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With tooth preparation, will the pulp resist surgical aggression?

or

Criteria to assess pulp reparative, reactional and defensive ability before surgical procedures.

or

Does the dentin-pulp complex age or does it adapt?

How to cite: Consolaro A. With tooth preparation, will the pulp resist surgical aggression? Criteria to assess pulp reparative, reactional and defensive ability before surgical procedures. Does the dentin-pulp complex age or does it adapt? J Clin Dent Res. 2016 Jul-Sep;13(3):18-38.

DOI: http://dx.doi.org/10.14436/2447-911x.13.3.018-038.bio

Submitted: August 16, 2016 - **Revised and accepted:** August 26, 2016.

Abstract: *Improper use of teeth speeds the process of aging up, leads to changes relative to color and shape, and modifies the morphological, imaging and biological characteristics of the pulp-dentin complex. Rapidly aged pulp has its reparative, reactional and defensive abilities decreased. This should be taken into account whenever assessing restoration and other surgical procedures planning, in which case the following doubt arises: should endodontic treatment be carried out or will the dental pulp resist damage caused by extensive wear inherent to surgical procedures? This article aims at providing biological grounds to answer the question, in addition to understanding how pulp-dentin complex aging occurs and which the potential clinical implications are.* ***Keywords:*** *Dental pulp. Aging. Dentin sclerosis. Reactive dentin. Darkening. Pulp calcification.*

The dental pulp is a specialized soft connective tissue, surrounded by the hard and mineralized dentin. When producing dentine, to some extent, the odontoblasts are included in its structure, as cytoplasmic extensions inside the dentinal tubules.

The dental pulp has very specific morphological and functional aspects and, together with the dentin, it composes the dentin-pulp complex, a structural and functional unit that interacts with the enamel and cementum. In the apex, the pulp naturally continues along with the connective tissue of the periodontal ligament, without a structure or measure to separate them in a regular or measurable way, that is, without exact limits (Fig 1).

The apical termination of the dental pulp corresponds to the point where the last odontoblast on the dentin surfaces is found. From that point, on the internal surface of the root canal, there are cementoblasts depositing incremental cemental layers over the dentin. The most apical portion of the root canal is occupied by periodontal tissue and, when the dental pulp is removed, it will be recognized as periodontal stump. The line represented by the limit between cementum and dentin (CDC junction), three-dimensionally tends to be irregular in its occlusoapical position.

One of the purposes of the cytoplasmic extensions of the odontoblasts in the tubules (Fig 2 and 3) is keeping the hydric and ionic balance of the dentin and providing defense mechanisms against external aggressive agents. In the coronary portion, each extension reaches up to 3,5mm length with 1 to 3μm diameter (Fig 4 and 5). The closer they are to the pulp, the larger their tubular diameter is.

Although the dentin-pulp complex (Fig 2 and 3) is a functional and structural unit, regarding the treatment and prognosis of diseases that affect the tooth, the pulp must be ad-

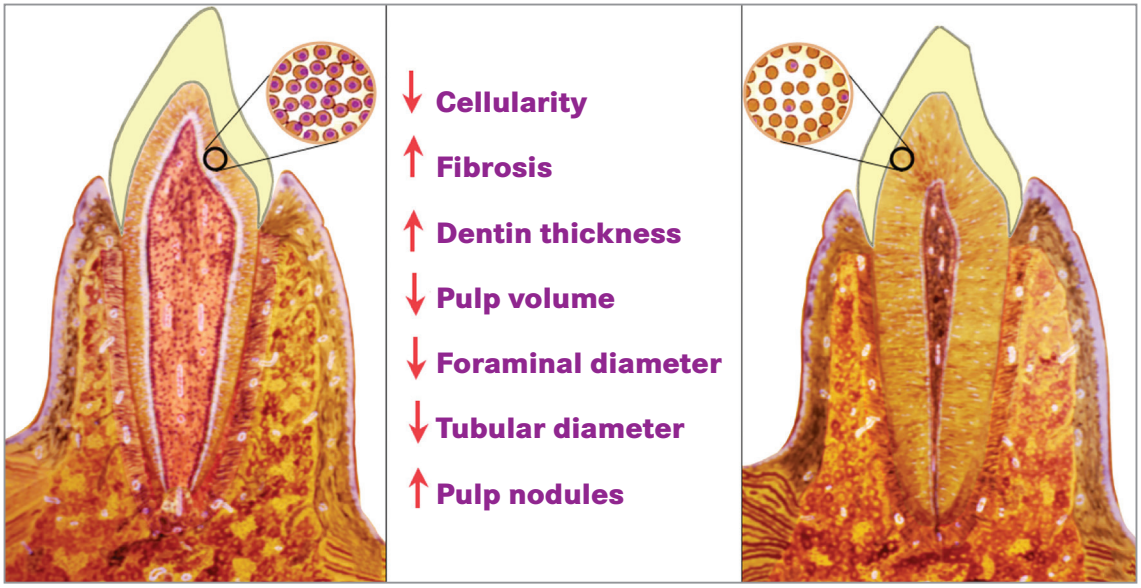

Figure 1: Adaptive dentin-pulp alterations or alterations proper of accelerated natural aging.

dressed separately from the dentin. Outlining and estimating the limits between the dentin and the pulp means establishing parameters for certain therapeutic and prognostic conducts.

Unconditionally replacing the terms dentin and/or pulp by the term dentin-pulp complex is unfeasible from the clinical and therapeutic point of view, for the dentin and the pulp are topographically distinct. The term dentin-pulp complex should be applied mainly when referring to embryonic and functional matters, as they are functionally integrated (Fig 2 and 3).

HOW A TOOTH AGES RAPDLY

Adaptive dentin-pulp alterations represent accelerated aging

The main pulp alterations are inflammatory because, in a singular way, the dental pulp in the human body does not give rise to benign or malign neoplasia. That is amazing!

The dentin-pulp complex changes its structure with age, but these changes (Fig 1) will only be significant when accelerated by factors which are a consequence of the intensified or improper use of the teeth over the years, such as:

1. attrition, with or without bruxism;
2. abrasion;
3. erosion;
4. abfraction;
5. minor traumas, such as concussion;
6. dental caries;
7. surgical and restorative procedures.

The orthodontic movement, periodontal disease and occlusal trauma do not represent factors that accelerate the aging process.[2]

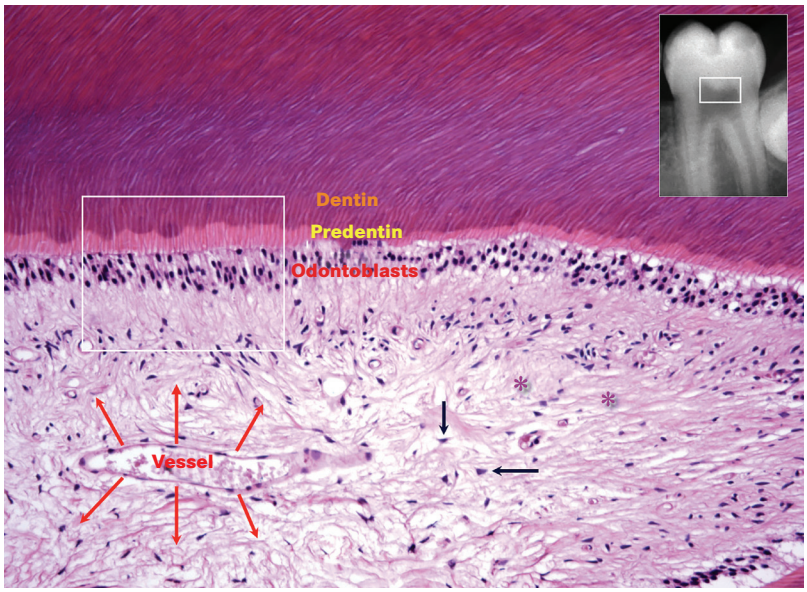

Figure 2: Dentin-pulp complex: the pulp vessels let the plasma out to form the interstitial liquid (red arrows) that nourish all the cells and compose the extracellular matrix (*), establishing positive intra-pulp pressure. The black arrows point to undifferentiated cells or young ones that are present in young pulps. In white, enlarged area in the next figure (HE, 10x).

Age, singly, with the improper use of healthy teeth, leads to minor alterations from the morphological point of view, without modifying significantly its reactional and restorative potential. Normal teeth show prevalence of 5,5 to 7% of pulp nodules, regardless of age. Nonetheless, when it comes to teeth with a history of caries, restorations and traumas, prevalence increases to 73 to 76,7%.

It would be more adequate applying the term 'Adaptive alterations of the pulp' instead of using the term 'pulp aging', for there is no correlation between the age group of the carrier and the condition of the tooth analyzed.[6]

For decades, it was thought that many alterations observed in the dentin-pulp complex were related to age. Nowadays, however, it is known that the main 'aging agent' of the pulp is the intensified and/or improper use of the teeth over the years (Fig 1). Age, singly, does cause changes, but these changes are subtle and minimally relevant from the clinical and biological point of view.

The intensified and/or improper use of the teeth throughout life causes changes that, for a long time, were called 'degenerative'. That was an inadequate term because, in fact, the structures are just adapting to a functional requirement. For many years, these changes were also named 'involutive', but involuting may imply backtracking, going backwards.

These dentin-pulp changes can be more accurately called 'alterations proper of natural or accelerated aging', for they are adaptive answers of the dentin-pulp complex to these superimposed situations – age and intensified and/or improper use – over the years (Fig 1).

Clinical Importance

The clinical importance of these alterations proper of aging or intensified and/or improper use of the teeth throughout life is related with:

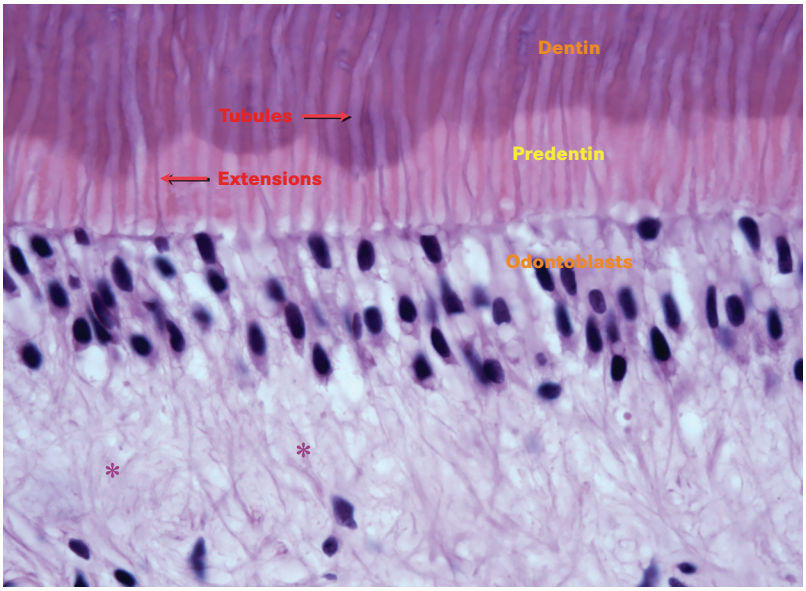

Figure 3: Dentin-pulp complex, highlighting the odontoblastic extensions and the dentinal tubules, with empty spaces among them, filled by dentinal fluid, coming from the pulp interstitial liquid (*extracellular matrix in the zone insufficient in pulp cells) (HE, 40x).

1. Decrease of the reactional and restorative capacity of the pulp against external aggressors, to the extent of influencing the prognosis of pulpitis, accidental exposure of the pulp and pulp reactions after deeper and/or more comprehensive cavity and prosthetic preparations.
2. Alterations of color and form which cause or are associated with size reduction and dentin exposure.

ADAPTIVE DENTIN-PULP ALTERATIONS OR ALTERATIONS PROPER OF AGING

The main alterations (Fig 1) proper of aging are:

1. increased dentin thickness;
2. secondary physiological or reactional deposition of dentin;
3. reduction of the pulp cavity volume;
4. decrease of the dentinal tubules diameter;
5. dentinal sclerosis;
6. dead tracts of dentin;
7. reduction of pulp cellularity;
8. reduction of pulp vascularization and innervation;
9. interstitial fibrosis and pulp hyalinization occurrences;
10. dystrophic calcification or pulp nodules.

The main aging factors of the dentin-pulp complex are the ones that activate their defense mechanisms, which are:

- Physical-chemical lesions of the dental tissues, due to attrition with or without bruxism, abrasion, erosion and abfraction.
- Chronic dental caries, due to invasion of bacteria and their products.
- Restorative and surgical procedures.
- Frequent and minor traumas.

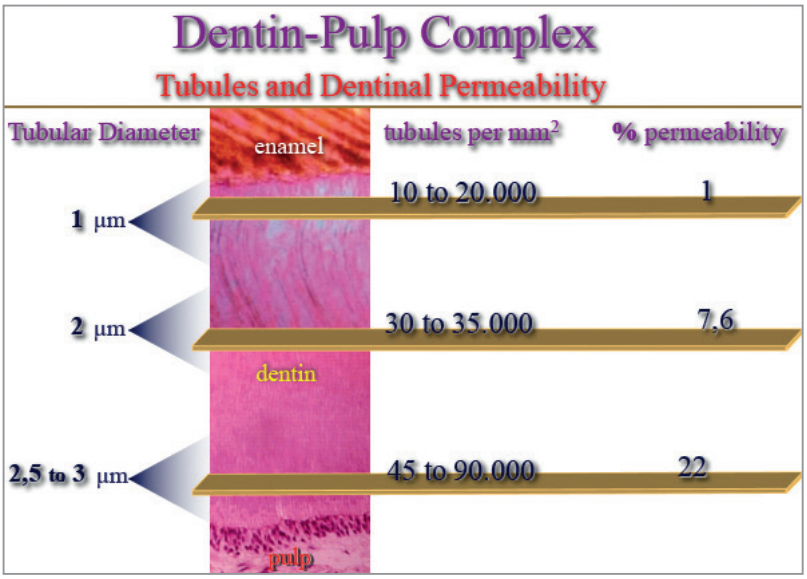

Figure 4: Direct relations between the degree of dentinal permeability and the density diameter per mm2 of the dentinal tubules, in accordance with the degree of proximity to the pulp.

Once again: the orthodontic treatment, chronic inflammatory periodontal disease and occlusal trauma are not efficient pulp aging agents[2].

The defense mechanisms of the dentin-pulp complex, despite being efficient when it comes to the maintenance of pulp vitality, will have as a natural consequence a higher or lower degree of pulp aging. The aging factors of the pulp act in a cumulative and simultaneous way.

Analysis criteria and expressions of accelerated aging of the dentin-pulp complex

1. Increased dentin thickness, due to the cumulative deposition of secondary dentin on the pulp wall, as a whole, or the localized deposition of reactional dentin, also known as pathologic secondary dentin. There will be a consequential and evident decrease of pulp volume.
2. Reactional dentin, also referred to as pathologic secondary dentin. Composed in the more accelerated depositions of dentin by the same odontoblasts that deposited it, previously and continuously, to the physiological secondary dentin, now stimulated more intensively by some external irritative agent, such as caries, surgical preparations, attrition and others (Fig 6 and 7).
3. Reduction of the pulp volume, which diminishes the pulp's capacity of containing an exudate and a bigger inflammatory infiltrate necessary to destroy the aggressor in order to, afterwards, promote repair.

The reduced pulp volume (Fig 1) significantly limits the capacity of containing, for longer and in a more effective way, the exudate and the inflammatory infiltrate proper of pulpitis, without compression of the venules and blood flow arrest. Thus, pulp volume reduction contributes to the decrease of defensive and restorative efficiency of the pulp.

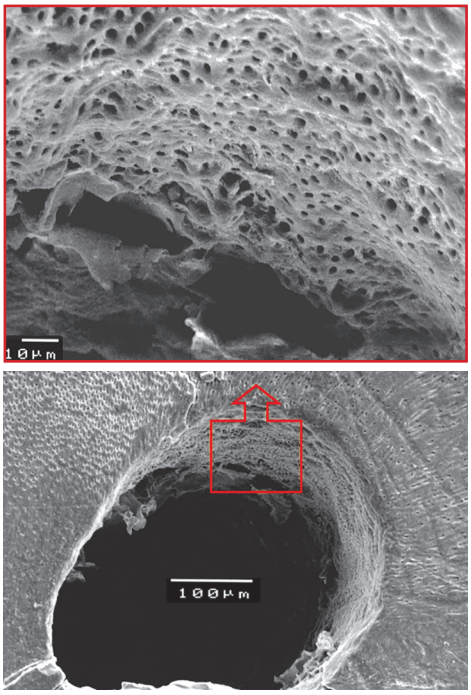

Figure 5: Dentinal tubules on the pulp wall seen with an electronic scanning microscope. Irregularity due to undulation on the dentinal surface and the density of tubules per area stand out.

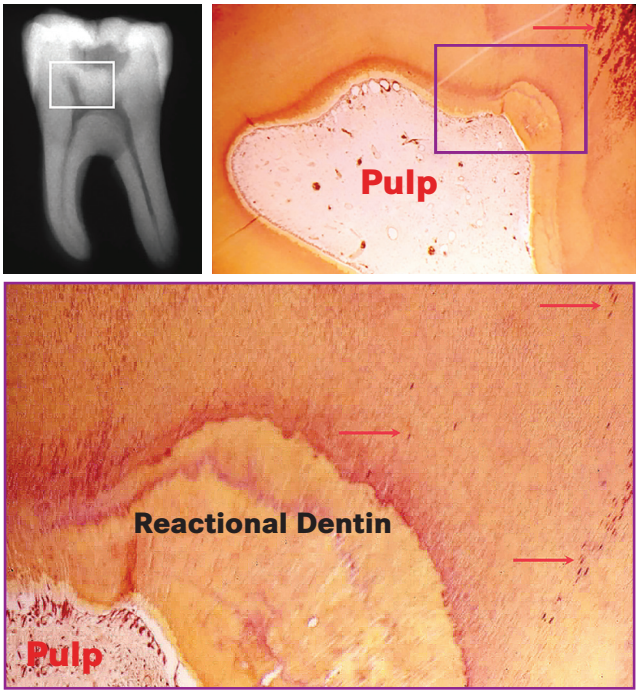

Figure 6: Caries in the dentin, with bacteria (arrows) in the tubules and deposition of reactional dentin on the pulp wall (BB; 10x e 25x).

The normal pulp volume for each tooth is, on average, 0,02ml. All the pulps of a human being should contain in its total space 0,05ml. Each 'ml' corresponds to approximately 20 drops. Reduced pulp volume decreases these figures even more.

4. Decrease of the dentinal tubules diameter, due to accumulation or acceleration of the continuous deposition of peritubular dentin and reduction of dentinal permeability[1,4].

The significant decrease of the dentinal tubules diameter may reduce the circulation and efficiency of dentinal fluid as a defense element (Fig 8 and 9) regarding its quantity, quality and velocity of flow in the tubular structure. The dentinal fluid flow interferes in the degree of penetration of substances that are applied to the dentinal surface during restorative and surgical procedures.

Once the odontoblastic layer is altered and the dentin surface is exposed, intra-pulp pressure gets higher than intra-oral atmospheric pressure (Fig 8). Dentinal flow from the inside outwards makes it difficult not only the penetration of the applied substances, but also the infiltration of bacteria and their products, such as acids, enzymes, toxins and metabolic products (Fig 10). The dentinal fluid contains, for its turn, defensive antimicrobic elements, such as plasmatic proteins, including immunoglobulins.

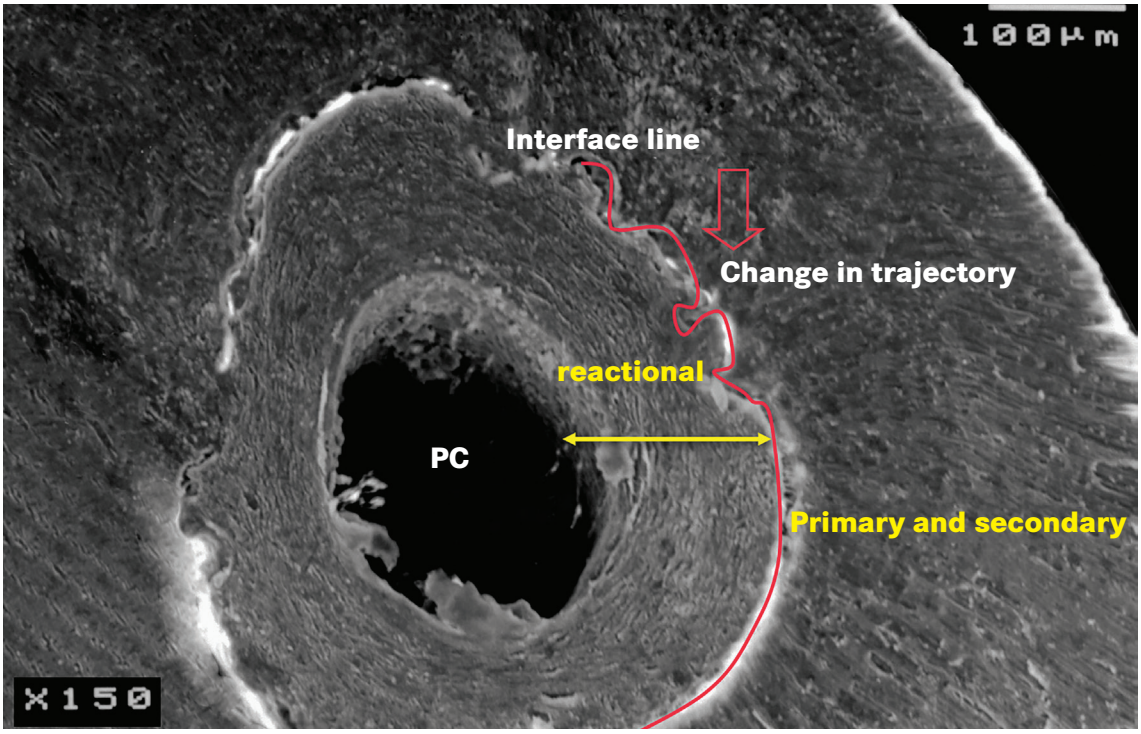

Figure 7: In the pulp cavity (PC), it is possible to see the deposition of reactional dentin (double arrow) over the primary and secondary dentin, resulting in an interface line (line) that interrupts the light of the dentinal tubule and hampers the arrival of bacteria and their products to the dental pulp (SEM).

5. Dentinal sclerosis, which represents the closing of light of the dentinal tubules by the accelerated and disorganized deposition of peritubular dentin against aggressive agents already mentioned[1,4,7]. Dentinal sclerosis reduces, hinders or blocks the penetration of chemical products and bacteria towards the pulp (Fig 11 and 12). The dentin that becomes sclerosed does not increase its physical resistance and gets less permeable[1]. The color of sclerosed dentin is darker than normal.
6. Dead tracts of dentin: in some situations, the odontoblastic extensions, instead of producing peritubular dentin, may retreat, so the tubules will be empty, what characterizes the dead tracts of dentin (Fig 11 and 12). The empty tubules under a harmed dentinal area are named dead tracts of the dentin. Such condition probably favors the penetration of bacteria into caries or of chemical substances related to restorative or surgical procedures.
7. Reduction of pulp cellularity on the pulp tissue, which results in the decrease of the restorative capacity against conservative procedures, such as capping, pulp curettage and pulpotomies (Fig 13). In order to form restorative or tertiary dentin in these procedures and areas, it is necessary differing

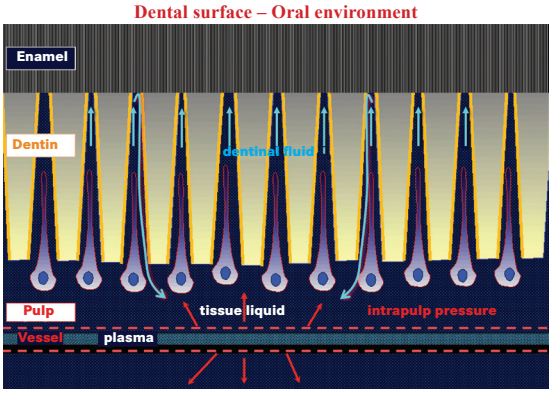

Figure 8: The dentinal fluid is formed by the positive intra-pulp pressure that makes the interstitial liquid circulate between the odontoblastic extensions and the walls of the dentinal tubules. When the dentinal fluid reaches the enamel, it reflows to the pulp, irrigating and thus nourishing the odontoblastic extensions and, at the same time, creating a defense mechanism by its composition and the flow itself, making it difficult for chemical substances, bacterial products and bacteria themselves to get to the pulp.

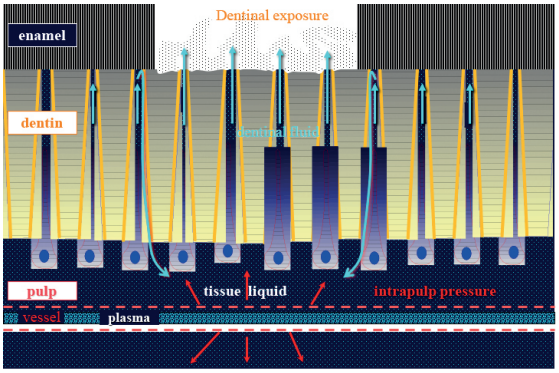

Figure 9: In cavities with exposed dentin, the dentinal fluid leaves through the tubules and accumulate on the dentinal walls, and its components may have antimicrobic action, buffer effect and may become part of the layer of dentin mud. The dentinal fluid, thus, hinders the arrival of chemical substances, bacterial products and bacteria to the corresponding dentin pulp.

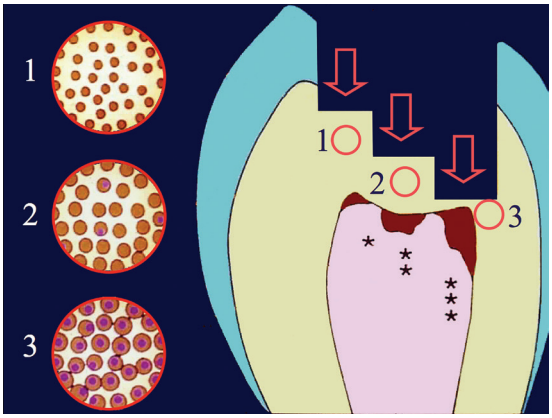

Figure 10: The preparations and the substances (arrows) used during the surgical and restorative procedures involving the dentin tend to be more aggressive as they approach the pulp, due to the increase of the diameter of the dentinal tubules and their density per area (circles). The inflammation degree and the damage caused to the dental pulp varies (*) according to how deep the prepared cavities are and, in the same proportion, the deposition of subjacent reactional dentin.

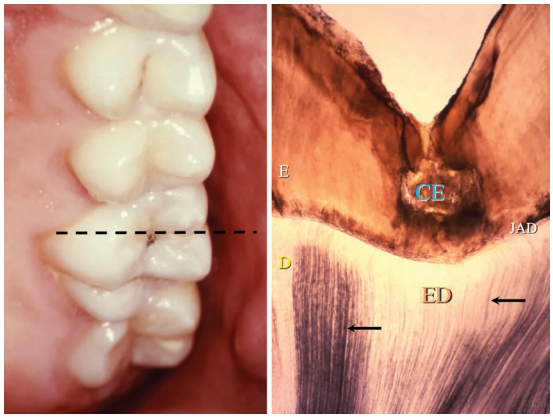

Figure 11: Caries in the enamel (CE) leading to dentinal sclerosis (DE) characterized by the closure of the tubules by peritubular dentin. The dead tracts of dentin are dark empty tubules (arrows). The dashed line represents the cutting analyzed with a microscope in B. (E = enamel, D = dentin and AC = amelocementary junction) (CD; 10x).

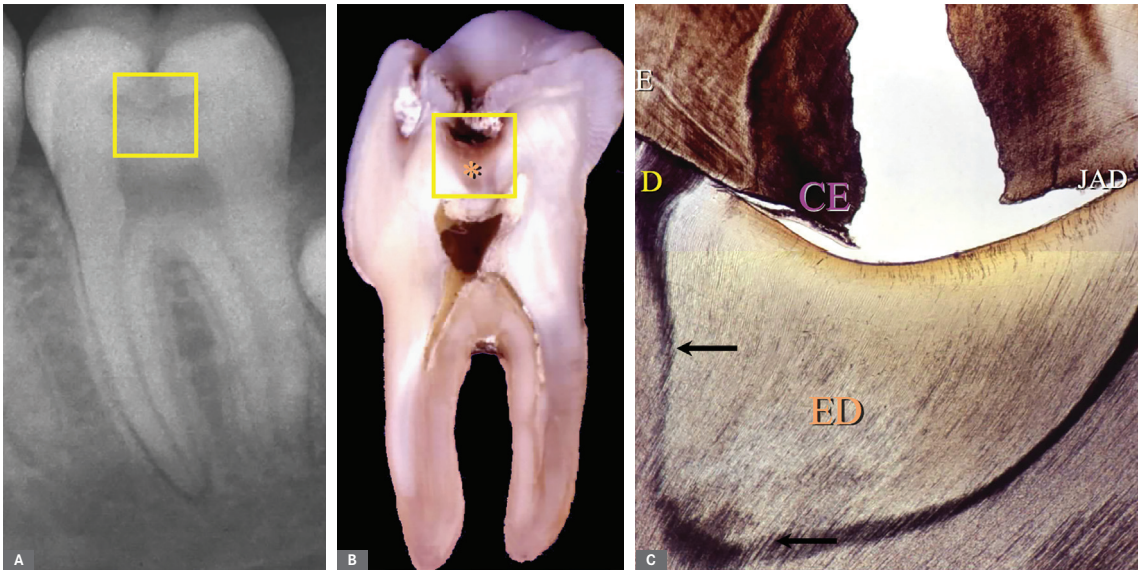

Figure 12: Caries in the enamel and the dentin (CE) with dentinal sclerosis (ED) and dead tracts of dentin (arrows) well characterized. Macroscopically, the sclerosed dentin is darker than its neighboring one, which was not affected (*). On the pulp wall there was deposition of reactional dentin. E = enamel, D = dentin and AC = amelodentinary junction) (CD; 10x).

the undifferentiated cells of the pulp in a third generation of odontoblast, in order to rebuild a new layer of cells and deposit the barrier of mineralized tissue under the material placed on the ulcered and operated area.

8. Reduction of pulp vascularization and innervation, reducing sensitivity and operative bleeding as the pulp aging occurs[3]. The angiogenic capacity of the pulp also decreases with the reduction of number of vessels, which influences its restorative capacity negatively.
9. Interstitial fibrosis and pulp hyalinization occurrences. The increase of the fibrous component associated with the reduction of pulp cellularity and vascularity (Fig 13) will decrease the collagen renovation velocity, followed by biochemical alterations of the extracellular matrix. The biochemically altered matrix results in small or large hyalinization areas. The hyaline areas may act as a crude organic matrix to be irregularly mineralized, creating pulp nodules, by dystrophic calcification in the central region of the pulp.
10. Pulp nodules and diffuse pulp calcifications: the pulp nodules, together with the reduction of pulp volume, are imaging indicators of pulp aging, especially under confirmation that they are associated to clinical situations that point to the occurrence of pulp aging factors.

In certain esthetic and/or restorative procedures, greater defensive and restorative capacity of the pulp can be required. However, if it is aged, it may indicate the need of previous

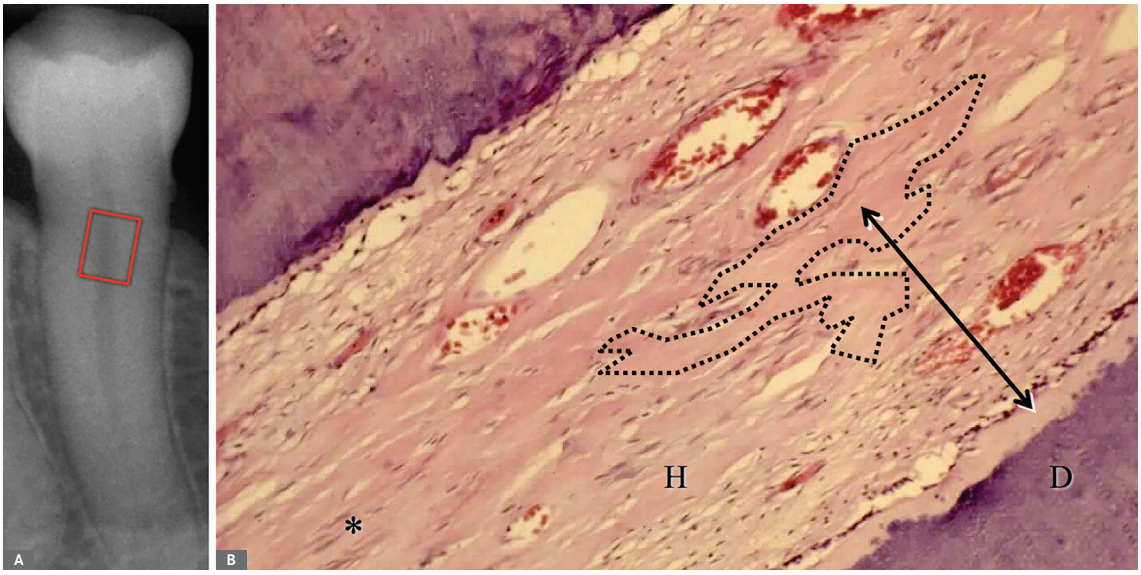

Figure 13: Dental pulp with low cellularity, pulp fibrosis (*) and hyalinization areas (H), highlighting the largest one with dashed lines. The double arrow reveals similarity of the hyaline area with the predentin also collagenic, a region of deposition of mineral salts (D = dentin) (HE; 25x).

endodontic treatment. Without adequate space, with reduced cellularity and vascularization, it becomes difficult to recompose pulp's normality.

PULP NODULES AND OTHER PULP CALCIFICATIONS: Differential diagnosis, meanings and clinical implications

Tissues calcification should be identified more precisely as mineralization, given that calcium is not the only mineral that takes part in the process. Nevertheless, the frequent use of calcification for referring to mineralization processes has made these terms almost synonyms (Fig 14 and 15).

Body tissues calcification may be normal or pathologic. In the second case, tissues calcification can be classified in accordance with two criteria.

By adopting the first criterion, distribution in the affected areas, tissues calcification can be classified as:

a) Focal: in some points of the dental pulp there may be pulp nodules, that is, focal pathologic calcifications;
b) Diffuse: when multiple and minuscule pulp areas suffer aleatory deposition of minerals, regardless of the cause, there will be a diffuse pathologic pulp calcification.

The second very important criterion for classifying pathologic tissues calcifications refer to the previous normality of the tissues where minerals will be deposited, since those tissues will play the role of an organic matrix.

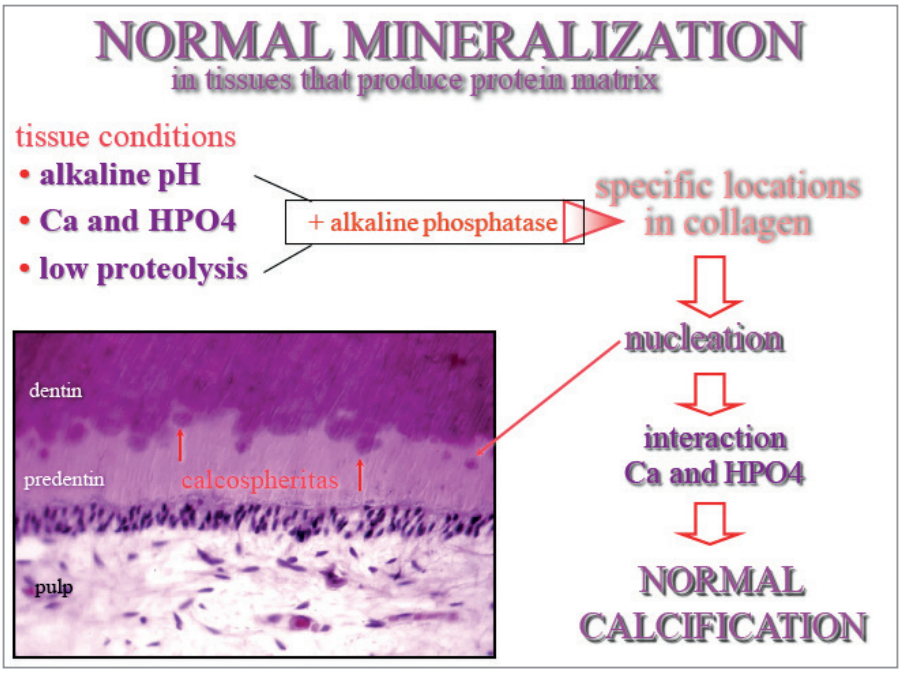

Figure 14: Conditions and stages of normal mineralization, as in the predentin.

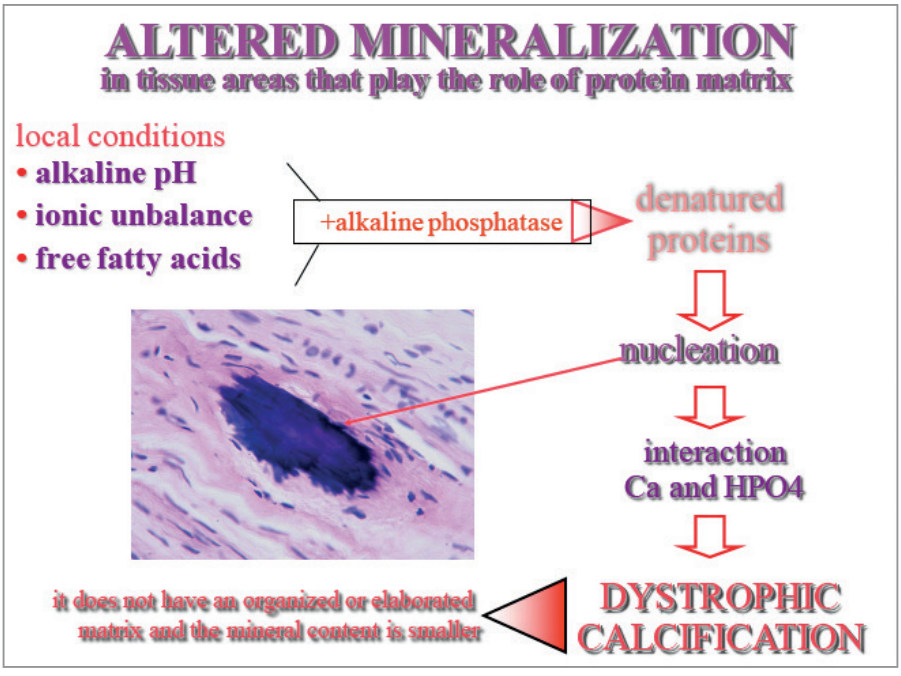

Figure 15: Conditions and stages of dystrophic calcification, as in the dental pulp.

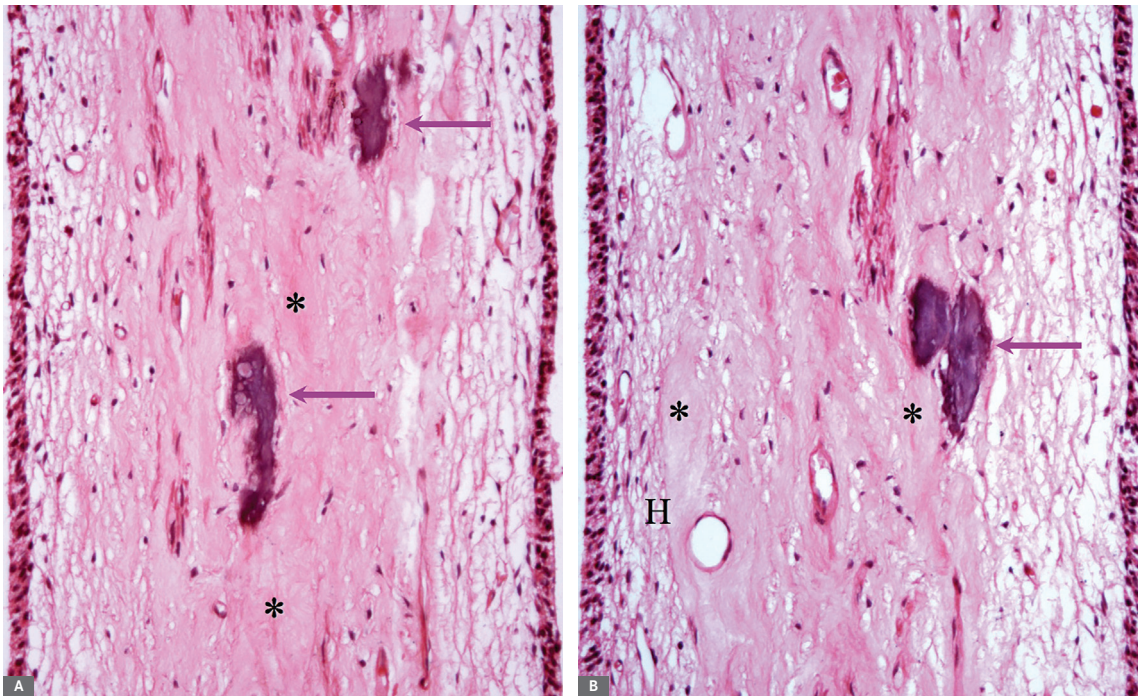

Figure 16: Fibrosis and pulp hyalinization (*), with low cellularity, of a same tooth, highlighting basophilic areas of focal dystrophic calcification or pulp nodules (arrows) (HE; 25x).

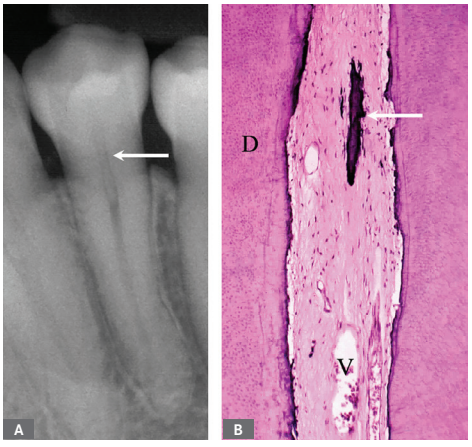

Figure 17: Needle-shaped pulp nodules (arrow) are a result of the dystrophic calcification on walls of vessels and nerves which, during aging, have their walls hyalinized and can be an area of deposition of mineral salts (D = dentin, V = vessels) (HE; 25x).

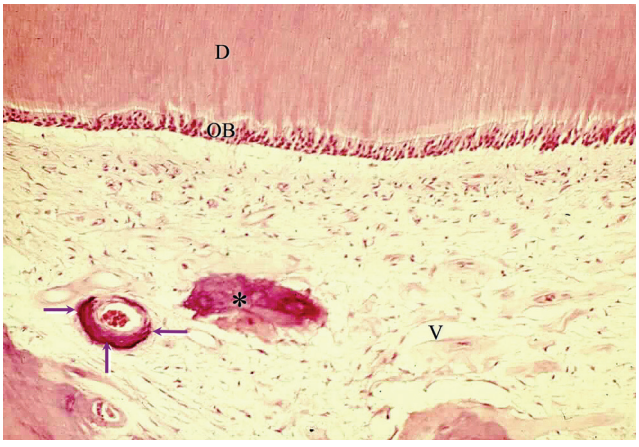

Figure 18: Dystrophic calcification (arrows) on the wall of blood vessels, which represents one of the ways for the beginning of pulp nodules, especially the needle-shaped ones, as shown in the previous figure. Near it, it is possible to notice a pulp nodule (*). (D = dentin, V = vessel, OB = odontoblasts) (HE; 25x).

a) dystrophic calcifications: if that happens on previously altered tissues (Fig 15);
b) metastatic calcifications: if the minerals precipitate on normal tissues.

Formation mechanism of pulp nodules

In the dental pulp precociously aged due to the decreased functional requirement, there is a sharp loss of its cellularity and the pulp extracellular matrix excessively gains a lot of dense collagen fibers (Fig 13). In these precociously aged pulps, in cases of abrasion, attrition and erosion, big restorations and prosthetic preparations, collagen shows a low renovation rate and large areas without cells are established.

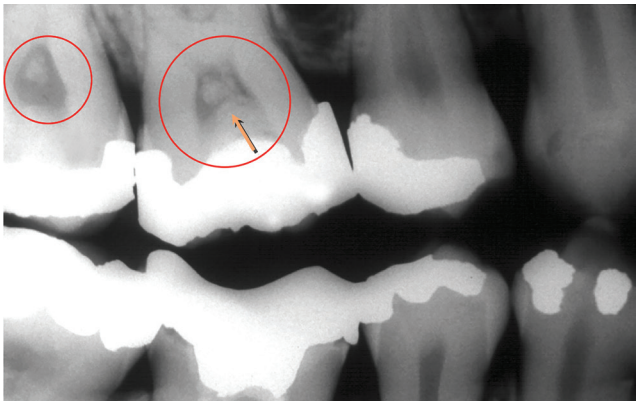

Figure 19: Pulp nodules, associated to pulp space reduction, with reactional dentin (arrow) and cause-effect relationship well determined and represented by extensive restoration.

These pulp areas without cells and rich in collagen are given, due to their pinkish aspect, the name of hyaline areas or hyalinization areas, and represent the most common region for

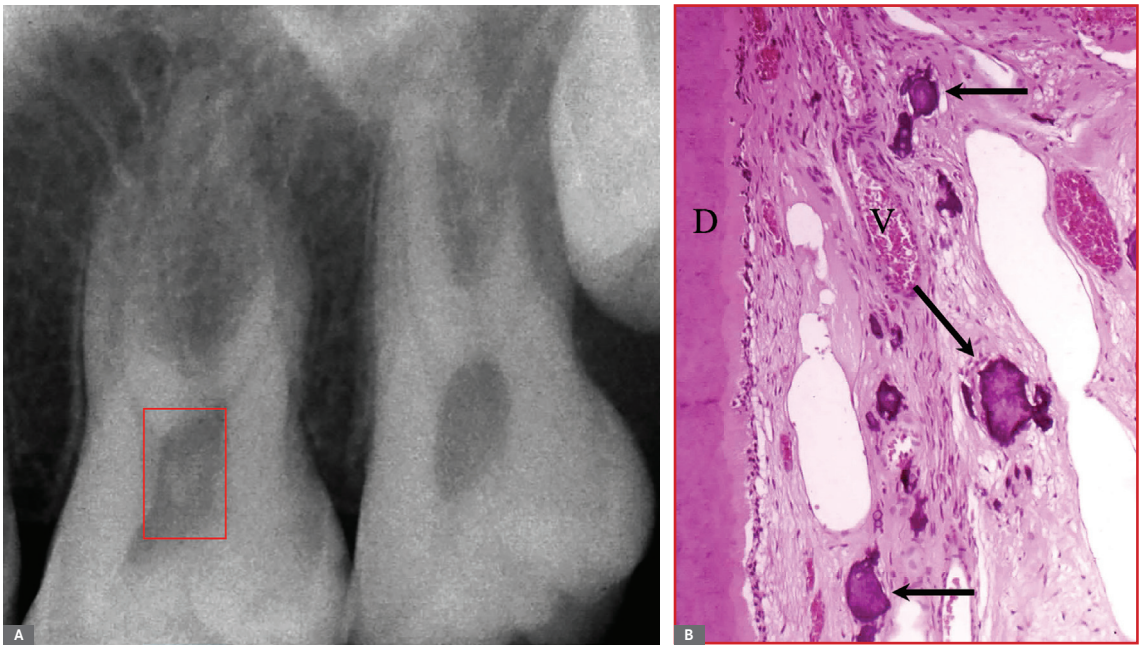

Figure 20: Pulp nodules in an area corresponding to box in A, in which just a few appear individualized in the image, for they generate signals only when exceeding the 200µm diameter, or 0,2mm (arrows) (D = dentin, V = vessel) (HE; 25x).

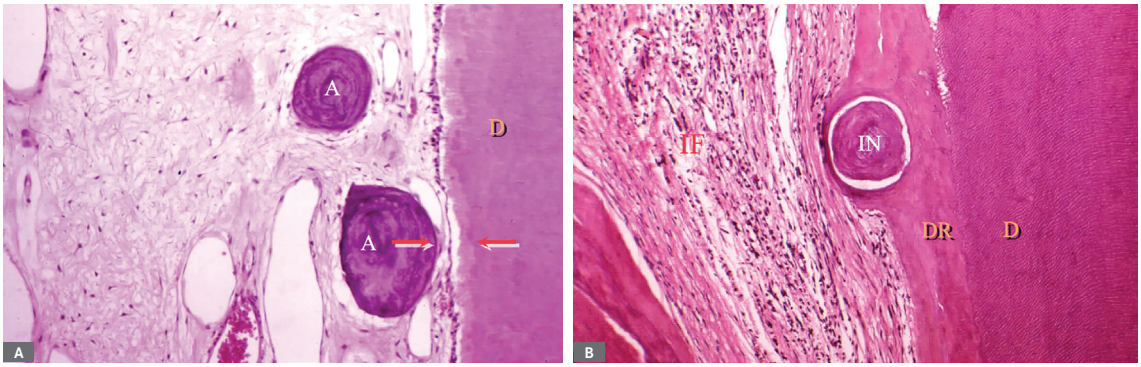

Figure 21: Pulp nodules: free (F), practically adhered (A) and inserted (IN) in the dentinal wall, by the continuous and accelerated deposition of reactional dentin (RD), associated to the chronical and subclinical inflammatory process (IF). Both arrows reveal the proximity between the dentinal wall and the pulp nodule (D = dentin) (HE; 25x).

the deposition of mineral salts into the pulp, as they act as an organic matrix (Fig 16). This is the most common mechanism for the formation of the pulp nodules (Fig 15).

The pulp nodules can also be formed from the hyalinization of vessels and nerves walls in precociously aged pulps (Fig 17 and 18), when they assume a needle-shaped radiopaque linear image[5].

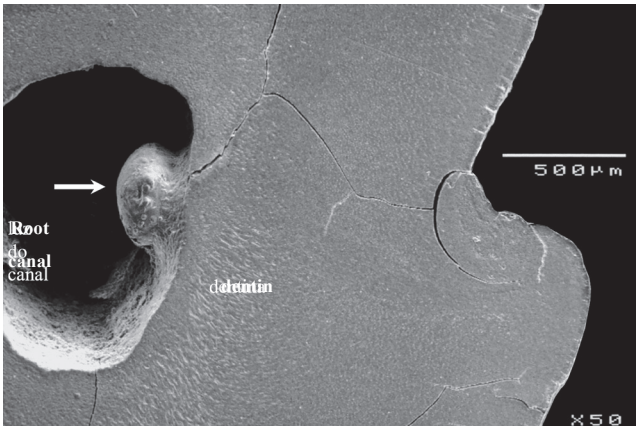

Figure 22: Adhered pulp nodule (arrow), to the electronic scanning microscope.

The growth of the pulp nodules inside the pulp tends to be concentric and slow, without the compression of the vessels and nerves in their evolution, thus, not leading to necrosis or pain. Regarding the pulp wall, the nodules are classified as free, adhered and inserted or interstitial. Its imaging appearance only happens when it reaches the diameter of approximately 200 µm or 0,2mm (Fig 18 to 22).

Formation mechanism of the pulp diffuse calcification

It represents the other form of dystrophic calcification in the pulp, besides the pulp nodules. When precocious pulp aging is intense and had its beginning a long time before, the connective tissue gets very fibrous and hyalinized; cellularity is minimum. In these cases, multiple, numerous and minuscule areas of deposition of mineral salts happen simultaneously and are randomly distributed along the pulp tissue, being named as Diffuse pulp calcification, for it does not assume a nodular and focal form (Fig 23 and 24).

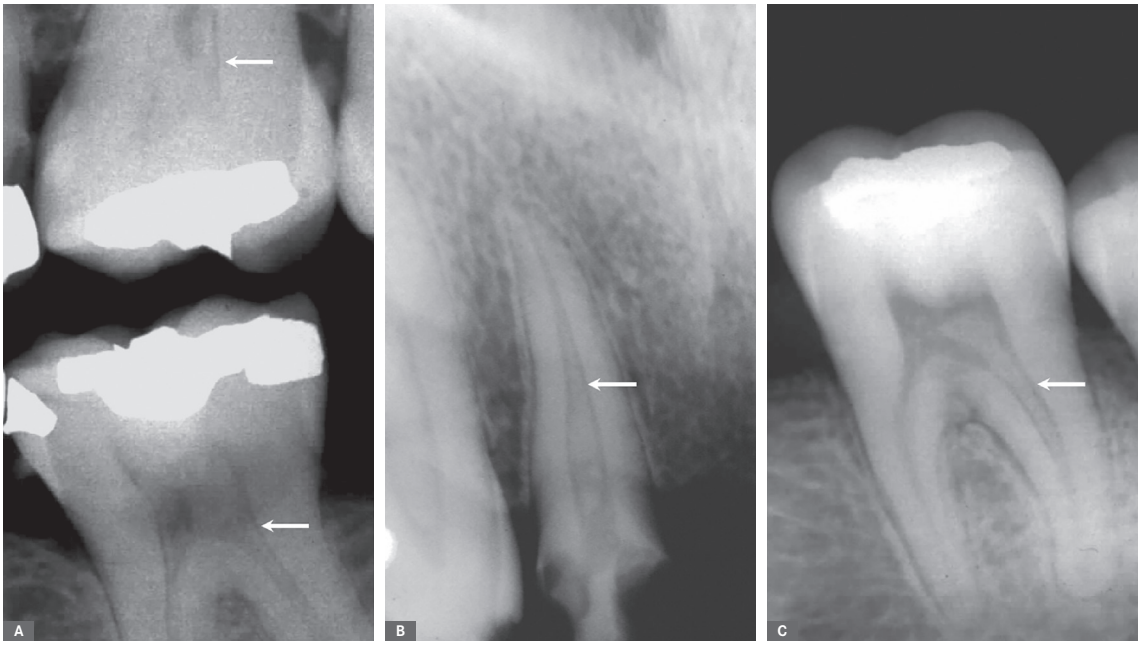

Figure 23: Diffuse pulp calcifications represented by multiple and small areas and of dystrophic calcifications in rapidly and precociously aged pulps, as it can be microscopically seen in the Figure. Highlighting the evident maintenance of the limits or of the pulp walls (arrows).

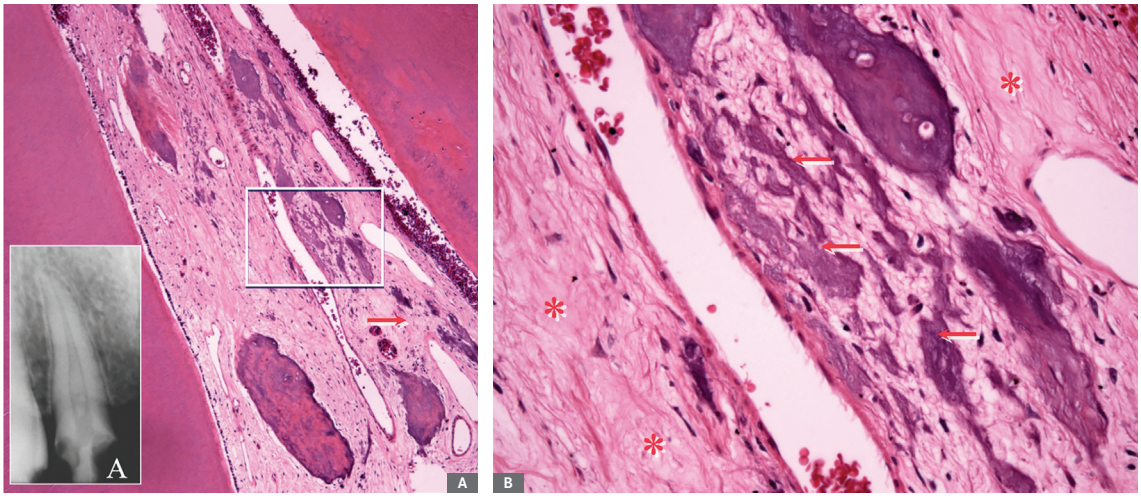

Figure 24: Diffuse pulp calcifications, as multiple and small areas and of dystrophic calcifications (arrows) in pulps with high degree of fibrosis and hyalinization of their extracellular matrix (*) (HE, 4x e 40x).

In the pulp calcification, the odontoblastic layer is represented by only one or two layers of cells, but it is there. Imagiologically, this 'persistence' of the odontoblastic layer explains why the pulp limits are present and possible to be outlined in the Diffuse pulp calcification, what differs it from the Calcific Pulp Metamorphosis, whose limits are progressively extinguished until the end (Fig 23 and 24).

It is important to highlight: Calcific Pulp Metamorphosis does not represent aging!

Calcific pulp metamorphosis represents the aleatory and dysplastic deposition of dentin on almost the entire pulp space, in cases of traumatized teeth or teeth with partial lesion of the vascular bundle that enters the pulp.[2]

Calcific pulp metamorphosis represents a reactional state to a mild dental trauma[2]. In order to survive in conditions of low blood supply, the pulp cells, in a general way, suffer metaplasia or a phenotypic transformation into odontoblasts which start depositing aleatory dentin being included to it, reducing their metabolism.

The pulp limits will be extinguished and the pulp spaces disappear gradually: a process also known as 'canal obliteration'. Calcific pulp metamorphosis has no etiologic connection with accelerated pulp aging.

Formation mechanism of metastatic pulp calcifications

On normal tissues, when there is a great amount of calcium in the blood, the minerals may deposit themselves in normal tissue components of the connective tissue – with collagen fibers, vessels, nerves and cartilages – without these being previously altered or modified. This type of pathologic calcification represented by the abnormal deposition of mineral salts in previously normal tissues is referred to as Metastatic Pathologic Calcification (Fig 25 and 26).

However, for that to happen, excess of calcium in the blood should be really huge. That happens in extreme and very serious situations of:

- hyperparathyroidism;
- multiple trauma;
- disseminated malignant neoplasias;
- osteomyelitis;
- other serious pathologic situation.

Metastatic pulp calcifications are rarely detected. Eventually, a professional may find patients having all the pulps, or most of them, obliterated, but always keeping the pulp limits distinct in the images. Once a situation of bruxism and generalized attrition is rejected – which would point to rapidly induced aging –, it is necessary to check with the patient for their systemic situation, in search of some serious disease that could increase, exuberantly, the blood calcium level, such as the ones aforementioned.

Although the systemic diseases that increase the blood calcium level are common in medical day to day practice, metastatic pulp calcifications are not common. In the cases in which the diagnosis of metastatic pulp calcification is concluded, there is no therapeutic need, for itself, but only orientation and monitoring, since, there are no symptoms and the affected teeth are not jeopardized.

Differential diagnosis must also be done in case of Calcific Pulp Metamorphosis, which, in general, affects only one or two neighboring teeth (Fig 25 and 26).

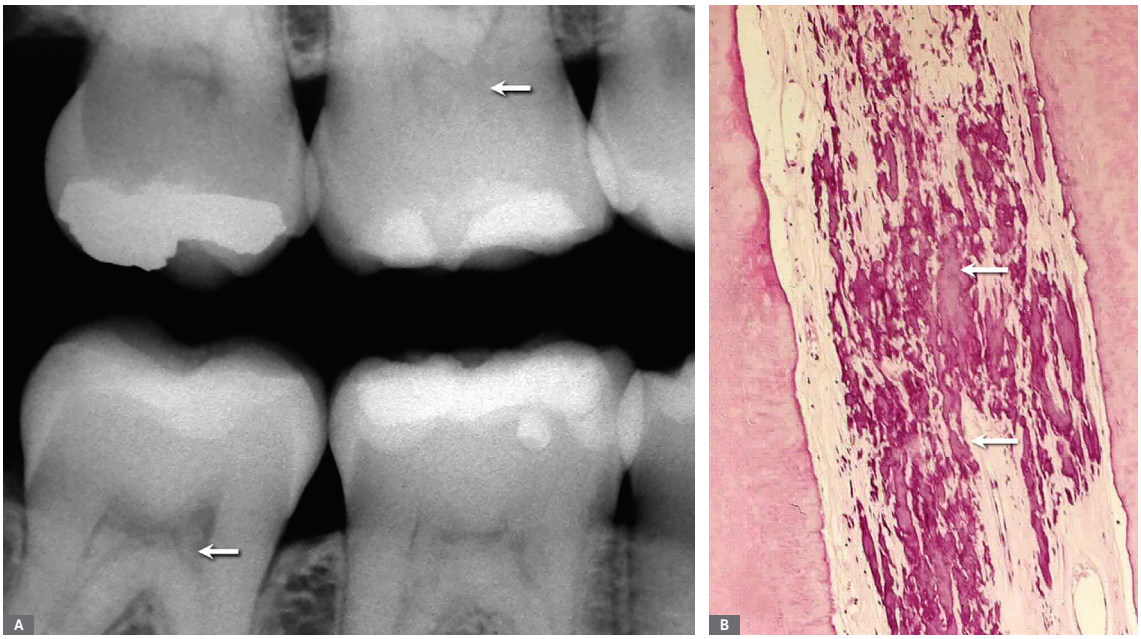

Figure 25: Metastatic pulp calcifications in a patient with a systemic disease which increased considerably the blood calcium level, as in B, where it was a carrier of kidney-related malignant neoplasia. Highlighting the number of teeth affected and the maintenance of the pulp limits. (HE, 25x).

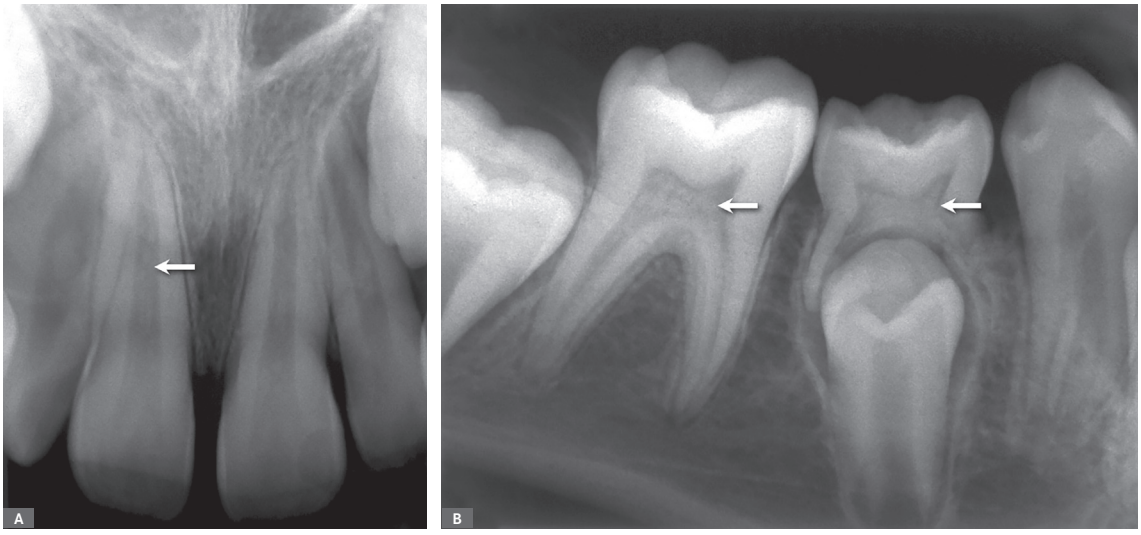

Figure 26: The presence of multiple diffuse pulp calcifications, with no cause-effect relationship and in a patient with good dental conditions, must immediately lead to medical examinations to check for the existence of systemic diseases which may cause considerable blood calcium level, as in the case of this 9-year-old boy, presented like this, including the deciduous teeth (by Prof. Dr. Fabrício, Ribeirão Preto/SP).

Criteria of differential diagnosis among pulp nodules, diffuse calcification, metastatic calcification and calcific pulp metamorphosis

Pulp nodules and diffuse pulp calcification represent the classical expression of dystrophic calcification of and in the dental pulp. The pulp nodules represent the deposition of mineral salts in areas previously altered by the pulp, that is, they represent dystrophic pulp calcification.

Yet, when it comes to great dental traumas, such as concussion and dental dislocation, the pulp nodules do not represent the main consequence to be feared or escaped. That consequence is actually the calcific pulp metamorphosis, that is, metaplasia of the pulp tissue into odontoblasts and dentin randomly deposited, obliterating the chamber and the root canal, and also gradually 'extinguishing' the imaging limits of the dental pulp.

In dental pulp showing pulp nodules, even when they are multiple and take over all the pulp space, the limits of the internal walls can be outlined and identified by imaging in periapical and interproximal radiograph. That identification is possible even when dystrophic pulp calcification is represented by multiple and small areas also identified as diffuse pulp calcification.

In the differential diagnosis between diffuse pulp calcification – one of the types of dystrophic pulp calcification – and calcific pulp metamorphosis, identifying the pulp limits represents an important reference point through the imagological point of view. When the pulp limits are outlineable and present, even if the pulp space is filled by radiopaque structures that indicate broad and total occupation of the pulp space, the diagnosis that must prevail is that of diffuse pulp calcification.

But if the pulp limits are diffuse, not possible to be outlined and without clearness, and the pulp space is being homogeneously hidden and gradually reducing pulp 'volume', the most recommended diagnosis is the one of calcific pulp metamorphosis.

In several situations, we face a radiographic image separately from a certain tooth. But the context in which the tooth with pulp alterations of that nature is inserted helps us to determine the diagnosis in a precise way:

1. Calcific pulp metamorphosis almost always affects one or two teeth that differ from the others, with normal pulp spaces. Diffuse pulp calcification affects many teeth, and the same happens to the rare metastatic pulp calcification.
2. In the calcific pulp metamorphosis, in general, there is a previous history of dental trauma, represented by concussion or dislocation. In many cases in which the patient does not report the previous trauma, the tooth presents enamel fracture and, very often, an adjacent tooth with aseptic pulp necrosis and chronical periapical lesion, when there are no coronary fractures. Patients tend to be younger.
3. When it comes to diffuse pulp calcification, the teeth show excessive deterioration, such as attrition or abrasion, broad restorations and prosthetic preparation, almost always associated to bruxism and clenching. The group of affected teeth is not related to the previous history of trauma. Patients tend to be older.
4. As for diffuse pulp calcification and calcific pulp metamorphosis, there is no association with systemic diseases. Whenever this association occurs, it is necessary to establish a

diagnostic relation with metastatic pulp calcification and several completely rigid teeth are impaired. That situation is clinically very rare and is associated to systemic diseases with high level of blood calcium.

Acute Dental Caries versus Chronical Dental Caries: Young teeth and pulps may be caught by surprise!

In chronical caries, the process lasts for months, maybe years, to reach the dental pulp to the extent of jeopardizing its vitality. The greatest thickness of the dentinal wall, the deposit of reactional dentin and dentinal sclerosis in naturally longer and more narrow tubules delay pulp impairment.

In recently erupted teeth in the oral environment, the dentinal wall is much thinner and the dentinal tubules are very broad in their diameter, besides being short. The pulp is exuberantly younger and has great space for its structures.

Nevertheless, caries in those teeth spread rapidly towards the pulp because, with broad and short tubules, bacteria and their products soon reach the internal dentin wall, for there is not time for depositing peritubular dentin for sclerosis, nor forming reactional dentin to increase the thickness of the dentinal wall. Pulpitis settles precociously and, surprisingly, pulp necrosis comes quickly.

Another example: pulpotomies only in young teeth

Regarding completely developed teeth, including the apex, pulp cellularity tends to reduce gradually, increasing its fibrous component, and its vascularization decreases. That gets very accelerated if the tooth is subjected to deterioration with attrition and abrasion, caries and restorations.

In conservative pulp procedures, such as capping and pulpotomies, abundant cellular proliferation and angiogenesis are required, as well as cellular migration and reorganization.

If the matrix is fibrous, that is a physical obstacle to cellular migration; without abundant vascularization, angiogenesis will not be exuberant and, without a great number of cells, there will not be fast neoformation of the odontoblastic layer. In aged pulps, the prognosis of conservative procedures tends not to be as good as in young teeth that have incomplete apex or have recently finished their root formation.

Final Considerations

The dentin-pulp complex's restorative and reactional capacity is directly related to its functional and morphological characteristics. The dentin-pulp complex modifies itself subtly due to its age, but without affecting, significantly, its reactional and restorative capacity.

Natural aging of both the pulp and the individual expose the dentin-pulp complex to external factors and, in order to preserve its integrity, it activates its defense mechanism (Fig 27). Nonetheless, every time these mechanisms are activated, there will be a little bit more aging. The more they are activated, the faster pulp aging will be.

Some factors caused by improper use of the teeth throughout life considerably accelerate the natural aging process and dramatically modify cellularity, degree of pulp fibrosis and the dimensions of the chamber and the root canal. The modifications related to precocious and accelerated aging of the dentin-pulp complex reduce pulp reactional capacity significantly, even against an aggressor of low intensity and, at the same time, decrease very much the restorative potential when facing conservative procedures, such as pulp capping and pulpotomies.

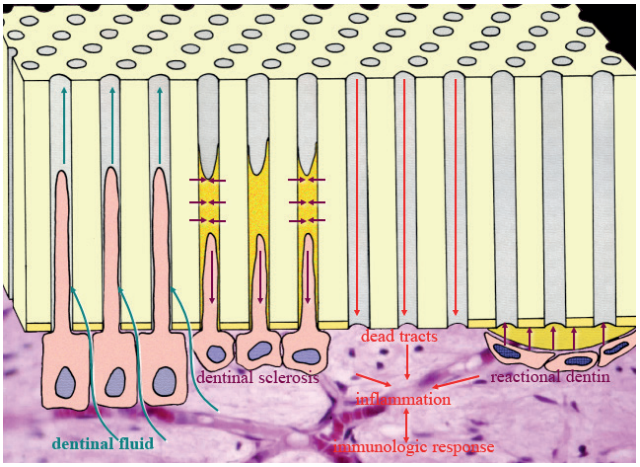

Figure 27: Pulp-dentin defense mechanism schematically represented: when activated, they cause accelerated pulp aging.

Evaluating the morphologic characteristics of the dentin-pulp complex during the planning of surgical and therapeutic procedures is essential for obtaining a higher degree of precision in the prognoses. Many of the aspects mentioned in this text, such as the changes related to precocious and accelerated aging of the pulp complex may be observed directly or indirectly in radiographic and tomographic images.

The cases of pulp calcification must be more accurately diagnosed in radiographic reports and clinical records, by making use of terminology in a more precise way, for there are four clinically and radiographically specific situations and with meaning, as well as clinical and therapeutic implications, which are different and distinct.

The changes due to precocious and accelerated aging of the dentin-pulp complex explain why the teeth tend to enhance their color that stems from the dentin and its thickness. Dentin's thickness minimally and gradually rises with age, but accelerated aging due to improper use intensifies the depositing of dentin, giving the tooth a more intense color shade.

In the attrition areas, with or without bruxism, the prominence of the exposed dentin is due to its natural color, but specifically from the sclerosis that its exposure to the oral environment causes, besides the depositing of reactional dentin in the adjacent pulp region. In those teeth with calcific pulp metamorphosis, likewise, the filling of the pulp space by dysplastic dentin causes intense darkening.

The teeth may be altered with age, but improper use is what makes their aesthetic appearance poor. Odontology must improve its clinical and imaging evaluation criteria regarding mouth aging, especially the teeth. What would aged teeth be from the clinical point of view? How to prevent that from happening? What to do in order to reverse the process? Ivo Pitanguy once stated: 'Before doing any intervention on a patient's face, I ask them to see a dental surgeon. Missing or ugly teeth call people's attention more than a smooth skin free from wrinkles'.

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