Oral Biology
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Lecture - 03
Tooth and it's Supporting Structures - Part 1

Hi everyone today's topic is Tooth and its Supporting Structures.

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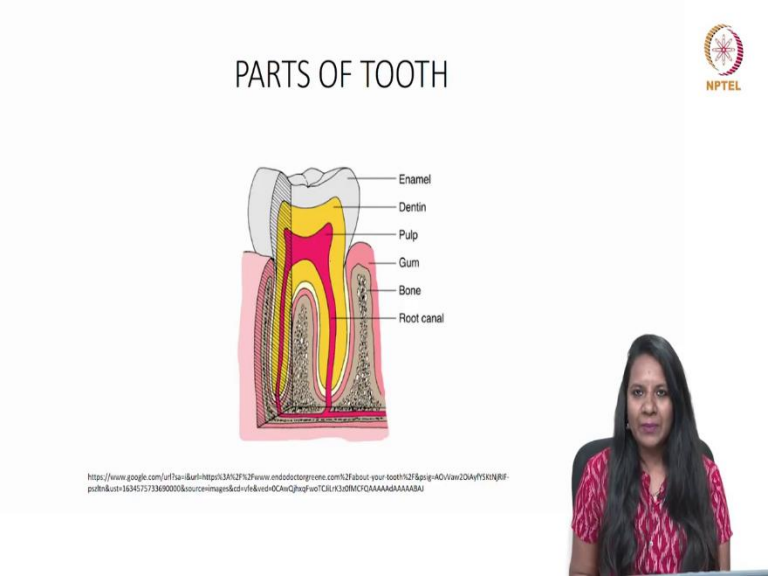

Tooth consist of enamel, dentin, pulp, cementum and supporting structures alveolar bone and periodontal ligament.

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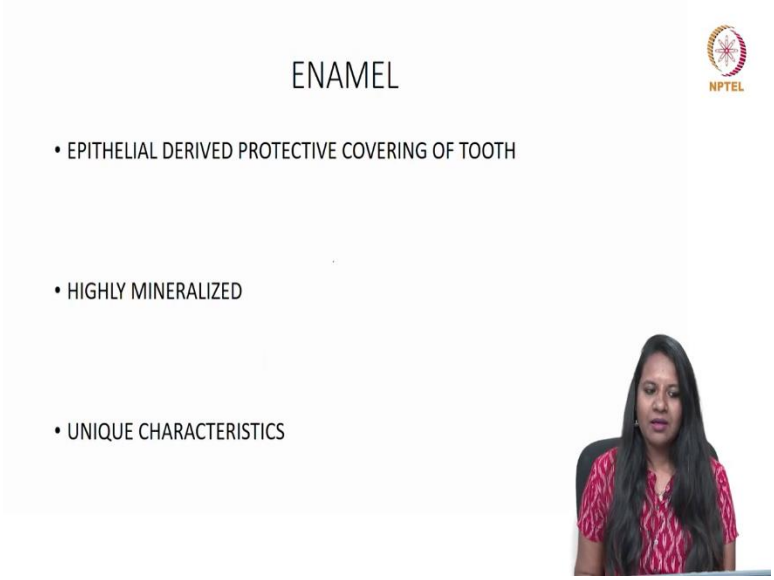

Moving to enamel; enamel is an epithelially derived protective covering of tooth and it is the most highly mineralized tissue in the body. It is unique because once it is formed it is not formed again in the lifetime. he enamel forming cells that is the ameloblast or lost once the tooth erupts into the oral cavity and it will not renew.

And this highly mineralized tissue which is formed by the matrix proteins, once the enamel has been formed the entire organic matrix were removed as bulk. So, these are the two features why enamel is unique.

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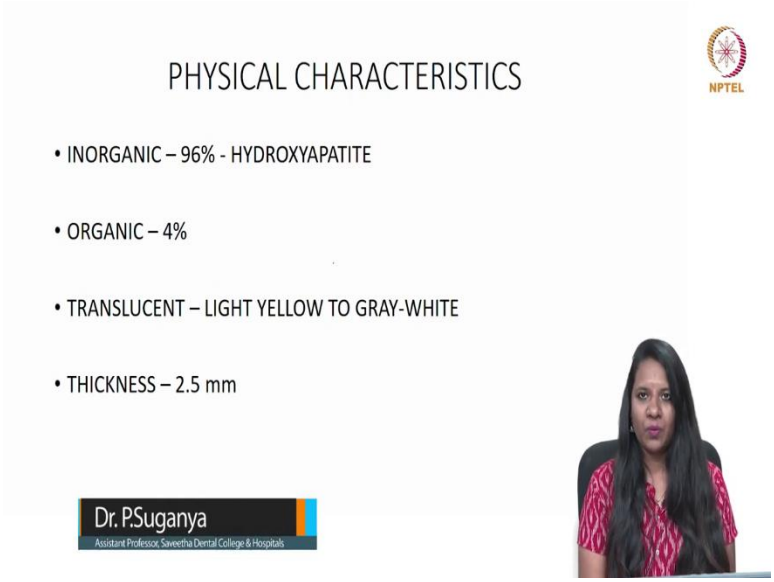

Moving to the physical characteristics; the enamel consists of 96 percent of inorganic which comprises of hydroxyapatite crystals that is calcium, phosphate and carbonate ions. And traces of

selenium lead and fluoride were formed if these elements were present during the formation of an enamel and consist of 4 percent of organic material.

Dissolution of these ions leads to the formation of dental caries. This provides the chemical basis for the dental caries. And this high mineralized tissue helps the enamel to withstand heavy occlusal forces during function and the color varies from light yellow to gray white, it is generally translucent. The thickness varies from 2.5 mm on the occlusal aspect to the feather edge or the cervical area.

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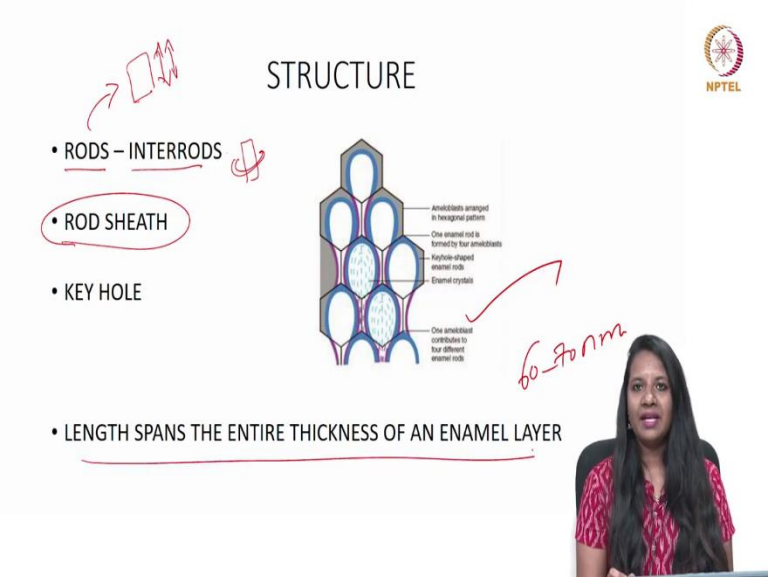

So, the structure is very important, the fundamental organization of an enamel is composed of rod, inter rod which is surrounded by rod sheath. Rods will be cylindrical in shape with crystals arranged parallel to the long axis of the rod whereas inter rods surround the rod and the orientation of crystals will be different than that of the longitudinal axis of the rods.

So, the rods and inter rods together will give a keyhole appearance. So, enamel comprises of a long ribbon like crystals which will be 60 to 70 nanometer in length, that is the length of the length spans the entire thickness of an enamel layer.

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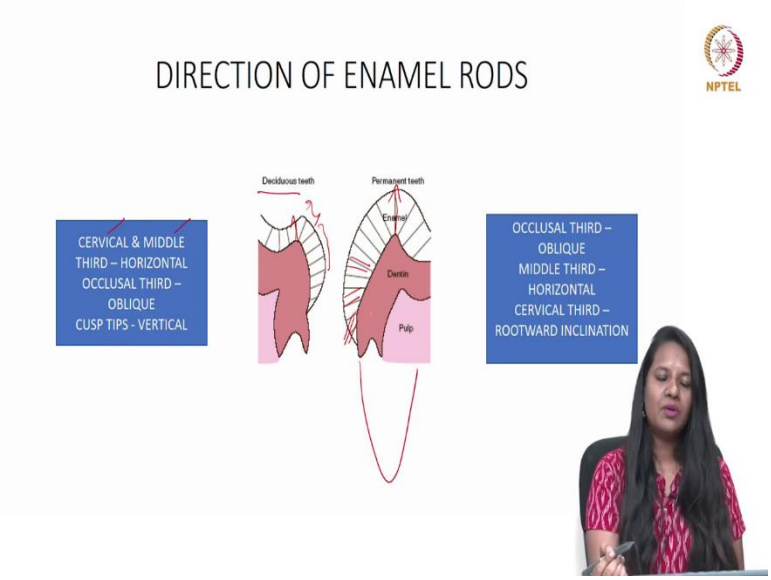

Moving to the direction of enamel rods, how these enamel rods were present in the deciduous and in the permanent teeth. So, in the deciduous teeth, in the cervical and middle third it will be horizontal ok and it will be oblique in the occlusal third and in the cusp tips it is almost vertical whereas, moving to the permanent teeth in the cusp tip it is again vertical and in the middle third it is horizontal and in the cervical it will incline towards the root. So, the rods will be inclined towards the root in the permanent teeth.

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Moving to amelogenesis. Amelogenesis is nothing but the formation of enamel. It will occur in two processes. The first is the initial mineralization where it is mineralized only up to 30 percent then it is mineralized to 96 percent by the concurrent accretion of the minerals.

Ameloblasts have a unique life cycle which is characterized by progressive phenotype change; that is, it will have its own primary activity throughout each phase of the cycle and they secrete matrix proteins that provides an environment favorable to mineral deposition.

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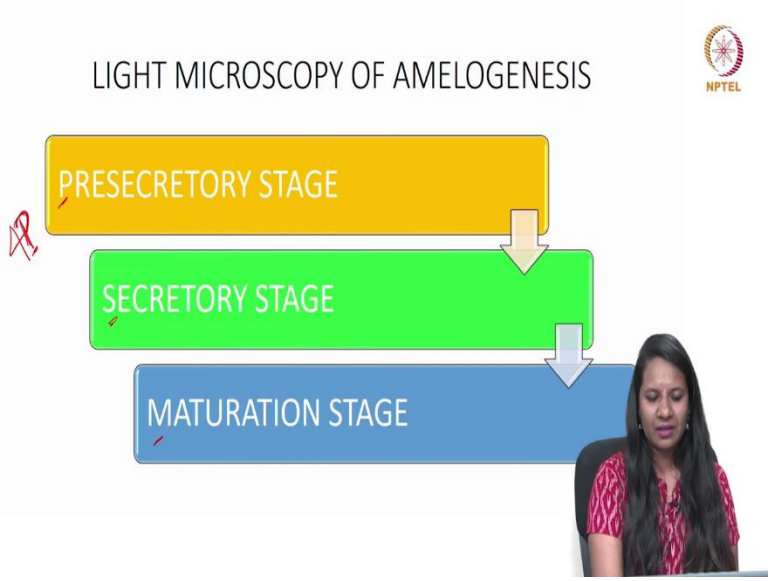

Moving to the light microscopy of amelogenesis, which is broadly classified into presecretory, secretory and maturation stage. In the presecretory remember 4 Ps that is the ameloblast will acquire phenotype, change polarity and it has high protein synthetic apparatus and it will permit or it will prepare the ameloblast to secrete the organic matrix.

In the secretory stage it will elaborate and organize the highly ordered tissue and in the maturative stage it will modulate and transfer ions, so that concurrent accretion of mineral will occur.

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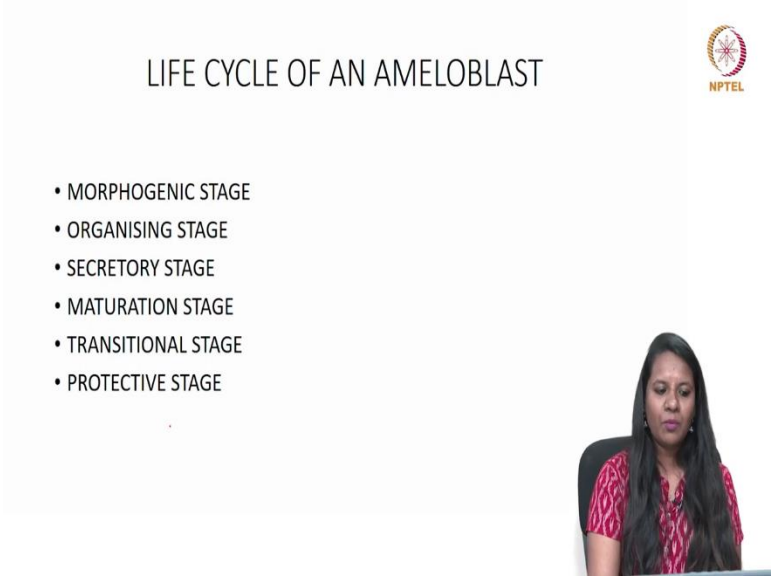

The life cycle of an ameloblast consists of morphogenic stage, organizing, secretory, maturation, transitional and protective.

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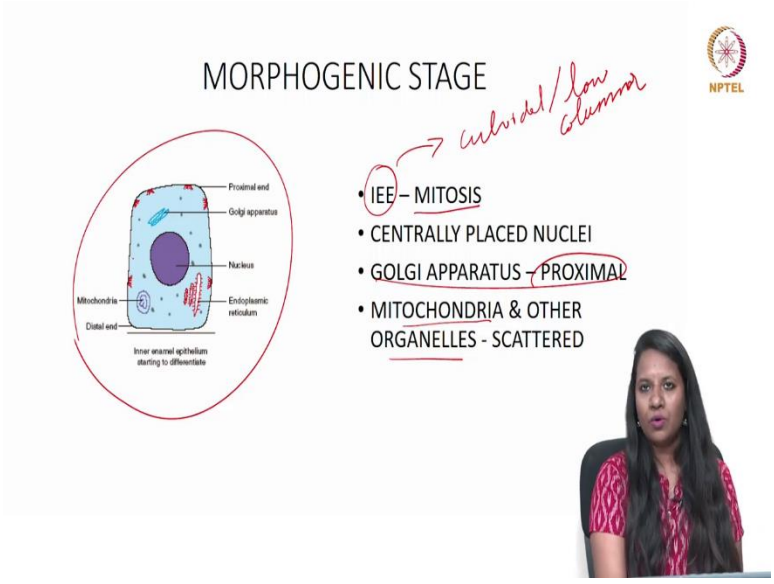

We will go through each stage the first being the morphogenic stage; that is the inner enamel epithelial cells will be either cuboidal or it will be low columnar with a centrally placed nuclei and it will undergo mitosis. Golgi apparatus will be seen in the proximal area and mitochondria and other organelles will be scattered. Here this is the picture of your morphogenic stage ameloblast where the Golgi apparatus were in the proximal area and the other organelles were just scattered.

Look at the size of the ameloblast which is either cuboidal or low columnar.

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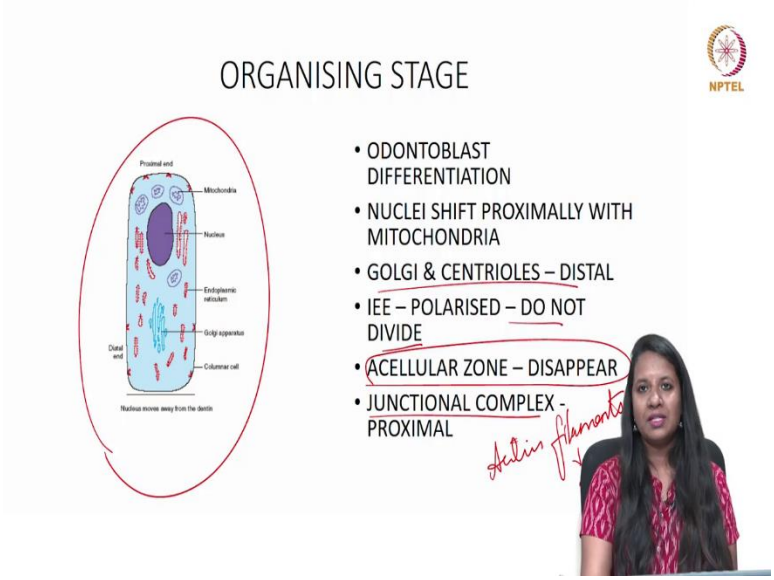

Moving to the organizing stage. Now you can see the difference in the size of the cell. So, the nuclei will shift proximally with mitochondria, Golgi and centrioles will be present on the distal side. The inner enamel epithelium will be polarized that is it will not divide after this. And there will be formation of odontoblast differentiation that is, as soon as the inner enamel epithelial cells polarized, it will interact with the dental papilla cells and differentiate into odontoblast.

Thus, a cellular zone which is present will disappear at this process. Then these ameloblasts will come closer together by the formation of a specialized structures called junctional complexes. It will be present both in the distal and the proximal end; namely distal junctional complex and proximal junctional complex.

And these junctional complexes will have something called actin filaments which will radiate from here to the cytoplasm of the ameloblast and then it will form something called terminal web. The purpose of this terminal web is that it makes the ameloblast remain attached together till the formation of an enamel. And this also decides what is to go in and what to go out of an enamel during its formation.

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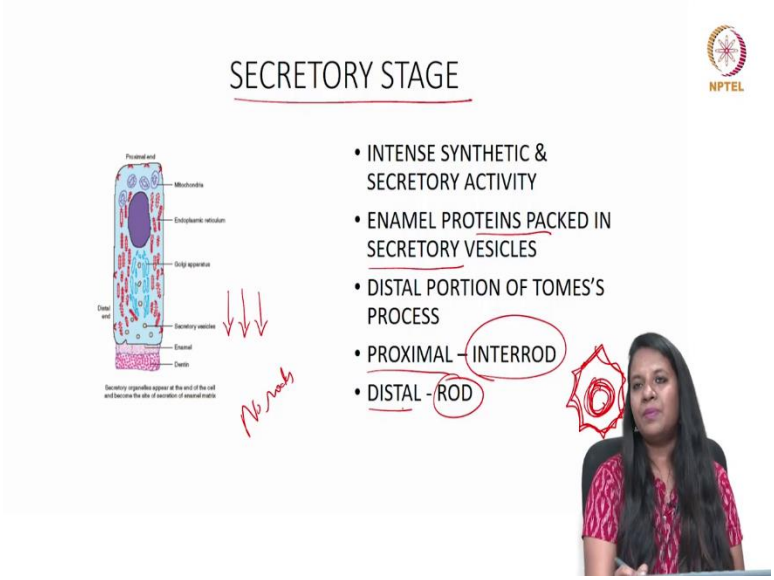

Moving to the secretory stage. As the name suggests, it reflects intense synthetic and secretory activity. So, the enamel proteins were packed in secretory vesicles. And they have been thrown out into the distal tombs process and it will lay down enamel. The first formed enamel will not have any rods or interrod.

It is the first formed enamel which is formed against the dentin and the whole ameloblast will move away once the enamel has been formed. And the proximal surface will lead to the formation of something called enamel partition; that is interrods.

The distal part will form rods, which will be filling in & the outer layer is the partitions which will be formed by the proximal and the inner pit will be filled by the distal, contributing to the rod and interrod.

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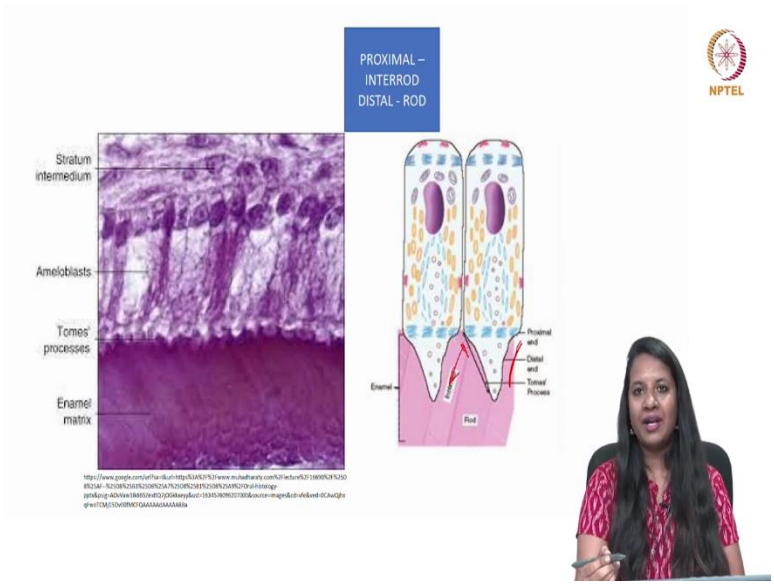

So, this picture clearly depicts the interrod from the proximal surface and the rod from the distal surface. So, inter rod is formed ahead of rod.

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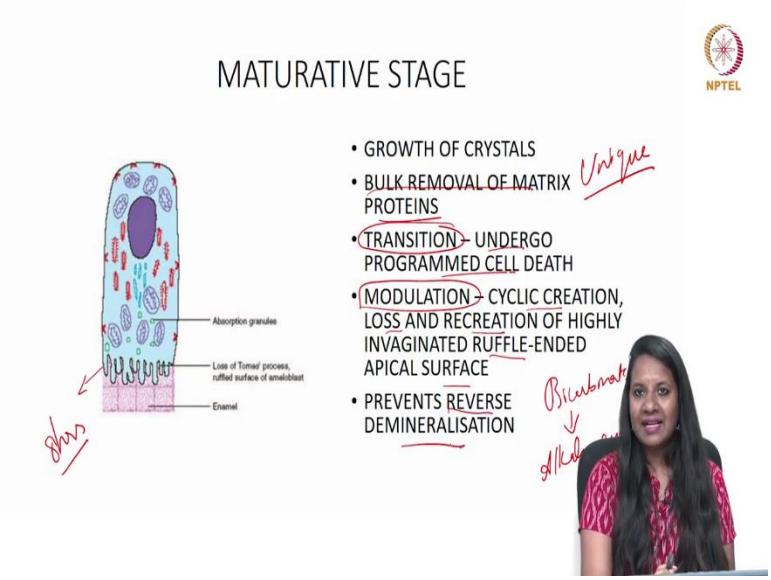

Moving to the maturative stage. Maturative stage is nothing but the growth of the preexisting crystals. So, with when the growth has been occurred there will be a bulk removal of matrix proteins. As I have already mentioned this is the unique feature of an enamel that is after the whole mineralization the maturation occurs by the prolonging the width of the crystals and removing of the matrix protein.

And there is a stage called transition where the ameloblast will reduce in size and the organelles will also be reduced and undergoes programmed cell death. That is 50 percent of ameloblast will undergo programmed cell death during its transitional stage and other 50 percent during the final maturative stage.

There is a special feature called modulation. Modulation is nothing but cyclic creation loss and recreation of highly invaginated ruffle ended apical surface. That is the ameloblast will be changing from the ruffle ended to smooth ended which will occur once in 8 hours.

Due to the formation of the enamel, that is due to the formation of more minerals, acidification occurs. So, this ruffle ended will secrete bicarbonate ions, which will alkalize the enamel fluid, thus preventing reverse demineralization. So, modulation is one of the main important processes which occurs within the ameloblast, to maintain the alkalic nature of the enamel fluid, to prevent reverse demineralization.

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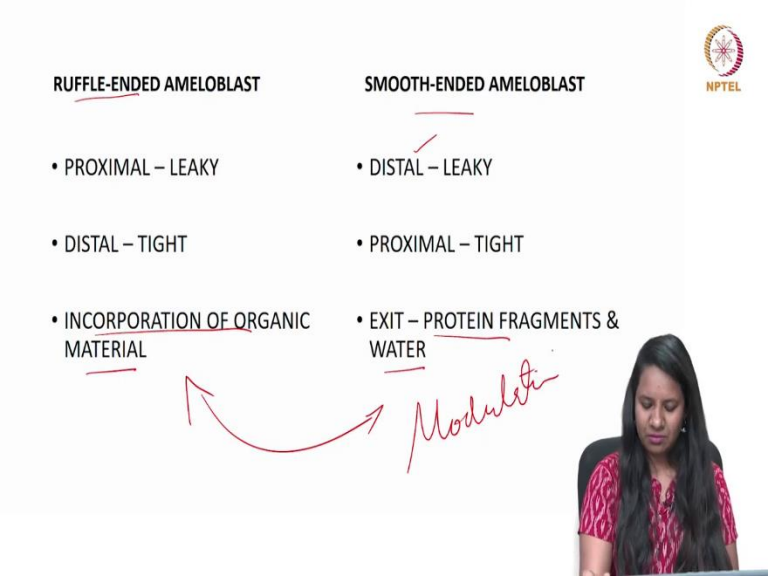

Moving to the differences between ruffle ended and smooth ended ameloblast. In the ruffle ended, the leaky region is the proximal surface whereas, in the smooth ended the leaky is the distal surface. And the tight is the distal in case of ruffle ended and the tight is the proximal in case of smooth ended. Ruffle ended leading to incorporation of organic material and smooth ended will help in the exit of protein fragments and water.

And the change of ruffle ended to smooth ended and smooth ended to ruffle ended is known as modulation.

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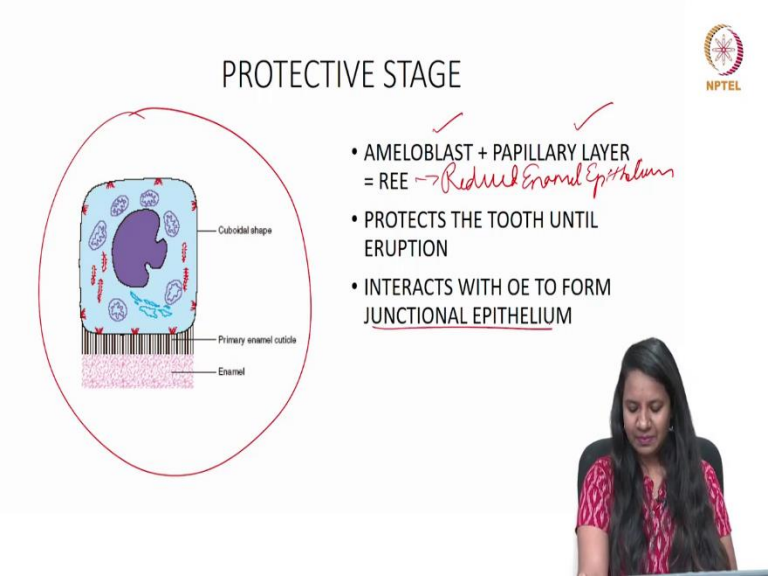

Moving to the final stage that is the protective stage. In the protective stage the ameloblast is reduced in size and these ameloblast along with the papillary layer combine to form reduced enamel epithelium. As the name suggest the ameloblast reduce in size. It is protected until the

tooth erupts into the oral cavity and once it is erupted into the oral cavity it interacts with the oral epithelium to form junctional epithelium.

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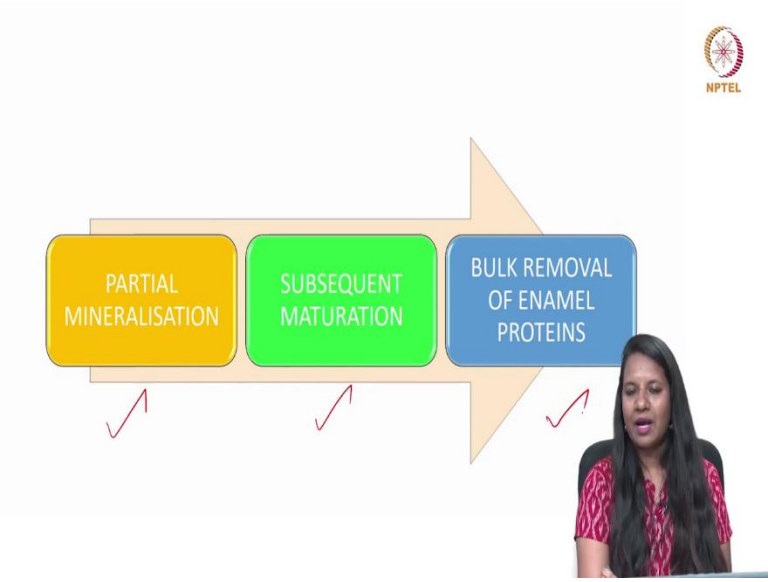

Hence the amelogenesis is nothing but initial partial mineralization and subsequent maturation with the increase in the width of the crystals with bulk removal of enamel proteins.

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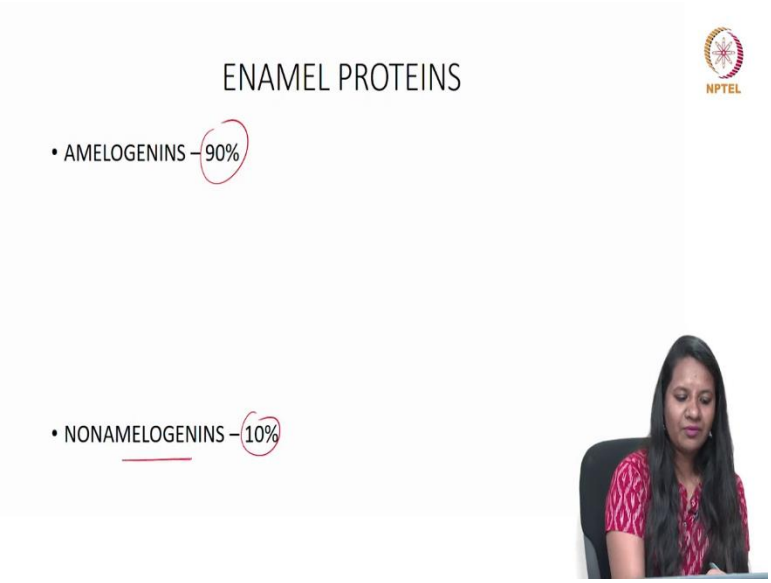

Enamel proteins which play a main role in the formation of an enamel, consist of 90 percent of amelogenin and 10 percent of nonamelogenin.

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Enamelysin, Kallikrein, Amelotin and Apin were few of the nonamelogenin where enamelysin belongs to matrix metalloprotease family and the it helps in the short-term processing of newly secreted matrix proteins. And kallikrein 4 belong to serine protease family and it is the bulk digestive enzyme and helps to remove the organic matrix.

Then comes the amelotin and apin. As the name starts with 'A', it is present in the apical surface of modulating ameloblast and hence it helps in the adhesion of enamel organ to the ameloblast. Everything starts with **A** and it is easy to remember **A**melotin, **A**pin which is present in the apical surface of modulating **A**menoblasts and it is helps in the **A**dhesion of enamel organ to the ameloblast.

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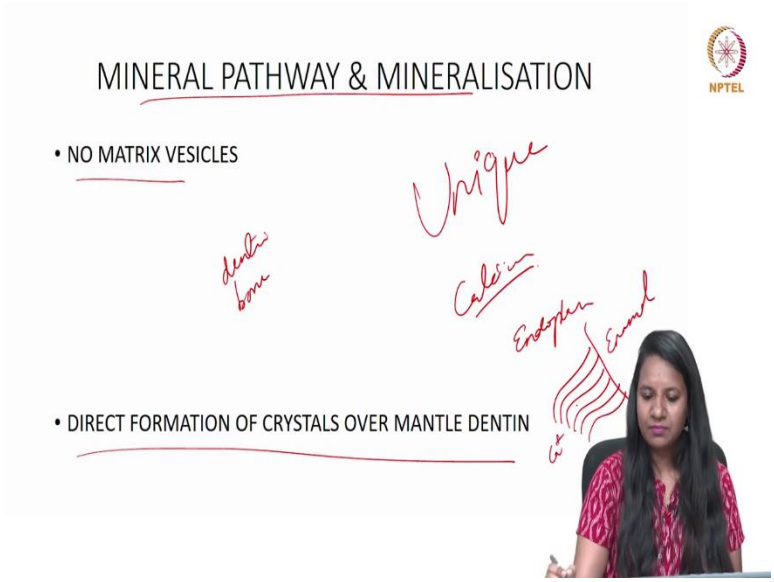

The mineral pathway and mineralization is very unique, because it does not have matrix vesicles, which plays main role in the mineralization and maturation of collagen based calcified structures such as dentin or bone. So, there is no equivalent formation of predentin or osteoid here in enamel.

It is just the direct formation of crystals over mantle dentin. So, then how come the calcium just pass through the enamel. It is passed through the enamel with the help of endoplasmic reticulum and there will be smooth sinusoidal pores through which the calcium gets into the enamel surface.

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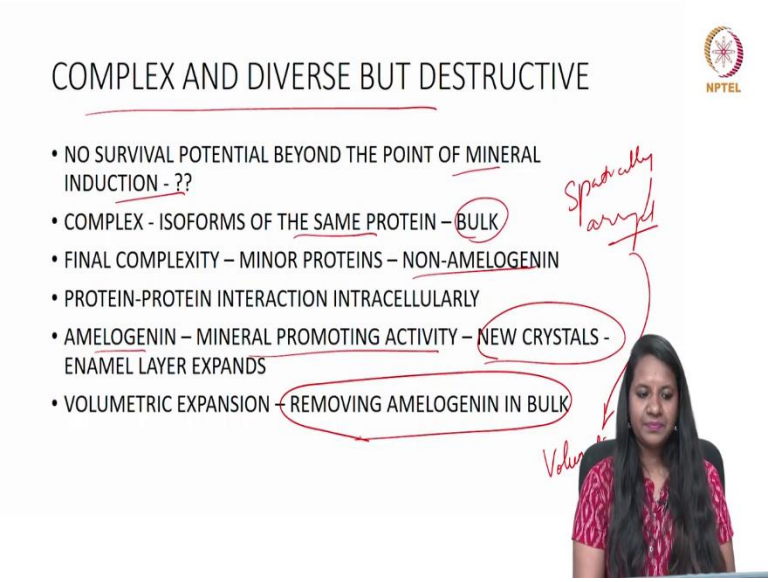

Finally, the enamel formation is something complex and diverse and also destructive. Why ameloblast should spend so much time in forming as highly mineralized structure with so much complexity, but there is no survival potential beyond the point of mineral induction, is still remains a puzzle enamel structure itself is complex?

Because it is formed by the isoforms of the same protein which leads to the bulk of the enamel. The final complexity was formed by the nonamelogenin. So, it is mainly the protein protein interaction intracellularly. And there is no equivalent of predentin or osteoid were form, it is just protein protein interaction.

Then it forms new crystals which leads to the expansion of the enamel layer. Once the crystals were spatially arranged the final expansion occurs called volumetric expansion, which occurs after removing this amelogenin in bulk which has a mineral promoting activity.

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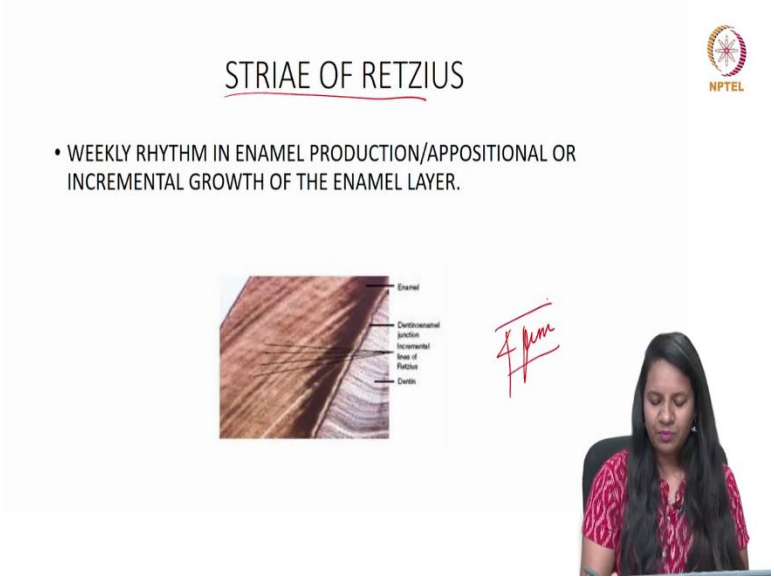

Now, moving to the histological structures that is present in the enamel. The first one being the striae of retzius; otherwise called incremental layer of retzius which is nothing but the weekly rhythm in enamel production or appositional or incremental growth of the enamel layer. So, the distance between each striae is 4 micrometer.

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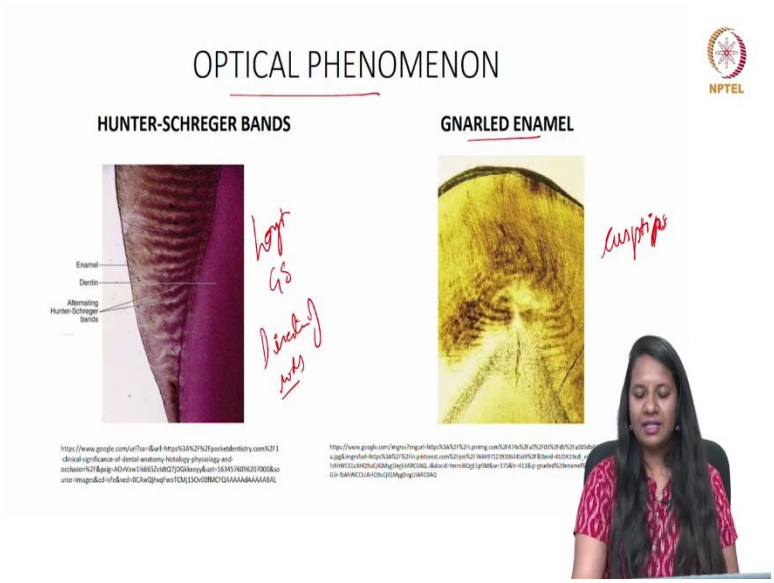

Next is the optical phenomenon and the first one being the Hunter Schreger bands. It is seen in the longitudinal ground section. And it is nothing but the alternate light and dark bands under incident illumination which can be reversed by altering the incident illumination and it is due to the change in the direction of rods. And next is the gnarled enamel which will be seen in the cusp tips where the enamel rods will twist and have a complex arrangement.

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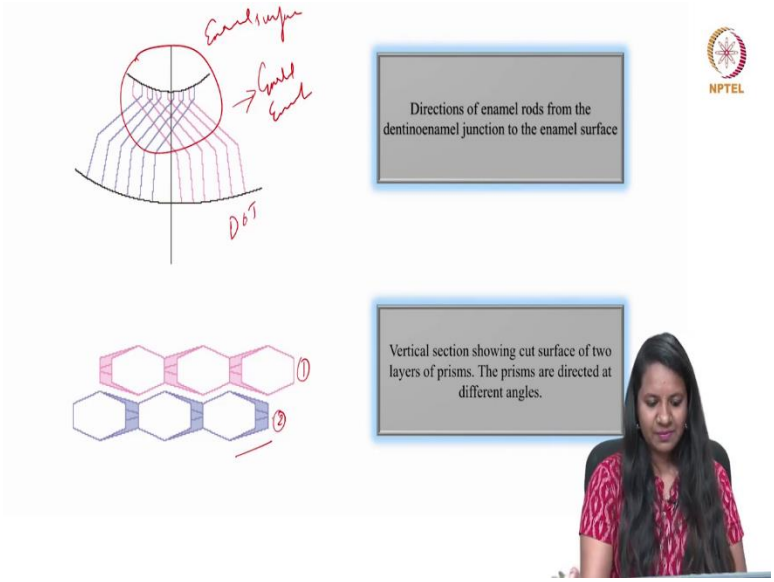

This picture depicts the direction of enamel rods from the DEJ to the enamel surface. And at the surface it is getting twisted up and produce a complex structure. So, when we see it under the microscope will give an optical phenomenon, called gnarled enamel. And the other picture shows cut surface of two layer of prism directed at different angles.

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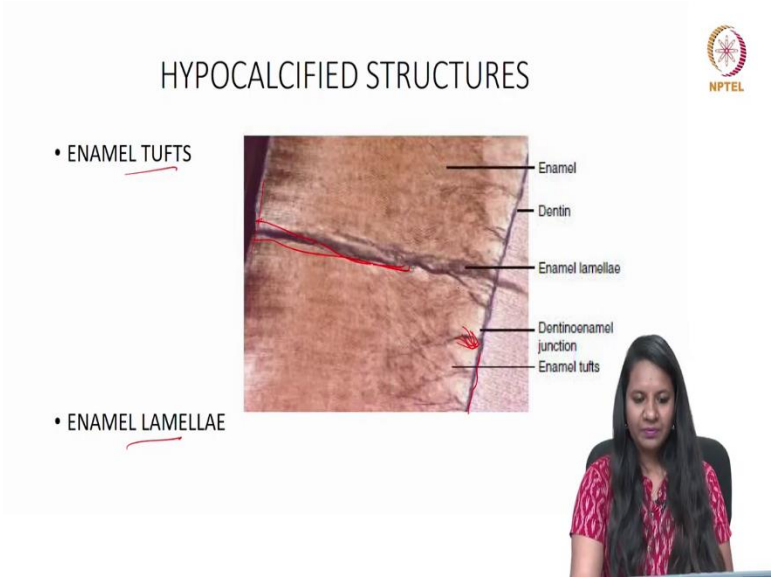

Moving to the hypocalcified structures called enamel tuft and enamel lamellae. Enamel tuft will raise from the DEJ to the enamel surface and it will have an appearance of tuft like grasses whereas, enamel lamellae are cracks which appear from the enamel surface to the DEJ.

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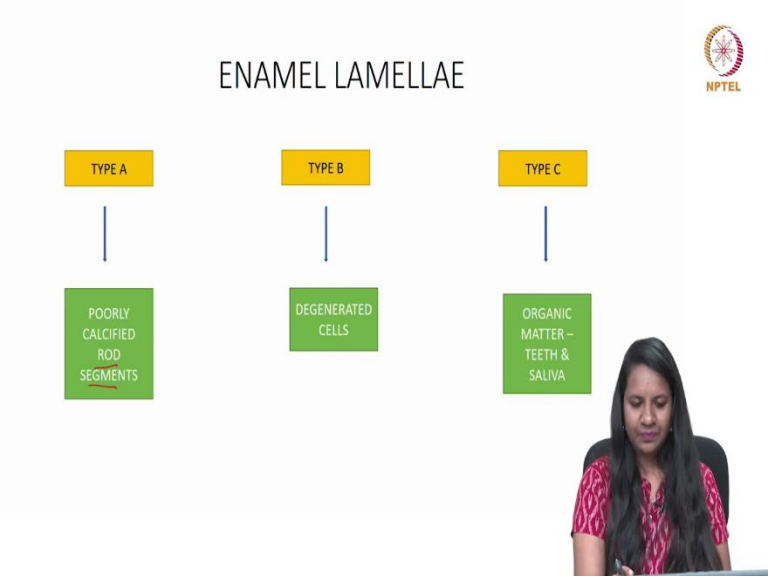

Enamel lamellae are of three types; type A, B and C. The type A consists of poorly calcified rod segments in the crack whereas, type B consists of degenerated cells and type C consists of organic matter which is derived from either tooth or saliva. So, it differs by the presence of content in the crack.

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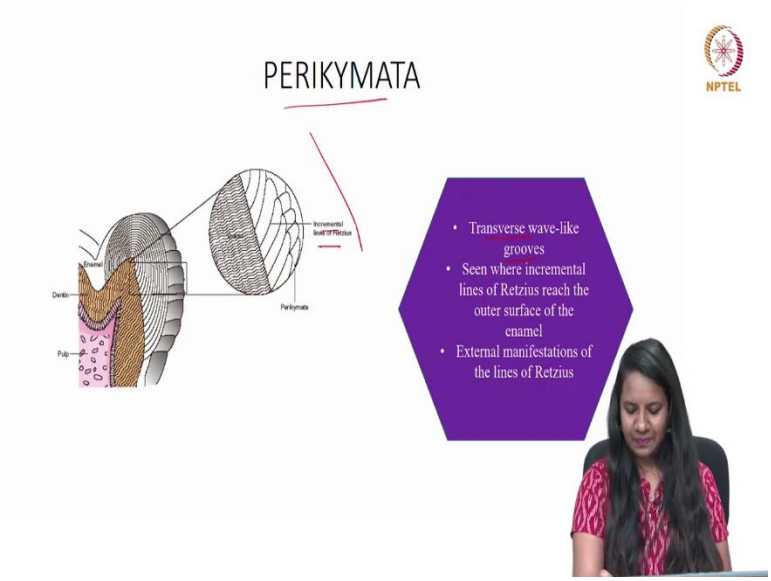

Perikymata is nothing but transverse wave like groups. It is seen where incremental line of Retzius reach the outer surface of enamel and it is called as external manifestation of striae of Retzius.

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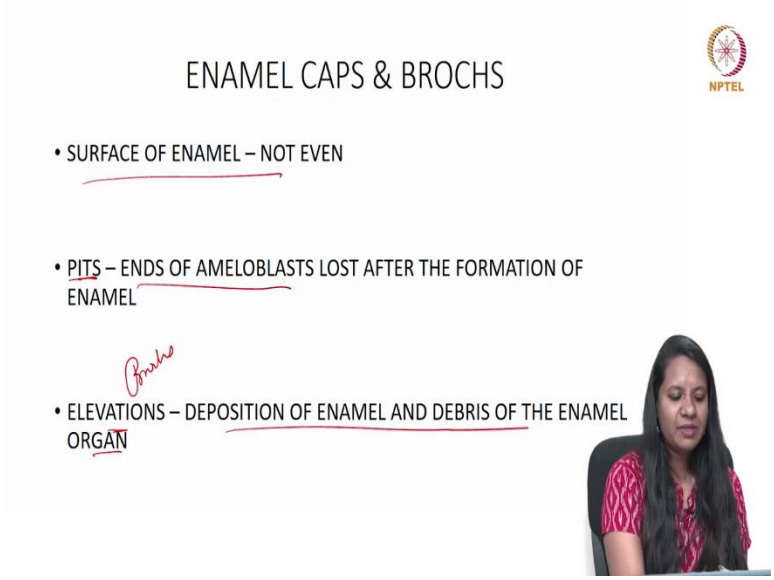

Moving to enamel caps and brochs, the surface of an enamel is not even. So, it will have a dip and an elevation. So, dip is nothing but pit and elevation is nothing but broch. Pit represents the lost ends of ameloblast after the formation of an enamel. Then elevation is due to the deposition of enamel and debris of the enamel organ is added. So, the surface of enamel is not smooth, it will be either pit or there will be either elevation.

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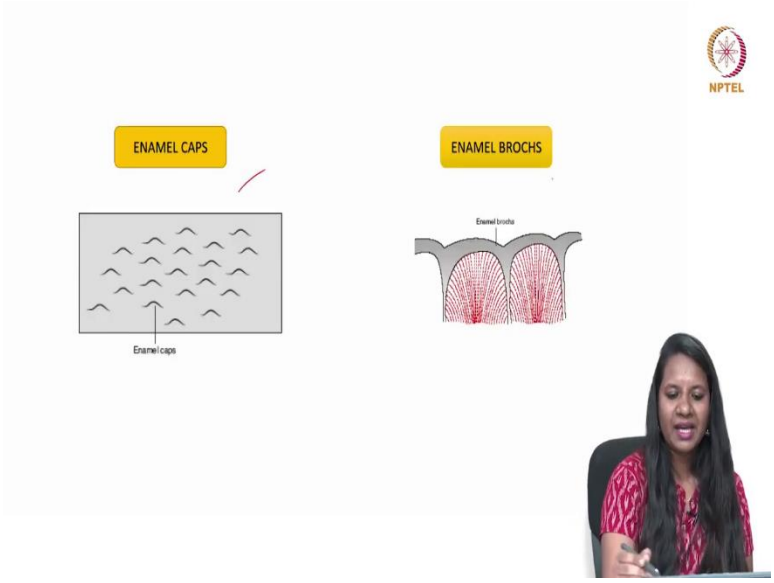

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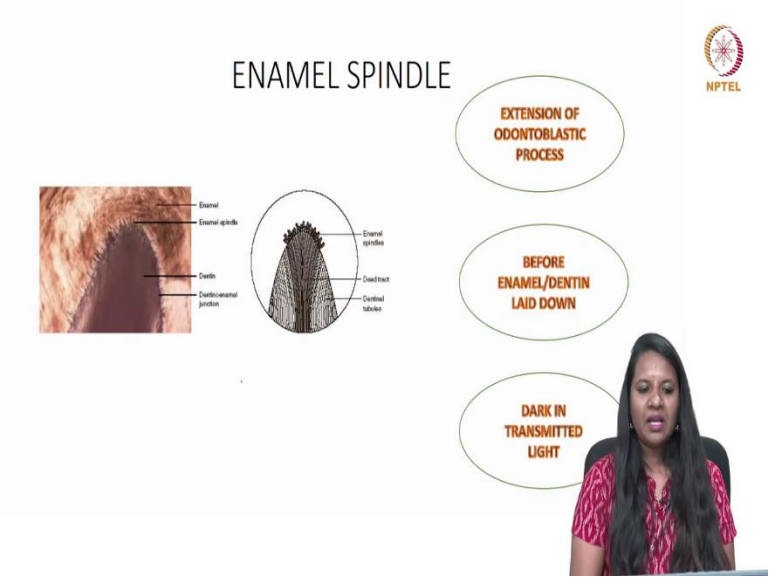

Enamel spindle is nothing but the extension of odontoblastic process to the enamel surface before enamel or dentin is laid down and it will be dark in transmitted light. So, here in this picture, numerous enamel spindles were noticed that is the odontoblastic process extending towards the enamel surface.

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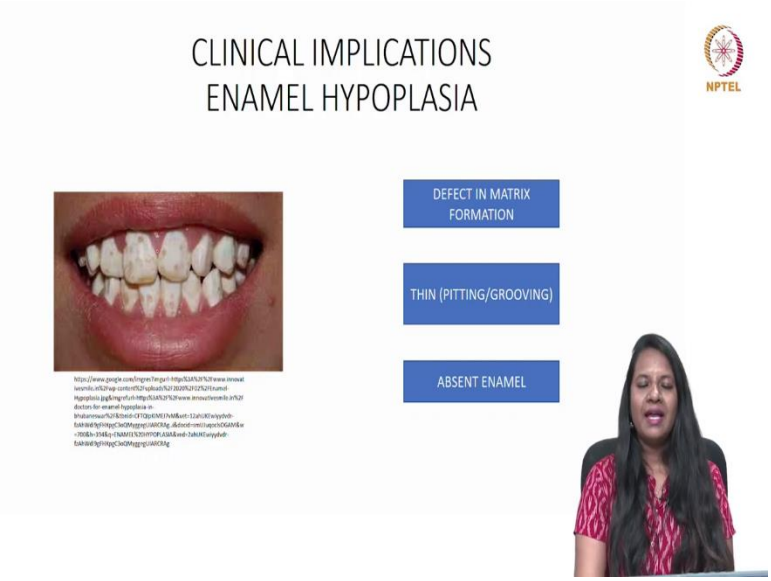

Moving to the clinical implications; Enamel hypoplasia is a defect in the matrix formation. So, there will be either thin enamel, either with pitting or grooving or there will be total absence of enamel. This picture shows pitting of enamel that is the brownish pits all over the enamel.

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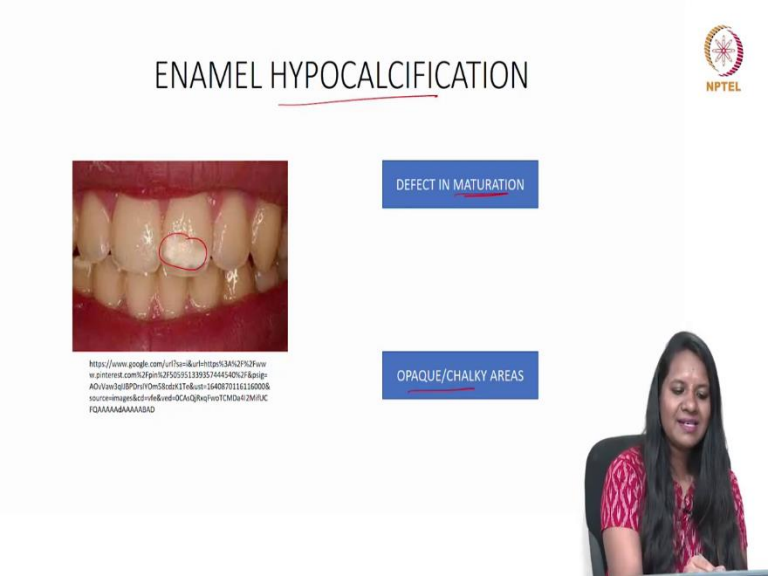

Enamel hypocalcification is a defect in the maturation. So, this type of enamel will appear opaque or chalky areas as given in this picture.

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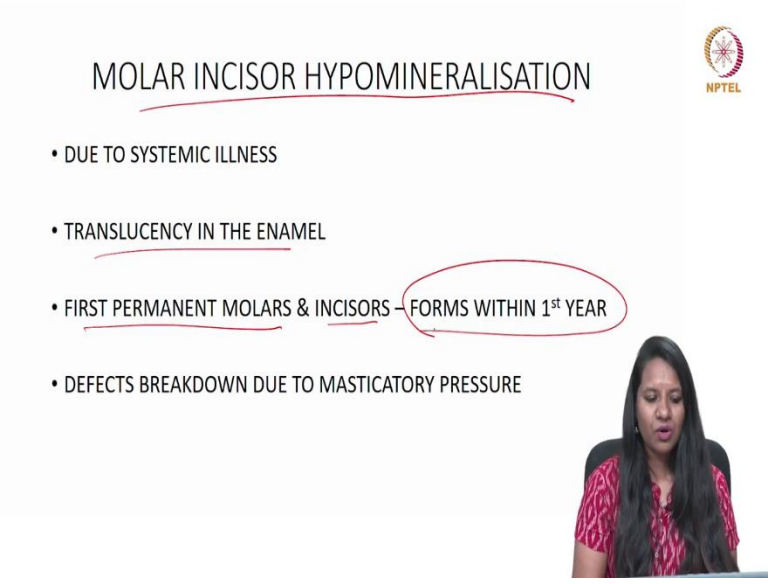

Moving to molar incisor hypo mineralization which will occur due to systemic illness during the formation of an enamel, and it will affect mostly the first permanent molar and incisor that is formed within the first year. So, if any kid within the first year of life have any systemic illness, then this condition can be more common. So, this defect will break down due to the masticatory pressure, since the enamel is very soft. So, patient will have a complaint of sensitivity.

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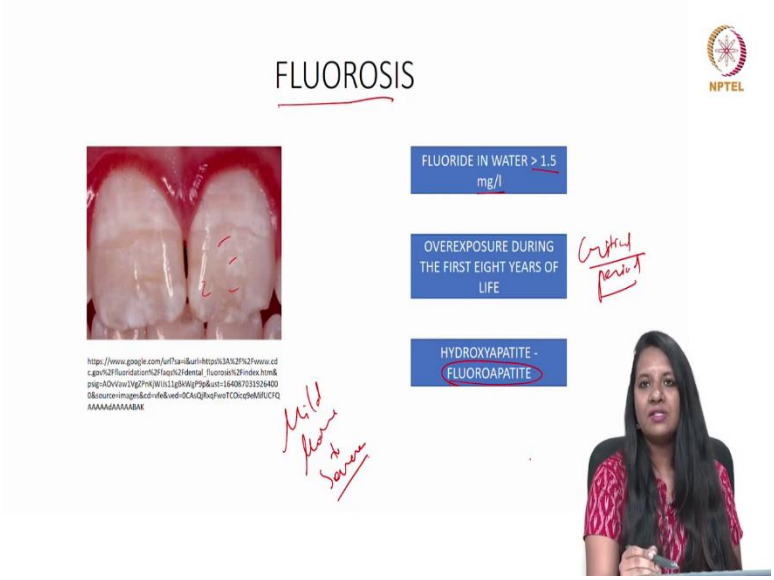

Fluorosis occurs when the fluoride in water exceeds more than 1.5 milligram per liter, there will be formation of fluorapatite rather than hydroxyapatite. Then the fluorosis will appear such as white patches or brownish patches all over the teeth. So, it will vary from mild, moderate to severe. The critical period is the first 8 years. So, when the kid is over exposed to fluoride during the first 8 years of life might have higher chances of getting fluorosis.

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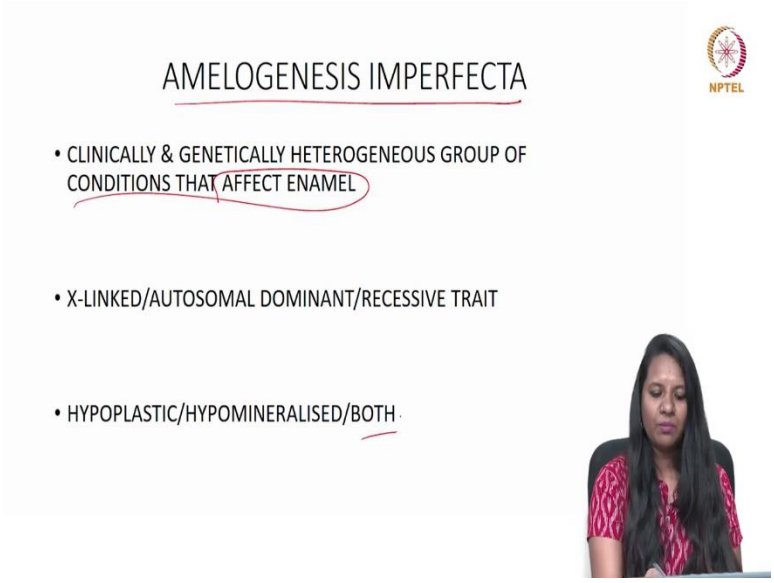

Amelogenesis imperfecta; very common term, it is nothing, but clinically and genetically heterogeneous group of conditions that affect enamel. It could be either X-linked or dominant or recessive trait. So, the trait could be hypoplastic or hypo mineralized or it might have both.

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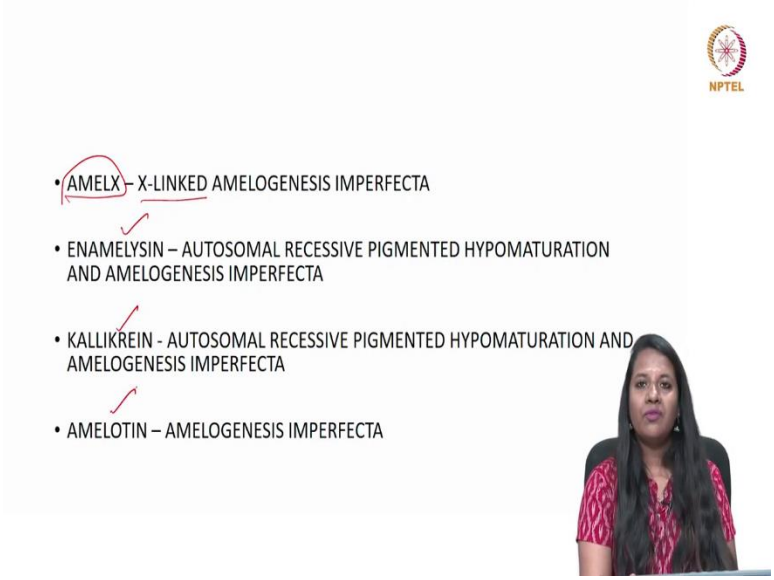

A defect in amelx leads to X-linked amelogenesis imperfecta and defect in enamelysin and kallikrein will have autosomal recessive pigmented hypo maturation amelogenesis imperfecta. And defect in amelotin will also leads to formation of a genetic disease named amelogenesis imperfecta.

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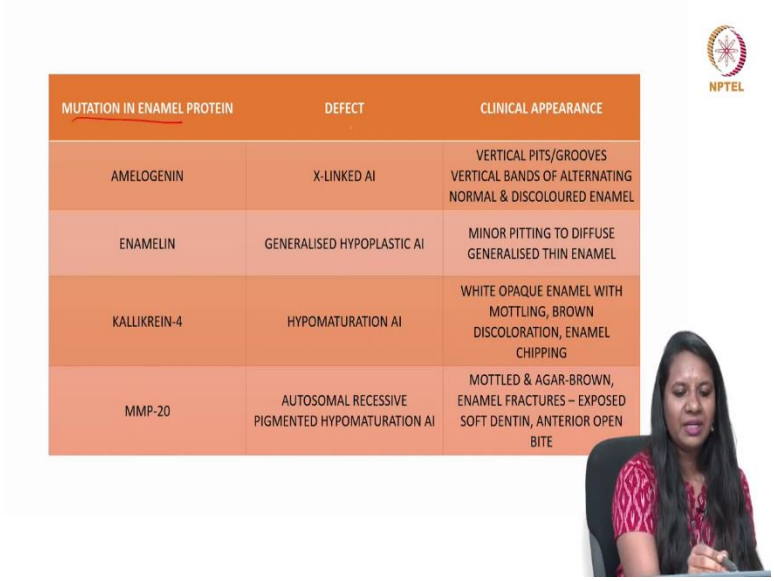

This table clearly depicts the mutation in enamel protein defect and their clinical appearance. Amelogenin leads to X-linked amelogenesis imperfecta and the teeth will have vertical pits and grooves, and there will be vertical bands of alternating normal and abnormal enamel. Enamelin, leads to generalized hypoplastic amelogenesis imperfecta, where there will be minor pitting to diffuse generalized thin enamel.

Kallikrein 4 leads to hypo maturation amelogenesis imperfecta where the enamel will be white opaque with mottling or they have brownish discoloration and there will be regular chances of enamel chipping. MMP-20 that is matrix metalloprotease 20 which also helps in the formation of enamel. And defect in this leads to autosomal recessive pigmented hypomaturation amelogenesis imperfecta. So, it will be mottled and characteristically have an agar brown appearance, the enamel fractures exposing the soft dentin. So, there will be higher chances of sensitivity and one unique feature is these patients will have anterior open bite.

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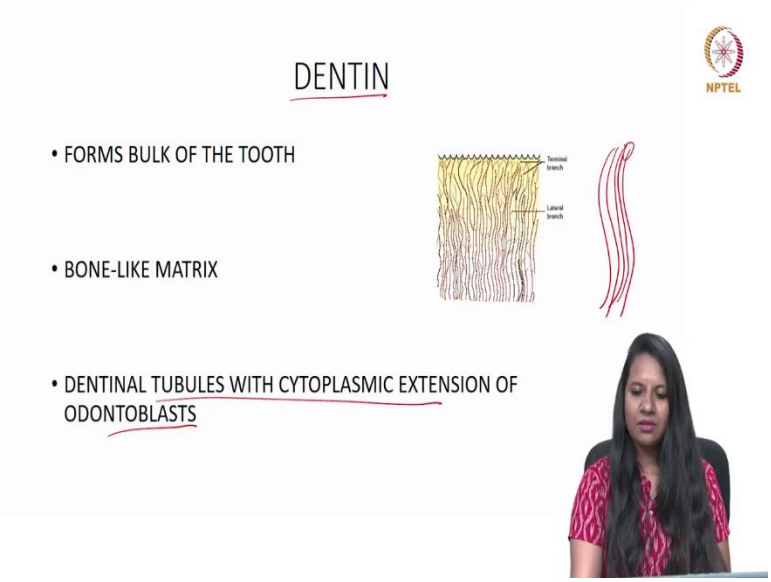

Dentin forms the bulk of the tooth. It is bone like matrix which has odontoblast traversed throughout its matrix and dentinal tubules with cytoplasmic extensions of the odontoblast were present. And it is it almost have a S-shaped or sigmoid shaped dentinal tubules into which the odontoblast will be traversing throughout the length.

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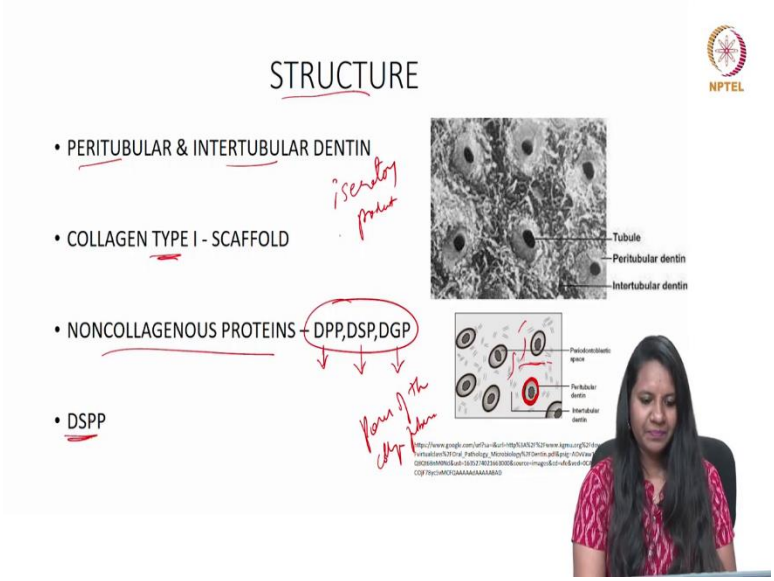

Dentin consists of 70 percent of inorganic 20 percent of organic and 10 percent of water. It is yellowish in color. The structure of dentin consists of peritubular dentin and intertubular dentin. As the name suggests peritubular dentin, delimits the tubule and it is highly calcified matrix whereas the intertubular dentin is the one which is present in between the peritubular dentin and this forms the bulk of the dentin and this is the primary secretory product. And it is formed in between the collagen fibrils that is mainly type 1. So, this type 1 collagen act as a scaffold upon which this intertubular dentin is laid down.

And there are a few noncollagenous proteins such as dentin phosphoprotein, dentin sialoprotein and dentin glycoprotein which helps in forming dentin thus closing the pores of the collagen fibers. All three will be depicted as a single molecule in the genome level that is dentin sialophosphoprotein.

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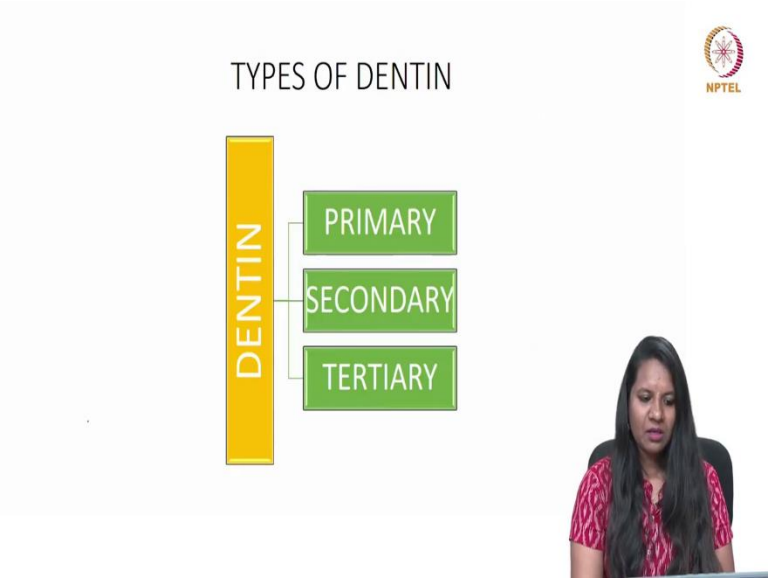

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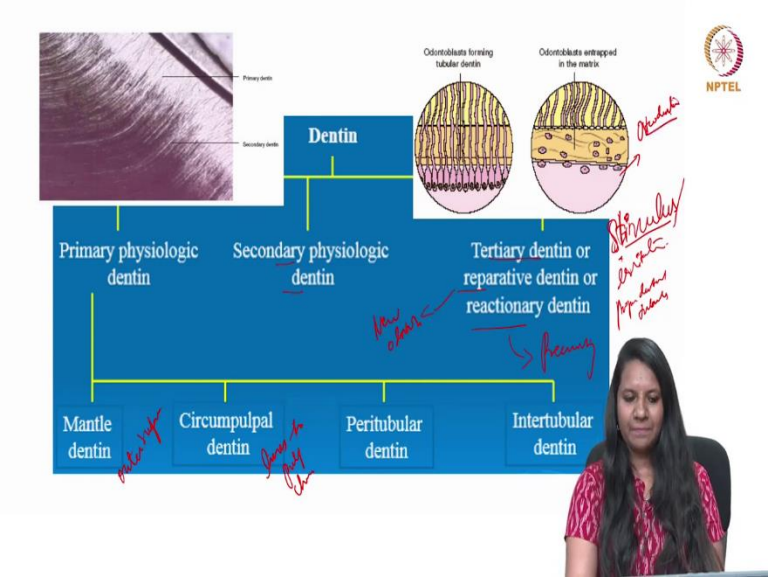

Types of dentin includes primary, secondary and tertiary. Under primary, circumpulpal dentin lines the pulp chamber and mantle dentin form the outer surface of coronal dentin. And secondary dentin is formed after the root formation, deposition is much slower when compared to the primary dentin.

And tertiary dentin is formed only when there is stimulus or irritation; such as a caries or any restorative material. So, this type of dentin will not have a proper dentinal tubule. It might be either sparse or it is totally absent.

And when the such type of dentin is formed from the preexisting odontoblast it is called as reactionary. And when it is formed from the new odontoblast it is called as reparative. And when these odontoblasts while secreting the dentin will get embedded in the dentinal matrix and such type of dentin is called as osteodentin.

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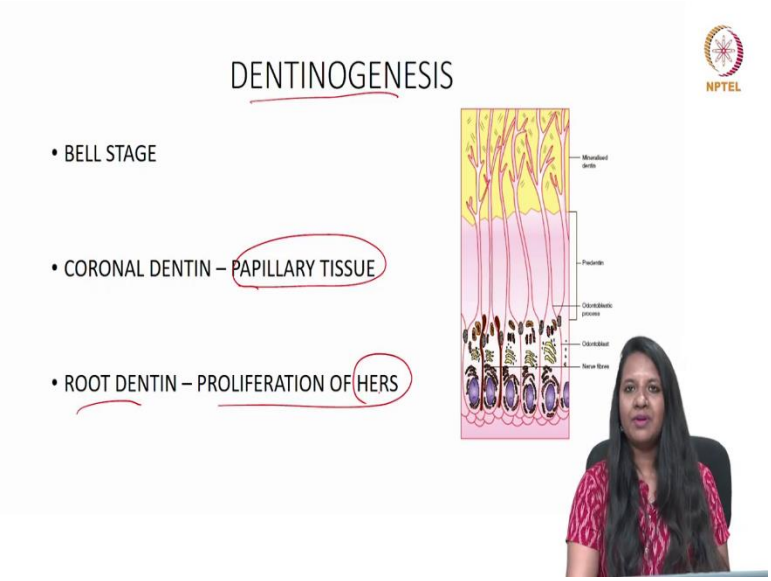

Dentinogenesis is the formation of dentin. So, it usually occurs in the bell stage. Once the inner enamel epithelial cells reach its polarity, these papillary tissues that is the dental papilla tissues will start differentiating into odontoblast and starts forming dentin. And root dentin needs proliferation of hertwig's epithelial root sheath from the cervical loop area.

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The first sign of dentin formation is the presence of large diameter fibrils, which is nothing but Von Korffs fibers. And then the dentin is laid down over the fibers by the odontoblast and they have matrix vesicles which is absent in case of enamel. Since these were collagen based calcified tissues, they all mineralize with the help of a matrix vesicle.

The matrix vesicle mineral phase starts as a single crystal which will grow inside it then break the confinement and it will fuse with the adjacent crystals to form a homogeneous mass to form dentin.

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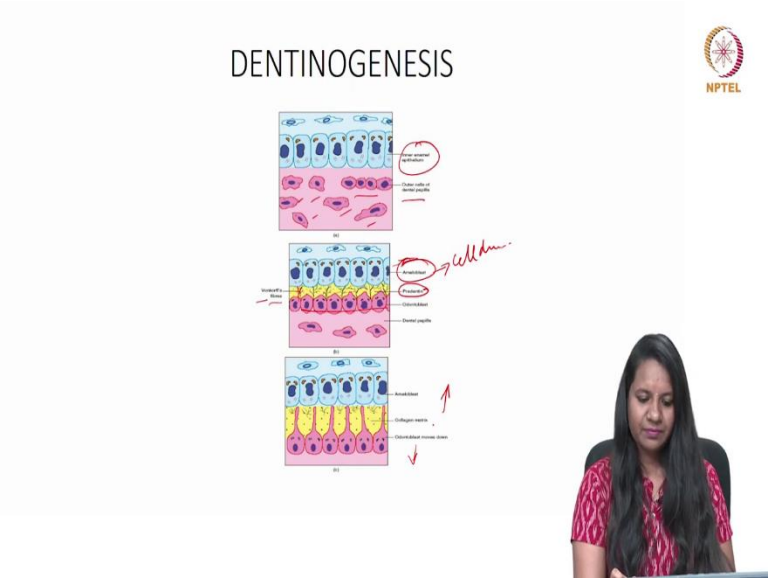

This picture shows the formation of dentin. The dental papilla cells were organized and differentiated into odontoblasts once inner enamel epithelium reach polarity. The formation of Von Korff fibers occur immediately and the odontoblast will start laying down dentin. The first form dentin is predentin. Later the odontoblast moves down as the dentin has been formed.

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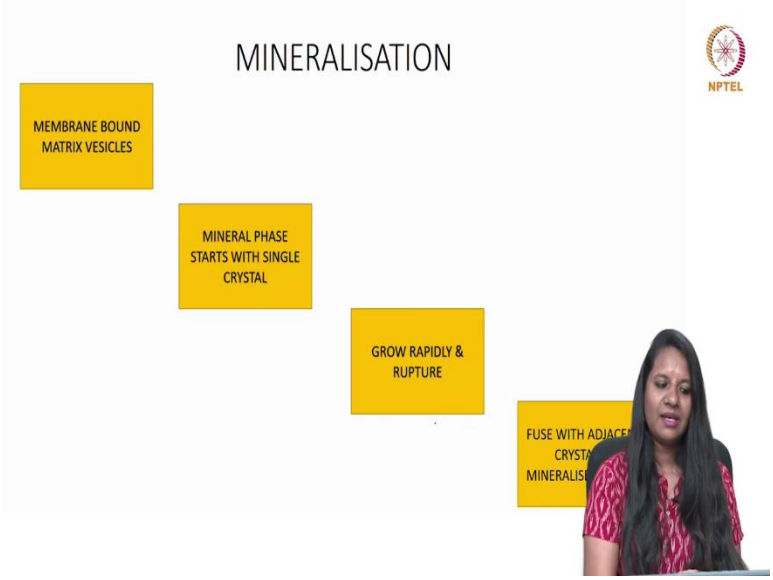

The mineralization starts with the help of membrane bound matrix vesicles, and the mineral phase starts with a single crystal which will grow rapidly and rupture the confinement of the matrix vesicle it will fuse with the adjacent crystals to form a homogeneous mineralized matrix.

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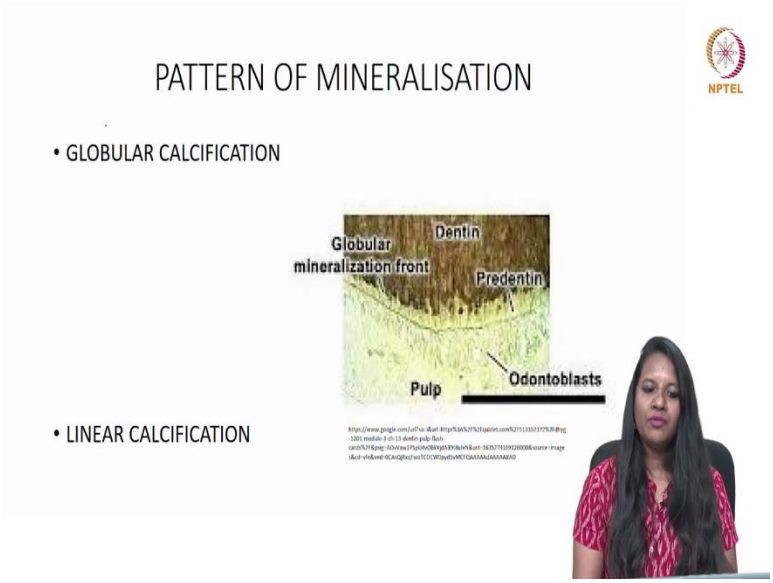

Pattern of mineralization could be either globular or linear. Globular calcification is nothing but the globules were fused together to form an homogeneous mass whereas, when the pattern of calcification is slowed down or when it is organized, it is called as linear calcification.

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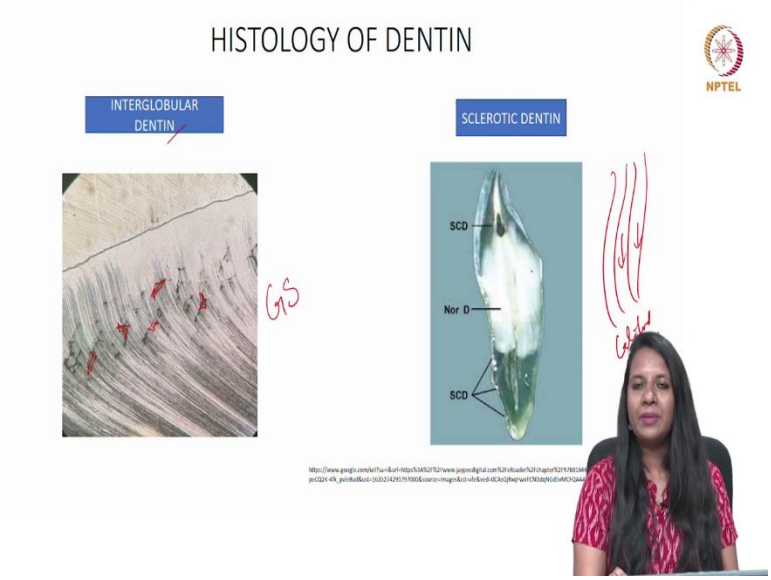

Moving to the histological aspects of dentin, that is, the structures we can see under microscope. Interglobular dentin which can be seen in the ground section when the globules fail to fuse and form a homogeneous mass and will appear black in transmitted light. The sclerotic dentin is a transparent dentin where the dentinal tubules were filled with calcified material. It will appear sclerotic and it will most commonly occur in the old age people.

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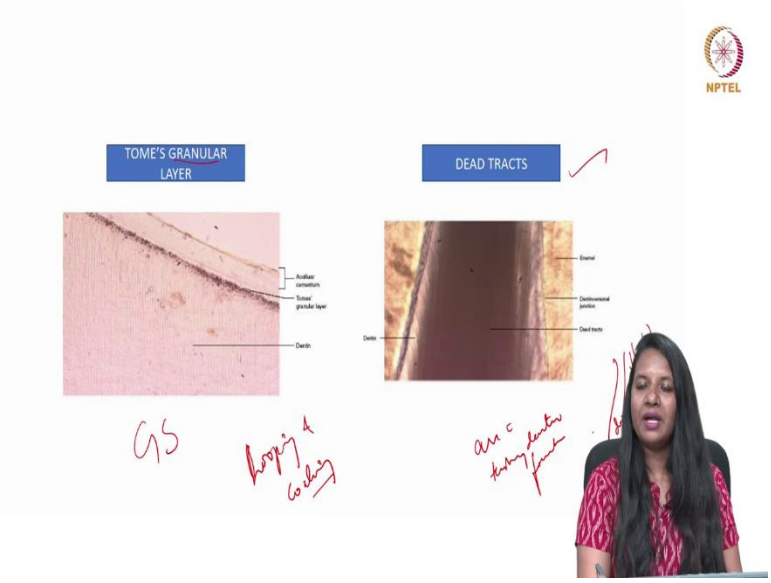

Tome's granular layer is the looping and coalescing of terminal branches of dentinal tubules that is seen in ground section as granular layer; hence it is termed as tomes granular layer.

Dead tracts occur due to the occlusion of dentinal tubules by any calcified material leading to the dyeing of the odontoblast present there. So, it will appear black in transmitted light. Mainly it is

associated with tertiary dentin formation; that is when there is any stimulus the odontoblast coalesces and die.

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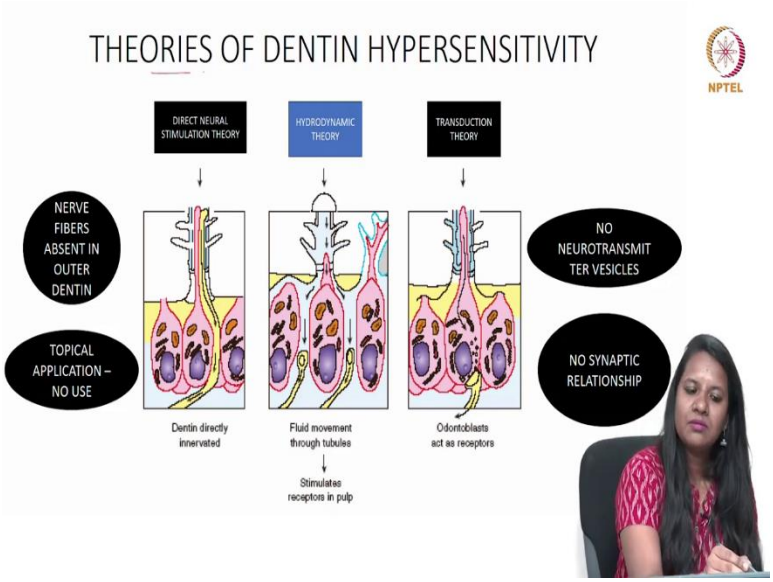

Dentin hypersensitivity is one of the main important symptoms which many patients will come across. So, dentin is sensitive because it is exposed. Many theories have been formulated to explain the dentin hypersensitivity.

The first one being the direct neural stimulation which suggests that the nerve fibers which is present along with the odontoblast, carry the pain sensation and thus it is depicting it as dentin hypersensitivity. This theory is rejected, because the nerve fibers were absent in the outer dentin and when there is topical application of analgesics, it does not provide any symptomatic relief.

The hydrodynamic theory is widely accepted theory that states that the dentinal fluid inside the tubules stimulate receptors in pulp leading to dentin hypersensitivity. The transduction theory states that odontoblast act as a receptor and it will simulate the pain. But there is no neurotransmitter vesicles in the odontoblast and there is no synaptic relationship between the odontoblast and the nerve endings to perceive it as a pain. So, the only accepted theory is the hydrodynamic theory which states that the dentinal fluid movement is responsible for the receptor stimulation in the pulp.

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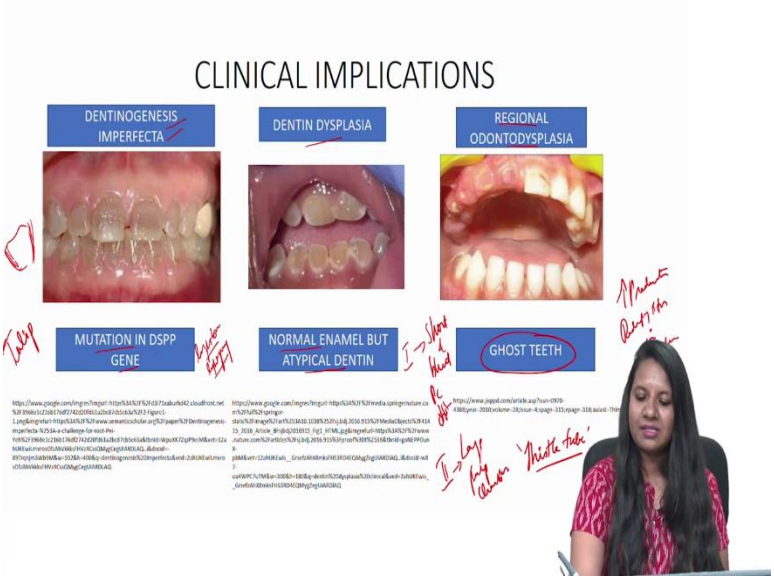

As amelogenesis imperfecta had defect in enamel, Dentinogenesis imperfecta have defect in dentin which is due to the mutation in the DSPP gene. It will affect both deciduous and the permanent teeth. So, it will have a light brown to light gray in color and it appears like a tulip. That is the crown will be broader with much increased cervical constriction.

Dentin dysplasia is another defect with normal enamel, but atypical dentin. It is of two types; in type 1, the roots were short and blunt and the pulp chambers were obliterated whereas in type 2, it will have large pulp chambers and radiographically there will be an appearance of thistle tube. The roots are said to have thistle tube appearance.

Regional odonto dysplasia, otherwise known as ghost teeth, which leads to the retarder eruption of most of the tooth. There will be normal enamel, but increased predentin with a greater number of radiopaque structures in the pulp chamber and there will be presence of interglobular dentin also as there is a defect in the fusing of the globules of minerals.