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The mechanisms of tooth eruption

Abstract

Tooth eruption is a complex multifactorial process. Association of alveolar bone remodeling, root elongation, cementum apposition, and periodontal ligament formation include a series of events. The objective of this review includes the characterization of an initial pre-eruptive phase, pre-occlusal and post-occlusal eruption, then, the tooth becomes functional. Gubernacular cord and canal are involved in the tooth eruption process. This is including vascular pressure, maturation of the periodontal ligament, changes in the alveolar bone, collagen fibers maturation, and cementum formation. Transcription factors and growth factors are integral parts of these multifactorial events. Hormones such as of IL-1 and PTHrP are located in the stratum reticulum (SR). Metalloproteinases suggest that tooth eruption is also implicated in these multifactorial processes. Genesis of osteoclasts and osteoblasts, tooth eruption molecules, biological aspects of the periodontal ligament are involved in this process. Up to now, a single process for tooth eruption has not been identified, but complex effects combine that shed lights on the different mechanisms involved in tooth eruption.

Keywords: alveolar bone, pre-eruptive tooth, eruption, periodontal ligament, cementum apposition, stellated stratum, gubernaculum, transcription and growth factors, osteoblasts, osteoclasts, metalloproteinases

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Introduction

Tooth eruption is a continuous multifactorial process, associated with alveolar bone remodeling, root elongation, cementum apposition and periodontal ligament formation.

There is no consensuses in the mechanisms involved, but include five successive stages:

Preeruptive movement

Intra-osseus

Pre-and post- occlusal eruption[1]

Other events may also be implicated: such as pulpal pressure, pulpal growth, root lengthening, traction by periodontal fibroblasts acellular and cellular cementum formation, and vascular pressure.

Developmental anomalies of tooth are recognized, including flexion, dilacerations, concrescence, fusion, dwarted roots, accessory roots, hypercementosis, and enamel pearl (Figure 1).[2]

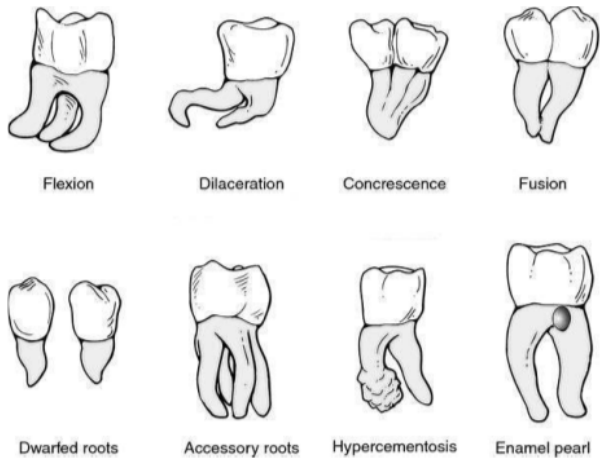

Figure 1 Developmental anomalies of the roots.[2]

First, the crown part of the tooth is formed. Then, from the cervical region (labial or lingual parts) starts the formation of a tongue of epithelial cells that contributes to the division of the cervical space into two (for mandibular molars). Figure 2 or three divisions of the cervical space[3] and Figure 3 are these events are leading to the formation of furcation dentin and to the onset of root formation.[1]

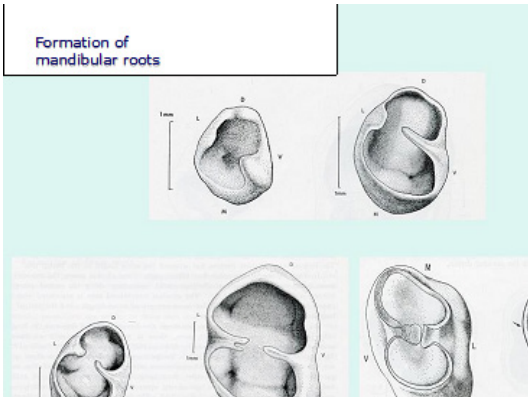

Figure 2 or three divisions of the cervical space.[3]

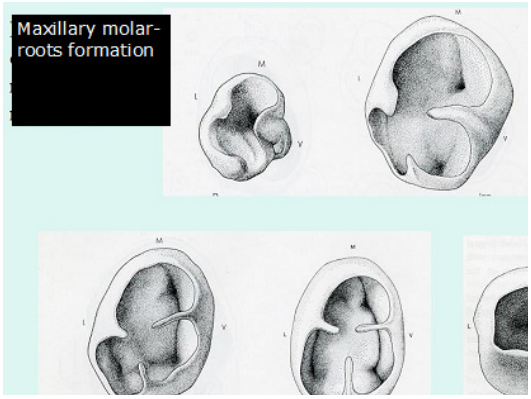

Figure 3 Formation of a maxillary molar (three roots).

Tooth eruption implies 3 different phases

Pre-eruptive phase, also named primary eruption. Pre-eruptive tooth movement implies that the second molar is moving backward, whereas anterior teeth are moving forward.[1] (Figure 4)

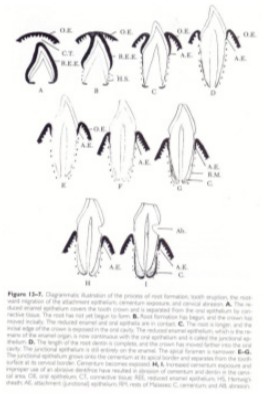

Figure 4 Tooth eruption implies 3 different phases.[2]

Eruptive phase: Two successive stages are reported**:**

An intra-osseous stage, with a parallel formation of roots, together with alveolar bone,

An extra-osseous compartment, these events include 4 stages: root formation, movement, penetration in the oral cavity, ending by occlusal contact.

Post-eruptive phase:

That maintain the jaws continue to grow, and compensate for occlusal and proximal wear.

Two sets occur in human (diphiodont): (deciduous vs permanent dentition). At some moment: the two dentitions are present (occurrence of a mixed dentition)

The formation of the furcation dentin, followed by the merge and occurrence of contact between lower and upper molars. This signify the end of the tooth eruption[4] (Figure 5).

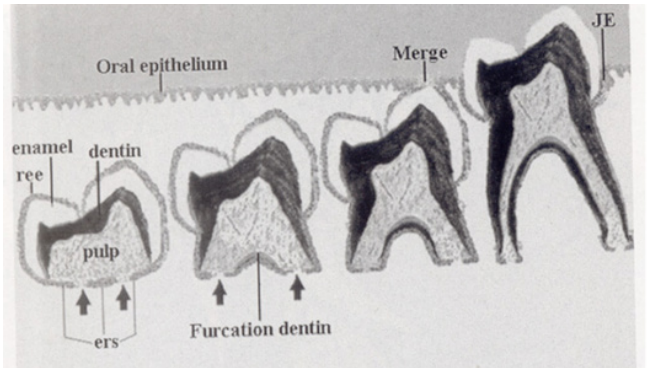

Figure 5 The formation of the furcation dentin.

Four erupting tooth movements are recognized. They are due to:

a) root formation

b) incisally or occlusally movements

c) movement of the crown

Gubernacular cord and canal are involved in tooth eruption: vascular pressure, periodontal ligament maturation, changes at the alveolar bone level. Widening of the gubernacular canal allows the tooth to erupt. The rate of tooth eruption depends on two different phases:

Intraosseous phase: Rate 1-10 µm/day

Extraosseous phase: Rate 75 µm/day

Mechanisms of eruptive movement involve a multifactorial process due to:

Root formation: Tongues formed from the internal and external border (the 4 layers, are reduced to two layers forming the Nasmith's cuticule which are at the origin of alveolar bone, periodontal ligament and cementum. The formation of apical foramen, involved in growth and development toward the eruptive zone is an important step during these phenomenons (Figures 6 & 7).[5]

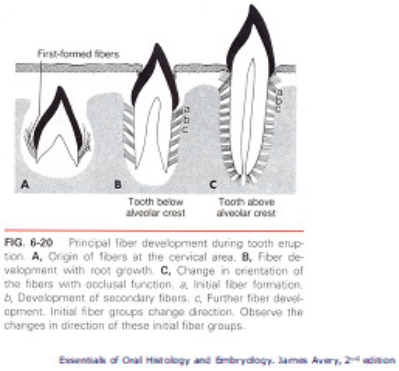

Figure 6 The formation of apical foramen.

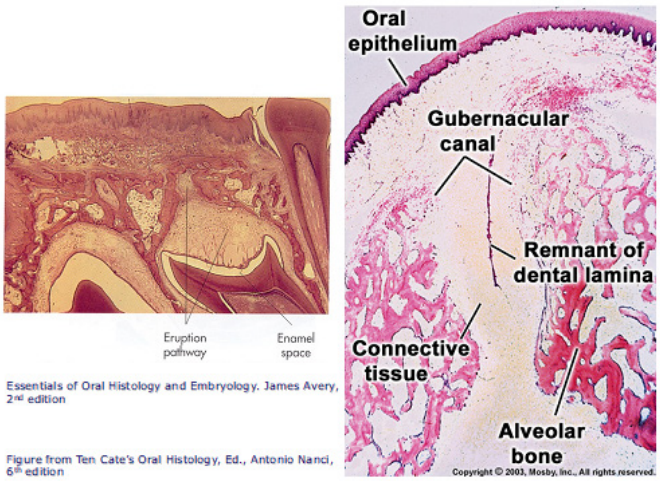

Figure 7 Eruption pathway and gubernacular canal facilitating tooth eruption in the oral cavity.[6]

Bone remodeling

Defects in osteoclast differentiation

Periodontal ligament (PDL) is present, but the tooth is not erupting, however, it is obvious that rootless teeth are erupting, despite the lack of root formation (Figures 8 & 9).[6]

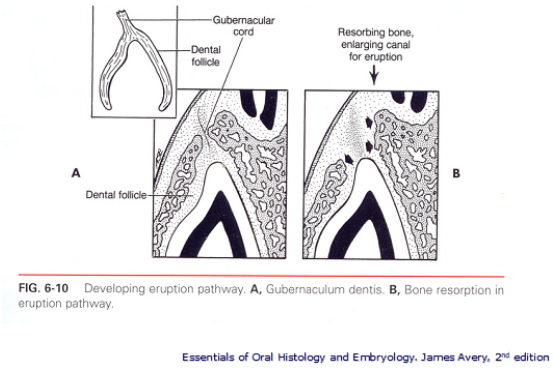

Figure 8 Periodontal ligament includes five different groups of fibers present in multi-rooted teeth.[9]

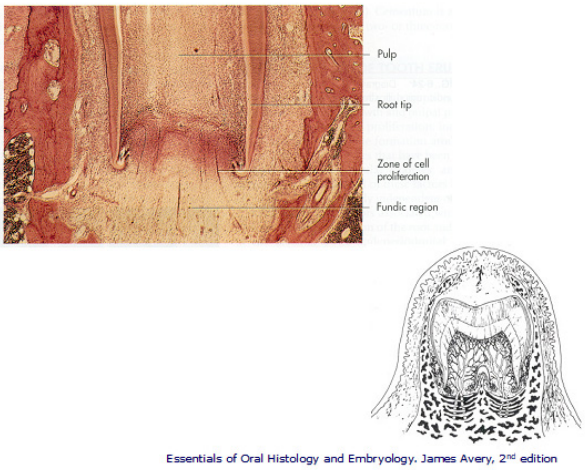

Figure 9 Epithelial hammock and apical zone of proliferation.[10]

Alveolar crest, horizontal fibers, oblique fibers, apical fibers, interradicular fibers

In single rooted teeth, groups of fibers are forming:

Dento-gingival, alveolo-gingival, circular and dento-periosteal groups

Lately, molars are sliding mesially.

Control factors of the mesial drift due to:

a) Contraction of the transseptal fibers

b) Adaptability of bone tissue

c) Occlusal forces: forward directed forces generated from intercuspal forces.

d) Pressure from tongue is pushing teeth mesially.

Mechanisms implicated in tooth eruption

Active eruption: compensates lengthening of clinical crown

Passive eruption:_recession of the gingiva and of the underlying alveolar bone (increased lengthening of the clinical al crown)Four events are implicated in tooth eruption.

Root formation: The crown migrates in the bone. The fibrous hammock contribute to tooth eruption. (Figure 10)[7] Implication of contraction of the transseptal fibers, adaptability of bone tissue, and occlusal forces generating intercuspal forces**.** Future apical foramen facilitates eruption.4 eruption and bone resorption, vascular pressure, Maturation/Migration fibroblasts, contraction of collagen gels; Maturation of the alveolo dental ligament, from the native collagen to mature collagen fibers (1000 µm of the native collagen, shortening to 670 µm in length of the mature collagen, after enzymatic ablation of the N- and C- termini).

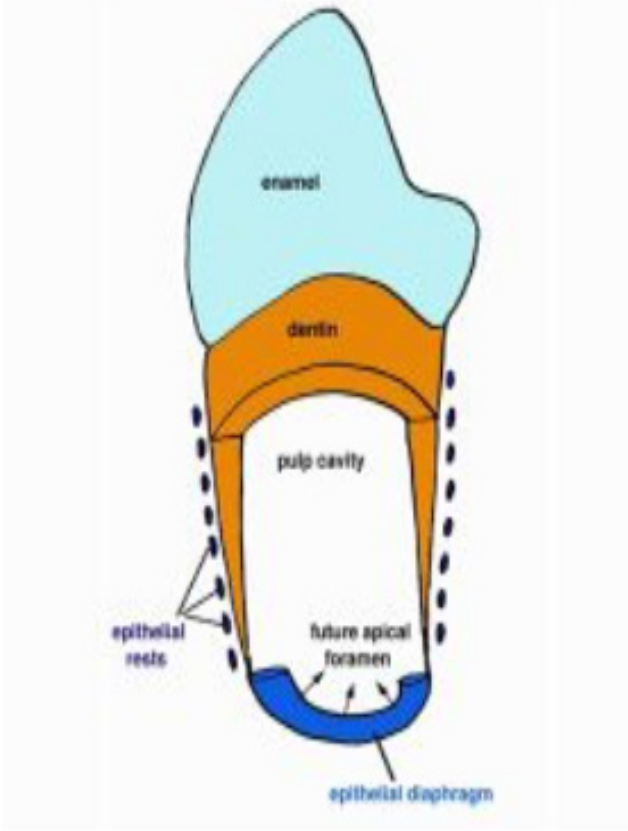

Figure 10 Implication of contraction of the transseptal fibers.

Changes in alveolar bone:

First, compression sites

Second, resorption at compression sites

Many theories the mechanisms of tooth eruption have been reported, such as root elongation, pulp cell proliferation, bone deposition, and tissue fluid pressure. It was also suggested that the force of eruption might come from the PDL.[6]

The mechanisms involved in tooth eruption are numerous, including:

Dental follicle in also implicated in tooth eruption. The follicle plays a lesser role. According to Wise and Lin, Cahill & Marks[7,8] ablation or removal of dental follicle may influence or coordinate the process of tooth eruption. There is evidence that dental follicles play an important role in this cascade because they produce several factors including tumour necrosis factor-a (TNF-a), transforming growth factor-b (TGF-b), interleukin-1(IL-1), colony-stimulating factor 1

(CSF-1) and receptor activator of nuclear factor kB ligand (RANKL). TNF-α, CSF-1 and RANKL are released by dental follicle cells and stimulate the migration and differentiation of bone-marrow precursors or mononuclear cells into multinucleated osteoclasts

Periodontal ligament

From a biomaterials perspective, the periodontal ligament is a complex, fiber-reinforced substance that responds to force in a viscoelastic and non-linear manner.

Adjacent alveolar bone: A bone remodeling cycle has four phases: activation, resorption, reversal, and formation Inhibition of the molecules that promote osteoclastogenesis would serve to prevent and inhibit eruption.

Numerous reports indicated as key osteoclastogenic molecules, such as RANKL, osteoprotegerin, and VEGF are expressed in the periodontal ligament. The osteoprotegerin is secreted *in vitro* by the periodontal ligament fibroblasts and can inhibit osteoclast formation. During a period of tooth eruption, only the expression osteoprotegerin would need to be inhibited and bone resorption is occurring.

All these modifications are influenced by genes that expressed molecules such as:

Transcription factors: Runx2/Cbfa 1

Growth factors: insulin-like GF-1

Vascular endothelial GF (VEGF)

Tumor necrosis factor-alpha (TNF-alpha >>>stimulation of the monocyte 1 protein chemotactin and VEGF

Hormones: Growth Factor receptors and parathyroid related protein (PTHrP). Indeed, the parathyroid hormone–related protein (PTHrP) and its parathyroid hormone/PTHrP receptor PPR signaling appear to crosstalk with other signaling pathways.

Enzymes (Protein-kinase C; PKA)

Metallo Proteinases: MMP-8 and MMP-13 and Disintegrins (PGs are cleaved: e.g. versican is cleaved by ADAMTS 1, ADAMTS 4, ADAMTS 5)[9]

Potential role of MT1-MMP:

The MT1-MMP activity in the dental mesenchyme, are essential for proper tooth root formation and eruption. These studies point to an indispensable role for MT1-MMP-mediated matrix remodeling in tooth eruption through effects on bone formation, soft tissue remodeling and organization of the follicle/PDL region.

MT1-MMP, a transmembrane zinc-endopeptidase that breaks down extracellular matrix components, indicates that MT1-MMP activity in the dental mesenchyme is essential for proper tooth root formation and eruption. These studies point to a role for MT1-MMP-mediated matrix remodeling in tooth eruption through effects on bone formation, soft tissue remodeling and organization of the follicle/PDL region.[10]

In addition, resorption of deciduous teeth (by dentinoclasts) is associated to the eruption of permanent teeth

Bone growth: Marks and Cahill[7] interpreted their experiments, with the eruption of metal and silicone replica, providing evidence for alveolar bone growth (apposition and resorption) passively 'carrying' the tooth through bone into function.

The first evidence of eruption is bone resorption beneath the calcified crown, in a direction opposite to the one in which the tooth is expected to move. Such cellular activity is not unexpected if a significant 'origin' of force within the follicle becomes displaced apically subsequent to calcification of the crown. The mechanics of tooth eruption lead to the conclusion that forces from the apical vasculature are the likely source of the eruptive force. As tooth eruption can be explained by the action of forces acting in a dynamic relationship with bone remodeling, many factors modify the rate and direction of the process.

Role of dental follicle: Cellular events – Molecules involved in tooth eruption:

Dental Follicle-95 is selectively degraded at the onset of tooth eruption.[6]

E**ruption molecules are implicated in tooth movements**: EGF, TGF-α and EGF utilize the same receptor. The Colony-stimulating factor-1 (CSF-1) is also contributing to tooth migration. The presence of IL-1 and PTHrP in the stratum reticulum (SR) suggests that the SR portion of the enamel organ play such a role, DF-95 is present at the onset of eruption and then decline. CSF-1 is expressed in the SR, and has a role in eruption. The cells are regulated by autocrine signaling by parathyroid hormone–related protein (PTHrP) and its parathyroid hormone/PTHrP receptor PPR. This PTHrP-PPR signaling appears to crosstalk with other signaling pathways and regulates proper cell fates of mesenchymal progenitor cell populations. Disruption of this autocrine PTHrP-PPR signaling in these cells leads to defective formation of the periodontal attachment apparatus, tooth root malformation, and failure of tooth eruption in molars, a rare genetic disorder exclusively affecting tooth eruption. Analyses at different stages of premolar eruption indicate that selective fragmentation of dental follicle protein DF-95 correlates with the presence of elevated levels of follicular collagenase and stromelysin.[11]

Mononuclear cells (osteoclast precursors) must be recruited into the dental follicle prior to the onset of eruption. These cells fuse to form osteoclasts that resorb alveolar bone, forming an eruption pathway for the tooth to exit its bony crypt.

Recruitment of the mononuclear cells to the follicle may require colony-stimulating factor-one (CSF-1) and/or monocyte chemotactic protein-1 (MCP-1). Osteoclastogenesis is needed for the bone resorption and enhancement of receptor activator of NFκB ligand (RANKL), in the adjacent alveolar bone and/or in the follicle. Paracrine signaling by parathyroid-hormone-related protein and interleukin -1α, produced in the stellate reticulu, may also play a role in regulating eruption. Osteoblasts might also influence the process of eruption, the most important physiologic role likely being at the eruptive site, in the formation of osteoclasts through signaling *via* the RANKL/OPG pathway. Evidence supports a role for an osteoblast-specific transcription factor, Cbfa1 (Runx2), in molecular events that regulate tooth eruption. Cbfa1 is also expressed at high levels by the dental follicle cells.[5]

Putative tooth eruption molecules:

EGF, EGF-R CSF-1, CSF-R, IL -1α, IL-1R, c-Fos, NF_kB

MCP-1, TGF-β1, PTHrP, Cbfa1, OPG/OCIF, RANKL

Fibroblasts may play a possible role in tooth eruption. Interstitial fluid pressure may also be related to tooth eruption

Mononuclear CSF-1 and MCP-1 are candidates for recruiting osteoclast precursors. Osteoblasts differentiation Osf2 or Cbfa1

(Core-binding factor a1) is a key transcriptional regulator of osteoblast differentiation during bone formation.

Primary Failure of Eruption (PFE): Eruption results from interactions between cells of the dental enamel organ, the follicle, and alveolar bone. (Figures 11 & 12)[12]

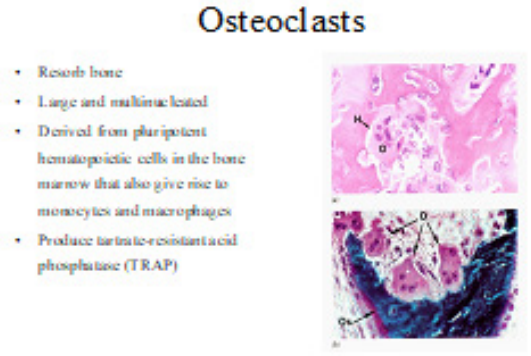

Figure 11 Multinucleated osteoclastic cells.

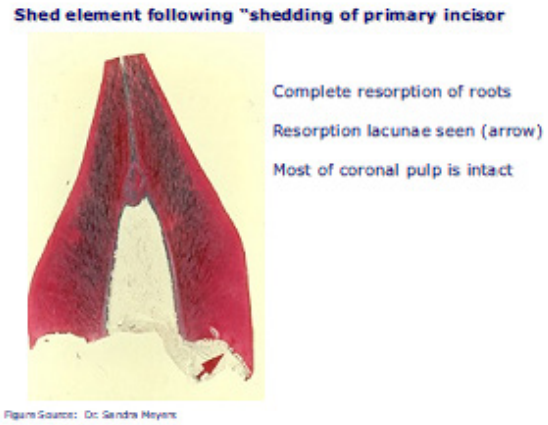

Figure 12 Shedding of primary incisor, interactions between cells of the dental enamel organ, the follicle and bonny alveolar.

Conclusion

Tooth eruption is a multifactorial event. Collagen maturation constitutes the basis for periodontal ligament maturation. Ablation of the collagen C and N termini leads to a reduced length of fibers firstly synthetized as pro-collagen (1000 µm), then reduced to a 670 Å periodicity (mature collagen). This shrinkage may contribute to the periodontal ligament maturation and consequently to tooth eruption. Changes were also identified in hormones (PTHrP), growth and transcription factors (TNF -a, -b, CSF-1, RANKL) metalloproteinases (MMP-8, 13, disintegrins, MT1-MMPs), enzymes expression of the surface of the bonny socket, all being expressed inside the ligament and in cementum (acellular and cellular enzymes expression at the surface of the bonny socket). The expression of proteases in the stellate reticulum (SR) seems also to be crucial. Membrane localization of Baz, Par6-a PKC and Crb depend on Baz. Cdc-42 drives morphogenesis by conferring apical identity of Par6. (Nunes de Almeida et al., 2019). The apical STEM cells contribute also to the mechanisms of tooth eruption. The process is not limited to root lengthening, but involves also some other factors.

Acknowledgments

None

Conflicts of interest

The authors have no conflict of interests.

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