itself has no etiopathogenic relationship with the dental pulp, nor any symptoms.

from a concussion, in its milder form, to avulsion and reimplantation, in its most severe form. Dental trauma can be:

a) Accidental, when it occurs in leisure activities, car crashes, violence and other incidents.

b) Surgical, as in cases of untimely luxation of unerupted canines and during procedures aimed at removing unerupted third molars positioned over second molars.

c) By the action of laryngoscopes on the teeth during the process of intubation in general anesthetic procedures.

An additional, very specific causative factor refers to teeth that remain unerupted for long periods of time, whose excessive atrophy of the periodontal ligament may set the stage for the occurrence of alveolodental ankylosis and subsequent replacement resorption.

Even multiple tooth resorptions are not systemic or hereditary

In humans, cell and tissue events are triggered by information contained in genes, but this does not give a hereditary connotation to all these events. Root resorption, like all biological phenomena, involve genetically mediated cell and tissue events, but not necessarily implying inheritance or development of individual or familial predisposition.

In humans there are no diseases, conditions or susceptibilities transmitted from father to children whereby tooth resorption is potentially facilitated or prevalent. The causes of tooth resorption are local and necessarily affect the cementoblasts and epithelial rests of Malassez. In endocrine pathologies as well as in other systemic diseases, tooth resorptions are not part of the clinical or imaging manifestations.

Apex and root morphology – the crown-root ratio –

as well as the shape of the alveolar bone crest have a bearing on the predictability of tooth resorption in orthodontic treatment. If necessary, one might go as far as asserting that patients with tapering tooth roots, pipette-shaped or torn apices and rectangular bony ridges are more predisposed or susceptible to root resorption during orthodontic treatment, but such proclivity is of a morphological – not genetic or hereditary – nature.

On the treatment and prognosis of tooth resorptions

Therapy of inflammatory tooth resorptions entails primarily the elimination of causative factors. When the inflammatory process and cellular stress cease in the resorption area, with the bone remodeling units and their osteoclasts undergoing demobilization and leaving the root surface, mediators disappear. The pH of the region returns to neutral state and new cementoblasts are formed, recolonizing root surfaces in a few days. New cementum is then formed through the reattachment of collagen fibers at the center of the new layer of cementoblasts. The root surface once again becomes biologically normal.

If the cause is contamination by bacteria via root canal, appropriate endodontic treatment should eliminate the cause while the inflammatory resorption repairs itself. If the causative factor is an orthodontic force, the process is stopped by deactivating the orthodontic appliance or through force dissipation. When one eliminates the possible causative factor and still the inflammatory tooth resorption does not cease, this would imply that the real cause has not been eliminated.

Replacement resorption always follows alveolodental ankylosis and once established there is no way the process can be stopped. When ankylosis is detected before it has evolved into replacement resorption, luxation followed by extrusion can in most cases restore the periodontal ligament on the bridges or bone-tooth connection foci. But if replacement resorption occurs when part of the tooth has been resorbed and replaced by bone, physical overlapping will prevent a cleavage to occur between them.

In summary: Inflammatory tooth resorption can be controlled, cured and has a positive prognosis, but replacement resorption has a poor prognosis, because sooner or later tooth loss is bound to occur.

Tooth resorptions do not induce pain or necrotic pulp

As close as they may be to pulp tissue, neither inflammatory root resorption nor replacement resorption causes any pain. The number of mediators present in order for resorption of mineralized tissues to occur is not sufficient to induce pain and discomfort in the patient. If there is pain sensitivity in teeth undergoing resorption, some other cause must be sought to explain it: Tooth resorption is an asymptomatic, "silent" biological process.

Tooth resorption may be further compounded or associated with microbial contamination, occlusal trauma, and pulp and periapical pathologies that can be symptomatic, but tooth resorption is not a causative factor in any of these conditions.

The same mediators, phenomena and bone resorption cells are present in tooth resorption but they do not cause pain. In the human skeleton, between 1 and 3 million bone remodeling units are acting on and resorbing the skeleton continuously with no symptoms.

Although very close to the pulp – or even in cases where tooth resorptions occur within the structure of the pulp itself as in internal resorption – tooth resorptions do not induce necrosis of dental pulp tissue. The process of tooth resorption does not release toxic products into the cells. Resorption of mineralized tissues is only aimed at deconstructing these tissues in order to recycle their mineral and non-mineral components, which will be reused as ions, amino acids and peptides.

Tooth resorptions are clinically asymptomatic and of themselves do not induce pulp, periapical and periodontal changes, as they are – more often than not – consequences and not causes of the latter.

Final considerations: The concept of tooth resorption

Resorptions in the body as a whole are phenomena that can be present in various clinical situations and refer to a mechanism whereby mineralized tissues are structurally removed. At the interface between osteoclasts and odontogenic mineralized tissue there occurs a release of acids and enzymes, and the resulting molecules are transported through the cytoplasm into vacuoles by a process known as transcytosis and secreted into the extracellular space in the form of amino acids,

peptides and ions. In the extracellular matrix and in body fluids such as blood and lymph these components are reused by other organs, tissues and cells.

Tooth resorption is a process whereby mineralized odontogenic tissues are dismantled through the agency of bone cells located on their surfaces when the protective structures of the teeth in relation to bone remodeling are eliminated, especially cementoblasts and epithelial rests of Malassez. Resorptions consist of a pathological manifestation in permanent teeth and a physiological manifestation in primary teeth. In some clinical situations such as in orthodontic treatment tooth resorption is common and acceptable as long as anticipated and mitigated as part of the biological cost to have esthetically and functionally adequate teeth.

The mechanisms of tooth resorption are known and its causes well-defined. Tooth resorptions are clinically asymptomatic and of themselves do not induce pulp, periapical and periodontal changes, as they are – more often than not – consequences and not causes of these conditions. Tooth resorptions are local, acquired changes and do not reflect dental manifestations of systemic diseases.

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