Age Changes of Enamel, Dentin, Pulp, Cementum

Dr J. Jananee MDS¹, Dr Deepika .R²

¹Professor, Asan Memorial Dental College & Hospital, Chennalpattu
²CRRI, Asan Memorial Dental College & Hospital, Chennalpattu

Abstract: Age changes of tooth structure are so of so much clinical value for practitioners. Age changes in the different tooth structures play an important role tooth related diseases their treatment and prognosis. So this article enumerates the tooth related changes in various aspects

Keywords: Enamel, Dentin, pulp, cementum

A detailed review of tooth structures and age related changes have been discussed with special emphasis on enamel, dentin, pulp and cementum.

1. Age Changes of Enamel

The most apparent age change in enamel is attrition or wear of the occlusal surfaces and proximal contact points as a result of mastication. This is evidenced by a loss of vertical dimension of the crown and flattening of the proximal contour. The surfaces of unerupted and recently erupted teeth are covered with pronounced rod ends and perikymata. At the points of highest contour of the surfaces these structures soon begin to disappear. This is followed by a generalised loss of the rod ends and flattening of the perikymata. Finally, the perikymata disappear completely.

Structure loss: It depends on the location of the surface of the tooth and location of the tooth in the facial and lingual surfaces lose their structure much more rapidly than proximal surfaces, anterior teeth lose their structure more rapidly than the posterior teeth.

As a result of age changes in the organic portion of enamel, the teeth become darker, and their resistance to decay may be increased, fluid permeability of older teeth is reduced.

2. Age Changes of Dentin

Dead tracts: in dried ground sections of normal dentin the odontoblast processes disintegrate, and the empty tubules are filled with air. They appear black in transmitted and white in reflected light. Odontoblast degeneration is often observed in the area of narrow pulp horn because of crowding of odontoblasts. Again, where reparative dentin seals dentinal tubules at their pulpal ends, dentinal tubules fill with fluid or gaseous substances.

In ground sections such groups of tubules may entrap air and appear black in transmitted and white in reflected light. Dentin areas characterized by degenerated odontoblast processes give rise to dead tracts. Dead tracts are probably the initial step in the formation of sclerotic dentin.

Sclerotic or transparent dentin: in cases of caries, attrition, abrasion, erosion, or cavity preparation, sufficient stimuli are generated to cause collagen fibers and apatite crystals to begin appearing in the dentinal tubules. This condition is prevalent in older individuals.

Apatite crystals are initially only sporadic in a dentinal tubule but gradually the tubule becomes filled with a fire meshwork of crystals. Gradually, the tubule lumen is obliterated with mineral, which appears very much like the peritubular dentin. The reparative indices of dentin in which the tubules are occluded are equalized, and such areas become transparent.

Transparent or sclerotic dentin can be observed in the teeth of elderly people, especially in the roots, it may also be found under slowly progressing caries. Sclerosis reduces the permeability of the dentin and may help prolong pulp vitality.

Features of sclerotic dentin: it is harder than normal dentin, its elastic properties were not altered, but its fracture toughness was reduced. The crystals present in the sclerotic dentin were smaller than those present in the normal dentin. It appears transparent in transmitted light and dark in reflected light.

Reparative dentin: If by extensive abrasion, erosion, caries, or operative procedures the odontoblast processes are exposed or cut, the odontoblasts die or survive, depending on the intensity of the injury. If they survive the dentin that is produced is known as reactionary or regenerative dentin. Those odontoblasts that are killed are replaced by the migration of undifferentiated cells arising in deeper regions of the pulp to the dentin interface. It is believed that the origin of the new odontoblast is from cells in the cell-rich zone. The newly differentiated odontoblasts then begin deposition of reparative dentin.

This action to seal off the zone of injury occurs as a healing process initiated by the pulp, resulting in resolution of the inflammatory process and removal of dead cells, this hard tissue thus formed is best termed reparative dentin although the terms tertiary dentin, response or reactive dentin are also used.
Bacteria living or dead, or their toxic products, as well as chemical substances from restorative materials, migrate down the tubules to the pulp and stimulate pulp response, leading to reparative dentin formation.

**Vitality of dentin:** Since the odontoblast and its process are an integral part of the dentin, there is no doubt that dentin is a vital tissue. Again, if vitality is understood to be the capacity of the tissue to react to physiologic and pathologic stimuli, dentin must be considered a vital tissue.

Dentin is laid down throughout life, although after the teeth have erupted and have been functioning for a short time, dentinogenesis slows, and further dentin formation is at a much slower rate. Pathologic effects of dental caries, abrasion, attrition or the cutting of dentin of operative procedures cause changes in dentin. These are described as the development of dead tracts, sclerosis, and the addition of reparative dentin.

**3. Age Changes of Pulp**

**Pulp stones (denticles):** pulp stones, or denticles, are nodular, calcified masses appearing in either or both the coronal and root portions of the pulp organ. They often develop in teeth that appear to be quite normal in other respects. They usually are asymptomatic unless they impinge on nerves or blood vessels. They have been in functional as well as embedded unerupted teeth.

Pulp stones are calcified, according to their structure as true denticles or false denticles. True denticles are similar in structure to dentin in that they have dental tubules and contain the processes of the odontoblasts that formed them, usually located close to the apical foramen.

False denticles do not exhibit dentinal tubules but appear instead as concentric layers of calcified tissue. They appear within a bundle of collagen fibers. In the center of these concentric layers of calcified tissue there may be remnants of necrotic and calcification of thrombin in blood vessels, called phleboliths, may also serve as idi for the false denticles.

**Fibrosis:** in the aging pulp accumulations of both diffuse fibrillar components as well as bundles of collagen fibers usually appear. Fiber bundles may appear arranged longitudinally in bundles in the radicular pulp, and in a random more diffuse arrangement in the coronal area.

The increase in fibers in the pulp organ is gradual and is generalised throughout the organ.

**Vascular Changes:** vascular changes occur in the aging pulp organ as they do in any organ. Atherosclerotic plaques may appear in pulp vessels. In other cases the outer diameter of vessel walls become greater as collagen fibers increase in the medial and adventitial layers. Calcification in the walls of blood vessels is found most often in the region near the apical foramen.

The capillary endothelium shows changes due to age. The endothelium in the elderly shows numerous pinocytic vesicles, microvesicles and microfilaments.in addition lipid like vacuoles, glycogen granules and many golgi complexes are present. Blood flow decreases with age.

**Cell Changes:** In addition to the appearance of fewer cells in the aging pulp, the cells are characterized by a decrease in size and number of cytoplasmic organelles. The typical active pulpal fibrocyte or fibroblast has abundant rough-surfaced endoplasmic reticulum, notable Golgi complex, and numerous mitochondria with well-developed cristae. The fibroblasts in the aging pulp exhibit less perinuclear cytoplasm and possess long, thin cytoplasmic processes. The intracellular organelles are reduced in number and size; the mitochondria and endoplasmic reticulum are good examples of this.

**4. Age Changes of Cementum**

**Hypercementosis:** It is an abnormal thickening of the cementum. May be generalised or localised, diffused or limited. Hypercementosis is termed cementum hypertrophy if the overgrowth improves the functional qualities of the cementum and is termed cementum hyperplasia if it is not correlated with increased function.

**Cementicles:** they are ovoid or round calcified structure that are formed as a result of calcification of the degenerated periodontal tissue or the epithelial rests of Malssez. Cementicles may be,

1) Free in the periodontal ligament
2) Attached to the cementum
3) Embedded in the cementum

**Permeability:** the permeability of cementum decreases gradually by age. The permeability from the periodontal side is lost except in the most recently formed layer of cementum, while that from the dentin side remains only in the apical region.

**Cementum Resorption and Repair:** cementum resorption can occur after trauma or excessive occlusal forces. After the resorption ceases the damage is usually repaired. If the repair establishes the former outline of the root surface it is called anatomic repair. However if only a thin layer of cementum is deposited and the root outline is not constructed it is called functional repair.

**5. Conclusion**

Depending on age changes treatmet mortality also changes. So the future dentists should have an clear knowledge of the above discussed chapters.

**References**
