



Alberto Consolaro^{1,2}, Mauricio de Almeida Cardoso³

¹ Universidade de São Paulo, Faculdade de Odontologia de Bauru (Bauru/SP, Brazil).

² Universidade de São Paulo, Faculdade de Odontologia de Ribeirão Preto, Programa de Pós-graduação em Odontopediatria (Ribeirão Preto/SP, Brazil).

³ Faculdade de Medicina e Odontologia São Leopoldo Mandic, Programa de Pós-graduação em Ortodontia (Campinas/SP, Brazil)

Crowing ages the mouth: is it normal or pathological? Teeth do not contact or push each other

20

Abstract: Teeth neither have contact nor touch their mineralized structure with another mineralized structure! A tooth does not push another tooth by contact. A tooth does not directly resorb another tooth: there will always be soft tissue interposition, even with the presence of a gap or interface between teeth of which imaging and tomographic examination suggests direct contact between two teeth. Crowding resulting

from an imbalance among factors that keep the dental arch stable might be taken as a natural aging-related process or as a pathological condition. Such conceptual difference regarding its nature needs further elucidation by evidence not yet available. Even in the event of crowding, with teeth being as near each other as possible, periodontal space is kept by function exerted by epithelial rests of Malassez releasing

EGF. When caused by the fact of teeth being near one another, resorption results from biological phenomena related to the presence of pericoronal follicle between teeth. Concrescence of teeth does not result only from excess proximity between roots, but by epithelial rests of Malassez death caused by trauma. **Keywords:** Tooth crowding. Tooth resorption. Unerupted teeth. Concrescence of teeth.

How to cite: Consolaro A, Cardoso MA. Crowding ages the mouth: is it normal or pathological? Teeth do not contact or push each other. J Clin Dent Res. 2018 Jan-Mar;15(1):20-8.

DOI: <https://doi.org/10.14436/2447-911x.15.1-020-028.bes>

Submitted: February 21, 2018 - **Revised and accepted:** February 26, 2018.

Tooth crowding in the anteroinferior region is explained by many as the result of direct pressure exerted by posterior teeth, especially third molars, over teeth in front of them in the dental arch. This explanation nearly resembles the classic cascade falling successively or the so-called domino effect of domino tiles falling in chain reaction. The present study aims at clarifying how this actually happens in pericoronal, periodontal and bone tissues.²

According to one's imagination, mandibular third molar "pushes" the second molar; subsequently, second molar "pushes" the first molar, and thus successively until force produced at the site is opposed, in the midline, to force produced

on the other side of the dental arch. Third molars involved in the situation are normally partially or completely unerupted, with some degree of second molar impaction.

Whenever an unerupted tooth is near another one, they neither touch nor have their mineralized structure in contact. This is not found either between permanent teeth or between permanent and deciduous teeth¹ (Fig 1). A tooth does not push – whether physically, mechanically or directly – another tooth. Before this even happens, phenomena leading to root resorption occur at the soft tissue found between both teeth.

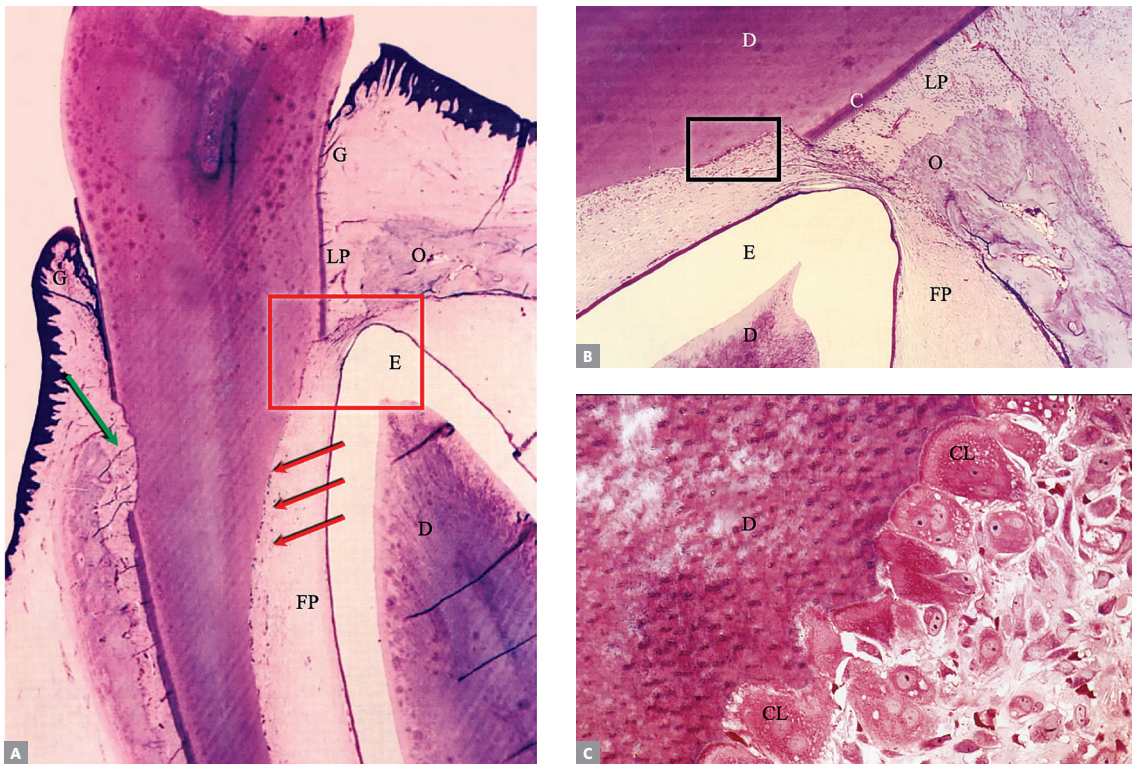


Figure 1: Mineralized structures of teeth do not touch: despite the presence of small spaces in between teeth, note connective tissue with numerous clasts promoting resorption, as shown by the photograph. Resorption of deciduous tooth was sped up by the permanent tooth pericoronal follicle mediators on the lingual surface (red arrows). On the buccal surface of the deciduous tooth, resorption is slower and less associated with death of cementoblasts. It is not sped up by permanent tooth pericoronal follicle mediators (green arrow) (A=16X; B=40X; C=400X). E = enamel; D = dentin; C = cementum; G = gingiva; PL: periodontal ligament; B = alveolar bone; LP = pericoronal follicle; CL = clasts (source: Reviere,⁷ 2000).

Despite radiographic and/or tomographic imaging examination strongly suggesting that a tooth touches another one, this image is not real (Figs 2 and 3). Should microscopic examination be carried out in those situations, fibrous connective tissue cells and strips full of clasts will always be revealed between teeth.⁷ Those clasts are occasionally known as osteoclasts, cementoclasts, dentinoclasts or even odontoclasts.

In the interface between soft tissue and neighboring tooth clasts, there are the Howship lacunae and other cells associated with resorp-

tive function, such as osteoblasts and macrophages. Those sets of “clasts-osteoblasts-macrophages” are known as bone modeling units.¹

Cells resorbing mineralized tissues, including cartilage, can merely be referred to as clasts, as they represent the same cell of the same macrophage lineage coming from the bone marrow. Clasts might have only one nucleus, but they are usually multinucleated, with an average of 10 to 15 nuclei^{3,4} (Fig 4). Morphologically, as revealed by a light microscope, clasts can be easily identified; however, researchers use immunocyto-

22

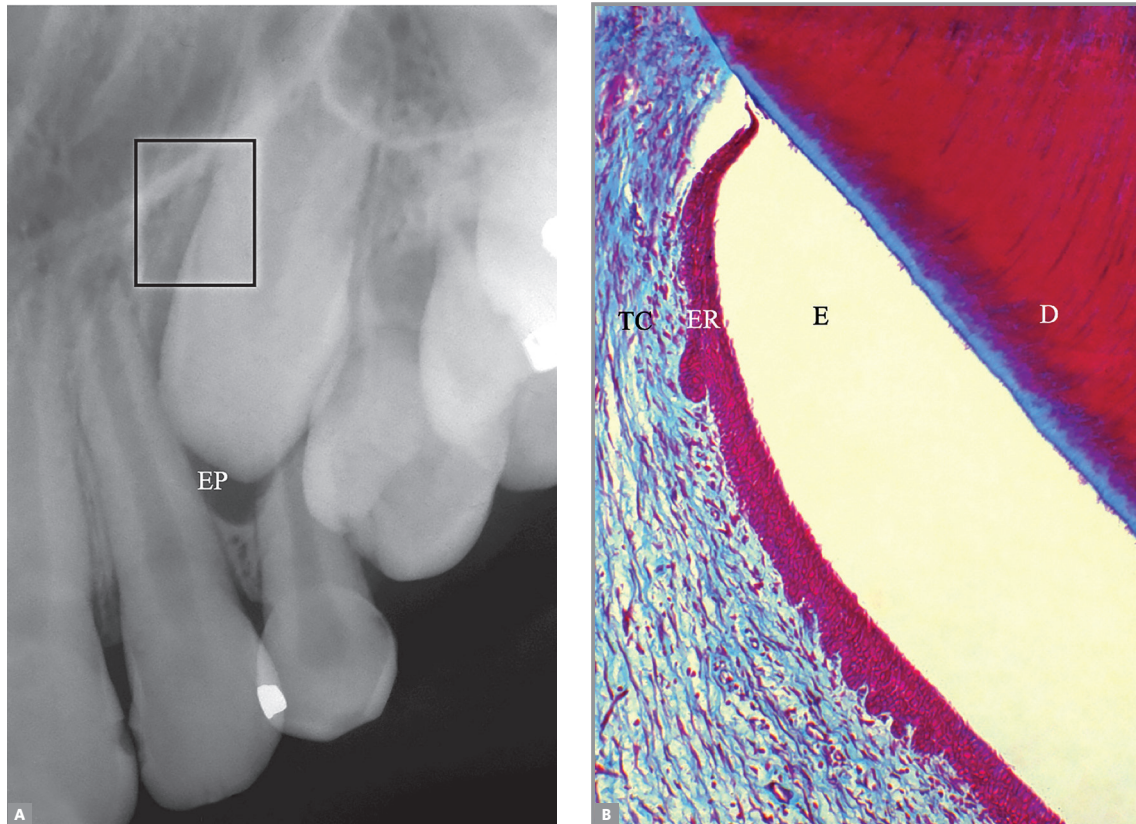


Figure 2: Maxillary canine only “apparently” touches the lateral incisor. Imaging examination taken with different X-ray beam angles as well as tomographic scans will reveal a gap between crown and root. Every crown is coated by follicle found in the pericoronal space as revealed by imaging examination. In **B**, note reduced enamel epithelium adhered to the enamel and supported by fibrous connective tissue. This is what comprises most part of the follicular structure. E = enamel; D = dentin; RE = reduced enamel epithelium; CT = connective tissue; PS = pericoronal space. (TM, 100X).

chemical markers with the very aim of including, in counting and morphometrics, clasts having only one nucleus or those not meeting morphological patterns, due to cut planes of examined tissues.

In transmission electron microscopy, mono- or multi clasts have the same structures that are necessary to exert resorptive function^{3,4} (Fig 4). The following are worth highlighting: 1) fringed active edge in the interface with underlying mineralized tissue; 2) sealing provided by interaction between membrane proteins and proteins

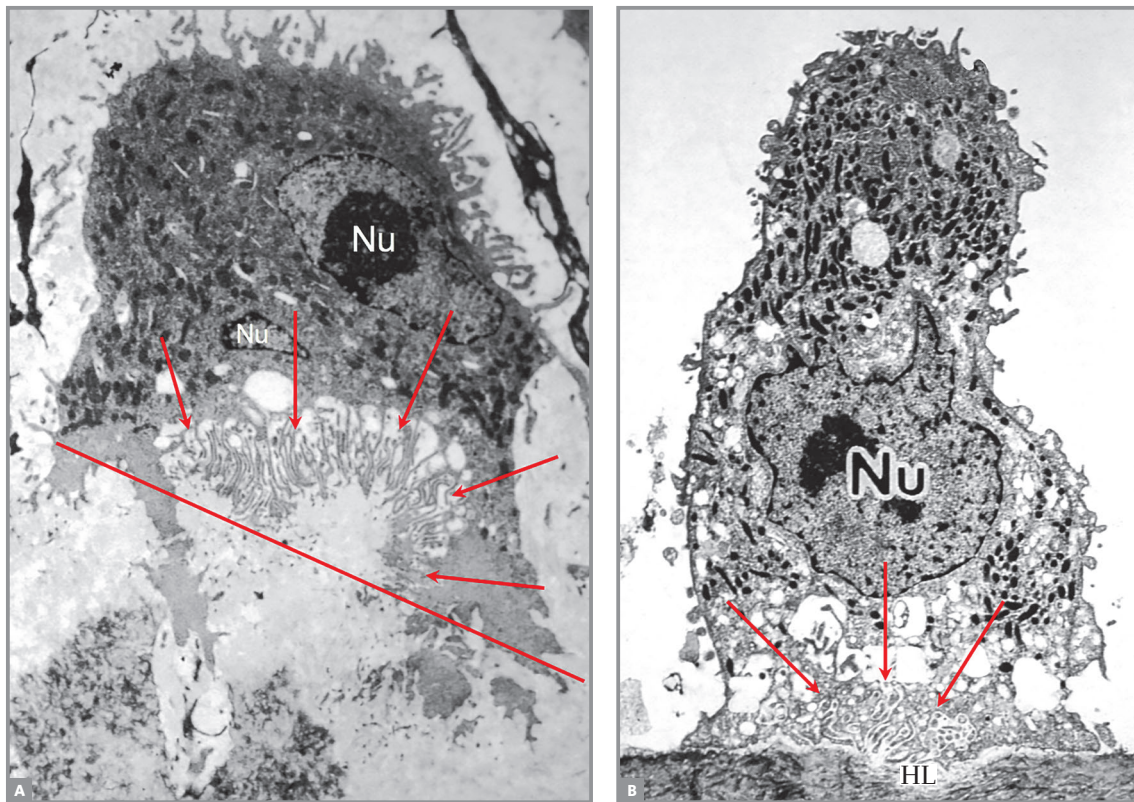
from mineralized matrix aiming at setting the limit of action taken by acids and enzymes released at site.

IN BETWEEN A PARTIALLY OR COMPLETELY UNERUPTED TOOTH AND AN ERUPTED TOOTH “APPARENTLY” TOUCHING WHAT GOES ON WITHIN SUCH SPACE?

On unerupted tooth crown surface reduced epithelium is adhered to enamel (Fig 2). In fibrous connective tissue providing support to the ep-



Figure 3: Tomographic scans obtained in slices or 3D reconstructions allow for clear view of a gap between mineralized structures of teeth, although some slices or reconstruction angles might suggest contact between them, as in **A**. In **B**, transverse slice reveals induced resorption in lateral incisors (arrow), but with not contact between teeth. Teeth do not cause resorption of other teeth, but clasts aligned in fibrous connective tissue found between them do.



24

Figure 4: Multinucleated clast (A) and mononucleated clast (B). Arrows signal the fringed active edge in both photographs. The red line is the surface of interface with mineralized tissue. Slices of mononucleated clasts were obtained in a sequence and included the entire cell, thus revealing the single nucleus. Howship lacunae (HL) reveals resorptive activity with the active edge. Nu = nucleus. (Transmission electron microscopy, Source: Domon et al³).

ithelium, there are dental lamina clusters of epithelial cells. Those epithelial components promote EGF, or epidermal growth factor, production at site. EGF is a mediator that not only stimulates the activity of clasts, but also interacts with other local mediators with the same purpose.

Reduced enamel epithelium and the underlying fibrous connective tissue, with clusters of epithelial cells, form pericoronal follicle which is

the major structure responsible for tooth eruption. Unerupted teeth move towards the occlusal plane thanks to pericoronal bone resorption made possible by the pericoronal follicle.

Experimental pericoronal follicle removal leads to absence of tooth eruption. All other dental structures can take part in the eruption process; however, they are not essential, since their experimental removal does not affect the process.

An unerupted tooth, with its forces (also known as vectors) going on in one direction, compresses the pericoronal follicle against a neighboring tooth root (Fig 2). Such compression gradually decreases and reduces or eliminates light from periodontal blood vessels, thus promoting death of cementoblasts at the site. Those cementoblasts covered the cementum and prevented clasts from lining up on the root mineralized surface.

Without cementoblasts elimination, it is impossible to induce root resorption, not even by bringing the pericoronal follicle near, as previously suggested.⁵ Death of cementoblasts is necessary, and pericoronal follicle alone does not lead to necrosis or elimination of cementoblasts at the site. The only factor leading to their elimination is compression of periodontal ligament vessels causes by eruption-related forces or vectors.

Death of cementoblasts leads the underlying root mineralized structure to attract and, as a result, become colonized by clasts that attach to it and trigger resorptive activity. The clasts are stimulated mainly by EGF and other follicle mediators, such as TGF-beta, CSF-1 and interleukin-1, which induce the formation of clasts and recruitment of precursors.

Normal periodontal space width ranges from 0.20 to 0.40mm, which corresponds to 200 to 400µm or 20 to 40% of a millimeter. One clast might have its longest axis measuring up to 0.06mm, which corresponds to 60µm or 6% of a millimeter.⁷

It is possible to claim that those spaces are reduced and hardly identified by radiographic and tomographic imaging examinations (Figs 2 and 3). Being aware of what goes on in the interface between two teeth does not allow – neither by analogy nor reference – for diagnosis or description in reports, feedbacks,

and articles,⁶ as if there was direct physical or imaging contact between them, as it often is the case!

WHAT SHOULD BE DONE IN CASES OF A PARTIALLY OR COMPLETELY UNERUPTED TOOTH THAT “APPARENTLY TOUCHES” AN ERUPTED TOOTH?

In order to end an ongoing resorptive process, displacing or moving the unerupted away while re-directing the course of tooth eruption is enough. Pericoronal follicle displacement allows for the following:

1. Relief of pressure produced on periodontal ligament vessels, with the latter having blood circulation recovered.
2. Resorption mediators molecules detachment. As a result, they are no longer able to reach the root surface to stimulate the clasts.

In this new environment set up in the interface between teeth, within hours or days, clasts are detached from the root surface and neighboring cementoblasts proliferate and cover the mineralized root surface, thus forming new cementum with reinsertion of periodontal fibers. As a result, normality of periodontal physiology is restored in this region.

HOW ABOUT DECIDUOUS TEETH? DO THEY TOUCH PERMANENT ONES?

Up to this date, many clinicians have shared the following assumption: a permanent tooth touching a deciduous one is somewhat part of the root resorption process.

The fact of permanent teeth being near deciduous ones allows for pericoronal follicle and resorption mediators to be near. The follicle mediators favor clasts placement on deciduous teeth root surface in many areas that are exposed due to apoptosis of deciduous teeth cementoblasts¹ (Fig 1).

Apoptosis is a slow and random process that occurs in all deciduous teeth surfaces as soon as formation is complete. Additionally, apoptosis is a genetically controlled process in our species and aims at eliminating cells that no longer are of any interest to the organism at several sites in one's body.¹

**IN-BETWEEN ERUPTED TEETH THAT
“APPARENTLY” TOUCH:
WHAT GOES ON WITHIN SUCH SPACE?
IS CROWDING INEVITABLE?**

Teeth remain extremely near one another for many years, and periodontal structures remain separate and interdependent (Fig 5). Teeth are stable in terms of position, even if the supporting bone is undergoing continuous mutation resulting from bone remodeling. Such balance occurs within a dynamic system of forces and functions, and it is typical of the tensegrity phenomenon.

Stable tooth position or tensegrity results from balance among:

1. soft tissues forces on the lingual side;
2. soft tissues forces on the buccal side;
3. teeth on the mesial side;
4. teeth distally positioned; and
5. occlusal forces of opposing teeth.

The mandible undergoes a remodeling process also associated with forces resulting from cartilaginous activity and adaptations TMJ condyles go through. The result of those forces could be referred to as vector. From a conceptual point of view, one could claim that there is “residual and ongoing mandibular growth” throughout one's life. Loss of vertical dimension, tooth wear and other process undergone by the bone and soft tissue lead to continuous adaptation.

Residual mandibular growth tends to move teeth forward towards the midline. Should there not be perfect balance among the five major sources of force produced over the tooth in

the dental arch, more simple, delicate and single-rooted teeth, such as mandibular incisors, tend to undergo malalignment and crowding over time. Lateral rotation and rotation can also occur over time.

Should tooth crowding occur in teeth of which position has never changed as a result of orthodontic treatment, what we have is an esthetical sign of aging, since this should be a natural process in the elderly only. The aforementioned also applies to teeth subjected to orthodontic movement: they might undergo crowding as well!

In both cases, with or without history of orthodontic treatment, crowding represents loss of balance among the five major factors responsible for preserving tooth position stability in the dental arch. That is, it represents loss of tensegrity. Not all of those who have complete dental arches and age above 60 years old present tooth crowding. In other words, tooth crowding is not inevitable.

Some studies in Orthodontics believe crowding is inevitable and inherent to aging, i.e., it is normal. They advocate patients continuously use retainers from canine to canine in the lower arch after the end of treatment with a view to preventing crowding over time.

Nevertheless, many other orthodontists believe crowding to be loss of balance in the normal position of teeth in the dental arch. This represents a disease or an abnormal or pathological change. Those clinicians do not advocate unrestricted use of retainers, except for very particular situations.

Although vectors of forces resulting from residual mandibular growth tend to move teeth towards the midline region, teeth do not touch one another, even in the event of crowding. Mineralized structures of teeth do not meet. And despite being extremely near one another, roots do not undergo resorption nor ankylosis, let alone concrescence of teeth. Why would that not be the case?



Figure 5: Mandibular tooth crowding in a young adult patient.

Periodontal space is where periodontal ligament is found. The former is preserved due to ongoing release of EGF, or Epidermal Growth Factor, by epithelial rests of Malassez. The peptide, which is released around this basketball-net-like epithelial network surrounding the root, permeates and “moistens” the periodontal ligament. EGF function in the periodontal ligament is to stimulate bone resorption in the periodontal surface of the socket, in the event of deposition of bundle bone new layers being too near the tooth. Thus, periodontal space always remains with thickness ranging between 0.2 and 0.4 mm.¹

This explains why alveolodental ankylosis does not occur with aging, even after decades or significant closeness between alveolar bone and tooth root. Even in cases of crowding with teeth being dangerously too near one another, resorption and alveolodental ankylosis do not occur: thanks to epithelial rests of Malassez.¹

ence regarding its nature needs further elucidation by evidence not yet available in the literature. Even in the event of crowding, with teeth being as near each other as possible, periodontal space is preserved by function exerted by epithelial rests of Malassez releasing EGF.

A tooth does not directly push nor touch another tooth. When caused by the fact of having teeth near each other, resorption results from biological phenomena, such as the presence of pericoronal follicle between teeth. Concrescence of teeth does not result only from excess proximity between roots, but also by epithelial rests of Malassez death or atrophy caused by trauma.

28

FINAL CONSIDERATIONS

Teeth neither have contact nor touch their mineralized structure with another mineralized structure! In short, a tooth does not push others as a result of contact. A tooth does directly resorb another tooth: there will always be soft tissue interposition. Soft tissues are represented by fibrous connective tissue full of clasts as well as other cells associated with resorptive processes of mineralized tissues, such as osteoblasts and macrophages. The aforementioned is found even with the presence of a gap or interface between teeth of which imaging and tomographic examination suggests direct contact between two teeth, whether permanent or deciduous.

Crowding resulting from an imbalance among factors that keep the dental arch stable might be taken as a natural aging-related process or as a pathological condition. Such conceptual differ-

References:

1. Consolaro A. Reabsorções dentárias nas especialidades clínicas. 3ª ed. Maringá: Dental Press; 2012.
2. Consolaro A, Cardoso MA. Dentes não encostam em dente e nem reabsorvem outros dentes. *Rev Clin Orthod Dental Press*. 2017 Dez-2018 Jan;16(6):112-7.
3. Domon T, Sugaya K, Yawaka Y, Osanai M, Hanaizumi Y, Takahashi S, et al. Electron microscopic and histochemical studies of the mononuclear odontoclast of the human. *Anat Rec*. 1994 Sept;240(1):42-51.
4. Domon T, Osanai M, Yasuda M, Seki E, Takahashi S, Yamamoto T, et al. Mononuclear odontoclast participation in tooth resorption: the distribution of nuclei in human odontoclasts. *Anat Rec*. 1997 Dec;249(4):449-57.
5. Ericson S, Bjerklin K, Falahat B. Does the canine dental follicle cause resorption of permanent incisor roots? A computed tomographic study of erupting maxillary canines. *Angle Orthod*. 2002 Apr;72(2):95-104.
6. Ericson S, Kuroi PJ. Resorption after ectopic eruption of maxillary canines: a CT study. *Angle Orthod*. 2000 Dec;70(6):415-23.
7. Riviere HL. *Lab manual of normal oral Histology*. Chicago: Quintessence; 2000.