The four mechanisms of dental resorption initiation

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The aim of this study is to present a classification with a clinical application for root resorption, so that diagnosis will be more objective and immediately linked to the source of the problem, leading the clinician to automatically develop the likely treatment plan with a precise prognosis. With this purpose, we suggest putting together all diagnosed dental resorptions into one of these four criteria:

1) Root resorption caused by cementoblast cell death, with preservation of the Malassez epithelial rests.
2) Root resorption by cementoblasts and Malassez epithelial rests death.
3) Dental resorption by odontoblasts cell death with preservation of pulp vitality.
4) Dental resorption by direct exposure of dentin to gingival connective tissue at the cementoenamel junction gaps.

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Dental resorptions are traditionally classified according with the mechanism of maintenance and evolution into:

a) Inflammatory.
b) By replacement.

Inflammatory dental resorptions are maintained by inflammatory mediators that stimulate BMUs — Bone Multicellular Units — where clastic cells gradually resorb the dentin surface free of cementoblasts and odontoblasts, eliminated as a consequence of the resorption process. The therapeutic principle of these dental resorptions is based on the identification and elimination of its cause, therefore, the resorption process will evolve to the repair phase. This is how we see inflammatory resorption related to orthodontic movement in each activation period.

Dental resorptions by replacement are maintained by systemic and local mediators of bone tissue which regulate the remodelling process or turnover. This resorption occurs always as a consequence of alveolodental ankylosis because of the death of Malassez epithelial rest cells — induced by dental trauma, especially by daily concussions. Since there is no way to eliminate the local mediators for bone turnover, the prognosis of dental resorption by replacement almost always involves tooth loss. It is important to highlight that orthodontic movement and occlusal trauma does not induce Malassez epithelial rests death.

To facilitate the clinical and etiological understanding of root resorptions, it was proposed a classification for each case, using as criteria its mechanism of induction and initiation of the process. Figures 1 and 2 illustrate and explain it.

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Using this classification in each clinical case allows a direct and precise diagnosis, immediately linked with its cause, leading to an automatic reasoning of the likely treatment plan with an accurate prognosis.

Root resorptions are grouped as follows:

1. Root resorption by cementoblast cell death with maintenance of Malassez epithelial rests.
   - Inflammatory root resorptions during orthodontic movement.
   - Apical Inflammatory root resorption in chronic periapical lesions.
2. Root resorptions by cementoblast and Malassez epithelial rests death.
   - Resorption by replacement in dental trauma.
   - Resorption by replacement in periodontal ligament atrophy of unerupted teeth – especially canines.
3. Root Resorption by odontoblast cell death with maintenance of pulp vitality
   - Internal Inflammatory root resorption by dental trauma.

4. Root resorption by direct exposure of dentin to the gingival connective tissue at the cementoenamel junction gaps
   - External cervical inflammatory resorption by accidental trauma, especially concussion.
   - External cervical inflammatory resorption by trans-operative dental trauma as in impacted canine traction and during intubation in general anesthetic procedures.
   - External cervical inflammatory resorption in association with internal whitening procedures.

FINAL CONSIDERATIONS
The application of the proposed classification for dental resorption to every clinical case will help the development of a direct diagnosis promptly linked with its cause. This will lead to a treatment plan with a precise prognosis.

REFERENCES