CHAPTER 2

Dental Anatomy and Dental Histology

1. TEETH DEVELOPMENT AND ABNORMALITIES

Q. 1. Discuss in detail the development of tooth. (*TNMGR, March 2010; Guwahati Uni., May 2011; RGUHS, May 2014; Sumandeep Uni., April 2015; MUHS, Dec. 2017*)

Q. Add a note on various theories of tooth development. (*NTR, Uni., May 2019*)

Q. Write a short note on enamel organ and its function. (*TNMGR, March 2007*)

Q. Add a note on development disturbances of the enamel. (*Gujarat, Uni., July 2017*)

Ans. Development of tooth is a complex process. Tooth formation starts in 6th week of IUL with formation of primary epithelial band. At about 7th week primary epithelial band divides into a lingual process called **dental lamina** and a buccal process called **vestibular lamina**. All deciduous teeth arises from dental lamina, later permanent successors arise from its lingual extension and permanent molars (accessors) from its distal extension. Tooth germ includes all the formative tissues for tooth and its supporting structures and has three main components:

- Enamel organ: Ectodermal component that gives rise to enamel.
- Dental papilla: Ectomesenchymal component that gives rise to dentin and pulp.
- Dental follicle or dental sac: Ectomesenchymal component giving rise to cementum, periodontal ligament (PDL) and part of alveolar socket.

Stages of Tooth Development (Fig. 1.1)

1. Bud stage
2. Cap stage
3. Bell stage
   - Early bell stage.
   - Late or advanced bell stage

Bud Stage: Enamel organ is bud shaped with peripheral cuboidal cells and central polyhedral cells. Peripheral cells of enamel organ are separated from ectomesenchymal components by a basement membrane. All the cells are attached to each other by desmosomal junctions. Ectomesenchymal condensation adjacent to enamel organ forms **dental papilla**. Marginal condensation of ectomesenchymal cells enclosing dental papilla and enamel organ is called **dental follicle or dental sac**.

Cap Stage: Enamel organ increases in size and attain shape of a cap by invagination of deep portion of bud. Cells lining the convexity or periphery of the cap are cuboidal in shape and are called **outer enamel epithelium (OEE)**. The cells lining the concave or invaginated portion change to columnar cells, **inner enamel epithelium (IEE)**. Central polyhedral cells transform into network of star-shaped cells called **stellate reticulum**. Dental papilla gets partially enclosed by invaginated portion of enamel organ. Cells of dental papilla undergo proliferation and condensation of ectomesenchymal cells and become more fibrous and denser.

Early Bell Stage: Enamel organ enlarges further and invagination deepens changing shape to that of a bell and four different layers of cells are seen in enamel organ. Cells lining IEE is composed of single layer of tall columnar cells that differentiate to *ameloblasts* (enamel forming cells). **Stratum intermedium (new)** is located between IEE and stellate reticulum and is composed of 2–3 layers of squamous cells. Cells of OEE lining periphery of enamel organ flattens to low cuboidal cells. At cervical region of enamel organ OEE loops inward to join with IEE called **cervical loop**. During early bell stage enamel organ loses its connection to oral ectoderm due to degeneration of dental

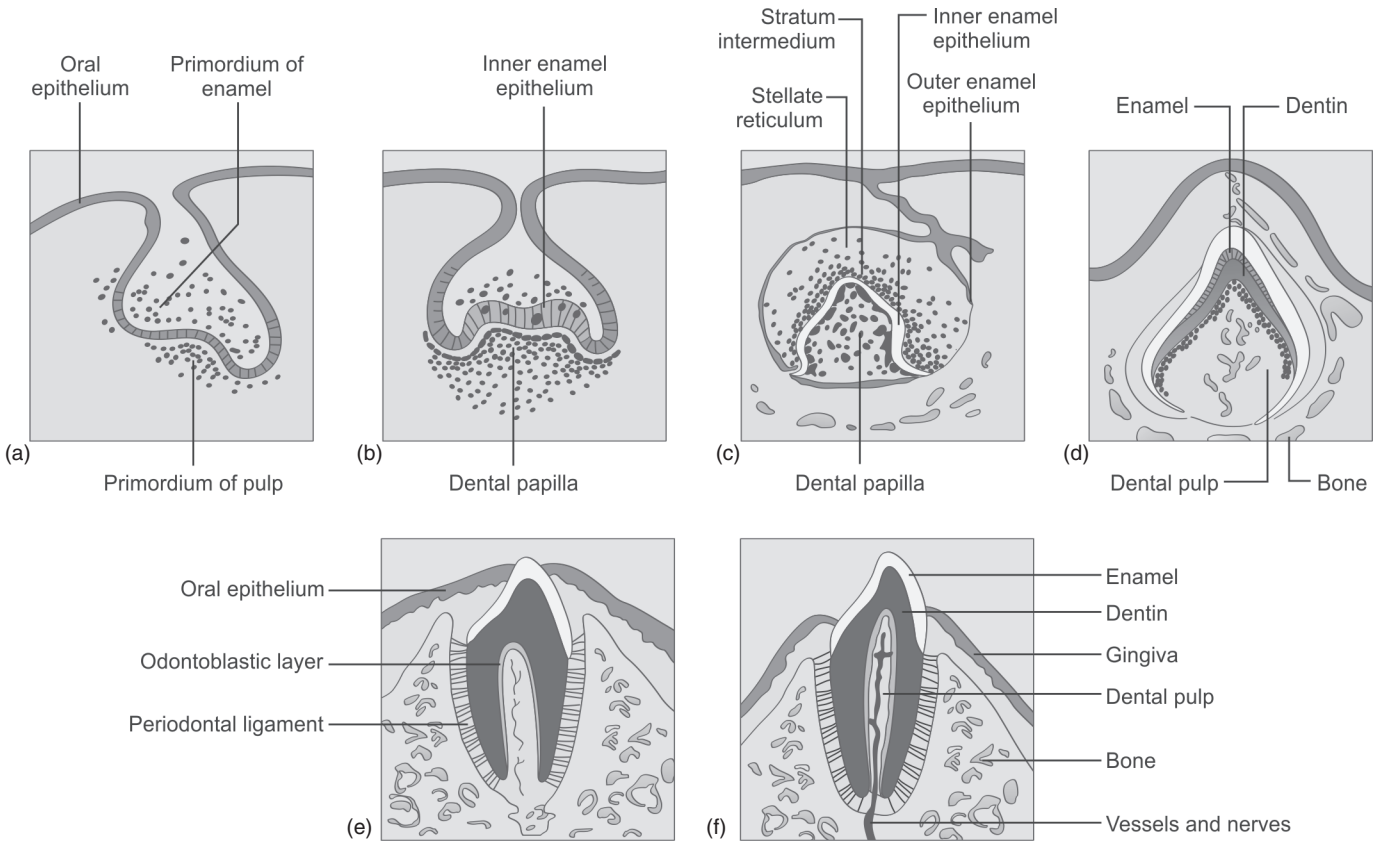

Fig. 1.1: Stages of tooth development: a. Bud stage; b. Cap stage; c. Early bell stage; d. Advanced bell stage; e. Eruption stage; f. Fully formed stage

lamina. Remnants of dental lamina are called **cell rests of serres**. Successional lamina develops at this stage which is the primordium for the permanent successor. Peripheral cells of dental papilla differentiate into *odontoblasts* (dentin forming cells) under the organizing influence of IEE cells. Dental follicle becomes more fibrous with 3 layers, i.e. inner cellular, outer fibrous layer and middle loose connective tissue.

Advanced Bell Stage: Differentiating feature between early and advanced bell stage is formation of hard tissues. Enamel organ shows four different layers, IEE, stratum intermedium, stellate reticulum and OEE. Histological difference from early bell stage are: Hard tissue formation, collapsed stellate reticulum and folding of OEE bringing capillaries of dental follicle nearer to ameloblasts. Dental papilla shows differentiated odontoblast at the periphery.

Q. 2. Write a short note on stellate reticulum.

(*RGUHS, April 2006*)

Ans. Stellate reticulum/enamel pulp: Polygonal cells located in the center of epithelial organ, between outer and inner enamel epithelium; begin to separate due to water being drawn into enamel organ from surrounding dental papilla as a result of osmotic force exerted by glycosaminoglycans (GAG) contained in ground substance. As a result, the polygonal cells become star shaped but maintains contact with each other by their cytoplasmic process. The spaces in between are filled with mucoid fluid, rich in albumin, giving a cushion-like consistency, protecting the delicate enamel forming cells. The cells in the centre of enamel organ are densely packed and form enamel knot and vertical extension of **enamel knot** is known as enamel cord. Both are temporary structure; act as a reservoir of dividing cells for growing enamel organ (Fig. 1.2).

Functions:

1. It provides elasticity and resistance.
2. Acts as buffer against forces that might distort developing dentinoenamel junction (DEJ).
3. It permits only limited flow of nutritional elements from overlying blood vessels to formative cells.
4. Acts as a shock absorber that may support and protect delicate enamel forming cells.

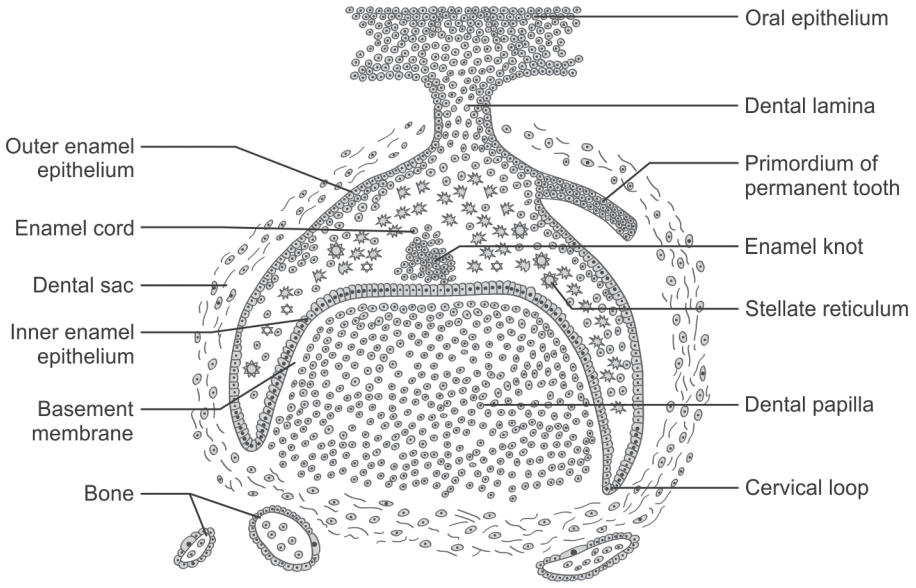

Fig. 1.2: Tooth bud showing star-shaped stellate reticulum, enamel knot and enamel cord

Q. 3. Write a short note on developmental anomalies in tooth morphology. (*TNMGR, Sept. 2007; AHSUC, May 2018*)

Ans. Developmental anomalies: An abnormality where pathology starts in embryonic stage of human life before formation of dentition. These include:

1. One or more teeth may be absent. Complete absence is called **Anodontia**.
2. Supernumerary teeth may be present.
3. Individual teeth may be abnormal. They may be too large (**Macrodont**) or too small (**Microdont**).
4. Two (or more) teeth may be fused to each other (**Gemination/Fusion**).
5. **Concrescence:** Union of teeth by cementum only.
6. Dilacerations: Sharp bend in root or crown of tooth.
7. Talon cusp: Accessory cusp projecting from cingulum of incisors.
8. Dense in dente: Invagination in surface of tooth crown before calcification.
9. Dense evaginatus: Proliferation and evagination of an area of IEE during tooth development.
10. Taurodontism: Body of tooth is enlarged at expense of roots.
11. The alignment of the upper and lower teeth may be incorrect (**Malocclusion**). This may be caused by one or more of the above anomalies or by defects of the jaws.
12. Eruption of teeth may be precocious (i.e. too early). Lower incisors may be present at birth (natal/ neonatal teeth).
13. Eruption of teeth may be delayed. The third molar frequently fails to erupt.
14. Teeth may form in abnormal situations, e.g. in the ovary or in the hypophysis cerebri.
15. There may be improper formation of the enamel (**Amelogenesis imperfecta**) or dentin (**Dentinogenesis imperfecta**) of the tooth.

Q. 4. Write a short note on theories of tooth eruption. (*TNMGR, Oct. 2012; KUHS, June 2013; RUHS, May 2015; Sumandeep Uni., June 2017; AHSUC, May 2018; DYP Uni., May 2019*)

Q. Write a short note on gubernacular cord. (*NTR Uni., May 2018*)

Ans. Tooth eruption is defined as the movement of a tooth from its site of development within the alveolar process to its functional position in the oral cavity.

Phases of tooth eruption:

1. **Pre-eruptive phase:** This phase begins in early bell stage and ends at beginning of root formation. Made by deciduous and permanent tooth germs within tissues of jaw before they begin to erupt.
2. **Eruptive phase** (Fig.1.4): It starts with initiation of root formation and made by teeth to move from its position within bone of jaw to its functional position in occlusion. This has intraosseous and extra-osseous compartments.
   a. **Active eruption:** It is gradual appearance of tooth in oral cavity due to axial occlusal movement of tooth.

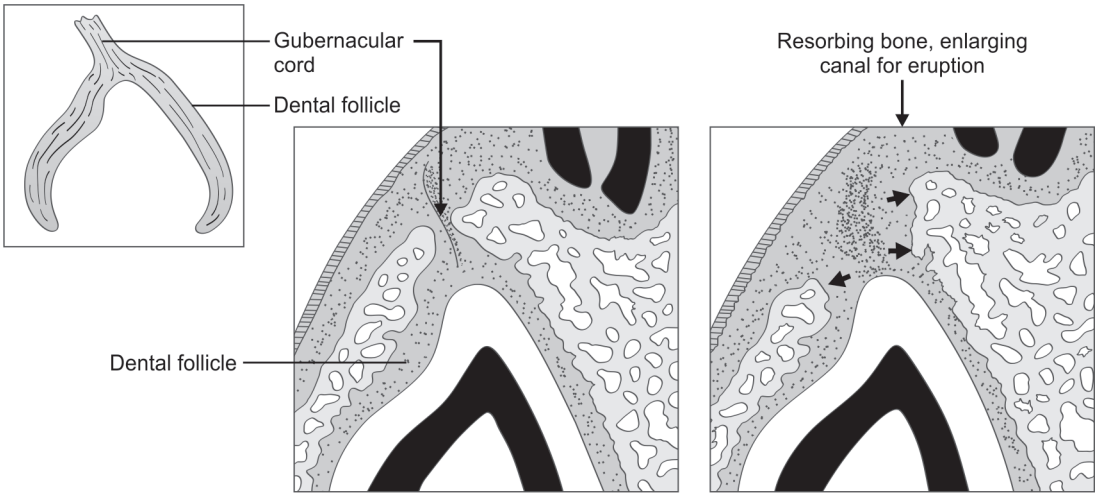

Fig. 1.4: Eruption of tooth through gubernacular cord

b. **Passive eruption:** It happens due to gradual retraction of attachment epithelium from tooth surface.

c. **Gubernacular cord:** As deciduous tooth erupts, permanent tooth germ become situated apically and is entirely enclosed by the bone except for a small canal that is filled with connective tissue and often contains epithelial remnants of the dental lamina known as "**gubernacular cord**".

d. **Gubernacular canal:** Holes noted in a dry skull lingual to primary teeth in jaws that represent openings of gubernacular cord. After removal of any overlying bone there is loss of intervening soft tissue between reduced enamel epithelium covering crown of tooth and overlying oral epithelium.

3. **Posteruptive phase:** Takes place after the teeth are functioning to maintain the position of erupted tooth in occlusion while jaws are continuing to grow and compensate for occlusal and proximal tooth wear.

Mechanism of tooth movements/theories of tooth eruption

1. **Bone remodeling theory (Brash, 1928):** Simultaneous bone deposition and bone resorption in area around tooth causes its axial movement.
2. **Root elongation theory:** The apical growth of roots results in axial directed force that leads to tooth eruption.
3. **Vascular pressure theory (V. Korff, 1935):** Alteration in local vascular supply and increase in local tissue pressure in PDL leads to tooth eruption.
4. **Periodontal ligament traction theory (Thomas, 1967):** The contractility of fibroblasts present in the PDL provides force for tooth eruption.
5. **Pulp constriction theory:** The growth of root dentin and constriction of pulp causes sufficient pressure to move tooth occlusally.
6. **Dental follicle theory (Marks and Cahill, 1984):** Specific cellular changes occurring in and around the follicle leads to tooth eruption.

Factors affecting tooth eruption:

1. **Genetic:** Genetic factors definitely controls tooth emergence, as most of them delay permanent teeth eruption; others are associated with complete failure teeth to erupt.
2. **Gender:** In girls permanent teeth erupt earlier, average 4–6 months than in boys.
3. **Nutrition:** Chronic malnutrition is correlated with delayed teeth eruption.
4. **Preterm birth:** Preterm children have delayed primary and permanent teeth eruption.
5. **Socioeconomic factors:** Children from higher socioeconomic backgrounds show earlier tooth emergence than children from lower socioeconomic classes.
6. **Body height and weight:** The taller and heavier children show early eruption of teeth as compared to children with normal body mass index.
7. **Craniofacial morphology:** Formation and eruption of maxillary teeth, especially molars, are delayed in skeletal Class III patients.
8. **Hormonal factors:** Hypothyroidism, hypopituitarism, hypoparathyroidism, and pseudohypoparathyroidism are associated with delayed permanent teeth eruption. Accelerated dental development

has been noted in association with increased adrenal androgen secretion.

9. **Systemic disease:** Most of the systemic diseases are associated with delayed tooth eruption, except diabetes accelerates tooth eruption.

Q. 5. Write a note on factors influencing shedding and eruption of primary teeth.

(*TNMGR, Nov. 1995; March 2009; BBD Uni., April 2014*)

Ans. Shedding is the physiologic process resulting in elimination of deciduous dentition with replacement by their corresponding permanent successors. Shedding involves resorption of hard and soft tissue. In soft tissue resorption, apoptotic cell death is involved.

Pressure generated by erupting permanent tooth guides pattern of deciduous tooth resorption. Initially, pressure is against root surface of deciduous tooth and resorption occurs on lingual surface. In mandibular incisors apical positioning of tooth germs does not occur and permanent tooth erupts lingually.

Resorption of deciduous molars: Resorption of the roots of deciduous molars first begins on their inner surfaces because early developing bicuspids are found between them. With continued growth of jaws and occlusal movement of deciduous molars, the successional tooth germs lie apical to deciduous molars. When the bicuspids begin to erupt, resorption of deciduous molars is again initiated and continues until roots are completely lost and tooth is shed.

Table 1.5a: Sequence and chronology of deciduous teeth

Jaw	*Tooth*	*Calcification begins* (months *in utero*)	*Crown completed post-natally* (months)	*Time of emergence* (months)	*Root completed* (years)	*Emergence sequence*
Max. (upper)	i^1	3–4 months	2	7–10	2.5	2
	i^2	4 months	2–3	8–11	2.5	3
	c^1	4–5 months	9	16–19	3.5	7
	m^1	4 months	6	12–15	3	5
	m^2	5 months	11	25–28	4	10
Mand. (lower)	i_1	3–4 months	2–3	6–8	2.5	1
	i_2	4 months	3	9–13	2.5	4
	c_1	4–5 months	9	17–20	3.5	8
	m_1	4 months	6	12–16	3	6
	m_2	5 months	10	20–26	3.5	9

Table 1.5b: Sequence and chronology of permanent teeth

Jaw	*Tooth*	*Calcification begins*	*Crown completed* (years)	*Time of emergence* (years)	*Root completed* (years)	*Emergence sequence*
Max. (upper)	I1	3–4 months	4–5	7–8	10	4
	I2	10–12 months	4–5	8–10	10–11	6
	C	4–5 months	6–7	11–13	11–14	12
	P3	1–2 years	6–7	10–12	12–14	8
	P4	2–3 years	7–8	10–12	13–14	10
	M1	At birth	4–5	6–7	9–10	2
	M2	2–3 years	7–8	11–13	15–16	14
	M3	7–9 years	12–16	17–20	18–25	16
Mand. (lower)	I1	3–4 months	3–4	6–7	9	3
	I2	3–4 months	4–5	7–8	9–10	5
	C	4–5 months	5–6	8–10	12–13	7
	P3	1–2 years	6–7	10–12	12–14	9
	P4	2–3 years	7	11–13	14–15	11
	M1	At birth	3–4	6–7	9–10	1
	M2	2–3 years	7–8	11–13	14–15	13
	M3	8–10 years	12–16	17–20	18–25	15

Resorption of cementum and dentine: Resorption involves a loss of organic as well as mineral constituent of matrix and is characterized by presence of osteoclasts.

Resorption of root: Root resorption seems to be initiated and regulated by stellate reticulum and dental follicle of underlying permanent tooth via secretion of stimulatory molecules, i.e. cytokines and transcription factors. The primary root resorption process is regulated in a manner similar to the bone remodeling, involving the same receptor ligand system known as RANK/RANKL (receptor activator of nuclear factor—kappa B/RANK ligand).

Mechanism of resorption and shedding: Pressure from erupting successional tooth and appearance of odontoclasts at site of pressure. Membrane of ruffled borders acts as proton pump → adding hydrogen ions to extracellular region → acidification → mineral dissolution. Increased forces of mastication with increase in jaw size leading to trauma to PDL → degeneration of PDL.

Q. 6. Write a short note on development of root. (*TNMGR, Sept. 2010; KUHS, June 2013*)

Q. Write a short note on Hertwig's epithelial root sheath. (*NTR Uni., Aug. 2008*)

Ans. The development of the roots begins after enamel and dentin formation has reached the future cemento-enamel junction (CEJ). The enamel organ forms Hertwig's epithelial root sheath (HERS), which molds the shape of roots and initiates radicular dentin formation. HERS consists of outer and inner enamel epithelia only. The cells of inner layer remain short and initiate differentiation of odontoblasts, which forms radicular dentin. Just after this, HERS loses its structural continuity and its remnants persist as epithelial network of strands called **rests of Malassez** (Fig. 1.6).

The root sheath prior to elongation in apical direction forms an epithelial diaphragm, which is a horizontal extension at the future CEJ. Subsequently, epithelial cells disintegrate and move away from surface of dentin so that connective tissue cells come into contact with

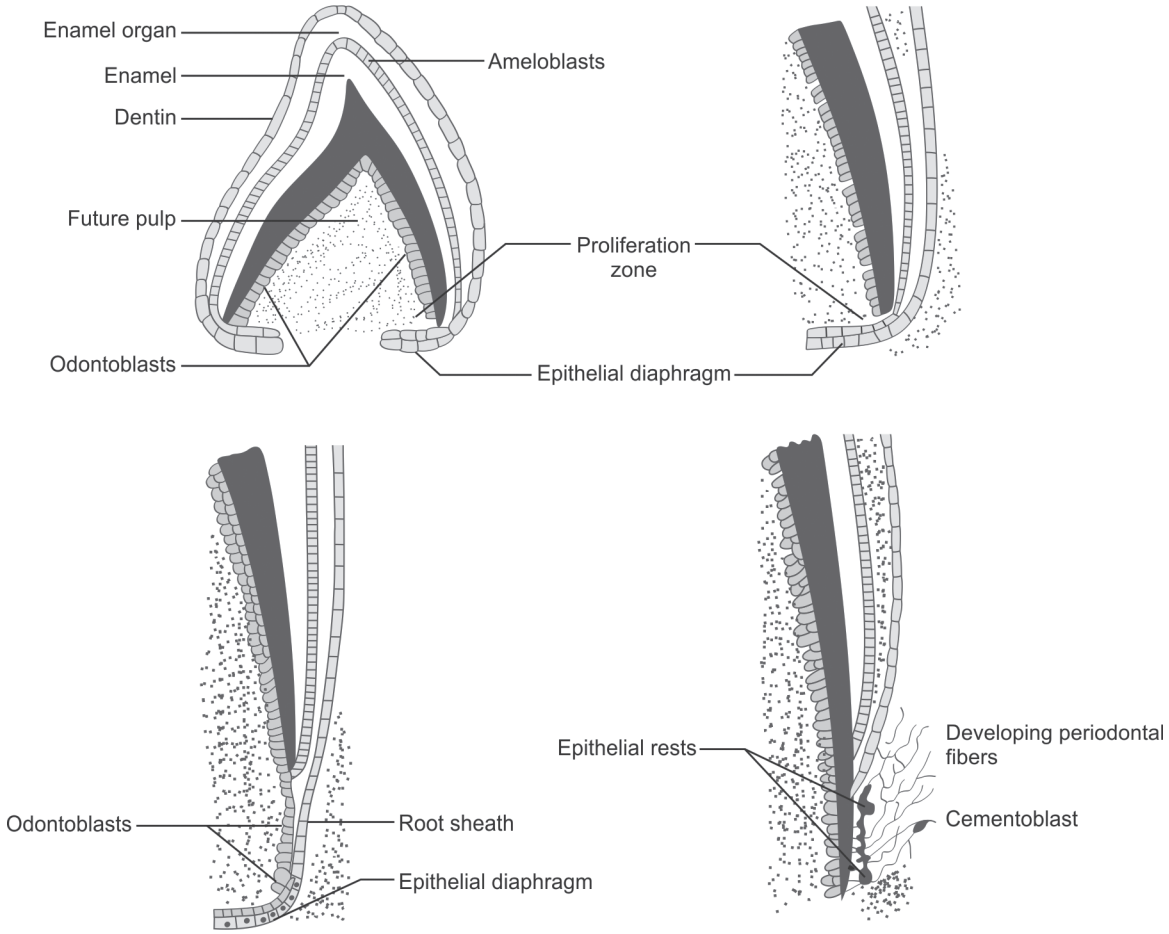

Fig. 1.6: Root formation

outer surface of dentin and differentiate into cementoblasts, that deposit a layer of cementum.

Q. 7. Write a note on anatomy of root apex and its significance. (*Sumandeep, Uni., April 2014, 2015*)

Ans. The terminal part of a tooth root exhibits four distinct landmarks:

Apical constriction (AC): Apical part of root canal with narrowest diameter. The distance between AC and apical foramen (AF) ranged between 0.4 and 1.2 mm, while its reported location in relation to root apex ranged between 0.5 and 1.01 mm. AC is mostly located either in dentin or at CDJ level and less frequently in cementum. The shape of AC in longitudinal sections has four possible configurations: Single, tapered, multi-constricted and parallel.

Apical foramen (AF): Main apical opening of root canal. Deviation of AF from root apex is common. Average distance between AF and root apex was found to be less than 1 mm.

Roots apex (anatomic and radiographic): Anatomic apex differs from radiographic apex in that former is root end as identified morphologically and latter is identified radiographically. It has been suggested to extend root canal instrumentation to 1 mm short of the radiographic apex, which would ensure closer proximity to position of the AF. However, continuous cementum deposition alters the position of the radiographic apex to the AF (Fig. 1.7).

Cementodentinal junction (CDJ): CDJ is the line of union between dentin and cementum at which pulpal tissue ends and periodontal tissue starts. During tooth development, cementum deposition follows that of dentin, resulting in a line of delineation separating the two tissue types. The CDJ is the ideal termination point for RCT.

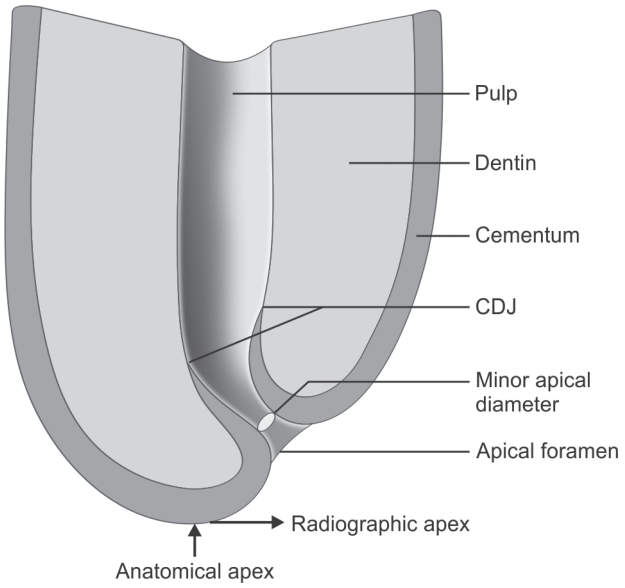

Fig. 1.7: Root apex and its parts

Q. 8. Write about theories of mineralization. (*BBD Uni., May 2011;UOK, June 2019*)

Ans. Mineralization is deposition of mineral salts in and around organic matrix to make it a calcified structure. The various proposed theories of mineralization are:

1. **Robinson's alkaline phosphatase theory:** The enzyme is responsible for mineralization.
2. **Cartier's theory:** According to Cartier alkaline phosphatase has very little role in mineralization and that ATPase is extremely powerful in inducing mineralization.
3. **White and Hers theory:** White and Hers made surprising discovery that bone and especially dentin still possessed possibility of splitting phosphate easters even ion removing and destroying all the enzymes.
4. **pH of cartilage explaining mineralization:** One of the oldest suggestions for mechanism of mineralization is that pH of cartilage is higher than that of other tissues which would favor precipitation of calcium phosphate.
5. **Seeding mechanism:** According to this mechanism, there are certain substances called seeding or nucleating having resemblance to apatite. These substances act as mould or template upon which crystals are laid down, after which crystallization proceeds automatically. This process is known as **epitaxy**. The following substances have been considered as possible seeding substances: collagen, chondroitin sulphate, lipid substances, phosphoproteins.
6. **Matrix vesicle concept:** Matrix vesicles are organelles of cellular origin that can be observed electron microscopically in the matrix of cartilage, bone, and other hard tissue.

Q. 9. Discuss ectomesenchymal interactions. (*Sharda Uni., July 2016, 2017*)

Ans. Epithelial mesenchymal interactions (EMIs) are described as a series of programmed, sequential and reciprocal (complex and multiphase) communications between the epithelium and mesenchyme with its heterotypic cell population, that result in differentiation of one or both cell populations. EMI plays a major role in the following conditions.

1. **In odontogenesis:** Odontogenesis can be described as a complex physiological process of tooth development from ectodermal and mesodermal appendages. Enamel organ derives from ectoderm, whereas dental papillae and dental sac are of mesodermal origin. For the development of teeth, interaction between these ectodermal and mesodermal tissues is essential. Odontoblasts differentiate only in the presence of the enamel epithelium. For EMIs to occur, messenger system is required between epithelium and mesenchyme, which may be: Direct cell–cell communication (cytoplasmic process and gap junctions), matrix vesicles, ions (K^+, Ca^{+2}), extracellular matrix molecules (collagen IV, I, III, fibronectin, tenascin, epithelial cadherin, laminin), hormones and growth factors (bone morphogenetic factor, fibroblast growth factor, epidermal growth factor, TGF), autocrine and paracrine regulators, mRNA, genes (pitx2, p21, Msx2, Lef1, Edar, Lhx6, Lhx7, Dix1, Dix2, Paz9, Gli1, Gli2, Gli3, Barx1 and Runx2)
2. **In dentinoenamel junction:** Enamel originates from epithelial tissue by ameloblast; dentine derives from mesenchymal tissue by odontoblast. The interdigitation of ameloblasts and odontoblasts is essential for union of dentino-enamel junction (DEJ).
3. **In salivary gland development:** Salivary gland is an epithelial modification and is made up of epithelial components, such as acinar and ductal cells, mesenchymal structures, such as fibrous stroma, vascular elements and a fibrous capsule. Hence, EMIs are required for development of the salivary gland tissue.
4. **In palatogenesis:** During 6th week of palatogenesis, palatal shelves grow rapidly along the vertical plane by proliferating mesenchymal cells, which undergo a sudden elevation to bring them into a horizontal apposition above the flattening tongue followed by fusion of opposing palatal shelves along midline through a series of interactions in cell adhesion molecules. This is considered to be the EMIs in palatogenesis.
5. **In oral cancer:** The new mass of tissue requires angiogenesis for their sustained proliferation and growth. As a consequence of these requirements, epithelial and mesenchymal cells need to have an interactionary signaling mechanism for their growth and proliferation. Basement membrane can be said to maintain the normal histogenesis and morphogenesis of epithelium. In the case of malignant tissue, where there is loss of basement membrane contiguity, normal histogenesis is lost; and the involved epithelial tissue adversely proliferates.

Q. 10. Discuss tooth numbering system.

(UHSR, June 2019)

Ans. There are several **tooth numbering** systems:

1. **Universal Notation System**
   a. *For primary teeth:* Universal system of notation for primary dentition uses 'upper case letters' for each of the primary teeth. The ADA in 1968 officially recommended the "universal" numbering system.

Right	A	B	C	D	E	F	G	H	I	J	Left
	T	S	R	Q	P	O	N	M	L	K	

   b. For permanent teeth: Universal system of notation for permanent dentition uses 'numbers' for each of the permanent teeth.

Right	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	Left
	32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17	

2. **Zsigmondy/Palmer Notation System:** In this system, arches are divided into quadrants: ⌟upper right; ⌞upper left; ⌝lower right; ⌜lower left.
   a. For deciduous dentition:

E	D	C	B	A	A	B	C	D	E
E	D	C	B	A	A	B	C	D	E

   b. For permanent dentition:

8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8

3. **Federation Dentaire Internationale (FDI):** Two-digit system, proposed by FDI and is adopted by WHO.
   a. For primary dentition: First digit indicates quadrant: 5 to 8. Second digit indicates tooth within a quadrant, 1 to 5 for primary teeth.

55	54	53	52	51	61	62	63	64	65
85	84	83	82	81	71	72	73	74	75

   b. For permanent teeth: First digit indicates the quadrant: 1 to 4 for permanent dentition. Second digit indicates the tooth within a quadrant:1 to 8 for permanent teeth.

	Upper Right								Upper Left							
	18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
	48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38
	Lower Right								Lower Left							

2. OCCLUSION AND SKULL BONE DEVELOPMENT

Q. 1. Write about development of occlusion from birth to adolescence. *(BFUHS, May 2010; KUHS, Nov. 2015; HP Uni., May, 2017; April 2019)*

Q. Discuss role of tongue in development and maintenance of normal occlusion. *(DRMLA Uni., July 2008; Gujarat Uni., April 2019)*

Q. Define occlusion. Discuss development of occlusion and malocclusion and factors affecting development of occlusion. *(CDER-AIIMS, Dec. 2016; BVP, June 2018)*

Ans. Occlusion is defined as the contact relationship of the teeth in function or parafunction.

Malocclusion: A condition in which there is a deflection from normal relation of teeth to other teeth in the same arch and/or to teeth in the opposing arch.

Periods of occlusal development:

1. **Pre-dental period:** During this period, neonates have no teeth. It lasts for 6 months after birth. It has following features:
   a. **Gum pads** (Fig. 2.1): The alveolar process at the time of birth is known as **gum pads**. They are horse shoe shape, pink and firm, and develop in two parts: i. Labio-buccal portion; ii. Lingual portion, separated from each other by **dental groove**. Each gum pad is divided into ten segments, each containing deciduous tooth sac, by **transverse grooves**. **Gingival groove** separates gum pad from palate and floor of mouth. **Transverse groove** between canine and first deciduous molar segment is called **lateral sulcus**. Upper gum pad is both wider and longer than lower gum pad. On closing, contact occurs in first molar region, and space exists anteriorly (infantile open bit), which helps in suckling.
   b. **Status of dentition:** Neonates are without teeth for about 6 months. Initially there is crowding of developing teeth, but during first year of life, they grow rapidly, allowing the proper alignment of teeth.
2. **Deciduous dentition period:** From 6 months to 2–3 years.
   The sequence of eruption is: A-B-D-C-E.
   Between 3 and 6 years of age, the dental arch is relatively stable. Other normal features during this period are:
   a. Physiological or developmental spacing in anterior region: **Primate or Simian space** (wider spaces between mesial to maxillary canine and distal to mandibular canine).
   b. Flush terminal plane: Distal surface of maxillary and mandibular deciduous second molars are in same vertical plane.
   c. Deep bite.
3. **Mixed dentition period (6–12 years):** It starts with eruption of first permanent molar (6 years). It has been classified into 3 phases:
   i. **First transitional period (6–8 years):** Emergence of first permanent molar and exchange of deciduous incisors with permanent incisors. The permanent incisors are larger than deciduous, the excess space needed than present is called **incisal liability** (for maxillary 7 mm and for mandibular arch 5 mm). This is compensated by:
      a. Utilization of interdental spaces seen in primary dentition.
      b. Increase in inter-canine width.
      c. Change in incisor inclination.
   ii. **Inter-transitional period:** Both upper and lower arches consist of sets of deciduous and permanent teeth, this phase is relatively stable.
   iii. **Second transitional period (10–12 years):** In this phase, there is replacement of deciduous molars

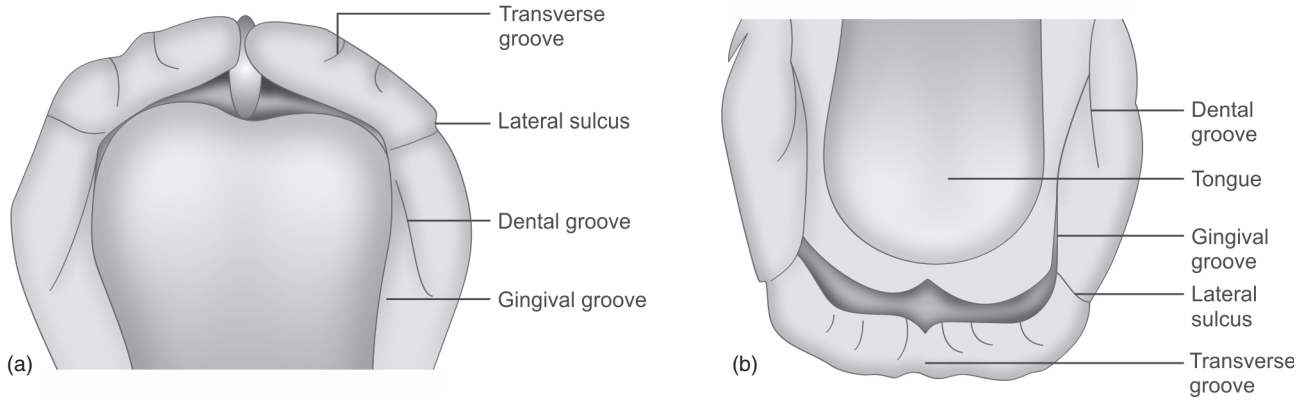

Fig. 2.1: (a) Maxillary gum pad; (b) Mandibular gum pad

and canines by premolars and permanent cuspids respectively. The space difference between combined width of deciduous canine and molars and mesiodistal width of permanent canine and premolars (**Leeway space of Nance**) is greater in mandibular arch, which is utilized for mesial drift of mandibular molars.

Ugly duckling stage (Broadbent's phenomenon, 1937): It is transient or self-correcting malocclusion seen in maxillary incisor region between 8 and 9 years of age, seen during the eruption of permanent canines. There is flaring of lateral incisors, maxillary midline diastema.

4. **The permanent dentition period:** The eruption sequence of permanent dentition in maxillary arch:

   6-1-2-4-3-5-7 or 6-1-2-3-4-5-7

   The eruption sequence of permanent dentition in mandibular arch:

   6-1-2-3-4-5-7 or 6-1-2-4-3-5-7

Factors affecting development of occlusion:

A. General factors:
   1. Skeletal factors: Conditions that affect jaw growth; pathological, inherited or acquired, trauma or infection
   2. Muscle factors: Lip form and activity, tongue size, position and function, adaptive swallowing, thumb sucking, neutral zone.
   3. Dental factors: Size of teeth; early loss of teeth leads to over eruption of opposing teeth.

B. Local factors:
   1. Aberrant developmental position of individual teeth: Trauma, malposed crown, dilacerated roots.
   2. Presence of supernumerary teeth: Supplemental, conical, tuberculated
   3. Developmental: Hypodontia affects form and position of teeth and growth of jaw.
   4. Upper labial frenum: Median diastema

Role of tongue in malocclusion: Position of tongue and its function plays an important role in development of dental malocclusion.

1. Microglossia: Dental arch is collapsed and reduced. Crowding in premolar area with severe class II malocclusion.
2. Macroglossia: Mandibular prognathism/Class III, buccal tipping of posterior teeth.
3. Abnormal posture leads to generalized spacing of teeth.
4. Genioglossus reflex initiated by large tongue, tonsils, mouth breathing leads to sustained jaw opening → sustained tongue posture → malocclusion (proclination, open bite, prevention of tooth eruption, posterior open bite, deep overbite).
5. Tongue thrust habit: Proclination of anterior teeth, anterior open bite, bimax dental protrusion, posterior cross bite.

Q. 2. Enumerate various theories of growth.

(UOK, July 2017)

Q. Write a short note on neurotropism.

(UOK, July 2017)

Ans. Growth refers to an increase in size/number (Profitt). **Development** is progress towards maturity (Todd).

Theories of growth:

1. Remodeling theory (Brash): Craniofacial skeletal growth occurs exclusively by bone remodeling.
2. Genetic theory (Brodie, 1941): Growth process is under influence of genetic control and is pre-programmed.
3. Sutural theory (Weinmann and Sicher, 1952): Craniofacial growth occurs at the sutures.
4. Cartilaginous theory (James Scott): Cartilage play primary role in craniofacial growth.
5. Functional matrix theory (Melvin Moss, 1962): Bone growth is influenced by function, as soft tissue grows; both bone and cartilage react and grow in response to soft tissue.
6. Multifactorial theory (Van Limborgh, 1970): Six factors control growth: Genetics, intrinsic growth factors, cartilage, suture, adjacent structures, and muscle function.
7. Enlow's principle of growth: Most of the bones have V-shaped, bone deposition on inner side and resorption in outer surface.
8. Enlow's counterpart principle: Growth in one region of skull influence the growth in others.
9. Neurotrophism (Behrents, 1970): It states that the nerve impulse involving the axoplasmic transport has direct growth potential and has an indirect effect on osteogenic growth by influencing soft tissue growth.

   Neurotrophic mechanism:

   a. Neuroepithelial tropism: Epithelial growth is normally controlled by release of certain neurotrophic substances by nerve synapses. Lack of this neurotrophic process causes abnormal epithelial growth, orofacial hypoplasia and malformation, etc.
   b. Neurovisceral tropism: At the myoblast stage of differentiation, the embryonic myoblasts establish a neural innervation without which further myogenesis usually cannot continue.

c. Neuromuscular tropism: The periosteal matrices generally determine the apparent localized neurotrophically controlled genomes. The attributing factors that form basis of neurovisceral tropism, e.g. the salivary glands, fat tissue and other organ, regulate the embedded passive position of the skeletal units.

Q. 3. Discuss role of hormone and vitamins in growth and development. *(UOK, July 2017; HP Uni., April 2019)*

Q. Discuss factors affecting growth and development. *(RUHS, May 2018)*

Ans. Factors affecting growth and development

I. Heredity and genetics: Phenotype, characteristics of parents, race, sex, biorhythm and maturation, genetic disorders.
II. Environmental factors: Physical surroundings, social factors.
III. Prenatal environment (maternal nutritional deficiency, endocrine disorders, etc.), postnatal (nutrition, infections, trauma, emotion, etc.)

Hormone:

1. Group 1: Those influencing the skeletal bone growth: Growth hormone, insulin, thyrotrophic hormone.
2. Group 2: Responsible for ossification of long bones: Parathormone.
3. Group 3: Responsible for pubertal growth spurts: Androgens, progesterone and estrogen.
4. Group 4: Prolactin.

Q. 4. Write about prenatal and postnatal growth of cranial base. (*TNMGR, Sept. 2009*)

Ans. Cranial base (also known as base of skull, skull base; Latin: *basis cranii*) is the most inferior part of skull forming the floor of cranial cavity. The cranial base is formed by five bones: Ethmoid, sphenoid, occipital, both frontal and both temporal bones.

A. **Prenatal growth:** The earliest evidence of formation of cranial base is seen in post or late somitic period (4th–8th week of IUL). During this late somitic period mesenchymal tissue derived from primitive streak, neural crest and occipital sclerotomes condense around developing brain. Thus a capsule is formed around the brain called **ectomenix** or ectomeningeal capsule. From around 40th day onwards, this **ectomeningeal capsule** is slowly converted into cartilage which heralds onset of cranial base formation. This occurs in 4 regions.

1. **Parachordal:** Chondrification centers forming around cranial end of notochord.
2. **Hypophyseal:** Cranial to termination of notochord, hypophyseal pouch develops which gives rise to anterior lobe of pituitary gland. On either side of hypophyseal stem, two hypophyseal (post-sphenoid) cartilages develop, which fuse together to form posterior part of body of sphenoid.
   Cranial to the pituitary gland, two presphenoid or trabecular cartilages develop with fuse together and form anterior part of body of sphenoid. Anteriorly, pre-sphenoid cartilage forms mesethmoid cartilage which gives rise to perpendicular plate of ethmoid and crista galli.
   Lateral to pituitary gland, chondrification centers are seen which form lesser wing (orbito-sphenoid) and greater wing (ali-sphenoid) of sphenoid.
3. **Nasal:** Initially during development, a capsule is seen around nasal sense organ which chondrifies to form cartilages of nostrils.
4. **Otic:** A capsule seen around vestibulo-cochlear sense organs chondrifies and later ossifies to give rise to mastoid and petrous portions of temporal bone.
   The initially separate centers of cartilage formation in cranial base fuse together into a single irregular and greatly perforated cranial base. The early establishment of the various nervous, blood vessels, etc. from and to brain results in numerous perforations or foramina in the developing cranial base. The ossifying chondrocranium meets ossifying desmocranium (cranial vault) to form neurocranium.
   Chondrocranial ossification: The cranial base, which is now in a cartilaginous form, undergoes ossification.
   Occipital bone: Both endochondral and intramembranous ossification from 7 centers.
   Temporal bone: Both endochondral and intramembranous ossification from 11 centers.
   Ethmoid bone: This bone shows only endochondral ossification from three centers.
   Sphenoid bone: Endochondral and intra-membranous ossification from 15 ossification centers.
   Cranial base or chondrocranium is important as a junction between cranial vault and facial skeleton and is relatively stable during growth compared to cranial vault and face.

B. **Post-natal growth of the cranial base:** The cranial base grows post-natally by complex interaction between the following three growth processes.

1. **Cortical drift and remodeling:** The cranium is divided into a number of compartments by bony elevation and ridges present in cranial base. These elevated ridges and bony partitions show bone deposition, while predominant part of floor shows bone resorption to accommodate growing brain. The foramina that allow passage of nerves and blood vessels undergo drifting by bone deposition and resorption so as to constantly maintain their proper relationship with growing brain.
2. **Elongation at synchondroses:** Most of the bones of cranial base are formed by a cartilaginous process, later replaced by bone. However, certain bands of cartilage remain at junction of various bones, known as synchondroses. The important synchondroses found in cranial base are:
   a. **Sphenooccipital synchondrosis:** It is the cartilaginous junction between sphenoid and occipital bones and considered to be the most important growth site of cranial base. It is believed to be active up to age of 12–15 years. Sphenoid and occipital segments then become fused in midline area by 20 years of age. As endochondral bone growth occurs at sphenoccipital synchondrosis, sphenoid and occipital bones increase in length and width.
   b. **Sphenoethmoid synchondrosis:** This is a cartilaginous band between sphenoid and ethmoid bones. It is believed to ossify by 5–25 years of age.
   c. **Intersphenoidal synchondrosis:** It is a cartilaginous band between 2 parts of sphenoid bone. It is believed to ossify at birth.
   d. **Intraoccipital synchondrosis:** This ossifies by 3–5 years of age.
3. **Sutural growth:** Cranial base has a number of bones that are joined to one another by means of sutures. Some of them include:
   a. Sphenofrontal, b. Frontotemporal, c. Sphenoethmoid, d. Frontoethmoid, e. Frontozygomatic.
   As the brain enlarges during growth, bone formation occurs at the ends of bone.
   Timing of cranial base growth: By birth, 55–60% of adult size is attained; By 4–7 years, 94% of adult size is attained; By 8–13 years, 98% of adult size is attained.

Q. 5. Discuss the prenatal and postnatal growth of maxilla and mandible. (*TNMGR, March 2007; Sept. 2008; UHSR, May 2016; June 2018; BVP, June 2018; NTR Uni., May 2019*)

Q. Write a note on role of condyle in mandibular growth. (*HP Uni., May 2018*)

Q. Write a short note on nasomaxillary complex. (*RHUS, May 2018*)

Ans. Around 4th week of IUL, a prominent bulge appears on ventral aspect of embryo corresponding to developing brain. Below the bulge, a shallow depression corresponding to the primitive mouth appears, called **stomodeum**. The floor of stomodeum is formed by the **buccopharyngeal membrane** that separates stomodeum from foregut. Mesoderm covering developing forebrain proliferates and forms a downward projection that overlaps the upper part of stomodeum known as **frontonasal process**. Mandibular arches of both sides form lateral walls of stomodeum, gives off a bud from its dorsal end called **maxillary process**. The ectoderm overlying frontonasal process shows bilateral localized thickenings above stomodeum, **nasal placodes**. These placodes soon sink and form nasal pits. The formation of these **nasal pits** divides the frontonasal process into two parts: a. Medial nasal process, b. Lateral nasal process.

The two mandibular processes grow medially and fuse to form lower lip and lower jaw. As maxillary process undergoes growth, frontonasal process becomes narrow so that the two nasal pits come closer. The line of fusion of the maxillary process and medial nasal process corresponds to nasolacrimal duct.

Development of palate *(MUHS, Dec 2016)*: The palate is formed by contributions of the:

a. Maxillary process.
b. Palatal shelves given off by maxillary process.
c. Frontonasal process.

Ossification of palate: Ossification of palate occurs from 8th week of IUL. This is an intramembranous type of ossification. Palate ossifies from a single centre derived from maxilla. The most posterior part of palate does not ossify, forming **soft palate**. The mid-palatal suture ossifies by 12–14 years.

Development of Maxillary Sinus *(RGUHS, Oct. 2010)*

Prenatal embryology of mandible *(CDER-AIIMS, May 2015)*: About 4th week of IUL, pharyngeal arches are laid down on the lateral and ventral aspects of cranial most part of foregut that lies in close approximation with stomodeum. Initially there are six pharyngeal arches, but fifth one usually disappears. Each of these five arches contains:

1. A central cartilage rod that forms skeleton of arch.
2. A muscular component termed branchiomere.
3. A vascular component.
4. A neural element.

Mandibular process of both sides grows towards each other and fuses in midline forming lower border of stomodeum, i.e. lower lip and lower jaw.

Meckel's cartilage *(UHSR, April 2015)*: Meckel's cartilage is derived from 1st branchial arch around 41st and 45th day of IUL. It extends from cartilaginous otic capsule to midline or symphysis and provides template for guiding growth of mandible. A major portion of Meckel's cartilage disappears during growth and remaining part develops into following structures:

1. Mental ossicles, 2. Incus and malleus, 3. Spine of sphenoid bone, 4. Anterior ligament of malleus, 5. Sphenomandibular ligament.

Endochondral bone formation: Endochondral bone formation is seen in 3 areas of mandible:

- **Condylar process** *(CDER-AIIMS, Dec. 2016)*: At 5th week of IUL, area of mesenchymal condensation can be seen above ventral part of developing mandible. This develops into a cone-shaped cartilage by 10th week and starts ossification by 14th week and then migrates inferiorly and fuses with mandibular ramus by about 4 months.
- **Coronoid process:** Secondary/accessory cartilages appear in region of coronoid process by 10–14 weeks of IUL. It grows in response to developing temporalis muscle and gets incorporated into intramembranous bone of ramus and disappears before birth.
- **Mental region:** In the mental region, on either side of symphysis, one or two small cartilages appear and ossify in 7th month of IUL to form numbers of mental ossicles in fibrous tissues of symphysis. These ossicles become incorporated into intra-membranous bone when symphysis ossifies completely during the first year of post-natal life.

Postnatal growth of maxilla: Growth of **naso-maxillary complex (NMC)** is produced by:

1. **Displacement:** Growth of cranial base has a direct bearing on NMC as maxilla is attached to cranial base by number of sutures. Primary displacement is seen in forward direction by growth of maxillary tuberosity in posterior direction. Passive/secondary displacement of NMC occurs in downward and forward direction as cranial base grows. This results in whole maxilla being carried anteriorly.
2. **Growth at sutures:** Maxilla is attached to cranium and cranial base by number of sutures. These sutures include:

   a. Frontonasal suture, b. Frontomaxillary suture, c. Zygomaticotemporal suture, d. Zygomatico-maxillary suture, e. Pterygopalatine suture.

   These sutures are all oblique and more or less parallel to each other. This allows downward and forward repositioning of maxilla as growth occurs at these sutures. As growth of surrounding soft tissue occurs, maxilla is carried downwards and forward. This leads to opening up of space at sutural attachments, leading to new bone formation on either side of suture and hence overall size of bones increases.
3. **Surface remodeling:** The following are bone remodeling changes that are seen in NMC.
   - Resorption occurs on lateral surface of orbital rim leading to lateral movement of eyeball. To compensate, there is bone deposition on medial rim of orbit and on external surface of lateral rim.
   - Bone deposition occurs along posterior margin of maxillary tuberosity, causing lengthening of dental arch and enlargement of anteroposterior dimension of entire maxillary body. This helps to accommodate erupting molars.
   - Bone resorption occurs on lateral wall of nose leading to an increase in size of nasal cavity. Bone resorption is seen on floor of nasal cavity. To compensate there is bone deposition on palatal side. Thus a net downward shift occur leading to increase in maxillary height.
   - Zygomatic bone moves in posterior direction, achieved by resorption on anterior surface and deposition on posterior surface.
   - Face enlarges in width by bone formation on lateral surface of zygomatic arch and resorption on its medial surface.
   - Anterior nasal spine prominence increases due to bone deposition and resorption from periosteal surface of labial cortex. As a compensatory mechanism, bone deposition occurs on endosteal surface of labial cortex and periosteal surface of lingual cortex.
   - As teeth start erupting, bone deposition occurs at alveolar margins. This increases maxillary height and depth of palate.
   - The entire wall of sinus except mesial wall undergoes resorption. This results in increase in size of the maxillary antrum.

Postnatal growth of mandible *(KUHS, Jan. 2014; CDER-AIIMS, May 2015)*

Basal bone or body of mandible forms one unit, to which is attached alveolar process, coronoid process, condylar process, angular process, ramus, lingual tuberosity and chin.

- **Ramus:** It moves progressively posterior by combination of deposition on the posterior region and resorption on anterior part of ramus.

- **Corpus or body of the mandible:** Displacement of ramus results in conversion of former ramal bone into posterior part of body of mandible.
- **Angle of mandible:** On lingual side of angle of mandible, resorption takes place on postero-inferior aspect and deposition occurs on antero-superior aspect. On buccal side, resorption occurs on anterio-superior part and deposition takes place on postero-superior part. This results in flaring of angle of mandible as age advances.
- **Lingual tuberosity:** It is a direct equivalent of maxillary tuberosity. The combination of resorption in lingual fossa and deposition on medial surface of tuberosity accentuates prominence of lingual tuberosity.
- **Alveolar process:** As teeth erupt, alveolar processes develop and increase in height by bone deposition at margins.
- **Chin:** In infancy, chin is usually underdeveloped. Mental protuberance forms by bone deposition during childhood. Its prominence is accentuated by bone resorption that occurs in alveolar region above it, creating a concavity.
- **Condyle** *(CDER-AIIMS, Dec. 2016, 2018):* Mandibular condyle has been recognized as an important growth site.
  a. It was earlier believed that growth occurs at surface of condylar cartilage by means of bone deposition and hence condyle grows towards cranial base. As condyle pushes against cranial base, entire mandible gets displaced forwards and downwards.
  b. It is now believed that growth of soft tissues including muscles and connective tissue carries mandible forwards away from cranial base. Bone growth follows secondarily at condyle to maintain constant contact with cranial base.

  Condylar growth rate increases at puberty reaching a peak between 12 and 14 years. The growth ceases around 20 years of age.
- **Coronoid process:** Growth of coronoid process follows the 'V' principle. Viewing longitudinal section of coronoid process from posterior aspect, it can be seen that deposition occurs on lingual surface of left and right coronoid process. Although additions take place on lingual side, vertical dimension of coronoid process also increases.

Q. 6. Write a note on growth spurts.

Ans. There are accelerated periods of growth known as growth spurts that occur at specific times during which period of growth shows a definite increase. The physiological alteration in hormone is believed to be the cause for such accentuated growth. The timings of growth spurts differ in boys and girls. The timings of growth spurts are as follows: 1. Just before birth, 2. One year after birth, 3. Mixed dentition growth spurt: Boys: 8–11 years; Girls: 7–9 years, 4. Pre-pubertal growth spurt: Boys: 14–16 years; Girls: 11–13 years.

Importance: Pubertal increments offer best time for orthodontic and orthopaedic treatment.

Q. 7. Write a note on methods of studying growth.

(RGUHS, May 2006; UHSR, May 2007;CDER-AIIMS, May 2015; NITTE Uni., April 2017; HP Uni., May 2017)

Q. Write a short note on growth assessment.

(UHSR, April 2013; AHSUC, May 2018)

Ans. Growth is defined as increase in size or number. According to Profitt:

A. **Measurement approaches:**
1. Craniometry: It is based on measurements of skull of human skeletal remains. Precise measurements can be made on dry skulls, only for cross sectional studies.
2. Anthropology: Various landmarks established in studies of dry skull are measured in living individuals by using soft tissue points overlying bony landmarks. Measurements obtained would be of different results. Growth of an individual can be followed over a period of time with repeated measurements.
3. Cephalometric radiography: It is a standardized radiographic technique in craniofacial region, introduced by Broadbent in 1931. This is based on precise orientation of head before a cephalostat. It allows direct measurement of skeletal dimensions. Disadvantages include two-dimensional representations of structures, technique sensitive and not all measurements are possible.
4. Comparative anatomy: It is carried out through comparisons with other species.
5. 3-D Imaging: Computed tomography, CBCT

B. **Experimental approaches:**
1. Vital staining: It involves administration of dyes to experimental animals. Dyes used are alizarin red 5, tetracycline, trypon blue, and lead acetate.
2. Autoradiography: It is a technique in which a film emulsion is placed over a thin section of tissue containing radioactive isotope, and then is exposed in dark by radiation. The location of radiation in film indicates site of growth. Commonly used autoradiographic labels are: 3H Thymidine, 3H Proline, Bromodeoxyuridine.
3. Radioisotopes: Radioisotopes of certain elements are often used *in vivo* markers. When injected into tissue, get incorporated into the developing bone

and can be detected by means of Geiger counter, e.g. Technetium-33, Calcium-45, Potassium-32.

4. Metallic implants: Inert metal pins generally made of titanium are placed in growing bones of skeleton, including face and jaws. These metal pins are well tolerated by skeleton and become permanently incorporated into bone. These serve as reference points to study amount, direction and manner of growth.
5. Natural markers: Certain histological features present in normal bone such as nutrient canals, lines of arrested growth.

Methods of collecting growth data

1. **Longitudinal studies:** These are measurements made of same person or group at regular intervals through time. **Advantages:** Temporary problems are smoothed with time, variability in development within a group is put in proper perspective, and serial comparison makes study of specific developmental pattern of individual possible. **Disadvantages:** Time consuming, expensive, sample loss.
2. **Cross sectional studies:** These are measurements made of different samples or different individuals and studied at different periods. **Advantages:** Quicker, less expensive, statistical treatment of data is easier, studies can be readily repeated, Method can be used in archeological data. **Disadvantages:** Variation in development among individuals within the sample cannot be studied.
3. **Semi-longitudinal studies:** Longitudinal and cross sectional studies can be combined to seek advantages of both. In this way one might compress 15 years of study into 3 years of gathering growth data.

Q. 8. Write about different ways of age estimation.
(UHSR, May 2007)

Q. Write a short note on skeletal maturity indicators.
(DRMLA Uni., July 2013; BBD, April 2014; Gujarat Uni, June 2018)

Ans. Age estimation is an important factor in biological identification in many forensic fields, such as forensic odontology, forensic medicine, forensic anthropology, and forensic osteology.

Types of age

I. **Chronological age or real age:** It is measured by the calendar.
II. **Height and weight:** Age of a person can be roughly determined from standard charts of height and weight, but is least accurate and reliable.
III. **Skeletal age:** Determined by degree of ossification/development of various bones known to occur at particular time in average individual.

Skeletal maturity indicators

a. **Hand and wrist radiograph:** The hand wrist region is made of numerous small bones. The appearance, ossification and union of these bones from birth to maturity show an orderly sequence of events in predictable schedule pattern. It is indicated when there is marked discrepancy between chronologic and dental age. Methods used are: Atlas method by Greulich and Pyle (1959); Bjork, Grave and Brown method (1976); Julian Singer's Method (1980); Fishman's skeletal maturity indicators (1982); Hagg and Taranger method (1982).
b. **Cervical vertebrae:** Shape of cervical vertebrae changes according to each level of skeletal development. Methods used are: Lamparki's method; Hassel and Farman method.
c. **Tooth mineralization:** Stage of root formation and mineralization has close relation with skeletal maturation of an individual. Methods used are: Nolla's stage of calcification, Goldstein and Tanner method.
d. **Mid-palatal suture ossification.**

IV. **Dental age:** Determined by studying development of various teeth from the time of crypt is visible till the time of root completion.

Dental Age Estimation Methods:

a. **Morphologic/Visual Examination:** Morphological methods are based on assessment of teeth (*ex vivo*). Hence, these methods require extracted teeth for microscopic preparation. Gustafson (1950), Dalitz (1962), Bang and Ramm (1970), Johanson (1971), Maples (1978), Solheim (1993) are a few morphological methods.
b. **Radiographic Examination:** Radiographic assessment of age is a simple, non-invasive and reproducible method that can be employed both on living and unknown dead. Various radiographic images that can be used in age identification are intraoral periapical radiographs, lateral oblique radiographs, cephalometric radiographs, panoramic radiographs, digital imaging and advanced imaging technologies. The radiological age determination is based on assessment of various features as follows:
   - Jaw bones prenatally.
   - Appearance of tooth germs.
   - Earliest detectable trace of mineralization or beginning of mineralization.
   - Early mineralization in various deciduous teeth during intrauterine life.
   - Degree of crown completion.
   - Eruption of crown into oral cavity.

- Degree of root completion of erupted or unerupted teeth.
- Degree of resorption of deciduous teeth.
- Measurement of open apices in teeth.
- Volume of pulp chamber and root canals/ formation of physiological secondary dentine.
- Tooth-to-pulp ratio.
- Third molar development and topography

c. **Histological examination:** Dentin translucency method, incremental lines of cementum.

d. **Biochemical examination:** The biochemical methods are based on the racemization of amino acids. L-aspartic acids are converted to D-aspartic acids and thus levels of D-aspartic acid in human enamel, dentine, and cementum increase with age. Some of the methods are: 1. Helfman and Bada method (1975, 1976), 2. Ritz et al. method (1995).

3. DEVELOPMENT OF DENTAL TISSUES

Q. 1. Explain the formation, structure, chemical composition and physical properties of enamel. Describe the hydroxyapatite crystal.

(UHSR; Sumandeep Uni., April 2014)

Ans. Enamel is a highly mineralized structure covering the anatomic crown of tooth.

A. **Physical properties:**
1. It appears bluish-white or grayish at thick opaque areas and yellow-white at thin areas reflecting underlying dentin.
2. Enamel forms a protective covering of 2–2.5 mm thickness over crown and knife edge thickness at cervical region.
3. It is the hardest calcified tissue in human body, 5–8 KHN.
4. It is selectively permeable.
5. The specific gravity is 2.8.
6. Density decreases from surface of enamel to DEJ

B. **Chemical properties:** Inorganic material: 96%; Organic substance and water: 4%.

i. **Inorganic contents:** Hydroxyapatite (calcium phosphate), ions (Sr, Mg, Pb, Fl)

ii. **Protein content:** a. Amelogenin (90%, rich in prolin, histidine, glutamin, leucine); b. Non-amelogenin (10%, protein: Ameloblastin, tuftelin, enamelin)

Hydroxyapatite crystals: $Ca_{10}(PO_4)_6(OH)_2$; Crystals unite to form enamel rod or prism. Closely packed, long, ribbon-like carbonate crystals, arranged approximately parallel to long axis of rods. It comprises 88–90% of tissue by volume and 95% by weight. Present in form of crystallites; Length: 0.05–1µm; width: 90 µm. Shape is hexagonal in maturing enamel and irregular in matured enamel. It has central core or C-axis of hydroxyl ion around which calcium and phosphorous ions are arranged in form of triangle. During formation, Mg can replace Ca and carbonate can replace hydroxyl ion. Concentration of ions increases from surface of enamel towards dentin, that of fluoride decreases from surface of enamel towards dentin.

C. **Structure:** *(HP Uni., April 2019)*
1. **Enamel rod:** Enamel is composed of enamel rods or prisms (5–12 million). In cross section rods are hexagonal in shape (key hole/paddle shaped). Each enamel rod is built up of segments separated by dark lines. Generally rods are directed at right angles to dentin surface. In deciduous teeth, direction of rods is horizontal in cervical and central parts of crown. Near incisal edge or tip of cusp they gradually increase in oblique direction and almost vertical in cusp tip region. In permanent teeth, in occlusal 2/3rd of crown, direction of rods is oblique. In cervical region rods deviate from horizontal in apical direction.
2. **Rod sheath:** Thin peripheral layer, darker and less calcified than rod.
3. **Gnarled enamel:** This optical appearance of enamel is observed in oblique cut section as bundles of rods seem to interwine more irregular near dentin in region of cusps or incisal edge.
4. **Hunter-Schreger bands:** The regular change in direction of rods produces alternating dark and light strips.
5. **Incremental lines of Retzius:** Successive apposition of enamel during formation produces brownish bands.
6. External manifestations of Retzius striae is known as **Perikymata (imbrication lines)**
7. **Neonatal line:** Accentuated incremental line of Retzius marking the boundary between two portions of enamel of deciduous teeth formed partly before and partly after birth.
8. **Enamel cuticle/Nasmyth's membrane:** Delicate membrane covering the crown of newly erupted tooth.
9. **Enamel lamellae:** Thin leaf-like structures that extend from enamel surface toward DEJ.
10. **Enamel tufts:** Thin ribbon-like structures arising at DEJ and reaching into enamel.
11. **Enamel spindle:** Odontoblast process crossing DEJ into enamel.

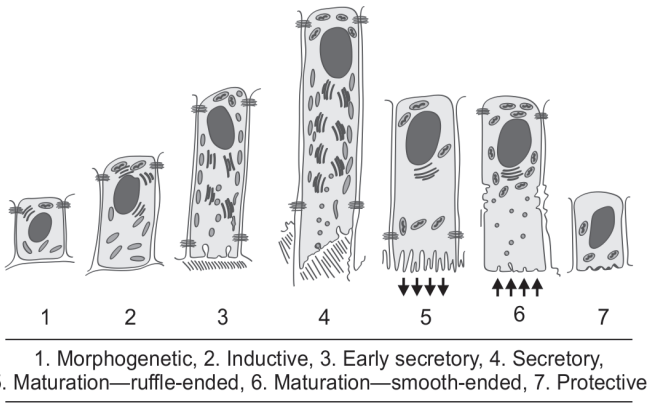

Fig. 3.1: Life cycle of ameloblasts

D. **Life cycle of ameloblasts** (Fig. 3.1): According to their function, ameloblasts can be divided into following stages:
   1. Morphogenic: Short columnar cell with large oval nucleus.
   2. Organizing: Cell become longer with reversal of polarity and nutritional stream.
   3. Formative: This starts after first layer of dentin is formed, development of Tome's process, key hole pattern of enamel rods.
   4. Maturative: Absorption of protein and deposition of minerals, Tome's process disappears.
   5. Protective: Radicular dentin formation, HERS breaks mineralized enamel in contact with dental follicle.
   6. Desmolytic: IEE stimulates dental follicle to form osteoclast activating factors, epithelia degenerates with tooth eruption.

Q. 2. Write a short note on amelogenins.

(RUHS, June 2017)

Ans. Enamel matrix proteins are generally classified into:
1. Amelogenin group: A 20-kDa hydrophobic protein
2. Enamelin group: A 65-kDa acidic protein and tuftelin.
3. Non-amelogenin, non-enamelin group: Ameloblastin (amelin or sheathlin).

Amelogenins constitute about 90% of total enamel matrix proteins and play a major role in mineralization and morphological changes in enamel. Human amelogenin gene has been located on X-chromosome at Xp22.1–p22.3 and on Y chromosome at Yp11.2.

Basic structure: Amino-terminal domain-A (hydrophobic) and carboxy-terminal domain-B (hydrophilic)

- **Role of amelogenin in enamel formation:** During enamel development and mineralization, secreted amelogenin are lost from tissue by specific proteases and replaced by mineral ions, calcium and phosphorus, which eventually results in fully mineralized hard and mature enamel.
- **Forensic dentistry:** Females have two identical amelogenin genes present on X-chromosome, whereas males have two different genes, present on both the sex chromosomes.
- **Regeneration of tissues:** Distinct isoforms of amelogenin have also been discovered in places other than enamel-like dentin matrix, odontoblasts, in remnants of Hertwig's root sheath and in periodontal ligament (PDL) cells, long bone cells such as osteocytes, osteoblasts, osteoclasts, some bone marrow cells, and articular cartilage, chondrocytes of articular cartilage and in cell layers of epiphyseal growth plate.
- **Future perspective as tumor markers:** Study of amelogenin gene and protein expression in odontogenesis and odontogenic neoplasms can be used as potentially useful polypeptide for identification of odontogenic epithelial components.

Q. 3. Write a short note on amelogenesis.

(TNMGR, March 2009; BBD Uni., April 2015)

Ans. Amelogenesis or development of enamel consists of two phases:

a. **Formation of enamel matrix:** The ameloblasts begin their secretory activity when a small amount of dentin has been laid down. The projection of ameloblasts into enamel matrix is called ***Tomes process***, gives junction between enamel and ameloblast a **picket fence** or **saw tooth appearance**. Two ameloblasts are involved in the synthesis of each enamel rod. The newly formed enamel matrix has two proteins: Amelogenin and enamelin.

b. Mineralization and maturation: Two stages
   1. First/primary mineralization stage: Immediate partial mineralization occurs in matrix segment and in interprismatic substance (25–30%).
   2. Second maturation stage: Gradual completion of mineralization. It starts from height of crown and progresses cervically. Each rod matures from depth to surface, and sequence of maturing rods is from cusps or incisal edge toward cervical line.

Applied Dental Anatomy

1. Defects in amelogenesis: Amelogenesis imperfecta.
2. Grooves and fissures on the occlusal surfaces are more prone to caries.
3. Lamellae, tufts and spindles may facilitate caries progression.
4. Enamel hypoplasia.

5. Enamel hypocalcification.
6. Fluorosis.

Q. 4. Write a short note on age changes in enamel. (*UOK, 2016; HP Uni., April 2019*)

Ans. Age changes in enamel:

1. Attrition or wear of occlusal surfaces and proximal contact points as a result of mastication.
2. Generalized loss of enamel rod ends.
3. Flattening of perikymata.
4. Finally complete disappearance of perikymata.
5. Localized increase of nitrogen and fluorine.
6. Teeth become darker due to increase in organic content and deepening of dentin colour.
7. Increase in resistance to decay.
8. Reduced permeability.
9. Enamel may become harder with age.

Q. 5. Write a short note on events in dentinogenesis with its anomalies. (*RGUHS, Oct. 2010*)

Ans. Dentinogenesis is formation of dentin by odontoblasts of mesenchymal origin located at periphery of dental pulp. Dentinogenesis begins when tooth germ reaches bell stage. Dental papilla is the formative organ, separated from IEE by cell free zone. Odontoblasts, dentin forming cells differentiate from ectomesenchymal cells of dental papilla following induction from IEE. Dentinogenesis consists of:

a. **Formation of collagen matrix (predentin):** Dentinogenesis begins at cusp tips after odontoblasts have differentiated and begin collagen production. Odontoblasts change their shape and size and give rise to several processes, which join together and become enclosed in a tubule. Collagen matrix formation continues, till the formation of crown and root formation. Initial dentin deposition along the cusp tips is known as **Korff's fibers**. Odontoblasts secrete both the collagen and other components of extracellular matrix.

b. **Mineralization:** Earliest crystal deposition is in the form of very fine plates of hydroxyapatite on surface of collagen fibrils and in the ground substance, subsequently within fibrils.

Anomalies of dentin formation

1. Dentinogenesis imperfect: Type I (Along with osteogenesis imperfect), II, III (Brandywine type)
2. Dentin dysplasia (rootless teeth): Type I—radicular; Type II—coronal.
3. Regional odontodysplasia (ghost teeth on radiograph).
4. Dens in dente (tooth within tooth).
5. Tetracycline pigmentation.

Q. 6. Write a short note on life cycle of odontoblasts. (*BBD, April 2013*)

Ans. Odontoblast is cell of neural crest origin that is a part of outer surface of pulp and its biological function is dentinogenesis. It is the 2nd most prominent cell in pulp. Odontoblast is a large columnar cells arranged in palisading pattern at periphery of pulp rich in rough endoplasmic reticulum (RER), Golgi complex with unidirectional secretory pattern and interconnected by macula adherens and gap junctions. No. of odontoblast = no. of dentinal tubules (59,000–76,000/mm^2). Odontoblast deposits 4 μm of dentin/day.

Life cycle: Fig. 3.6

1. **Odontoblast differentiation/preodontoblast stage:** In late bell stage, under the inductive influence of

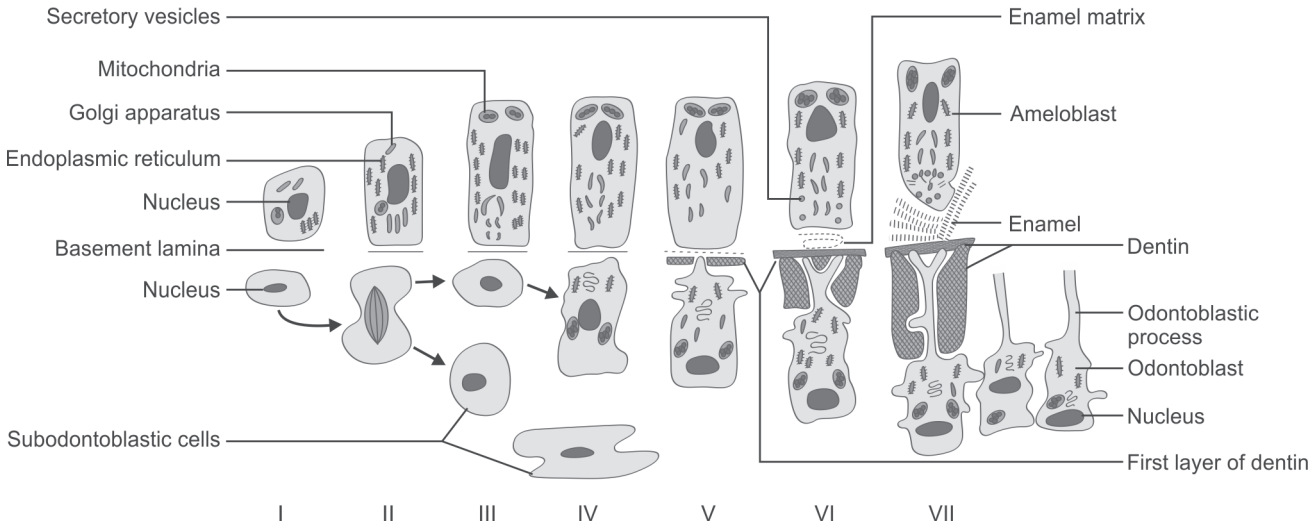

Fig. 3.6: Life cycle of odontoblasts: I–IV: Differentiation stage; V–VII: Formative stage

IEE, peripheral ectomesenchymal cells differentiate into preodontoblasts. They assume columnar shape, aligned as single row along the basement membrane with several projections.

2. **Formative/synthetic/active/secretory stage:** Secretory odontoblasts are aligned along the periphery of pulp. Functionally, it has cell body in which synthesis of proteins occurs and cell process whereby secretion occurs. The first sign of dentin formation is the appearance of distinct, large-diameter collagen fibrils called ***von Kroff's fibers*** (collagen type III). They originate deep among the odontoblasts; extend toward IEE, and immediately below epithelium.
3. **Quiescent/resting/aged odontoblast stage:** Stubby cells, scanty cytoplasm, dark closed faced nucleus, with absent secretory granules. This stage occurs after completion of circumpulpal dentin. The odontoblast loses most of their protein forming organelles to accommodate the decrease in their function. The fully differentiated and actively secreting odontoblasts decrease slightly in size and the cell process stop to elongate as dentin formation is reduced.

Fate of Odontoblasts: Life span of odontoblasts is equal to that of a viable tooth because once differentiated they cannot undergo further cell division. Resting odontoblasts involved in secondary dentinogenesis is renamed "**odontocytes**" because their function and properties are similar to osteocytes. These odontocytes may participate during reactionary dentinogenesis. Gene DMP1 is involved in differentiation of secretory odontoblasts into odontocytes.

Clinical Significance

1. Pathological differences in functional life of odontoblasts lead to dentinogenesis imperfecta.
2. Shell/thistle-tube teeth: Pre-odontoblasts do not differentiate into odontoblasts.
3. Pulpal obliteration: Odontoblasts do not differentiate into osteocytes.
4. Pink tooth: Outward resorption of dentinal tubules by odontoclasts results in pulpal tissue appearing pink through thin enamel.

Q. 7. Write a short note on age changes in dentin.

(TNMGR, March 2009; Oct. 2012;UHSR, May 2009; HP Uni., May 2015,April 2019; UOK, 2016; Sumandeep Uni., June 2016, 2017; CDER-AIIMS, May 2019)

Q. Write a note on structure of dentin and its clinical significance. *(RGUHS, July 2017)*

Ans. Dentin is the mineralized hard tissue forming main bulk of tooth, covered by enamel in crown and cementum in root.

A. **Physical properties:**
   1. Dentin is pale yellow to white in colour.
   2. Thickness ranges from 3 to 10 mm.
   3. Modulus of elasticity ranges from 15 to 20 GPA.
   4. Hardness is 68 KHN.
   5. Dentin is less radiopaque than enamel.

B. **Chemical properties:**
   Inorganic: 70%; Organic: 20%; Water: 10%

   Inorganic: Calcium hydroxyapatite crystals (mainly); Salt: Calcium carbonate, sulphate, phosphate, etc.; trace elements: Cu, Fe, F, Zn.

   Organic: Collagen (90%) Type I > III, V; non-collagen matrix proteins (phosphoproteins—phosphoryn; glycoproteins—sialoprotein, osteonectin, osteocalcin; Proteoglycans—chondroitin sulphate enzymes: Acid phosphatase, alkaline phosphatase; Lipids: Phospholipids, glycolipids).

Types of Dentin: (Fig. 3.7a and b)

1. **Primary dentin:** Dentin formed before complete root formation, forms most of the tooth portion. Primary dentin lining pulp chamber is referred as **circumferential dentin**. The outer layer is more mineralized is known as **mantle dentin**.
2. **Secondary dentin:** This is dentin formed after root completion. It is formed at slow rate and contains less number of tubules than primary dentin. It protects pulp from exposure in older teeth.
3. **Tertiary dentin/reparative/reactive/irritation/replacement/adventitious dentin:** This is formed when odontoblasts die during any operative procedure, erosion, dental caries, from newly formed odontoblasts, from underlying undifferentiated perivascular cells in deeper pulpal tissue. It has fewer tubules and more twisted.

Structure of Dentin *(RGUHS, July 2017)*

- Dentinal tubules: It is the unit structure of dentin, forms shallow 'S' shape at middle part of crown, straight at cusp and root portion of tooth. Their density increases towards pulp.
- Periodontoblastic space: Potential space between tubule wall and odontoblastic process contains nerves, collagen fibrils, plasma proteins, glycoprotein and mitochondria.
- Lamina limitans: Organic sheath lining the dentinal tubules.
- Dentinal fluid/dentin lymph: It is ultra-filtrate from pulp capillaries present between dentinal tubules and odontoblastic process.
- Predentin: First formed dentin consists of non-mineralized matrix.

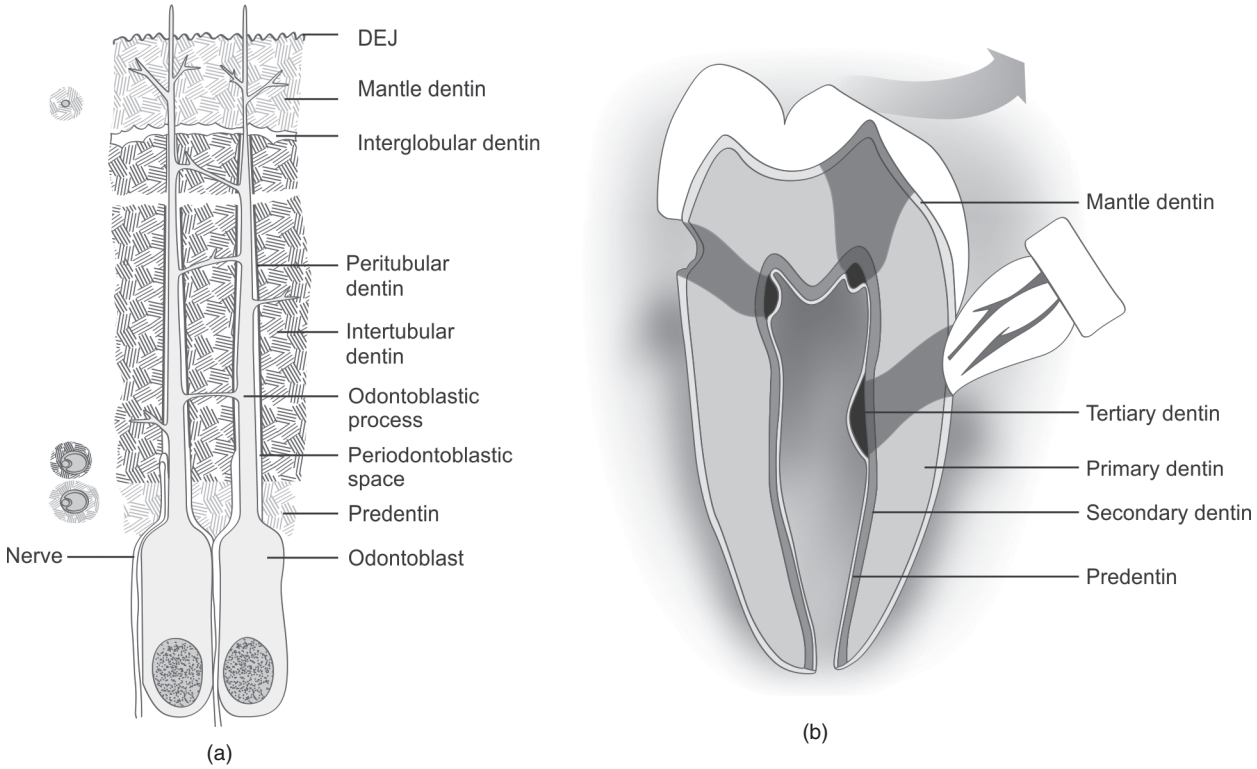

Fig. 3.7: (a) Structure of odontoblasts; (b) Dentin and its types

- Peritubular dentin: The wall of dentinal tubules surrounding the odontoblastic process.
- Intertubular dentin: The dentin in between dentinal tubules.
- Interglobular dentin: Areas of hypomineralization due to failure of fusion of mineral globules.
- Incremental lines of von Ebner/imbrication lines: These are fine striations perpendicular to dentinal tubules, due to daily rhythmic deposition of dentin.
- Lines of Schreger: Congruence of primary curvatures of dentinal tubules.
- Granular layer of Tomes': Smaller areas of interglobular dentin due to interference in mineralization of firstly formed layer of dentin, leading to looping of terminal ends of tubules.
- Contour lines of Owen: Accentuated incremental lines due to disturbance in matrix formation.
- Neonatal lines: Accentuated incremental lines due to disturbance in calcification, reflects abrupt changes in environment at birth.
- Dentinoenamel junction (DEJ): Scalloped interface between dentin and enamel, with convexity towards dentin.

Age Changes:

1. Physiologic secondary dentin formation.
2. Reparative dentin formation.
3. **Dead tracts:** Due to any mechanical injury, odontoblastic process may be lost, which appears black in transmitted light and white in reflected light.
4. **Sclerotic or transparent dentin:** Any external stimulus sometimes leads to increase deposition of collagen fibers and apatite crystals in the tubules, leading to complete obliteration.
5. **Eburnated dentin:** Exposed portion of reactive sclerotic dentine due to slow caries. It is hard, darkened, cleanable and resistant to further caries.

Innervation of dentin:

1. Numerous nerve endings in predentin and inner dentin.
2. Myelinated nerve fibres of pulp (Aδ) reach brain via trigeminal nerve.

Q. 8. Write a short note on dentin hypersensitivity. *(RGUHS, Oct. 2008; TNMGR, March 2011; Oct. 2013; Oct. 2016)*

Q. Describe the various theories and management of dentinal hypersensitivity. *(RGUHS May 2015; MUHS, June 2017)*

Ans. The only type of sensation obtained on dentine pulp complex is pain. Theories of dentinal hypersensitivity (Fig. 3.8).

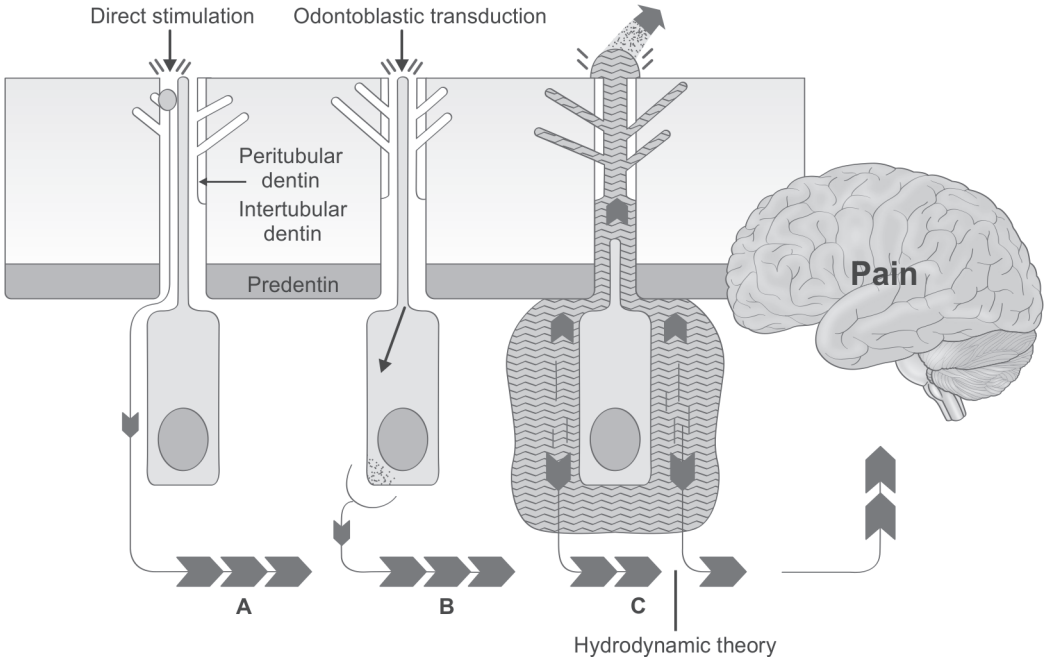

Fig. 3.8: Theories of pain transmission through dentin. A. Direct neural stimulation, B. Transduction theory, C. Hydrodynamic theory

1. **Direct neural stimulation (Scott Stella, 1963):** A stimulus reaches the nerve endings in the inner dentin. This theory assumes that nerve fibers extend to DEJ.
2. **Transduction theory:** This theory presumes that the odontoblasts process is the primary structure excited by the stimulus and impulse is transmitted to the nerve endings in the inner dentin. This is supported by fact that odontoblasts have neural crest in origin.
3. **Fluid or hydrodynamic theory (Gysi 1900, Brannstorm, 1967):** Any stimuli can affect the fluid movements in the dentinal tubules, this fluid movements further stimulates the pain mechanism in the tubules by mechanical disturbances of nerves closely associated with odontoblasts and its process. (most popular).

Management of hypersensitivity: Block the dentinal tubules. Topical fluoride application, fluoride iontophoresis, night guards, restorations, soft tissue grafting, lasers.

Q. 9. Write a short note on pain receptors in dental pulp. *(TNMGR, April 2012)*

Ans. Sensory nerve fibers of dental pulp are afferent endings of trigeminal cranial nerve. These fibers reach the root canal through apical foramen, going to root pulp in lumps. These lumps are often associated with blood vessels in a collagen sheath, forming neurovascular bundle. On approaching subodontoblastic region, fibers form an intricate network known as the ***plexus of Raschkow***. After this, myelinated fibers lose their myelin sheath and emerge as free nerve endings. These sensitive fibers act as nociceptors and belong to:

1. **C fibers:** Unmyelinated, sympathetic, found in close association with blood vessels, vasoconstriction and have a low conduction velocity, a smaller diameter, and a higher excitation threshold. They are located deeper than myelinated fibers and are principally activated by heat, causing slow, diffuse, and durable pain. If the pain stimulus intensity increases, the sensory C fibers are recruited and the pain becomes a burning sensation. The C fiber reaction shows that pulp damage is irreversible.
2. **Aδ fibers:** Small myelinated, fast conduction, low stimulation threshold, are superficial (located in pulp and dentin junction), transmit pain directly to thalamus, and generate a sharp and stabbing pain that is easily localized. These characteristics make them the first nerve fibers to react and transmit pain impulse even when there is no irreversible tissue damage.
3. **Aβ fibers:** Large myelinated functionally similar to Aδ fibers but are stimulated at lower electrical threshold. These fibres are located at pulp-dentin border or in close proximity to odontoblast cell body.
4. **Parietal layer of nerves or plexus of Raschkow:** Formed of myelinated and non-myelinated fibers.

Q. 10. Discuss functions of pulp and its response to various stimuli. (*TNMGR, April 2013; MUHS, June 2017*)

Ans. Pulp is a mass of connective tissue that resides within the center of tooth, directly beneath the layer of dentin. It is a part of "dentin-pulp" complex, and also known as **endodontium.**

Functions of Pulp:

1. **Inductive:** Induces oral epithelium to differentiate into dental lamina and enamel organ.
2. **Formative:** It produces dentin through odontoblasts.
3. **Nutritive:** It nourishes dentin, by means of its rich vascular supply.
4. **Protective:** It responds to various types of stimuli, by forming secondary and tertiary dentin, which increases coverage of pulp.
5. **Defensive or reparative:** It responds to any irritation by forming reparative dentin.
6. **Sensory:** Changes in temperature, vibration and chemical that affect the dentin and pulp.

Response of pulp to stimuli: The pulp is highly responsive to any stimuli. Even a slight stimulus will cause inflammatory cell infiltration, hyperemia or localized abscess. Hemorrhage may be present. The odontoblast layer is either destroyed or greatly disrupted. Compound containing calcium hydroxide induces reparative dentin formation. Closer the restoration to pulp, greater will be the pulp response.

Q. 11. Write a short note on age changes in pulp. (*TNMGR, March 2009; KUHS, Jan. 2014; Sumandeep Uni., June 2016, 2017; UHSR, May 2017; HP Uni., April 2019*)

Ans. The physiology of pulp has been known to change over time due to the aging process, resulting in distinct phenotypic differences. Compromised circulation and innervation occurs due to increased deposition of secondary dentin at apical root, constricting apical foramen.

1. Decrease in number as well as size of pulp cells with aging.
2. Increase fibrosis and collagen fibers in pulpal tissue.
3. Appearance of atherosclerotic plaques and calcification in pulpal vessels.
4. Progressive mineralization of nerve sheath.
5. Formation of pulp stones or denticles.
6. Formation of diffuse calcifications in pulp chamber.
7. The number of cells in pulp decreases as cell death occurs with age.
8. The volume of pulp chamber decreases with continued deposition of dentin.
9. In some cases, pulp chamber can be obliterated with aging.

Q. 12. Write a short note on calcifications of pulp. (*TNMGR, Oct. 2012*)

Q. Write a short note on pulp stones. (*BFUHS, Nov. 2007*)

Ans. Calcification is hardening of tissue by deposition of or conversion into insoluble calcium salts or compounds.

Diffuse calcifications: They appear as irregular calcific deposits in pulp tissue, usually following collagenous fiber bundles or blood vessels. The pulp chamber may appear normal, with these calcifications in roots. These calcifications may be classified as **dystrophic calcifications**.

Pulp stones (Denticles): Nodular, calcified masses appearing in both coronal and root portions of pulp organ. They are usually asymptomatic. **True denticles** are similar in structure to dentin, as they have dentinal tubules. They are rare and usually located close to apical foramen. **False denticles** do not exhibit dentinal tubules. They appear as concentric layers of calcified tissue. Pulp stones may be classified as **free, attached** or **embedded.** Pulp stones may appear close to blood vessels and nerve trunks. Their incidence as well as size increases with age. They are found more commonly in coronal pulp (Fig. 3.12).

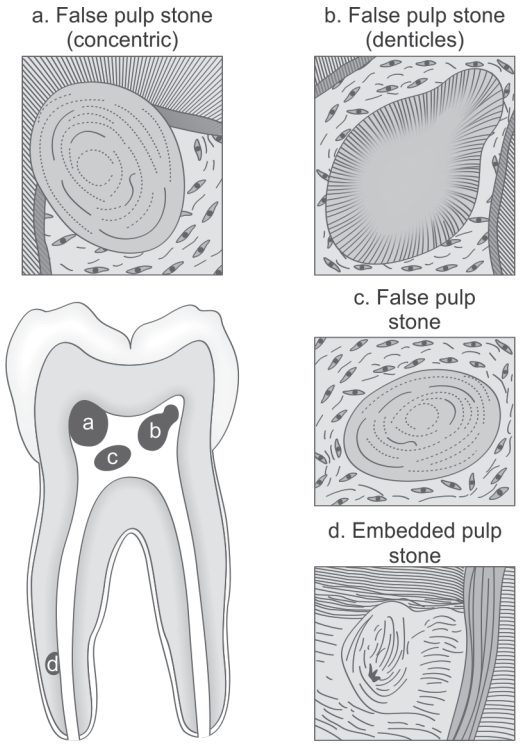

Fig. 3.12: Pulp stone types and its location

Clinical Significance

1. Presence of pulp stones may alter the internal anatomy of pulp cavity, making access opening of tooth difficult.
2. Pulp stone in large number may indicate chronic irritation of pulp.

Q. 13. Discuss pulpo-dentinal complex as "marriages are made in heaven." *(BBD Uni., April 2014; RGUHS, May 2014)*

Q. Discuss histopathology of dental pulp. *(Sumandeep Uni., April 2015)*

Ans. Dentin and pulp are embryologically, histologically, and functionally the same tissue and therefore are considered as a complex. Both pulp and dentin have a common origin from dental papilla. Structure and response of dentin to injury are largely functions of odontoblasts and other cells in pulp, but these cells are dependent on dentin for their protection and state of differentiation. The embryonic dental papillae are responsible for formation of this coupled tissue. The response of pulp to any restorative material will be influenced by its surrounding dentin also. Dentinal fluid in the tubules, which is continuous with extracellular fluid of pulp, serves as a medium for relaying injurious agents to pulp to induce an inflammatory response.

Pulp: Parts of pulp:

Pulp chamber/coronal pulp: Located in crown of tooth.

Root canal/radicular pulp: Pulp located in root area.

Apical foramen: Opening from pulp at apex of tooth.

Accessory/lateral canal: Extra canal located on lateral portions of root.

Microscopic Zones in pulp (zones from outer to inner zone) (Fig. 3.13):

1. Odontoblastic layer: Lines outer pulpal wall and consists of cell bodies of odontoblast. Secondary dentin may form in this area from apposition of odontoblast.
2. Cell-free zone (zone of Weil): Fewer cells than odontoblastic layer. Nerve and capillary plexus located here.
3. Cell-rich zone: Increased density of cells as compared to cell-free zone and also a more extensive vascular system.
4. Pulpal-core: Located in center of pulp chamber, which has many cells and an extensive vascular supply, similar to cell-rich zone.

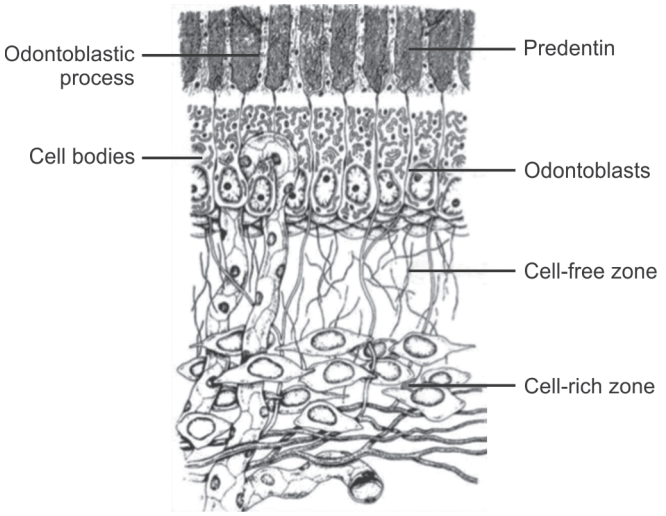

Fig. 3.13: Pulp dentin complex

Contents of the Pulp

1. Cells: Odontoblast, fibroblast, white-blood cells, undifferentiated mesenchymal cells, macrophages and lymphocytes. No fat cell.
2. Fibrous matrix: Mostly reticular fibres and collagen fibres (type I and type III).
3. Ground substance: Act as medium to transport nutrients to cells and metabolites of cell to blood vessels.

Vascularity and nerves of pulp: Pulp organ is extensively vascular with vessels arising from external carotids to superior or inferior alveolar arteries. It drains by same vein.

Nerves: Several large nerves enter apical canal of teeth. This trunks transverse radicular pulp, proceed to coronal area and branch peripherally.

Q. 14. Write about portal of infections in dental pulp. *(Sumandeep Uni., April 2014)*

Ans. The various routes by which the microorganisms reach the pulp are as follows.

1. **Dentinal tubules:** After a carious lesion or during dental procedures, microorganisms may use the pathway in a centripetal direction to reach pulp.
2. **Open cavity:** Direct pulp exposure due to operative procedures, breaks physical barrier imposed by dental structures and leaves pulp in contact with septic oral environment.
3. **Periodontal membrane:** Microorganisms from gingival sulcus may reach pulp chamber through PDL, using lateral channel or apical foramen as pathway.

4. **Blood stream:** A transient bacteremia in blood may get attracted to pulp following trauma or operative procedure that produced inflammation without causing pulp exposure (***anachoresis***).
5. **Faulty restoration:** In obturated tooth with gutta percha and sealer, contamination may occur if the temporary seal is broken or if the tooth structure fractures before final restoration, or if final restoration is inadequate.
6. **Extent:** Microorganisms might reach the principal and/or lateral canals migrating from an infected tooth to a healthy pulp as a consequence of contiguousness of tissues.

Q. 15. Write a short note on cementum.

(*TNMGR, Oct. 2013*)

Q. Write a short note on histology/ultrastructure of cementum.

(*UHSR, 2013; TNMGR, Oct. 2018*)

Ans. Cementum is the mineralized dental tissue covering the anatomic roots of human teeth. It is formed by connective tissue cells (cementoblasts) of dental follicles, which comes in the contact of newly formed radicular dentin. It is light yellow in colour and softer than dentin.

Physical Characteristics:

1. Calcified structure whose calcification and hardness is less than dentin.
2. More permeable than dentin.
3. Light yellow in color.
4. Lacks luster and is dark than enamel.
5. Less readily resorbed than bone.
6. Begins at cervical portion of tooth at CEJ and continues to apex.
7. Thickest at apical region and thinnest at CEJ.

Chemical Composition:

1. Organic content and water: 50–55%.
   a. Collagen (type I, III, V, IX), GAGs (chondroitin 4-sulphate, dermatan sulphate).
   b. Non-collagenous proteins: Alkaline phosphatase, bone sialoprotein, fibronectin, osteocalcin, osteonectin, osteopontin, vitronectin, cementum derived attachment protein, insulin like growth factor-I
2. Inorganic content: 45–50%.
   a. Calcium and phosphate as hydroxyapatite.
   b. Trace elements like Cu, F, Fe, Pb, K, Si, Na, Zn. *Cementum has the highest fluoride content.*

Classification

A. **Location:**
   1. Coronal: Formed over enamel covering the crown.
   2. Radicular: On root surface.

B. **Presence/absence of cells:**
   1. Cellular cementum: Secondary cementum formed after tooth reaches occlusal plane, less calcified, contains cementocytes; more frequent on apical half.
   2. Acellular cementum: First formed before tooth reaches occlusal plane, more calcified, devoid of cementocyte; more frequent on coronal half of root.

Histology:

A. **Cells, fibres, ground substance:** Cementoblast (synthesis collagen and organic matrix), cementocytes (present in cellular cementum), cementoclasts (cementum resorption and repair)

B. **Incremental lines of Salter:** Accentuated lines of highly mineralized areas with less collagen due to rhythmic periodic deposition of cementum.

C. **Cementoenamel junction (CEJ):**
   1. Cementum overlaps enamel: 60–65% of the teeth.
   2. Cementum meets enamel in a sharp line: 30% of the teeth.
   3. Cementum and enamel does not meet at all: 5–10% of the teeth.

D. **Cementodentinal junction (CDJ):** Terminal apical area of cementum where it joins internal root dentin.

Functions:

1. It furnishes a medium for attachment of collagen fibers, bind tooth to alveolar bone.
2. It serves as major reparative tissue for root surfaces.
3. It helps in functional adaptation of teeth.

Q. 16. Write a short note on cementogenesis.

(*RGUHS, Nov. 2011*)

Ans. Cementum formation (cementogenesis) is preceded by deposition of dentin along the inner aspect of HERS. The newly formed dentin comes in contact of connective tissue of dentin follicle, forming cementoblast. Cementoblasts synthesize collagen and protein polysaccharides, which make up cementum matrix. After this, mineralization of matrix starts, by deposition of calcium and phosphate ions present in tissue fluids.

Stages of cementogenesis (Fig. 3.16)**:**

Phase I: Laying down of cementoid tissue (matrix formation)

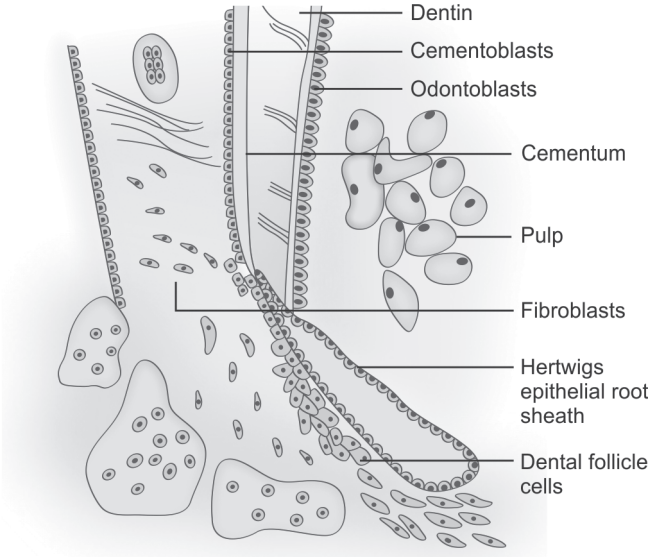

Fig. 3.16: Cementogenesis

Phase II: Mineralization: Apatite crystals are deposited along fibrils.

Cementum formation takes place rhythmically. A thin layer of cementoid is seen on surface of cementum lined by cementoblasts. These fibers are embedded in cementum and attaches tooth to surrounding bone. (***Sharpey's Fibers***)

Cementum is laid down much slowly while the tooth is erupting. This cementum is **acellular or primary**. When tooth comes in occlusion, more cementum forms around the apical 2/3rd of root, which has greater proportion of collagen. The cementoblasts become trapped in lacunae within this matrix. This cementum is called **cellular (secondary)** cementum. The rate of formation of cellular cementum is much more rapid than that of acellular cementum.

Q. 17. Write about role of cementum in health and diseases. (*Suman Vidyapeeth, April 2010*)

Ans. Cementum in health:

1. Cementum is more resistant to resorption than bone.
2. Cementum resorption can occur after trauma or excessive occlusal forces.
3. After resorption the damage is repaired by formation of new cementum.
4. Transverse fracture of the root is repaired by formation of new cementum.
5. In cases of gingival recession, cementum may get hypermineralized.

Cementum in disease:

1. **Developmental anomalies:**
   a. Concrescence: Union of roots by cementum.
   b. Ectopic enamel: Presence of enamel on root cementum.
   c. Enamel pearl: Hemispheres of enamel with dentin and pulp present in furcation area.
   d. Cervical enamel projections: Dipping of enamel from CEJ towards bifurcations (mandibular molars).
   e. Hypercementosis: Non-neoplastic deposition of excessive cementum, seen in abnormal occlusal trauma, arthritis, Paget's disease.
   f. Ankylosis: Anatomic fusion of tooth cementum or dentin with alveolar bone.
2. **Regressive alterations of teeth:**
   a. Abrasion: Pathologic wearing of tooth substance by abnormal mechanical process, usually on exposed root surfaces.
   b. Cementicles: Dystrophic calcifications which lie free in periodontal ligament.
   c. Root caries: Soft progressive lesion found anywhere on root surface, that is exposed to oral environment.
   d. Calculocementum: Calculus embedded deeply in cementum.
3. **Due to periodontal pathology:**
   a. Subsurface alteration: Due to gingival inflammation, alterations in structure and composition.
   b. Cervical root resorption.
   c. Bacterial contamination due to exposure to oral environment.
   d. Pathologic granules: Areas of collagen degeneration.
   e. Areas of increased mineralization/demineralization.
   f. Cellular resorption of cementum.
4. **Neoplasms of cementum:**
   a. Benign cementoma.
   b. Periapical cemental dysplasia.
   c. Central cementifying fibroma.
   d. Gigantiform cementum.
   e. Focal cemento-osseous dysplasia
5. **Systemic disease**
   a. Cleidocranial dysplasia: Absence of cellular cementum.
   b. Hypophosphatasia: Absence of cementum.
   c. Hyperpituitarism: Hypercementosis.
   d. Hypothyroidism: External resorption of roots.
   e. Hyperparathyroidism: Loss of lamina dura and root resorption
   f. Paget's disease: Hypercementosis, loss of lamina dura and root resorption
6. In forensic odontology: Age estimation from incremental lines of cementum (acellular cementum).

Q.18. Write a short note on development of periodontal ligament.

(*MAHE, Dec. 1997; TNMGR, Sept. 2007; BFUHS, Nov. 2007; UHSR, April 2015, May 2017*)

Ans. Development (Fig. 3.18): Enamel organ is surrounded by a condensation of ecto mesenchymal cells called **dental sac**. The part of dental sac immediately close to enamel organ is called **dental follicle**. Once HERS disintegrates leaving behind **epithelial rests of Malassez**, cells of dental follicle come close to surface of newly formed dentin. The dental follicle cells then differentiate into cementoblasts and lay down cementum on dentin on the developing root. The other cells of dental follicle differentiate into fibroblast and lay down fibers and ground substance of periodontal ligament. As crown approaches the oral mucosa during tooth eruption, these fibroblasts become active and start producing collagen fibrils. They initially lack orientation, but they soon acquire an orientation oblique to tooth. The first collagen bundles appear in the region immediately apical to CEJ and give rise to gingivodental fiber groups. As tooth eruption progresses, additional oblique fibers appear and become attached to newly formed cementum and bone. The transseptal and alveolar crest fibers develop when tooth merges into oral cavity. Alveolar bone deposition occurs simultaneously with PDL organization. ***Sharpey's fibers*** are fewer in number and more widely spaced than those emerging from cementum. At later stage, alveolar fibers extend into middle zone to join lengthening cemental fibers, attain their classic orientation, thickness and strength when occlusal function is established.

Q. 19. Write a short note on periodontal ligament (PDL). Write about its age changes.

(*RGUHS, April 2006; TNMGR, March 2010; RUHS, June 2017*)

Q. Discuss the architecture variability and clinical considerations with respect to periodontal ligament.

(*UHRS, 2013; HP Uni., April 2019*)

Ans. The periodontal ligament (PDL) is a specialized connective tissue which occupies the space between root and alveolar bone of tooth socket. Width range is 0.15–0.38 mm, thinnest around middle third of root. (***Hourglass shaped***)

Cells of Periodontal Ligament (PDL):

A. Synthetic cells: Osteoblasts, fibroblasts, cementoblasts.
B. Resorptive cells: Osteoclasts, fibroblasts, cementoclasts.
C. Other cells: Epithelial rests of Malassez, defense cells (mast cells, macrophages, eosinophils).

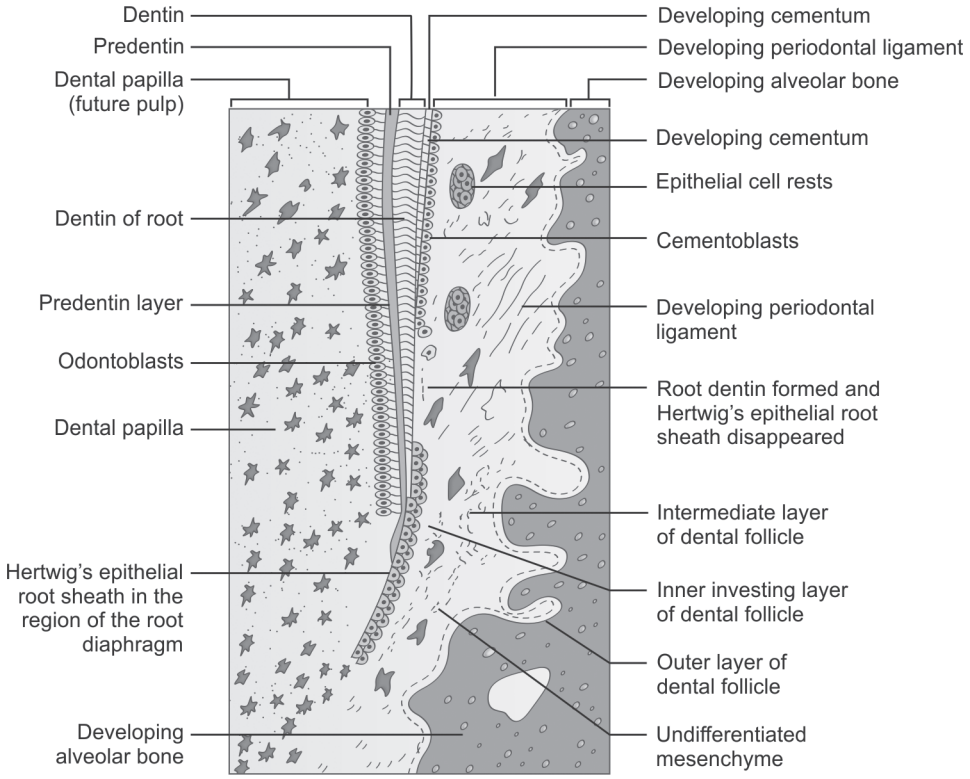

Fig. 3.18: Development of periodontal ligament (PDL)

Extracellular Substances

A. Connective tissue fibers: Collagen fibers (type I, III, XII), oxytalan fibers, reticular fibers, elastic fibers.
B. Ground substance: Proteoglycans (chondroitin sulphate, dermatan sulphate, heparin sulphate, hyaluronic acid), glycoprotein (fibronectin, laminin, tenascin)

Blood supply: Perforating arteries arising from intra-alveolar vessels.

Nerve supply: A and C fibers from trigeminal nerve. These fibers end as:

a. Free nerve ending: Present along the length of root, nociceptors.
b. Ruffini's endings: Appear dendrite and end in terminal expansions among PDL bundle mechano-receptors.
c. Meissner's corpuscles: Coiled form in mid-region, tactile perception.
d. Encapsulated spindle like endings: Associated with root apex temperature receptor.

Fibers of PDL: Principal fibers consist of individual fibers forming a continuous anastomosing network between tooth (cementum) and bone (Fig. 3.19).

1. **Transseptal group**: Extend interproximally in cementum of adjacent teeth, resist tooth separation, mesial or distal.
2. **Alveolar crest group:** Prevent extrusion of teeth and resist lateral movement.
3. **Horizontal group:** Resist horizontal and tipping forces.
4. **Oblique group:** Largest group, resist apically directed masticatory forces.
5. **Apical group:** Prevent tooth tipping; do not occur on incompletely formed roots.

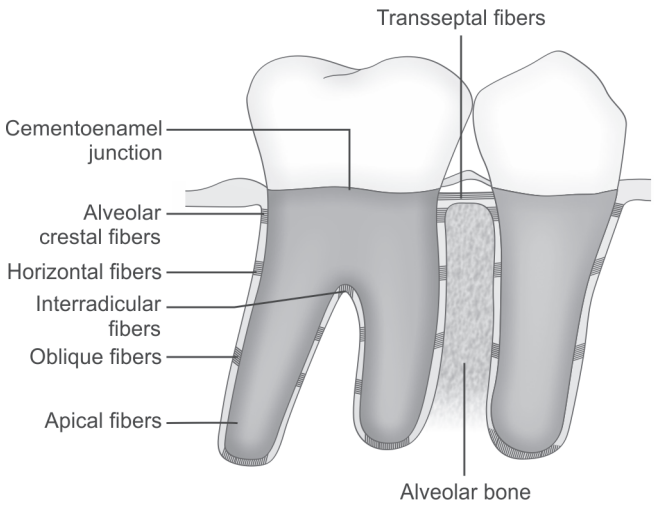

Fig. 3.19: Principal fibers of PDL

6. **Interradicular group:** Resist forces of luxation and rotation, present onto the furcation areas of multi-rooted teeth.

Functions:

1. Provision of a soft tissue "casing" to protect vessels and nerves from injury by mechanical force.
2. Transmission of occlusal forces to bone.
3. Attachment of the teeth to bone.
4. Maintenance of gingival tissues in their proper relationship to teeth.
5. Resistance to impact of occlusal forces (shock absorption)
6. Formative: Helps in formation of collagen, cementum, and bone.
7. Sensory: PDL transmits sensation of tactile, pressure and pain.
8. Nutritive: Supplies nutrition to cementum, bone and gingiva.
9. Homeostatic.

Age Changes:

1. Reduction in vascularity, elasticity and reparative capacity.
2. Decreased number of fibroblasts with more irregular structure is seen.
3. Decreased collagen synthesis.
4. Decrease in no. of periodontal fibers.
5. The fiber bundles become thicker, broader and more highly organized.
6. Presence of areas of hyalinization.
7. Decreased organic matrix production and epithelial cell rests.
8. Increased amount of elastic fibers.
9. Reduction in width of PDL space.

Q. 20. Write a short note on fibroblasts.

Ans. Fibroblasts are principal cells of PDL characterized by their rapid turnover of extracellular compartment, collagen. Collagen fibrils of bundles are continuously being remodeled by fibroblasts, which are capable of simultaneously synthesizing and degrading collagen.

Structure: Fibroblasts stem from a mesenchymal origin and have an elongated spindle or stellate shape with a multitude of cytoplasmic projections. Within the cytoplasm is an abundance of rough endoplasmic reticulum (RER) and large Golgi apparatus.

Products of fibroblasts

- Collagen type I, III, and IV, proteoglycans, fibronectin, laminins, glycosaminoglycans, metalloproteinases, and prostaglandins.
- Transcription growth factor-alpha and beta (TGF-α and TGF-β), platelet-derived growth factor (PDGF),

granulocyte macrophage colony-stimulating factor (GM-CSF), epidermal growth factor (EGF) and tumor necrosis factor (TNF).
- Fibroblasts are known for their plasticity; adipocytes, pericytes, endothelial and epithelial cells, can de-differentiate into fibroblasts.
- They can transform into myofibroblasts, present in both healthy and pathologic tissues and contain features of fibroblasts and smooth muscle cells.

Functions
- Activated fibroblast can be induced by appropriate stimuli from macrophages, lymphocytes, mechanical force, and bacteria.
- In connective tissue remodeling, fibroblasts are capable of synthesis and phagocytosis of collagen and components of extracellular matrix.
- Cytokines produced by fibroblasts have capacity to mediate tissue destruction and stimulate osteoclastic bone resorption.
- Fibroblast-derived proinflammatory mediators and cytokines are implicated in periodontal tissue destruction by promoting fibrosis, granuloma formation or bone resorption.

Q. 21. Write a short note on alveolar bone.

(*TNMGR, March 2007; KLE Uni., Dec. 2008; NTR Uni., May 2019*)

Q. Write a short note on bone cells.

(*TNMGR, Sept. 2010*)

Q. Discuss alveolar bone in health and disease.

(*UHSR, May 2016; May 2017; HP Uni., May 2017; April 2019*)

Ans. Alveolar bone may be defined as that part of maxilla and mandible that forms and supports sockets of teeth. It is also known as "**functional bone**" as it is susceptible to functional changes and is lost after tooth extraction.

Development: Near end of 2nd month of IUL, mandible and maxilla form a groove that opens towards the surface of oral cavity. As tooth germs start to develop, bony septa form gradually and alveolar process starts developing during tooth eruption.

Structure

a. Alveolar bone proper: Thin lamella of bone that surrounds root of tooth and gives attachment to principal fibers of PDL, perforated by multiple openings that carry nerves and blood vessels into PDL, so it is known as "**Cribriform plate**."
b. Supporting alveolar bone: Bone that surrounds alveolar bone proper and gives support to socket.
   1. Cortical plates—compact bone, forming inner and outer plates of alveolar processes. Much thicker in mandible than maxilla and thickest in premolar, molar region on buccal side.
   2. Spongy bone—fills the area between cortical plates and alveolar bone proper, not found in anterior teeth region.

In health, the distance of 1.5–2 mm is always maintained between alveolar crest and adjacent teeth.

Cells:
1. **Osteoprogenitor cells:** Undifferentiated mesenchymal cells and hematopoietic stem cells divide and transform into osteoblasts and osteoclasts.
2. **Osteoblasts:** Bone forming cells. Formed from multipotent mesenchymal cells. They secrete type I collagen and bone matrix (osteoid). They exhibit a high level of alkaline phosphatase.
3. **Osteoclasts:** Bone resorbing cells. Multinucleated, found in Howship's lacunae, derived from circulating monocytes and local mesenchymal cells.
4. **Osteocytes:** Entrapped osteoblasts in lacunae are called osteocytes. They resorbs surrounding bone to form spaces called *osteocytic lacunae*.

Age changes:
1. More irregular periodontal surface of bone
2. Less regular insertion of collagen fibers.
3. Osteoporosis.
4. Decreased vascularity.

Alveolar bone in disease: Conditions of alveolar bone loss:
1. Extensive gingival inflammation.
2. Trauma from occlusion
3. Systemic conditions: Vitamin D deficiency, diabetes, hyperparathyroidism, etc.
4. Periodontitis/abscess
5. Tooth extraction
6. Overhang restoration
7. Ill fitting prosthesis

Q. 22. Write a short note on bundle bone.

(*TNMGR, Sept. 2007*)

Ans. The alveolar bone proper consists of lamellated and bundle bone. Bundle bone is that bone in which the principal fibers of the PDL are anchored. The term bundle bone was chosen because the bundles of the principal fibers continue into the bone as Sharpey's fibers. It is characterize by scarcity of fibrils in intercellular substance, which are arranged at right angles to Sharpey's fibers. It contains fewer fibrils than lamellated bone, and therefore, it appears dark in H&E stained sections. The bundle bone contains more

calcium salts per unit area than other types of bone tissues, such areas are seen as dense radiopacities (lamina dura), radiographically

Q. 23. Write a short note on lamina dura.
(*TNMGR, Oct. 2013*)

Ans. Lamina dura (LD) is a radiographic landmark viewed largely on periapical radiographs (PR). The terminology LD (or alveolus) is applied to the thin layer of dense cortical bone, which lines the roots of sound teeth. It appears as a well-defined radiopaque (white) layer in radiograph. The term lamina dura or "hard layer" is derived from the fact that it is more radiopaque than adjacent bone. Presence of LD is an indication of health of teeth. Radiographically it is seen as a thin radiopaque line running around the length of roots. Adjacent to the LD, on tooth side, a thin dark shadow represents the space occupied by periodontal membrane, known as periodontal space. PR has mainly been used to assess both periodontal ligament (PDL) space and LD. The presence or absence of LD and PDL space on radiographs may also be affected by any variations in angulations of X-ray beam. The convexity or concavity of proximal tooth surfaces, the curvature of roots, level of cementoenamel junction and thickness of alveolar bone may also cause variations in thickness and clarity of the LD.

Q. 24. Write a short note on cusp of Carabelli.
(UHSR, June 2017)

Ans. It is accessory lingual cusp located on mesio-palatal cusp of maxillary second deciduous molars and first, second and third permanent molars. It may be unilateral or bilateral, with marked deviation in size. In some cases, accessory cusp is seen occasionally on mandibular permanent or deciduous molar, this is called ***protostylid***. Carabelli's trait was found in 1842 by Sir Georg Carabelli. Carabelli's trait is one of the most studied nonmetric traits. The Carabelli's trait has been used as a critical ethnic indicator for several decades, most likely because it can be simply observed in both living individuals and skeletal material, and can, therefore, be used to show major ethnic differences in dentition. Dahlberg's classification is the most commonly applied method for determining degree and expression of Carabelli cusps. (Fig. 3.24). For permanent dentition, Carabelli's trait appears to be generally the most common among the European populations, followed by African populations and American Indians, with lowest prevalence occurring in other Mongoloid races.

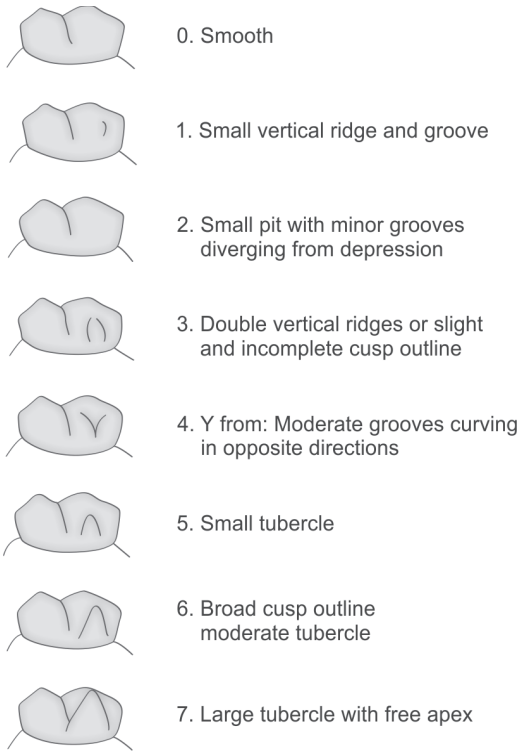

Fig. 3.24: Dahlberg's (1963) scale for the determination of degree and expression of Carabelli cusps

4. ORAL MUCOUS MEMBRANE, GINGIVA AND MISCELLANEOUS

Q. 1. Write a note on oral mucous membrane in health and diseases. (*TNMGR, April 2013*)

Q. Write a short note on oral mucosa.
(*KLE, June 2007; TNMGR, Oct. 2012*)

Q. Describe microanatomy/histology of oral mucosa/ buccal mucosa. (*HNBG Uni., June 2016; MUHS, June 2017*)

Ans. It is a protective lining of oral cavity consisting partly of epithelium and partly of connective tissue. Anatomically, it begins at the vermilion border of lip and extends up to a point where the pharynx ends.

Oral mucous membrane in health (Fig. 4.1)

Role of oral mucosa

1. It is protective mechanically against both compressive and shearing forces.
2. It provides barrier to various pathogens.
3. It has a role in immunological defense.
4. Minor salivary glands within the mucosa provide lubrication and buffering as well as secretion of some antibodies.
5. Mucosa is richly innervated, providing inputs for touch, properiception, pain and taste.

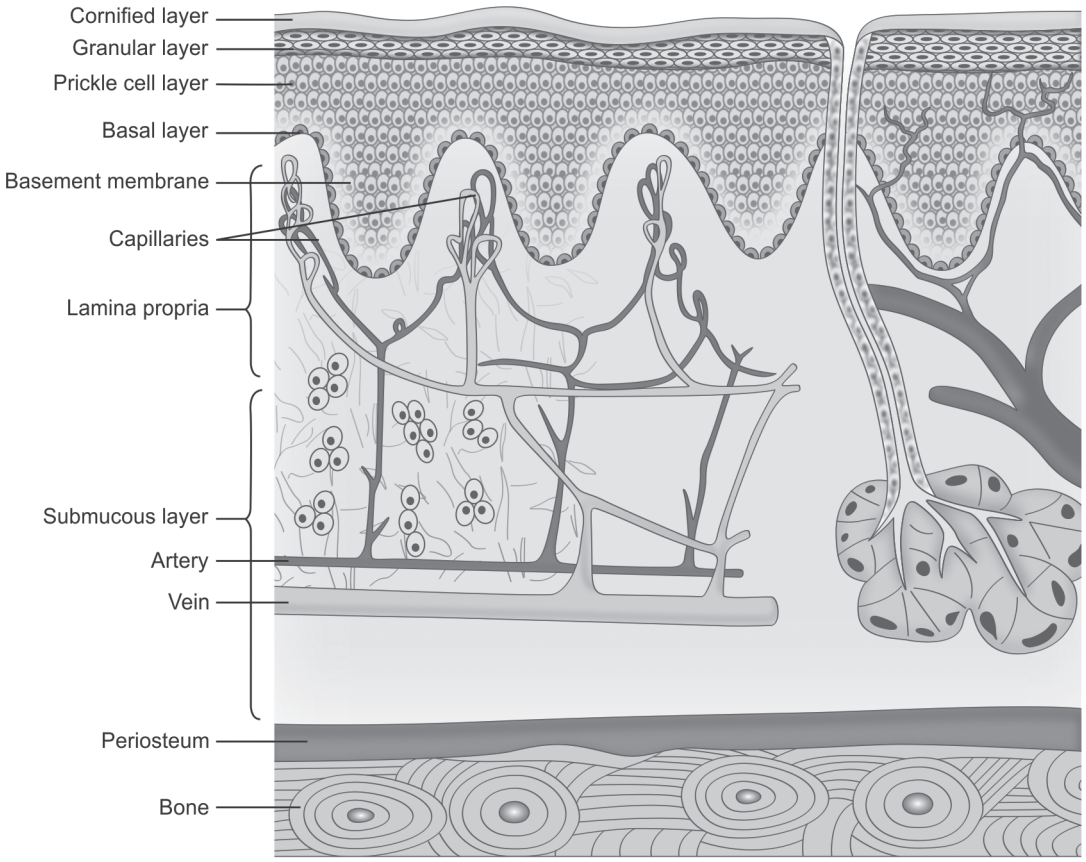

Fig. 4.1: Oral mucous membrane

6. Reflexes such as gagging, salivation are initiated by receptors in the oral mucosa.

Development: Primitive oral cavity develops by fusion of embryonic stomatodaeum with foregut after rupture of buccopharyngeal membrane. Structures from branchial arches like tongue, epiglottis and pharynx covered by epithelium are derived from endoderm. Epithelium covering palate, cheeks and gingiva are of ectodermal in origin.

Oral mucosa can be divided into:

a. **Masticatory mucosa**: Gingiva and hard palate.
b. **Lining or reflecting mucosa**: Lip, cheek, vestibular fornix, alveolar mucosa, floor of mouth, soft palate.
c. **Specialized mucosa**: Dorsum of the tongue, taste buds.

It consists of surface epithelium and underlying connective tissue, lamina propria.

The epithelium: Derived from ectoderm. It can be **keratinized** or **non-keratinized**.

A. Keratinized epithelium consists of four layers:

- Stratum basale: Cells are cuboidal or low columnar, adjacent to basement membrane, most active mitotically.
- Stratum spinosum (prickle cell layer): Spherical or elliptical cells.
- Stratum granulosum: Flat and wide cells, keratohyaline granules.
- Stratum corneum (surface layer): Flat cells devoid of nuclei, filled with keratin
  1. Orthokeratinized epithelium: No nuclei in stratum corneum and presents a well-defined stratum granulosum.
  2. Parakeratinized epithelium: Surface cells have pyknotic nuclei; in this stratum corneum and stratum granulosum are absent.

B. Non-keratinized epithelium: In this stratum corneum and stratum granulosum are absent. It includes lips, buccal mucosa, alveolar mucosa, soft palate, ventral surface of tongue, floor of mouth.

The lamina propria: It has

1. Papillar layer: Large finger-like projections.
2. Reticular layer.

Oral mucous membrane in disease: The basic considerations in oral mucosa are variation in tissue colour, dryness, smoothness or firmness and bleeding tendency of gingiva.

1. Periodontal pocket: It is a pathologically deepened gingival sulcus as a response to plaque toxins and subsequent immunologic response.

2. Restorative dentistry: In young patients, when clinical crown is smaller than anatomic crown, it is difficult to prepare a tooth for an abutment or crown.
3. Gingival recession: May result in cemental/root caries and sensitivity of the exposed dentin.
4. Keratinisation of gingiva: Can be achieved by massage or brushing, thus helping in stimulation and minimizing plaque accumulation.
5. Discoloration of gingiva: Metal poisoning by lead or bismuth causes characteristic discoloration.
6. Blood dyscrasias can be diagnosed by characteristic infiltration of oral mucosa.
7. Viral diseases like measles manifest as typical lesions of oral mucosa.
8. Changes of tongue: In scarlet fever, atrophy of lingual mucosa causes peculiar redness of **Strawberry tongue**. Systemic diseases such as vitamin deficiencies lead to typical changes as **Magenta tongue** and **beefy red tongue**.
9. Macule: A flat spot/stain/discoloration of oral mucosa, e.g. amalgam tattoo, nevus, rash of secondary syphilis.
10. Papule: Small rounded pimple-like variably colored, e.g. White variably patterned elevations of lichen planus.
11. Plaque: Slightly raised clearly demarcated area that may be smooth, pebbly cracked or fissured, e.g. leukoplakia, erythroplakia.
12. Vesicle: Small circumscribed elevated blister not more than 5 mm in diameter with covering layer of epithelial cells and containing an accumulation of fluid, e.g. herpes labialis.
13. Pustule: Vesicle predominantly containing pus.
14. Bulla: Large vesicle or blister, e.g. pemphigus and drug reactions. May appear white due to necrosis of epithelium forming pseudomembrane.
15. Ulcer: Sore characterized by loss of epithelium yielding a punched out area, e.g. traumatic ulcers, aphthous stomatitis, cancer and tuberculosis.
16. Fissure: Narrow linear crack of epidermis with an ulcer at its base, e.g. fissured tongue.
17. Erosion: Partial loss of upper layers of epithelium, e.g. toothbrush trauma, erosive lichen planus.
18. Cyst: Cavity lined by epithelium containing fluid or cells, e.g. gingival cyst.
19. Nodule: Localized elevated mass of tissue projecting from surface, e.g. fibroma, mucocele.
20. Tumour: Swelling of part of an organ. Inflammatory, developmental or neoplastic. Carcinoma is a malignant tumour of epithelial cells.
21. Wheal: Pruritic, reddened, edematous papule.
22. Sinus/sinus tract: Leading from underlying cavity cyst or abscess and opening onto surface.
23. Scar: White depressed mark, line or area representing healing after injury, e.g. gingivectomy, apicoectomy, deep inflammation, previous trauma.

Q. 2. Normal variants of oral mucosa.

(NTR Uni., July 2017)

Ans.

A. **Normal anatomic variants:** Linea alba; Leukoedema; Normal oral pigmentation; Lingual tonsils/Foliate papillae; Lymphoid aggregates; Varicosities; Fordyce's granules; Hairy tongue; Fissure tongue

B. **Developmental anomalies:** Fordyce's granules; Congenital lip pits; Ankyloglossia; Cleft lip; Bifid tongue; Double lip; Torus platinus; Torus mandibularis; Multiple exostosis.

Q. 3. Write a short note on keratinization.

(UHSR, May 2016)

Q. Add a note on the consequence of loss of integrity of epithelium-basement complex.

(NTR Uni., May 2018)

Ans. Keratinization/cornification, is a process of cytodifferentiation in which keratinocytes, when proceeding from their postgerminative state (stratum basale) to finally differentiated, become hardened cell filled with protein, constituting a structurally and functionally distinct keratin containing surface layer such as stratum corneum. Keratins that form the intermediate filaments are expressed exclusively in epithelial cells regardless of germ layer origin of these cells and are useful as markers of differentiation. The keratins are broadly divided:

A. **Primary and secondary:**
   1. Primary keratins: Synthesized by epithelial cells on a regular basis, e.g. K8/18 in simple epithelia, K5/14 in stratified epithelia.
   2. Secondary keratins: Additional keratins produced by epithelial cells, e.g. K7/19 in simple epithelia, K15, and K6/16 in stratified epithelia.

B. **Based on distribution:**
   1. Soft keratin: Allows some stretching but returns to normal upon relaxation of tension.
   2. Hard keratin: Very little flexibility owing to presence of many cysteine disulfide cross links.

C. **Based on X-ray diffraction pattern:**
   1. Alpha
   2. Beta
   3. Feather keratins
   4. Amorphous keratins

D. **Based on amino acid sequence:**
   1. Type I family: Keratins numbered from 9–20, acidic.
   2. Type II family: Keratins numbered 1–8, basic.

E. **Based on molecular weight:**
   1. Low molecular weight keratins: Molecular weight of 40 kDa.
   2. Intermediate molecular weight keratins: Molecular weight 40 kDa–57 kDa.
   3. High molecular weight keratins: Molecular weight of 57 kDa.

Functions of Keratins

1. Keratins influence architecture and mitotic activity of epithelial cells.
2. Keratins and associated filaments provide a scaffold for epithelial cells and tissues to sustain mechanical stress, maintain their structural integrity, ensure mechanical resilience, to protect against variations in hydrostatic pressure and establish cell polarity.
3. Keratins and its filaments are involved in cell signalling, cell transport, cell compartmentalization and cell differentiation.
4. Keratin filaments influence cell metabolic processes by regulating protein synthesis and cell growth.
5. Keratins are involved in transport of membrane bound vesicles in cytoplasm of epithelial cells.

Keratinization Disorders with Predominant/Associated Oral Lesions

1. **White Sponge Nevus/Cannon's disease/familial white folded dysplasia:** Occurs due to a mutation in gene encoding for K4/13 that is expressed in spinous cell layer of non-keratinized mucosa of oral cavity.
2. **Pachyonychia Congenita (PC) (Greek-thick nails from birth):** Mutation in gene encoding for K6/16 and K17 that typically affects nails and palmo-plantar skin and often oral mucosa, tongue, larynx, teeth and hair.
3. **Dyskeratosis congenita (DC)/Cole-Engmen syndrome/Zinsser-Cole-Engmen syndrome:** Mutation in DKC1 gene, thus disrupting normal maintenance of telomerase.
4. **Hereditary benign intraepithelial dyskeratosis/ Witkop-Von Sallman syndrome**
5. **Darier's disease (keratosis follicularis)**
6. **Pemphigus:** Defects in keratin associated protein, desmosomes.
7. **Keratinizing lesions of oral cavity:** These demonstrate hyperkeratosis on histopathology.
   a. Reactive lesions: Frictional keratosis, smokeless tobacco-induced keratosis, nicotine stomatitis, hairy tongue, hairy leukoplakia

Table 4.3: Distribution of major keratins

Keratin distribution in epithelia	
K5/14	Basal cell layer of both the keratinized and non-keratinized stratified epithelium
K1/10	Keratinized epidermis
K6/16	Spinous cell layer of keratinized mucosa
K4/13	Intermediate layer of non-keratinized epithelium
K19	Basal layer of non-keratinized epithelium
K9	Suprabasal cells of palmar and plantar epidermis
Keratin expression in gingival	
K5/14	Basal cell layer
K19	Basal cell layer of junctional epithelium and gingival margin
K8, 18, 13, 16, 19	Superficial layers of junctional epithelium
K4, 13, 16	Superficial layer of gingival margin
K1/10, K6/16 and K2p	Superficial layers of outer gingival epithelium
Distribution of keratin in the dorsal aspect of the tongue (Dale et al 1990)	
Keratin similar to hair	Posterior portion of filiform papilla
K4/13	Interpapillary region
K4/13 and K1/10	The anterior portion

   b. Immune mediated lesions: Lichen planus, discoid lupus erythematosus, graft versus host disease.
   c. Pre-neoplastic and neoplastic diseases: Actinic cheilosis, leukoplakia, proliferative verrucous leukoplakia, verrucous carcinoma, squamous cell carcinoma.
   d. Infections: Squamous cell papilloma, verruca vulgaris, condyloma accuminatum, molluscum contagiosum and verruciform xanthoma.

Q. 4. Write a short note on biosynthesis of collagen *(Sumandeep Uni., April 2014)*

Q. Write a short note on collagen and its degradation. (*TNMGR, March 2009*)

Ans. Collagen (Greek: To produce "Glue") is a major structural protein of extracellular matrix constitutes about 25–30% of protein of mammals. Collagen is fibrous protein that possesses high tensile strength and cannot be stretched. There are at least 12 different types of collagen, each exhibiting certain specific and unique chemical characteristics.

Type I: Skin, bone, scar, tendon, blood vessel, cornea.

Type II: Cartilage, inter-vertebral disc, vitreous body.

Type III: Fetal skin, vessels, granulation tissue.

Type IV: Basement membrane.

Type V: Cell surface, hair, placenta.

Type VII: Beneath stratified squamous epithelia.

Type IX: Cartilage.

Type XII: Tendon, ligaments

Distribution in oral tissues:

1. Alveolar bone: Type I
2. PDL: Type I, III
3. Cementum: Type I
4. Dentin: Type I
5. Pulp: Type I, III
6. Gingiva: Type I, IV
7. TMJ: Type I, II, III

Basic structure: Basic structural unit of collagen is trimer of polypeptides called tropocollagen that forms a triple helix. Tropocollagen is a rod-shaped molecule, 300 nm long and 1.5 nm thick consists of 3 helical polypeptide chains having 1050 amino acids—"α-chains". Peptide bonds are internal-resistant to digestion by proteases. Triple helix is stabilized by hydrogen bonds between the peptide bonds of different chains.

Biosynthesis of collagen: Collagen is one of the proteins that functions outside the cell. Polypeptide precursors of the collagen molecule are formed in fibroblasts, osteoblasts and chondroblasts. These are secreted into extracellular matrix (Fig. 4.4).

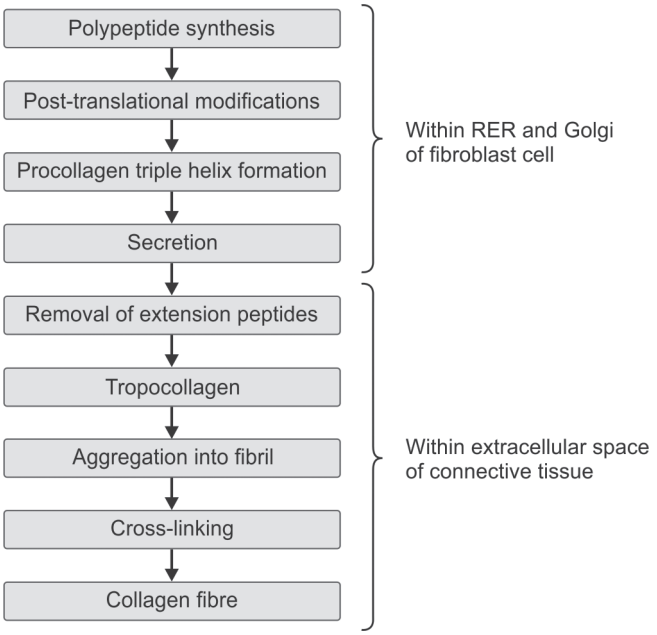

Fig. 4.4: Sequence of collagen synthesis

Degradation: The degradation of collagen is done by enzyme collagenase. Collagenase is secreted as a proenzyme that is activated by specific neutral proteases. Collagenolysis activity takes place outside osteoclast and occurs at a specific site on tropocollagen molecule. The broken fragments of collagen are further decalcified by other proteases.

Conditions associated with defects in collagen synthesis and metabolism:

1. Ehlers-Danlos syndrome
2. Osteogenesis imperfecta
3. Epidermolysis bullosa
4. Dermatosparaxis
5. Alport syndrome
6. Schmid metaphyseal chondrodysplasia
7. Scurvy
8. Menke's disease
9. Lathyrism.

Q. 5. Write a short note on salivary immunoglobulins.

(*TNMGR, March 2010*)

Ans. The predominant salivary immunoglobulin is secretory IgA or sIgA. It differs from serum IgA in that it exists as an 11S dimer consisting of two IgA molecules joined by a J chain, plus a secretory component, whereas serum IgA exists as a 7S monomer. Secretory IgA is a product of two different cell types where plasma cell synthesize polymeric IgA containing J chain of about 1.5 kD and glandular cell synthesize a glycoprotein secretory component of 7 kD. Secretory component is a receptor for polymeric IgA containing J chain; the IgA binds to secretory component below the tight junction of glandular epithelial cells and is then transported across the luminal surface. The presence of secretory component makes IgA resistant to proteolytic enzymes. Purified salivary IgA and IgG fractions have been found with agglutinating activity against oral isolates of α-hemolytic streptococci.

These immunoglobulins are produced locally by plasma cells in connective tissue stroma of the glands. It is the first line of defense of the host against pathogens which invade mucosal surfaces. Salivary IgA antibodies could help oral immunity by preventing microbial adherence, neutralizing enzymes, toxins and viruses; or by acting in synergy with other factors such as lysozyme and lactoferrin. In addition, low levels of salivary IgA have been presented as a risk factor for upper respiratory infection and have also been associated with an increased risk for periodontal disease and caries.

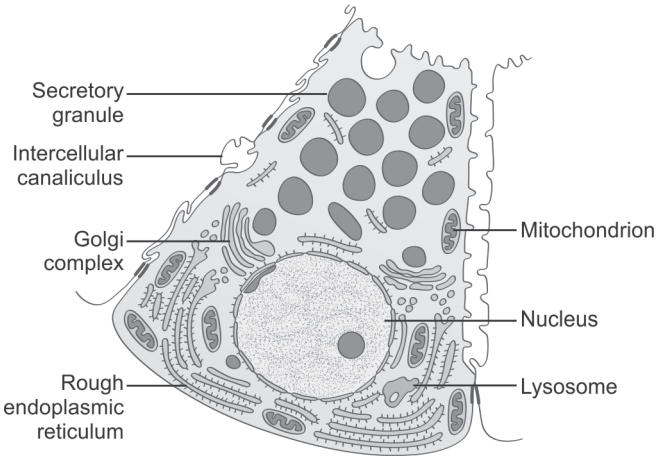

Fig. 4.6: Structure of serous cell

Q. 6. Write about ultrastructure of serous cell. *(TNMGR, April 2011)*

Ans. Serous cells are specialized for the synthesis, storage and secretion of proteins. The typical serous cell is pyramidal in shape, with its broad base resting on thin basal lamina and its narrow apex bordering on the lumen. The spherical nucleus is located in the basal region of the cell; occasionally binucleated cells are observed. There is accumulation of secretory granules in the apical cytoplasm. The basal portion of the cytoplasm is filled with ribosome studded endoplasmic reticulum. Golgi apparatus is located apical or lateral to nucleus. Mitochondria are found throughout the cell. (Fig. 4.6).

Q. 7. Discuss microscopic and macroscopic appearance of gingiva. *(AHSUC, July 2016)*

Q. Development of gingival sulcus. *(NTR Uni., May 2019)*

Ans. Gingiva is that part of oral mucosa that covers alveolar processes of jaws and surrounds neck of teeth (Carranza).

Functions

A. As part of oral mucosa: It protects supporting tissues from oral environment.
B. As part of peridontium: Its fibres secure against rotational forces; maintain periodontal health by its defense mechanism.

Gingival sulcus: It is shallow crevice around tooth, bounded by surface of tooth on one side and epithelial lining of marginal gingiva on other side. Probing depth is 2–3 mm.

Development of gingival sulcus: After enamel formation completes, the crown is covered with reduced enamel epithelium (REE), which terminates at CEJ. The basal lamina lies in contact with enamel directly. When tooth penetrates oral mucosa, REE fuses with oral epithelium. Shortly the epithelial mass at tip of crown degenerates, resulting exposure of crown in oral cavity. With tooth eruption, this united epithelium condenses along crown and ameloblasts from inner layer of REE become squamous cell with time. The gingival sulcus is formed when tooth erupts into oral cavity. Gradually this united epithelium transforms into junctional epithelium (JE), which occurs in apical direction.

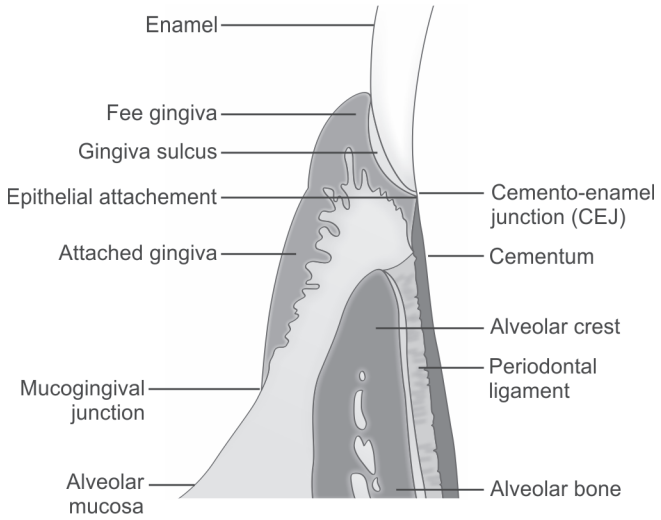

Fig. 4.7: Gingiva and its parts

Macroscopic/anatomical structure of gingiva:

1. **Marginal/unattached gingiva:** Terminal edge of gingiva (Fig. 4.7) surrounding the teeth in collar like fashion. *Free gingival groove* is formed by functional folding of free gingival margin during mastication.
2. **Attached gingiva:** It is continuous with marginal gingiva, is firm resilient and tightly bound to underlying periosteum of bone. On facial aspect, it is demarcated from alveolar mucosa by *Mucogingival junction.*
3. **Interdental gingiva:** Pyramidal or "**Col**" shaped, occupies interproximal space below area of tooth contact.

Microscopic structure of gingiva: Stratified squamous epithelium with core of connective tissue made up of collagen fibers and ground substance.

A. **Gingival epithelium:** *(UOK, May 2015)*
   1. **Oral epithelium:** Faces oral cavity; Covers crest and outer surface of marginal gingiva and surface of attached gingiva. On average, oral epithelium is 0.2–0.3 mm in thickness. It is keratinized or parakeratinized or presents various combination.
   2. **Sulcular epithelium (SE):** Faces the tooth, lines the gingival sulcus. It is a thin, non-keratinized

stratified squamous epithelium without rete pegs. It extends from coronal limit of JE to crest of gingival margin. It shows many cells with hydropic degeneration. The SE acts as a semi-permeable membrane, not heavily infiltrated by PMNs.

3. **Junctional epithelium (JE):** Provides contact between gingiva and tooth, consists of a collar-like band of stratified squamous non-keratinizing epithelium. The thickness of JE increases with age. JE tapers from its coronal end to its apical termination.

   JE is formed by confluence of oral epithelium and REE during tooth eruption. JE is attached to tooth surface (epithelial attachment) by means of an internal basal lamina. It is attached to gingival connective tissue by an external basal lamina.

   Attachment of JE to tooth is reinforced by gingival fibers, which brace marginal gingiva against tooth surface. For this reason, JE and gingival fibers are considered functional units referred to as ***dentogingival unit***.

   Functions: JE firmly attached to tooth surface forming an epithelial barrier against plaque, bacteria; allows access of gingival fluid inflammatory cells, and components of immunologic host defense to gingival margin; JE cells exhibit rapid turnover, contributes to host–parasite equilibrium and rapid repair of damaged tissue.

Cells of gingival epithelium:

1. **Principal cells:** Keratinocytes
2. **Clear cells/non-keratinocytes:** Langerhans cells, Markel's cells, melanocytes, inflammatory cells.
3. **Melanocytes:** These are dendritic cell located in basal and spinous layer of gingival epithelium. They synthesize melanin in organelles: Premelanosomes or melanosomes which contain *tyrosinase* which hydroxylates tyrosine to dihydroxyphenylalanine (DOPA), which in turn is progressively converted to melanin. Melanin granules are phagocytosed and contained within other cells of epithelium and connective tissue called melanophages or melanophores.
4. **Langerhans cells:** These are dendritic cells located among keratinocytes at all supra-basal levels. They belong to mononuclear phagocytes system (reticuloendothelial system) as modified monocytes, derived from bone marrow. They contain elongated granules and are considered macrophages with possible antigenic properties. They have an important role in immune reaction as antigen-presenting cells for lymphocytes. They contain ***Birbeck's granules*** and have marked adenosine triphosphate activity. They found in oral epithelium of normal gingiva and in smaller amounts in sulcular epithelium; they are probably absent from junctional epithelium of normal gingiva.
5. **Merkel cells:** They are located in deeper layer of epithelium, harbors nerve endings, act as tactile receptors and connected to adjacent cells by desmosomes.

B. **Connective tissue of gingiva** *(UOK, May 2015):* Connective tissue of gingiva is known as **lamina propria**. It has: a. Papillary layer; b. Reticular layer.

Ground substance: Proteoglycans (hyaluronic acid, chondroitin sulfate); glycoproteins (fibronectin, laminin).

Fibres: Collagen type I and IV; reticular; elastic (oxytalan, elaunin and elastin fibers).

Cells: Fibroblast, mast cells, macrophages and eosinophils, plasma cells, lymphocytes and neutrophils.

Blood supply: Supraperiosteal arterioles, vessels of periodontal ligaments, arterioles from crest of interdental septa.

Lymphatics: a. Mandibular incisor gingiva → Submental node; b. Maxillary palatal gingiva → Deep cervical nodes; c. Buccal gingiva of maxilla and buccal and lingual gingiva in mandibular premolar-molar → Submandibular lymph nodes.

Nerve supply: Branches of trigeminal nerve in PDL as: Meshwork of terminal argyrophilic fibers; Meissener tactile corpuscles; Krause end bulbs and encapsulated spidles.

Q. 8. Write a short note on dentogingival unit.
(UHSR, May 2012; HP Uni., April 2019)

Q. Write a short note on gingival fibers.
(MUHS, Dec. 2018; UHSR, May 2019)

Ans. Dentogingival unit comprises junctional epithelium (epithelial attachment), connective tissue and gingival fibres. A term frequently used to describe the dimensions of soft tissues that face the teeth is biologic width of soft tissue attachment. Biologic width of attachment varied between 2.5 mm in normal case and 1.8 mm in advanced disease case. The connective tissue of marginal gingiva is densely collagenous, containing a prominent system of collagen fibres bundle called, gingival fibers, consists of type I collagen (Fig. 4.8).

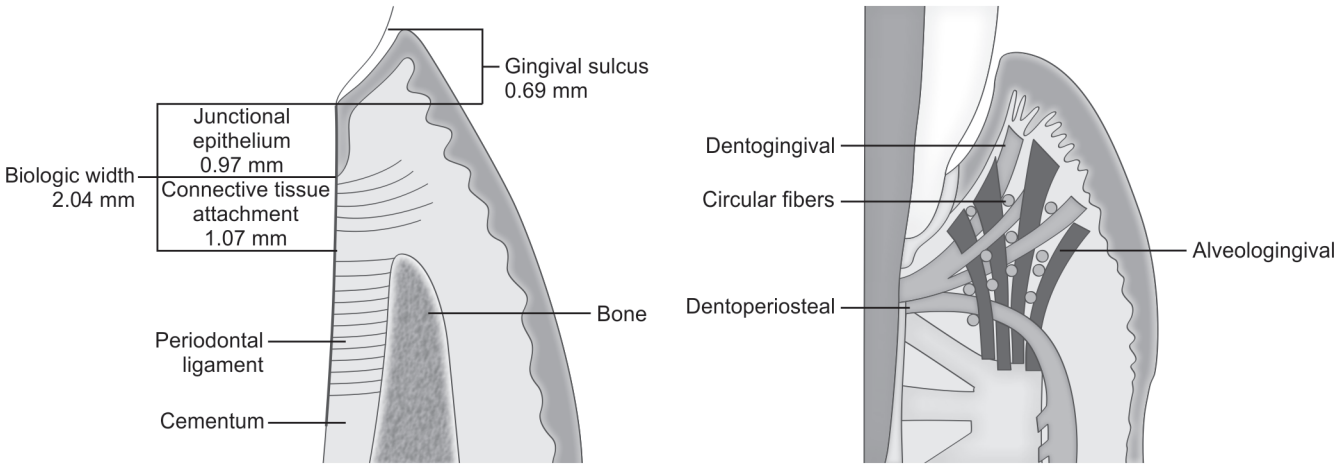

Fig. 4.8: Dentogingival unit

Functions

a. To brace the marginal gingiva firmly against tooth.
b. To provide rigidity.
c. To unite free marginal gingiva with cementum and adjacent gingiva.

Gingival Fibers:

A. **Principal:**
   1. Circular fibers (CF): Run their course in the free gingiva; encircle the tooth in ring-like fashion.
   2. Dentogingival fibers (DGF): Embedded in cementum of supra alveolar portion of root, project from cementum in fan-like configuration out into free gingival tissue.
   3. Dento-periosteal fibers (DPF): Embedded in cementum of supra alveolar portion of root, but run apically over vestibular and lingual bone crest and terminate in attached gingiva.
   4. Transseptal fibers (TF): Extends between supra alveolar cementum of adjacent teeth.

B. **Secondary:** Periosteogingival, transgingival, intercircular, intergingival, semicircular

Q. 9. Write a short note on gingival crevicular fluid.

(TNMGR, Oct. 2011; UOK, July 2013; RUHS, May 2015; AHSUC, May 2017)

Ans. Gingival crevicular fluid (GCF) is an inflammatory exudate that can be collected at the gingival margin or within the gingival crevice. Gingival crevicular fluid (GCF) can be found in the physiologic space (gingival sulcus), as well as in the pathological space (gingival pocket or periodontal pocket) between the gums and teeth. In the first case it is a transudate, in the second it is an exudate. The constituents of GCF originate from serum, gingival tissues, and from both bacterial and host response cells present in the aforementioned spaces and the surrounding tissues. The collection and analysis of GCF are the noninvasive methods for the evaluation of host response in periodontal disease. These analyses mainly focus on inflammatory markers, such as prostaglandin E2, neutrophil elastase and beta-glucuronidase, and on the marker of cellular necrosis—aspartate aminotransferase. Further, the analysis of inflammatory markers in the GCF may assist in defining how certain systemic diseases (e.g. diabetes mellitus) can modify periodontal disease, and how periodontal disease can influence certain systemic disorders (atherosclerosis, preterm delivery, diabetes mellitus and some chronic respiratory diseases). The gingival fluid is believed to: Cleanse material from the sulcus. Contain plasma proteins that may improve adhesion of the epithelium to the tooth. Possess antimicrobial properties. Exert antibody activity to defend the gingiva.

Clinical Significance

1. Its presence in clinically normal sulcus signifies inflammation.
2. Amount of GCF is proportional to severity of inflammation.
3. GCF follows circadian rhythm more from 6 am to 10 pm.
4. Female sex hormone increases GCF.
5. Drug excreted through GDF like tetracycline, metronidazole may help in local therapy.
6. GCF production is more during healing period after periodontal surgery.
7. Mechanical stimuli increase GCF production.
8. Smoking produces marked but transient increase in GCF.

Q. 10. Write a short note on cell adhesion molecules.
(Sumandeep Uni., April 2015)

Ans. Cell adhesion molecules (CAMs) are glycoproteins located on the cell surface. They are typically transmembrane receptors and are composed of three domains: Intracellular domain with cytoskeleton, transmembrane domain with CAMs, extracellular domain with extracellular matrix. CAMs help cells to stick to each other and to their surroundings. Cell adhesion receptors enable cells to recognize and bind molecules on other cells or in the extracellular matrix. Cell adhesion receptors can form: Homophilic (or homotypic) adhesions or heterophilic (or heterotypic) adhesions.

Types:

1. Cadherins: Calcium-dependent molecules, cell adhesion by forming desmosomes. Subclass: a. Neural, b. Placental, c. Epithelial.
2. Ig super family CAMs: Calcium-independent transmembrane glycoproteins. Subclass: ICAM, VCAM-1 PECAM-1, NCAM.
3. Selectins: Divalent cation dependent glycoproteins. Subclass: a. Endothelial, b. Leukocyte, c. Platelet.
4. Integrins: Large group of heterodimeric glycoproteins, e.g. α and β.
5. Mucins: Group of serine and threonine-rich protein and hydroxyproline enabling post-translational O-glycosylation.

Functions of CAMs:

1. Involvement in inflammation.
2. Tumorigenesis.
3. Establishment of the blood–brain barrier.
4. Involvement in lymphocyte homing.
5. In regulation of apoptosis.

Malfunctioning of CAMs leads to breast cancer, leukocyte adhesion deficiency (LAD) syndrome, and epithelial cell cancer.

Q. 11. Write a note on proteoglycans.
(TNMGR, May 2018)

Ans. "Proteoglycans" (Balaza, 1967): A family of macromolecules composed of one or more Glycosaminoglycans (GAGs) covalently bound to a protein core.

Structure of proteoglycans: GAGs + Core Protein

A. **Glycosaminoglycans:** Principal carbohydrate component of proteoglycans, extend perpendicularly from the core in a brush-like structure. GAGs are composed of repeating disaccharide units of: Uronic acid (D-Glucuronic acid or L-Iduronic acid) and Hexosamine (D-Glucosamine or D-Galactosamine). GAGs have been classified mainly into:
   1. Sulfated glycosaminoglycans: Chondroitin sulfate, dermatan sulfate, heparin and heparin sulfate; keratan Sulfate.
   2. Non-Sulfated Glycosoaminoglycans: Hyaluronan
B. **Linkage sugars:** Oligosaccharides.
C. **Core Proteins:** Proteoglycan core proteins range in size from 10–300 KD with one to several hundred attached glycosaminoglycan chains.

Proteoglycan synthesis: Protein core is synthesized in RER with hydrophobic N-terminal leader sequence that is removed during translation of RNA. Addition of GAG occurs after the specific glycopeptide linkage has been formed and later modifications to GAG chains eventually lead to completion of synthetic process. Initiation of GAG chain elongation occurs through sequential addition of sugars that are transported to Golgi and are formed in the cytosol of cell.

Types of Proteoglycans

A. **Based on their GAG composition**
   i. Small dermatan sulfate proteoglycans
   ii. Large aggregating chondroitin sulfate proteoglycans.

B. **Based on location**
   i. Matrix Organizers: 1. Aggrecan, 2. Versican, 3. Perlecan
   ii. Tissue space fillers: Leucine Rice—interstitial proteoglycans (Decorin, Biglycan, Fibromodulin, Lumican)
   iii. Cell surface proteoglycans (or) intracellular proteoglycans of hematopoietic cells: Syndecans, Cd44, Glypicans, Betaglycan, Serglycin

Functions

1. Extracellular matrix proteoglycans and tissue-organizing proteoglycans are principally associated with conferring physicochemical properties to tissues.
2. Large proteoglycans serve to maintain tissue hydration.
3. Smaller extracellular matrix proteoglycans serve important functions in binding to other matrix molecules.
4. Cell surface proteoglycans provide the necessary means for cells to attach to their matrix.
5. Intracellular proteoglycans of hematopoietic cells, important in enzyme packaging in order to prevent non-specific enzymatic activity (or) autolysis of cell.
6. Cell surface proteoglycans and matrix proteoglycan have ability to bind and regulate growth factor activity.

Proteoglycans of Periodontium

1. Gingiva: Chondroitin-4-sulfate + dermatan sulfate (predominant in gingival connective tissue) + hyaluronic acid + heparan sulfate (60% in epithelium)
2. Inflamed gingiva: The gingival proteoglycans in inflammation leading to decrease in dermatan sulfate and increase in chondroitin sulfate, and degradation of their proteoglycan core proteins and hyaluronic acid.
3. Hyperplastic gingiva: There is increased synthesis of proteoglycans.
4. Periodontal ligament: Dermatan sulfate and chondroitin sulfate (mainly). As development progresses, concentration of hyaluronic Acid in PDL decreases and levels of dermatan sulfate and chondroitin sulfate proteoglycans increases.
5. Cementum: Hyaluronan, dermantan sulfate, chondroitin sulfate and keratin sulfate, exclusively in precementum and precementocyte lacunae.
6. Bone: Chondroitin sulfate-4 (major), hyaluronic acid, chondroitin sulfate-6, dermatan sulfate, keratan sulfate, biglycan, decorin (minor)
7. Epithelial attachment: Heparan sulphate (major)
8. Sulcular fluid: Non-inflamed sites: Hyaluronic acid only. Inflammatory sites: Hyaluronic acid, dermatan sulfate, chondroitin sulfate.

Q. 12. Write a short note on osseointegration. *(NTR Uni., May 2018)*

Ans. The concept of osseointegration was developed and term was coined by Dr PerIngvar Branemark. It is defined as "Direct structural and functional connection between ordered, living bone and surface of a load carrying implant".

Mechanism: The mechanism involves three phases:

1. Osteoconduction: Migration of osteogenic cell by chemotaxis to surgical sit is known as osteoconduction.
2. New bone formation (osteogenesis):
   a. Stage 1: Osteogenic cells secrets organic matrix.
   b. Stage 2: Organic matrix provides nucleation site for calcium phosphate mineralization.
   c. Stage 3: Growth of calcium phosphate crystal and initiation of collagen fibers assemble.
   d. Stage 4: Calcification of individual collagen fibrils.
3. Bone remodeling: Osteoclastic resorption followed by lamellar bone deposition to maintain health skeletal mass.

Q. 13. Discuss self-protective features of human dentition. *(NTR Uni., May 2018)*

Ans. Protective functional form of teeth includes:

1. **Proximal contact areas:** All teeth contact adjacent teeth at a proximal contact area. Types of proximal contact: Point contact and contact area. Because of contact areas, food will not be packed between teeth causing inflammation to supporting tissues, thereby causing gingivitis and periodontitis. It helps to stabilize dental arches by combined anchorage of all teeth in arch in positive contact with each other.
2. **Interproximal areas (formed by proximal surface in contact):** These triangular shape areas are normally filled with gingival tissues and avoid any food impaction.
3. **Embrasures (spillways):** The curvature formed by two adjacent teeth in an arch from a spillway space.
4. **Height of contours:** Labial and buccal contours at cervical 3rd, and lingual contour at middle 3rd.
5. **Curvature of cementoenamel junction:** Curvature of cervical lines on mesial and distal surfaces.

Q. 14. Write a short note on CPITN. *(UOK, May 2015)*

Ans. Community Periodontal Index of Treatment Needs (CPITN) was developed for joint working family of WHO and FDI by Jukka Ainamo, David Barmes, George Beagrie, Terry Cutress, Jean Martin and Jeniffer Sardo-Infirri in 1982.

Scope and purpose: CPITN procedure is recommended for epidemiological surveys of periodontal health. It provides guidance on planning and monitoring of effectiveness of periodontal care programme and dental personnel required. CPITN records the common treatable conditions, namely periodontal pockets, gingival inflammation, dental calculus and other plaque retentive factors. It does not record irreversible changes such as recession, tooth mobility or loss of periodontal attachment.

Advantages: Simplicity; speed and international uniformity.

Limitations: Partial recording, Exclusion of some important signs of past periodontal breakdown, absence of any marker of disease activity or susceptibility.

Procedure for CPITN

- The dentition is divided into six parts called *sextants*.
- Each sextant is given a score.

- For epidemiological purposes, score is identified by examination of specified index teeth.
- For clinical practice, the highest score in each sextant is identified after examining all teeth.
- Essentially CPITN considers periodontal treatment needs of each sextant with respect to:

i. No need for care (score 0)
ii. Bleeding gingivae on gentle probing (score 1)
iii. Presence of calculus and other plaque retentive factors (score 2)
iv. Presence of 4–5 mm pockets (score 3)
v. Presence of 6 mm or deeper pockets (score 4)