DEVELOPMENT OF TOOTH

CONTENTS

6. Hertwigs’ Epithelial Root Sheath & Root formation
7. Amelogenesis
8. Dentinogenesis
9.Cementogenesis
10. Developmental disorders of Teeth
11.Agents affecting tooth development
12.Clinical Application and coclusion

INTRODUCTION

- OVERVIEW OF DENTAL TISSUES

- Enamel,
- Dentin,
- DEJ,
- Pulp,
- CEJ
- Cementum,
- Alveolar bone,.

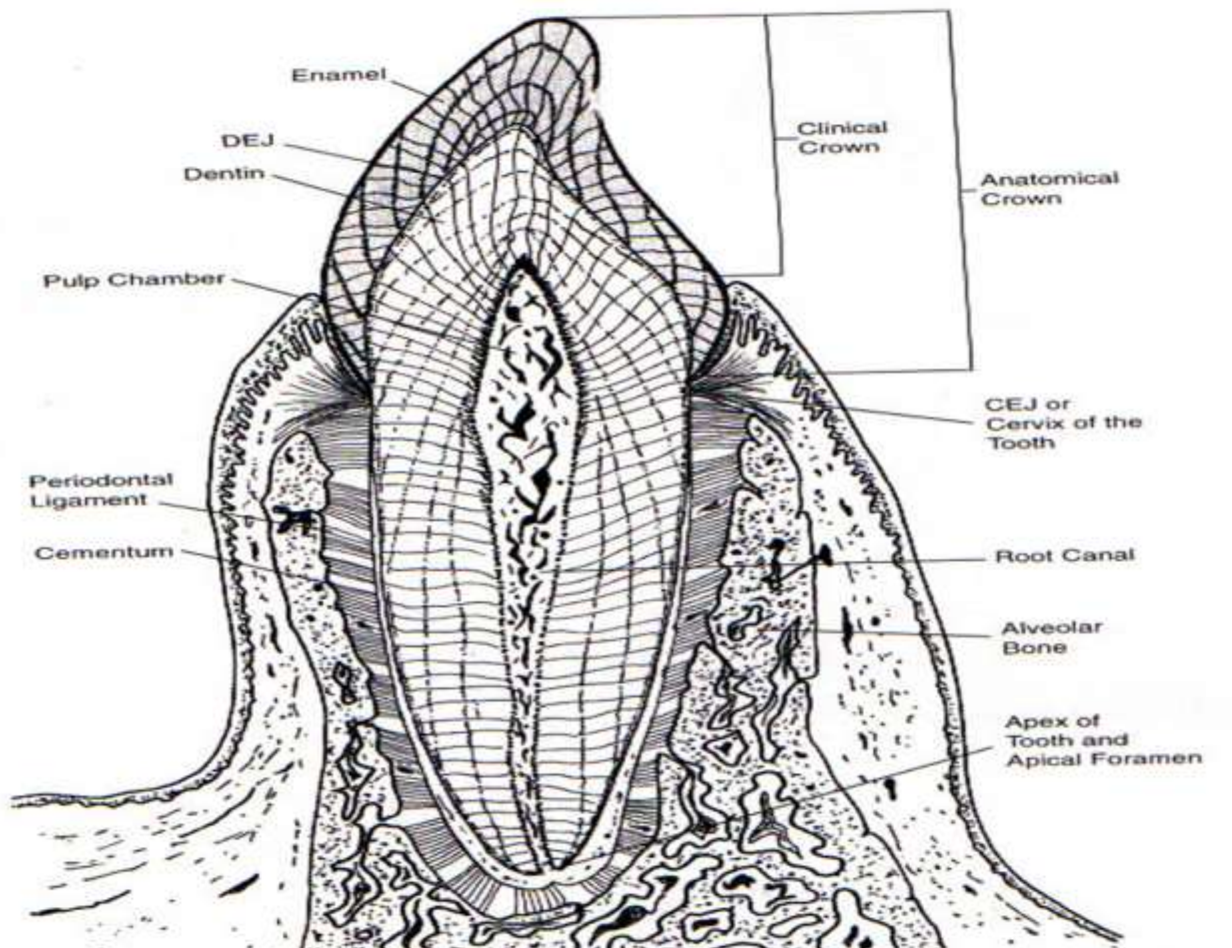


Figure 5–1. A diagram of longitudinal section of an incisor in situ. Note the anatomical boundaries between the mineralized tissues. DEJ, dentin-enamel junction. Observe the incremental lines in enamel and dentin.

2.Overview of Tooth Development

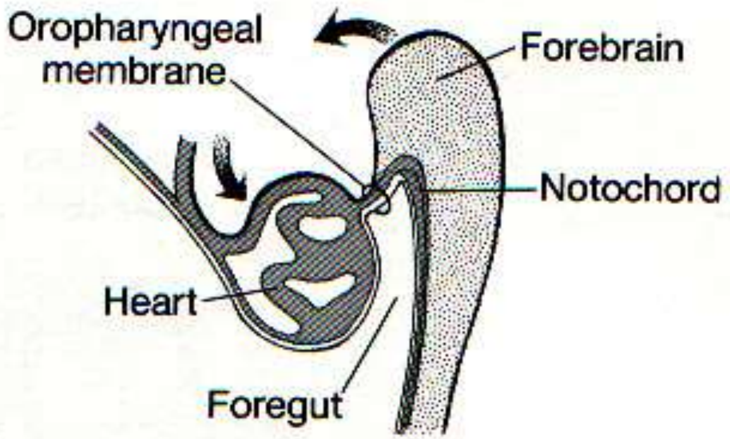


Figure 2–2. Anterior growth of brain vesicles, 2.5 weeks.

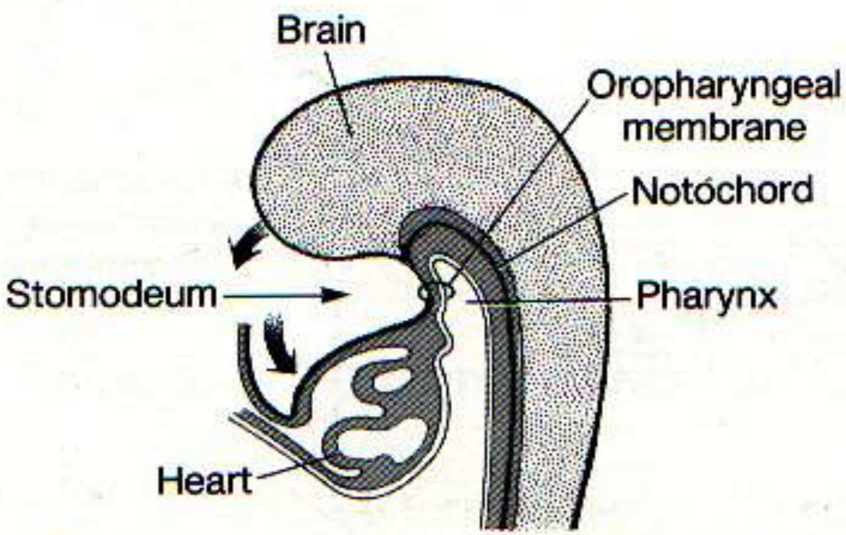


Figure 2–3. Further growth of the brain anteriorly, 3 weeks.

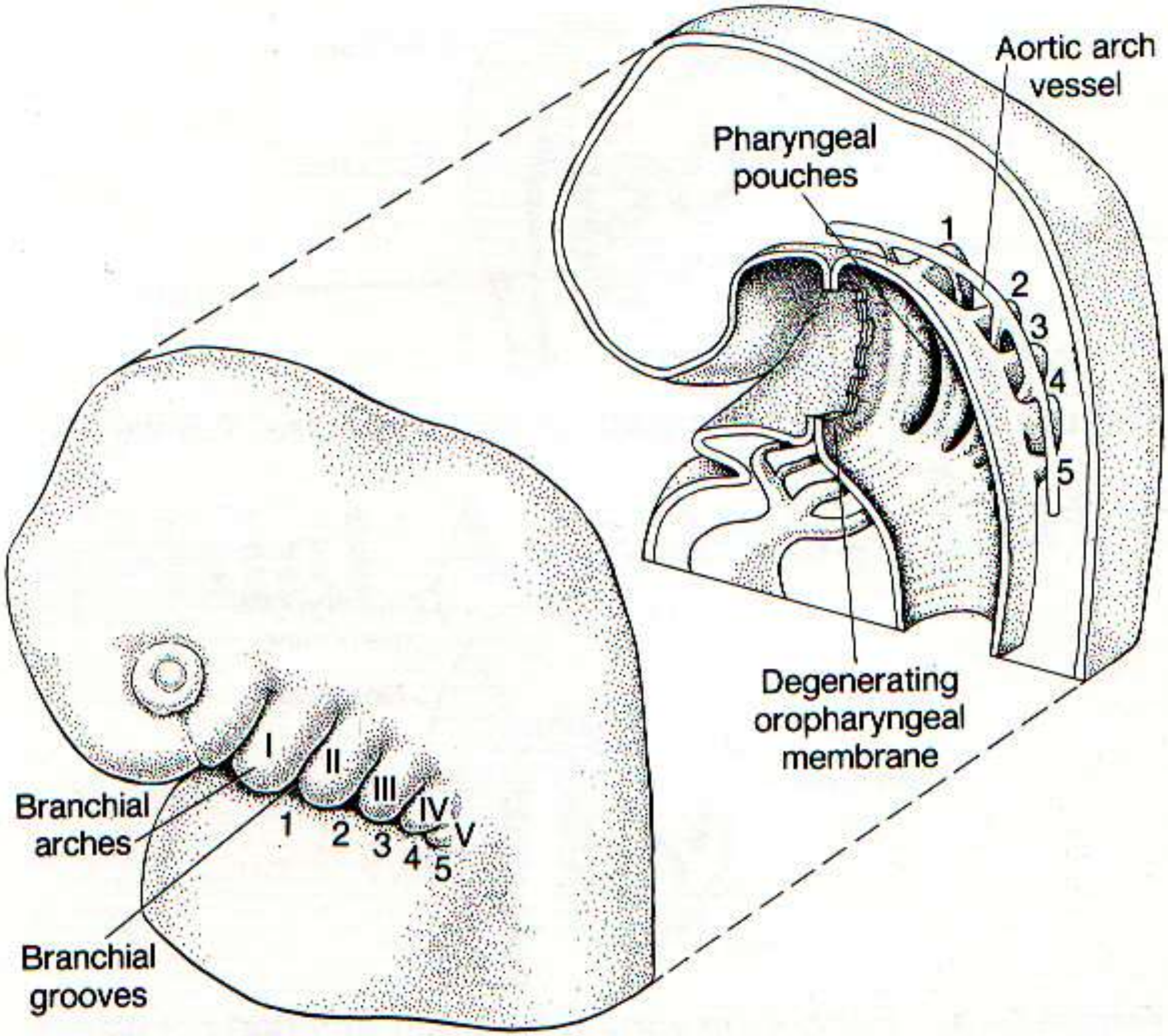


Figure 2–5. Sagittal view of branchial region at 4 weeks. Observe the blood vessels that arise from the heart below and pass through each branchial arch.

Primary Epithelial Band

- Forms 6 weeks after development of embryo.
- Bands are roughly horse shoe shaped.
- Each band of epithelium called primary epithelial band.
- Which gives rise to

  1. Vestibular Lamina
  2. Dental Lamina

Vestibular Lamina :

- No vestibule or cheek seen at 6 week of development.
- Forms as a result of proliferation of vestibular lamina in to ectomesenchyme.
- It is also termed as ***Lip Furrow Band***.

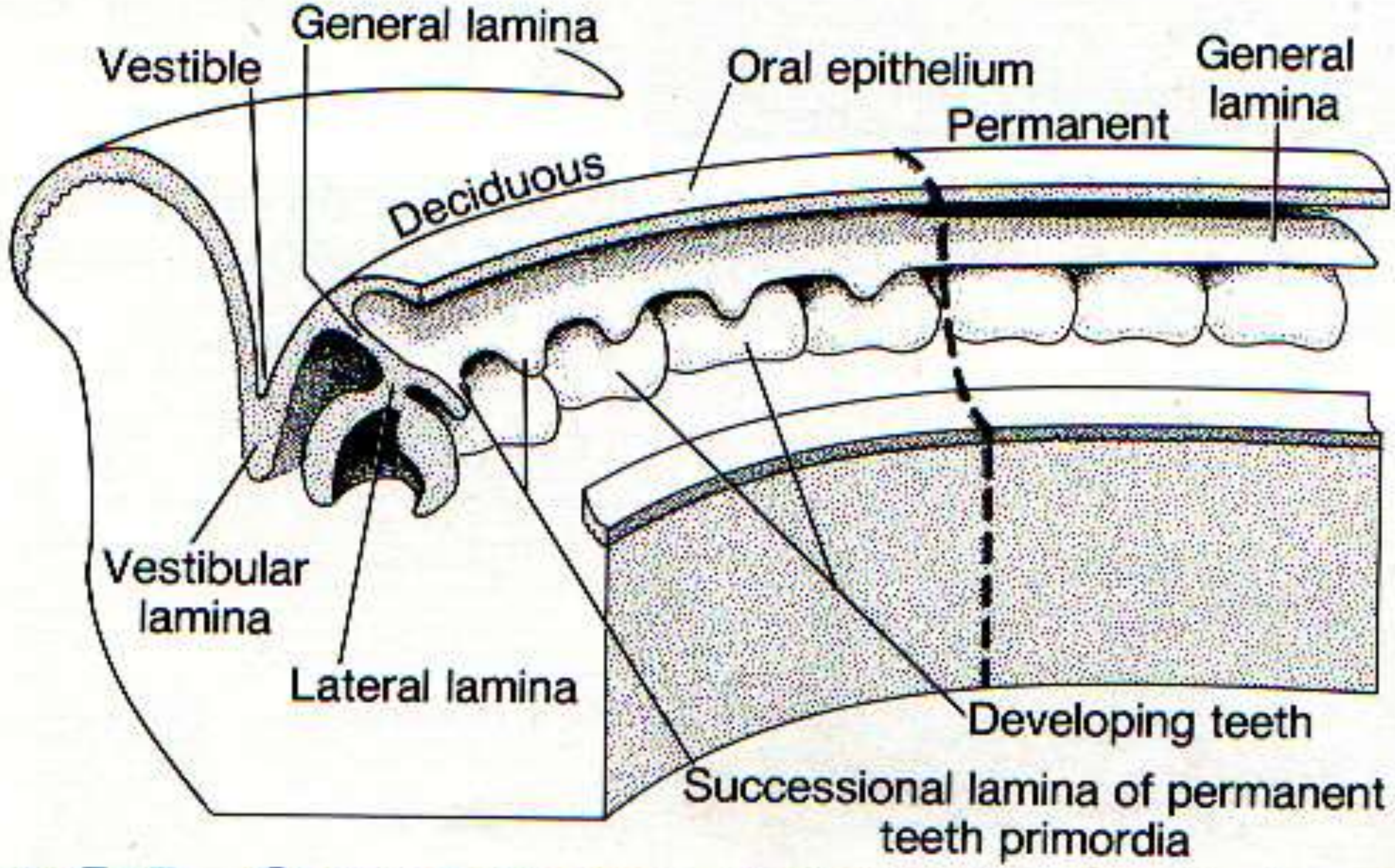


Figure 5–5. Stylized diagram depicting the continuity of the dental lamina system for deciduous and permanent teeth. Note the permanent molars arise from the general lamina and not the successional lamina.

Dental Lamina

- Is a band of epithelium that has invaded the underlying ectomesenchyme along each of horse shoe shaped dental arches.
- Serves as primordium of deciduous teeth.
- Distal proliferation of dental lamina is responsible for location of the germs of the permanent molars in ramus of mandible & tuberosity of maxilla.
- Successor of deciduous teeth develop from lingual extension of the dental lamina called ***successional lamina*** and develops from the fifth month in utero (Permanent central incisor) to the tenth month of age (second premolar)

<u>Fate of Dental Lamina:</u>

- Total activity of dental lamina extends over a period of 5 years
- Any particular portion of dental lamina functions for a much briefer period
- However , dental lamina may still be active in third molar region after it has disappeared elsewhere
- As teeth develop they loose their continuity with dental lamina
- Remnants of dental lamina – persist as epithelial pearl or islands within jaw as well as in the gingiva called ***<u>Cell rest of serres</u>***

Tooth Development :

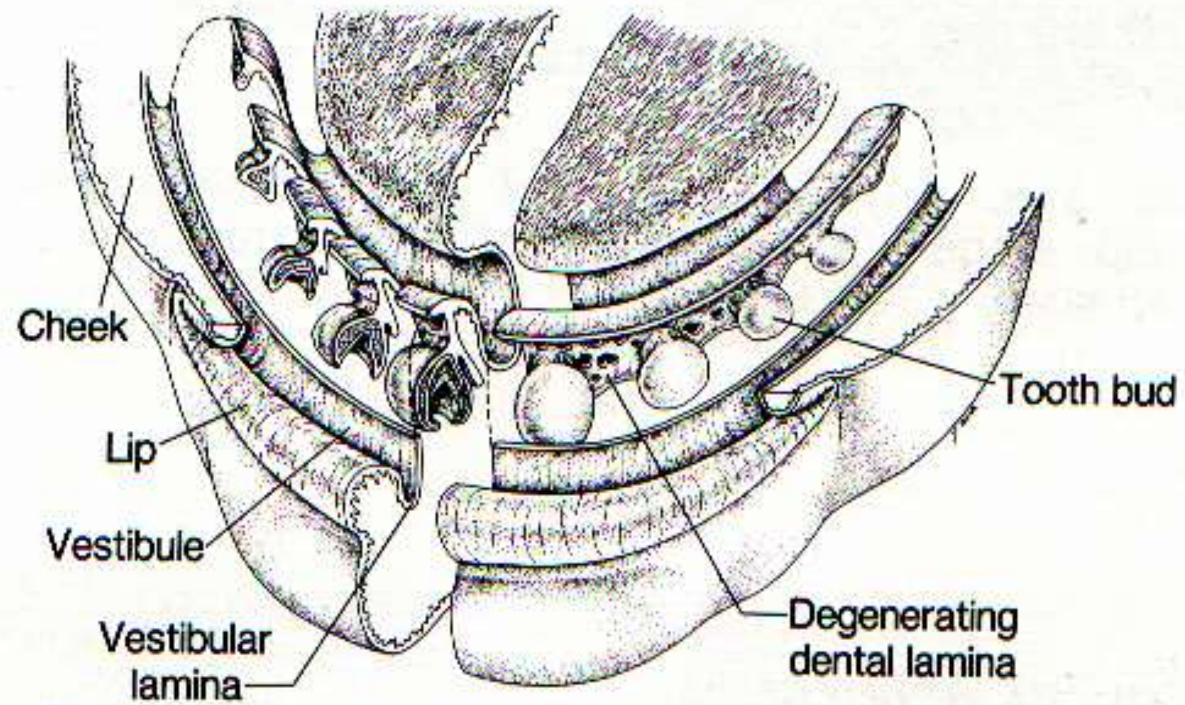


Figure 5–6. Development of tooth buds in developing alveolar processes.

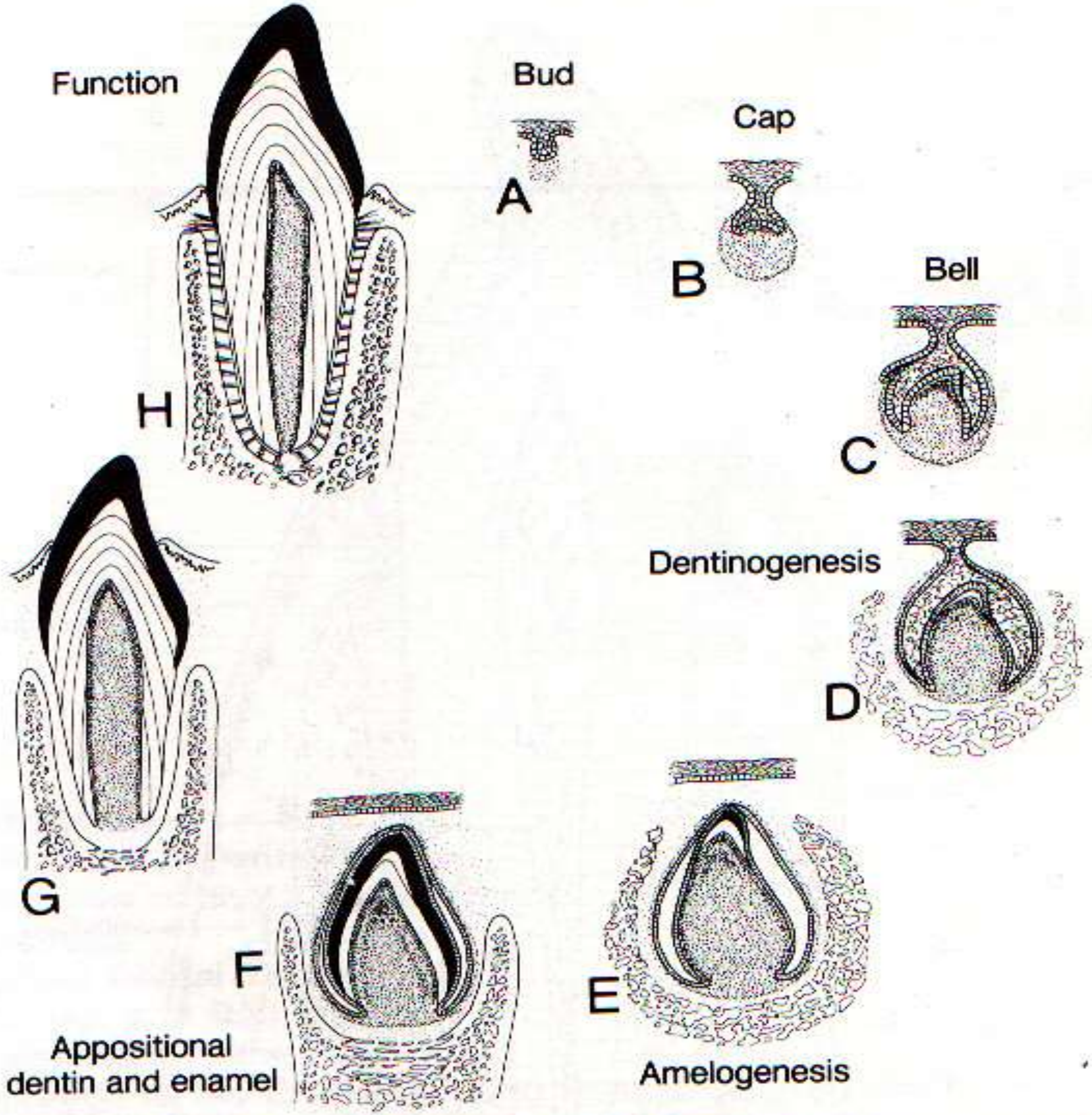


Figure 5–2. (A-H). Diagram depicting the stages of tooth development beginning with the bud stage (A).

Developmental Stages

- Although tooth development is a continuous process, the development history is divided into several morphological stages.
- While the size & shape of individual tooth are different they pass through same stages of development.
- They are named after shape of epithelial part of tooth germ & are called Bud, Cap, Bell Stages, advanced Bell Stage

Bud Stage :

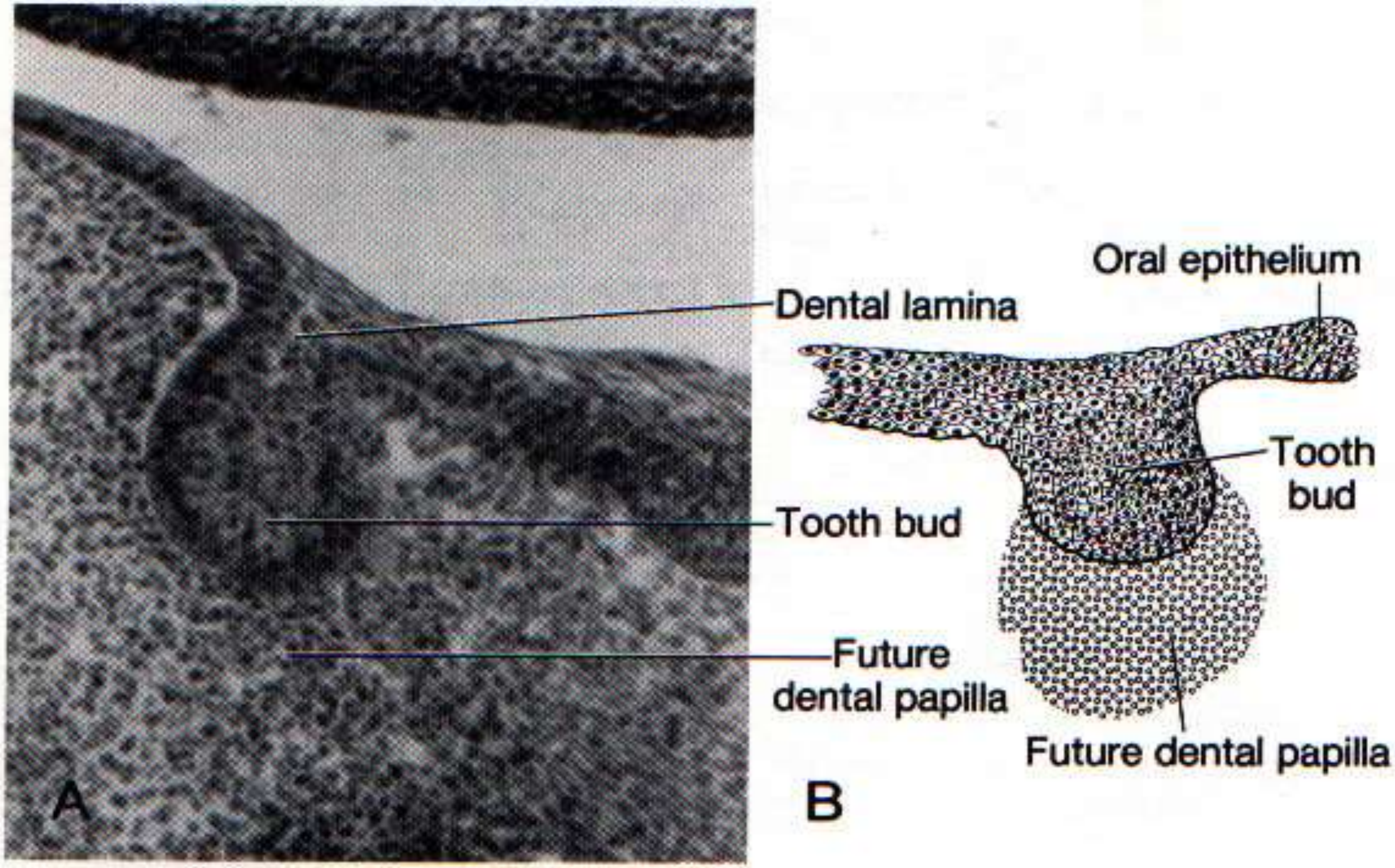


Figure 5–7. (A). Histology of tooth development at the bud stage. (B). Diagram of tooth development at the bud stage.

Cap Stage:

- **Outer & inner enamel epithelium**
  - Enamel niche – enamel organ is seen to have a double attachment to dental lamina to the overlying oral epithelium
- **Stellate reticulum (enamel pulp)**
  - Star shaped cells
  - Acts as shock absorber
    - Since the spaces in stellate reticulum are filled with albumin rich mucoid fluid
  - Transitory Structures
  - ❖ Enamel Knot – densely packed cells at the center of enamel organ
  - ❖ Enamel Cord – vertical extension of enamel knot
  - ❖ Enamel Septum – when enamel cord extends to meet outer enamel epithelium
  - ❖ Enamel navel – outer enamel epithelium at the point of meeting

- These temporary structures acts as reservoir of dividing cells of dividing enamel organ
- Enamel knot acts as signaling center

- **Dental papilla –**
  - Formative organ of dentin and the primordium of pulp
  - Epithelium exerts a dominating influence over the adjacent connective tissue
  - It shows active budding of capillaries and mitotic figures
  - Its peripheral cells adjacent to inner enamel epithelium enlarge and develops in odontoblasts
- **Dental sac –**
  - Also called as dental follicle
  - There is marginal condensation in ectomesenchyme surrounding the enamel organ & dental papilla forming primitive dental sac

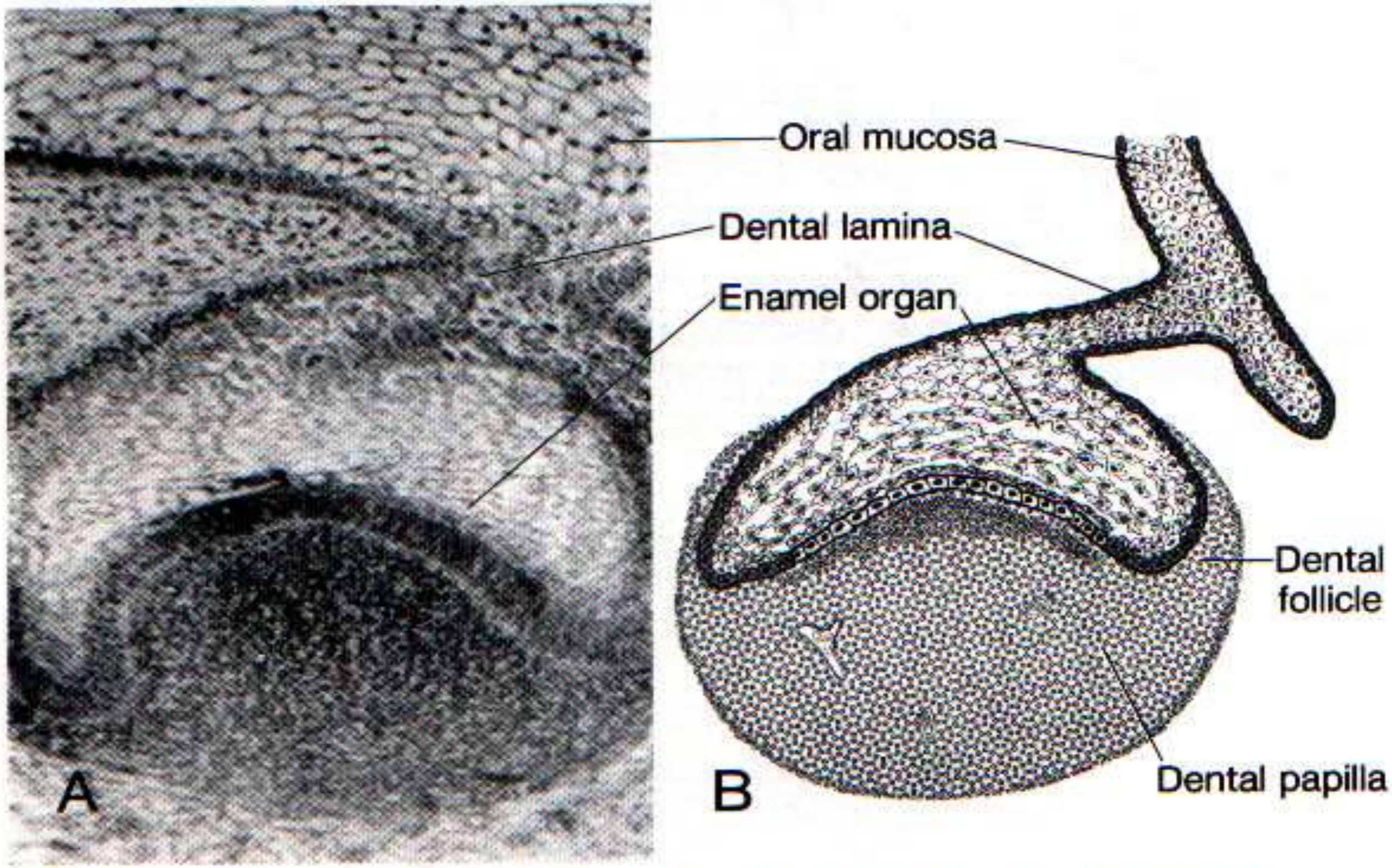


Figure 5–8. (A). Histology of tooth development at the cap stage. (B). Diagram of tooth development at the cap stage.

Bell Stage:

- Four different types of epithelial cells can be distinguished on light microscopic examination of the bell stage of the enamel organ.

1. **Inner enamel epithelium**
    1. Tall columnar, develops into ameloblast
2. **Stratum intermedium :**

   Well developed cytoplasmic , acid mucopolysaccharides, and glycogen deposits
3. **Stellate reticulum.**
4. **Outer enamel epithelium** : provide nutritive supply to avascular enamel organ

- **Dental papilla:**
  - The basement membrane that separates the enamel organ & the dental papilla just prior to dentin formation is called ***membrana preformativa***
- **Dental sac:**
  - Before formation of dental tissue begins the dental sac shows a circular arrangement of its fibers & resembles a capsular structure
  - With the development of root , the fibers of dental sac differentiate into periodontal fibers
- Shape of crown is determined at this stage
- The junction between inner & outer enamel epithelium is called cervical loop (area of intense mitotic activity)

ADVANCED BELL STAGE

- Commencement of mineralization and root formation
- Formation of dentin occurs first as a layer along future DEJ proceeds pulpally and apically
- The enamel formation proceeds coronally and cervically
- Cervical portion of enamel organ gives rise to Hertwig's epithelial root sheath

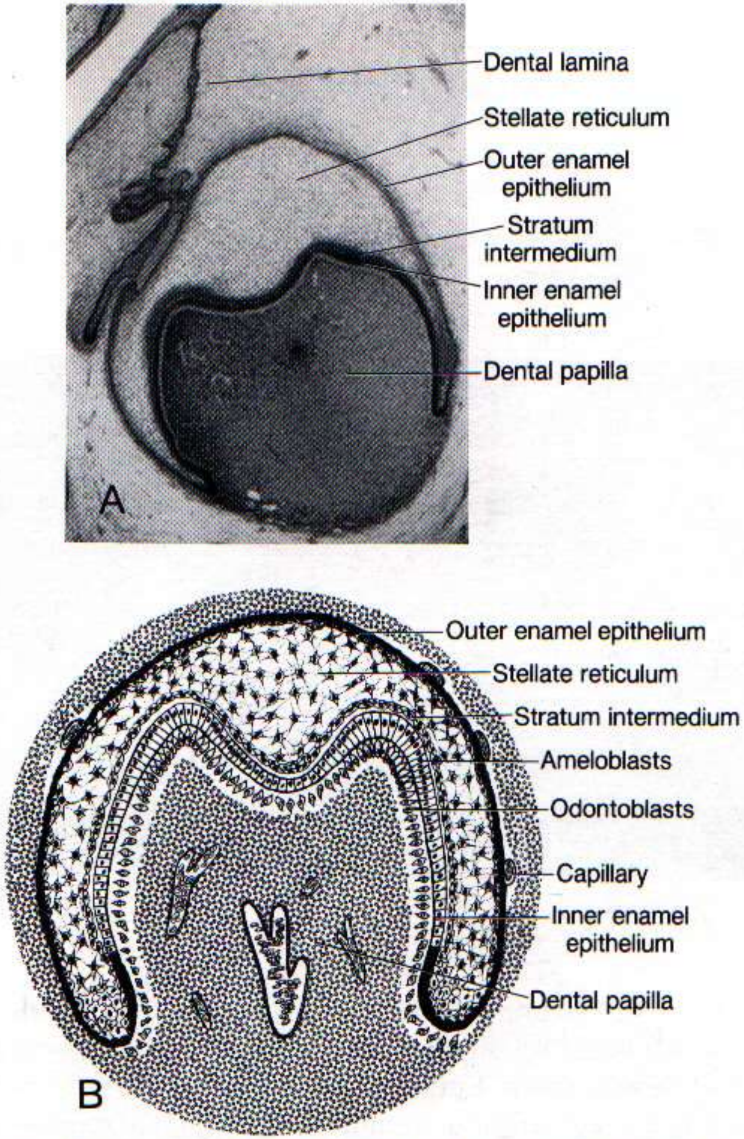


Figure 5–9. (A). Histology of tooth development at the bell stage. (B). Diagram of tooth development at the bell stage.

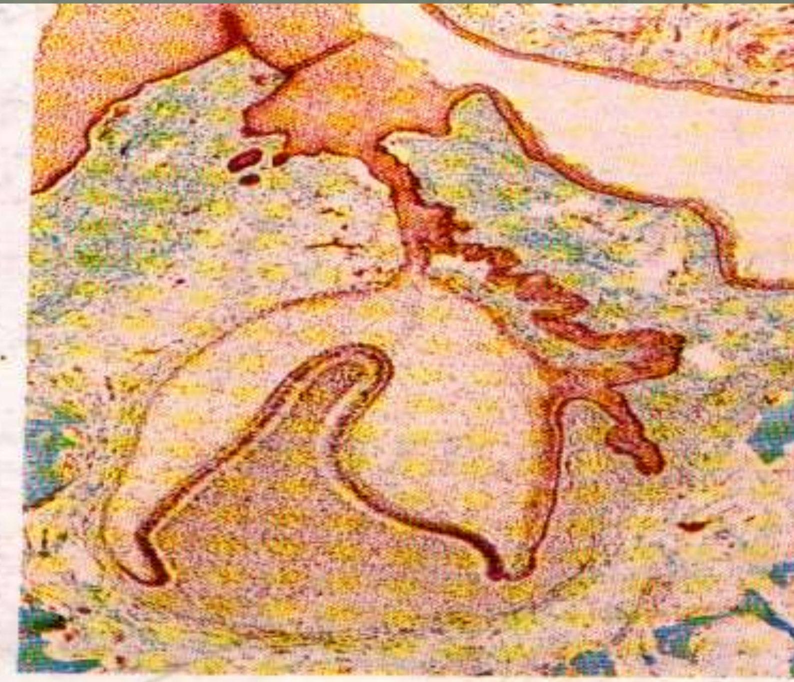
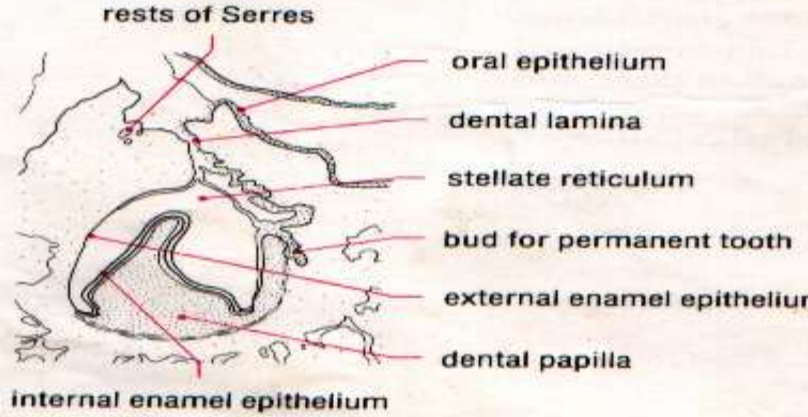


Fig. 1.2 Developing tooth germ at the bell stage. The dental lamina remains attached to the overlying oral epithelium. Trichrome stain.



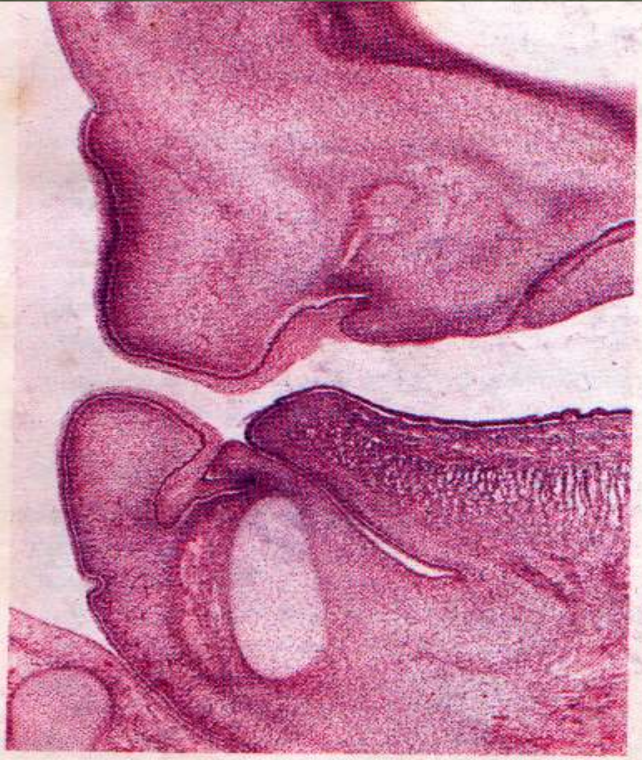


Fig. 1.1 Histology of the jaws of a fetus showing a developing tooth germ in the cap stage.

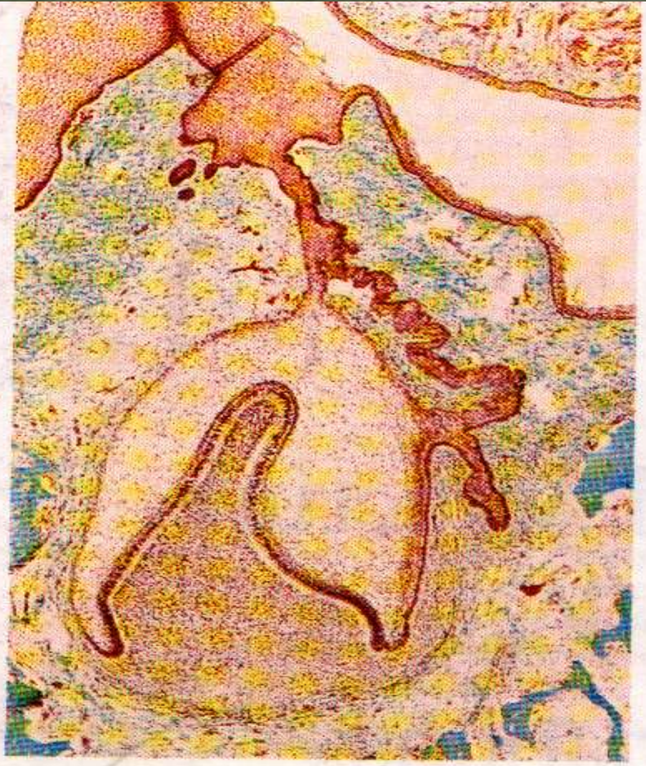


Fig. 1.2 Developing tooth germ at the bell stage. The dental lamina remains attached to the overlying oral epithelium. Trichrome stain.

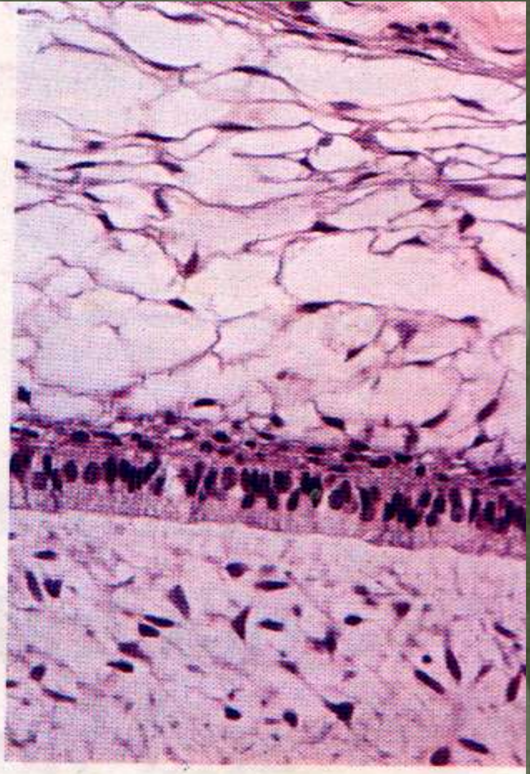
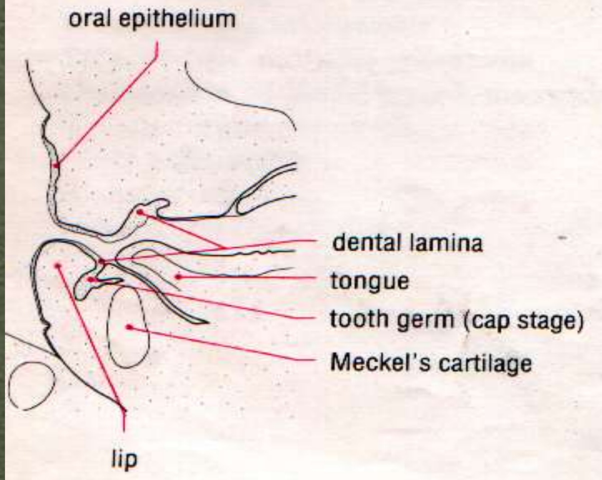
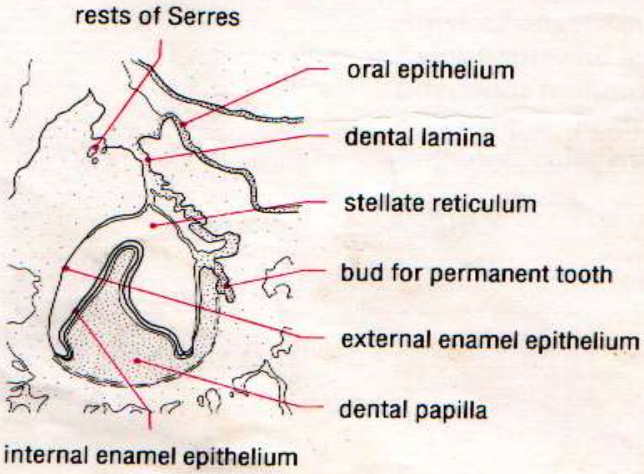


Fig. 1.3 High power view of the interna enamel epithelium.





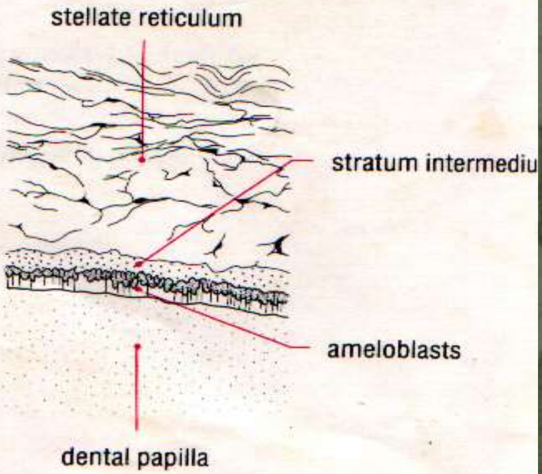


Fig. 1.4 Histology of advanced bell stage (about 18 weeks *in utero*).

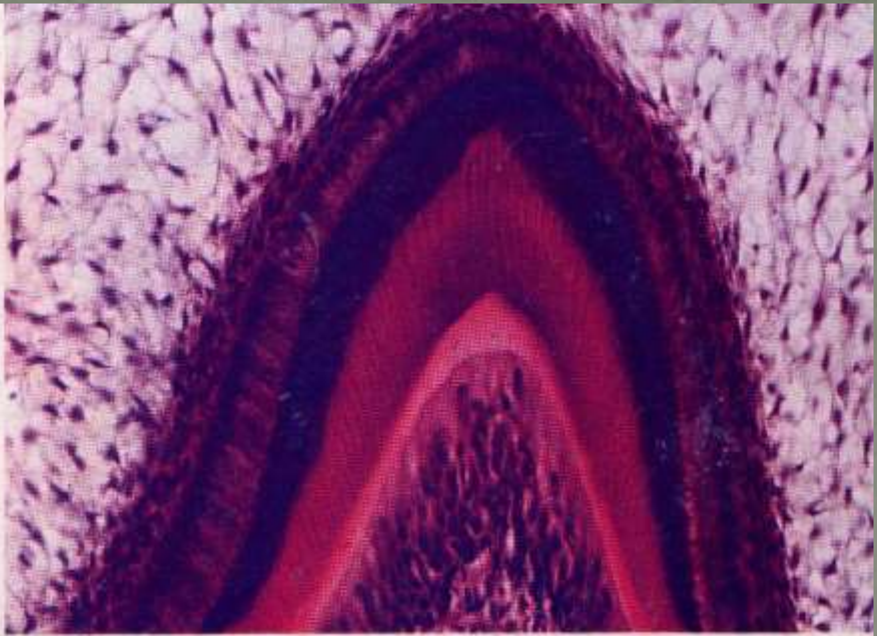
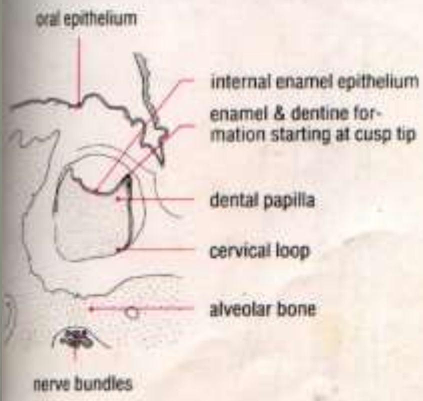
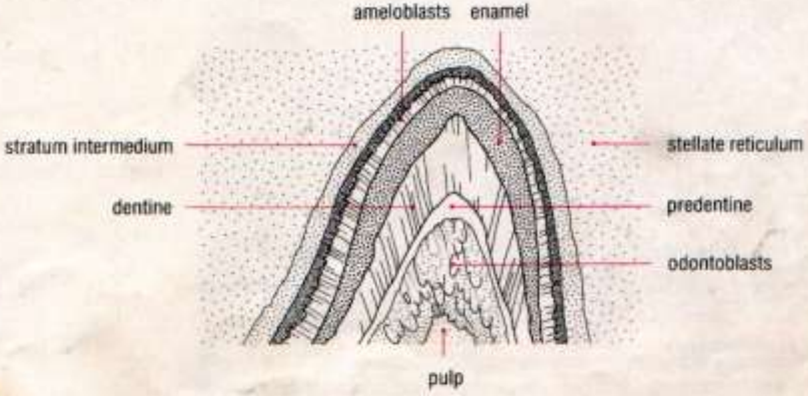


Fig. 1.5 High power view of advanced bell stage showing early hard tissue formation.



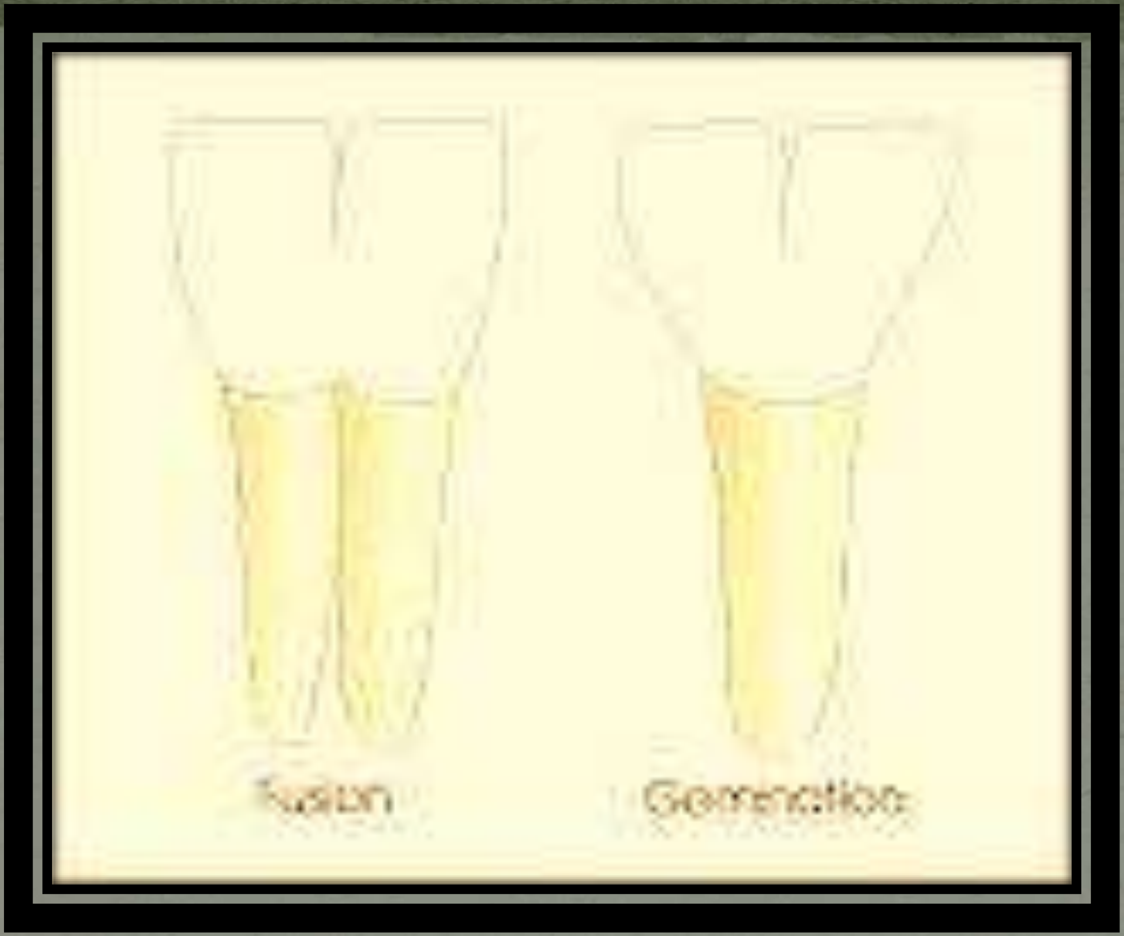


HISTOPHYSIOLOGY

- INITIATION:
  - Different teeth are initiated at definite times
  - Initiation induction requires ectomesenchymal-epithelial interaction
  - Lack of initiation results in **anodontia**
  - Abnormal imitation results in **supernumerary** teeth .

ANODONTIA

SUPERNUMARARY TEETH



- PROLIFERATION:
  - Proliferative growth causes regular changes in size & proportions of the growing tooth germ

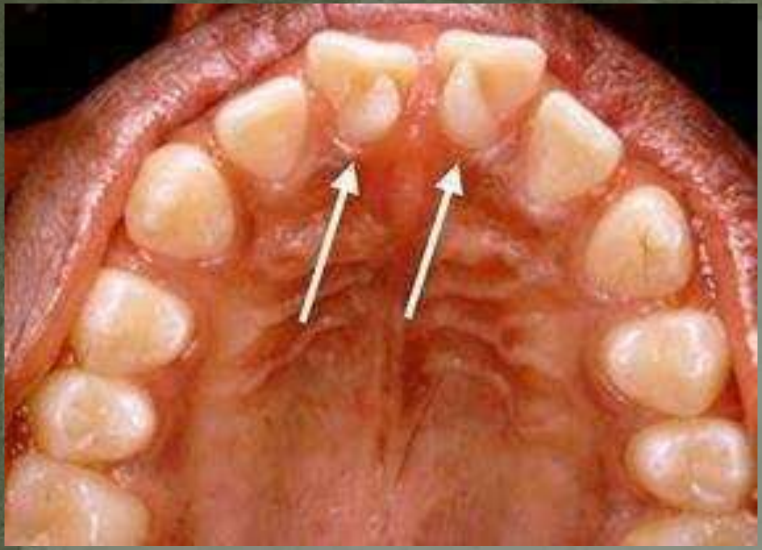
HISTODIFFERENTIATION

- The formative cells of the tooth germs developing during the proliferative stage undergo definite morphologic as well as functional changes and acquire their functional assignment
- This phase reaches its highest development in bell stage of enamel organ
- The organizing influence of inner enamel epithelium causes the differentiation of adjacent cells of dental papilla into odontoblasts
- With the formation of dentin , the cells of the inner enamel epithelium differentiates into ameloblasts & enamel matrix is formed opposite to the dentin
- Enamel does not form in absence of dentin
- Disturbance in this stage causes dentinogenesis imperfecta or atypical dentin

Dentinogenesis imperfecta

MORPHODIFFERENTIATION:

- Basic form and relative size of future tooth is determined
- The dentinoenamel & dentinocemental junctions , which are different and characteristic of each tooth , acts as a blueprint pattern
- Disturbance causes
  - Supernumarary cusps (**talons cusp**)
  - Twinning
  - Loss of cusps or roots
  - Malformed or peg shaped tooth (**Hutchinson's incisor**)
  - **Dens in dente**
  - **Macrodontia**
  - **Microdontia**



Talons cusp

Hutchinson's incisor

- Dens in dente

- MACRODONTIA

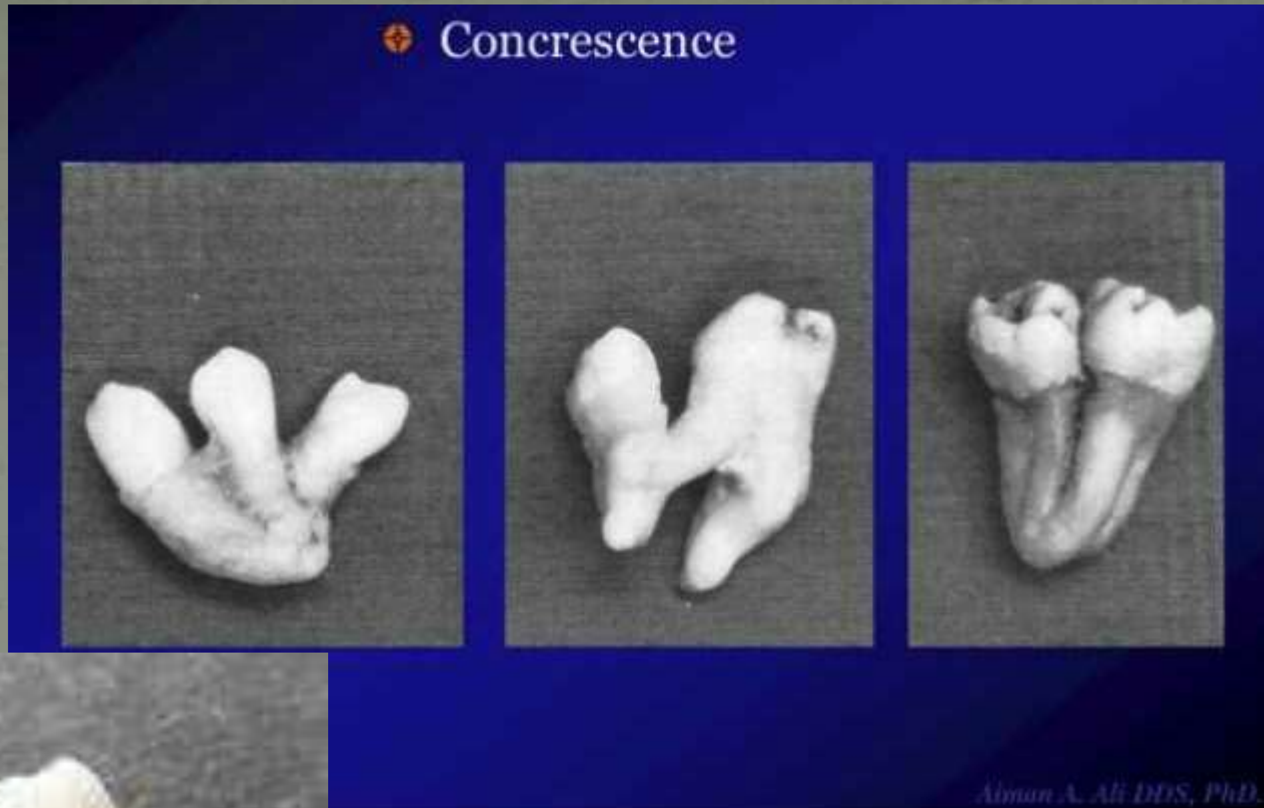
- MICRODONTIA

APPOSITION

- Apposition is the deposition of the matrix of hard dental strctures
- Appositional growth is characterized by regular and rhythmic deposition of extracellular matrix, which is of itself incapable of further growh
- Disturbance causes :
  - Enamel hypoplasia
  - Hypocalcification
  - Intrinsic staining
  - Concrescence


Enamel Hypoplasia
ToothIQ.com


Intrinsic Staining


Concrescence
Aiman A. Ali DDS, PhD.

Stages in tooth Development

Morphologic stages	Physiologic processes
Dental Lamina ⟷	Initiation
Bud Stage	
Cap Stage	Proliferation
Bell Stage (early)	Histodifferentiation
Bell Stage (advanced)	Morphodifferentiation
Formation of enamel And Dental Matrix	Apposition

Hertwig's root sheath:

- Hertwig's root sheath which is the successor of the cervical loop of the tooth organ controls root formation.
- It is responsible for determining the size, shape & number of roots formed.
- Root development begins after enamel & dentin formation has reached the future CEJ.
- Hertwigs RS consist of outer & inner enamel epithelia only, it dose not include stallete reticulum & stratum intermedium.

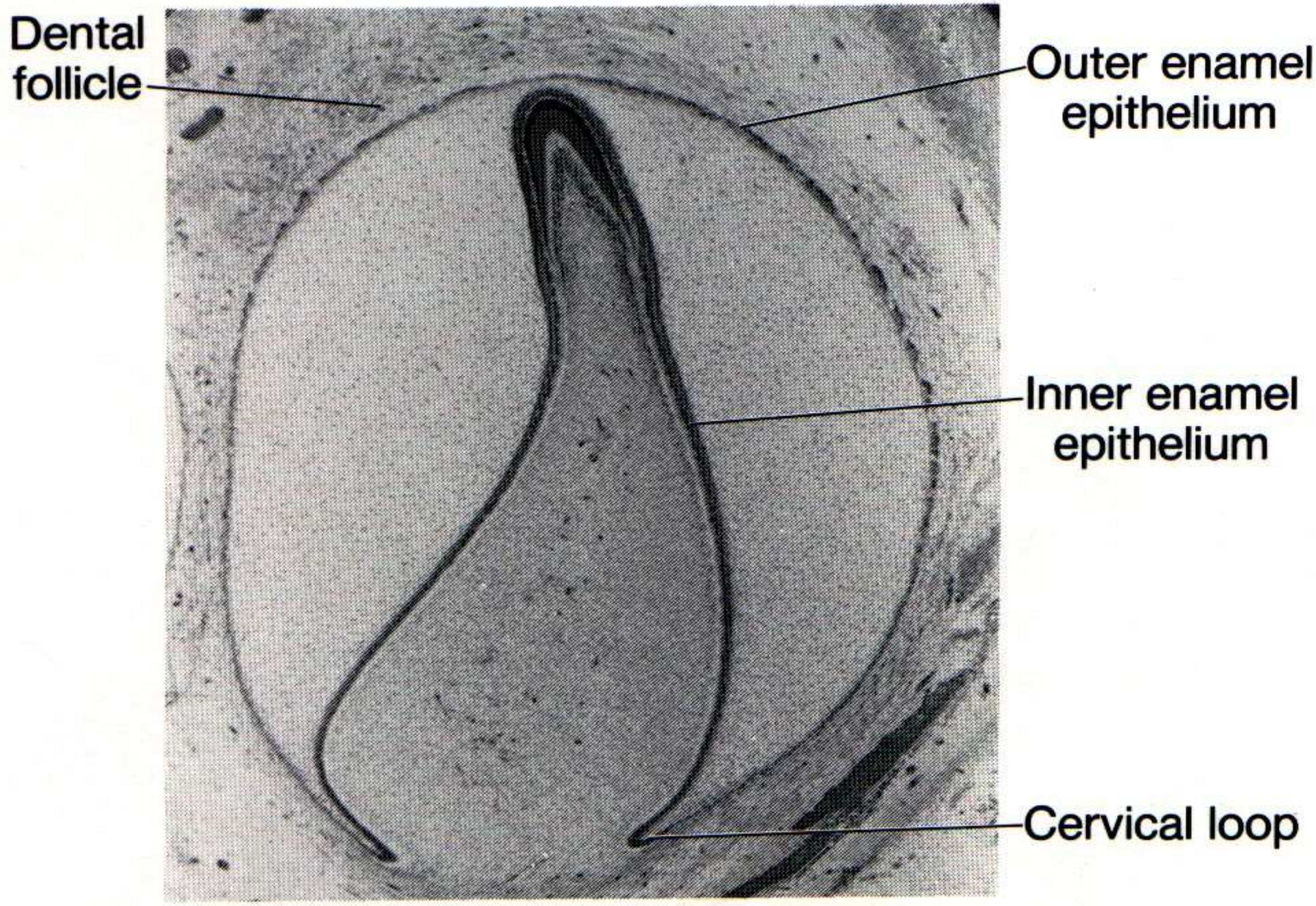


Figure 6–1. Formation of cervical loop.

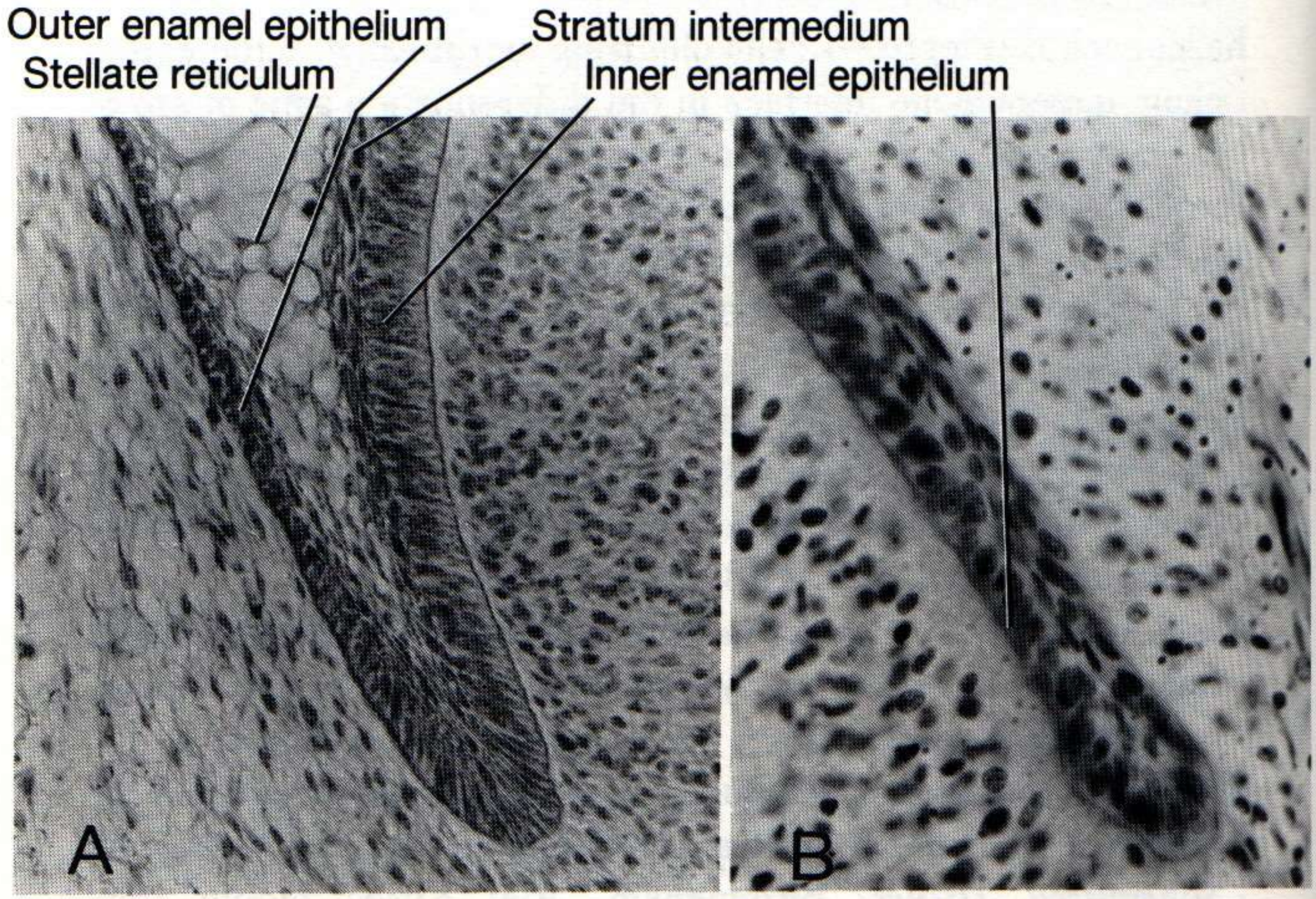


Figure 6–2. Higher magnification of cervical loop (A) and root sheath (B).

- The cells of inner layer remains short & normally do not produce enamel. When these cells induce the differentiation of radicular cells into odontoblast & the first layer of dentin has been laid down, the epithelial RS looses its structural continuity & its close relation to the surface of the root. Its remnants are found in pdl of erupted teeth & are called ***rests of Malassez***.

- Prior to the beginning of root formation, the root sheath forms the epithelial diaphragm. The outer & inner enamel epithelia bend at the future CEJ into a horizontal plane, narrowing the wide cervical opening of the tooth germ.

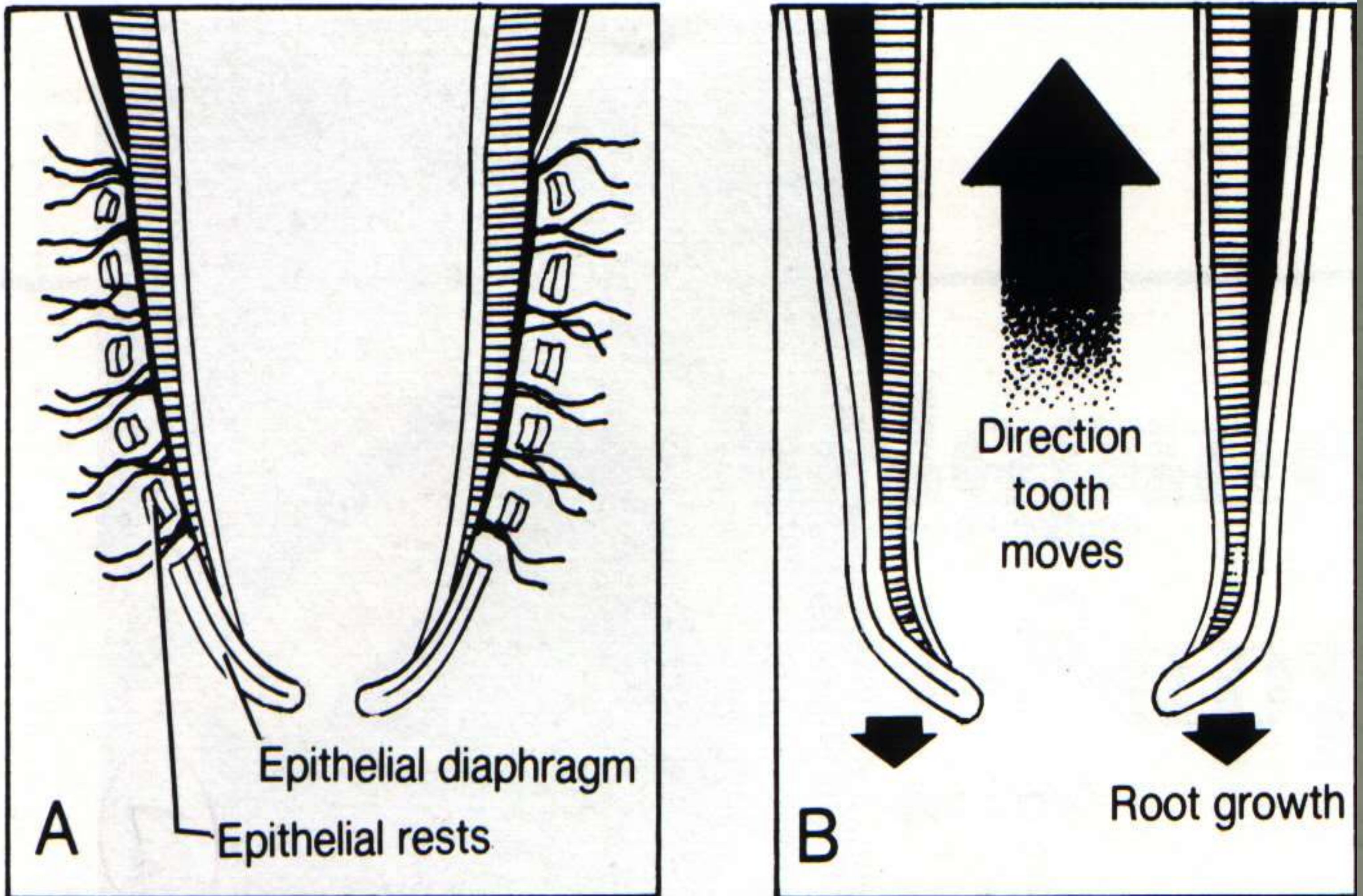


Figure 6–5. Root elongation (A) and tooth eruption (B)

- Proliferation of the cells of the epithelial diaphragm is accompanied by proliferation of the cells of the connective tissue of the pulp.The free end dose not grow into the connective tissue, but epithelium proliferates coronally to the epithelial diaphragm.
-
- The differentiation of odontoblast & the formation of dentin follow the lengthening of the root sheath. At the same time the connective tissue of the dental sac surrounding the RS proliferates & divide the continuous double epithelial layer into a network of epithelial strands.

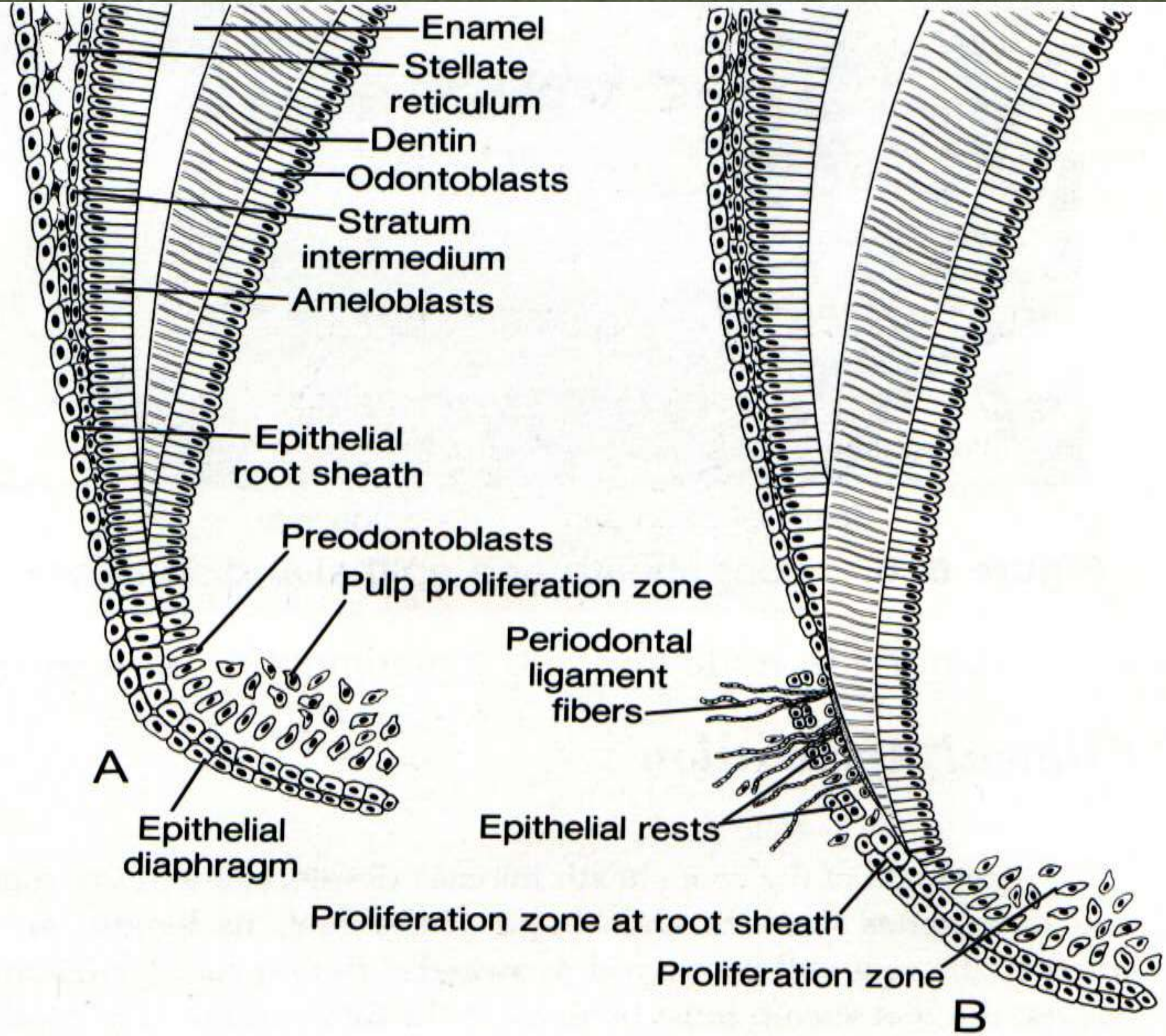


Figure 6–4. Formation of epithelial diaphragm. (A). Initial root formation. (B). Later root formation.

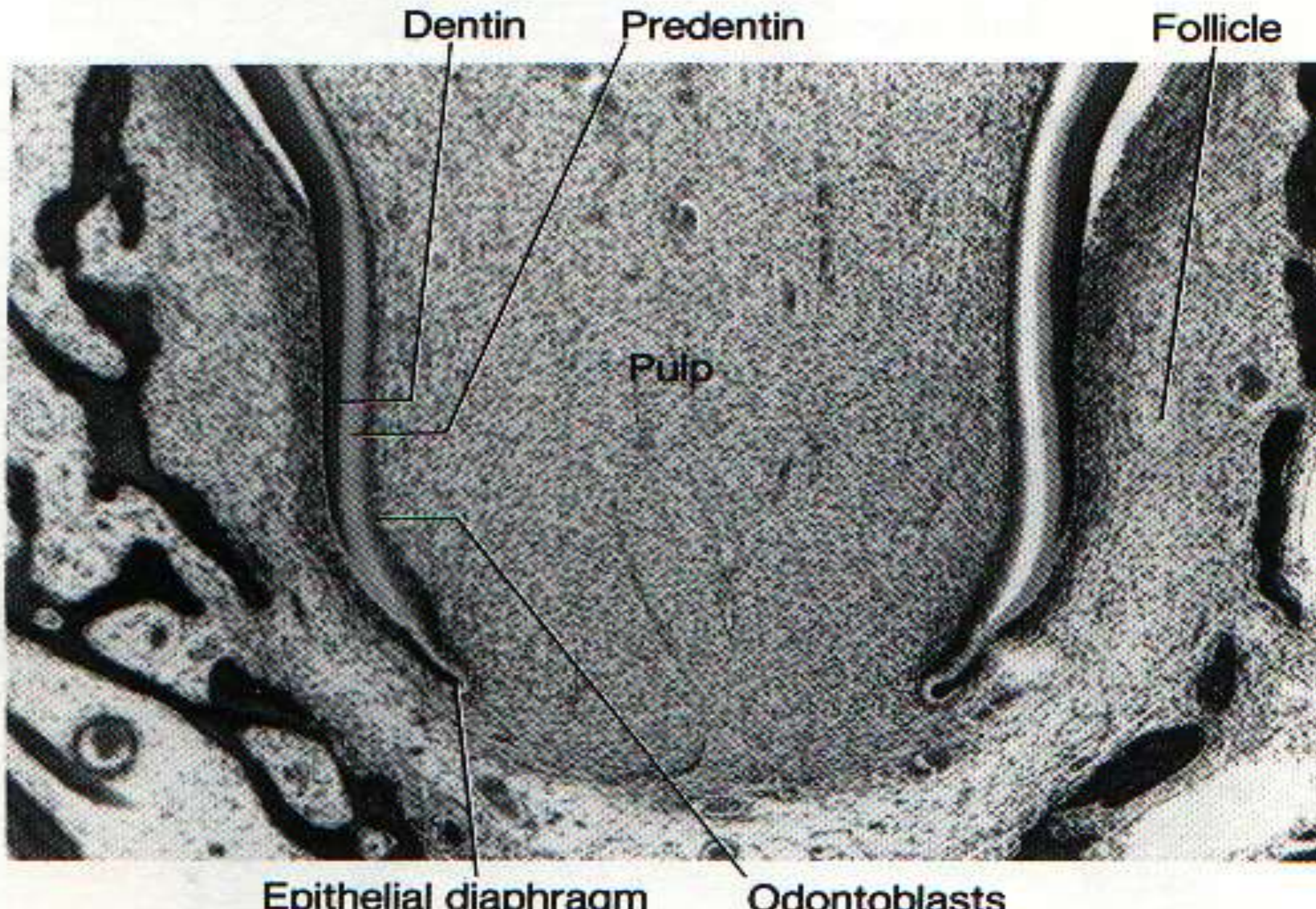


Figure 6–6. Root sheath and epithelial diaphragm.

- The epithelium is moved away From the surface of the dentin so that connective tissue cells come into contact with the outer surface of the dentin & differentiate into cementoblast that deposit a layer of cementum onto the surface of the dentin.

- In the last stage of root formation, the proliferation of the epithelium in the diaphragm lags behind that of the pulpal connective tissue.

- The wide apical foramen is reduced first to the width of the diaphragm opening itself & later is further narrowed by apposition of dentin & cementum to the apex of the root.

- During the growth of the enamel organ the expansion of its cervical opening occurs in such a way that long ***tongue like*** extension of the horizontal diaphragm develop.Two such extension are found in the germs of upper molar.

- Before the division of the root trunk occurs, the free ends of these horizontal epithelial flaps grow towards each other & fuse. The single cervical opening of the coronal enamels organ is then divided into two or three openings.

- On the pulpal surface of the dividing epithelial bridges, dentin formation starts.

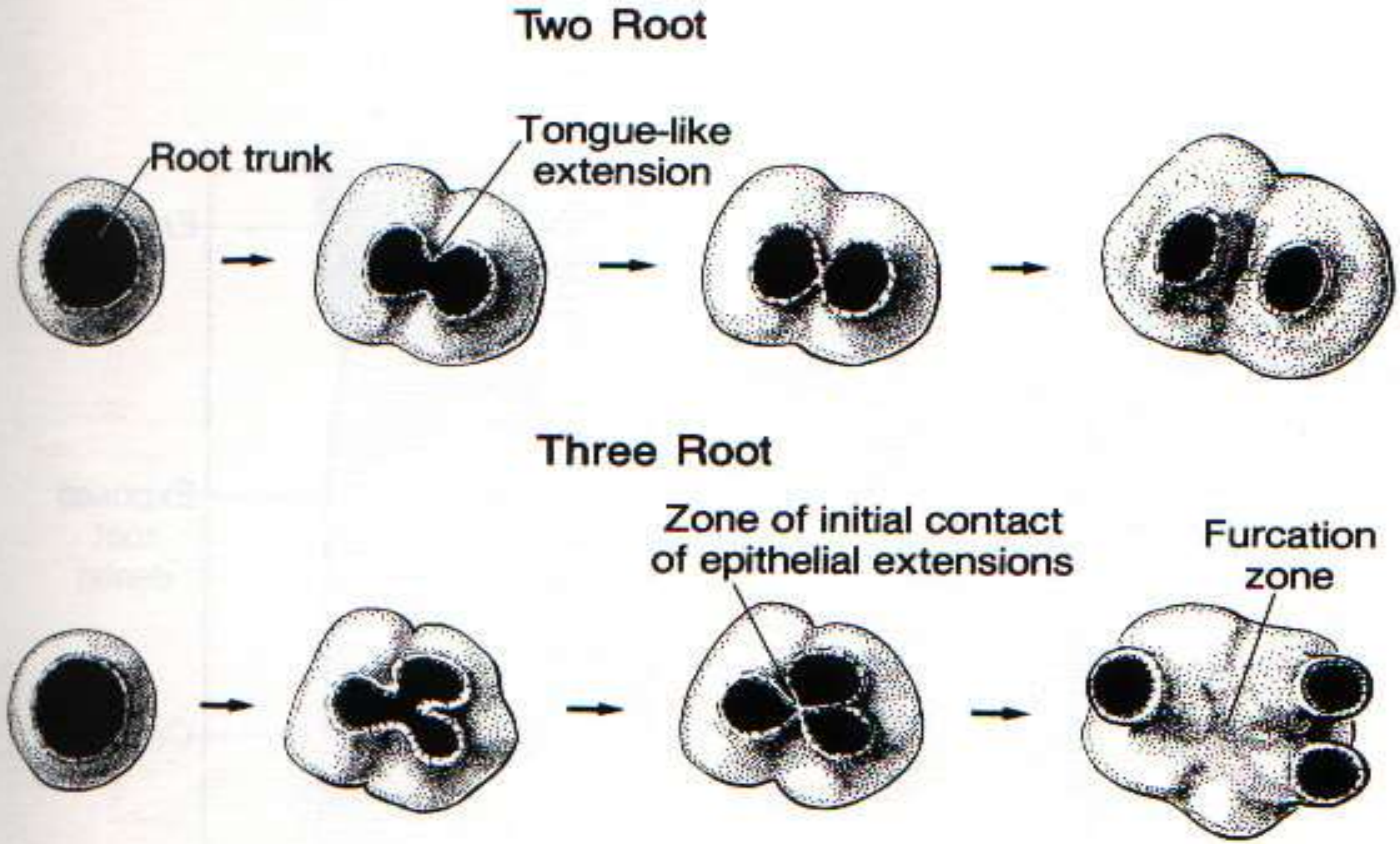


Figure 6–7. Multiple-root development. Note that the number of tonguelike extensions dividing the single root on left is equal to the number of roots to be formed on the right.

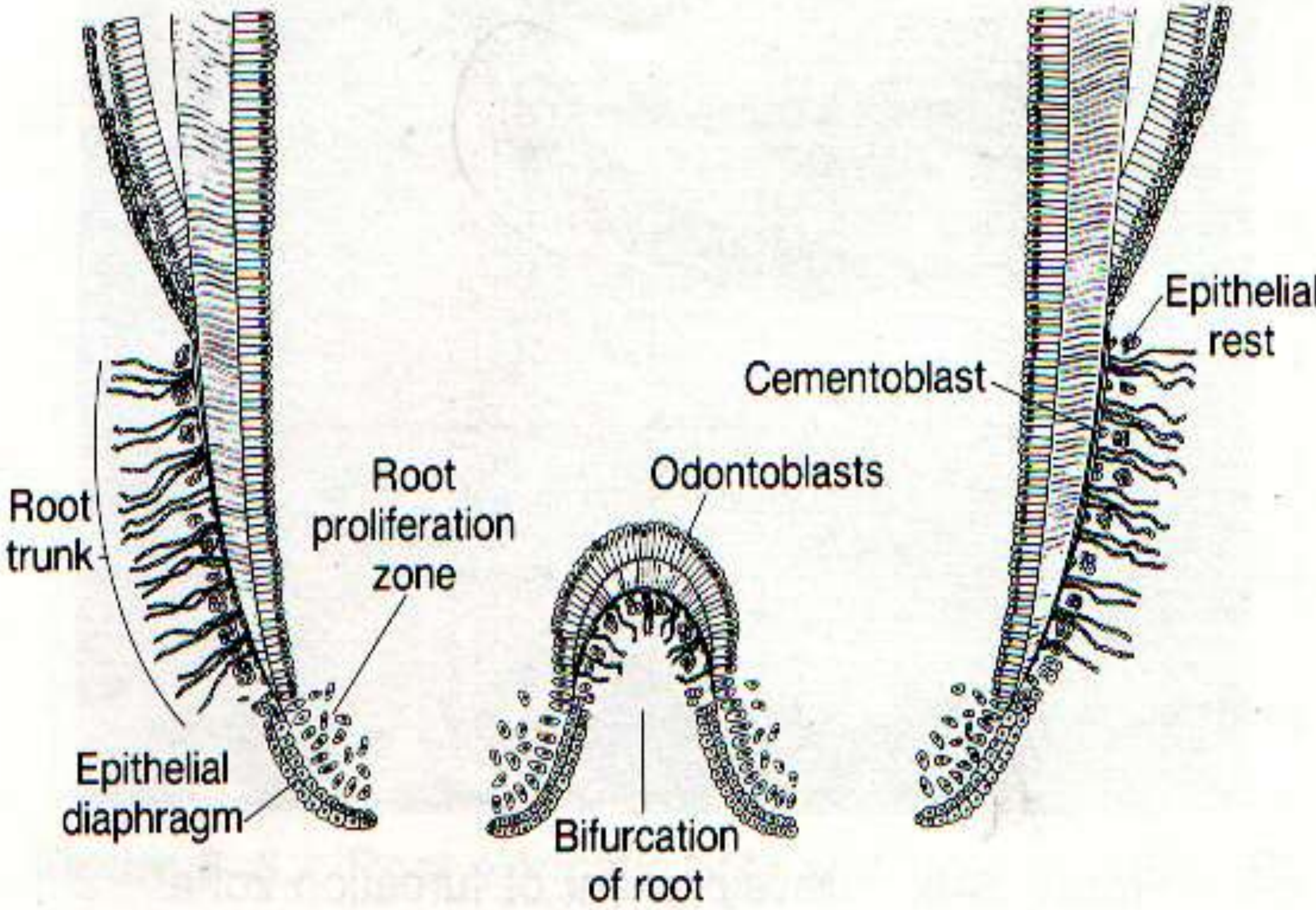


Figure 6–11. Development of individual root.

- If the cells of the epithelial RS remain adherent to the dentin surface, they may differentiate into fully functioning ameloblast & produce enamel called ***enamel pearls***, found in the areas of furcation of the roots of PM.

- If continuity of Hertwig's RS is broken or is not established prior to dentin formation, a defect in the dentinal wall of the pulp ensures. This accounts for development of accessory root canals opening.

- If continuity of Hertwig's RS is not broken even after dentin formation it results in exposure of root dentin to oral cavity.

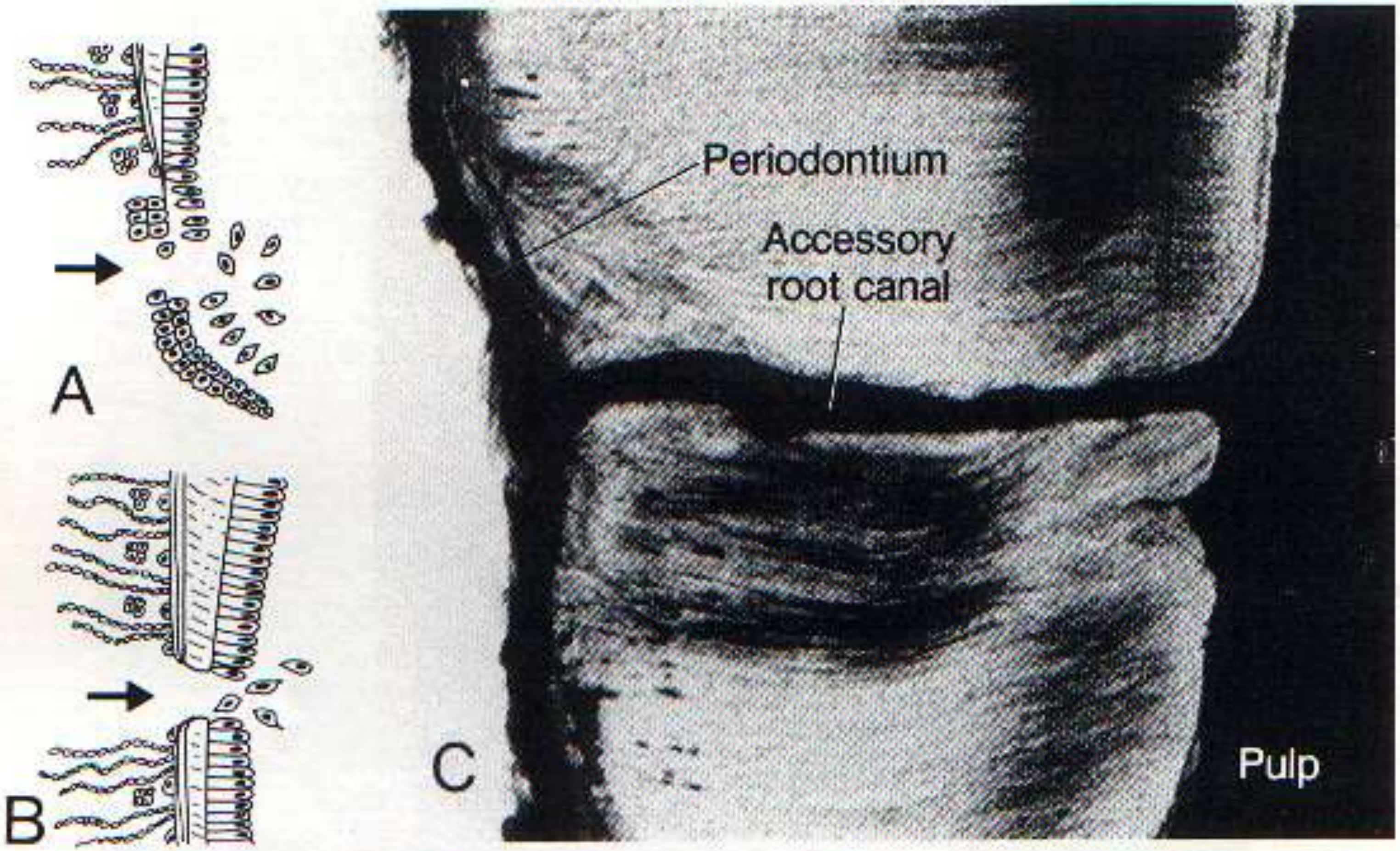


Figure 6–12. (A). Formation of defective root sheath. (B). Lack of odontoblast differentiation and formation of dentin. (C). Resulting accessory canal in mature tooth.

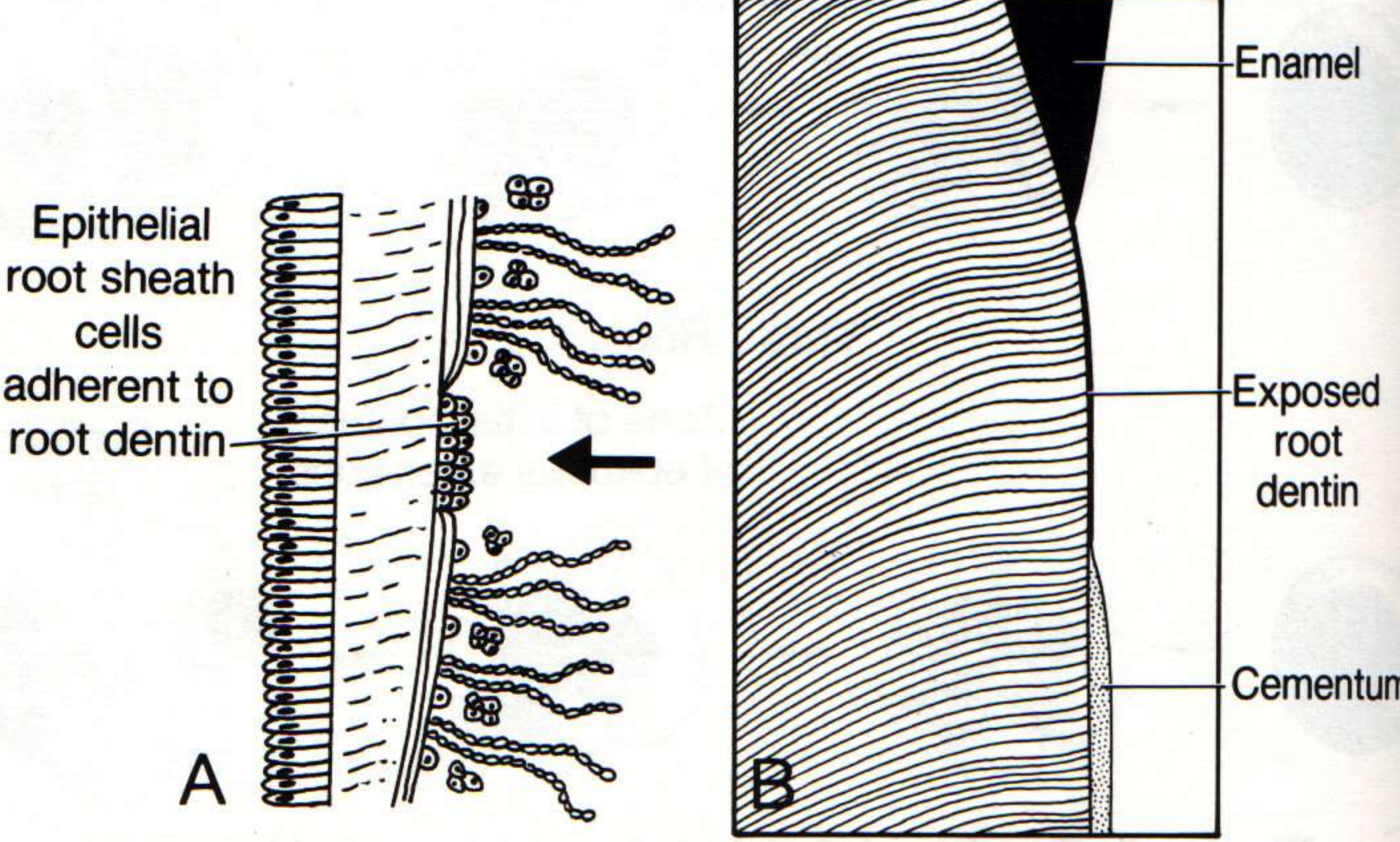
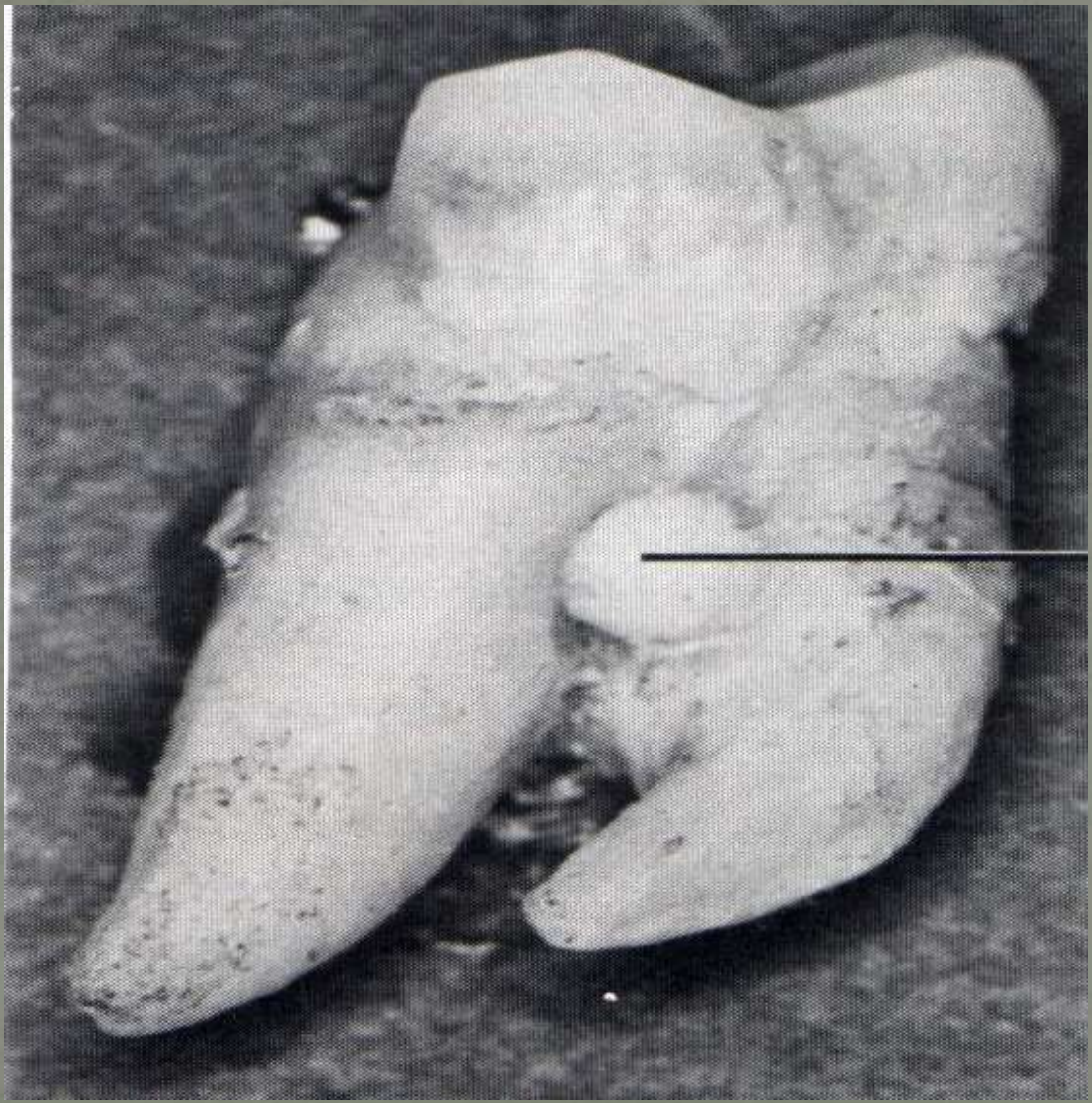


Figure 6–13. (A). Root sheath cells fused to dentin. (B). Area of exposed dentin.

ENAMAL PEARL



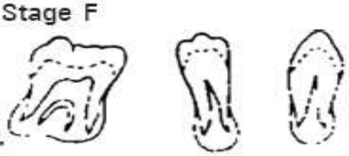
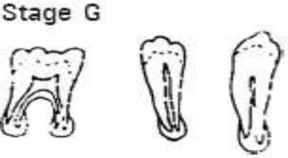
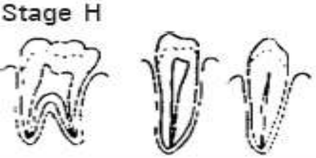
MOLECULAR INSIGHTS IN TOOTH DEVELOPMENT

- Fgf 8- localizes the sites of oral ectoderm and antagonize the Bmp4
- Bmp4 – specify the sites of tooth initiation
- Pax 9 – establish the position of prospective tooth mesenchyme

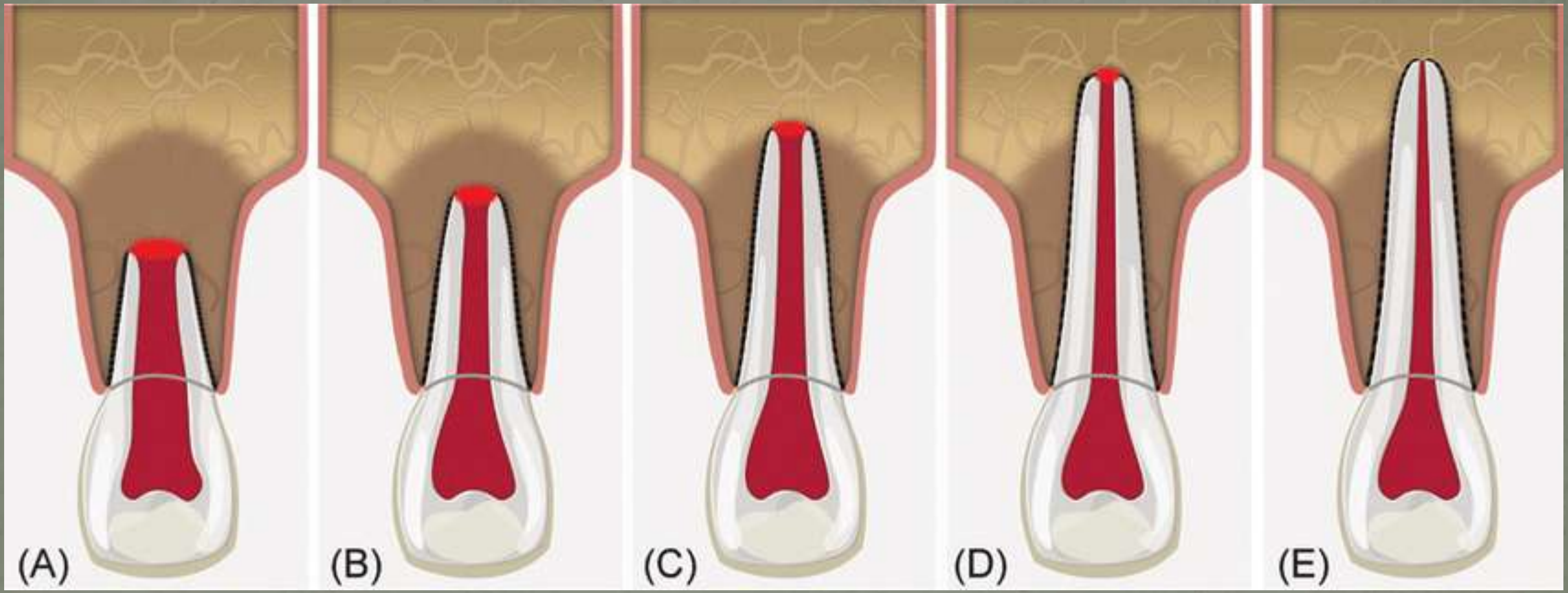
- Incisors- Msx -1, Msx -2
- Canine – Msx – 1, Msx-2, Dlx-2
- Molars – Barx -1 & Dlx -2

NOLLA'S STAGES OF TOOTH DEVELOPMENT

- Stage 0: Absence of crypt
- Stage 1: Presence of crypt
- Stage 2: Initial calcification
- Stage 3: 1/3rd crown completed
- Stage 4: 2/3rd crown completed
- Stage 5: Crown almost completed
- Stage 6: Crown completed
- Stage 7: 1/3rd root completed
- Stage 8: 2/3rds root completed
- Stage 9: Root completed, apex open
- Stage 10: Apical foramen closed

Demirjian stage	Description
Stage 0	No evidence of calcification
Stage A 	Cusp tips are calcified but have not yet fused
Stage B 	Calcified cusps are united, so an outlined occlusal surface is well defined
Stage C 	Enamel formation is complete at the occlusal surface. Dentinal deposition has commenced. The outlines of the pulp chamber are curved
Stage D 	Crown formation is complete to the cementoenamel junction. The pulp chamber in the uniradicular teeth is curved, being concave toward the cervical region. In the molars, the pulp chamber has a trapezoid form. The pulp horns are beginning to differentiate. Root formation is seen
Stage E 	The walls of the pulp chamber are straight and the pulp horns are more differentiated. The root length is less than the crown height. In molars, the radicular bifurcation is visible
Stage F 	The walls of the pulp chamber now form an isosceles triangle. The apex ends in a funnel shape. The root length is equal to or greater than the crown height. In molars, the bifurcation has developed sufficiently to give the roots a distinct outline with funnel shaped endings
Stage G 	The walls of the root canal are now parallel and its apical end is still partially open (distal root in molars)
Stage H 	The apical end of the root canal is completely closed (distal root in molars). The periodontal membrane has a uniform width around the root and the apex

CVEK'S STAGES OF ROOT DEVELOPMENT



Schematic of Cvek's stages of root development. (A) Group I, < 1/2 root length; (B) Group II, 1/2 root length; (C) Group III, 2/3 root length; (D) Group IV, wide open apical foramen and nearly completed root length; (E) Group V, closed apical foramen and completed root development. Groups I, II and III shows wide and divergent apical openings

AMELOGENESIS

- As the preameloblast differentiate to become a secretory ameloblast it also polarizes. Intracellular changes involves lengthening of the cell, proliferation of ER, and redistribution of cellular organelles (basal migration of the nucleus and apical migration of the golgi apparatus).

- As enamel matrix is deposited, the ameloblast migrates in an outward direction and acquires a set of apical and basal terminal bars, as well as a specialized apical process, **Tomes‘ process.**

- Tomes' process can be defined as that part of the ameloblast, apical to the apical terminal bars.

- Tomes process contains numerous secretion granules.Tomes' process can be divided into two portions, a proximal and distal part. The proximal part of Tomes' process contacts adjacent ameloblasts. The distal part also called the interdigitaing part, is surrounded by the enamel matrix.

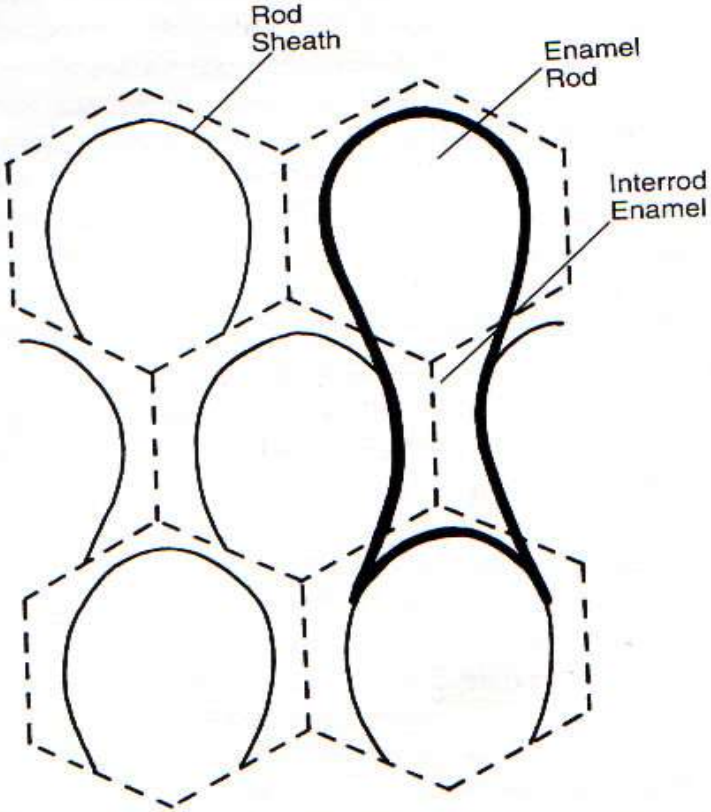


Figure 5–19. (A). Diagram depicting enamel rods. The arcades represent the rod sheaths and a line connecting the open ends encloses an enamel rod; the rest is interrod enamel. The hexagonal profile represents the secretory territory of one ameloblast. Note that it takes 4 ameloblasts to form the outlined keyhole structure but only one forms an enamel rod.

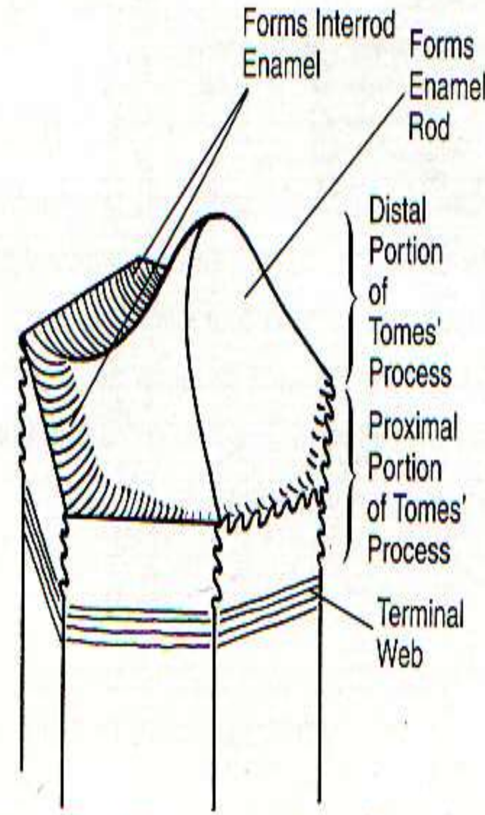


Figure 5–19. (B). Diagram of the Tomes' process of a secretory ameloblast. The distal part projects into the enamel (see also Fig. 5–22) and forms the enamel rod. The proximal part rests on the enamel surface forming interrod enamel.

- The secretary ameloblast like the odontoblast is a polarized cell having a secretory or apical end and a nonsecretory or basal end. It migrates in an outward direction away from the DEJ and secretes enamel. The initial or first-formed enamel is prismatic. As the ameloblast develops and acquires a Tomes' process enamel prisms are formed.

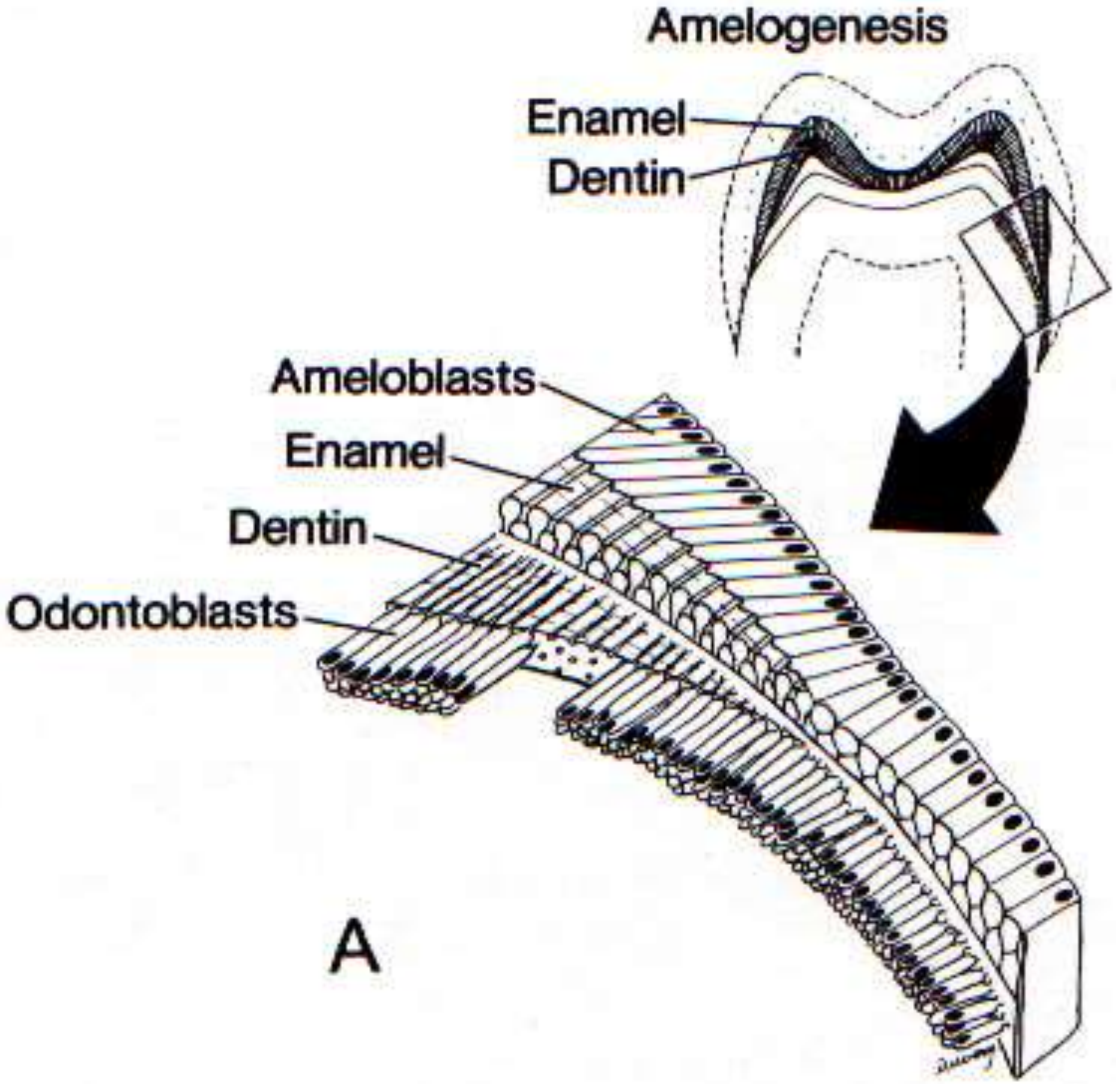


Figure 5–15. Diagram of histodifferentiation within the developing tooth. (A). Sites of initial dentin and enamel formation. (Figure continued on next page).

- Unlike dentin, enamel matrix is partially mineralized as soon as it is secreted. Two major types of proteins, the **amelogenins** and the **enamelins** have been found in the enamel matrix. These proteins are believed to play important roles in the orientation & growth of enamel crystals.

- Amelogenins have been detected in secretory ameloblasts. The nature of enamelins is controversial. Recently, a new enamel protein with the characteristics of enamelin, **tuftlin**, has been found in enamel.

Enameling bind tightly to hydroxyapatite and may in some way regulate crystal growth.

- Unlike dentin, enamel crystals are arranged in a pattern and are much larger than those of dentin.

- **Enamel Rod :**

- Are arranged in keyhole, fish or paddle like pattern. This pattern is not seen near the DEJ nor near the enamel surface. These areas represent areas of prism less enamel.
- When enamel secretion occurs along a flat secretory front, during the initial and final secretion of enamel, there are no prisms in the enamel.

- When enamel secretion occurs by fully developed secretory ameloblasts in the presence of a Tomes' process enamel rods are formed.

- There are two different concepts of an enamel rod. 4 different ameloblasts contribute to the synthesis of one enamel rod or keyhole.With this view there can be no interrod enamel. one enamel rod-one ameloblast. This corresponds to the head of the keyhole and it is made by one ameloblast. The "tail“ would then represent interrod enamel,

- Enamel rod represents the path an ameloblast took during its outward migration from the DEJ.
- Initial enamel is formed when the basal lamina is being eliminated and lacks enamel rods and is aprismatic.
- The bulk of the enamel, inner enamel, is secreted during the secretion stage of amelogenesis when the ameloblast have a fully developed Tomes' process. Enamel rods are formed at this time.

- The Final enamel is produced during the time the Tomes' processes are regressing, and the ameloblast are in the stage of post secretory transition and maturation.

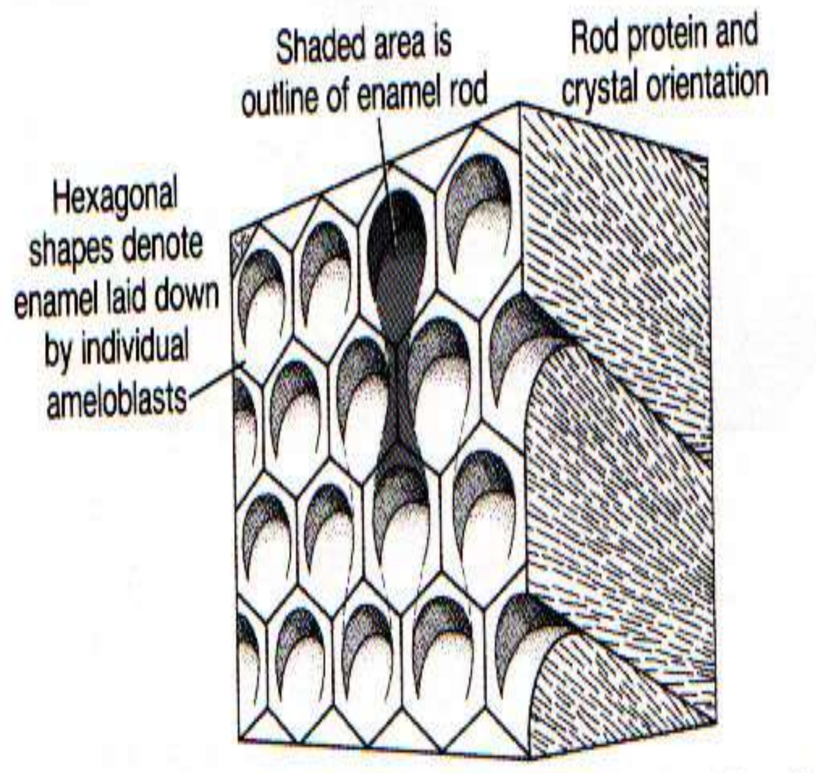


Figure 5–21. Diagram of the emeloblast-enamel rod interface.

Maturation Stage :

- During the process of maturation, enamel becomes fully mineralized. The organic and water content of enamel becomes reduced and the inorganic component (principally hydroxyapatite) increases.
- Enamel matrix becomes mineralized as soon as formed and continues to mature until completely developed. This feature distinguishes it from dentin and bone.
- During the secretion stage, enamel nearest the DEJ, being developmentally older, is more mineralized

or mature. This stage is first recognized by the formation of a ruffled apical border in ameloblasts.

- Spaces between enamel crystals diminish in size with an increase in crystal diameter and length. The enamel rod or head of the rod appears to contain the most mineral in later maturation. spaces between rods, which indicates a lower mineral content.
- Mature rod size is 5 X 9µm. Final enamel trickiness (from 2 to 2.5 mm over the cusps) is attained after the ameloblast completes enamel formation.

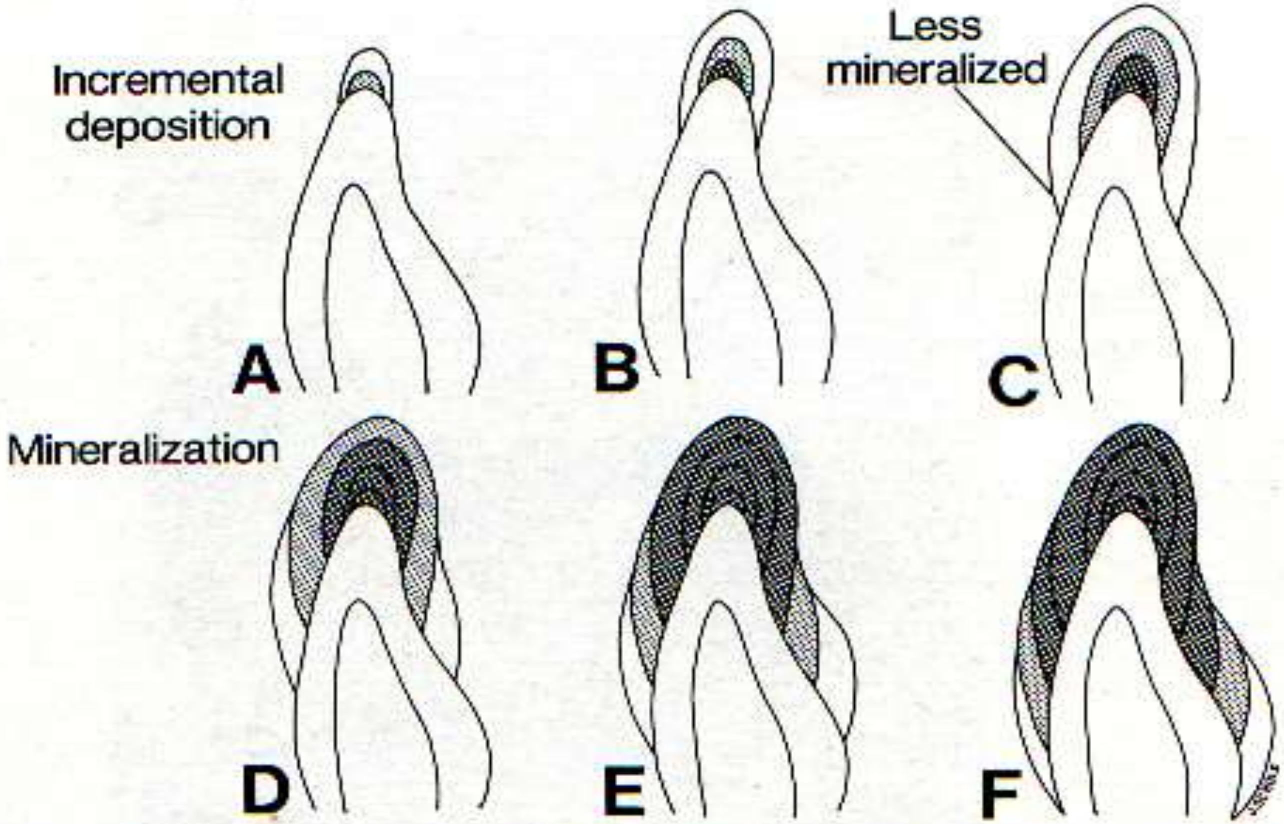


Figure 5–26. Summary of the stages of enamel mineralization. Initial enamel is formed in A and becomes more mature (more calcified) in B as further matrix is formed. (C). Further increments are formed. (D). Mineralization and matrix deposition increases. (E). Enamel matrix is formed on the side of the cusps. (F). Final matrix is formed and progresses cervically.

Dentinogenisis :

- Oval or polygonal cells located near the basal lamina That Separates the enamel organ from the dental papilla are the Preodontoblast. These cells elongate and become young Odontoblast.

- In the process of differentiation, odontoblasts become columnar polarized cells having a secretary or apical pole and a nonsecretory or basal pole.

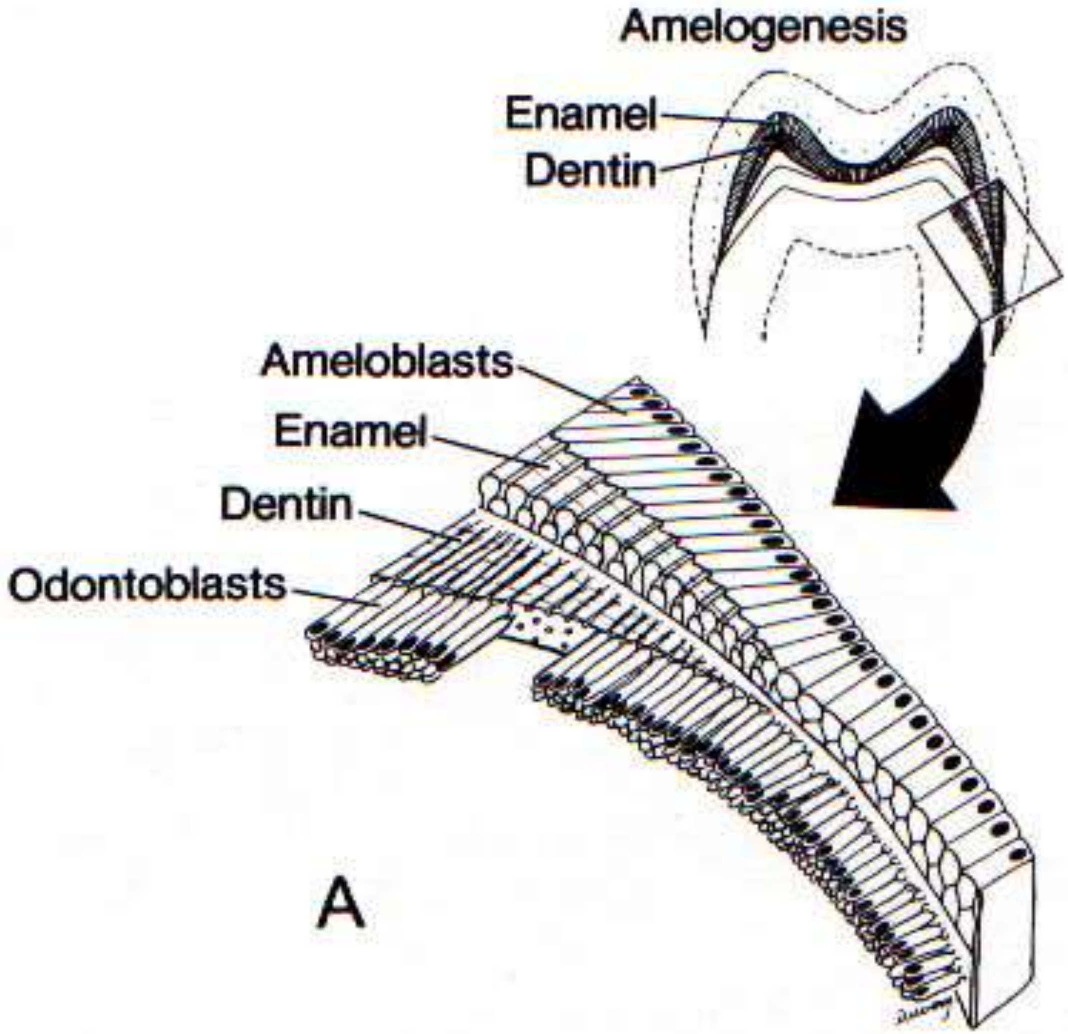


Figure 5–15. Diagram of histodifferentiation within the developing tooth. (A). Sites of initial dentin and enamel formation. (Figure continued on next page).

- An extensively developed endoplasmic reticulum and Golgi apparatus are characteristic of the active secretory odontoblast.

- The odontoblast process, also known as **Tomes' fiber**, becomes embedded in the extra cellular matrix of the predentin & elongates as the odontoblast retreats from the ameloblast layer of the enamel organ.

- As the odontoblast elongates their nuclei occupy a basal position in the cell, & the organelles become more evident towards the apical ends of the cell.

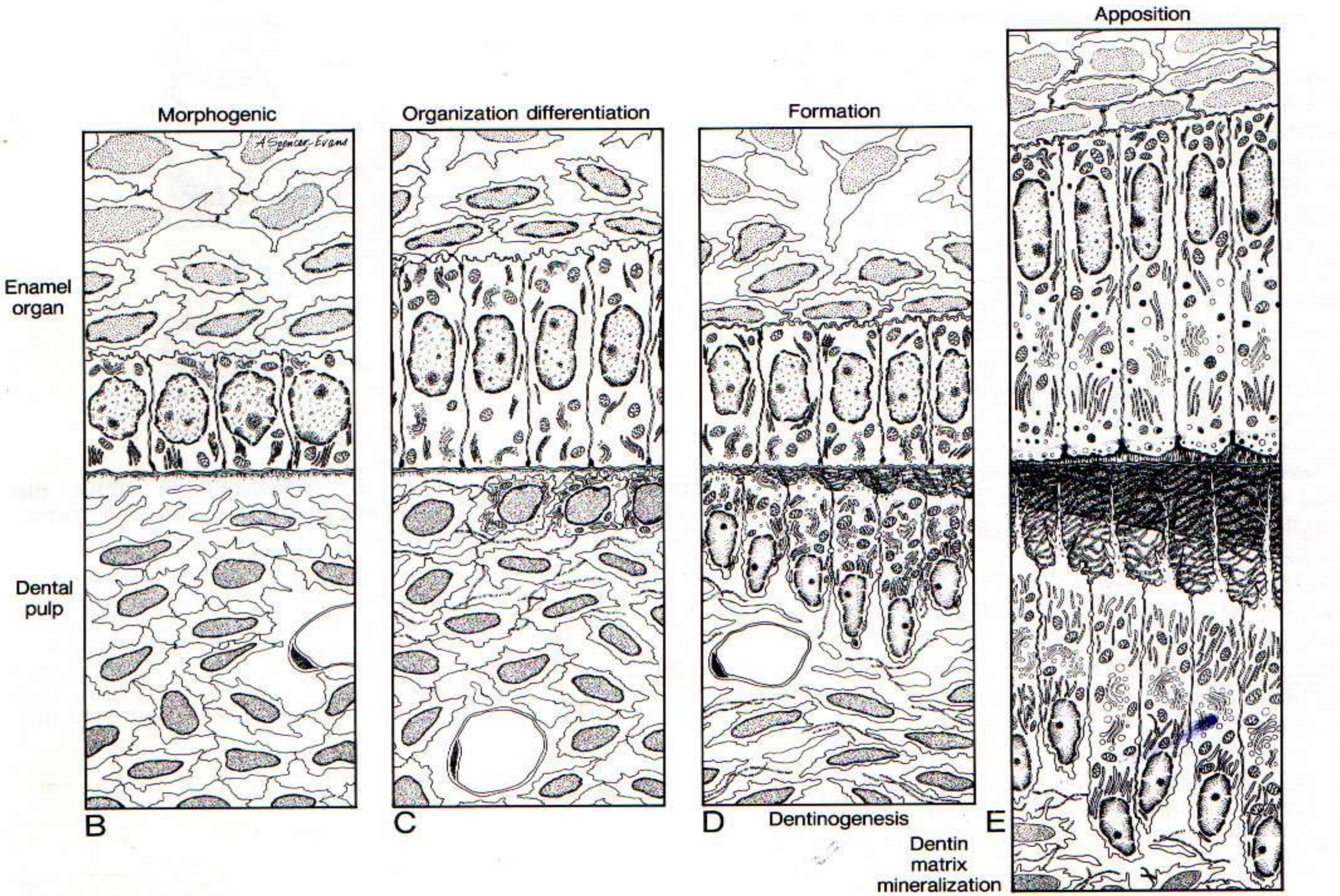


Figure 5–15. *Continued.* (B to D). Represent the early stages of odontoblast differentiation and dentin formation. (B to I). Represent important stages of ameloblast differentiation and the formation of enamel. Note in G and H the ameloblast differentiation and the formation of enamel. Note in G and H that the ameloblasts modulate alternating these forms during amelogenesis.

- Odontoblast immediately begin forming the collagenous dentin matrix which is not mineralized when it is first formed & is therefore termed Predentin.

- During differentiation some of the first formed collagen fiber passes between the differentiating odontoblast & extend towards the basal lamina where they appears to end in fan like arrangement “**kroffs fibers**”. As this collagen matrix formation proceeds the odontoblast move away from what will become the DEJ.

- The ends of odontobastic processes maintain their positions and as a result the odontobastic process lengthens. The dentin-enamel junction lies at the former junction between the inner enamel epithelium and dental mesenchyme.The matrix which forms around the elongated cell processes eventually mineralizes and the odontobastic process will lie within a dentinal tubule.

Mineralization:

- Of the first-formed predentin is thought to occur in one of two ways:

1. Small mineral crystals appear, and mineralization spreads from these sites throughout the first-formed predentin.
2. Small mineral crystals are nucleated in spaces which exist in collagen fibrils ,Crystals are oriented along the long axis of these fibrils. These minute crystals grow and spread throughout the predentin, until the newly formed band of collagen along the pulp is uncalcified.

- As each daily increment of predentin forms along the pulpal boundary, the more peripheral adjacent predentin, which formed during the previous

day, mineralizes and becomes dentin.

- As the predentin calcifies and becomes dentin the dentin-predentin junction becomes established. Following the establishment of the dentin-predentin junction, the dental papilla becomes the dental pulp.
- Predentin is continuously formed along the pulpal border during crown formation and following eruption, and is calcified along the predentin-dentin junction This results in a decrease in the volume of the pulp cavity.

- During the period of crown development and during eruption, approximately 4 μm of dentin is laid down daily. After the teeth reach occlusion, the deposition rate decreases to a level of less than 1 μm per day.
- The amount of predentin formed each day along the pulpal boundary is termed an increment.
- Incremental deposition and mineralization of dentin begins at the tips of the pulp horns at the DEJ and proceeds by the rhythmic deposition of layers in the cusps until the crowns are completely formed

GENES INVOLVED

- MAP1B – odontoblast differentiation
- PHEX - Dentin mineralization

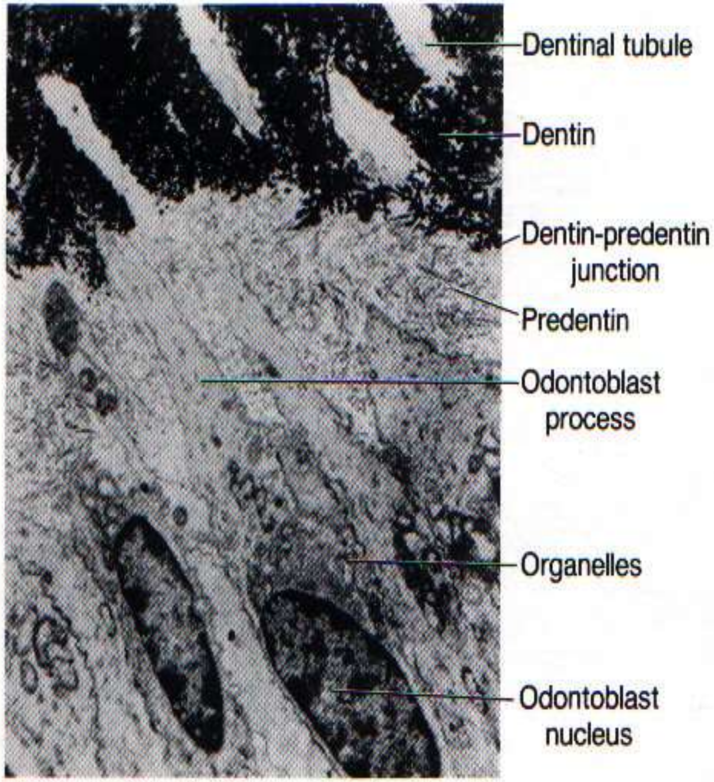


Figure 5–17. Transmission electron micrograph of the mineralization front or dentin-predentin junction.

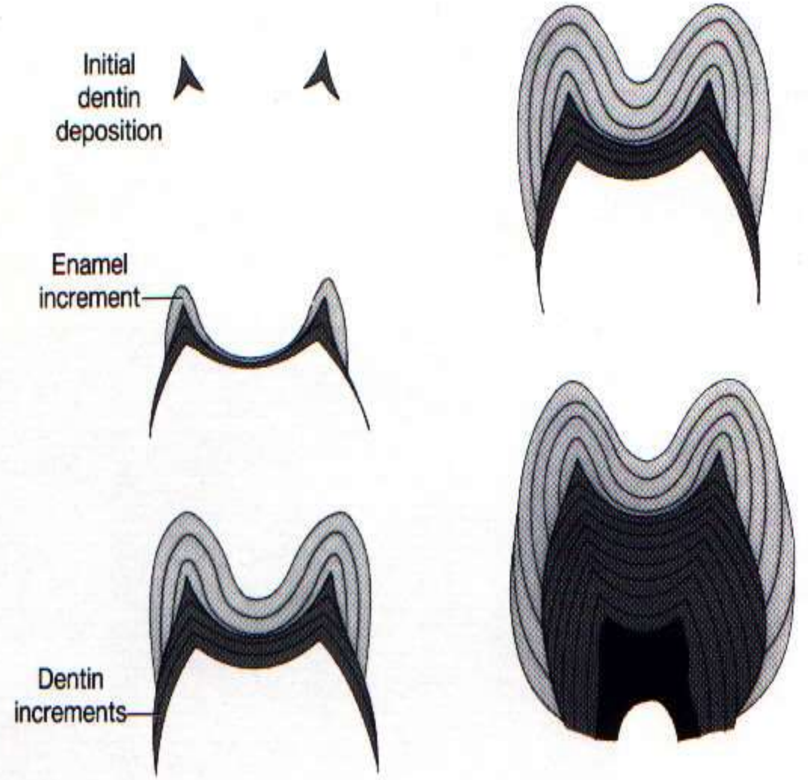


Figure 5–18. Diagram depicting the incremental deposition of dentin and enamel.

Developmental Disorders of Teeth :

1. Size of teeth : Microdontia & Macrodontia.
2. Shape of teeth : Gemination, Fusion, Concrescence, Dilaceration, Talon cusp, Dense in dent, Dens evaginatus & taurodontism.
3. Number of teeth : Anodontia, Supernumerary teeth, Predeciduous dentition .
4. Structure of teeth : Amelogenesis Imperfecta, Environmental enamel hypoplasia, Dentinogenisis Imperfecta & Dentin Dysplasia.

Developmental Disturbance in size of Teeth

1.Microdontia

a.True generalized microdontia

b.Relative generalized microdontia

c.Microdontia involving single tooth.

2.Macrodontia

Developmental Disturbance in shape of Teeth

Gemination, Fusion, Concrescence,Dilaceration, Talon cusp, Dense in dent, Dens evaginatus & Taurodontism.

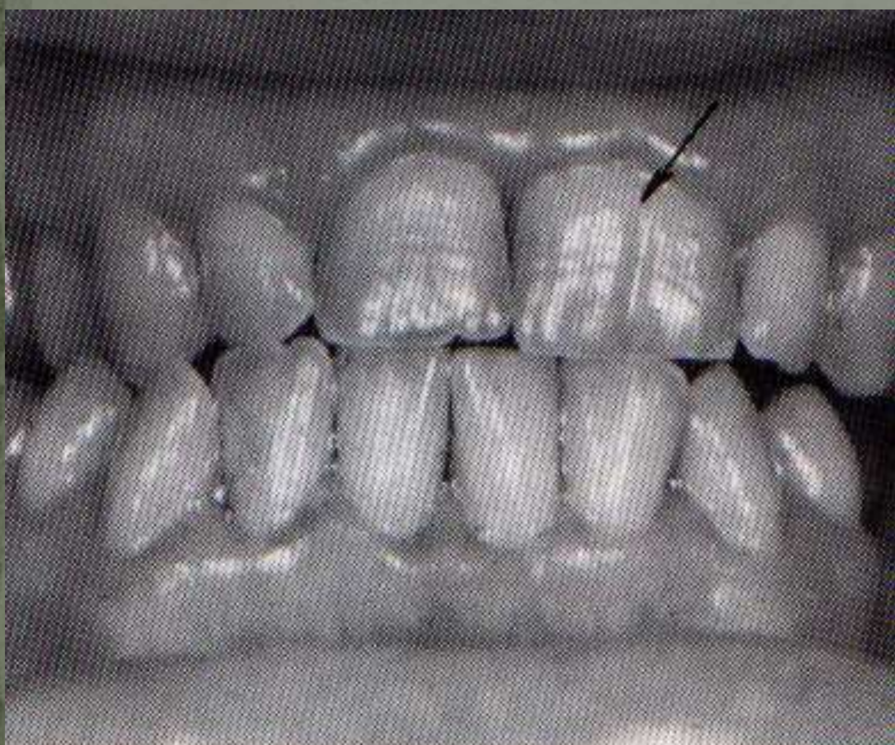


Figure 1–31. *Fusion of teeth.*

Figure 1–32. *Dilaceration.*
Examples of various types of curves and angles involving roots of teeth.

Figure 1–33. ***Talon cusp.***

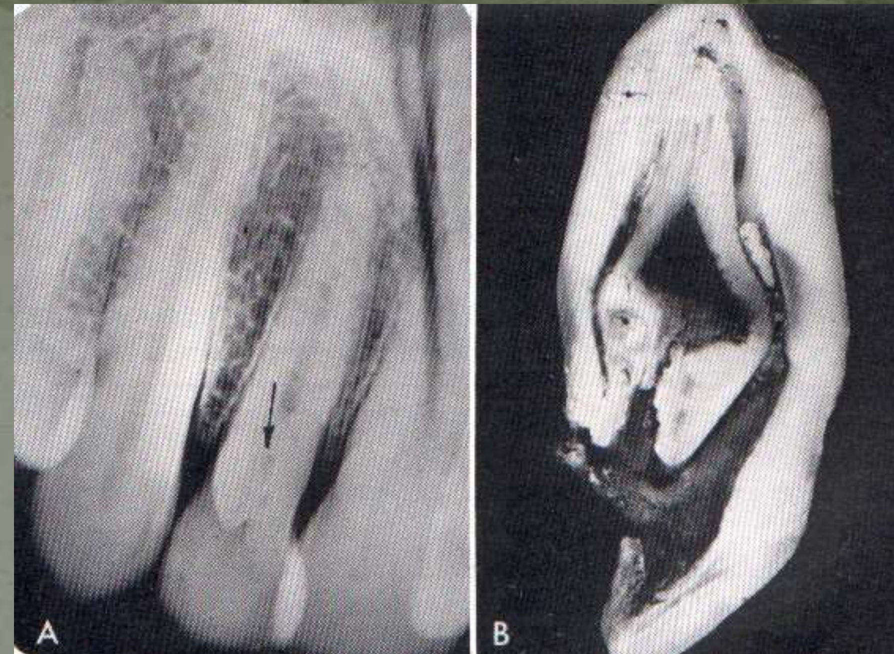


Figure 1–36. ***Dens evaginatus.***

Figure 1–37. ***Taurodontism.***

Developmental Disturbance in number of Teeth

Anodontia, Supernumery teeth, Predecidious dentition & Post permanent dentition.

Figure 1–42. *Supernumerary tooth.*
A, The supernumerary tooth between the maxillary permanent central incisors is called a mesiodens. *B*, The intraoral roentgenogram.

Developmental Disturbance in structure of Teeth

1. **Amelogenesis Imperfecta**

- It is an hereditary defects of enamel. It is ectodermal disturbance, since the mesodermal components of the teeth are normal.
- Development of enamel occurs in three stages : **a.** Formative stages, **b.** Calcification stage,

  c. Maturation stage.
- Accordingly 3 basic types of Amelogenesis Imperfecta are recognized :
- **a.** Hypoplastic type, **b.** Hypocalcification type,

  c. Hypomaturation type. (**C/F**)

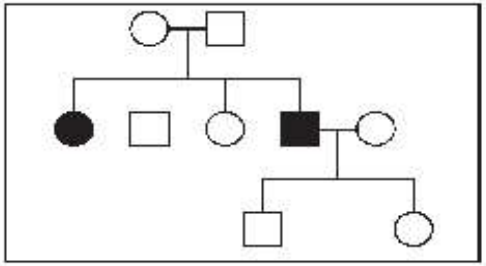
CLASSIFICATION

Weinmann *et al.*, 1945 [4]	*Two types based solely on phenotype: hypoplastic and hypocalcified*
Darling, 1956 [5]	*Five phenotypes based on clinical, microradiographic and histopathological findings.* Hypoplastic Group 1 – generalised pitting Group2 – vertical grooves (now known to be X-linked AI) Group 3 – Generalised hypoplasia Hypocalcified Type 4A – chalky, yellow, brown enamel Type 4B – marked enamel discolouration and softness with post-eruptive loss of enamel Type 5 – generalised or localised discolouration and chipping of enamel
Witkop, 1957 [6]	*Classification based primarily on phenotype. 5 types:* 1. Hypoplastic 2. Hypocalcification 3. Hypomaturation 4. Pigmented hypomaturation 5. Local hypoplasia Added mode of inheritance as further means of delineating cases.
Schulze, 1970 [7]	*Classification based on phenotype and mode of inheritance.*

Amelogenesis imperfecta: a classification and catalogue for the 21st century. Oral Diseases.2007

Amelogenesis Imperfecta

Autosomal Recessive



ENAM mutation

Hypomineralized ARAI

Mutations

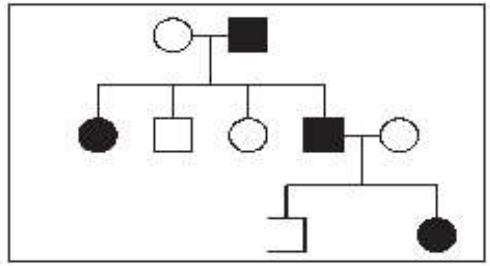
- *KLK4*
- *MMP20*

Hypoplastic ARAI

Mutations

- *ENAM*

Autosomal Dominant



FAM83H mutation

Hypoplastic ADAI

Mutations

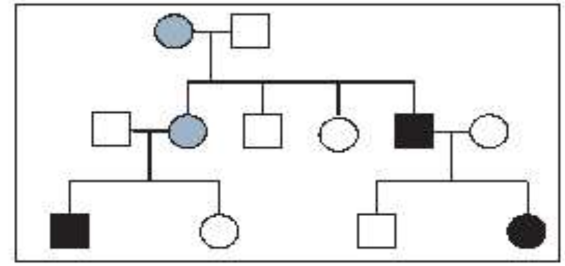
- *ENAM*

•Hypocalcified ADAI

Mutations

- *FAM83H*

X-linked



AMELX mutation

Hypoplastic XLAI

Mutations

- *AMELX*

Fig. 2.1 Amelogenesis imperfecta (hypoplastic type): the enamel is thin, rough in texture and stained but what little there is, is hard and relatively normal in structure. Three sisters were similarly affected and the trait has been traced back through three generations.

Fig. 2.2 Amelogenesis imperfecta (sex-linked dominant type): in this affected female, the typical vertically ridged enamel can be seen.

2. Environmental Enamel Hypoplasia :

"May be defined as an in complete or defective formation of organic enamel matrix of teeth".

- To basic types of Enamel Hypoplasia exist
- a. Hereditary type,

  b.Those caused by environmental factor.
- In hereditary type both decidious & permanent dentitions are usually involved & generally only the enamel is affected. In contrast when the defect is caused by environmental factor, either dentition may be involved & some times only a single tooth. Both enamel & dentin are affected.

- Environmental factor which are responsible for causing enamel hypoplasia are.

1.Nutritional deficiency:Vit-A,C & D.

2.Exanthematous disease: measles, chicken pox, scarlet fever.

3.Congenital syphilis

4.Hypocalcemia

5.Birth injury, Rh incompatibility

6.Local infection or trauma

7.Fluorides

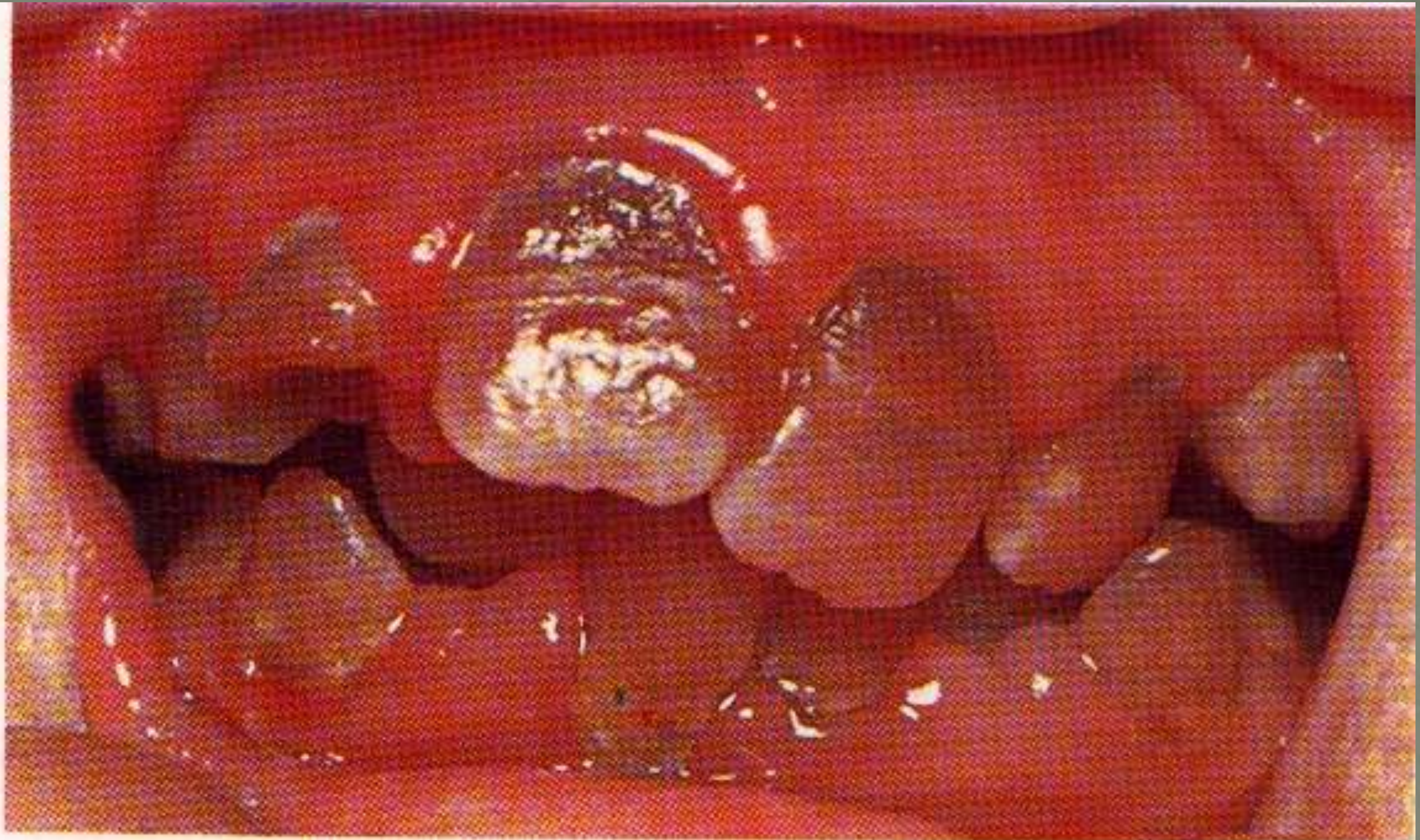


Fig. 2.26 Erythropoietic porphyria: the characteristic reddish-brown stain of the enamel is produced by deposition of porphyrin in the developing dental tissues. There was also extensive facial scarring.

Enamel Hypoplasia due to Fluoride (Mottled Enamel)

- It is caused by ingestion of fluoride containing drinking water during the time of tooth formation. Severity of mottling increases with increasing amount of fluoride in water. There is little mottling of any significance at a level below 0.9 to 1 ppm.
- This type of hypoplasia is due to a disturbance of the ameloblast during the formative stage of tooth development.

- Depending upon the level of fluoride in the water supply. There is a wide range of severity in the appearance of mottled teeth varying from

1. White flecking or spotting of enamel.
2. Mild changes manifested by white opaque areas involving more of the tooth surface area.
3. Moderate & sever changes showing pitting & brownish staining of the surface.
4. Corrode appearance of the teeth.

Fig. 2.28 Mottled enamel (fluorosis): in this severe example, the enamel has widespread pitting defects and brown discoloration.

Fig. 2.27 Mottled enamel (fluorosis): in this moderately severe case the teeth show paper-white opaque areas and a little brown stain. The texture of the enamel is otherwise normal.

Dentinogenisis Imperfecta :

Type I: always occurs in families with osteogenesis Imperfecta.

Type II: never occurs in families with osteogenesis imperfecta. Most frequently referred as Hereditary opalesent dentin. It is most common dominant inherited disorder in human.

Type III: Is Brandy wine type, characterized by multiple pulp exposure in decidious teeth.

"DSPP" gene involved from chromosome no. 4

- Decidious teeth are more severely affected than the permanent teeth in type I. In type II & III both dentition are affected.
- Colour of teeth may range from brownish yellow & exhibit a characteristic translucent or opalescent hue.
- R/F most striking feature is the partial or total obliteration of the pulp chamber & root canals by continued formation of dentin.
- The affected patients in type III DI had features characterised as “Shell Teeth.” enamel of the tooth appears normal, while dentin is extremely thin, & the pulp chamber are enormous.

Fig. 2.5 Dentinogenesis imperfecta: the teeth are greyish in colour but, unlike the tetracycline-stained teeth in Fig. 2.29, are abnormally translucent.

Fig. 2.7 Dentinogenesis imperfecta: in this 14-year-old, the teeth have worn down to gum level but the pulp chambers have become obliterated as part of the disease process. Some enamel remains around the necks of the posterior teeth.

Dentin Dysplasia

Characterized by normal enamel but atypical dentin formation with abnormal pulp morphology. (hereditary)

- Type I: Radicular dentin dysplasia

Both dentition are affected. Teeth exhibit extreme mobility & are commonly exfoliated prematurely or after minor trauma as a result of short roots.

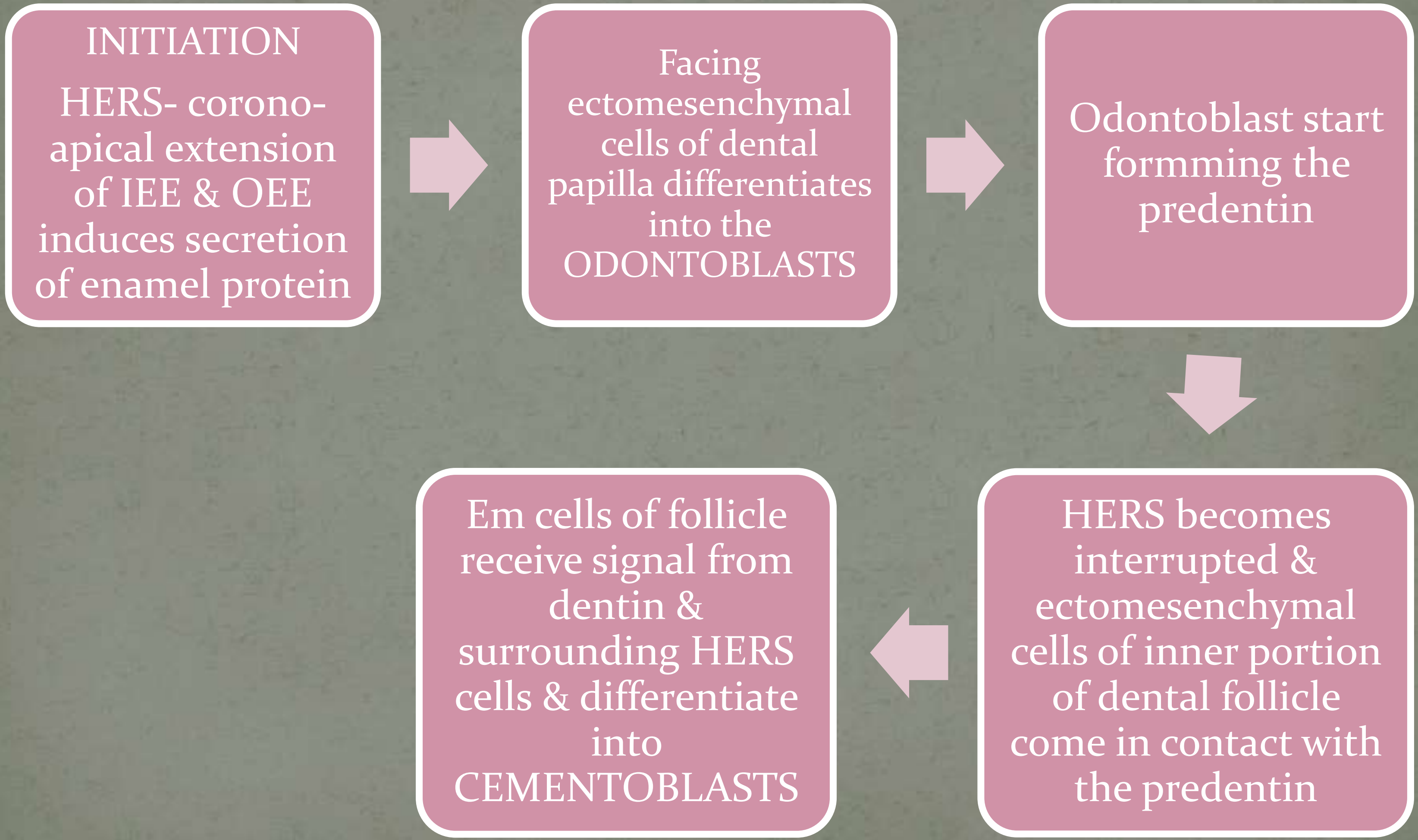
- Type II: Coronal dentin dysplasia

- Abnormal large pulp chamber in coronal portion often described as Thistle tube

- Dentin dysplasia Type 1
  - Enamel and coronal dentin are normal.
  - Radicular dentin loses it's organization.
  - Permanent > Deciduous
  - Extreme mobility , Exfoliation , Delayed Eruption.
  - H/F : STREAM / LAVA flowing Across Boulders appearance.
- R/F : Primary teeth are more affected. Roots are short / absent.
- Little or no detectable pulp.

- Dentin dysplasia Type 2
  - Synonyms : Coronal Dentin Dysplasia.
  - Root length is normal in both dentition.
  - Tooth demonstrate a blue to amber to brown translucency.
  - H/F : Radicular dentin is atubular , amorphous and hypertrophic.
  - Pulp stones develop
- R/F : Bulbous crowns. Cervical constriction. Thin roots.
- Alteration of pulp as tube or Flame shaped

CEMENTOGENESIS



MOLECULAR FACTORS REGULATING CEMENTOGENESIS

MOLECULE	BIOLOGICAL ACTIVITY
IGF- 1	Proliferation ,Differentiation, Matrix synthesis
FGF	Proliferation ,Differentiation, Matrix synthesis, Angiogenesis
PDGF	Proliferation ,Differentiation, Matrix synthesis
TGF- beta	Matrix synthesis, Angiogenesis, Chemotaxis
BMPs	Differentiation, Matrix synthesis, Bone formation
EGF	Proliferation ,Differentiation
CGF	Proliferation ,Differentiation

MATRIX COMPONENT	
Collagen	Cell adhesion, differentiation , regulates proliferation
BSP	Cell adhesion, differentiation , Mineralization
OPN	Cell adhesion, regulates differentiation & survival
Fibronectin	Cell adhesion, differentiation , regulates proliferation
osteonectin	Regulates Angiogenesis, differentiation & proliferation
Cementum- attachment protein	Cell adhesion, differentiation

Agents affecting Tooth Development

Introduction:

- Certain vitamin & hormone deficiencies if present during tooth formation will adversely affect formative cells & matrix that they produce.
- Reduced organic matrix content results in production of hypoplastic tissue.
- Excessive levels of tetracycline or fluoride may become incorporated into mineralizing teeth & interfere with the mineralization process.

- The extent of defect depend on the nature of the substance, the degree of excess or deficiency and the developmental time.
- Vitamin-A, C, D, Parathyroid hormone, Tetracycline & Fluoride are discussed in terms of their relation to matrix development.

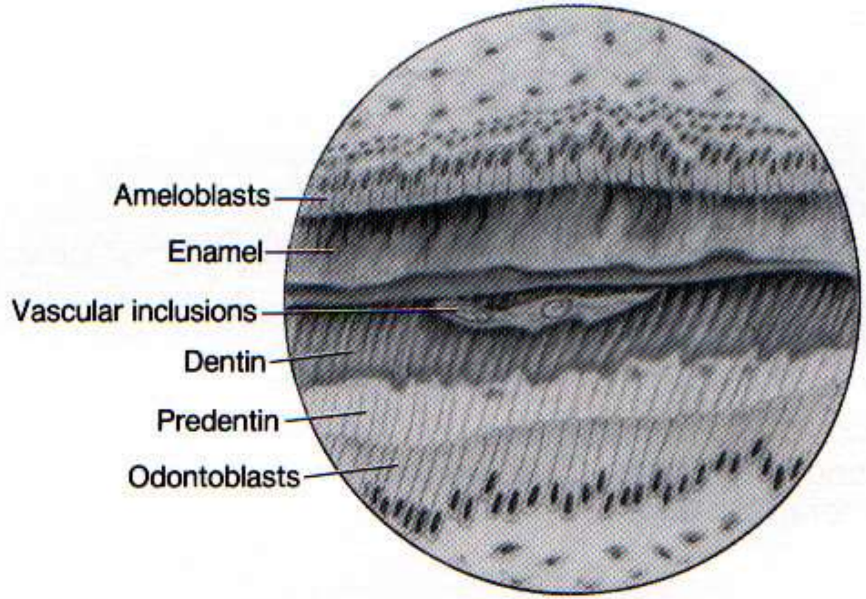


Figure 8–2. Illustration of vitamin A deficiency indicates shortened ameloblasts, enamel matrix deficiency, and vascular inclusions in dentin at the dentinoenamel junction.

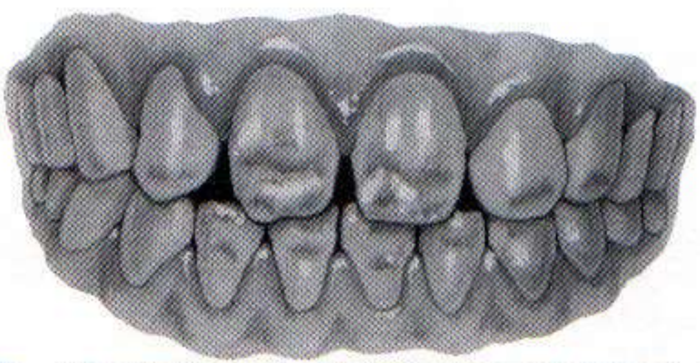


Figure 8–3. Illustration of clinical view of defective enamel resulting from vitamin A deficiency.

Figure 8–5. Appearance of defective dentin formation resulting from vitamin C deficiency, with vascular inclusions and degenerated odontoblasts.

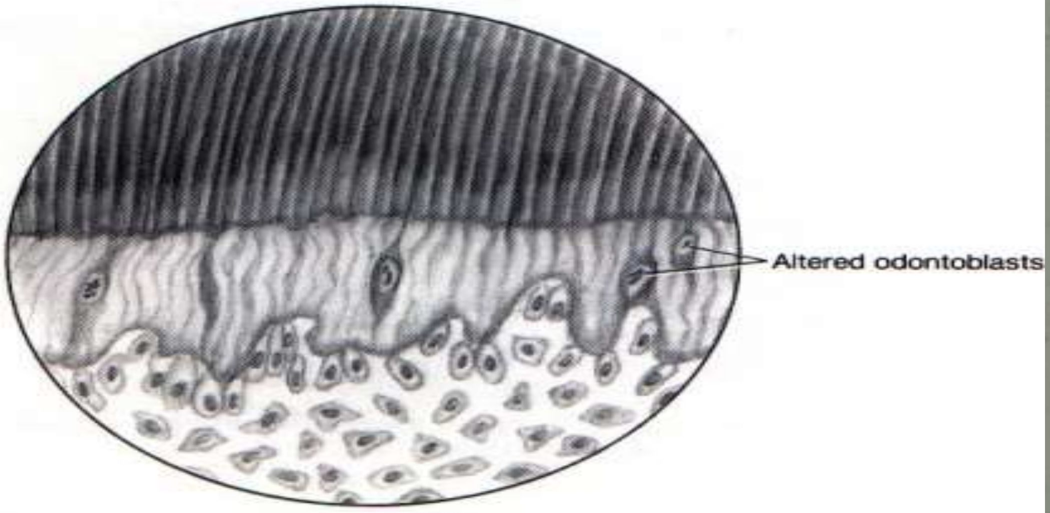


Figure 8–6. Illustration of vascular inclusions and altered odontoblasts resulting from vitamin C deficiency.

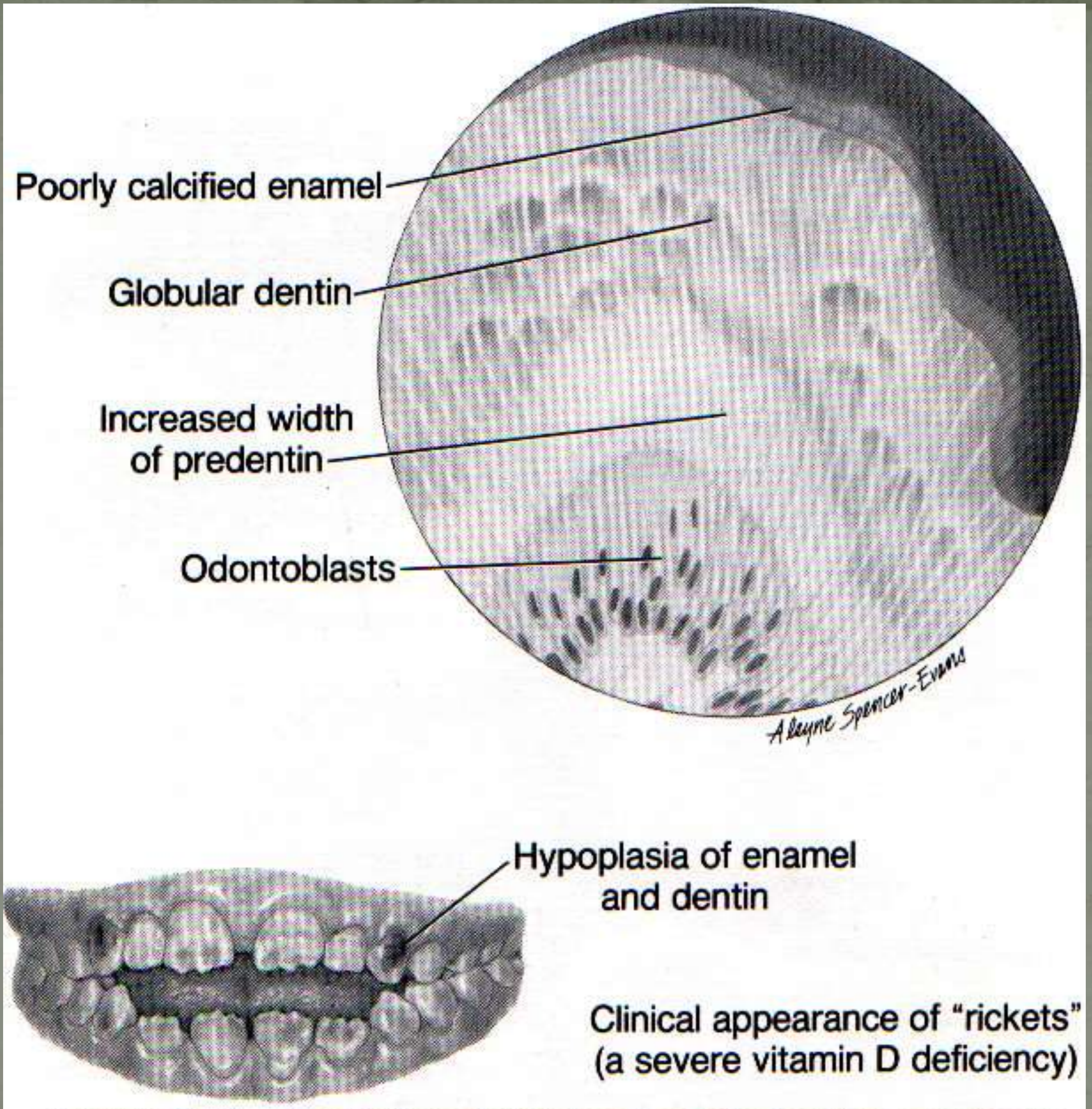


Figure 8–7. Illustration of histology and clinical view of vitamin D deficiency.

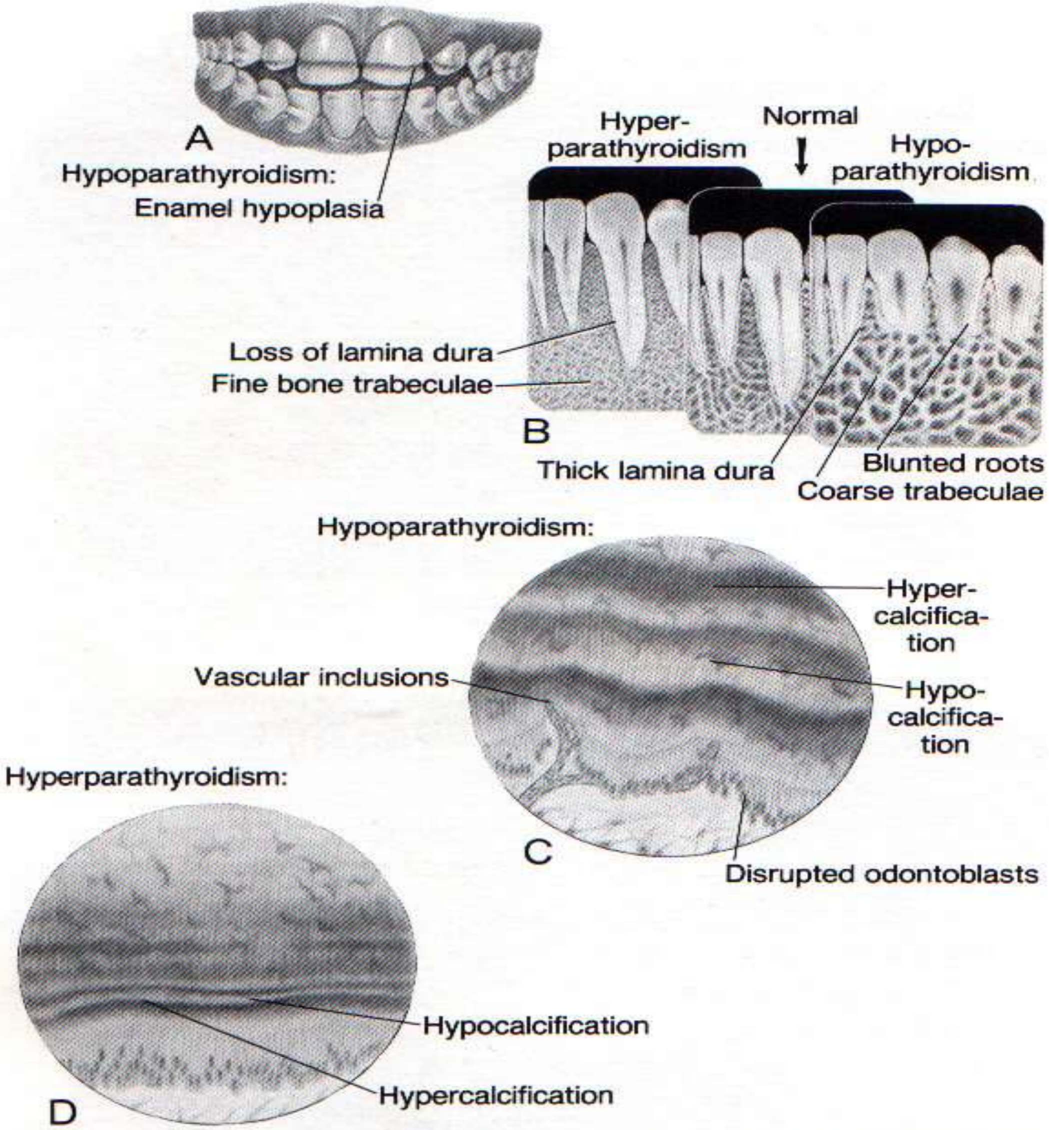


Figure 8–9. (A). Illustration of clinical effects of parathyroid hormone. (B). Diagrams of radiographs indicate altered supporting bone. (C) and (D) are the histological appearance of alternating bands of hypocalcified and hypercalcified dentin. These bands may demonstrate the clinical appearance of hypocalcified tooth and bone.

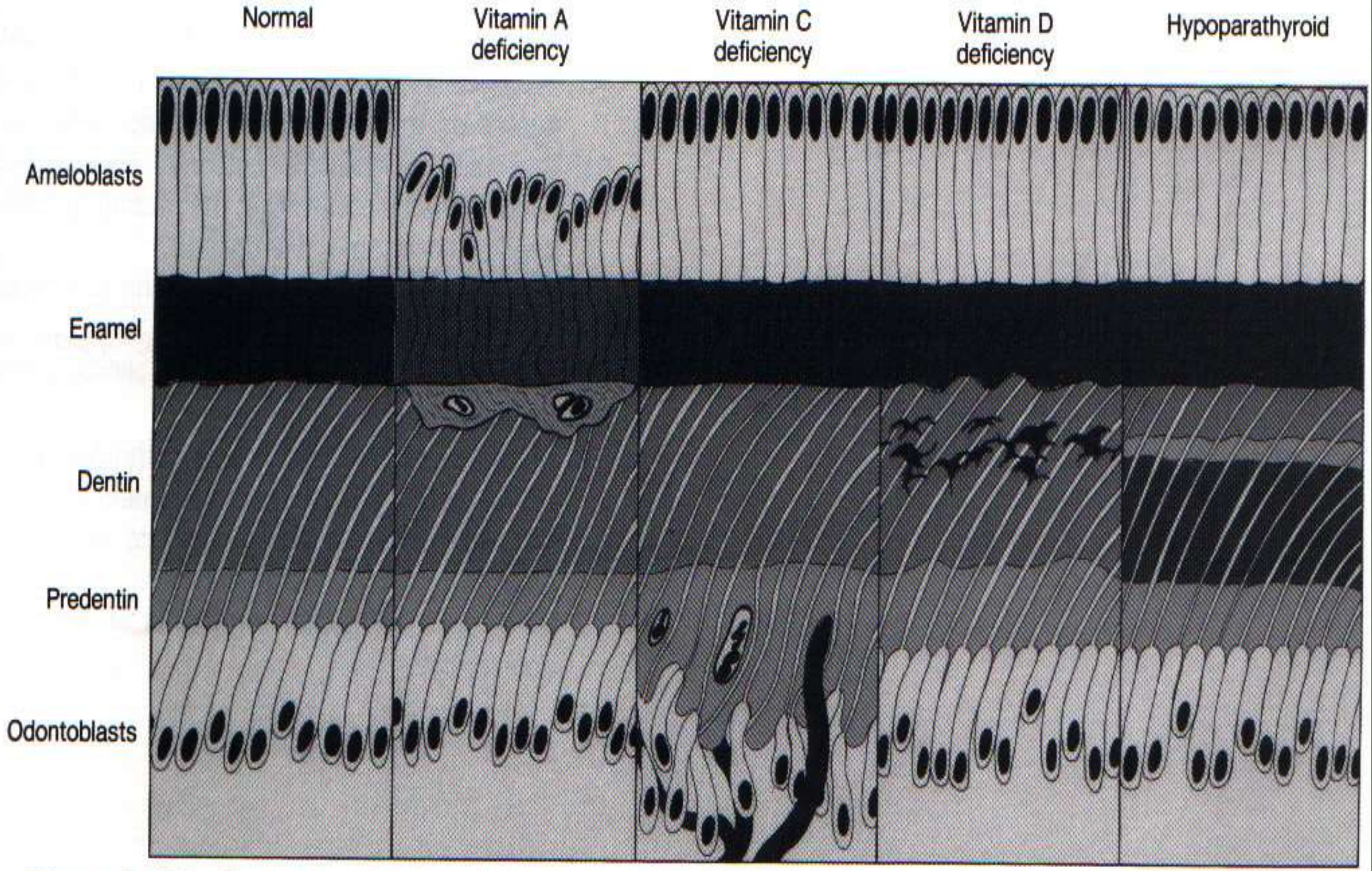


Figure 8–20. Summary of effects caused by vitamin A, C, and D deficiencies and hypoparathyroidism.

Tetracycline & Fluoride:

- If available during the mineralization phase, may be incorporated in dentin, enamel, cementum & bone.
- Fluoride is a binary compound of fluorine useful as an anticaries substance. Tetracycline on the other hand is used as an antibacterial agent. Both are deposited along with minerals in developing hard tissue.
- On prolonged exposure to light, tetracycline stained dental tissue will change in colour to

- A brown to gray. Other effects of tetracycline is hypoplasia or absence of enamel. Staining is most notable in dentin, especially in the first formed dentin at the DEJ. Staining is more notable under UV light.
- Amount of damage is directly related to the magnitude & duration of the dosage.

Mechanism: It is believed that a chelate of calcium & tetracycline forms. At higher concentration in both ameloblast & odontoblast protein synthesis is impaired. This in turn will result in hypoplasia of the enamel & dentin matrix.

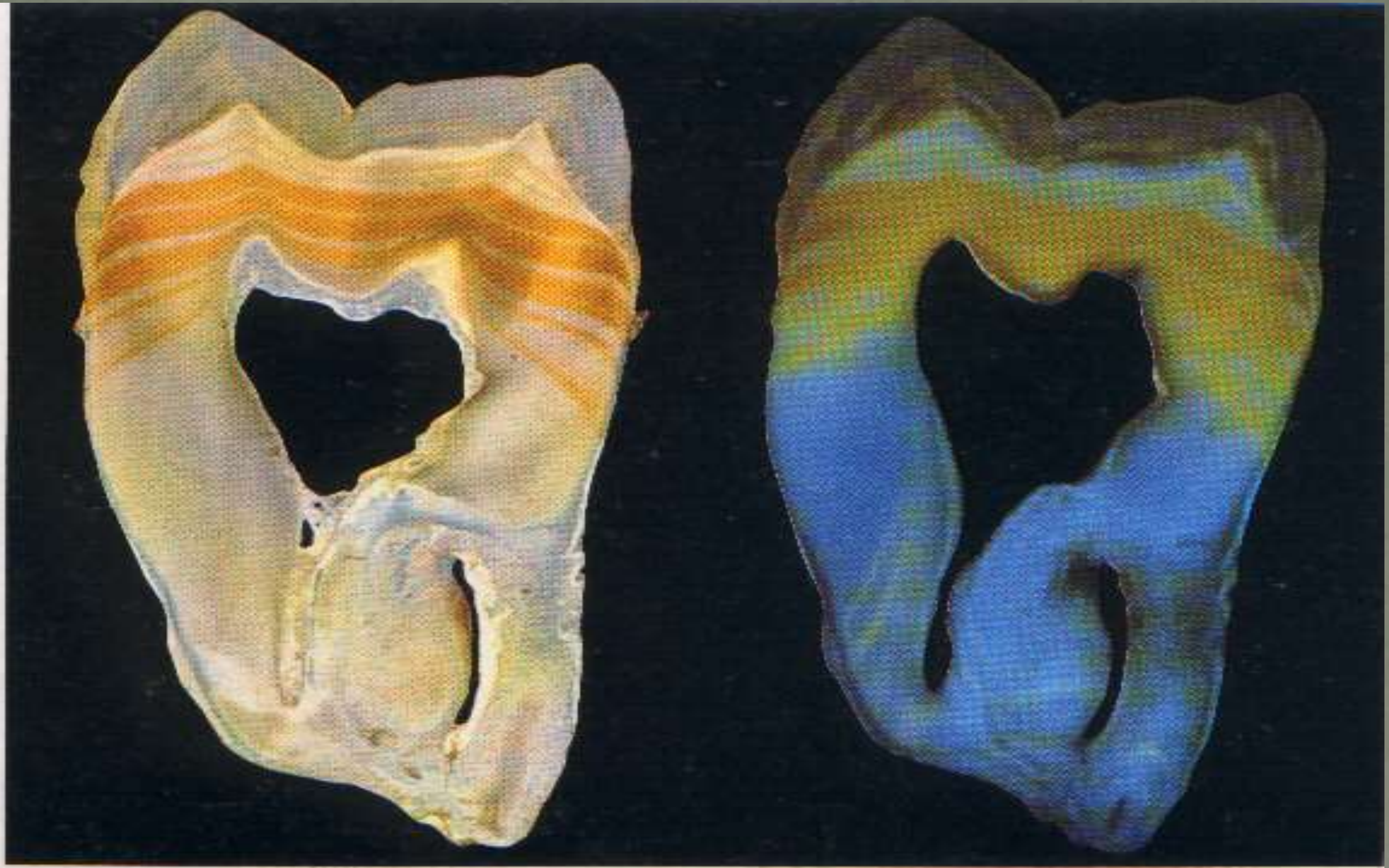


Fig 2.30 Tetracycline pigmentation: hard section (left) shows the broad bands of tetracycline deposited along the incremental lines of the dentine; (right) same tooth viewed under ultraviolet light shows fluorescence of the bands of tetracycline.

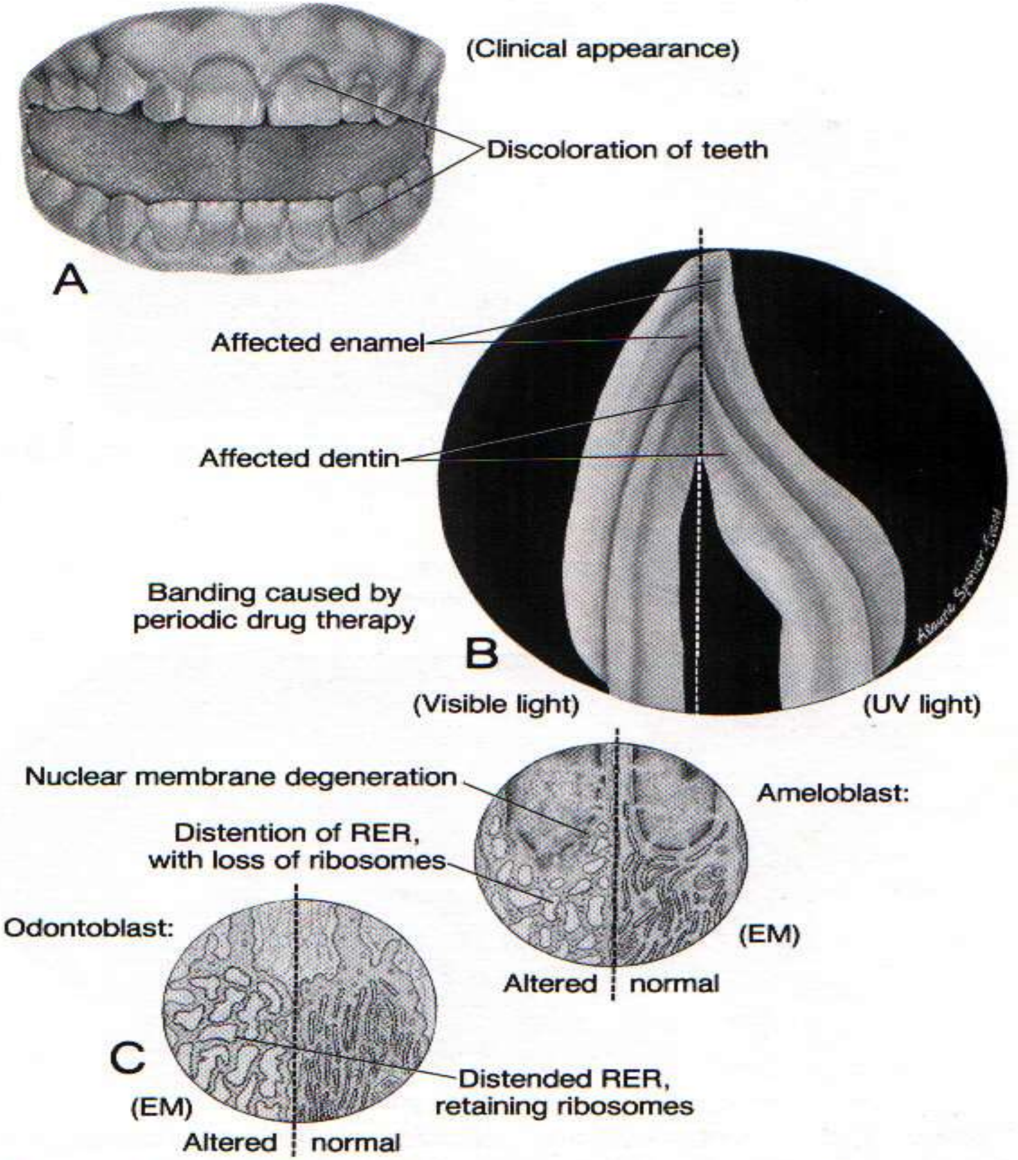


Figure 8–11. Effects of tetracycline in human teeth result in staining and hypoplastic enamel. Staining is evident in first-formed dentin. Both ameloblast and odontoblast endoplasmic reticula are altered.

- Tetracycline & to limited extent Sodium Fluoride cross the placental barrier.
- If pregnant female consumes fluoridated water during mineralization of the fetal teeth. Such teeth exhibit higher resistance to dental caries.
- If on the other hand tetracycline antibiotics are administered to the mother during the period of tooth mineralization, the decidious teeth may later be stained. Tetracycline staining of teeth is permanent, but staining of bone is not permanent as bone is remodeled continuously.

- Cervical staining is more characteristic of tetracycline because this agent deposits primarily in the dentin.

- Fluoride is most beneficial to the teeth in concentration of approximately 0.5 to 1 ppm of water. Higher concentration such as 5 ppm causes mottling & hypoplasia of the enamel & hypo mineralized dentin.

- Less amount of fluoride is being found in primary teeth than in permanent teeth because the maturative stage last for 1-2 years in primary teeth & 4-5 years in permanent teeth.

Fig. 2.29 Tetracycline pigmentation: the newly erupted permanent teeth are yellow the deciduous teeth have changed to a di grey. Tetracycline had been given for long periods during the mother's pregnancy ar during the child's infancy.

- Tetracycline has been widely used to visually record growth in experimental animal. Daily deposition of dentin can thus be recorded by measuring the width of dentin between each fluorescent line.

- Tetracycline may be used to evaluate tooth movement by reveling bone & dentin formation.

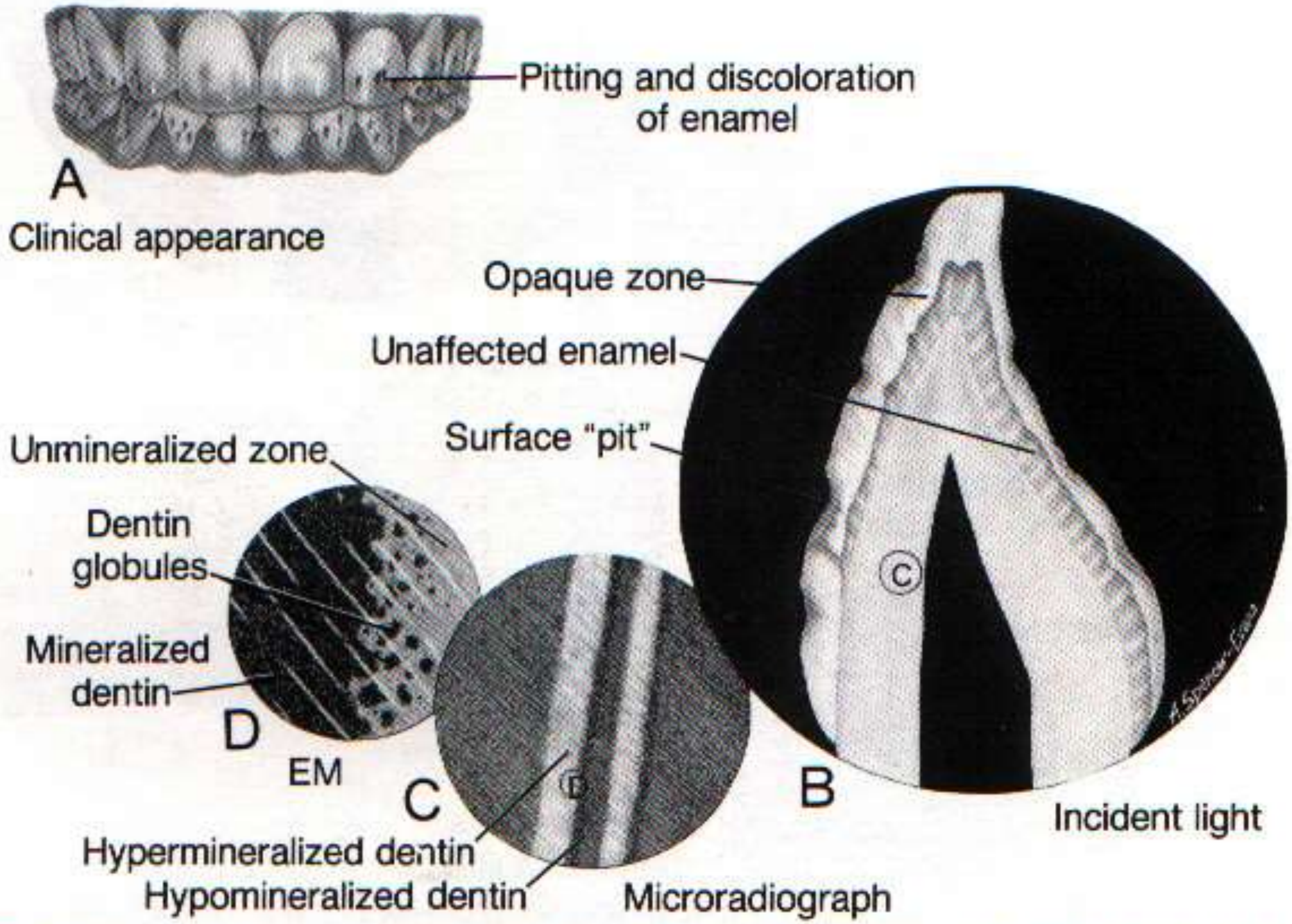


Figure 8–14. Illustration of effects of sodium fluoride on developing teeth. (A). The clinical picture reveals brown-stained hypoplastic pits. (B) illustrates the appearance of these pits in a ground section of the tooth seen in (A). (C) illustrates altered incremental lines of the encircled zone in (B). (D) illustrates the hypocalcified dentin of the encircled zone in (C), at higher magnification of the electron microscope.

Clinical Application :

1. The size of the tooth is depends upon both proliferative & secretory activities of the cell. Macrodontia, Microdontia are the result of factors affecting the growth of the tooth germ at the cap & bell stages. True macro & microdontia may be the result of over & under secretion of pituitary hormones.
2. Absence of teeth, partial or total anodontia is due to factors that disrupt tooth development at the initiation stage. In genetic disorder ectodermal dysplasia there is total anodontia.

3. Distal proliferation of dental lamina is responsible for the location of the germs of the permanent molars in the ramus of the mandible & tuberosity of maxilla.
4. If the cells of the epithelial RS remain adherent to the dentin surface, they may differentiate into fully functioning ameloblast & produce enamel called enamel pearls, found in the areas of furcation of the roots of PM.
5. If continuity of Hertwig's RS is broken or is not established prior to dentin formation, a defect in the dentinal wall of the pulp ensures.this accounts for development of accessory root canals opening.

6. If continuity of Hertwig's RS is not broken even after dentin formation it results in root dentin exposure to oral cavity.
7. Developmental disorders of teeth causes clinical problems related to appearance, spacing, crowding, & periodontal treatment.
8. In intercuspal areas, during enamel secretion, the ameloblasts may become strangulated when their bases begin to touch one another. These areas becomes pits & fissures in erupted tooth. They are extremely difficult to clean.

9. Clinically,vit-C deficiency is manifested orally by gingival bleeding & loosening the teeth. Weakness, anemia, bone loss, & susceptibility to hemorrhage.
10. Osteoporosis results when calcium loss because of resorption is greater than calcium deposition. This may be evident orally with loss of alveolar bone & loosening of teeth.
11. Certain antibiotics, like the tetracyclines, have an affinity for calcified tissues. They may become incorporated within the mineral phase during maturation, & cause discoloration of the enamel & underlying dentin.

12. Brown staining or defect in the enamel of the incisal third of crowns indicates the presence of toxic substance in the body at the time of initial mineralization of the teeth. Staining in the cervical area relates to induction of a toxic substance at a time of final crown mineralization.

13. The cervical region of the crown and the central grooves are the last zones to mineralize. Ameloblasts in these regions may lose functional ability before mineralization is complete. Lack of complete mineralization of the central grooves or cervical areas is believed to be a reason for caries in these areas.

Summary :

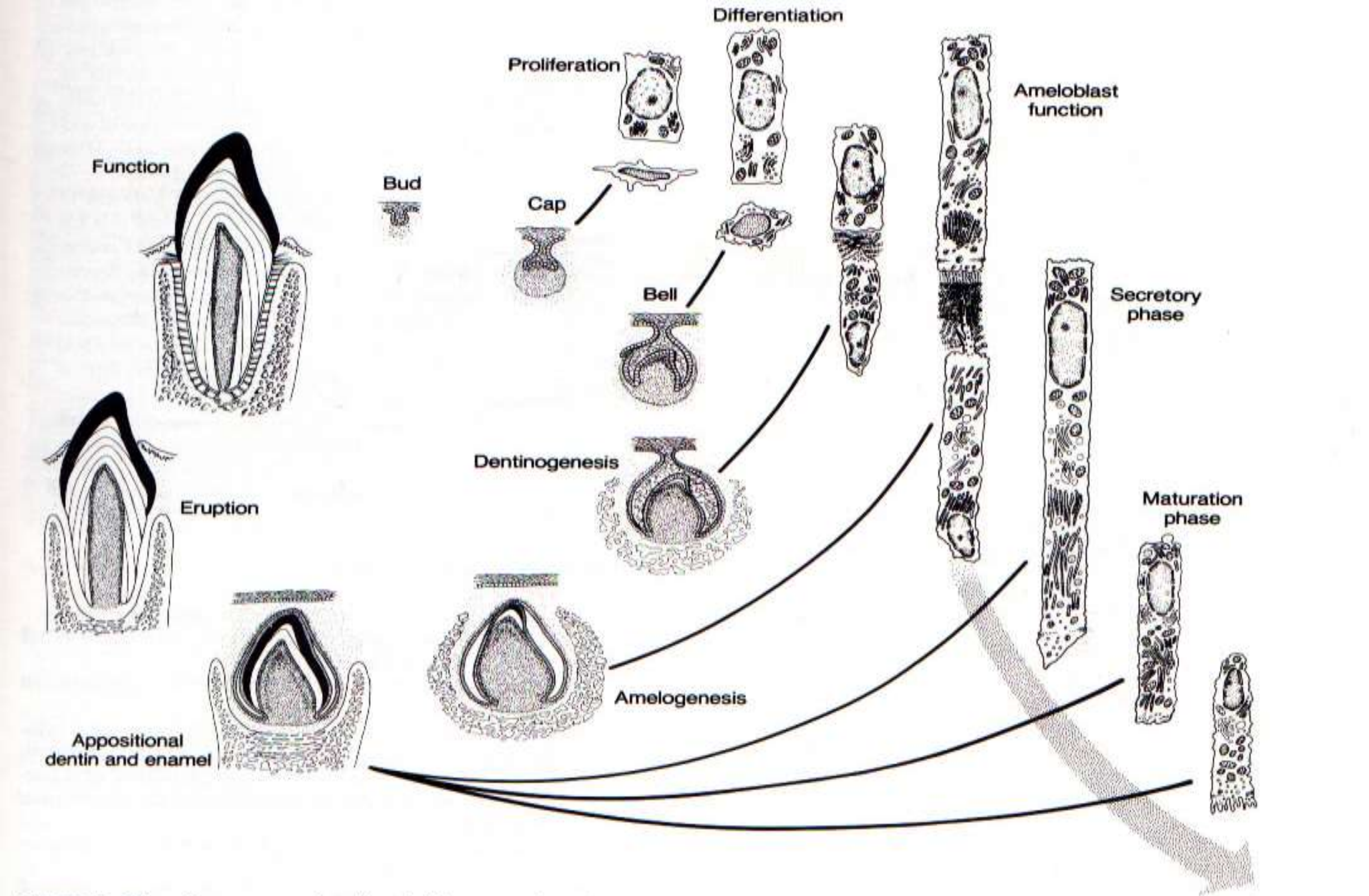


Figure 5–31. Summary of cell activities correlated to early stages of tooth formation which are important to the development, eruption, and function of teeth. Ameloblast and odontoblast differentiation and function are seen on the right and tooth development stages on the left.

❖Refrences :

1. Oral Histology & Eeditionmbryology
   by Orbans 13th edition
2. Oral Histology & Development
   by Tencate.
3. Oral Pathology
   by Shafer Hine Levi 6th edition
4. Internet


Thank
you!