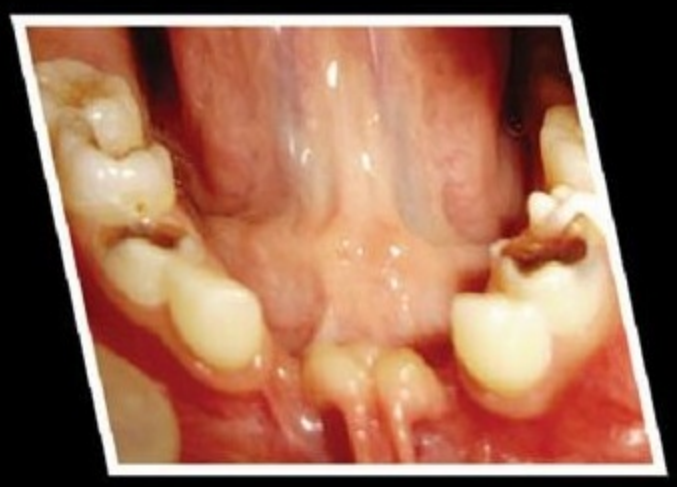
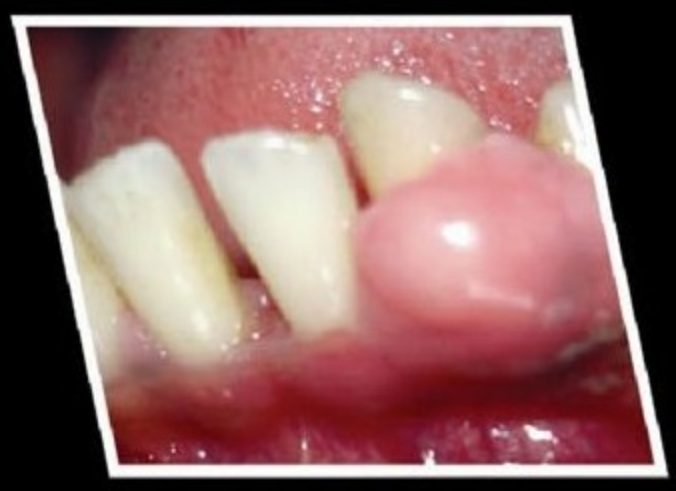
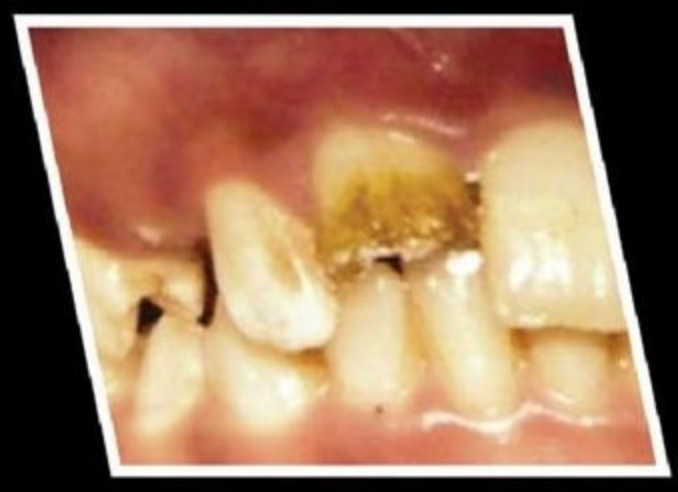
Dental Diseases

Differential Diagnosis







Priya Verma Gupta

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Differential Diagnosis of Dental Diseases

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Preface

Two decades back dental surgery was a growing branch but now it has grown up well. Previously dental surgeons used to prefer extraction of tooth but now they are being paid to save the tooth. In order to achieve they should be able to assess, diagnose the disease and treat accordingly. To differentiate two similar dental diseases one should know the pros and cons of the specific disease which will help the students and clinicians.

I would like to thank my mentors Drs (Profs) N Sridhar Shetty and Amita Hegde for the knowledge given to me by them.

There is always some scope to improve upon and for that healthier suggestions are always welcome.

I am thankful to Shri Jitendar P Vij, Chairman and Managing Director, Jaypee Brothers Medical Publishers, for giving me the opportunity to write this book.

Priya Verma Gupta

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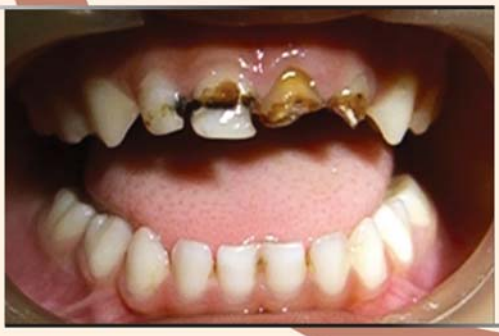


CHAPTER 1

Morphology of Primary Dentition

INTRODUCTION

Primary teeth are often called deciduous teeth. The word "deciduous" comes from a Latin word "decidere" – meaning, "to fall off". Deciduous teeth fall off or are shed like leaves from a deciduous tree. These teeth are shed and then replaced by permanent successors. This process of shedding the deciduous teeth and replacement by the permanent teeth is called exfoliation. Exfoliation begins 2 or 3 years after the deciduous root is completely formed. At this time the root begins to resorb at its apical end and resorption continues in the direction of the crown until the entire root is resorbed and the tooth finally exfoliates.

Importance of Primary Dentition

1. The loss of primary teeth tends to disturb the eruption sequence of permanent teeth.
2. The primary teeth are used for performing mastication of food, digestion and assimilation during one of his most active periods of growth and development.
3. Primary dentition is very important for the maintenance of proper diet.
4. Maintenance of adequate spacing and arch continuity

for the emergence of permanent teeth is one of the most important functions of primary teeth.
5. Flared roots of the primary molars resist the mesial displacement of the coronal portion of the tooth and helps in preserving sufficient space for the premolars and permanent canines.
6. The primary teeth also performs a function that stimulates the growth of the jaws through mastication, especially in the development of the height of the dental arches.
7. Another important function of the primary teeth is the development of speech. Early and accidental loss of the primary anterior teeth may lead to difficulty in pronouncing the sounds 'f', 'v', 's', 'z', and 'th' thus requiring speech correction.
8. Primary teeth also serve a cosmetic function by improving the appearance of the child.
9. Maintains a normal facial appearance.
10. Resorption helps in guiding the erupting permanent tooth into the proper location.
11. Prevents the migration of adjacent teeth thus maintaining the integrity of arch.

MORPHOLOGICAL DIFFERENCES BETWEEN PRIMARY AND PERMANENT DENTITION (FIGS 1.1 AND 1.2)

The Crown

1. The primary tooth has a shorter crown than the permanent tooth.

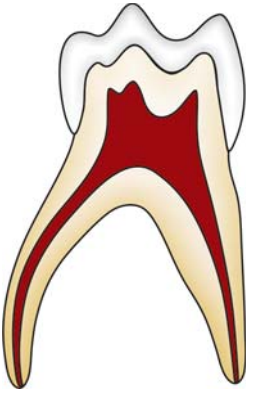


Fig. 1.1: Primary tooth

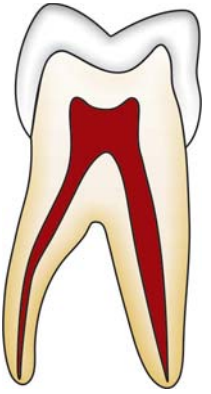


Fig. 1.2: Permanent tooth

2. The enamel and dentin layers are thinner in the primary tooth.
3. The occlusal table of a primary tooth is relatively narrower than the permanent tooth.
4. The primary tooth is much more constricted in the cervical portion of the crown.
5. The enamel rods in the gingival third extend in a slightly occlusal direction from the DEJ (Dentino-enamel-junction) in primary teeth whereas they extend slightly apically in the permanent dentition.
6. The contact areas are very broad and flat.
7. The color of the primary teeth is usually whiter than the permanent teeth.
8. The crowns of the primary anterior teeth are wider mesiodistally than the cervicoinsical length of the permanent teeth.

9. The buccal and lingual surfaces of the primary molars are flatter, thus providing a broader contact with the adjacent tooth.
10. The buccal and lingual surfaces of the molars, especially the first molar converge towards the occlusal surface.
11. The buccolingual diameter of the occlusal surface is much less than the cervical diameter.
12. The cervical ridge of enamel in the anterior crown labially and lingually is much more prominent in primary dentition.
13. The cervical prominence gives primary crown a bulbous appearance and accentuates the narrow cervical portion of deciduous roots.
14. There is less tooth structure protecting the pulp in primary teeth.
15. Usually there are no depressions on the labial surface of the crowns of the incisors i.e. Mamelons are absent.
16. The cingulum of anterior teeth is prominent.
17. The cusps are short, the ridges are not pronounced and the fossae are correspondingly shallow.
18. The buccal cusps on molars are not sharp, with their cusp slopes meeting at an obtuse angle.
19. The second primary molars are larger than the first molars.
20. In totality the crowns of primary teeth are seen short when compared with the permanent teeth.

THE PULP

1. The pulp of the primary tooth is larger in relation to the crown size that of the permanent tooth.
2. The pulp horns of the primary tooth are closer to the outer surface of the tooth.
3. The mesial pulp horn appears to be in a closer approximation of the surface than does the distal pulp horn of the primary tooth.
4. The mandibular molar has larger pulp chambers than the maxillary molar in the primary tooth.
5. The form of the pulp chamber follows the surface of the crown.
6. Usually there is a pulp horn under each cusp.

THE ROOT

1. The root of the primary anterior tooth is narrower mesiodistally.
2. The roots of the posterior primary tooth are longer and more slender.
3. The roots of the primary molar flare more as they approach the apex.
4. The roots of the anterior teeth bend labially in their apical one third by as much as 10°.
5. The second molar roots are spread more widely than the first deciduous molar.
6. There is absence of a root base in the primary molars.
7. The roots erupt directly from the crown and there is no root trunk.
8. The position of the apical foramen is variable due to resorption.

It has been thought that the primary teeth are capable of a greater inflammatory response to insult because of the greater blood supply. They are also considered to be less sensitive to pain because of incomplete development of the neural network.

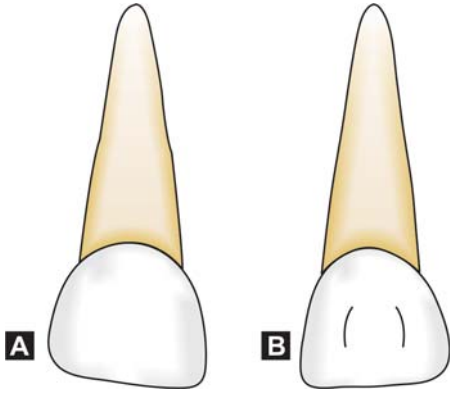
MORPHOLOGY OF INDIVIDUAL TEETH

Maxillary Central Incisor (Figs 1.3A and B)

- Number of pulp horns – 3
- Number of roots – 1
- Number of developmental lobe – 1

Labial Aspect

1. Mesiodistal diameter is greater than its cervicoinsical length.



Figs 1.3A and B: (A) Labial aspect, (B) Lingual aspect

2. Mamelons are absent on the deciduous teeth.
3. The labial surface is unmarked by grooves, depressions, or lobes.

Lingual Aspect

1. Well developed marginal ridges.
2. Highly developed cingulum.
3. The depression between the marginal ridges and the cingulum forms the lingual fossa.
4. The cingulum is convex and occupies the cervical 1/2 to 1/3 of the surface.

Mesial and Distal Aspects

1. The crown appears wide in relation to its total length.
2. The labiolingual measurements make the crown appear thick.
3. The curvature of cervical line, is distinct, curving toward the incisal ridge.

Incisal Edge

1. The incisal edge is centered over the main bulk and is relatively straight.
2. The incisal edge is proportionately long.
3. The mesial surface joins the incisal edge at an acute angle and the distal surface at a more rounded, obtuse angle.
4. The incisal edge is formed from one developmental lobe.

Root

1. The roots are S-shaped, bending lingually in the cervical third to half and labially by as much as 10° in the apical half.
2. The root is much longer relative to the crown length with tapered end.

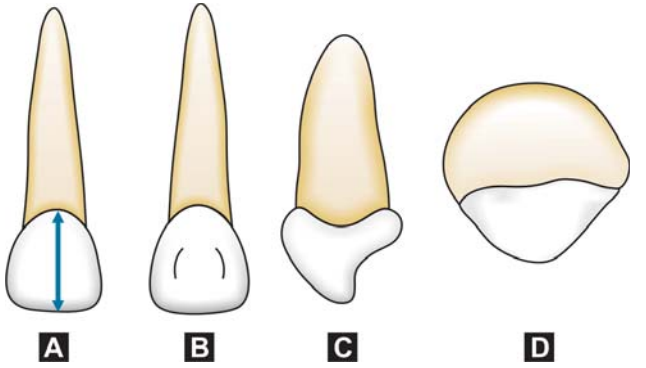
Pulp Cavity

1. The pulp cavity conforms to the general outside surface of the tooth.
2. The chamber tapers cervically in its mesiodistal diameter.
3. It is widest at the cervical ridge labiolingually.
4. Both pulp chamber and canal are large when compared to permanent tooth.
5. The pulp canal tapers evenly until it ends in the apical foramen.

MAXILLARY LATERAL INCISOR (FIGS 1.4A TO D)

- Number of pulp horns - 3
- Number of root - 1
- Number of developmental lobe-1

1. A lateral incisor's crown is smaller than a central incisor's crown in all dimensions.
2. Only the cervicoincisal length is greater than its mesiodistal width.
3. Distoincisal angles of lateral incisors are more rounded.
4. The labial surface when viewed from the incisal aspect is more convex.



Figs 1.4A to D: (A) Labial aspect, (B) Lingual aspect, (C) Mesial aspect, (D) Incisal aspect

5. The lingual fossa is deeper as compared to central incisor.
6. The marginal ridges are more pronounced as compared to central incisor.
7. The outline of the crown from the incisal aspect is almost circular.
8. The pulp chamber follows the contor of the tooth, so does the canal.
9. There is a slight demarcation between the pulp chamber and the canal.

Root

1. The root appears much longer in proportion to the crown.
2. The root appears constricted at its cervical third.
3. There is a mesial concavity on the root surface.
4. The distal surface is generally convex.

MAXILLARY DECIDUOUS CANINE (FIGS 1.5A TO D)

- Number of pulp horns-3.
- Number of root-1.
- Number of cusp-1.
- Number of developmental lobes-4.

Labial Aspect

1. It is bulkier than the primary incisors in every aspect.
2. The crown is more constricted at the cervix.
3. More convex on its mesial and distal surfaces.
4. The root is more slender.
5. The canine is greater in mesiodistal diameter.
6. The crown has a diamond shaped appearance.
7. It has a long, well developed, sharp cusp.

Fig. 1.5A: Labial aspect

Fig. 1.5B: Lingual aspect

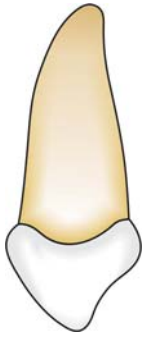


Fig. 1.5C: Mesial aspect

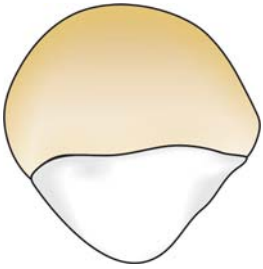


Fig. 1.5D: Incisal aspect

8. Maxillary canine cusps are often very sharp with two cusp ridges.
9. The mesial slopes of the canines are longer than the distal cusp slopes.
10. These mesial cusp slopes are flat to concave and less steeply inclined than the shorter distal slopes which are more convex.

Contact Areas

1. Distal contact areas of primary canines rest against the mesial surfaces of primary molars.
2. Primary maxillary canines have mesial proximal contacts more cervical than the distal proximal contacts.

Lingual Aspect

1. A tubercle extends from the cusp tip to the lingual ridge.
2. The lingual ridge extends from the cusp tip to the cingulum.
3. It divides the lingual surface into mesiolingual and distolingual fossae.

4. The cingulum on a maxillary canine crown is bulky with well-developed cusp.
5. The lingual surface of the crown may present a slight concavity called the lingual fossa.
6. Primary canine has a 'fang-like' appearance.

Mesial and Distal Surfaces

1. Canine is much wider at the cervical third of the crown.
2. The crown and the root at the cervical third are wider labiolingually.
3. There is increased length of the mesioincisal edge.
4. The labial and the lingual surfaces converge as they approach the cervical area.
5. The increase in crown dimension, in conjunction with the root width and length permits resistance against forces that the tooth must withstand during function.
6. The function of this tooth is to punch, tear and apprehend food material.

Incisal Aspect

1. The crown is rhomboidal-like a square that has been slightly shifted.
2. The labial ridge is relatively pronounced.
3. The cingulum is obvious.
4. The tip of the cusp is slightly distal to the center of the tooth.
5. The mesial cusp slope is longer than the distal cusp slope.

Root

1. The root is long, thick in diameter and slightly flattened on proximal surfaces.
2. The root is bulky in the cervical and middle third.
3. Tapering occurs mostly in the apical third region where the root tip is bent labially.

Pulp Cavity

1. The pulp cavity conforms to the contor of the tooth.
2. The central pulpal horn is projecting incisally farther than the remainder of the pulp chamber.
3. Distal horn is larger than the mesial projection.
4. Very little demarcation between the pulp chamber and the canal can be seen.

MAXILLARY FIRST MOLAR (FIGS 1.6A TO D)

- Number of roots-3
- Number of pulp horns-3
- Number of cusps-3
- Number of developmental lobes-4

Labial Aspect

1. The deciduous maxillary first molar is a blend of premolar and molar.
2. It is wider buccoligually than mesiodistally.
3. It has two major cusps – a mesiobuccal and a mesiolingual.

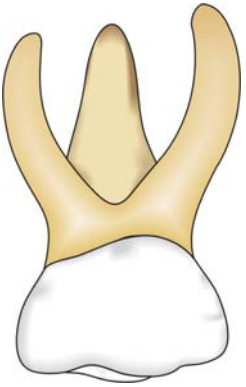


Fig. 1.6A: Labial aspect

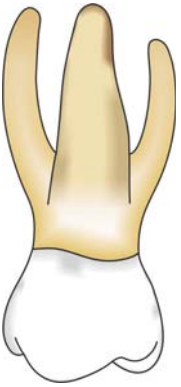


Fig. 1.6B: Lingual aspect

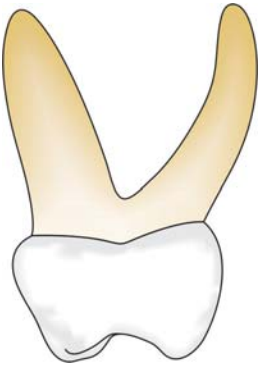


Fig. 1.6C: Mesial aspect

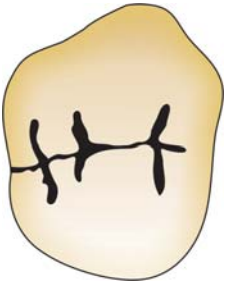


Fig. 1.6D: Occlusal aspect

4. There is a distobuccal cusp which is smallest of the entire cusp.
5. The buccal surface is convex in all directions.
6. The buccal surface is divided by the buccal groove.
7. There is a well developed buccal ridge present on mesiobuccal cusp.
8. A less developed ridge is present on the distobuccal cusp.
9. It is much smaller in all measurements than the second molar.
10. Its relative shape and size suggest that it was designed to be a "premolar section" of the primary dentition.
11. In function it acts as a compromise between the size and shape of the anterior primary teeth and the molar area.
12. The crown appears squat since the mesiodistal diameter is considerably greater than the crown height.
13. The mesial moiety of the crown has a greater height in consequence of its more cervical projection onto the root area.
14. The lingual root is positioned exactly midway between the two buccal roots.
15. The cervical third of the buccal margin bulges and is called the buccal cervical ridge.

Lingual Aspect

1. The crown of a first molar converges toward the lingual surface.

2. The mesiobuccal cusp is always the longest but second sharpest.
3. The mesiolingual cusp is the sharpest but second longest.
4. The distolingual cusp is small and rounded, if present.
5. A tiny tubercle can sometimes be seen on the mesiolingual cusp.
6. The shorter diameter of the lingual cusp, leads to a narrower lingual diameter.

Mesial Surface

The mesial surface is greater in diameter at the cervical border than at the occlusal. This is true for all molar forms, but it is more pronounced on primary teeth than on permanent teeth. The mesiobuccal and lingual roots are visible only from the mesial aspect. The distobuccal root is hidden behind the mesiobuccal root. The mesial marginal ridge groove is sharp and deep in profile and continues in the form of a shallow, narrow depression up the crown surface toward the cervical line.

DISTAL ASPECT

1. The crown appears to be narrower distally than mesially.
2. It is narrow occlusally than cervically. The marginal ridge is fairly well developed and is crossed by a prominent distal groove.
3. All three roots may be seen from this angle, as the distobuccal root is superimposed on the mesiobuccal root so that only the buccal surface and the apex of the latter may be seen.

Occlusal Aspect

1. Crown outline converges lingually.
2. The crown converges distally also.
3. The occlusal surface has a central fossa, a mesial triangular fossa.
4. It has well developed buccal developmental groove.
5. Sometimes there is a well-defined triangular ridge - oblique ridge.
6. The occlusal surface has three pits-central, mesial and distal.
7. The mesial pit-deepest and well defined, distal pit shallowest poorly defined.
8. The distobuccal cusp is separated from the mesio-buccal cusp by a buccal groove.
9. The occlusal pit-groove pattern is frequently H-shaped.
10. Supplemental grooves can be seen.
11. The crown may have three or four cusps.
12. On the 3–cusp form there is only a central and a mesial pit and an oblique ridge which often unites the mesiolingual with the distofacial cusps. The central groove connects the two fossa, the central fossa and the mesial triangular fossa.
13. The distal, facial and mesial developmental grooves radiate from the central pit.
14. On the 4-cusp form, there are three fossa – mesial, central and distal. A small pit is usually present in each fossa. Grooves originating at the distal pit are the distofacial triangular, distolingual, and the distal marginal grooves.
15. An oblique ridge runs from the distobuccal cusp to the mesiolingual cusp.

Pulp Cavity

1. Consists of a chamber and three canals.
2. The chamber consists of three or four pulpal horns and is more sharply pointed than the outer contour of the cusp.
3. Mesiobuccal is the largest of all the pulpal horns.
4. The mesiolingual pulpal horn is angular and sharp.
5. The distobuccal horn is the smallest.
6. It is sharp and occupies the extreme distobuccal angle.

Root

1. They are long and slender and they spread widely.
2. The roots are three in number – mesiobuccal, distobuccal and lingual.
3. The lingual root is the longest and diverges in a lingual direction.
4. The distobuccal root is the shortest.
5. The trifurcation or bifurcation of roots begins immediately at the site of the CEJ (cemento-enamel-junction).

MAXILLARY SECOND MOLAR (FIGS 1.7A TO D)

- Number of roots – 3
- Number of pulp horns– 4/5
- Number of cusps – 4/5
- Number of developmental lobes-5.

Buccal Aspect

1. The tooth resembles a permanent maxillary first molar, although much smaller in size.

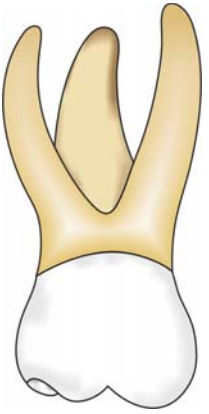


Fig. 1.7A: Buccal aspect

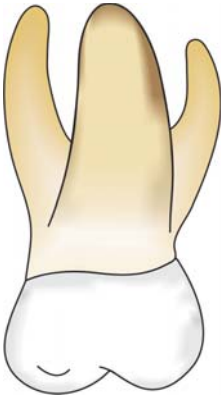


Fig. 1.7B: Palatal aspect

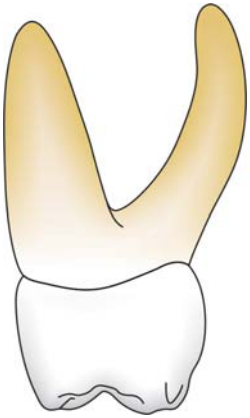


Fig. 1.7C: Mesial aspect

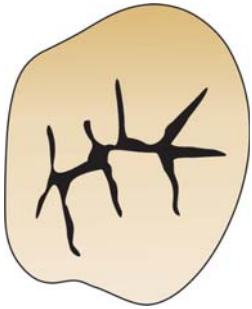


Fig. 1.7D: Occlusal aspect

2. Two equivalent buccal cusps can be seen from this aspect with a buccal groove between them.
3. A primary second molar is much larger than a primary first molar.
4. The crown is narrower at the cervix.
5. The roots from this aspect appear slender.
6. They are much longer and heavier than those of the maxillary first molar.
7. The crown of the second primary molar is trapezoidal in outline.
8. A well defined cervical ridge which extends the full diameter of the buccal surface.
9. The buccal surface is divided by the buccal groove into a mesiobuccal and a distobuccal cusp, the mesio buccal being the larger.
10. The morphological concordance between both maxillary and mandibular second primary molars and first permanent molars has been termed isomorphy.

Palatal Aspect

1. It is convex, inclining slightly as it approaches the occlusal border.
2. It is divided by the lingual groove.
3. The mesiolingual is higher than the distolingual cusp.
4. A fifth cusp, when present, occupies the mesiolingual area at the middle third of the crown- referred to as carabelli's cusp.
5. A lingual developmental groove separates the mesiolingual and distolingual cusps.

Mesial Aspect

1. The crown is 0.5mm longer and about 2mm wider buccolingually.
2. The roots are up to 2mm longer.
3. The cusp of carabelli is visible lingual and apical to the mesiolingual cusp.
4. The mesiobuccal root from this aspect appears broad and flat.
5. The lingual root has the same curvature as the root of the first deciduous molar.
6. The mesial surface presents a fairly high marginal ridge.
7. The mesiobuccal angle is acute, the mesiolingual angle is obtuse.
8. The surface is convex occlusocervically and less so buccolingually.

Distal Aspect

1. It is convex occlusocervically and flattened in its cervical portion.
2. Contact with the upper first permanent molar is in the form of an inverted crescent with the convexity toward the occlusal surface.
3. The distal calibration of the crown is less than the mesial measurement.
4. Distobuccal and distolingual cusps are about the same length.
5. A rather straight cervical line is evident both distally and mesially.

Occlusal Aspect

1. It has four well-developed cusps – mesiobuccal, distobuccal, mesiolingual, distolingual and a cusp of carabelli.
2. It is somewhat rhomboidal.
3. The mesiobuccal cusp is the largest and the disto-lingual is the smallest, except for the fifth cusp.
4. The occlusal surface has a central fossa with a central pit, a well-defined mesial triangular fossa.
5. Well–developed groove called the central groove connecting the mesial triangular fossa with the central fossa.
6. The buccal developmental groove extends buccally from central pit.
7. The oblique ridge is prominent and connects the mesiolingual with the distobuccal cusp.
8. Distal to the oblique ridge one finds the distal fossa - distal developmental groove.
9. The distal groove acts as a demarcation between the mesiolingual and distolingual cusps.
10. The occlusal surface has three pits. The central pit is large and deep which joins the shallow mesial pit and the distal groove, which traverses the oblique ridge to join the distal pit. The distal pit is deep.

Root

1. They have 3 roots – mesiobuccal, distobuccal and a palatal root.
2. They are thinner and flare more as they approach the apex.

3. The distobuccal root is the shortest and narrowest of the three.
4. The palatal root is the longest.
5. The point of bifurcation between the mesiobuccal root and the lingual root is 2-3mm apical to the cervical line of the crown.
6. The point of bifurcation between the distobuccal and the lingual root is more apical than any other points of bifurcation.
7. Unlike the first molar the mesiobuccal root may be as long as the lingual.

MANDIBULAR CENTRAL INCISOR (FIGS 1.8A TO D)

- Number of roots-1
- Number of pulp horns-3
- Number of developmental lobe-1

Labial Aspect

1. The labial aspect has a flat face with no developmental grooves.
2. The crown is wide in proportion to its length in comparison with that of its permanent successor.
3. It is smaller than the maxillary central incisor.
4. Tooth resembles the permanent maxillary lateral incisor.
5. The proximal sides of the crown taper evenly from the contact areas.
6. The root is long, evenly tapered down to the apex.
7. The root is almost twice the length of the crown.

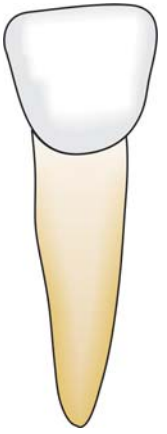


Fig. 1.8A: Labial aspect

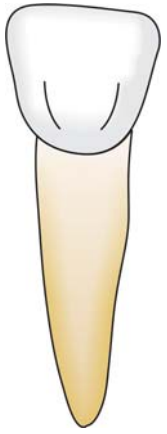


Fig. 1.8B: Lingual aspect

Fig. 1.8C: Mesial aspect

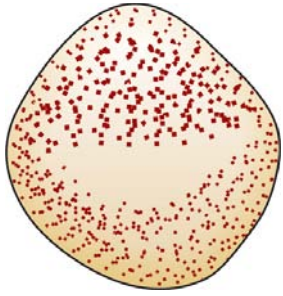


Fig. 1.8D: Occlusal aspect

8. The mesio and distoincisal angles are both sharp forming almost 90° angles.
9. The incisal edge of the newly erupted tooth is perfectly straight in the horizontal plane.

Lingual Aspect

1. On the lingual surface, the marginal ridges and the cingulum may be located easily.
2. The lingual surface at the middle third and incisal third may have a flattened surface level.
3. It may present a slight concavity called the lingual fossa.
4. The cingulum may be extending almost halfway up to the crown.

Mesial Aspect

1. Shows the typical outline of an incisor tooth.
2. The convexity of the cervical contors presents labially and lingually at the cervical third is pronounced.
3. The mesial surface of the root is nearly flat and is evenly tapered.
4. The incisal ridge is centered over the center of the root.

Distal Aspect

1. The outline is the reverse of that found from the mesial aspect.
2. These surfaces are convex labiolingually.
3. Contact with the adjacent teeth is on the incisal third.
4. The cervical line is positioned more apically on the lingual than on the labial side.

Pulp Cavity

1. The pulp canal is oval in shape.
2. The pulp cavity conforms to the general surface contor of the tooth.
3. The roof of the pulp chamber is widest mesiodistally.
4. Labiolingually the chamber is widest at the cingulum.
5. There is a definite demarcation between the pulp chamber and the canal.

MANDIBULAR LATERAL INCISOR (FIGS 1.9A TO D)

- Number of roots-1.
- Number of pulp horns-3.
- Number of developmental lobes-4.

Fig. 1.9A: Labial aspect

Fig. 1.9B: Lingual aspect

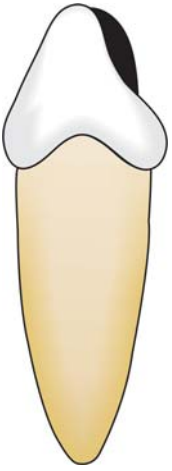


Fig. 1.9C: Mesial aspect

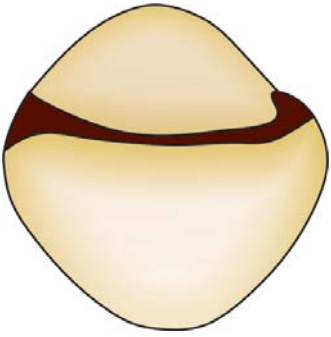


Fig. 1.9D: Occlusal aspect

Labial Aspect

1. The fundamental outline is similar to those of the primary central incisor.
2. The lateral incisor is somewhat larger in all measurements.
3. The cingulum may be a little more developed than that of the central incisor.
4. The incisal margin slopes downward distally in contrast to the central incisor.
5. Its distoincisal angle is rounded (obtuse) rather than sharp.
6. The distal margin of the crown is more rounded.

7. The slightly greater height and lesser mesiodistal diameter of the crown gives the crown a more rectangular and narrow appearance.
8. The mesioincisal angle is sharp.

Root

1. The root of the primary mandibular lateral incisor is longer, narrower.
2. Roots of the mandibular incisors, in general are straight in their cervical half, but then bend labially about 10^{o} in their apical half.
3. This bend helps in making space for the developing secondary incisors which should be erupting in a lingual and apical position.

MANDIBULAR CANINES (FIGS 1.10A TO D)

- Number of roots-1.
- Number of pulp horns-3.
- Number of developmental lobes-4.

The mandibular primary cuspid has the same general contor form as the maxillary cuspid, but is not as bulbous labiolingually or as broad mesiodistally.

Labial Aspect

1. It is convex in all directions.
2. It has a central lobe which terminates incisally, extends cervically to the cervical ridge.
3. The labial surface of mandibular cuspid is much flatter than the maxillary cuspid.

Fig. 1.10A: Labial aspect

Fig. 1.10B: Lingual aspect

Fig. 1.10C: Mesial aspect

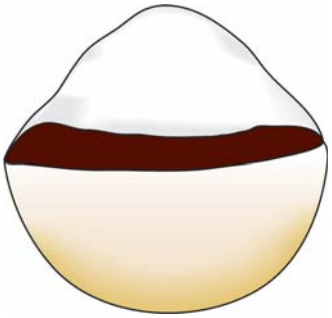


Fig. 1:10D: Occlusal aspect

4. The crown is perhaps 0.5 mm shorter and the root is at least 2mm shorter.
5. The cervical ridges are not quite as pronounced as maxillary cuspid.
6. The labiolingual diameter is much small.
7. The cingulum is much reduced in size and prominent and occupies less than the cervical third of the height of the crown.
8. Mandibular canines are longer incisocervically than mesiodistally.

Lingual Aspect

1. The lingual surface is made up of three ridges.
2. The marginal ridges are less prominent than on the maxillary cuspid.
3. The distal marginal ridge is slightly longer than the mesial.
4. The most obvious difference is the presence of one lingual fossa.

Mesial and Distal Surfaces

1. They are convex in the cervical third.
2. The mandibular canines are not as wide labiolingually resulting in smaller proximal surfaces.
3. Contact with the adjacent teeth is on the incisal third of the tooth.
4. The mesial and distal heights of contor are much nearer the cervix.

Incisal Aspect

1. The incisal edge is highest at the apex of the cusp and precedes cervically both mesially and distally.
2. From this aspect, the canines have a diamond shape.
3. The distal cusp slope is longer than the mesial slope.
4. The opposite arrangement is true for maxillary canines. This makes for proper intercuspation of these teeth during mastication.

Pulp Cavity

1. The pulp cavity conforms to the general contour of the tooth.
2. There is no differentiation between chamber and canal.

Root

1. The roots of the deciduous canines are almost twice as long as their crowns and are thicker than the roots of the incisors.
2. The mandibular root is slightly shorter than the maxillary root and is more tapered.
3. The root is triangular in cross-section.
4. In the apical third root is bent labially, similar to mandibular incisors.

MANDIBULAR FIRST MOLAR (FIGS 1.11A TO D)

- Number of roots-2.
- Number of cusps-4.
- Number of pulp horns-4.
- Number of developmental lobes-4.

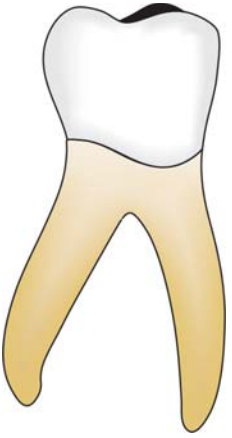


Fig. 1:11A: Buccal aspect

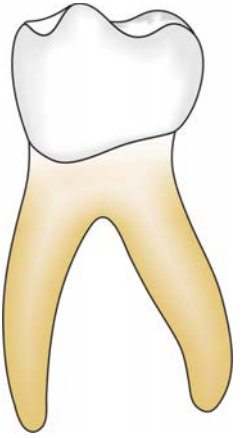


Fig. 1.11B: Lingual aspect

Fig. 1:11C: Mesial aspect

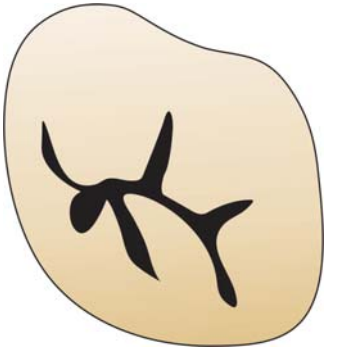


Fig. 1.11D: Occlusal aspect

This tooth does not resemble any of the other teeth, deciduous or permanent. Because it varies so much from all others, it appears strange and primitive. Its chief differing characteristic is its overdeveloped mesial marginal ridge. This ridge somewhat resembles a fifth cusp. The outline of the tooth is rhomboidal.

Buccal Aspect

1. The mesial outline of the first molar is almost straight from the contact area to the cervix, constricting the crown very little at the cervix.
2. The distal outline converges towards the cervix.
3. The distal portion of the crown is shorter than the mesial portion.
4. The two buccal cusps are rather distinct; the mesial cusp is larger than the distal cusp.
5. Instead of a groove, a developmental depression is present between the cusps.
6. The buccal surface presents a prominent cervical ridge where it joins the mesial surface at an acute angle and the distal surface at an obtuse angle.
7. Buccolingually the tooth gives a constricted appearance.
8. The crown is wider mesiodistally than cervic-occlusally.
9. The roots are long and slender and they spread greatly at the apical third beyond the outline of the crown.
10. The mesial root curves slightly distally in the apical third.

Lingual Aspect

1. The tooth converges lingually to a marked degree on the mesial half whereas distally it is opposite.
2. The distolingual cusp is rounded.
3. The mesiolingual cusp is long and sharp at the tip.
4. The mesiolingual cusp almost centered lingually.
5. Mesial marginal ridge is so well developed that it is considered to be another cusp.
6. The lingual surface is traversed by a lingual groove, thus dividing the lingual surface into a mesiolingual and a distolingual cusp.

Mesial Aspect

1. There is extreme curvature buccally at the cervical third.
2. The cervical line slants upward buccolingually.
3. A transverse ridge, connecting the mesiobuccal and mesiolingual cusps can be seen from the mesial aspect.
4. The mesial marginal groove separates the mesial marginal ridge from the mesiolingual cusp ridge.
5. The outline of the mesial root from the mesial aspect does not resemble the outline of any other primary tooth root.
6. The buccal and lingual outlines of the root drop straight down from the crown tapering only slightly at the apical third.

Distal Aspect

1. The cervical line does not drop buccally.
2. The length of the crown buccally and lingually is more uniform.

3. Cervical line extends almost straight across buccolingually.
4. The distal cusps are not long or as sharp as the two mesial cusps.
5. The distal marginal ridge is not as straight and well defined as the mesial marginal ridge.
6. The distal surface is more convex than the mesial surface.
7. The distal contact area with the second molar is in the middle of the crown.
8. The mesial contact area is located more cervically.
9. The distal root is rounder and shorter than the mesial root, tapering apically.

Occlusal Surface

1. The occlusal outline is rhomboidal in shape.
2. Prominence of the mesiobuccal surface is evident.
3. The mesiolingual cusp may be seen as the largest and best developed of all.
4. The buccal developmental groove divides the buccal cusps evenly.
5. Buccal developmental groove is short, extending from the buccal cusp ridges to the central pit.
6. The central developmental groove joins buccal developmental groove at this point and extends mesially, separating the mesiobuccal cusp and mesiolingual cusp.
7. The central groove ends in a mesial pit in the mesial triangular fossa.
8. The mesiobuccal cusp exhibits a well-defined triangular ridge, which terminates in the center of the

occlusal surface buccolingually at the central developmental groove.

9. The lingual developmental groove extends lingually, separating the mesiolingual cusp and the distolingual cusp.
10. The occlusal surface has a small mesial triangular fossa and pit and a larger distal fossa that extends almost into the center of the occlusal surfaces.
11. In the distal fossa there is a central pit and a small distal pit near the distal marginal ridge. There is no central fossa.

Pulp Cavity

1. The pulp chamber has four pulpal horns.
2. The mesiobuccal horn, occupies a considerable part of the pulp chamber. It is rounded and connects with the mesiolingual pulpal horn in a high ridge.
3. The distobuccal pulpal horn lacks the height of the mesial horns.
4. The mesiolingual pulpal horn is long and pointed.
5. The distolingual pulpal horn is the smallest and more pointed than the buccal horns.
6. There are 3 pulp canals – mesiobuccal, mesiolingual, and distal canal.
7. The distal canal which projects in ribbon fashion is wide buccoligually and may be constricted in its center.

MANDIBULAR SECOND MOLAR (FIGS 1.12A TO D)

- Number of cusps-5.
- Number of roots-2.
- Number of pulp horns-5.

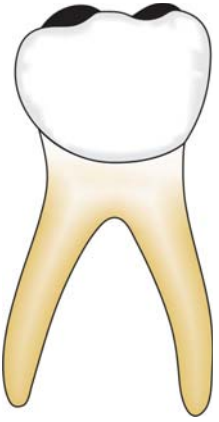


Fig. 1:12A: Buccal aspect

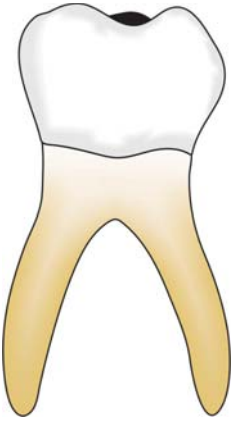


Fig. 1:12B: Lingual aspect

Fig. 1:12C: Mesial aspect

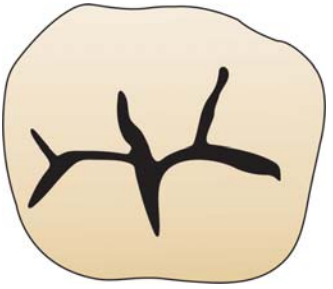


Fig. 1:12D: Occlusal aspect

- Number of developmental lobes-5.

The primary mandibular second molar has characteristics that resemble those of the permanent first molar, but its dimensions differ. The tooth is larger than the first primary molar and smaller than the first permanent molar.

Buccal Aspect

1. It has a narrow mesiodistal calibration at the cervical portion of the crown.
2. Mesiobuccal and distobuccal developmental grooves divide the buccal surface of the crown occlusally into 3 cuspal portions almost equal in size mesiobuccal, buccal and a distobuccal cusp.
3. The distobuccal cusp extends lingually at the occlusal border more than the buccal cusps to give a smaller occlusal area at the distocclusal surface.
4. The roots of a second molar are longer and slender, flaring mesiodistally at their middle and apical thirds.

Lingual Aspect

1. Two cusps with equal dimensions are seen between the short lingual groove.
2. The arrangement narrows the crown lingually.
3. The cervical line is relatively straight and the mesial portion of the crown seems to be a little higher than the distal portion of the crown from this aspect.

Mesial Aspect

1. The crest of contor buccally is more prominent on the primary molar, and the tooth seems to be more constricted occlusally because of the flattened buccal surface.

2. The marginal ridge is high, a characteristic that makes the mesiobuccal cusp and the mesiolingual cusp appear rather short.
3. The lingual cusp is longer than the buccal cusp.
4. The contact area with the first primary molar is in the shape of inverted crescent just below the notch of the marginal ridge.
5. The surface is constricted at the occlusal border.

Distal Aspect

1. The crown is not as wide as mesially. It is possible to see the mesiobuccal cusp as well as the distobuccal cusp from the distal aspect.
2. The distolingual cusp appears well developed.
3. The distal marginal ridge dips down more sharply and is shorter buccolingually than the mesial marginal ridge.
4. Contact with the first molar (permanent) is not as broad as contact on the mesial surface, being in the form of a round contact just buccal and cervical to the distal groove.
5. The distal root is almost as broad as the mesial root and is flattened on the distal surface. The distal root tapers more at the apical end than does the mesial root.

Occlusal Aspect

1. The occlusal aspect is somewhat rectangular.
2. The three buccal cusps are equal in size and so are the lingual cusps. However, the total mesiodistal width of the lingual cusp is less than the total mesiodistal width of the three cusps.

3. The buccal aspect is made up of 3 cusps – a mesiobuccal cusp (second in size), a distobuccal cusp (largest) and a small distal cusp.
4. The lingual aspect is made up of two cusps of about equal size, the mesiolingual and distolingual cusps which are divided by the distolingual groove.
5. The crown converges both distally and lingually.
6. The mesial marginal ridge is better developed and more pronounced than the distal marginal ridge.
7. There are three pits on this surface, of which the central pit is the deepest and best defined, followed by the mesial pit and the least well defined distal pit.

Pulp Cavity

1. It is made up of a chamber and usually three pulp canals.
2. The pulp chamber has five pulpal horns corresponding to the five cusps.
3. The mesiobuccal and mesiolingual pulpal horns are the largest, the mesiolingual pulpal horn being slightly less pointed.
4. The distobuccal pulpal horn is not as large as the mesio buccal pulpal horn but larger than the distolingual or distal horns.
5. The distal pulpal horn is the shortest and smallest
6. The two mesial pulpal horns are the shortest and canals leave the floor of the pulp chamber through a common orifice that is wide buccolingually and narrow mesiodistally. The common canal soon divides into a longer mesiobuccal canal and a smaller mesiolingual canal.

CHAPTER 2

Developmental Disturbances of Teeth

Developmental Alteration in the Size of Teeth

Although tooth size is variable among different races and between the sexes, there is usually symmetry of two sides of the jaws. When significant size variation is present, the entire dentition is rarely affected. Typically only a few teeth are altered significantly in size.

Microdontia

This term is applied only when the teeth are physically smaller than usual. Widely spaced normal sized teeth may appear small within the jaws that are larger than normal. This is termed relative microdontia. But this in true sense represents macrognathia, not microdontia. If all the teeth in both the normal sized arches are smaller than normal is referred to as generalized microdontia. True generalized microdontia is seen in pituitary dwarfism, Down's syndrome. Isolated microdontia/microdontia involving one or two teeth are far more common than generalized types. Most commonly affected teeth are maxillary lateral incisor or maxillary third molars. Maxillary lateral incisors typically appear peg shaped crown (conical) (Fig. 2.1).

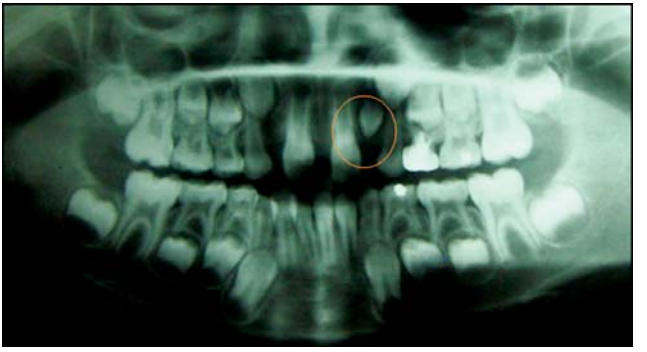


Fig. 2.1: Microdontia (peg-shaped lateral)

Macrodontia (Megalodontia, Megadontia)

In this condition, one or more teeth are physically larger than normal. This term should not be used when normal sized teeth are crowded with in a small jaw. In such case relative macrodontia is more appropriate. In addition, the term macrodontia should not be used to describe teeth that have been altered by fusion/gemination. Diffuse/true generalized macrodontia is seen in pituitary gigantism. Regional/localized macrodontia is occasionally seen on the affected side of the mouth in patients with hemifacial hypertrophy. Macrodontia of single tooth is seen, but is rare. Rhizomegaly/radiculomegaly are an uncommon type of macrodontia in which the root/roots of a tooth are considerably longer than normal. This condition is observed commonly in mandibular canines.

Developmental Alteration in Number of Teeth

Anodontia/Hypodontia

Anodontia: Congenital absence of all teeth.

Partial Anodontia/Hypodontia: Congenital absence of one or more teeth.
Oligodontia: Congenitally missing six or more teeth.

These terms cannot be applied to teeth that have developed, but have failed to erupt.

Many cases of hypodontia appear to be genetically controlled. Environment also influences the final out come or in some cases may be responsible completely for the lack of tooth formation. For example trauma, infection, radiation, chemotherapy, endocrine disturbances and severe intrauterine disturbances have been associated with missing teeth.

Ectodermal dysplasia (Fig. 2.2): This is an X-linked recessive disorder affecting essentially males. Defect is seen in all ectodermally derived structures. Patient exhibit decreased

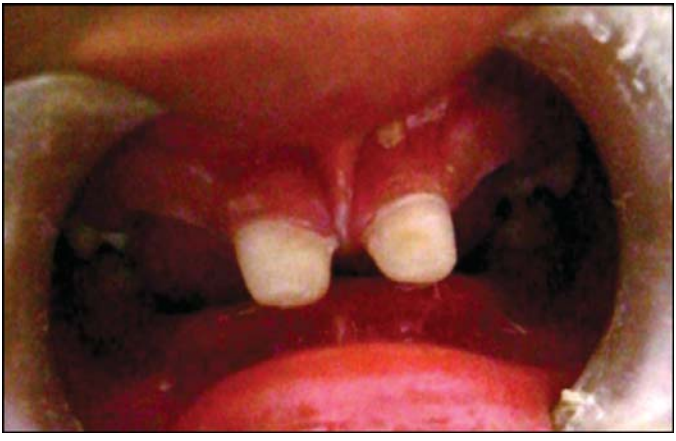


Fig. 2.2: Patient having hypodontia due to ectodermal dysplasia

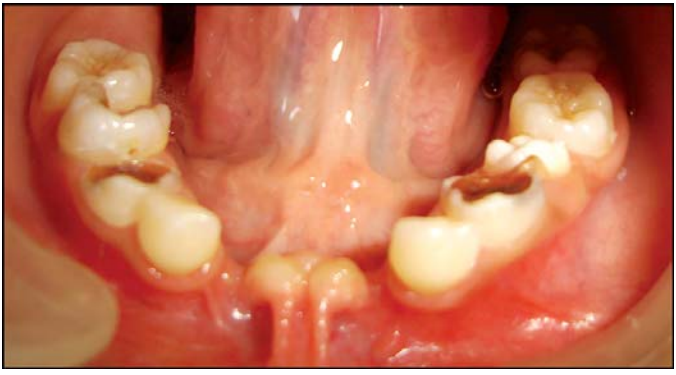


Fig. 2.3: Hypodontia i.e. congenitally missing mandibular central and lateral incisors along with double frenum

sweating, sparse hair, many congenitally missing teeth. Hypodontia involves most stable teeth like central incisor and first molars (Fig. 2.3).

Solitary median maxillary central incisor syndrome: Presents with a single central incisor in the midline. This condition may be associated with other midline disturbances such as cleft lip and palate, imperforate anus, umbilical hernia, choanal stenosis.

Hyperdontia/Supernumerary Teeth

More frequent in the permanent dentition. Ninety percent of the cases occur in the maxilla with strong predilection for the anterior region. The most common region is the maxillary central incisor followed by maxillary 4th molar. Most supernumerary teeth are unilateral. Supernumerary tooth located between maxillary central incisors is termed as **mesiodens (Fig. 2.4)**. Supernumerary 4th molar is called

distomolar. A supernumerary tooth situated lingually or buccally to a molar is called paramolar. Supernumerary teeth are morphologically classified as either supplemental or rudimentary. Supplemental teeth duplicate the typical anatomy of posterior or anterior teeth. Rudimentary supernumerary teeth are dysmorphic and can assume conical or tuberculate form. The eruption of supernumerary teeth is variable and depends on the space available. Seventy percent of supernumerary teeth in the anterior maxilla fail to erupt. In such situation midline diastema is common. Whenever there is a large midline diastema, radiographic evaluation to detect presence of supernumerary tooth is essential. Supernumerary teeth can also prevent eruption of adjacent teeth, displacement or rotation, crowding. Unerupted supernumerary teeth can be associated with cyst. At times they can erupt into nasal cavity as well. Meticulous clinical and radiographic examination is required to detect supernumerary teeth.

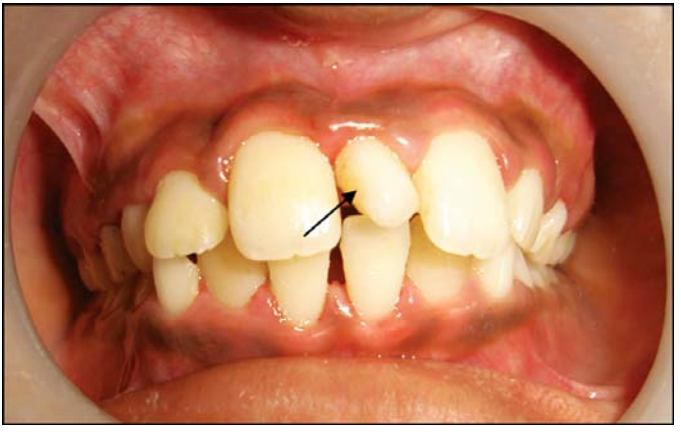


Fig. 2.4: Arrow indicating supernumerary tooth/mesiodens

Horizontal tube shift technique where two periapical radiographs are taken at different horizontal angulation is helpful in detecting the position of the unerupted teeth.

Developmental Alterations in Shape of Teeth

Double Teeth

Refers to two conjoined teeth. However this term should not be applied to teeth joined only through their root cementum. They are referred by other terms like "Bifid teeth", "connation", "joined teeth", "double formations".

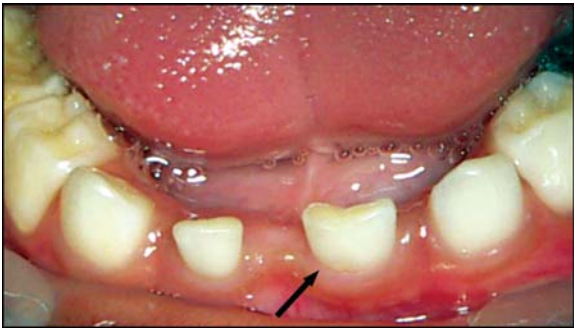
Fusion: Refers to union of two separate tooth germs during their formation. They may be joined only by enamel and/or dentin. Pulps may also be fused. Extent of union depends on the stage of development of teeth at the time of fusion. Fused teeth appear broader. They also frequently exhibit incisal notch or grove which may continue on to the root surface (Figs 2.5A and B).

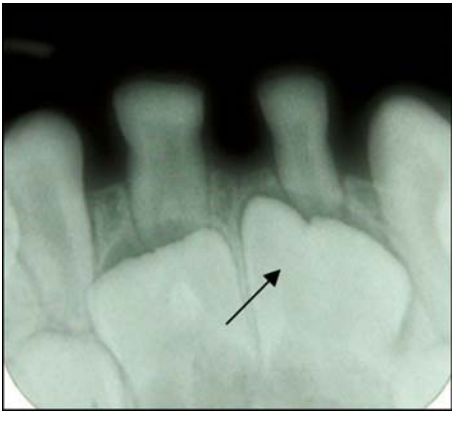
Gemination: Refers to a double teeth originated from one tooth bud. This is the result of incomplete splitting (Fig. 2.6).

Differential Diagnosis

There no flawless criteria to differentiate between fusion and gemination.
Following features can be used to distinguish between them:

i. *Morphology*: In case of gemination, fused crowns exhibit mirror image. Whereas fusion takes place at an angle causing crooked appearance.





Figs 2.5A and B: Clinical and radiological pictures of the fusion of central and lateral incisor

ii. *Pulpal anatomy*: Fused teeth tend to have double pulpal space and geminated teeth usually have an undivided pulp. However in both gemination and fusion the pulp spaces may or may not be separated.

iii. *Location in the jaw*: Double teeth in the mandible would almost exclusively represent fusion and germinations are more common in maxilla. But majority of fusions

of primary teeth and geminations were located in the mandible.

iv. *Crowding*: Fused teeth occupy less space than separate teeth would, causing spacing in the arch. Gemination results in extra tooth hence require more space thereby likely to cause crowding but fusion of normal tooth bud with a supernumerary would result in crowding. Hence neither crowding nor diastema seems decisive.

v. *Number of teeth*: In case of fusion there will be reduction in tooth number. Gemination does not reduce the tooth number. However fusion of normal tooth with supernumerary tooth will not result in reduction in tooth number.

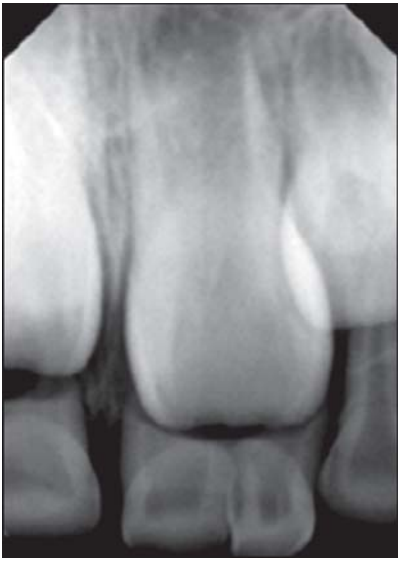


Fig. 2.6: Gemination

Concrescence

Concrescence is union of two adjacent teeth by cementum alone without union of the underlying dentin. Unlike fusion and gemination, concrescence can be developmental or post-developmental inflammation. More frequent in posterior maxilla. Usually between maxillary second and third molars.

Accessory Cusps or Supernumerary Cusps

Occasionally teeth exhibit additional/supernumerary cusps.

a. **Cusp of carabelli:** The most common example of this phenomenon is the cusp of carabelli located on the palatal surface of mesiolingual cusp of a maxillary molar. Most obvious on first molar.
b. **Dens evaginatus:** Can be defined as a tubercle or protuberance from the involved surface of the affected tooth. This is also referred by other terms like **occlusal tubercle, Leong's premolar, tuberculated premolar, occlusal enamel pearl**. This protuberance has an outer layer of enamel, a core of dentin and may also contain pulp extension into it. Most commonly seen on occlusal surface of premolars. May occur in the form of a drop, nipple or cylindrical cone. Occur predominately in people of mongoloid origin. This condition is clinically important because fracture or wear of the tubercle frequently leads to the major complications like pulp necrosis, periapical infection, often before the root completion (Fig. 2.7).

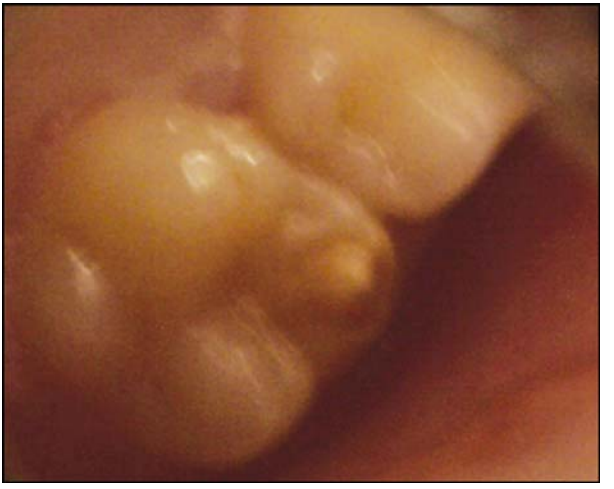
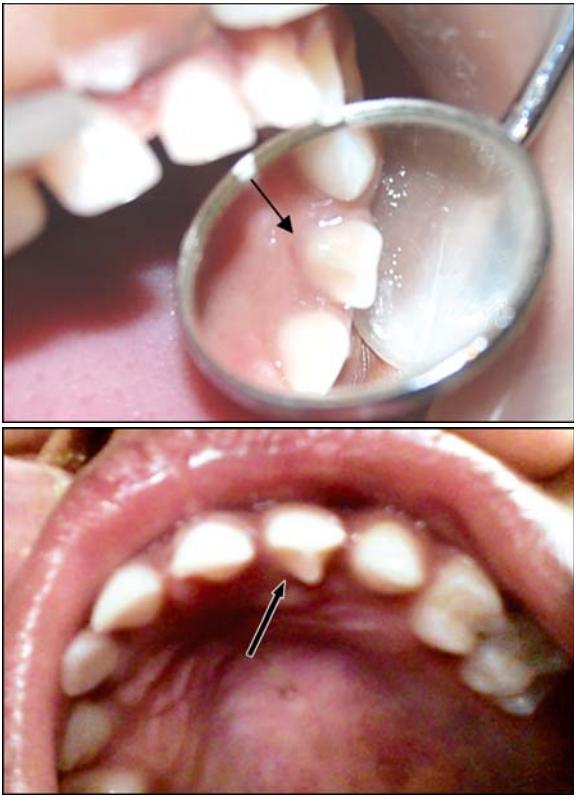


Fig. 2.7: Dens evaginatus

c. **Talons cusp:** Is a cusp like structure projecting from the cingulum of incisors. In shape it resembles an eagle's talon. It can be considered as dens evaginatus of anterior teeth. Predominately seen on maxillary permanent lateral incisors. Very rarely seen on deciduous teeth. Like dens evaginatus talon cusp is composed of enamel, dentin and pulp extension. Talon cusp is common in Chinese, Caucasians and African – Americans. Incidence of talon cusp is high in patients with Rubinstein-Taybi syndrome and Struge-Weber syndrome (Figs 2.8A and B).



Figs 2.8A and B: Both showing talons cusp in relation to left primary maxillary lateral incisor

Shovel Shaped Incisors

Affected incisors demonstrate prominent lateral margins creating a hollowed lingual surface resembling shovel. Typically the thickened marginal ridges converge at the cingulum, not uncommonly there is a deep pit or dens

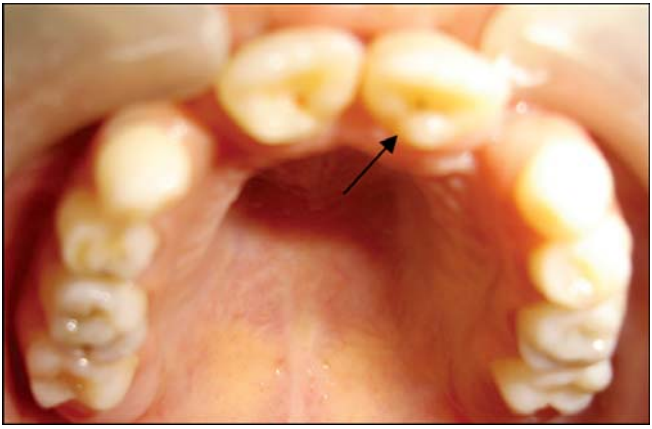


Fig. 2:9: Shovel shaped incisors

Invaginatus at this junction. Maxillary incisors are more commonly affected (Fig. 2.9).

Dens Invaginatus

Dens Invaginatus is a deep surface invagination of the crown/root that is lined by enamel (Fig. 2.10). Known by other terms like dens in dente, dilated odontome. Dens Invaginatus can be divided into coronal and Radicular type. Coronal type has been further divided into three types (Oehler 1957)

a. **Type I – invagination confined to the crown.**
b. **Type II – invagination extends to the root portion and ends in a blind sac that may or may not communicate with the adjacent dental pulp.**
c. **Type III – extends through the root and perforates in the apical or lateral root surface without any**

communication with the pulp. Radicular type is rare. Strip of enamel lined invagination extends into the root surface.

In most cases dens invaginatus is detected by chance on radiograph. Clinically an unusual crown morphology in the form of dilated peg shaped, barrel shaped crown or deep foramen cecum may be important hint. As maxillary lateral incisors are commonly involved, these teeth should be investigated thoroughly clinically and radiographically, at least in case of deep pit in foramen cecum region. If one tooth is involved contralateral tooth should also be investigated. In case of coronal type, pulpal involvement can occur shortly after the eruption of tooth, early diagnosis is mandatory.

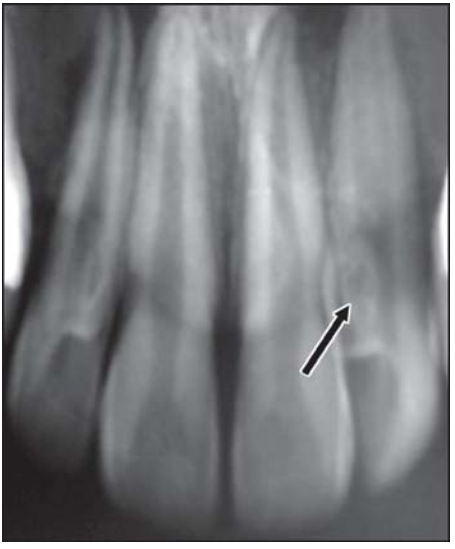


Fig. 2.10: Dens invaginatus

Taurodontism

Taurodontism is an enlargement of the body and pulp chamber of a multirooted tooth with apical displacement of the pulpal floor and bifurcation of the roots. Affected teeth tend to be rectangular in shape. There is increase apico-occlusal height of pulp chamber and furcation is close to the apex. The diagnosis is made from the radiographic appearance. Depending on the degree of apical displacement of the pulpal floor taurodontism can be mild, moderate or severe. If endodontic treatment is required in such tooth, the shape of the pulp chamber increases the difficulty of locating, instrumenting and obturating the pulp canals (Fig. 2.11).

Dilaceration

Dilaceration is an abnormal angulation or bend in the root or less frequently the crown of a tooth. Any tooth may be affected. Dilaceration do not interfere with eruption.

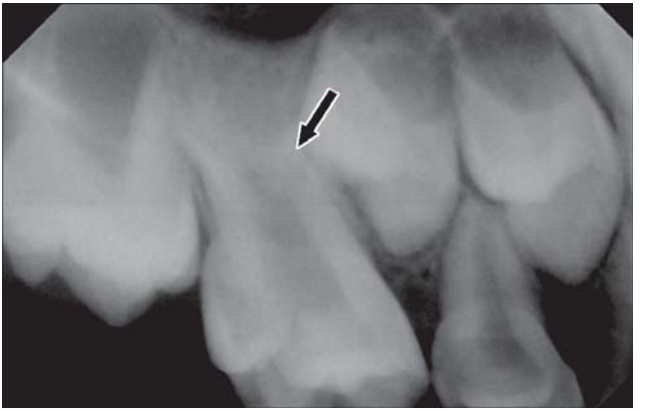


Fig. 2.11: Taurodontism of first maxillary molar

Dilacerated tooth erupt into complete occlusion, but follow altered path. Minor dilacerations of normal teeth requires no treatment. If eruption is delayed or abnormal, surgical exposure and orthodontic movement is required. Caution must be exerted during endodontic treatment of dilacerated teeth to avoid perforation of the root (Fig. 2.12).

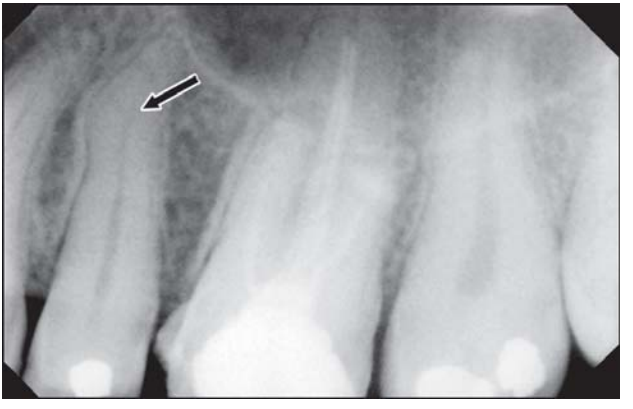


Fig. 2.12: Dilaceration seen in relation to maxillary premolar

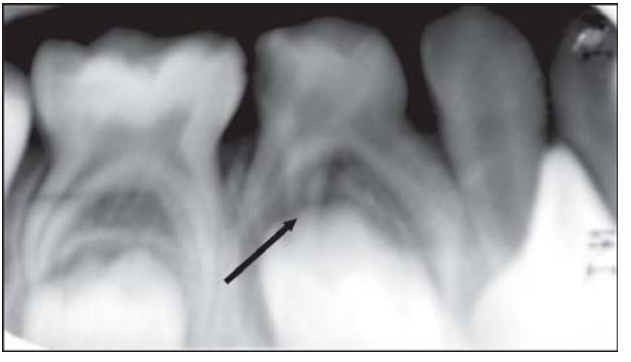


Fig. 2.13: Supernumerary root of primary mandibular first molar

Supernumerary Roots

Supernumerary roots refers to development of increased number of roots on a tooth compared with that classically described in dental anatomy. Both deciduous and permanent can be involved. Detection of supernumerary root is critical when endodontic therapy and exodontias is under taken. Otherwise no specific treatment is required (Fig. 2.13).

CHAPTER 3

Pain

INTRODUCTION

Dentistry owes its very beginning to the quest for pain relief and the identification, diagnosis and early elimination of pain remain important foundations to the dental profession.

Pain is a symptom in the completely subjective sense. It is a complex experience that includes a sensory—discriminatory component.

Pain is accompanied by the most widespread reactions and can sometimes be said truly to involve the whole being. The experience of pain is a subjective, private sensation, which can be described only imperfectly to others. Whereas it is possible to share other sensations by, for example, listening, testing or touching with reasonable certainty of the same experience as another person but this is not possible with pain. Pain also includes the reactions elicited by the stimulus. For instance, some of the reactions to a noxious stimulus include pain. Startle response, muscle reflex, vocalization, sweating, and increase in heart rate, in addition to blood pressure and behavioral changes.

Such reaction will be modified by a number of factors, including the emotional status of the patient, past experiences and memories of pain, magnitude of tissue

damage or stimulus other concomitant sensory experiences ability to comprehend the causes and consequences of the pain and ethnic group and culture.

Thus pain is not only complex and multidimensional but it is also protective, defensive and diagnostic which result in the accurate diagnosis and appropriate treatment that is often difficult in infants and young children.

Thus pain is a neuroscience; characterized by the following:

1. More the sensory nerves that supply to a particular region of body, the more acute will be the sensitivity in the area.
2. Higher the ratio of motor nerves to muscles, the greater will be the possible control finesse.

Different individuals feel and react to pain and suffering in different ways. Patient in pain therefore require individual treatment. Sensation of pain is perceived in our brain i.e. The brain receptor generates electrical impulses that travel through the spinal cord along with nerve impulses to brain.

Pain and other sensations are generally carried by three different types of fibers:

1. A beta—carry sensations like touch, pressure and position.
2. A delta—carry sharp pain with a speed of 12 to 20 m/sec.
3. C fibers—carry pain with a speed of 2 to 3 m/sec.

Pain can be acute or chronic in nature:

- Acute pain is a pain of sudden onset and short duration which is generally associated with tissue damage or threat of damage.

- Chronic pain may persist for long periods of time and are often associated with a variety of factors, including:
  1. Some definite past event (accident, infection).
  2. Some ongoing disease process (rheumatoid arthritis, temporomandibular dysfunction).
  3. Unknown cause in many cases.

Terms used to Describe Pain

The characteristic of a pain are difficult to define although an 'ache' is the term most frequently used.
Other terms includes:

1. Burning pain
2. Deep pain
3. Throbbing pain
4. Gnawing pain
5. Pricking pain
6. Stinging pain
7. Superficial pain
8. Pulsatile pain
9. Tickle.

The level of suffering may increase with the duration of pain, even though the intensity either remains the same or actually decreases.

Somatic pain often reflects a pathological lesion in a specific organ or tissue.

Neurogenic pain may indicate a disease process affecting a particular nerve fiber.

Visceral pain is transmitted mainly in the fibers which may accompany sympathetic nerves, most visceral pain impulses travel with sympathetic nerves.

Psychogenic pain: Psychic and emotional states influence pain perception and its expression. Such patients are emotionally disturbed and are very common in young women. Pain may continue for days together. A primary nociceptive input characterizes the pain emanating from the structures that hurt whereas secondary pain may comprise of pain that is felt from some distance from the actual lesion (e.g. somatic deep pain). All deep somatic pains are not alike. By contrast high threshold receptors innervate visceral structures so that pain is not usually felt until a certain threshold level in reached.

Most pains result from stimulation of nerves that innervates the site although some points occur spontaneously without any stimulation. Pain is therefore a complex series of phenomena that may be described as unpleasant emotional and sensory experiences, associated with either actual or potential tissue damage or described in terms of tissue damage.

MECHANISMS OF NEURAL PAIN PERCEPTION

Specificity Theory

Specificity theory of pain was considered a specific sensation with its own anatomically distinct receptors, primary and cortical reception areas. This theory contended that only free unmyelinated nerve endings in the orofacial area were activated by noxious stimuli. More recently, the anatomical specificity of receptors has been discarded as most receptors, if sufficiently stimulated can respond to noxious irritation.

Summation Theory

Summation theory of pain is an alternative theory postulates that pain does not depend on specific pathways but on excessive stimulation involving all types of receptors with the resultants central neural summation or convergence of activity. This summation theory was subsequently replaced by the **pattern theory** where by the pattern of neural impulses set up by the noxious stimuli were considered important.

Sensory Interaction Theory

Sensory interaction theory contended that pain result from the interaction of both nociceptive and non-nociceptive afferent fibers on central neurons, thus resulting in an excitatory or inhibitory interactions determining whether or not pain was perceived. Such a plethora of theories serves to emphasize the complexity of the neuronal component of pain sensation. In fact, the central and peripheral mechanisms for pain are both extremely complex and only partially understood. In fact, approximately 50% of sensory fibers and up to 20% of spinal motor roots can transmit pain. Also, most pain receptors are polyrather than unimodal. There are no central pain centers in the central nervous system analogous to the respiratory or vasomotor center.

The sensory pain receptors serving nociception may be divided into four main groups:

Pain Receptors

- Free receptors
- Proprioreceptor

- Exteroceptors
- Interoceptors

Free (Uncapsulated) Receptors

It is the predominant form of nociceptor, especially in the cutaneous tissues, oral mucosa and periodontal tissues. But although these free nerve endings are associated with pain sensation, similar responses can be elicited from other specialized nerve endings.

Proprioreceptor

Involved in automatic functioning and perceives movement, pressure and position.

1. **Free nerve endings:** Deep somatic pain and other sensations.
2. **Golgi tendon organs:** Mechano receptors in muscle tendon.
3. **Muscle spindles:** Mechano receptors.
4. **Pacinian corpuscles:** Perception of pressure.
5. Periodontal receptors perception of tooth movement.

Exteroceptors

Exteroceptors are stimulated by the immediate external environment, with most of the impulses being sensed at conscious level.

1. **Free nerve ending** : Tactile and superficial pain
2. **Krause's corpuscles** : Cold receptor
3. **Meisssner's corpuscles** : Tactile skin receptors

4. **Merkel's corpuscles** : Tactile receptor in the oral mucosa and sub mucosa of the tongue.
5. **Ruffini's corpuscles** : Pressure and warmth receptor.

Interoceptors

Located within the body cavities, they serve involuntary bodily function below conscious levels.

1. **Free nerve endings:** perception of visceral pain and other sensation.
2. **Pacinian corpuscles:** perception of pressure.

Central Connection of Pain

Oral or facial pain sensation is mediated centrally by afferent primary neurons that pass through the following:

1. The posterior roots of the fifth- Trigeminal, seventh- Facial, nineth- Glossopharyngeal and tenth- Vagus cranial nerves.
2. The first, second and third cervical spinal nerves
3. The visceral afferent that descends through the cervical sympathetic chain to pass through the posterior roots of the upper thoracic spinal nerves.

The nerve cell bodies of the sensory neurons are located in the posterior root ganglion of the respective nerves through which they pass. The pain fibers from the maxillofacial region terminate in the *nucleus caudalis*. This is the lower portion of the trigeminal spinal tract nucleus. The second order trigeminal neurons begin in the substantia gelatinosa of the nucleus caudalis and project to the thalamus. Third order neurons then project from the thalamus to the cerebral cortex.

During the transmission of nerve impulses through these tracts, however, they are subjected to numerous inhibitory and facilitatory impulses from the higher cerebral centers. The general intensity of suffering related to a number of factors, including attention, attitude, preconditioning experience and temperament, all of which exert pre-synaptic inhibitory influence on the painful stimuli reaching the *nucleus cacidalis*. Thus pain transmission from the face and mouth is not just a simple neural pathway a variety of intermediate influence may be exerted on the neural transmission.

Such a modulating system may involve the following:

1. A change in the character of the evoked discharge.
2. Alteration of the modalities to which the neuron responds.
3. Alteration of the characteristic of its receptive field.

There appears to be a general relationship between nerve fiber diameter and conduction velocity.

Large fibers conduct impulses more rapidly than small ones. There also appears to be some relation between fiber size and the type of impulse transmitted. A and C fibers appear to convey pain impulses in addition to other sensory modalities, where as fast conducting Aα, β and γ fibers primarily convey tactile and proprioceptive impulses but no pain. Sensory information may be rapidly conducted to CNS by transmission of nerve impulses or more slowly by means of neurochemical substances passing through the axonal transport system.

Chemical Mediators

A number of biochemical factors are also associated with the transmission of pain.

1. **Bradykinin** is an endogenous polypeptide released from the inflammatory reaction site. It serves as a powerful vasodilator and excites all types of receptors, sensitizing some high threshold receptors to respond to otherwise innocuous stimuli. It acts only in the presence of prostaglandin.
2. **Histamine** is a vasoactive amine – acts as a vasodilator which increases the permeability of the small blood vessels and functions as a central nervous system neurotransmitter.
3. **Prostaglandins:** Sensitize nociceptors to different types of stimuli, thus lowering their pain thresholds to all types of stimulation. Prostaglandin E increases the response of slowly adapting A methane receptors to non-noxious stimuli.
4. **Serotonin** is associated with vascular pain syndromes as an algogenic agent, an important endogenous anti-nociceptive mechanism.
5. **Substance P** polypeptide acts as an excitatory neuro-transmitter. It is released from spinal cord cells i.e. A δ and C fiber by afferent stimulation and by excitation of dorsal horn neurons.

Substance P modulating action on pain is both rapid and short lived.

Other agents: Acetylcholine, potassium and a variety of endogenous toxic substance serve as an algogenic agent.

Mechanics of Pain

At one time pain was considered to be evoked by noxious stimulation of neural structures, with the pain reaction involving a cerebral level modified by prior conditioning such as memory, emotional response and evaluative significance of pain. Pain is now considered to be a much more complex process, with neural impulses being altered, changed and modulated on passing to the higher centers in addition to being affected by excitatory and inhibitory influences.

Gate Theory

The gate control theory of pain is still the most accepted mechanistic theory of pain. It was devised by Melzack and Wall. According to this theory noxious stimulation of the substantia gelatinosa (SG) functions as a gate control system that modulates the afferent pathways before they influence the transmission of cells. The afferent patterns in the dorsal column system therefore function in part as a control trigger which activates the selective brain process that influence the modulating properties of the gate control system.

The transmission cells activate neural mechanisms that comprise the action system responsible for response and perception. On noxious stimulation of the skin impulses are transmitted centrally by both large A and small C fibers.

The nerve impulses passing in the large A fibers arrive at the substantia gelatinosa before those transmitted by the C fibers. These latter fibers activate the transmission cells initially; in addition to activating a negative feedback mechanism (reduces their effect). Large fiber impulses

therefore closes the gate, whereas small fiber impulses opens the gate.

Continuous ongoing bombardment by afferent nerve impulses by C fibers serves to hold the gate in a relatively open position. On noxious stimulation, the activity of the large fibers increases disproportionately to that of the small fibers and the stimulus is received as pain. Decreased large fiber activity will result in gate opening, whereas there will be increased pain perception if there is disproportionate small fiber activity.

A critical level of gate activity is therefore set by the large fibers. When this critical level is set high, pain is not perceived until the firing level in reached, causing a delay between stimulus and perception, termed ***temporal summation***. After reaching this critical firing level a transmission all barrage may occur explosively, causing pain that is wholly disproportionate to stimulus intensity. This is ***spatial summation***.

Deficient large fiber activity may therefore elevate the critical firing level of the gate control system reflected by summation effects and also result in gate being opened with the effect that lower intensity stimulation is perceived as pain.

Gate–setting and critical firing levels are therefore different faces of the pain mechanism, even though both affect the system. Presenting of the gate is also determined by higher central nervous system centers and may depend on factors like:

1. Past experience
2. Conditioning
3. Attention directed to suffering
4. The emotional status of the individual at a given time.

Presenting of the gate is also dependent on the stimulus. When stimulation occurs, certain extremely fast-conducting fibers bypass the substantia gelatinosa to synapse first in the thalamus and then to the sensory cortex. These very rapidly transmitted impulses convey data concerning the location and nature of the noxious stimulus before the same stimulus reaches the synaptic gate.

Such impulses have two effects:

1. Setting the receptivity of cortical neurons.
2. Resetting the gate itself.

There may be central modulation of the gate before the noxious impulses have had time to reach the gate. If such central inhibitory feedback control on the gate is total, no pain will be felt, if the central inhibitory system fails, however, all sensations may be perceived as pain. Finally, the second order neurons from the gate to the thalamus are another level where modulation may occur. Those neurons may transmit data concerning the nature of the noxious stimulus resulting in activation of both motor and sensory cortices.

The role of the cerebral cortex in either sensory perception in general or pain perception in particular is however still uncertain. Undoubtedly the cortex and other parts of the forebrain are implicated in motivation, emotion and pain memory, although their exact involvement in pain mechanism has yet to be defined.

Orofacial Pain

Orofacial pain may be carried by myelinated or unmyelinated fibers afferents from a receptive field.

1. High-threshold mechanoreceptive (A) afferent fibers activated only by intense mechanical stimuli.
2. Heat nociceptive (A) afferents that respond to intense heat.
3. Strong mechanical stimuli and polymodal nociceptive C afferent that are responsive to intense mechanical thermal and chemical stimuli.

These afferents are conveyed to the trigeminal ganglion where their primary afferent cell bodies are located. They then enter the brain stem and traverse the trigeminal spinal tract before entering the trigeminal nuclear complex.

Within this nucleus caudalis there appears to be three general types of nociceptive neurons:

1. Nociceptive—specific neurons that respond exclusively to noxious mechanical and thermal stimuli.
2. Wide-dynamic range neurons that respond to non-noxious as well as noxious stimuli.
3. Low-threshold mechanoreceptive neurons that respond to light touch, pressure or facial hair movement.

Cerebral cortex is involved in both general sensory perception as well as pain perception.

Secondary Effects of Pain

There is generally a close segmental relationship between the primary initiating pain and secondary effects of noxious stimulus. Most secondary symptoms occur in structures innervated by the same major nerve that mediates the primary pain. Sensory effects resulting in referred pain and secondary hyperalgesias. (E.g. angina pectoris radiates to the top of the left shoulder).

Referred Pain

The exact mechanism is unclear but appears to depend on convergence of afferent inputs on to central neurons from both the source and the referred sites, in addition to central summation.

For instance:

1. Afferent inputs from facial, tooth pulp, pharyngeal and laryngeal sites may converge on the trigeminal brain stem complex and underlie certain types of referred pain in the orofacial region.
2. Autonomic effects resulting in vasomotor and glandular symptoms (E.g. facial flushing with a dental abscess).
3. Motor effects resulting in trigger points and myospastic activity in segmentally related skeletal muscles. (E.g. masseter muscle pain associated with TMJ malfunction).

Etiology of Pain

Pain is therefore stated to have a multimodal etiology, some pathological processes that are commonly associated with pain are mentioned below:

Inflammation

Inflammatory pain is actually a reaction to the chemical inflammatory mediators of bradykinin and prostaglandins. These chemical mediators:

- Alter local receptor sensitivity and receptivity.
- Increase nociceptor sensitivity to stimulation.
- Higher-threshold mechanoreceptors may also become sensitized to a wider variety of stimuli.

Pain of inflammatory origin may involve different kinds of tissue innervated by receptors with different reactive responses. Superficial pain may be inflammatory in origin (gingivitis) where as visceral inflammatory pain may result from inflammatory changes in the adjacent tissues.

Musculoskeletal Pain

It is non-inflammatory in origin. It results from muscles spasm, it is reactive, and it is protective (e.g.) muscles splinting in a temporary protective mechanism resulting in muscle pain and weakness. Such splinting serves to rest the muscle until the symptoms have disappeared.

Vascular Pain (e.g. Migraine)

Display a characteristic emotional overlay with personality features of insecurity and tension.
Condition associated with vascular pain:

a. Vasodilatation and increased vascular permeability.
b. Tissue edema at the painful site.
c. Edema of the vessel wall and perivascular tissues.
d. Associated muscle pain.

Neural Pain

Cutting or crushing a peripheral nerve induces anesthesia due to afferent impulse interruption, and also including parasthesia, hyperesthesia, hyperanalgesia or spontaneous pain.

Chronic neural compression, however ultimately leads to myelin nerve sheath degeneration loss of the myelin sheath may result in impulses, traveling through the

demyelinated region passing to adjacent nerve fibers in a random manner often reflected as pain (e.g.) trigeminal neuralgia.

OTHER CAUSES OF PAIN

Pain may result from a variety of pathological events:

1. Spontaneous events without obvious cause.
2. Elevation of body temperature (e.g. fever).
3. A general response to abnormal allergic, emotional, endocrine, metabolic, or toxic conditions.
4. Direct noxious chemical, mechanical or thermal insults.

Categories of Facial Pain

It is classified into three:

1. **Somatic pain** results from stimulation of neural receptors or peripheral nerves. If superficial in origin its characteristics include;
   a. Bright pain with a stimulating quality.
   b. Accurate pain localization.
   c. Accurate correlation between site of lesion and pain source.
   d. Temporary pain abolition by topical anesthetic application to the site.

   If deep in origin:
   a. Dull pain, depressing in quality.
   b. Variable localization of diffuse pain.
   c. Site of pain may/may not correlate with site of lesion.
   d. Central excitatory effects.

2. **Neurogenic pain**
   Are generated within the nervous system itself; nerve receptor or nerve fiber stimulation is unnecessary.

Clinical Features

   a. Bright, burning pain.
   b. Good localization.
   c. Close correlation between site of pain and lesion.
   d. Accompaniment by other sensory, motor and/or autonomic symptoms.

3. **Psychogenic pain**
   May reflect somatic or neurogenic pain intensification or may be psychoneurotic manifestation.
   a. The site of pain often has no correlation with a possible cause.
   b. The clinical behavior and/or response to therapy may be non-physiological, unexpected (or) unusual.

4. Odontogenic pain (Tooth ache)
   A tooth is primarily innervated from the maxillary or mandibular division of the trigeminal nerve that passes essentially through the apical foramen. These nerve fibers are evident at the cap stage of odontogenesis. In the fully developed tooth some nerves terminate in the pulp proper, the majority pass towards the coronal pulpal walls and roof to form the subodontoblastic nerve plexus. Nerve fibers from this plexus then pass into the overlying odontoblastic and unmyelinated nerve fibers.

Thermal, osmotic, electrical, therapeutic and pharmacological stimuli result in pulpal nerve excitation, although

the degree of specificity remains obscure. In fact both myelinated (A) and unmyelinated (C) fibers pass through the apical foramen into the root canal and generally branch infrequently until the coronal aspect where they fan out towards the pulpal dentine border as Rashkow's plexus.

The density of plexus varies, tending to be greater, at the tips of the coronal pulp horns. From this subodontoblastic nerve plexus nerve fibers are distributed in the pulpodentinal border zones, with terminals showing a characteristic head like structure.

Some of the small nerve fibers in the odontoblastic zone are closely applied to the odontoblasts and others enter the predentine to pass through the dentinal tubules for variable distances. Some of these nerve fibers may however have an autonomic afferent rather than nociceptive afferent function. Also, there is little evidence that these fibers pass to the dentinoenamel junction. Thus although these nerve fibers may be associated with toothache, other mechanisms may also be responsible.

The odontoblastic transduction theory contends that a dental stimulus excites either an odontoblast or its process, which then transmits the excitation to adjacent nerve fibers. There is a close association between odontoblasts and subodontoblastic nerve plexus, in addition to the neural crest origin of the odontoblasts themselves.

The hydrodynamic theory contends that enamel or dentinal stimulation causes an outward or inward flow of dentinal tubular contents, the resultant disturbances being translated as pulpal nerve excitation. The innervation of the pulp includes both afferent neurons that conduct sensory impulses and efferent autonomic neurons that principally

provide neurogenic modulation of the blood flow in the pulp.

Myelinated axons associated with pain sensory function have the following properties:
1. Relatively fast conduction velocity.
2. Relatively slow stimulation threshold.
3. Convey impulses conceived as sharp and penetrating.

Pain sensory unmyelinated nerve fibers passing into the pulp tissue exhibit the following properties:
1. Relatively slow conduction velocity.
2. High stimulation threshold.
3. Convey impulses conceived as dull and lingering.

Pulpal stimuli results in neuronal activity in widely dispersed CNS regions include:
1. Trigeminal brain stem sensory nuclei.
2. Cranial nerve motor nuclei.
3. Cerebellum.
4. Reticular formation.
5. Ventrobasal thalamus.
6. Hypothalamus.
7. Somatosensory cerebral cortex.
8. Orbital cerebral cortex.

Pulpal stimulation thereby produces emotional, motivational and other behavioral changes in addition to the appropriate reflex responses. The innervations of tooth pulps exhibits marked variability with an increase in the number of myelinated axons from time of first eruption to sometime after eruption. This correlates with newly erupted healthy teeth being less sensitive than older healthy teeth.

With aging, however the number of axons entering a tooth generally decreases. This again correlates well with the clinical finding of reduction in pulpal sensitivity with increasing age.

Procedures for Control of Pain

1. **Pharmacological agents:** Local and general anesthetic agents, analgesic drugs (e.g. morphine).
2. **Therapeutic procedures:** Acupunctures, transcutaneous electrical stimulation, audioanalgesia, hypnosis.
3. Psychiatric counseling.
4. Neurosurgery.

All these methods are directed towards blocking pain transmission either at the periphery before the impulses enter the brain or within the brain.

CHAPTER 4

Pulp

INTRODUCTION

"Pulp is a soft tissue of mesenchymal origin with specialized cells odontoblasts, and number of tissue elements including vascular tissue, nerves, connective tissue fibers, ground substance, interstitial fluid, fibroblasts, immunocompetent cells, undifferentiated connective tissue cells and other cellular components".

The closed relationship between odontoblasts and dentin is referred to as the **(Pulp-Dentin Complex)** is one of the several reasons that why dentin and pulp should be considered as functional entity, certain peculiarities are imposed in the pulp by rigid mineralized dentin in which it is enclosed.

Pulp reacts to bacterial and other stimuli by inflammatory response. Certain anatomic features of this specialized tissue however tend to alter the nature and course of its response.

Thus, it is, necessary to know about the development, structure and function of the pulp-dentin complex in the hope that this knowledge will provide a firm biologic basis for clinical decision making.

ANATOMY OF THE PULP

General Features

- Pulp occupies the internal cavities.
- Pulpal outline corresponds to the general external outline of tooth.
- Pulp organs - 52.
- Total volume - 0.38cc.
- Average single pulp volume - 0.02cc.

Apical Foramen

- Size: 0.4mm - Maxillary.
- Size: 0. 3mm - Mandibular.
- Location and shape - changes as a result of functional influences.
- Provides entry for Nerves and blood vessels.
- Multiple foramina/accessory foramina (located twice the distance) of apical delta.

Accessory Canals

- Apical half/furcation area (Molars).

Radiographically

- It is composed of soft tissue.
- Appears radiolucent extending uniformly from interior of crown to apices of roots with great variations among individuals in size of pulp chamber and extent of pulp horns.
- Root canal may be apparent extending to root apex or may appear constricted in region of apex not discernible

in last 1 mm. Lateral canals exist just short of radiographic apex.
- At end of tooth development canal diverges and walls of root taper to knife edge.
- Developing root-divergent apex around dental papilla—enclosed by opaque bony crypt.

Embryologically

Pulp is derived from neural crest cells. Neutral crest arise from ectoderm along the lateral margins of neural plate and migrate extensively. The tooth papilla initially called as dental papilla. After dentin formation it is designated as pulp only.

In the earliest stages of development, neural crest cells is the area of proliferating future papilla, that causes oral epithelium to invaginate and forms enamel organ. These organs enlarge to enclose dental papillae in central position. The papillae further controls whether the forming enamel organ will be an incisor or a molar.

At the location of future incisor the development of dental pulp begins about the eighth week of embryonic life soon there after more posterior tooth organ begin differentiating as the cell density is great due to proliferation of cells within.

Young papilla is highly vascularized and well organized network of vessels appear by the time dentin formation occurs, and capillaries crowd among the odontoblasts during the period of active dentinogenesis. The cells of dental papilla appear as undifferentiated mesenchymal cells. Gradually they differentiate into stellate shaped

fibroblasts. After inner and enamel organ cells differentiated into ameloblasts, the odontoblasts then differentiate from peripheral cells of dental papilla and dentin production begins.

As this occurs the tissue is no longer called dental papilla but designated as pulp organ. Few large myelinated nerves are found in pulp until dentin of crown is well advanced. At that time nerves reach the odontogenic zone in pulp horns.

Gross Morphology of the Dental Pulp

An intact vital pulp may be removed from the root canal in one piece with barbed broach, on examination it was found-firm, cohesive and resilient unit, maintaining its original shape. This is because pulp is composed principally of gelatin like substance called ground substance, which is reinforced throughout by irregularly arranged and interlaced collagen fibres and fibre bundles embedded in its stroma are cells, blood vessels and nerve fibers that make up loose connective tissue categorized as 'Dental pulp organ'.

SPECIAL ENVIRONMENT OF DENTAL PULP

Connective tissue elements of pulp respond to changes in environment. The unique environment unyielding walls of dentin surrounding a resistant and resilient fiber reinforced ground substance makes dental pulp a special organ.

Pulp injury is frequently irreversible and painful because of certain restrictions in its environment:

1. Low compliance environment
2. Resilience of connective tissue
3. Ineffective collateral circulation.

Intrapulpal pressure in normal and pathological conditions given below:

Pulpal pathology	*Intra-pulpal pressure (mm Hg)*	*Nature of pulp*	*Clinical expression*
Normal pulp	10 (8-15)	Structurally Intact Resilient tissue Extripation in one piece	
Irreversible pulpitis	13	Intact vascular	Transitory pain, hyper active pulp requires stimulus
Irreversible (Painful pulpitis)	34.5	Intact Engorged Necrosis Abscess formation	Spontaneous pain persists after the removal of stimulus persistent response to cold stimuli
Suppurative Non-vital pulp	35.6	Fluid filled	Pain varies Heat sensitive Tenderness on percussion

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Pulpal pathology	*Intra-pulpal pressure (mm Hg)*	*Nature of pulp*	*Clinical expression*
Nonvital (dehydrated)	Unknown (negative pressure)	Dry granulated (mummy dust)	No response to vitality test Periapical radiolucency

HISTOLOGY

Dental pulp may be divided into three zones:

1. Central Zone: Pulp proper core of connective tissue containing the large nerves and blood vessels that begin to arborize towards peripheral pulpal areas.
2. Cell rich Zone: Area that is richly populated with reserved cells fibroblasts serves as reservoir for replacement of destroyed dentin producing cells. Observed in coronal pulp but can exist in radicular pulp.
3. Peripheral to cell rich Zone is sub-odontoblastic region of pulp, or **Zone of weil- cell free or cell poor zone**. This zone may diminish in size or disappear when dentin formation occur at rapid rate, or when reparative dentin present. 40 µm wide, abounds in nerve fibers that have lost myelin sheath which enter odontoblastic zone. The combined odontoblastic layer and sub-odontoblastic free network form a sensory complex (peripheral sensory unit).

Structural Elements of Dental Pulp

- Cells
- Intercellular substance
- Intercellular fluid.

Cells Fibroblasts

- Reserve cells
- Defense cells
- Special cells.

Fibroblast

Distributed throughout pulp (especially cell rich zone) produces gelatinous extra cellular matrix

- Young fibroblasts undergo mitosis to differentiate into replacement odontoblasts.
- Synthesized type I and type III collagen (responsible for collagen turnover),GAGs, proteoglycans.
- Stellate shape and extensive process that contact by intercellular junctions.
- Stain deeply with basic dyes, lighter stained cytoplasm appears homogenous.
- SEM-rough surface endoplasmic reticulum, mitochondria and other organelles.
- In young pulp cells divide and are active in protein synthesis.
- In older pulp cells appear rounded or spindle shaped with short process and exhibit few intracellular organelles→fibrocytes.
- In embryonic young pulp cellular elements predominate and in mature pulp fibrous component.
- Dual function.

Reserve Cells

- Found along capillaries and in cell rich zone.
- Larger than fibroblasts, polyhedral in shape with peripheral processes and large oval staining nucleic.
- Pluripotent cells.

Defense Cells

- Histiocytes or Macrophages.
- Mast cells.
- Plasma cells.
- Neutrophils (PMNs), eosinophils, basophils, lymphocytes and monocytes.

Macrophage

Irregularly shaped cell with short blunt process, small nucleus, rounded and darkly stained than fibroblasts, granular cytoplasm.

- In case of pulpal inflammation-exhibit granules and vacuoles in cytoplasm, nuclei increase in size exhibit prominent nucleolus.
- Associated with smaller blood vessels and capillaries.
- Ultra structurally exhibits rounded outline with short blunt process.
- Distinguishing feature is aggregation of vessels or phagosomes containing phagocytozed dense irregular bodies.

Lymphocytes and Eosinophils

These cells are found extra vascularly in normal pulp. During inflammation they increase in number. Mast cells seen along vessels in inflamed pulp, they have round nucleus and dark staining granules in cytoplasm. They increase in number during inflammation.

Plasma Cells

- Seen during inflammation of pulp.
- Small nucleus and concentric in cytoplasm.
- Chromatin adherent to nuclear membrane giving cart wheel appearance.
- Basophilic cytoplasm with light stained Golgi apparatus near nucleus.
- Densely packed rough endoplasmic reticulum.
- Mature cell has eccentric nucleus and abundant cytoplasm.
- Function in production of antibodies.

Dendritic Cells (Immunocompetent Cells)

- Antigen specific inflammatory reactions in pulp.
- Defense mechanism of pulp dentin organ.
- Lymphocytes - Recognize antigens.
- Dendritic cells - Capture antigens - presenting them to lymphocytes.
- Few associated with macrophages, also intact with nerves and vessels in pulp.
- Primary Immunosurveillance system.
- Regulating blood flow.

Metabolism

Metabolic activity can be described as measuring the rate of oxygen consumption and production of CO_2 or lactic acid by pulp tissue (Radio spirometry method/pulp oximetry).

- Sparse cellular composition of pulp low rate of O_2 consumption.

- High metabolic activity during active dentinogenesis, odontoblastic layer.
- Produce energy through glycolytic pathway, pentose phosphate shunt.
- ZOE, Amalgam, $Ca(OH)_2$ decreases O_2 consumption
- Orthodontic application decrease respiratory activity.

Odontoblast

These odontoblasts layer composed of cell bodies of odontoblasts.

- Highly specialized connective tissue cell of pulp.
- Plays a role in dentin and pulp function.
- Morphologic variations occur from tall columnar cell in crown to low columnar in midroot:
  - In root portion, cells are shorter and cuboidal.
  - Towards apex, cells are flattened like fibroblast.
  - In columnar region, they elaborate regular dentin and tubules.
  - In mid portion, cells are few and less regular.
  - In apical portion, cells are less differentiated, less tubular, more amorphous dentin.
- Intercellular space 200-300A°.
- Under SEM (scanning electron microscopy) nucleus **appears** ellipsoidal, contains chromatin and nucleolus surrounded by two thin membranes, extensive rough endoplasmic reticulum, well developed centrally located in Golgi complex, numerous mitochondria and numerous granules and vesicles.

Dentinoblasts forms palisaded arrangement of pulp in a single row at entire periphery of pulp; a compound pseudo stratified layer is present in pulp horn region due to

crowding of cells in narrowing lumen. They are in close physical contact with each other but also communicate via junctional complexes.

- If one odontoblast is injured others are immediately affected. They do not undergo mitosis (post mitotic cells).
- When they die, matrix secretary function is carried out by neighboring odontoblast or new cells that arises from pluripotent cells.
- Odontoblastic layer demarcated from dentin by hyper chromatic line at pulpo dentinal membrane. (An indication for close contact of cells at dentinal border).
- Dark dots or dense bars at these junctions are called terminal bars. These have several components like:
  - Zona occlusions
  - Zona adherence
  - Macule adherence
- Disruption of this membrane is evidence of pathologic change. It corresponds to point with which adjacent cells are attached. Modified junctional complex and its cytoplasmic fine filaments called terminal web—it picks up additional stain.

Junctional complex (Intercellular junctions) are classified as:

- Impermeable Junctions: Tight Junction, Zona occludens.
- Adhering Junctions: Intercellular bridges modified desmosomes.
- Communicating Junctions: Nexus type gap junctions.

Odontoblastic Process

Odontoblasts extend their cytoplasm and plasma membrane into dentinal tubules as odontoblastic process (Tomes fiber).

- Odontoblastic process completely fills the tubule at dentin predentin junction but farther into dentin separates from tubule wall. The Peri-odontoblastic space contains granular material and collagen fibers.
- It is larger at pulpal end than at periphery (4:1), circular in cross section.
- Average diameter of odontoblastic process is 2mm and average length is 2 mm.
- Cytoplasm is devoid of major organelles.
- Predominant structure—Microtubules (200-250A) Transport system for peri tubular dentin matrix.
- Vesicles present at periphery-contains transported matrix to be deposited at wall.
- Peri odontoblastic space is bathed by intercellular fluid form dental pulp.

Intercellular Components

- Collagen fibers
- Amorphous ground substance
- Intercellular tissue fluids.

Collagen Fibers

Type I, II, III- Interstitial collagen
IV- Basement membrane
Pulpal collagen—Mixture of type I and type III
Dentin collagen—Type I

Developing young pulp is smaller in length and diameter (reticular, argyrophilic, precollagenous) 100-120A°
Mature collagen—Increase in length and diameter 400-700A°.

- Larger—made up of several bundles.
- Show 640A° wide cross striations.
- Less likely to be destroyed and removed.
- Crabgrass effect (Fibrosis).

Ground substance contains mucopolysacchrides (complex protein carbohydrate compounds)

- Glycosaminoglycans or proteoglycans.
- Nonsulfated type mucopolysacchrides—hyaluronic acid is predominant in pulp.
- Sulfated type—chondroitin sulfate (A, B, C).
- Viscosity varies from slightly viscid fluid to soft gelatin like material.
- It is miscible with intercellular fluid.
- It is hydrophilic in nature, i.e. passage of water soluble and waste metabolite.
- Reinforcement with collagen fibres makes it rigid.
- Change in state of polymerization influence spread of inflammation and infection.

Van Hassel

- Viscosity of ground substance reinforced with collagen doesn't permit fluid to move easily from one part to another or pressure to be transmitted through out.
- Sets as barrier against microorganism.
- Inflammation moves apically by increments from a particular site.

- Chemical mediators (proteolytic enzymes, hyalurodinase, hydrolytic enzymes) edema, heat alter the ground substance by depolymerizing the molecules. Chemotactic factors, chemicals liberated from injured cells alter viscosity and effect PMN's and macrophages.
- Aging pulp has less bound water. Tissue is dehydrated and more viscous. O_2 and metabolites have difficulty traversing the tissue it diminishes the ability to defend, heal or repair.

Systemic Factors affecting Pulp

- Vitamin deficiency—fibroblasts become smaller, reduced number of intra cellular organ cells.
- Hormones and Hormonal imbalance; systemic steroid therapy—affects odontoblasts and reparative dentinogenesis.
- Diabetes affects the tissue repair, increase of glucose concentration in dentinal pulp fluid, degenerative inflammatory changes, and dentinogenesis affected.
- Thyroid deficiency decreases the vascularity of pulp, hyper mineralization of bone and dentin.
- Protein deficiency leads to decreased cellular elements interference with dentinal repair.
- A systemic virus infection causes injury to odontoblasts.
- Systemic manifestations are degeneration, necrosis, infection of pulp.

Functions of Pulp

Basic Function

1. Inductive: induces oral epithelial differentiation into dental lamina and enamel organ formation. Induces

developing enamel organ to become particular tooth or to acquire a particular shape.

2. Formative: Dentin formation throughout life at different rates.
   - Initial or primary dentin is tubular, regularly arranged.
   - As functional strain increase, dentin formation increases.
   - As dentin matrix gets secreted, odontoblasts retreat towards the pulp centre, they become more wavy and few tubules per unit area are formed (functional secondary dentin)
   - Environmental stimulation produces atypical dentin, i.e. it has irregular or absent tubules and can be defined as reparative, irregular, defensive or more accurately irritation dentin.
   - Severe trauma may activate forming cells leading to obliteration of canal– traumatic dentin (irritation).
   - Osteodentin- matrix is laid down so rapidly it entraps cells, tissues giving bone like appearance.
3. Nutritive function: Rich peripheral capillary network Provide oxygen and nutrients to dentinoblasts and their process and is continuous source of dentinal fluid.
   - Water soluble substance, components of plasma filter across capillary wall.
   - Pulp chamber vary from 2-5 mm at widest portion and arterioles enter foramina 0.1mm diameter.

Arteries and veins—Branches of superior and inferior alveolar vessels.

- Branch at all levels greater in coronal pulp. In multi-rooted teeth rich anastomoses is seen.

- No collateral circulation.
- Greatest capillary concentration in sub-dentinoblastic area to satisfy functional demands of large cellular population in neighboring dentinoblastic and cell rich zone (TCN).

Blood vessels are small, thin, delicate, largest arterial vessel— 50-100 μm characteristic layers - Outer (Adventitia).

Thin smooth muscle cell bed - tunica media
- Endothelial lining of flat cells - intima.

Terminal Arteriole

- Endothelium and spiraled smooth muscle (media) cells around supporting adventitia.
- Contraction of smooth muscle control the diameter of arteriole.
- Arterio venous anastomoses (AVA) - more in apical half that maintains the blood flow (decrease intrapulpal pressure).

Capillary structure: No tunica media or adventitia single layer can be seen. Flat endothelial cells continuous with lining arterioles and venules are surrounded by reticular and collagenous fibres.
- Fenestrations are present between cells.
- Selective permeability, fluid inter change, can lead to edema and increased intra pulpal pressure.
- Not innervated.
- Highest concentrations of loops capillary in peripheral plexus (TCN).
- Efferent venous system coalease into thinner venules and are more delicate.
- 50 um lumen surrounded by intima, thin media scanty or absent adventitia.

Lymph vessel—drained by regional lymph nodes – submaxillary and submandibular.

Carious teeth—removes fluid reduces pressure, resolve initial inflammation.

Intra Pulpal Pressure

Nature of turgid gelated ground substance limits pressure.

Increase in intra pulpal pressure not generalized response but a local phenomenon.

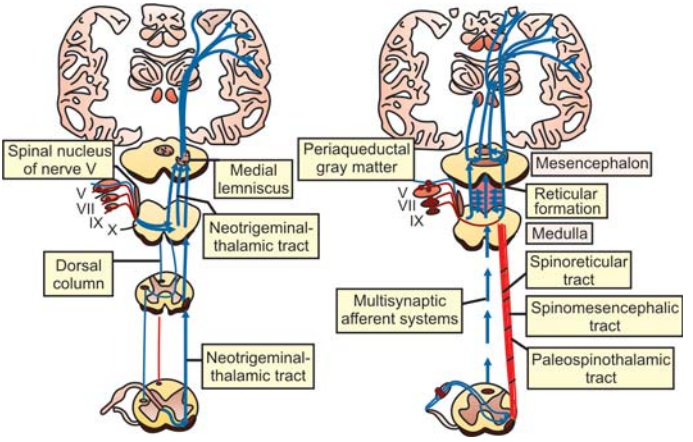

Fig. 4.1: The afferent efferent pathway

Theories of Pulpo-Dentinal Pain Fiber Excitation

1. Classical theory – Dentin innervation
2. Modified theory – transduction theory
3. Hydrodynamic theory

CLASSIFICATION AND FUNCTIONS OF FIBERS IN PERIPHERAL NERVES

Fiber	*Diameter (um)*	*Function*
Aα	6-20	Afferent fibres for touch pressure, proprioception, vibration
Aβ		(Mechanoreceptors)
Aγ	5-12	
Aδ	1-5	Afferent for pain and temperature visceral afferent fibers
B	1-3	Preganglionic visceral efferent fibers
C	0.4-10	Afferent fibrous for pain and temperature. Postganglionic visceral efferent fibers

PAIN FIBERS IN PULP (NOCICEPTIVE)

	A (δ) fibers	*C Fibers*
Diameter (um)	2-5 (measure of parent fibre)	0.3-1.2 same diameter through their length
Conductive velocity (m/sec)	5-10	4-2
Location of terminals	Superficial-terminals in odontoblastic, sub odontoblastic zone	Near blood vessels throughout pulp

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	A (δ) fibers	C Fibers
Pain characteristics	Sharp, pricking, unpleasant but bearable (fast, momentary)	Throbbing, aching less bearable, extremely unpleasant
Stimulation threshold	Low—does not take much to fire an impulse can be stimulated without injury	High stimulation—intense to damage tissue associated with inflammatory process (exudative lesion tissue damage)

RESPONSE OF A, B AND C NERVE FIBRES TO SPECIAL STIMULUS

Stimulus	*A-δ (Low threshold)*	*C (High threshold)*
Intra pulpal pressure changes	Increase response	Increase response
Sudden nerve Compression Prolong nerve	Decrease response (block Impulse)	Resistant to increase pressure (impulse continue)
Vitality test Electric	Positive (immediate)	Negative (except at high stimulus)
Cold	Positive (immediate)	Negative (no effect)
Rapid heat	Immediate : 1st response (Sharp, localized)	Delayed ; 2nd response (dull, radiating)
Slow and sustained heat	Negative	Positive (after 45-47 °C)
Resistance to O_2 depletion Hypoxia/Anoxia	Decreased fiber activity (Short survival time in pressure necrosis)	Fiber activity resistant to depletion (long survival time)

Pain Associated with

- Dry functional heat—cavity preparation dehydrates dentin and frictional stress pulls out the fluid—stretch odontoblasts and nerve terminals into tubules. Cavity should be kept moist with spray and compressed air.
- Sweets and candy bars: Sugar and hypertonic solution create osmotic gradient fluid movement from deeper areas of less concentration. There is initial sharp pain of Aδ fibers and if concomitant inflammation in pulp tissue-dull aches due to C fibers).
- Defective or loose margin: Fluid moves toward pulp due to plunger action against tubule orifice.
- Thermal stimulation: Causes movement of fluid with in tubule because of its 10 times greater coefficient of expansion than wall of tubule.
- Expansion and movement towards pulp—if closed tubule at outer surface and if open it occurs away from pulp—over flows its aperture.

Response Faster to Cold than Heat

- This is due to the rapid movement of the outward fluid.
- With heat large volume of dentin should be affected before pronounced dislocation of tubule content.
- In normal pulp—no pain—if present it is dull, longer duration due to vasodilatory effect.

Outward Flow can be Minimized by

- 5% potassium nitrate, 10% strontium chloride, potassium nitrate and sodium monoflurophosphate.

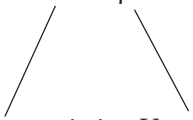
- Potassium oxalate (30%) forms Ca oxalate crystals and block hydraulic conductance by 98%.
- Smear layer decrease permeability, decrease fluid flow.
- Decrease nerve response by neurally acting agents like potassium salts and eugenol.

Reflex Phenomenon

Efferent response (effect to return impulse): Electric or chemical methods cause decrease blood flow and vasoconstriction. Activation of cervical sympathetic nerve fibers in arterial adventitial layer penetrate tunica media at NMJ (neuromuscular junction).

At NMJ (neuromuscular junction) they release norepinephrine (NE) and cause constriction of arterial lumen thus causing decrease flow of blood.

Pulp has α and β adrenergic receptors in arteriolar vessels



Vasoconstriction Vasodilatation

Humoral substance like catecholamine, prostaglandins, substance P, and bradykinin causes vasoconstriction and dilation. Vasodilatation can be due to reflex phenomenon following vasoconstrictions which is mediated by tissue metabolites.

Pathosis of mild/Moderate/ Normal condition	**Pathosis of Strong/ Sustained conditions**
Vasoconstriction action of NE at NMJ neuromuscular junction is inactivated and neutralized by	Excessive efferent impulse of smooth muscles of arterial vessel go in prolonged contraction
↓	↓
Accumulated catabolic products Lowering pH, decrease energy source	Great accumulation of catabolic products, decrease pH, depletion of energy source
↓	↓
Relaxes blood vessel smooth musculature	Muscle relaxation extended passive dilation
↓	↓
Vasodilatation, increase rate and increased volume of flow	Increase blood volume (hyperemia) in pulp Capillary gets engorged Decrease flow, vascular congestion Inflammation (pulpitis) intra pulpal pressure increases
↓	
Removal of waste products (O_2, H_2O)	
↓	
Replenish O_2 and energy source	

Role of Neuropeptides

Pulpal nerve fibers contain neuropeptides, such as calcitonin gene related peptide (CGRP), substance P (SP), neuropeptide Y, neurokinin A and vasoactive intestinal polypeptide.

- Release can be triggered due to tissue injury, compliment activation, antigen antibody, reaction or stimulation of inferior alveolar nerve.
- They produce vascular changes; SP and CGRP contribute to hyperalgesia and promote sound healing.
- Mechanical elimination and electrical stimulation of dentin produces vasodilatation within pulp – causing releasing of neuropeptides from inter dental sensory fibers.

Plasticity of Inter Dental Nerve Fibers

- Number, size and cytochemistry of fibers changes due to aging, tooth injury and caries.
- Nerve fibers sprout into inflamed tissue. Content of CGRP and SP increase their fibers.
- Inflammation subsides – decrease in member of sprouts.
- Regulation of these changes by NGF found on intra dental sensory fibers schwann cells.
- Synthesized by fibroblasts in cell rich zone particularly in tip of pulp horn.
- Maximum sprouting corresponds to area with increased production of NGF (Carious lesion).

Defense Function

Dentinoblasts when injured or irritated contribute to one or more defense function:

1. Dentinal pain – theories of excitation
2. Formation of smear layer
3. Tubular sclerosis.
4. Irritation (reparative dentin)
5. Inflammation of subjective connective tissue

Regulation of Pulpal Blood Flow

Occurs by nerve impulses and chemical regulation. Sympathetic (Adrenergic) nerve fibers releases nor epinephrine which cause vasoconstriction (α and β adrenoreceptors).

- Parasympathetic (Cholinergic) Nerves release acetylcholine causing dilation (β adrenergic)
- Axon reflex vasodilatation
- Electric stimulation of sympathetic nerve fibres to pulp cause decrease in blood flow detected by laser doppler flowmetry.

Rates of Blood Flow

- Blood flow of coronal pulp is twice as much as that of apical half.
- Coronal region—70 ml/min/100 g of pulp flow.
- Core region—15 ml/min/100 g.
- Shunting occurring in apical half—because of numerous shunts and U turn loops.

CLASSIFICATION OF PULPAL PATHOSIS

Ingle's classification	*Seltzer and Bendar (Histologic classification)*
1. Hyper reactive pulpagia • Hyper sensitivity • Hyperemia	1. Intact pulp with scattered chronic inflammatory cells (chronic pulpitis—incipient form)
2. Acute pulpalgia incipient (Reversible) moderate (referred) advanced (relieved by cold)	2. Acute pulpitis 3. Chronic partial pulpitis with partial necrosis 4. Chronic total pulpitis with liquefaction necrosis
3. Chronic Pulpalgia 4. Chronic Pulpalgia 5. Hyper plastic pulpitis 6. Pulp necrosis 7. Atrophic pulposis 8. Calcific pulposis	5. Chronic partial pulpitis (Hyper plastic form) 6. Pulp necrosis 7. Atrophic pulp 8. Dystrophic mineralization

Role of Dentinoblasts

Changes as injury increases progressively:

1. Increase permeability of dental tubules.
2. Disturbance of pulpodentinal membrane – aggregate break down of odontoblasts.
3. Disruption of palisaded odontoblastic layer, many odontoblasts affected during the stimulus.

4. Aspiration of odontoblastic nucleic into dentinal tubules.
5. Irreversible odontoblastic injury –release of tissue injury factors – hydrolyze cells and intra cellular substance.
6. Inflammatory changes in odontoblastic zones.
7. Sub odontoblastic inflammatory changes – Vasodilatation, leukocyte infiltration, edema.
8. Central zone inflammation reflects circumferential and apical progression and irreversible total pulpitis.

HISTOPATHOLOGY OF INFLAMMATION

	Mild reaction	*Moderate*	*Severe*
No. of cells	Increase in cell free zone	More cell	Marked infiltration, abscess formation
Type	Fibroblast, undifferentiated cells, few inflammatory cells	Neutrophils mononuclear cells	Neutrophils mononuclear leukocytes
Odonto blastic layer		Altered Pseudo stratified nuclei in tubules	Altered
Capillaries	Extravasated blood vessels	Increase	Numerous
Response	Localized to affected area	Localized	Localized to affected tubules

ULTRA STRUCTURAL OF PULPAL INFLAMMATION

Immediate	(15-60 min.)	Displacement of odontoblastic nucleic disturbance in sub-odontoblastic tissue
Post operative	(6 hours)	Engorged blood vessels, leukocytic, exudates Degenerative changes in odontoblastic layer dilated RER, swollen mitochondria Deterioration in odontoblastic layer
	(12 – 24 hours)	Necrosis Phagocytosis Degenerative changes in odontoblasts
	(4 hours)	Elongated cells, polarization of nucleus New or secondary odontoblasts Macrophage active at vital tissue border Collagen synthesis
	(3 – 8 days)	Healing phase Continued collagen synthesis Mineralization of inter tubular matrix

Healing is less in fiber rich than cell rich pulp.

DIFFERENTIAL DIAGNOSIS OF POTENTIALLY REVERSIBLE AFFECTED PULP

	Potentially reversible	*Probably irreversible*
Pain	Momentary: dissipates readily after removal stimulus (Cold, Aδ)	Continuous: Persists minutes to hours, presence of internal irritant, C fiber. Throbbing: arterial pulsation area of increase intra pulpal pressure
Stimulus	Requires external stimulus (cold, heat, sugar)	Spontaneous: does not require injured tissue in chambers or canal Intermittent: Spontaneous pain of short duration
History	Recent dental procedure cervical abrasion or erosion	Extensive pulp capping, deep caries, trauma, etc.
Electric	Premature response negative	Premature, delayed, mixed respond in advance stage (with acute apical periodontitis)
Referred	Negative minimally affected pulp tissue, short duration of pain	Common

Contd...

Contd...

	Potentially reversible	*Probably irreversible*
Lying down	Negative minimally affected pulp tissue	Common - increase cephalic pressure increases already excessive intra pulpal pressure
Color	Negative	May be present due to tissue lysis and intra pulpal hemorrhage
Radio-graph	(Restoration caries), periodontal pocket, cupping of alveolar crest periapex negative	Restoration, caries Periapex – widening of PDL

Acute Inflammation: Effects of Nerve Factors with Tissue Injury Factors

- Immediate and transitory pain perception.
- Vasodilatation.
- Increase pressure.
- Secondary pain response.
- Mechanism of pulp and periapical exudative (acute) inflammatory response.
- Vascular changes at injury site due to cellular and neurogenic response.

Retrogressive and Age Changes of Dental Pulp

1. Decrease in cellular components.
2. Dentinal sclerosis.
3. Decrease in quality and number of blood vessels and nerves.
4. Reduction in size and volume of pulp due to secondary and reparative dentin formation.

5. Increase in number and thickness of collagen fibers.
6. Increase in pulp stones and dystrophic mineralization.
7. Decrease in cellular components – due to decrease circulation regression of fibroblasts and odontoblasts (age).
8. Increase peritubular dentin- (Sclerosis) cell mediated age changes in apical third of root with aging, odontoblast reduce in numbers and disappears.
   - Dental caries (defense mechanism).
   - Erosion, abrasion, attrition.
9. Dead tracts in primary dentin – rapid caries – odontoblasts degenerate dentinal tubules devoid of living protoplasm.
10. Remaining odontoblasts cells seal these tracts by elaboration of reparative dentin.
11. Not highly mineralized as sclerotic dentin – more progression of caries.

Blood Vessels

- Arteriosclerotic changes – decrease blood supply hyperplasia of intima narrowing of lumen and elastic fibers, fine mineral deposits –complete obliteration of media and adventitia.
- Blood vessels decrease in number.

Nerves

Mineralization of nerve sheath and nerve itself decreases its branching in coronal pulp. Under caries, fibers become coarsened, irregular and argyrophilic varicosities formed.

Formation of Secondary Dentin

- **In molar—**at floor of pulp chamber, less on occlusal and lateral walls.
- **Upper anterior**:
  1. Lingual wall of pulp chamber.
  2. Incisal tip and then in wall of chamber.
- Irregular, few tubules.
- Shrink in occlusal radicular direction than mesio-distally.
- Horns also recede but less as tissues.

Formation of Reparative Dentin

- More amorphous, less tubular, less regular than primary dentin.
- Severe injury with inflammatory response – rapid reparative dentin.
- Found when involved dentin tubules – caries, attrition, abrasion, restoration.
- Hyper mineralized or Hypo mineralized zone present less abundant.
- Under caries:
  - More regular than under restoration.
  - Tubular structure more described.
  - Palisade fashion arranged odontoblasts.
- Rampant caries: Decayed reparative dentin as readily as primary
  - Marked off by a calci traumatic response.
  - Destroy odontoblastic layer.

Inflammatory cells (macrophage and lymphocyte) seen under reparative dentin in pulp.

- Under restoration:

More amorphous and irregular softer than primary dentin average daily reparative dentin formation

– 2.8 um – deciduous.
– 1.5 um – permanent (fischer et al).
– Quality – depend on depth of cavity.

Deep Cavity

- Lag period in onset of pre dentin and then in huge amount under the tubules, change in odontoblast regular palisaded arrangement.
- Cells are flattened in coronal portion and reduce in number.
- Calci traumatic response.
- Pulp undercut tubules show chronic inflammatory cells.

In Root Canals

Present in significant amount—in chronically inflamed and periodontally involved teeth. Excessively narrowed and almost obliterated.

Atrophy (Pulposis)

Advance Age

- Increase collagen fibers because decrease number of cells.
- Increase resistance of proteolytic enzyme, decrease in collagen solubility, water content, chemical reactivity.
- Decrease ratio of ground substance to collagen.

From Caries and Operative Procedure

- Decrease in size and number of cells.
- Pulp underlying large areas of reparative dentin seems to be burned out, coagulation necrosis.
- Dystrophic mineralization in coronal and radicular portion.
- Mineralization of blood vessel and perineural sheath.

From Periodontal Disease

- Small in size few in number.
- Impairment of nutrition supply.

Dystrophic Mineralization

Aging

- Ground substance alteration (decrease reactivity, increased less soluble macromolecules).
- Smooth spherical clusters packed around collagen fibers or intracellular debris in fibroblasts.
- Patterns as typical of hydroxyapatite crystals.
- Nucleus is deposited, further mineralization occurs by secretion.
- Occurs in mucopolysacchrides content of sheath around fibrils (sulfated).
- Seen in nerve sheath, vessel walls.
- When extripated feels wooden hard.

Due to Caries, Periodontal Disease and Trauma

- Caries – increase in coronal dystrophic mineralization.
- Chronically inflamed pulp – mineralized in region of liquefaction necrosis.

- Cells (odontoblasts, fibroblast, endothelial cells) show Smooth and folded surface – therefore interfere with exchange of nutrients – degenerate and become hollow.
- Fibers have beaded appearance; minerals found near degenerating fibers they coalesce to form large masses.
- It resembles cementum, bone, and dentin and clinically appears as yellowish hue compared to normal tooth.

Denticles (Pulp Stones)

Large mineralization may cause complete obliteration of pulp chamber or root canal.

HYPERACTIVE PULPALGIA

1. Hypersensitivity
2. Hyperemia
3. Acute pulpalgia
4. Chronic pulpalgia
5. Hyperplastic pulpitis
6. Necrotic pulp
7. Internal resorption
8. Traumatic occlusion
9. Split tooth or incomplete fracture.

Hyperactive pulpalgia is characterized by short, sharp shock. It is never spontaneous. Pain is of short duration even if irritant is there - may be cold or hot, sweet or sour. Ice cream excites the nerve in teeth and pain is referred to eyes.

Three different types of sensitivity of heat is noticed:

- A pulsating type or response in which discharge of fiber is synchronized with heart beat.

- Transient type of pain when nerve fiber is excited by heat over 43°C.
- Long lasting type of response started over 45°C.
- Sensation continues once temperature even comes down.

Fluids have a considerably greater coefficient of expansion than solids. A sudden rise of temperature 20°C at outer one-third may give rise to a sudden pain.

Hyperactive pulpalgia is common following the placement of new restoration. Patients also complain this after curettage. It may be associated with caries of tooth. Fractured teeth are more hyper reactive. In maxillary sinusitis maxillary teeth are involved.

Hyper reactivity is divided into two; hypersensitivity and hyperemia.

HYPERSENSITIVITY

The initiating factors of a hypersensitive pulp are usually cold food or drink or cold air or stimulation of exposed dentin on the root surface by cold, sweet or sour substances. Fruits and salts may also result it. The sensation disappears as soon as electrolyte is diluted away or metal is removed. Actually pain can be evoked from dentin by applying to it solutions which exert high osmotic pressure.

HYPEREMIA

As regards hyperemia all minor pulp sensations were at one time thought to be associated with hyperemia. Hyperemia is an increased blood flow in the pulp. The increased pressure against the sensory nerve endings in

the pulp produce the sensation associated with hyperemia. It will explain why the pain appears to be of different intensity and character with application of cold or heat, the cold producing hypersensitivity response and the heat producing true transient hyperemia.

Intra pulpal pressure decreases when patient has fell asleep and increases when awakened. Best treatment for hyperemia lies in its prevention. The insulating base under metallic restorations materially reduces most of hypersensitivity. Sensation usually diminishes gradually as irritational dentin builds to protect the dental pulp. Commercial toothpastes are available to reduce sensitivity. These contain either formalin or a strontium chloride preparation.

ACUTE PULPALGIA

Acute pulpalgia develops with the development of pulp inflammation or pulpitis. Increased intra pulpal pressure is the stimulus that is applied to the sensory nerves of the pulp and leads to severe toothache.

The mild discomfort after anesthesia wears off, following cavity preparation is a good example of incipient paraplegia. Teeth feel different.

Removal of the carious lesion followed by sedative cement for a few days may be all that is required to arrest incipient acute pulpalgia. Corticosteroids placed in the cavity following preparation or used on the dentin Surface prior to cementation of extensive restorations has proved effective for reducing postoperative pain.

Moderate Acute Pulpalgia

The pain is a true toothache. Pain is of nagging or of boring type. Pain to start is localized and later becomes diffuse or referred to another area. Pain does not resolve once the irritant is removed.

Moderate pulpalgia may start spontaneously from such a simple act as that of lying down. Hot food or drink excites the pain. Warm water will not relieve the pain and cold water sometimes makes it worse.

If the pain is constant for some time all the pulps on the affected side seem to ache and frequently two or three give the same response to the thermal testing. Radiograph may give immediate clue in the form of huge interproximal cavity or a restoration impinging upon a pulp chamber.

Treatment includes pulpectomy and endodontic therapy. One should try to save tooth.

Advanced Acute Pulpalgia

This pain is very severe the most excruciating acute pain known to man. Patient may even become hysterical from pain. Relief from this pain is embarrassingly by simple cold water.

The history is self-incriminating. Symptoms are violent. The involved tooth usually can be pointed out by patient and is tender to percussion too. Because the inflamed pulp reacts so violently to heat, the most decisive test is the heat test. As soon as not gutta-percha touches the involved tooth, the patient develops sub gluteal vacuum and patient rises up in the chair as if stabbed. Cold water is instantly applied and pain subsides. Local anesthesia gives blessed relief.

CHRONIC PULPALGIA

Patient tolerates grumbling pain for years. Often the pain can easily be kept under control with anti inflammatory.

The pain is diffuse and patient may not locate the cause. The pulp involved is not affected by cold. Pain persists till irritant is there. These are the lesions which cause pain until irritant is dislodged.

Radiograph often reveals interproximal or root caries or recurrent caries under a restoration. The apices of involved roots also show external resorption. Thermal tests are of little value in a positive sense.

Pulp extirpation and endodontic therapy if the tooth is to be saved, otherwise extraction is the line of treatment.

HYPERPLASTIC PULPITIS

The discomfort of a hyperplastic pulp results from compressing food against the exposed pulp during mastication. As regards treatment teeth involved in hyperplastic pulpitis are badly decayed, and restoration is virtually very difficult.

Necrotic Pulp

There are no true symptoms of complete pulp necrosis for the simple reason that pulp is destroyed along with its sensory nerves. Many cases of pulp necrosis are discovered because of discoloration of crown. This applies primarily to anterior teeth.

The radiography may be helpful if a periapical lesion exists for its presence and usually indicates associated pulp death. On radiograph necrotic pulp may exhibit only slight

periapical change. The tooth with a necrotic pulp may also be slightly painful to percussion.

INTERNAL RESORPTION

Internal resorption is an insidious process when the afflicted pulp is completely symptom free. Symptoms of internal resorption depend primarily on whether the process had broken through the external tooth surface. The pulp that erodes through the root surface may give vague pain on mastication. Percussion may be of little value pulpectomy is the only treatment for internal resorption.

TRAUMATIC OCCLUSION

A tooth traumatized by bruxism or traumatized because a restoration is in hyper occlusion often responds. Pulp is usually hypersensitive reactive primarily to cold.

The patient may complain of discomfort upon awakening in the morning or possibly of being awakened by the discomfort.

SPLIT TOOTH OR INCOMPLETE FRACTURE

Split tooth or incomplete fracture is a tooth that is split or cracked but not yet fractured. The tooth may be uncomfortable only occasionally during mastication. Pain may be like unbearable stab. This is when there is crack in the dentin and suddenly spreads as the cusp is separated from the remainder of tooth. Pulp may remain hypersensitive for years. Most frequent complaint is of pain while biting or contracting cold fluids. If the pulp is involved in fracture any exciting agent will bring on discomfort.

CHAPTER 5

Dental Caries

Dental caries is a widespread, chronic, infectious disease experienced by almost 80 percent of children by the age of 18 and by more than 90 percent of adult or in another way; it is the most prevalent chronic disease affecting the human race. Once it occurs, its manifestations persist throughout life even though the lesion is treated. There are practically no geographic areas in the world whose inhabitants do not exhibit some evidence of dental caries. It affects persons of both sexes in all races, all social-economic traits and every age group. It usually begins soon after the teeth erupt in the oral cavity. Persons who never develop carious lesions are designated "caries free. No satisfactory explanation of their caries resistance has been found.

Hundreds of dental research investigators for more than a century have studied various problematic aspects of dental caries. Despite this extensive investigation, many aspects of etiology are still under observation and efforts at preventions have been partially successful.

Hence dental caries can be defined as a microbial disease of the calcified tissues of the teeth, characterized by demineralization of the inorganic portion and destruction of the organic substances of the tooth resulting in cavitations.

Dental caries can occur on any aspect of the tooth but the surfaces which are more prone to the accumulation of plaque and bacteria, are the one which are more susceptible to caries. As this closely knitted plaque has a very complex structure and harbors variety of microorganisms.

Dental caries may occur in any area where plaque and food can adhere to the tooth. Certain bacterias found in mouth can break down sugars and starches and create an acid capable of destroying tooth enamel. So, formation of cavity requires acid producing bacteria, substrate, plaque, and host susceptibility factor (Fig. 5.1).

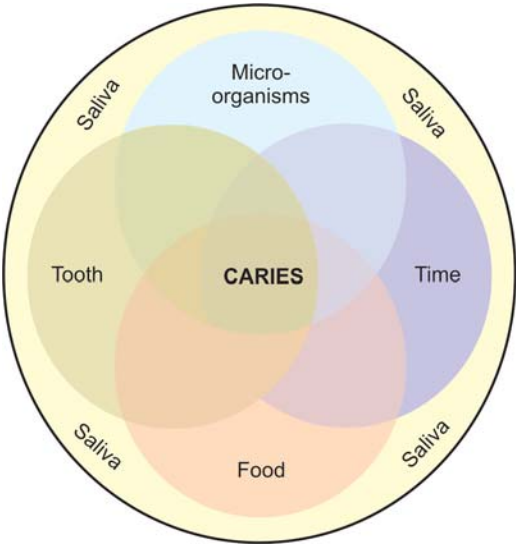


Fig. 5.1: Factors essential for the development of caries

Untreated caries may progress through the enamel and dentin thereby causing inflammation and death of dental pulp. Caries involving dental pulp can also cause intense pain. Decayed teeth that involve the dental pulp is a potential source of infection in human body. Dental examination for caries cannot be considered complete without radiographic examination.

EARLY THEORIES OF CARIES

Worms

According to the ancient Sumerian text, toothache was caused by a worm that drank the blood of the teeth and fed on the roots of the jaws. This legend of the worm was discovered on one of many clay tablets excavated. Niffer, Ur, and other cities within the Euphrates valley of the lower Mesopotamian area and estimated to date from about 5000 BC. The idea that caries is caused by a worm was almost universal at one time, as evidenced by the writing of Homer and popular lore of China, India, Finland, and Scotland.

Guy De Cahuliac

Guy De Cahuliac (1300-1368), was the greatest surgeon of Middle Ages believed that worms caused dental decay. He advocated fumigation with seeds of leek, onion and hyoscyamus as a cure for dental caries. The fumigation was also used in earlier times by Chinese and Egyptians.

Humors

The ancient Greeks considered that a person's physical and mental constitution was determined by the relative

proportions of the four elemental fluids of the body - blood, phlegm, black bile, and yellow bile which corresponds to the four humors – sanguine, phlegmatic, melancholic and choleric. All diseases, including caries could be explained by an imbalance of those humors.

Vital Theory

This theory considered dental caries as originating within the tooth itself, analogous to the bone gangrene. At the end of eighteenth century this theory remained dominant until the middle of the nineteenth century. A clinically well known type of caries is characterized by extensive penetrations into the dentin and even into the pulp, but with a barely detectable catch or a fissure.

Chemical Theory

Parmly (1819) rebelled against the vital theory and proposed that an unidentified "chemical" agent was responsible for caries. Support for the chemical theory came from Robertson (1835) and Regnart (1938) who actually carried out experiments with different dilutions of organic acids (such as sulphuric and nitric) and found that they corroded enamel and dentin.

He stated that caries began on the enamel surface in locations where the food putrefied and acquired sufficient dissolving power to produce the disease chemically.

Parasitic or Septic Theory

Parasitic or septic theory in 1843 Erdl described filamentous parasites in the "surface membrane" of teeth. Shortly

thereafter Ficinus, a Dresden physician observed filamentous microorganisms, which he called denticulate in material taken from carious activities. He implied that these bacteria caused decomposition of the enamel and then the dentin. Neither Erdl nor Ficinus explained how these organisms destroyed tooth structure.

Chemo–Parasitic Theory

This theory is a blend of the above two theories, because it states that caries is caused by acids produced by microorganisms of the mouth. It has been customary to credit this theory to W. D Miller (1890), whose writings and experiments helped to establish this concept on a firm basis.

The work of Willoughby D. Miller (1853-1907) at the University of Berlin has a most profound effect on the understanding of caries etiology and subsequent caries research.

In a series of experiments Miller demonstrated the following facts:

- Acid was present within the deeper carious lesions, as shown by reaction on litmus paper.
- Different kinds of foods (bread, sugar, but not meat) mixed with saliva and incubated at 37°C could decalcify the entire crown of a tooth.
- Several types of mouth bacteria (at least 30 species where isolated) could produce enough acid to cause dental caries.
- Lactic acid was an identifiable product in carbohydrate –saliva incubation mixtures.

- Different microorganisms (filamentous, long and short bacilli and micrococcus) invade carious dentin.
- Miller concluded that no single species of microorganism caused caries but rather that the process was mediated by an oral microorganism capable of producing acid and digesting protein.

Proteolytic Theory

According to this theory, the organic component is most vulnerable and is attacked by hydrolytic enzymes of microorganisms, this precedes the loss of the inorganic phase.

- Gottlieb (1944) maintained that the initial action was due to proteolytic enzymes attacking the lamellae, rod sheaths, tufts and walls of the dentinal tubules. He suggested that a coccus, probably *staphylococcus aureus*, was involved because of the yellow pigmentation that he considered pathognomonic of dental caries.
- Frisbie (1944) also described caries as a proteolytic process involving depolymerization and liquefaction of the organic matrix of enamel. The less soluble inorganic salts could then be freed from their "organic bond" favoring their solution, by acidogenic bacteria that secondarily penetrate along widening paths of ingress.
- Pincus (1949) contended that proteolytic organisms first attacked the protein elements, such as the dental cuticle and then destroyed the prism sheaths. The loosened prisms would then fall out mechanically. He also suggested that sulfatases of gram-negative bacilli

hydrolyzed "mucoitin sulfate" of enamel or chondroitin sulphate of dentin and produced sulfuric acid. The released sulfuric acid could combine with the calcium of the mineral phase. It should be noted that the composition of the organic components of enamel does not resemble that of connective tissue and an abundance of sulphated polysaccharides has not been demonstrated. The "Pincus" theory therefore remains, without any experimental support.

PROTEOLYSIS—CHELATION THEORY

According to this theory, decalcification is mediated by a variety of complexing agents, such as acid anions, amines, amino acids, peptides polyphosphates, and carbohydrate derivatives. Oral karatinolytic bacteria are thought to be involved in the process. Differences in the keratin content of the enamel in children with high caries and low caries experience are considered important. It should be noted that only a small fraction of the protein of enamel bears any resemblance to the keratin of hair.

Schatz and Martin challenged the chemo-parasitic theory and advocated the proteolysis – Chelation theory and stated that acid may prevent tooth decay by interfering with growth and activity of proteolytic bacteria.

ETIOLOGY

Caries has Multifactor Etiology (Figs 5.2A and B)

- *Micro flora:* Acidogenic bacteria that colonize the tooth surface

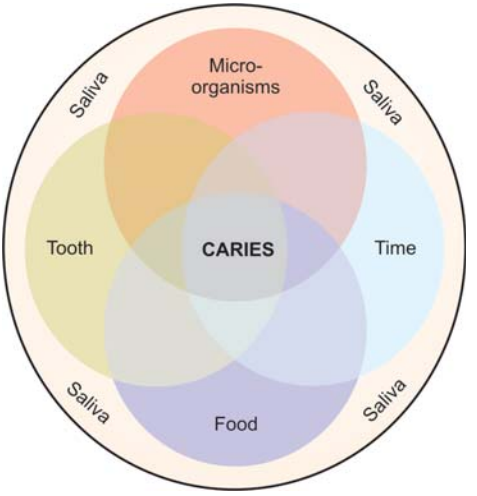


Fig. 5.2A: Keys diagram

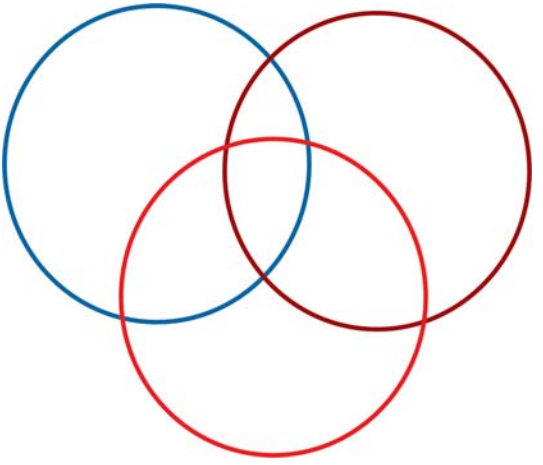


Fig. 5.2B: Modified keys diagram

- *Host:* quantity and quality of saliva, the quality of the tooth, etc.
- *Diet:* intake of fermentable carbohydrates, especially sucrose, but also starch.
- *Time:* Total exposure time to inorganic acids produced by the bacteria of the dental plaque.

Host Factors: Saliva

Saliva refers to the mixture of secretions in the oral cavity. The mixture consists of fluids derived from the major salivary glands (Parotid, submandibular, sublingual) from the minor glands of the oral mucosa and traces from the gingival exudates. There is no doubt that saliva significantly influences the caries process as evidenced by animal experiments in which the salivary glands are surgically extirpated. It is only fair to point out that removal of salivary glands is a drastic procedure affecting, in addition to saliva other factors which in themselves influence caries development includes:

1. Difference in food and water consumption.
2. Short eating intervals.
3. Greater food retention.
4. Possible alterations in the bacterial flora of the mouth.
5. Maturation of the enamel.

Xerostomia (Greek Xeros = Dry, Stoma = Mouth)

Xerostomia (Greek Xeros = Dry, Stoma = Mouth) was first described by Bartley in 1868.

Xerostomia is defined as the perception of oral dryness. It is often due to a reduction in salivary flow rates of the

major and minor salivary glands. Humans suffering from decreased or lack of salivary secretions often experiences an increased rate of dental caries and rapid tooth destruction. Xerostomia may be the consequence of a variety of different human pathological conditions like:

1. Congenital absence or malformation of salivary glands.
2. Sarcoidosis.
3. Sjogren's syndrome.
4. Therapeutic radiations of the head and neck.
5. Surgical removal of salivary glands.
6. Chronic administration of certain drugs.
7. Parkinson's disease.
8. Diabetes mellitus.
9. Acute virus infections involving salivary gland.
10. Anxiety.
11. Mental stress.
12. Depression.

Host Factors: Tooth

Tooth Morphology and Arch Form

A susceptible host is one of the factors required for caries to occur. Tooth morphology has long been recognized as an important determinant. For example attempts to induce caries in dogs have been unsuccessful mainly because of the wide spacing and the conical shape of the teeth.

On the basis of clinical observation it is known that the pit and fissure areas of the posterior teeth are highly susceptible to caries. Food debris and microorganisms readily gets impacted in the fissures. Investigations have

shown relationship between caries susceptibility and depth of the fissure. Certain surfaces of the tooth are more prone to decay whereas other surfaces rarely show decay. For example, in mandibular first molar the likelihood of decay in descending order is occlusal, buccal, mesial, distal and lingual, whereas in maxillary first molars the order is occlusal, mesial, palatal, buccal and distal (Fig. 5.3).

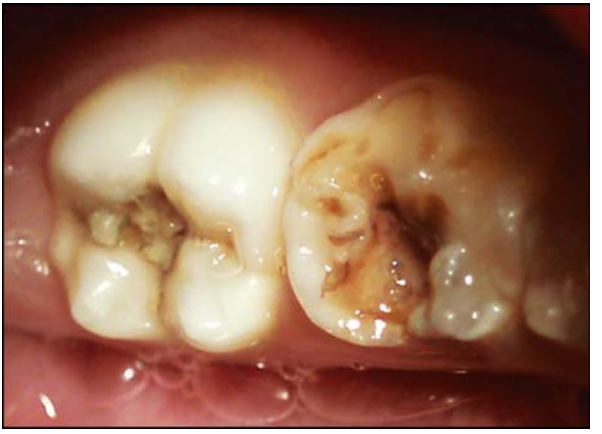


Fig. 5.3: Occlusal caries in newly erupted mandibular first permanent molar

An intraoral variation exists in susceptibility to caries between different tooth types. The more susceptible permanent teeth are the mandibular first molars closely followed by the maxillary first molars and the mandibular and maxillary second molars. The second premolars, the maxillary incisors and the first premolars are the next in sequence, whereas the mandibular incisors and canines are the least likely to develop lesions. Irregularities in arch

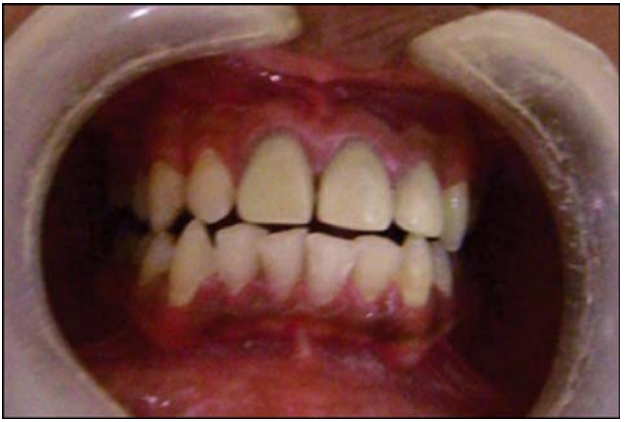


Fig. 5.4: Crowding and arch irregularities leads to development of dental caries due to poor oral hygiene

form, crowding, and overlapping of the teeth also favor the development of carious lesions (Fig. 5.4).

Tooth Composition

There is good evidence to indicate that enamel surface is more caries resistant than the subsurface. Microradiographs of initial carious lesions frequently reveal marked demineralization of the subsurface enamel beneath an outermost layer that is only slightly effective. Several hypotheses have been developed to explain this phenomenon. A pumping mechanism has been proposed whereby matter is transported from the inner enamel to the surface zone and from the surface zone to the saliva. There is net movement of inorganic mineral phase from the inner enamel to the oral cavity. The surface enamel appears unaltered

simply because it is continuously being regenerated by precipitation of solid phases. However, when "white spots" have been examined by scanning electron microscopy, the initial stage of such active carious lesions has been characterized by openings in the outer enamel surfaces through eroded focal holes.

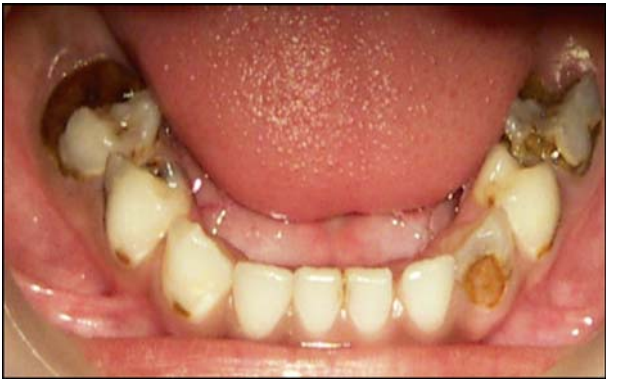
Accordingly, the concept of a relatively ineffective surface layer, described in transverse sections examined by microradiography or light microscopy, needs revision in view of the surface defects, which represents signs of focal demineralization. By transmission electron microscopy, the enamel surface is seen to be dented by irregular destruction of appetite crystals. Small microdefects starting at the enamel surface and reaching the deeper enamel layers have been observed.

Changes of the enamel such as decrease in density and permeability and an increase in nitrogen and fluoride content, occurs with age. These alterations are part of the post eruptive "maturation" process whereby teeth become more resistant to caries with time. The concentration of fluorides of the surface layer of enamel increases as the fluoride concentration of the drinking water increases, and such enamel is less soluble in acids. Furthermore, the higher the fluoride concentrations of the water supply, the lower the prevalence of caries.

Role of Specific Microflora

Microorganisms implicated in the etiology of dental caries must be acidogenic as well as acid-uric to initiate carious lesions. In enamel the microorganisms must be able to colonize the tooth surface and survive in competition with less harmful species, forming bio films –the so called Dental

Plaque. As early as 1960, Fitzgerald and keys showed that certain microorganisms isolated from human dental plaque when inoculated in germ-free rodents on a high – sucrose diet, resulted in the spread of rampant caries. Therefore dental caries should be regarded as an infectious transmissible disease.



Microbiology of Dental Caries

- Three cariogenic bacterias
  - Mutans streptococci (MS)
    - *S. mutans, S. sobrinus, S. sanguinis, S. salivarius, S. milleri*
  - *Lactobacilli*
    - *L. acidophilus, L. casei*
  - Actinomyces
- Although not the first ones to colonize the tooth, studies indicates *S. mutans* to be the main culprit in causing dental caries.

Figs 5.5A and B: Clinical picture showing dentinal caries and microorganisms responsible for the development of dental decay

There is abundant support for the so – called specific plaque hypothesis, introduced by Loesche (1982, 1986), which proposes that some specific species of the plaque flora is to be regarded as major pathogens in the etiology of dental caries. Included in the major pathogens are those bacteria associated with caries in humans and also able to induce carious lesions in experimental animals. The most important are the mutans streptococci, there are seven species, of which two *S.mutans* and *S.sobrinus*, are closely associated with caries in humans.

The second genus closely associated with caries is lactobacillus, commonly isolated from carious dentin. Also associated with the etiology of dental caries, but considered to be less cariogenic than *S.mutans*, *S.sobrinus* and Lactobacillus are *Actinomyces odontologica, Actinomyces naeslundi,* and some other species of mutant streptococci.

Streptococcus Mutans

In 1924 Clarke isolated a streptococcus that predominated in many human carious lesions and that he named *Streptococcus mutans* because of its varying morphology. Clarke noted that *S.mutans* adhered closely to tooth surfaces in artificially induced caries .For the next 40 years; *S.mutans* was virtually ignored, until the 1960s when it was "rediscovered" and its prevalence in plaque confirmed.

Characteristics of this group of streptococci have been described. They are *non motile, catalase-negative, gram – positive cocci* in short or medium chains. On mitis-salivarius agar, they grow as highly convex to pulvinate (cushion – shaped) colonies. These colonies are opaque; the surface

resembles frosted glass. These *S.mutans* variants also posses caries inducing properties, and when reisolated from infected animals they may resume the original rough colonial form.

When cultured with sucrose they form polysaccharides that are insoluble or can be precipitated with one part ethanol. This property of forming insoluble extra cellular polysaccharides from sucrose is regarded as an important characteristic contributing to the caries inducing properties of *S.mutans*. Mutants of *S.mutans*, which lack the ability to synthesize insoluble glucans or to stick to glass surfaces, do not cause smooth surface caries.

Streptococcus mutans exhibits several important properties:

1. It synthesizes insoluble polysaccharides from sucrose.
2. It is a homo-fermentative lactic acid former.
3. It colonizes on tooth surfaces.
4. It is more aciduric than other Streptococci.

Cariogenic strains of S.*mutans* contain lysogenic bacteriophage, which has not been isolated from non cariogenic strains. Non cariogenic mutants of S.*mutans* are unable to adhere to glass and have decreased ability to form insoluble polysaccharide. If these mutants are infected with lysogenic phages, they are transformed, acquiring the ability to adhere and form abundant insoluble polysaccharide.

S. mutans isolates can be grouped into three "chemo types" based on differences in the composition of the cell wall. At least seven sero types of *S. mutans* have been found. Compared to *S. sanguis*, *S. mutans* is more aciduric and can reproduce in a culture medium at a pH as low as 4.3.

S. mutans forms a homogenous group, based on physiological, ecological, and morphological characteristics, and has been recognized as a distant species by the National Communicable Disease Center. However, analysis of the guanosine and cytosine content and hybridization studies on the homologies of the DNA isolated from strains of *S.mutans* revealed significant differences. These cariogenic organisms, though phenotypically similar, are genetically heterogeneous, and were therefore divided into five genotypes or "genospecies".

However, because their nucleic acid base content and sequence are too disparate, these "mutans –like" organisms have been divided into separate species, namely *S. mutans, Streptococcus rattus, Streptococcus sobrinus, Streptococcus ferus* of the "mutans – group". *S. mutans, S. rattus, and S. cricetus have* been accepted and appear on the Approved Lists of Bacterial Names. *S. mutans* and S. *sobrinus* are most commonly found in human plaque. S. *cricetus* and *S. rattus* are less common in humans, and *S. ferus* only occurs in rats.

ORAL LACTOBACILLI

Lactobacilli are *gram positive, non spore – forming rods* that generally grow best under micro-aerophilic conditions. Isolation and enumeration of oral lactobacilli have been facilitated by use of selective agar medium which suppresses the growth of other oral organisms by its low pH 5.4

Lactobacilli are found mostly as transients in the mouth of infants. Lactobacilli represent about 1% of the oral flora. *L. casein* and *L. fermented* are the most common oral species.

The population of oral lactobacilli is influenced by dietary habits. A favorite habitat of lactobacilli is in the dentin of deep carious lesions.

Lactobacilli and its Role in Caries

Lactobacilli or organisms resembling lactobacilli have been reported in the oral cavity ever since Miller enunciated the chemo-parasitic theory. In 1925, Bunting and his collaborators claimed that bacillus Acidophilus was the specific etiological factor responsible for the initiation of caries. Subsequent investigators have isolated other types of lactobacilli besides *L. acidophilus* in saliva, plaque, and carious lesions. Small anaerobic lactobacilli (*L. minutus*) have been isolated consistently from pockets of patients with chronic periodontitis. The genus lactobacillus includes many species with a broad range of guanosine + cytosine content.

The following are the most commonly encountered in the mouth.

Homofermentative	**Heterofermentative**
L. casei	*L. fermentum*
L. acidophilus	*Lactobacillus braves*
Lactobacillus plantarum	*Lactobacillus buchneri*
Lactobacillus salivarius	*Lactobacillus cellobiosus*

In isolates of lactobacilli from human carious dentin, the homofermentative out numbered the heterofermentative variety. It was argued that lactobacilli are both acidogenic and aciduric and could, therefore, multiply in the low pH of plaque and carious lesions. Using selective culture media,

counts of lactobacilli in the saliva could be correlated with the prevalence of dental caries. Furthermore the growth site of lactobacilli was reported to correspond to the sites of clinically diagnostic carious lesions. When such lesions were restored, most of the growth sites of the lactobacilli were removed.

Acceptance of the doctrine that lactobacilli were the etiological agents of dental caries was not universal, however as more information on the microbial composition of dental plaque became available it was found that lactobacilli constitute only a minor fraction (1/10000) of the plaque flora. Amount of acid that can be formed by the relatively small number of lactobacilli present in plaque is almost insignificant in comparison with that produced by other acidogenic oral microorganisms. In humans lactobacilli can be isolated from the saliva, tooth surfaces, dorsum of the tongue, vestibular mucosa, and hard palate. *L. acidophilus* is most frequently isolated from saliva. Lactobacilli have a relatively low affinity for the tooth surface. The establishment of oral lactobacilli coincides with the development of carious lesions. *L. casei* is the predominating lactobacillus in dental plaque and carious dentin. Fitzgerald interprets such data to mean that lactobacilli are more a consequence than a cause of caries initiation.

Oral Actinomyces

Actinomyces is a *gram –positive, nonmotile, non-spore-forming* organisms occurring as rods and filaments that vary considering in length. Filaments are usually long and slender and are branching.

The species that have been found in the oral cavity are:

Facultative anaerobic	Anaerobic
A. naeslundi	*A.israelii*
A. viscosus	*Actinomyces meyeri*
	A. odontolyticus

All species of Actinomyces ferment glucose, producing mostly lactic acid, lesser amounts of acidic and succinic acid, and traces of formic acid. Most interest has centered on *A.viscosus* and *A.naeslundi* because of their ability to induce root caries, fissure caries, and periodontal destruction when inoculated into gnotobiotic rats. *A.viscosus* has been separated into two, and *A.naeslundi* into four, serological types.

Actinomyces is a good plaque former, capable of adhering to wires and forming tenacious deposits on the teeth. It is the most common group of microorganisms isolated from the subgingival microflora and from plaque of human root surfaces caries. It is found in the supragingival plaque of all children and comprises about 50% of all cells present. *A.naeslundi* predominates in the tongue, salivary flora, and in the plaque of young children, while plaque from teenagers and adults has a higher proportion of *A. viscosus*.

Classification of Dental Caries

This classification is based upon the clinical, radiographic and histological appearance of caries process and in some instances, the involvement of a particular tooth, group of teeth or a tooth surface (Table 5.1). Classification of dental caries according to size and site by Graham Mount is shown in Table 5.2.

Table 5:1 Classification of dental caries

Classification location	*Method of examination*
Class I Cavities in pits or fissures Occlusal surfaces of premolars and molars	Direct or indirect visual exploration
Occlusal two-third of facial and lingual surfaces of molars	Radiograph not useful
Class II Cavities in proximal surfaces of premolars and molars	Early caries: by radiographs only Moderate caries not broken through from proximal to occlusal. Visually by color changes in tooth and loss of translucency Exploration from proximal Extensive caries involving occlusal: direct visual
Class III Cavities in proximal surfaces of incisors and canines which do not involve the incisal angle	Early caries: by radiographs or transillumination. Moderate caries not broken through to lingual or facial: • Visual by tooth color change • Exploration • Radiograph Extensive caries: direct visual
Class IV Cavities in proximal surfaces of incisors or canines which involve the incisal angle	Visual transillumination
Class V Cavities in the cervical 1/3rd of facial or lingual surfaces (not pit or fissure)	Direct visual: dry surface for vision Exploration to distinguish decalcification: whether rough or hard and unbroken. Area may be sensitive to touch

Table 5.2: Classification of caries lesion according to size and site by Graham Mount

Site	*Size*			
	Minimal[1]	Moderate[2]	Enlarged[3]	Extensive[4]
Pit or fissure[1]	1.1	1.2	1.3	1.4
Contact area[2]	2.1	2.2	2.3	2.4
Cervical area[3]	3.1	3.2	3.3	3.4

Depending upon the Origin Dental Caries can be Classified as:

a. An incipient, initial or primary caries, defined as the first attack on a tooth surface (Fig. 5.6)
b. A recurrent or secondary lesion is the one which can be observed under or around the margins or surrounding walls of an existing restoration (Fig. 5.7).
c. Residual caries-Which is not removed during a restorative procedure.

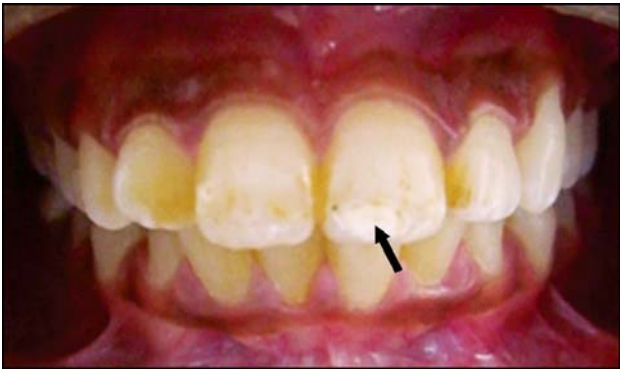


Fig. 5.6: Incipient lesions

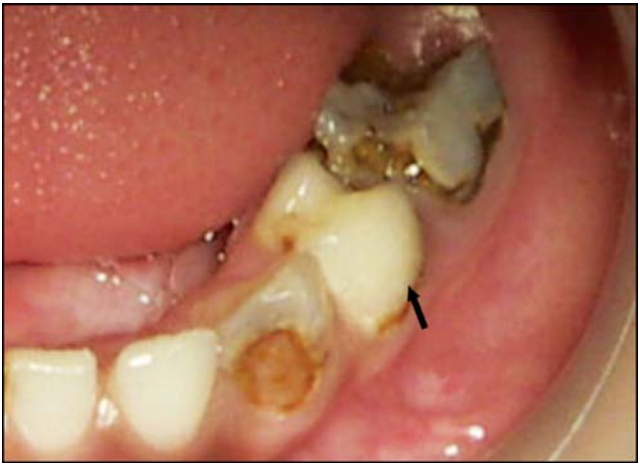


Fig. 5.7: Secondary caries

Depending on the Rapidity of Progress

a. Acute or rampant caries is a rapidly invading process that usually involves several teeth (Fig. 5.8)
b. *Chronic caries:* Which are of variable depth, long standing and tend to be fewer in number (Fig. 5.9).
c. *Arrested caries:* Described as caries which become static or stationary and does not show any tendency for further progression (Fig. 5.10).

Depending on the Location

a. *Pit and fissure caries:* Those originating in the pits and fissures found on the lingual surface of maxillary anterior teeth and on the buccal, lingual and occlusal surfaces of the posterior teeth (Fig. 5.11)

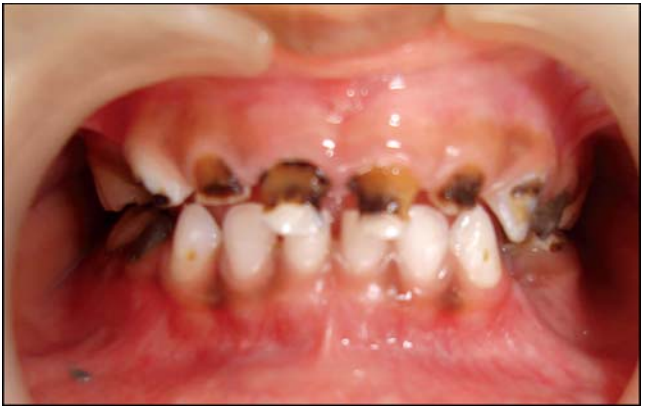


Fig. 5.8: Rampant caries

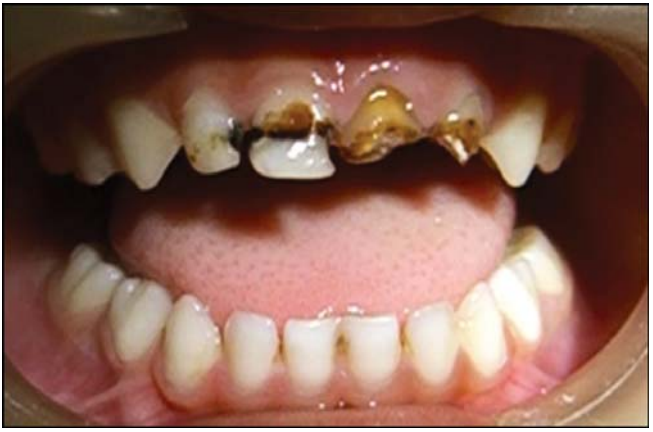


Fig. 5.9: Chronic caries

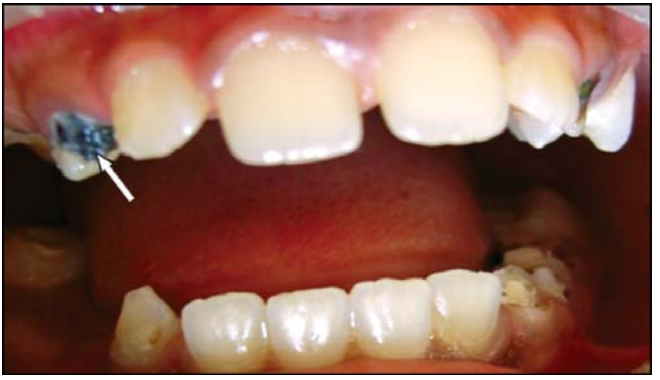


Fig. 5.10: Arrested caries

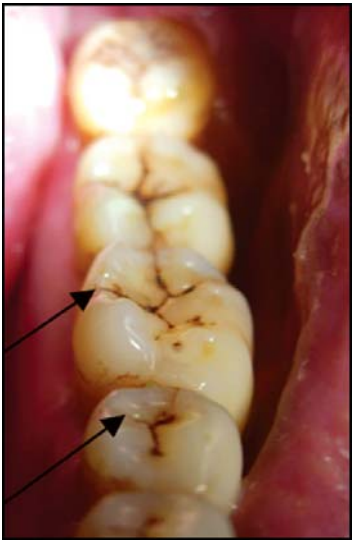


Fig. 5.11: Pit and fissure caries involving molars and premolars

b. *Smooth surface caries:* Carious lesions located on surface other than pits and fissures are classified as, smooth surface lesions.

- Smooth surface lesions may be further subdivided as:
  - Inter-proximal, occurring on mesial or distal contact points.
  - Cervical, occurring on buccal or lingual surface near the dentine-enamel junction.

Proximal Caries—Susceptible zone

This region extends from the contact point down to the height of the free gingival margin. It increases with the recession of the alveolar bone and gingival tissues (Figs 5.12 and 5.13).

Facial, buccal and lingual caries—Note the uniform enamel surrounding each radiolucent lesion (Fig. 5.14).

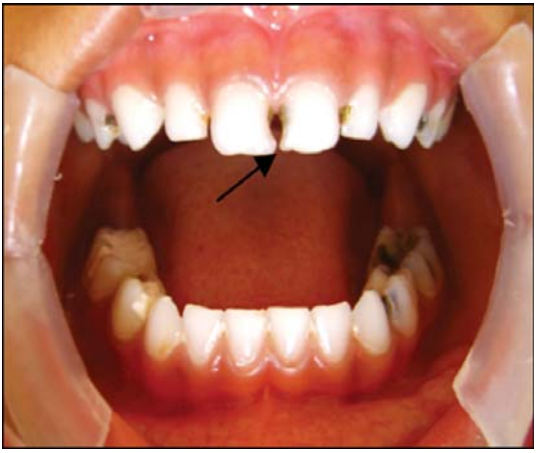


Fig. 5.12: Proximal caries involving maxillary incisors

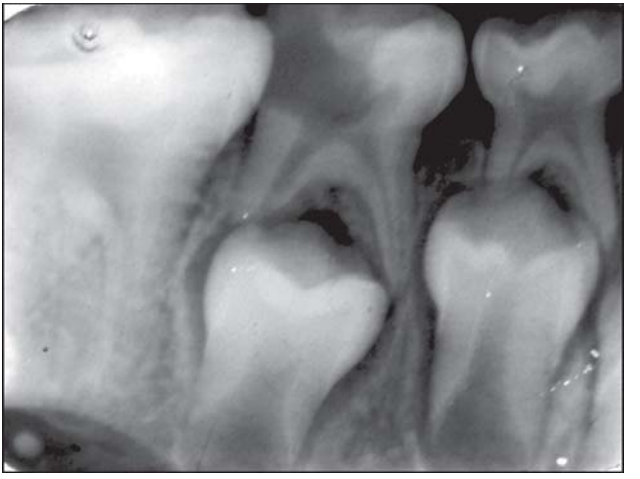


Fig. 5.13: Radiographically proximal caries encroaching pulp can be appreciated

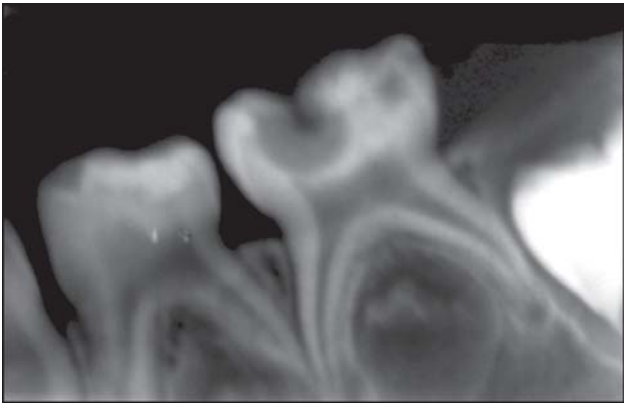


Fig. 5.14: Lingual caries involving the mesiolingual pulpal horn

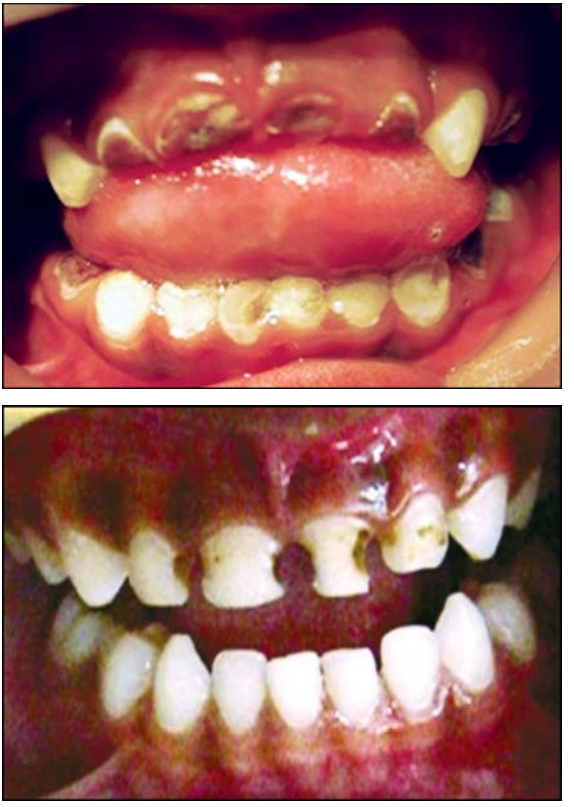
According to the Pathway of Dental Caries

a. *Forward decay or caries:* Where the caries starts in the enamel and then it involve the dentin i.e. wherever the caries in enamel is larger than in dentin.
b. *Backward decay or caries:* Where caries process in dentin progresses much further than it does in enamel i.e. when the spread of caries along the DEJ exceeds the caries in the contagious enamel, from the junction and is termed as backward caries.

Depending upon the Patient's Age

Infancy (Early Childhood or Nursing Bottle) Caries (Figs 5.15A and B)

Numerous reports by Pediatricians and Pedodontists describe a rapidly progressing type of dental caries that affects the primary teeth of children, usually during the first 2 years of life and sometimes as early as the first year. In children with infancy caries there is a unique distribution of dental decay. The 4 maxillary anterior incisors are affected first; these teeth are anatomically so positioned in the mouth as to be most frequently bathed by a feeding. If unchecked the decay may extend to the maxillary and mandibular molars. Initially, the lower anterior teeth may not be involved because of the protective environment of the mandibular salivary secretions and the cleansing action of the tongue muscles. Infancy caries is most often seen in children with an unusual dietary history such as the addition of syrup, honey or sucrose to the formula of the use of a pacifier dipped in honey or other. It has been reported that prolonged and unrestricted night time breast-feeding can result in increased caries rates.



Figs 5.15A and B: Both the pictures depicting the anterior teeth affected from early childhood caries (ECC)

Adolescent Caries

The characteristic features of this type of caries are:

- Lesions of teeth and surfaces those are relatively immune to caries.

- Relatively small opening in enamel with extensive undermining of enamel.
- Rapid penetration of enamel and extensive involvement of dentin.
- The rapid progression of the lesion which does not permit an effective pulpal response with little or no secondary dentin.

Senile Caries

Senile caries (Fig. 5.16) occurs during old age. This may be due to reduction in salivary secretion or exposure of roots following gingival recession.

Depending on the surfaces of tooth involved as:

a. Simple caries lesion (Fig. 5.17)—Involving only one surface of the tooth.
b. Compound caries lesion (Fig. 5.18)—Involving two surfaces of a tooth.

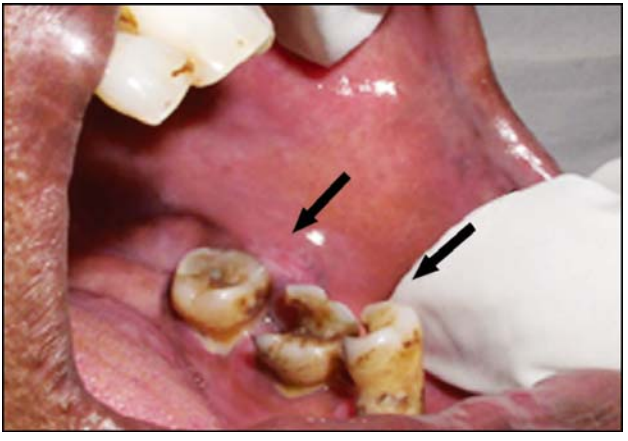


Fig. 5.16: Senile caries

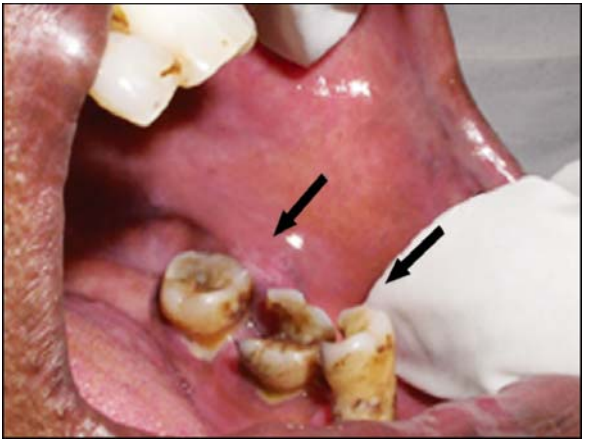


Fig. 5.17: Simple caries involving single tooth surface

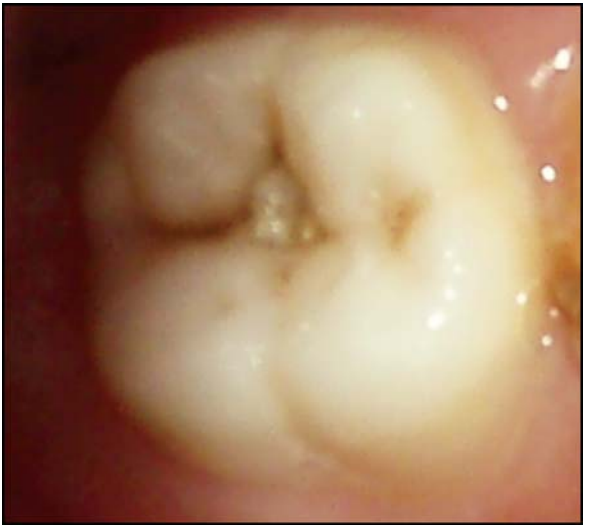


Fig. 5.18: Complex caries involving two surfaces of teeth

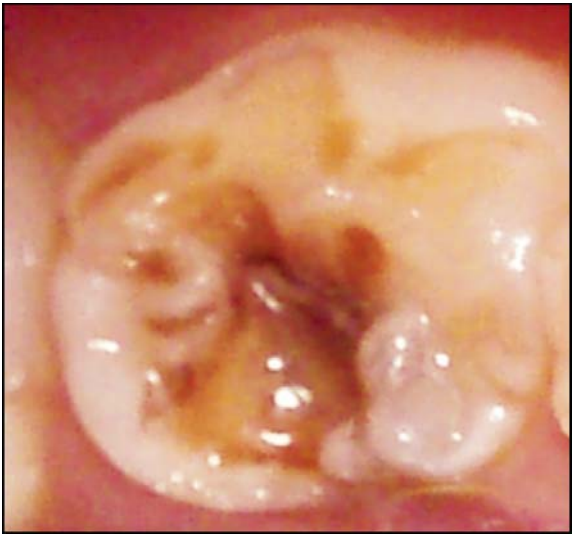


Fig. 5.19: Compound caries involving more than two surfaces of teeth

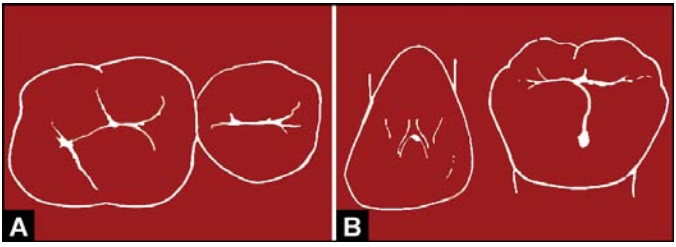
c. Complex caries lesion (Fig. 5.19)—Involving three or more than surfaces.

Blacks well known method of classification of cavities and also Graham Mounts new classification of cavities for operative dentistry is based on morphological considerations.

Classification based on GV Black's Treatment and Restorative Design

a. Class I-G.V. Black Classification (Figs 5.20A and B)
Pit and fissure (anterior and posterior)

- Caries begins in structural defects of the teeth i.e. pits, fissures and sometimes grooves.



Figs 5.20A and B: Class I caries affecting the occlusal surface of molars, premolars and the lingual pit of interiors

- Locations:
  - Occlusal surfaces of premolars and molars.
  - Occlusal 2/3rd of the buccal and lingual surfaces of molars and premolars.
  - Lingual surfaces of anteriors.
- By Black—Class I lesions occur in pits and fissures of all teeth, but this class is essentially intended for bicuspid and molars.

b. Class II-GV Black Classification
Proximal surface of posterior teeth (Fig. 5.21)
- Caries found are on proximal surfaces of bicuspids and molars.
- By GV Black—Class II lesion can involve both Mesial and Distal surfaces or only proximal surface of a tooth and is referred to as an Mo, Do or MOD.

c. Class III-G.V. Black Classification (Fig. 5.22)
Proximal Surface of Anterior Teeth
- Caries found on the proximal surfaces of anterior teeth that do not involve the incisal angle.
- By Black – Cavity may occur on mesial and distal surface of any incisor/cuspid.

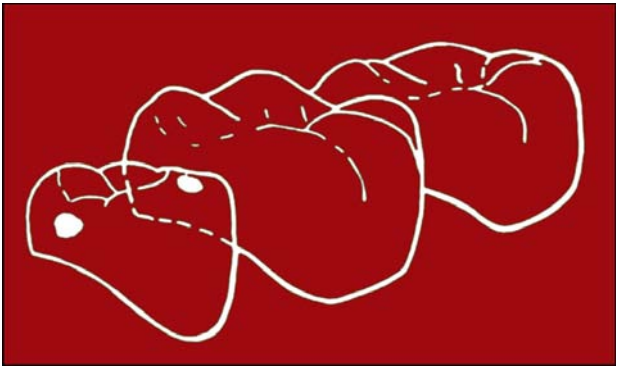


Fig. 5.21: Class II caries involving mesial and distal surfaces of the posterior tooth

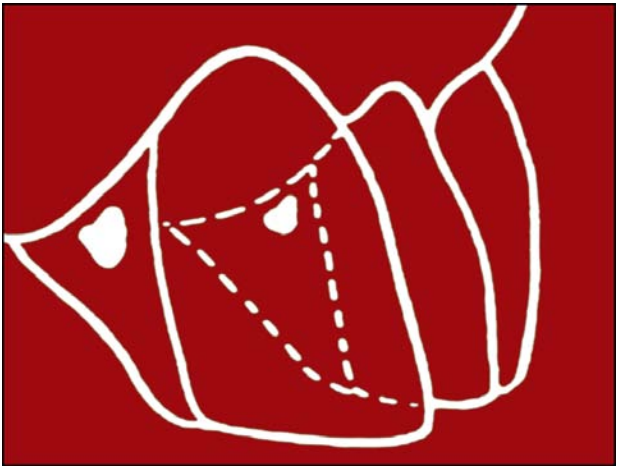


Fig. 5.22: Class III caries present on the proximal surface of incisors, without involving the incisal edge

d. Class IV-GV Black Classification (Fig. 5.23)
Proximal surface and incisal angle of anterior teeth
- Found on the proximal surfaces of anterior teeth that involves the incisal edge
- By Black—is a lesion on the proximal surface of an anterior tooth, in which the incisal edge is also involved.

e. Class V- GV Black Classification (Fig. 5.24)
Cervical area of anterior and posterior tooth
- Caries found at the gingival third of the facial and lingual surfaces of anterior and posterior teeth.
- By Black – can occur on either the facial or the lingual surfaces; however the predominant occurrence of these lesions is adjacent to the lip and cheeks rather than tongue.

f. Class VI-GV Black Classification (Fig. 5.25)
Occlusal Cusps of Posterior Teeth

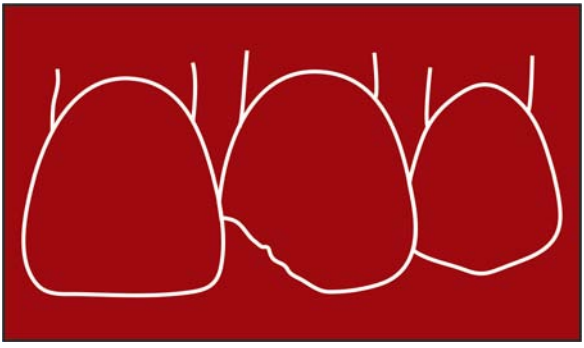


Fig. 5.23: Class IV caries on the proximal surface of the incisors, involving the incisal edge

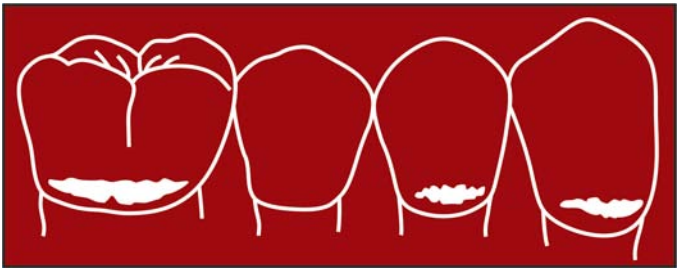


Fig. 5.24: Class V caries present at the cervical third of the facial and lingual surfaces

Fig. 5.25: Class VI caries involving occlusal cusp tips of molars and premolars, in addition to the incisal edge of anteriors

Incisal edge of anterior teeth:

- Defects found on incisal edges and cuspal tips.
- Also found on premolars and molar cuspal tips.

Depending on the tissue involvement as:

a. Enamel caries (Fig. 5.26)
b. Dentin caries (Fig. 5.27)
c. Cemental caries (root caries) (Fig. 5.28)

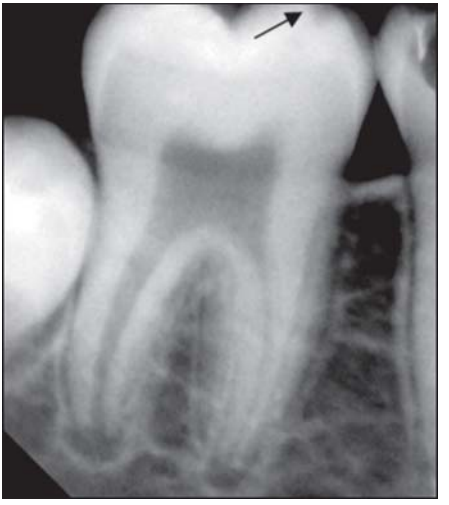


Fig. 5.26: Enamel caries - lesion restricted to enamel itself

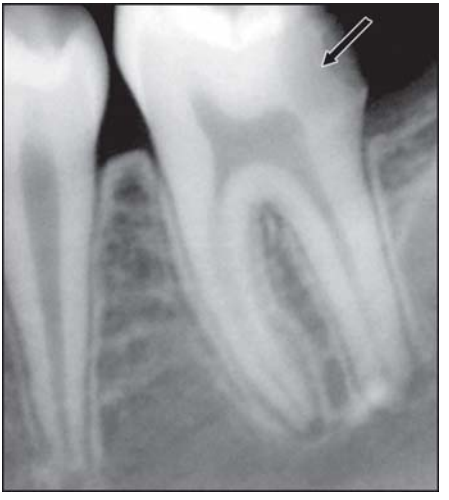


Fig. 5.27: Dentinal caries – lesion extended up to dentin involving enamel, dentin and pulpal horn

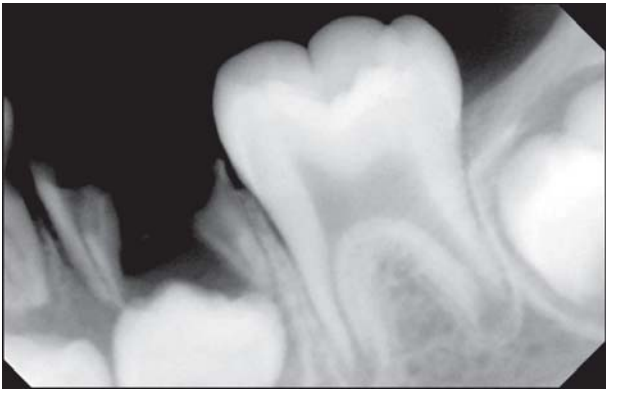


Fig. 5.28: Root caries – gross destruction of the coronal portion of the tooth, involving furcation and the radicular portion

Radiographic Caries Classification

A. **An incipient interproximal lesion** (Fig. 5.29) extends less than halfway through the thickness of enamel. The term incipient means beginning to exist or appear. An incipient lesion is seen in enamel only.
B. **A moderate interproximal lesion** (Fig. 5.30) extends greater than halfway through the thickness of enamel, but does not involve the DEJ. A moderate lesion is seen in enamel only.
C. **An advanced interproximal lesion** (Fig. 5.31) extends to the DEJ or through the DEJ and into the dentin, but does not extend through the dentin greater than half the distance toward the pulp. An advanced lesion affects both enamel and dentin.

According to Rate of Progression of the Lesions

Active

Color-pale yellow

Consistency-soft and cheesy

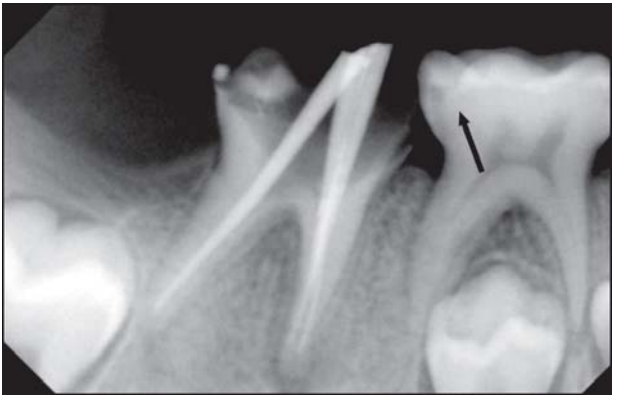


Fig. 5.29: "Notched" incipient proximal enamel caries

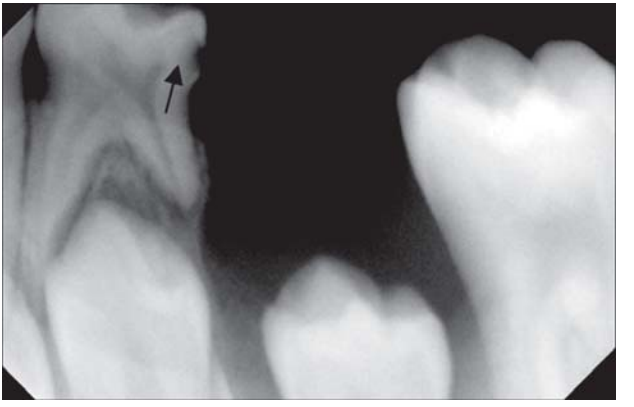


Fig. 5.30: Moderate enamel proximal caries

Pain-painful with sweets and on probing

Chronic

Color-dark center with pale periphery

Consistency-leathery center and soft edges

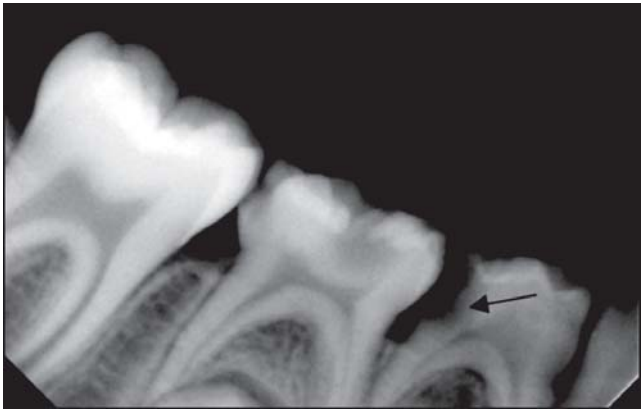


Fig. 5.31: Advanced proximal caries

Pain-generally no pain

Arrested
Color-dark brown or black
Consistency-hard
Pain-none

CRITERIA FOR DIAGNOSIS

Cavitation: It has to be distinguished from fractures and smooth lesions of erosion and abrasion.
No Frank Cavitation: It is difficult to diagnose. Criteria of diagnosis is being summarized in 3 categories:

i. *Detection of pit and fissure:* Area is carious when the dentist resists probe after insertion into a pit or fissure and is accompanied by one or more.
ii. *Detection of lesion on smooth area of facial and lingual surfaces:* Area is carious if surface is etched or if there is white spot as evidence of demineralization.
iii. Detection of lesions on proximal surfaces.

Table 5.3: Signs to look for dental caries

- Soft-end enamel adjacent to pit or fissure which may be scrapped out.
- Softness at the base of area.
- Opacity adjacent to pit or fissure due to demineralization.
- Area is carious if there is loss of normal translucency of enamel adjacent to pit.
- If there is penetration with explorer.
- Scraping away enamel with explorer.
- Areas exposed as marginal ridge show opacity as undermined enamel. Any discontinuity of enamel in which an explorer will enter is carious.
- Any definite radiolucency indicating a break in continuity of enamel.
- Loss of translucency.
- The initial clinical lesion seen in smooth surface enamel caries is a chalky white spot which is hard and shows no cavitation. This results in an increased porosity of tissue and leads to color change. White lesion becomes stained progressing to light brown and then darker brown. Color change takes place due to change in amino acid composition of the matrix.

Clinical signs for diagnosing dental caries are summarized in Table 5.3.

Actually stains and pigmentation should not be regarded as evidence of caries because these may occur on sound teeth too.

CONDITIONS RESEMBLING CARIES

Number of radiolucencies involving crown and roots of teeth are seen similar to caries.

Restorative Materials

It can be identified by its well-defined smooth outlines.

Abrasion (Fig. 5.32)

It is a wearing away of tooth structure from friction of a foreign object. Most commonly it is caused by tooth brushing and is seen at the cervical margin of teeth. It can be confused with root surface caries. On radiograph it is seen as well-defined horizontal radiolucency along the cervical margin of a tooth.

Cervical Burn Out

It is a radiolucent artifact seen on dental X-ray. It may be confused with dental caries and appears as collar or wedge-shaped radiolucency.

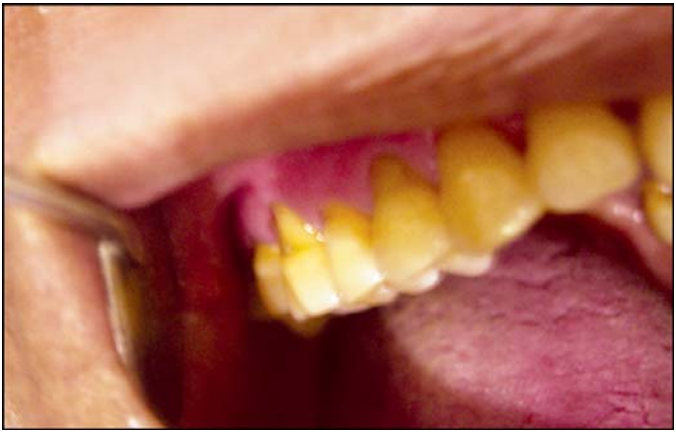


Fig. 5.32: Abrasion-cervical

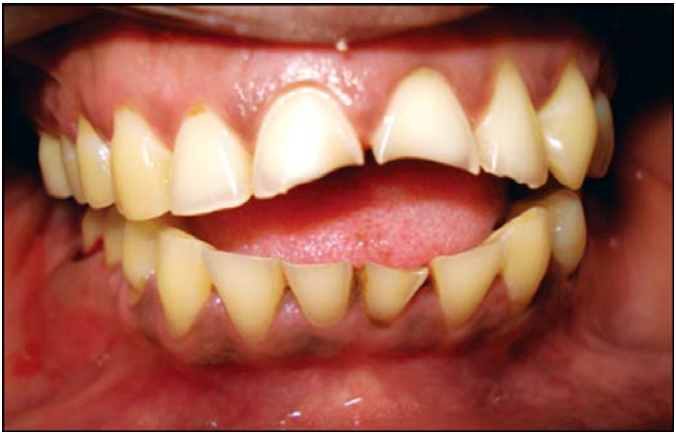


Fig. 5.33: Severe attrition of anterior teeth

Attrition (Fig. 5.33)

It is a mechanical wearing down of teeth and these concavities may resemble occlusal or incisal caries on a dental radiograph.

CHAPTER 6

Dental Stains and Discolorations

A tooth presenting with smooth and shiny surface, depicts a healthy tooth. Tooth color varies from individual to individual, i.e. it can range from pearl white to pale yellow. Any deviations from these colors and smooth surface anatomy can be termed as **dental stains or dental discolorations**.

Discolorations can occur in various forms:

- Stains limited to the external surface of the tooth
  - Due to external factors
- Stains incorporated within the tooth structure
  - Due to external factors
  - Due to internal factors
- Stains present due to formation of calculus and soft deposits.

Stains present due to exogenous reasons can be settled with prophylactic procedures like scaling and polishing but stains occurring within the tooth substance are hard to remove and routine procedures are of little help (Table 6.1).

EXTRINSIC STAINS

Yellow Stains

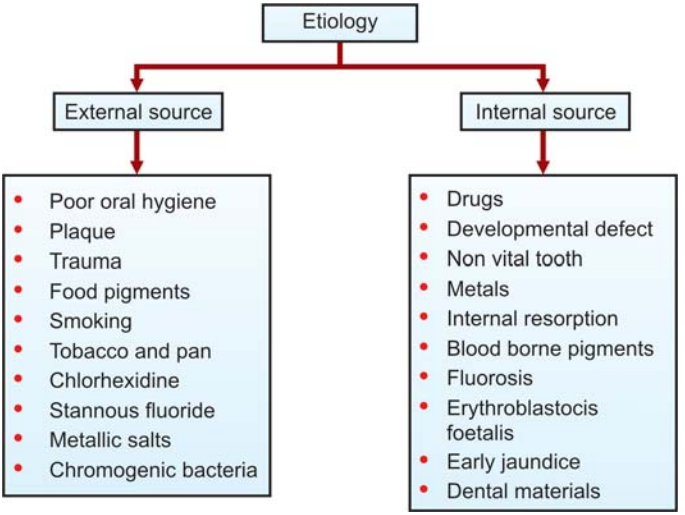
These stains generally occur from the food pigments and are also associated with the presence of bacterial plaque; it

Table 6.1: Classification of stains

According to the location of stains:
- Extrinsic stains
- Intrinsic stains

According to the sources of stains:
- Local/Environmental/Exogenous
  - Exogenous extrinsic
  - Exogenous intrinsic
- Systemic/endogenous
  - Endogenous intrinsic

Table 6.2: Etiology of dental stains and discolorations



has a dull, light yellow appearance due to bacterial plaque which is common to all ages and more prominent inpatients where personal oral hygiene is neglected.

Green Stains

The appearance of these stains varies from light or yellowish green to very dark green. These stains are generally embedded in the bacterial plaque extending from facial to proximal surfaces of the tooth. Generally facial one-third of the maxillary anterior teeth are involved.

These stains can occur in various forms such as:

- Curved line following the contour of the gingival crest
- Streaked, following the grooves in the enamel.
- Dispersed or smeared irregularly on the entire surface of the tooth.

These stains are composed of chromogenic bacteria, fungi (giving it a green appearance), decomposed hemoglobin and few inorganic elements such as calcium, potassium, sodium, magnesium and others in small quantity.

Although they can occur at any age but they are primarily found in childhood resulting from poor oral hygiene, gingival hemorrhage and chromogenic bacteria which gets embedded in the bacterial plaque and give rise to green stains.

D/ D for Green Stains

- Chlorophyll preparations
- Drugs
- Industrial metallic dusts.

Black Stains

A highly retentive calculus like stain, black or dark brown in color, occurring on the gingival third along the gingival margin also known as brown stain.

The stain appears as a continuous or interrupted line following the gingival margin. This line may be 1 mm wide with no appreciable thickness or it may also appear as heavy deposits which will be slightly elevated from the tooth surface and is easily detectable. The teeth frequently appear clean and shiny with a lower tendency to bleed.

Stains generally make their appearance on facial and lingual surfaces extending up to the proximal surfaces of the posterior teeth. Rarely the anterior teeth are involved.

Black stain has a calculus like appearance and is generally composed of microorganisms embedded deep in a matrix. These microorganisms are mostly gram positive rods, including a small percentage of cocci along with other bacteria. This differs from the composition of the calculus which predominantly contains cocci but the mineralization is quite similar to calculus.

Such stains can occur at any age but are more common in childhood, females and frequently found in clean mouth. These stains tend to recur despite maintenance of personal hygiene but the severity may reduce with meticulous plaque control methods.

Tobacco Stains (Fig. 6.1)

Clinically, these stains appear as light brown to dark brown or black. They may appear in various forms such as:

- Narrow crest following the gingival contour
- Wide band extending from the cervical third to the middle third of the tooth
- Diffuse.

They are primarily present on the cervical third of the tooth but may involve any surface including pits and

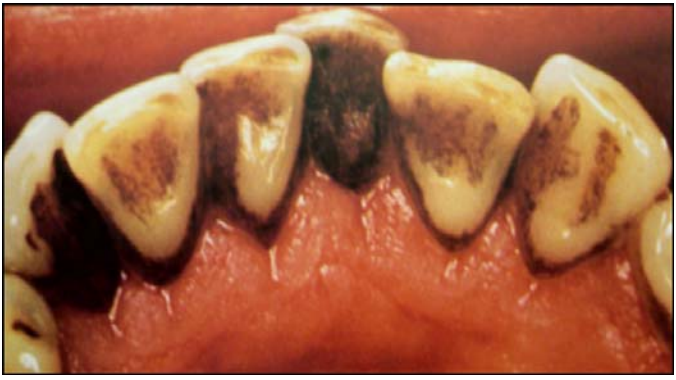


Fig. 6.1: Tobacco stains on the lingual aspect of lower anterior teeth

fissures and the lingual surface. These stains are generally formed from the smokeless tobacco and are mainly composed of tar and combustion products. The heavy deposits may penetrate the enamel and present as endogenous stains.

Orange and Red Stains

These stains appear at cervical third, occurs more frequently on anteriors than compare to posterior covering both facial and lingual surfaces of anterior teeth. These stains are generally caused by chromogenic bacteria.

Metallic Stains: Occurring due to

1. Metal present in drugs
2. Metals from metal industry

Metal Present in Drugs

These stains appear from the drugs that incorporate metal salts. These stains are generalized unlike other stains and may affect all the surfaces of the tooth. These drugs enter the plaque substance and impart its color to plaque and calculus, e.g. consumption of iron will give rise to black or brown stains, manganese will give rise to black stains.

Metals from Metal Industry

The populations affected with these kinds of stains are those in the vicinity of the industry. Cervical third of the anterior teeth are primarily affected by the inhalation of metallic dust through mouth, bringing them in contact with teeth, these metals along with bacterial plaque impart their color to the plaque present which occasionally may penetrate the tooth substance and give rise to endogenous stains. Copper will give bluish green stains, nickel will give green stain, and cadmium will give yellow or golden brown stains.

Other Exogenous Brown Stains

Stannous Fluoride

Stains appears sometimes as light brown or light yellow, which forms on to the tooth surface after the prolonged or the repeated use of stannous fluoride product which results in the formation of brown tin oxide due to the reaction of the tin ion and fluoride compound.

Brown Pellicle

This stain is smooth and structure less and recurs readily after removal. The pellicle can take on stains of various colors.

Food Pigments

Stains resulting from pigments leaching out of the food such as turmeric stains, stains from tea, coffee, and use of artificial colors give rise to various stains.

Chlorhexidine Mouth Rinses

Chlorhexidine mouth rinses are excellent agents for the plaque control, but repeated use of these agents may give rise to brownish stains on the tooth surfaces generally affecting the proximal surface of the tooth.

Betel Leaf (Fig. 6.2)

Use of betel leaf will give rise to stains which can vary in color from dark brown to deep mahogany brown to black.

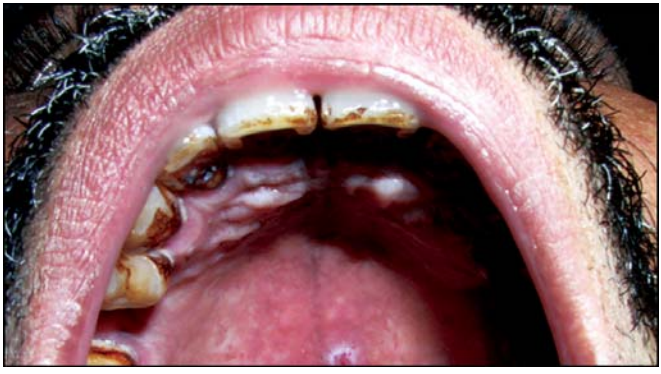
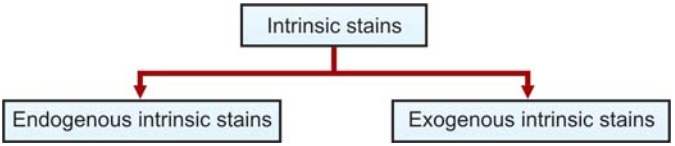


Fig. 6.2: Stains occurring due to prolong use of betel leaf

These stains generally contain microorganisms and mineralized content, which has the characteristic resemblance to the subginigival calculus.



Endogenous Intrinsic Stains

These stains are incorporated within the tooth structures that have occurred to take place during the development of the tooth and are more suggestive of developmental anomalies of tooth. As the name suggests these stains cannot be removed by regular prophylactic procedures.

Drugs

Tetracycline Stains (Fig. 6.3)

This drug was widely used once upon a time to fight variety of infections. At that point little was known about the side effects of the drug. This antibiotic has an affinity towards

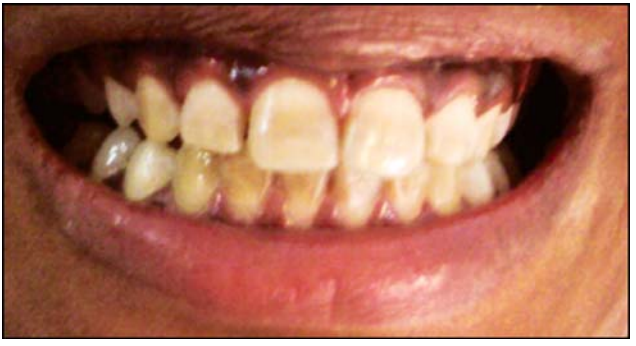


Fig. 6.3: Patient exhibiting tetracycline stains

the mineralized tissue and gets absorbed by the hard tissues of the body.

Since the drug also has a capability to pass across the placenta and enters the fetal circulation, so when the expecting mother is administered this drug during the third trimester, the drug gets deposited in the forming bones and teeth of the fetus. Discoloration can also occur when the drug is administered to the child in infancy and early childhood.

Discoloration may be generalized or limited to specific parts of the tooth, depending up on at what period of development it was administered. It also depends on the dosage duration and the type of the drug. The color of the tooth may very from light green to dark yellow or a grey brown. The stains appear as a thick band of discoloration present either on cervical, middle or incisal third depending upon the time of the administration of the drug during the development.

Pulpless Tooth/Non Vital Tooth

Not all the pulp less teeth discolor, this is attributed to the improved endodontic procedures that prevent discoloration associated to a necrotic pulp. Such pulpless teeth has a vide range of discoloration, which can range from light yellow to brown to grey or from black to bluish black to dark brown. The discoloration results from the hemorrhagic breakdown or from the decomposition of the pulpal tissue within the chamber. As the pulp chamber is non-compliant chamber, the decomposed hemoglobin stagnates within the pulp chamber and seeps in to the dentinal tubules of the tooth consequently resulting in intrinsic discoloration.

Developmental Defects of the Teeth

Genetic or environmental disturbances during the tooth formation may give rise to developmental defects like amelogenesis imperfecta or dentinogenesis imperfecta due to the defective development of ameloblastic and odontoblastic cells consequently affecting the tooth development.

Developmental Defects Occurring due to Genetic Factors

Amelogenesis Imperfecta (Fig. 6.4)

Genetic anomalies generally involve both primary and the permanent dentitions that affect either enamel or dentin, and most often it appears as a diffuse or even vertical derangement. Amelogenesis imperfecta is considered a hereditary structural anomaly with an incidence of

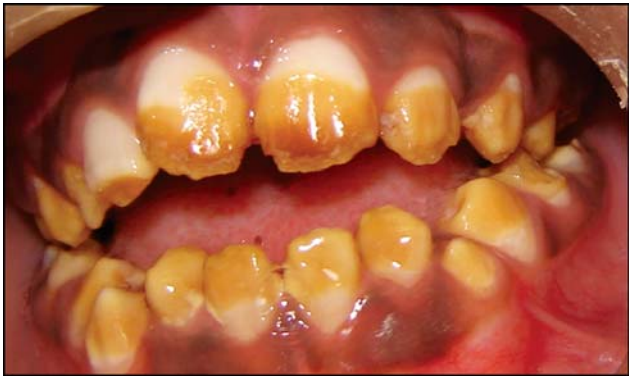


Fig. 6.4: Amelogenesis imperfecta: a clinical view depicting worn off appearance of enamel and exposed dentin in conjugation with open-bite

occurrence of 1 person in every 1,400. In such cases, the pulp and dentin are usually normal and the teeth are caries resistant.

Amelogenesis imperfecta can be divided into two broad categories:

1. Hypomineralization—qualitative enamel anomalies
2. Hypoplasia—quantitative enamel reductions but the mineralization is normal.

According to Schulze

- Hypomineralization of enamel
  - Autosomal dominant
  - Autosomal recessive
  - Autosomal dominant with occlusodentodigital dysplasia
- Hypoplasia of enamel
  - Sex-linked dominant
  - Sex-linked dominant with open-bite, which may represent dysgnathia, abnormal skull development
  - Sex-linked dominant, without open-bite
  - Autosomal-recessive, hypoplastic form
  - Autosomal-dominant, hypoplastic form
  - Autosomal-dominant with pleiotropic effects
  - Autosomal-dominant, aplastic form
  - Autosomal-dominant transmission with Morquio's syndrome.

Witkop added a new category (hypomaturation) to Schulze's classification based on clinical, genetic, and structural differences between hypoplasia and hypomineralization.

Characteristics of hypoplastic form of amelogenesis imperfecta:

- Reduction in the amount of enamel matrix
- Normal hardness but insufficient thickness
- Enamel surface is hard, rough, granular or lobulated
- Surface stains ranges from yellow-yellowish brown to yellowish red due to absorption of stain from food and other sources
- Females show less discoloration than males.

Characteristics of hypomineralized form of amelogenesis imperfecta:

- Disturbance in the mineralization process
- Teeth erupts normally but wears away with the period of time
- Affects mostly the labial surface of anterior teeth and molar cusp tips
- Low mineral content, decreased radiographic appearance
- Abnormally colored enamel varies from yellow to yellow red to brown flakes.

Dentinogenesis Imperfecta (Fig. 6.5)

Dentinogenesis imperfecta is a developmental defect, inherited as an autosomal dominant trait. In this condition the permanent and primary dentition are involved, enamel appears to be normal, roots are also affected.

- Post eruption, there is a rapid calcification of the pulp chambers and root canals which will lead to the darkening in the color of the clinical crown
- Characteristic findings consist of a peculiar opalescent purplish brown or gray color of the teeth

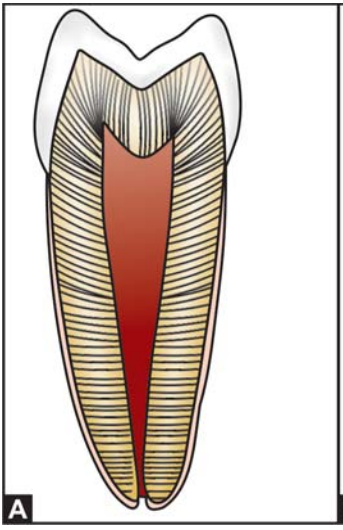


Fig. 6.5A: Normal healthy tooth

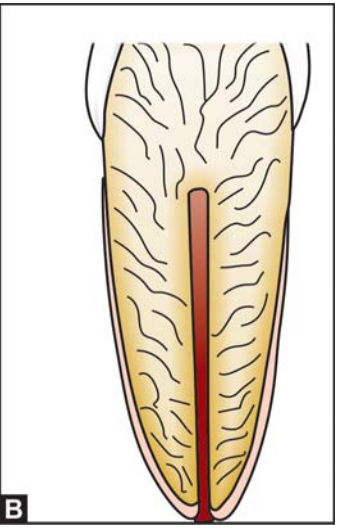


Fig. 6.5B: Tooth affected from dentinogenesis imperfecta having reduced sensitivity due to regression of pulp

- Rapid attrition of the crowns, and reduction in carious lesions.
- Enamel, cementum, and periodontal membrane show no microscopic abnormalities, but there are many changes noted in the dentin.
- The dentinoenamel junction is straight rather than scalloped which probably accounts for the weak attachment of the enamel to the dentin and the tendency for the enamel to chip, leaving exposed dentin.
- Irregular tubular arrangement can be seen.
- Smaller number of tubuli, decreasing toward the pulp.

- Occasional larger diameter of tubuli and cellular inclusions.
- Numerous branching of the tubuli.
- Poor calcification of the teeth.
- The circumpulpal dentine picture resembles irregular secondary dentine formation.
- The teeth are much smaller than normal, about one-half the dimensions or one-eighth the volume.
- Enamel appears as a very thin layer, and is quickly worn away in the mouth.
- Pulp chambers and canals show in the radiographs of deciduous teeth during formation, but are almost completely obliterated in erupted teeth of the permanent set.
- The teeth are not sensitive to grinding, indicating that the dentine does not contain vital fibrils.

Enamel Hypoplasia/ Enamel Hypocalcification (Fig. 6.6)

Enamel hypoplasia is a disorder affecting the structure of the teeth. It is often seen as one component associated with many syndromes or enamel hypoplasia may be mild enough resulting in a pitting of the enamel surface or a horizontal line across the enamel of the crown.

Systemic/Prenatal Hypoplasia

Results from the ameloblastic disturbance which occurs in two stages:

- During formation of enamel matrix—enamel hypoplasia

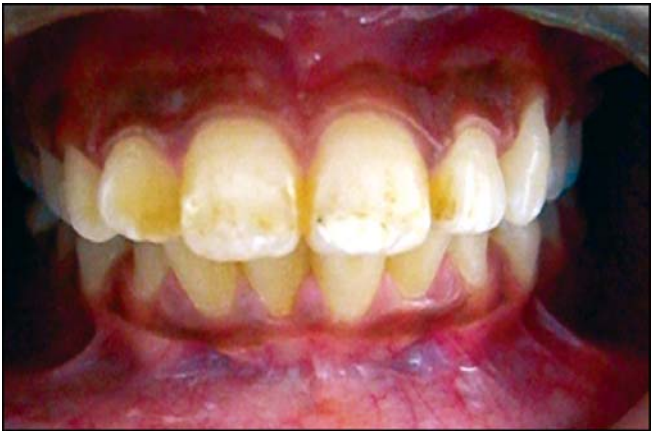


Fig. 6:6: Enamel hypoplasia (mild form)

- During calcification and maturation of matrix-enamel hypocalcification
- Presence of white spots or pits which may get discolored over a period of time.

Local/Postnatal Hypoplasia

- Generally affects single tooth
- White spots may become stained over a period of time as above mentioned in Figure 6.6.

Hypoplasia may Result due to Multiple Reasons

- From nutritional deficiencies
- Trauma or neurologic defects
- Cleft lip and palate
- Radiation
- Local infection
- Rubella embryopathy.

Colorado Stains/Brown Stains (Fig. 6.7)

Colorado stains was the name given Dr Frederick S. McKay for the condition dental fluorosis and described it in the literature as mottled enamel. Ingestion of excessive fluoride leads to enamel hypomineralization due to toxic damage to the ameloblastic cells. This may lead to development of pitting or crack in the tooth. The stains may range from isolated white flakes to smudged dark brown stains.

Exogenous Intrinsic Stains

These stains are present within the tooth substance but come from an outside source and not from within the tooth itself. Such stains present themselves as exogenous intrinsic stain such as:

- Stains occurring due to trauma (Fig. 6.8)
- Restorative materials such as:
  - Silver amalgam — grey to black
  - Copper amalgam — bluish green

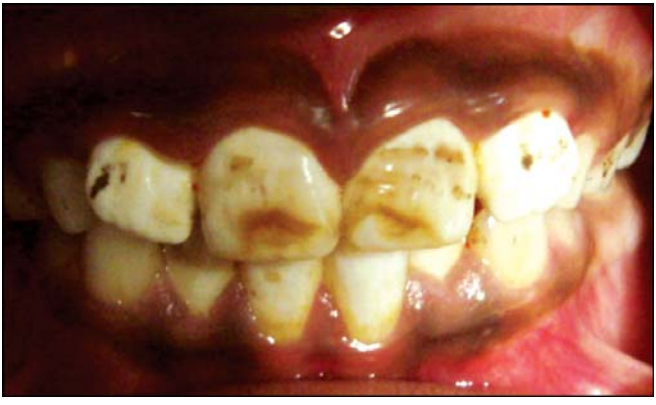


Fig. 6.7: Dental fluorosis/Colorado stains

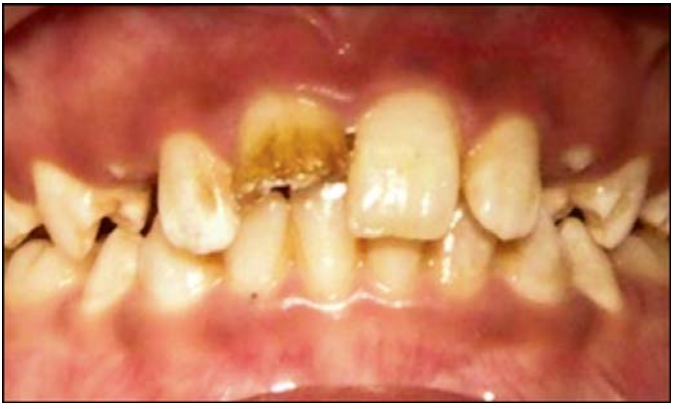


Fig. 6.8: Discolorations occurring due to trauma

- Mineral trioxide aggregate — grey
- Composites — light brown (Fig. 6.9)
- Cavit — moderate yellow/green stain
- IRM — reddish-orange discoloration.

Obturating material such as:

- Vitapex (calcium hydroxide with iodine)—yellow (Fig. 6.10)
- Root canal sealer (silver) — black
- Gutta percha—mild pinkish stain.

Drugs

- Stannous fluoride topical agent — dark brown
- Chlorhexidine — brown
- Ammonical silver nitrate — brown to black.

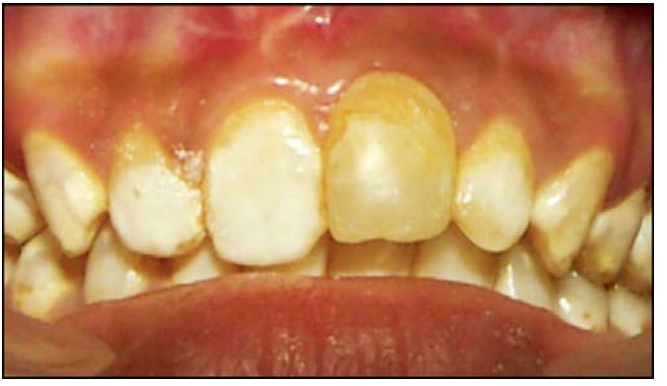


Fig. 6.9: Discoloration of maxillary anterior teeth post composite restoration

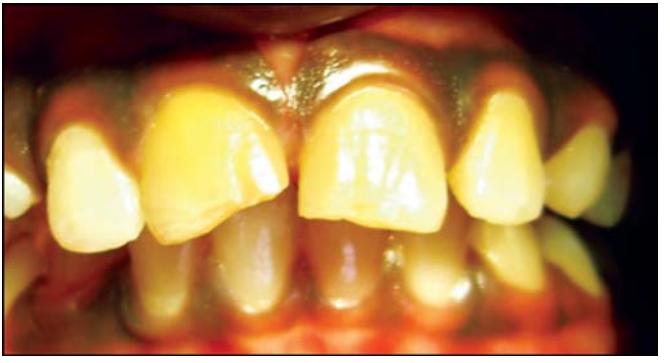


Fig. 6:10: Stains due to use of obturating material (Vitapex)

Other reasons:

- Internal resorption — pink
- Incipient caries — white chalky spots
- Cervical caries — black

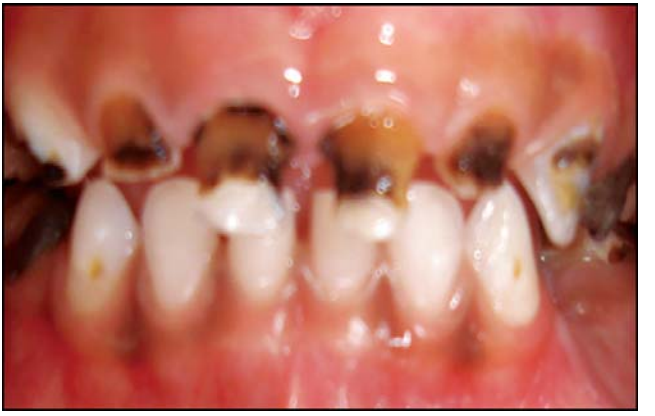


Fig. 6.11: Caries resulting in brownish discoloration

- Recurrent caries — grey to brown
- Rampant caries — black to dark brown (Fig. 6.11).

CHAPTER 7

Gingival Enlargement and its Management

The gingival and associated soft tissue of the periodontium may be enlarged in response to various interactions between the host and the environment. Although such enlargement usually represents an inflammatory response to dental plaque, or unreleased susceptibility may result because of various systematic conditions, medications and genetic predisposition. In rare instance certain benign and malignant neoplasm's may also cause enlargement of the tissue.

- Classification of gingival enlargement according to etiologic factors and pathologic changes.

INFLAMMATORY ENLARGEMENT

- Chronic
- Acute

ENLARGEMENT ASSOCIATED WITH SYSTEMIC DISEASES

Conditional Enlargement

- Pregnancy
- Puberty

- Vitamin deficiency
- Plasma cell gingivitis
- New specific conditional enlargement (Granuloma pyogenicum).

Systematic Diseases causing Gingival Enlargement

- Leukemia
- Granulomatous diseases.

NEOPLASTIC GINGIVAL ENLARGEMENT

- Benign lesions
- Malignant tumors.

False Enlargement

According to location and distribution:

- Localized
- Generalized
- Marginal
- Papillary
- Discrete.

GRADING OF GINGIVAL ENLARGEMENT

1. By Angelopoulos and Gouz (1972) and modified by Miller and Damm (1992).

   The height of gingival tissue was measured from the cementoenamel junction to true gingival margins.

Grade 0:	Normal gingiva
Grade 1:	Minimal enlargement that is less than 1 mm increase in size gingival covering the cervical third or less of anatomic crown.
Grade 2:	Moderate enlargement that is 2-4 mm increase in size or gingiva extends into the middle third of anatomic crown
Grade 3:	Severe enlargement that is nodular growth and increase in size 2/3rd of the crown.

2. By Seymour et al (1985) modified by Brunet et al (2001)—In bucco lingual direction. The increase in size of the papilla is measured from enamel surface at the interdental contact point to the outer papillary surface. Two score's, i.e. for buccal and lingual papilla are obtained.

Grade 0:	No sign of gingival enlargement
Grade 1:	Enlargement confined to under dental papilla
Grade 2:	Enlargement uniforms papilla and marginal gingiva
Grade 3:	Enlargement covers three quarters or more of the crowns

3. By Bokenkamp et al 1994

Grade 0:	Papillary thickness of less than 1 mm
Grade 1:	Papillary thickness—1 to 2 mm
Grade 2:	Papillary thickness—2 mm

INFLAMMATORY ENLARGEMENT

Chronic inflammatory enlargement (Fig. 7.1) is caused by prolonged exposure to dental plaque.

Clinical Features

- It originates as a slight ballooning of the interdental papilla and/or marginal gingiva.
- In early stages, a line preserving shape bulge is seen around the teeth. It progresses slowly uniting the part of the crowns. Is it usually painless, unless complicated by acute infection or trauma.
- Occasionally, discrete, sessile or pedunculated mass may be present, which may be inter proximal or on the marginal or attached gingival.
- Painful ulceration may occur by the folds of the mass and adjacent gingiva.

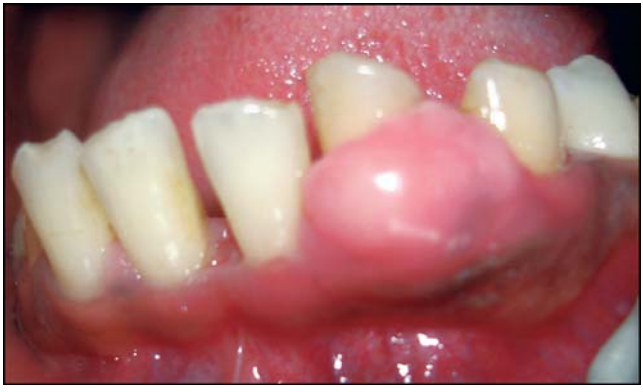


Fig. 7.1: Enlargement of inflammatory origin

- The gingiva may be deep red or bluish red in color, soft, shiny and friable in consistency, which may tend to bleed easily.
- Histologically, connective tissue shows preponderance of inflammatory cells. There is vascular engorgement with new capillary formation.
- The gingiva may be relatively firm, resilient and pink.
- Histologically, it shows greater fibrotic component with abundance of fibroblast and collagen fibers.

Mouth Breathing

Gingivitis and gingival enlargement may also be seen in individuals with mouth breathing habit.

- It is usually seen in maxillary anterior region
- It is probably attributed to irritation from surface dehydration
- Gingiva appears red and edematous with a diffuse surface
- Exposed area appears shiny
- Altered gingiva may be clearly demarcated from the adjacent unexposed gingiva.

ACUTE INFLAMMATORY ENLARGEMENT

Gingival Abscess

Localized purulent infection involving marginal gingiva or interdental papilla. It is a localized, painful, rapidly expanding lesion that is usually of sudden onset. It is generally limited to marginal gingiva or interdental papilla.

In early stages, it appears as a red swelling with a smooth, shiny surface. Within 24 hr to 48 hr the lesion

usually become fluctuant pointed and forms a surface orifice from which purulent exudates may be expressed.

In the later stages or in untreated cases, infection may spread to adjacent tissue spaces, which may ultimately result in the formation of space infection.

Periodontal Abscess

It is a localized purulent infection within the tissue adjacent to the periodontal pocket that may lead to destruction of PDL and alveolus bone. It appears as an avid lunation of the gingiva along the lateral aspect of root. Gingiva appears red and edematous, pus can be expressed from gingival sulcus by digital pressure.

DRUG-INDUCED GINGIVAL ENLARGEMENT

Gingival enlargement can also arise as consequences of administration of various drug regimens.

ANTICONVULSANTS

Phenytoin (5-Diphenylephentain) has been used to control seizures. Phenytoin selectively stabilizes manual excitation by blocking or interfacing with Ca^{+2} influx across the cell membrane. The clinical onset of gingival overgrowth is reported after 1 month of use of the drug. This reaches its maximum by 12 to 18 months.

Other anticonvulsants drugs which tends to show gingival overgrowth as their side effect includes; chronic use of valproic acid, carbamizapine or phenobarbitone in adult patients.

IMMUNOSUPPRESSANTS

Cyclosporine was first isolated in Switzerland in 1970 as a metabolite of the fungus species talypocladium but have little nature as an antifungal antibiotic. However as a cyclic polypeptide with potent immunes suppressive actions, cyclosporine, a survival of allergenic transplant involving skin, heart, kidney, liver, pancreas, bone marrow and lung. It is also used in the treatment of autoimmune disorder like Behcet diseases, rheumatoid arthritis, SLE, bullous pemphigoid and pemphigus, Crohn's disease and insulin—dependent diabetes.

Clinically and microscopically gingival hyperplasia induced by cyclosporine (Fig. 7.2) is similar to that induced by phenytoin.

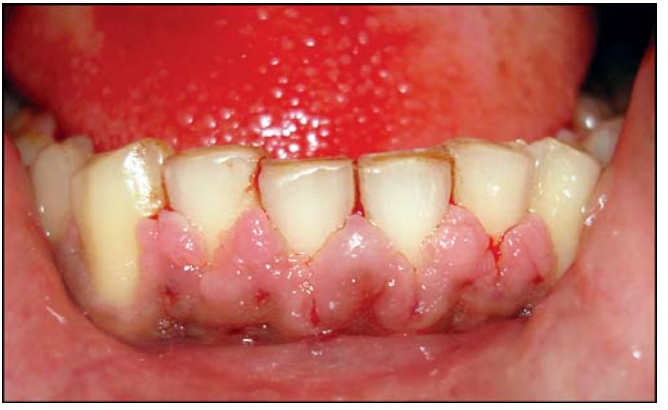


Fig. 7.2: Cyclosporine induced gingival enlargement

Calcium Channel Blockers

Nifedipine is an effective calcium channel blocker that has been effectively used in the management of cardiovascular disorders including hypertension, angina pectoris, coronary artery spasm and cardiac arrhythmia.

These drugs act by inhibiting calcium which can influx across the cell membrane of cardiac and smooth muscle cells, thereby blocking the mobilization of calcium intracellular. The primary side effects of calcium channel blockers result from excessive vasodilatation which manifests as facial flushing, dizziness, headache and edema.

Clinical Manifestation

Clinical Manifestation of nifedipine gingival enlargement (Fig. 7.3) frequently appears within 1 to 3 months after initiation of treatment with the associated medications. The

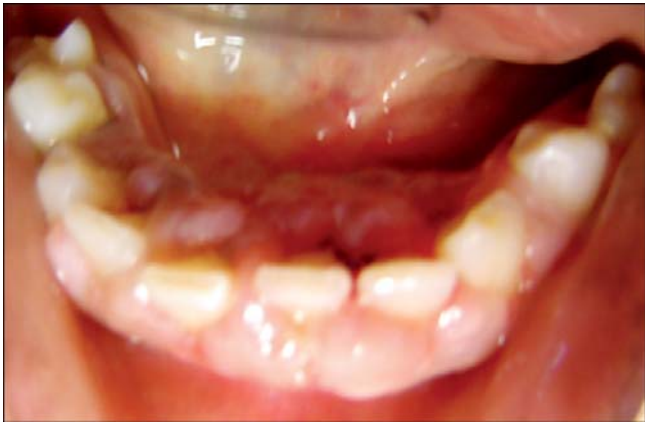


Fig. 7:3: Nifedipine induced gingival enlargement

growth starts as a painless, bead like enlargement of the facial and lingual gingival margins. As the condition progress, the marginal and papillary enlargement unites, they may develop into a massive tissue fold covering a considerable portion of the crowns and they may interface with occlusions.

When unaccompanied with a inflammation the lesion is mulberry shaped, firm, pale pink and resilient with a minutely copulated surface and no tendency to bleed. The enlargement characteristically appears to project from beneath the gingival margin from which it is separated by a linear groove.

The enlargement is generalized but is generally more severe in maxillary and mandibular anterior regions. The growth may interfere with aesthetics, mastication and speech dysfunctioning. Gingival overgrowth often impairs nutrition and access to oral hygiene, resulting in an increases susceptibility to oral infection, caries and periodontal disease.

Enlarged tissue may also result in malpositioning of teeth and also may affect the eruption sequence of teeth. Presence of enlargement makes plaque control difficult, often resulting in a secondary inflammatory process. Secondary inflammatory changes not only add to the size of the lesion caused by the drug but also produce a red lobulated surface demarcation and increased tendency towards bleeding.

- Phenytoin gingival overgrowth has also been observed in edentulous patients and beneath the boundaries of fixed partial dentures. Overgrowth has also been reported to occur around implant abutment.

- In an individuals who are immunosuppressed with cyclosporine, sometimes pebbly papillary lesion appear on the surface of larger copulation which have been associated with the presence of Candida albicans invading the gingival epithelium. Also, the affected gingival tissues are soft, bluish red, extremely fragile and bleed easily on probing as compared to tissue affected by Phenytoin. Also, cyclosporine induced overgrowth has not been reported in edentulous patients.
- Phenytoin and cyclosporine have immunosuppressant properties and as a consequence may protect the patient against periodontal breakdown.

IDIOPATHIC GINGIVAL ENLARGEMENT

Gingivostomatitis/Elephantiasis/Idiopathic fibromatosis/Hereditary gingival hyperplasia.

Familial Fibromatosis

Characterized by slowly progressive, non hemorrhagic enlargement of the maxillary and mandibular gingiva or may be localized to either jaw.

- Enlargement may be bilateral, thus the name, symmetrical gingival fibromatosis has been suggested by Wiktop 1971. Although the enlargement is not always symmetrical, the name was given as focal gingival fibromatosis.
- Gingiva is grossly enlarged and pink in color, firm and lathery in consistency with a minutely rebelled surface. Secondary inflammatory changes may be present.

- Teeth may be delayed in emerging and speech may be impaired
- Mastication may become painful, if enlargement extends to the occlusal surface.

Etiology

1. Unknown
2. Hereditary basis—autosomal dominant
3. Presence of teeth/plaque
4. Seen in sclerosis→A combination of
   Epilepsy + Mental deficiency + Angiofibroma

ENLARGEMENT IN PREGNANCY

Pregnancy associated enlargement may be marginal or gingival or may occur as a single or multiple tumors like masses. Enlargement is mainly related to elevated hormonal level during pregnancy which not only increases vascular permeability but also alter the sub gingival microflora with a predominance of p. intermediate.

Marginal Enlargement

It mainly occurs due to aggravation of previous inflammation and is seen in 10-70% of the cases. Clinically, there is generalized enlargement of marginal gingival which a more prominent interproximally, gingiva appears bright red and magenta in colures, soft and friable in consistency and has a smooth and shiny surface.

Bleeding occurs either spontaneously or on mild provocation.

Pregnancy (Granuloma) Tumor/Lobular Capillary Hemangioma/Angiogranuloma

This is a hyperactive inflammatory response due to bacterial plaque mediated by systemic condition, it usually occurs during the 1st and 3rd trimester of pregnancy.

Clinical Features

It usually appears as a mushroom like flattened spherical mass that protrudes from the gingival margin or interproximal space and attached by a sessile or pedunculated base. It tends to expand laterally and pressure from tongue and cheek attributes to its flattened appearance.

- It is usually seen in the upper anterior teeth.
- The granuloma appears dusky red or magenta in color (Rasberry like Appearance) with numerous deep red, pinpoint markings and a smooth, glistening surface (Fig. 7.4).

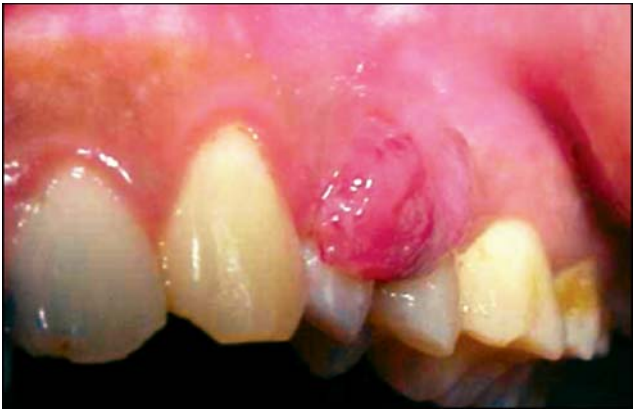


Fig. 7.4: Pregnancy induced gingival enlargement

- It has a semi firm consistency, but may be soft and friable, it does not innate the underlying bone.
- It bleeds spontaneously or even on mild provocation.
- It is usually painless, but painful ulceration may occur if it interferes with occlusion.

Etiology

- Pressure of local factors, i.e. microbial plaque is essential for the development of the lesion.
- It is also possible that inhabitation of collagens by progesterone result in accumulation of collagen within the connective tissue.
- An elevated level of sex hormones during pregnancy stimulates the macrophages to increase the production of ovarian factors, i.e. vascular endothelial growth factor (VEGF) and basic fibroblast growth factors (BFGF).

ENLARGEMENT IN PUBERTY

Gingival enlargement may be seen in males and females during puberty, in area of plaque accumulation. Enlargement is usually confined to marginal gingival with bulbous inter-proximal papilla seen more frequently on facial aspect than the lingual aspect. Features of inflammatory enlargement are seen as it undergoes spontaneous reduction after puberty, if plaque and calculus are removed.

ENLARGEMENT IN VITAMIN C DEFICIENCY/SCURVY

Vitamin C deficiency does not cause gingivitis but increases the severity of inflammation in minimal amount of plaque.

Clinical Features

- Gingival enlargement is marginal, appears bluish red in color, tender, grossly swollen and boggy
- It is soft and friable in consistency with a smooth and shiny surface
- Hemorrhage occurs spontaneously or on slight provocation in long-standing cases
- Gingiva develop a purple hue and surface necrosis with preudomembrane formation being a common feature
- Histopathology, the tissue shows chronic inflammatory cellular infiltration with a superficial acute response. There are scattered areas of hemorrhage with engorged capillaries.

PLASMA CELL GINGIVITIS

It was first reported in USA in 1986. It is considered to be allergic response or hypersensitivity reaction to some component of chewing gum, dentifrice or diet. It is also known as atypical gingivitis, plasma cell gingivostomatitis or plasma cell granuloma' (localized form).

Clinical Features

- More common is young females
- There is intense hyperemia and edema of free and attached gingiva
- Sharply demarcated from alveolar mucosa that appears red, soft and friable with fissuring of commissures
- Patient complains of burning reactions and is sensitive to dentifrices and seasoned foods.

PYOGENIC GRANULOMA

It is a tumor like non specific conditioned enlargement as response to minor trauma.

ETIOLOGY

Proportionally, it was thought to arise due to both mycotic infection or due to staphylococcal or streptococcal infections. Now, it is considered to be an exaggerated conditioned response to minor trauma.

Clinical Features

- It is usually seen on gingiva, but may also arise from lips, tongue and buccal mucosa.
- Lesion varies from a discrete spherical tumor like mass to a flattened, keloid like enlargement with a broad base.
- It is usually bright red or purple in color with a friable or soft consistency and which bleeds on slight provocation. The surface may show ulceration and purulent exudates may be present.

The growth may occur spontaneously to become a fibro epithelial papilloma.

LEUKEMIC GINGIVAL ENLARGEMENT

- Leukemic cells can infiltrate the gingiva and less frequently the alveolar bone.
- Enlargement is associated with:
  - Acute monocytic leukemia
  - Acute myelomonocytic leukemia
  - Acute lymphocytic leukemia

Enlargement is not reported to occur in edentulous patients.

Clinical Features

- Gingival enlargement may occur as a diffuse enlargement as extension of marginal gingiva or a discrete tumor like interproximal mass
- Gingiva appears bluish red and cyanotic with rounding of gingival margin
- Bleeding occurs either spontaneously or on a slight provocation
- Gingiva increases in size covering the entire crown
- Acute painful necrotizing ulcerating inflammatory involvement may occur in the crevice formed at the junction of enlarged gingiva and contiguous tooth structure.

WEGENER'S GRANULOMATOSIS

This was first described by Wegener 1936. It is characterized by granulomatous necrotizing lesions of the respiratory tract and kidneys including the oral defects.

Oral Manifestations

- Oral mucosal ulceration
- Abnormal tooth mobility
- Exfoliation of teeth
- Delayed healing
- Gingival enlargement may be localized to a single papilla or generalized.

- Gingiva is reddish purple in color and bleeds on stimulation.
- Appearance is of "Over Riped Strawberry".

Sarcoidosis

Sarcoidosis is a chronic granulomatous disease of unknown etiology which involves lymph nodes, lungs. It is seen in 2nd and 3rd decade of life and more common in blacks.

Oral Manifestations

- Sarcoid granuloma
- Sarcoid gingivitis
- Gingival tissues are hyperplastic with a granular appearance
- They are red in color and bleed on probing
- Painless enlargement.

Histopathologically, connective tissue shows disserts, non caseating epithelial cells and multinucleated foreign body type giant cells with peripheral mononuclear cells.

In some cases, servers and rapid periodontal destruction can occur.

Crohn's Disease

It is a granulomatous disorder characterized by non specific, sub mucosal alteration of the intestinal tract, accompanied by stenosis, necrotic breakdown and sccaring of mucosa.

Clinical Features

- Abdominal pain
- Pyrexia
- Intermittent diarrhea
- Joint pain
- Generalized malaise.

Oral Manifestations

- Aphthous ulceration is the most frequent lesions that are found
- Cobblestone appearance of oral mucosa
- Lesions are generally confined to buccal mucosa
- Diffuse erythematous, granular enlargement
- Gingival lesion are copulated
- Hypertrophy
- Ulceration may be present.

The cobblestone effect is due to a combination of hypertrophy and edema that produces an elevation of the mucosa. This soon becomes divided by linear fissures. Lesions in mucobuccal field resemble epulis fissuratum. Destruction may occur at angles of the mouth.

NEOPLASTIC GINGIVAL ENLARGEMENT

Neoplastic gingival enlargement comprises a small percentage of gingival enlargements in fact they comprise only 8% of all oral tumors occuring on gingiva.

Fibroma

Fibroma is a reacting hyperplasia of falcon's tissue rather than a neoplasm. It also known as "irritation fibroma", "traumatic fibroma" or "fibrous no duel".

Clinical Features

It appear as a well circumscribed smooth surfaced nodule about 1-2 cm in diameter. It may be firm and nodular and might be soft in nature. The growth is usually pedunculated.

- Variations
  - Giant cell fibroma
  - Peripheral ossifying fibroma.

Papilloma

Papilloma is a benign proliferation of surface squamous epithelium that may be caused by human papilloma virus (HPV-6, HPV-11) Papilloma appears as a solitary, pedunculated or sessile nodule exhibiting a cauliflower like surface. It may be white or slightly red in color.

Multiple papillomas like lesions may be observed in:

- Goltz and Gorlin syndrome
- Cowden's syndrome
- Down's syndrome.

Peripheral Giant Cell Granuloma

This arises interdentally or from the gingival margin usually present on labial surface. The growth may be sessile or pedunculated. It may vary in appearance from smooth regularly outshined mass to an irregularly shaped, multilobulated mass with protuberances and surface indentations.

- It is usually painless unless painful ulceration occurs due to trauma or infection.
- It may be spongy in consistency and color varies from pink to deep red or purplish red.

Gingival Cyst

Appear as localized enlargements that may involve the marginal and attached gingiva.

- It usually occurs in premolar area or on lingual surfaces.
- It occurs as a painless, soft tissue lesion but may erode the bone surface.
- Gingival cyst may from develop odontogenic epithelium or from surface extra traumatically implanted in the area.
- The cyst histologically shows a cavity lined by thin, flattened epithelium.

Other benign tumors known to occur on gingiva:

- Hemangioma
- Neurofibroma
- Neurilemmoma
- Ameloblastoma.

Squamous Cell Carcinoma

- It appears as an exophytic, irregular growth or may be flat, ulcerating lesion.
- It is usually painless unless secondary infection occurs.
- Early invasion is a common feature.
- It may also appear as a rapidly growing lesion emanating from extraction site.

FALSE ENLARGEMENT

False enlargement refers to the apparent increase in the size of gingiva due to increase in size of underlying osseous structures during teeth eruption.

Osseous Lesion

- Tori
- Exostosis
- Paget's dysplasia
- Central giant cell granuloma
- Ameloblastoma
- Osteosarcoma.

UNDERLYING DENTAL TISSUES

Labial gingiva may show a bulbous marginal distortion caused by super imploration of the bulk of the gingiva or as the normal prominence of enamel. Such an enlargement may persist only until the junctional epithelium has migrated to CEJ.

CHAPTER 8

Halitosis

Halitosis in general means bad breath, and is also known as oral malodor. The term halitosis originated from the Latin word *halitus*, breath, and the Greek – *osis*, meaning abnormal condition. Oral malodor is a common complaint that may periodically affect people of all age groups. The term oral malodor is a generic descriptor for foul smells emanating from the mouth which encompasses ozostomia, stomatodysodia, halitosis (either pathological and physiological, fetor oris or fetor ex ore). The latter term denotes local or systemic production of malodor. Oral malodor may rank only behind dental caries and periodontal disease, as the most common cause of patients visit to the dentist. Surprisingly a problem of this magnitude with such high priority to the public has traditionally been neglected by the dental profession. Oral malodor, which is commonly noticed by patients, is an important clinical sign and symptom that has many etiologies, which include local and systemic factors. It is often difficult for the clinician to find the underlying pathologies, since the bad breath usually comes from the mouth itself, the dentist should be the first professional whom individuals turn to for help.

DEFINITIONS

Halitosis is a general term denoting unpleasant breath arising from physiological and pathological causes from oral and systemic sources.

Some authorities make a distinction between the term Halitosis and Fetor oris. Bad breath not arising in the mouth is halitosis, whereas odors that are caused orally are fetor ex oris.

According to **Grant,** this distinction does not seem to be important and the term halitosis is used for any kind of bad breath.

According to **Glickman,** Halitosis also termed Fetor ex ore or Fetor oris is foul or offensive odor emanating from the oral cavity.

CLASSIFICATION

There are numerous classifications of halitosis which have been described by different authors:
Halitosis can be classified based on:

I. Etiology
II. Patients criteria

Based on Etiology

Dominic et al (1982) Categorized Halitosis as follows:

a. Halitosis due to local factors of pathologic origin.
b. Halitosis due to local factors of non-pathologic origin.
c. Halitosis due to systemic factors of non-pathologic origin.
d. Halitosis due to systemic factors of pathologic origin.
e. Halitosis due to systemic administration of drugs.
f. Halitosis due to xerostomia.

Bogdasarian (1986) Classification based on Etiology

a. Normal breath of physiologic mouth odor.
b. Odors from oral conditions.
c. Odors from nasopharynx, pharynx and lungs.
d. Odors excreted via lungs.

Dayan et al (1982) Divided Foul Odor into Three Groups Namely

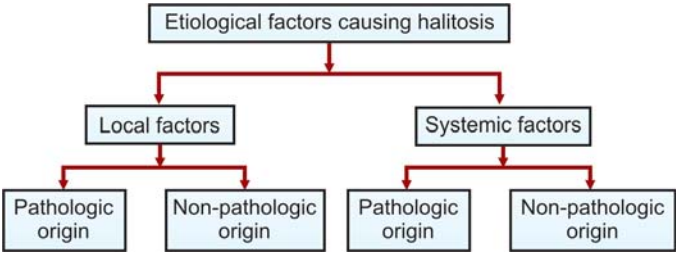
a. Odor eminating within the oral cavity.
b. Odor eminating from regions immediately adjacent to the oral cavity.
c. Odor eminating from lungs.

Based on Patients Criteria

Iwakuru et al (1994) Classified Halitosis as:

a. *Primary halitosis*: Patients do not actually have halitosis but suffer from imaginary halitosis.
b. *Secondary halitosis*: Patients actually have halitosis.

Halitosis, a condition that causes a severe social handicap to those who suffer from it.



Halitosis due to Local Factors of Pathologic Origin

Halitosis can be caused by local conditions such as:

- Poor oral hygiene
- Extensive caries
- Gingivitis
- Periodontitis
- Open contact allowing food impaction
- Vincent's disease
- Hairy or coated tongue
- Infectious malignant neoplasm's of the oral and pharyngeal cavities
- Healing extraction wounds
- Necrotic tissue from ulceration
- Dentigerous cyst
- Increase in gram-negative filamentous organisms.
- Increase in plaque pH to 7.2, formation of indoles and amines in the oral cavity
- Other causes such as chronic sinusitis, rhinitis, lethal granuloma, pharyngitis, tonsillitis, syphilitic ulcers, cancrum oris, tumors of the nose, dental abscess, cancerous tumors of trachea, bronchi, chronic fetid bronchitis, etc.

In adults, chronic periodontal disease is the major cause of halitosis. Periodontal pockets produce hydrogen sulphide, which gives off an offensive smell.

Halitosis due to Local Factors of Non-pathologic Origin

- Stagnation of saliva associated with food debris
- Dentures
- Excessive smoking
- Diet and dietary habits
- Endodontic patients

Halitosis due to Systemic Factors of Pathologic Origin

There are various systemic conditions/disease, which can cause halitosis:

- Diabetes mellitus
- Liver failure
- Acute rheumatic fever
- Lung abscess, tuberculosis, bronchiectasis
- Uremia, kidney failure
- Blood dyscrasias
- Syphilis
- Scurvy
- Non-lipid reticuloendotheliosis disorder, e.g. eosinophilic granuloma
- Letterer-Siwe disease and Hand-Schüller-Christian disease
- Inborn errors of metabolism, e.g. trimethylaminuria
- Cancer
- Acidosis.

Halitosis due to Systemic Factors of Non-pathologic Origin

- Metabolites from ingested food that are excreted through the lungs can cause halitosis.
- A vegetarian has fewer tendencies to produce halitosis than an excessive meat eater because there is fewer degraded waste by products of proteinacious substance in vegetables.
- Garlic, onion, leeks, alcohol, etc impart odors to the breath by being absorbed into the circulatory system.
- Excessive alcohol drinking leads to alteration of the microbial flora and causes proliferation of odor fermenting organisms capable of producing halitosis.

- Young children aged 2-5 years may have a sweet fetid mouth odor due to their tonsil crypts lodging food and bacteria as halitosis is also common following tonsillectomies.
- Vincent's disease and its odor are most frequently seen in teenagers.
- Some patients with dysmenorrhea suffer from a mousy odor resembling decayed clotted blood.
- From time to time, one may experience hunger odor with hunger sensations.

Halitosis due to Systemic Administration of Drugs

Systemic administration of drugs can also cause halitosis. Usually drugs containing iodine or chloral hydrate can reach the breath thus causing halitosis.

Due to Systemic Administration (Table 8.1)

Table 8:1: Halitosis due to systemic administration of drugs

Drugs	*Main therapeutic use*	*Mechanism*
Insosorbide dinitrate	Anginal therapy	Both intrinsic odor of the drug and metabolic end products of the drug
Ethyl alcohol	Anginal therapy, sedation	Odor of the drug reaches the breath via the systemic route
Chloral hydrate	Hypnotic sedation	
Amyl nitrate	Anginal therapy	
Diuretics	Anti-hypertension, anti-edematic	
Phenothiazine and their derivatives	Schizophrenia, antiemitics psycho-sedative	Due to xerostomia caused by drug
Tranquilizers	Sedation	

Halitosis due to Xerostomia

Xerostomia means decreased or absence of saliva, which can cause halitosis. It is seen in conditions like Sjögren's syndrome, salivary gland aplasia, Mikulicz's diseases, radiation therapy exceeding 800 rads, macroglobulinemia with salivary gland involvement, Hereford's disease, diabetes, systemic and metabolic disease with high fever and dehydration, emotional disturbances and poor oral hygiene. The various types of oral odor includes:

Morning Breath

Oral malodor is common on awakening (morning breath) usually as a consequence of low salivary flow and stagnation of saliva during sleep. Morning breath rarely has any significance and can be readily rectified by eating, tongue brushing, tooth brushing and by rinsing the mouth with fresh water.

Exogenous Malodor

Halitosis at other times of the day is often the consequence of eating various foods such as garlic, onion or spices (in curries), cabbage, brussels sprouts, cauliflower, radish or habits such as smoking or drinking alcohol. Avoidance of these foods and habits is the best means of prevention.

True Oral Malodor (Endogenous Malodor)

Halitosis that is not due to the above simple causes is most often a consequence of oral bacterial activity typically arising from anaerobes arising is due to:

- Poor oral hygiene
- Gingivitis (ANUG)
- Periodontal disease
- Infected extraction sockets
- Oral sepsis
- Postoperative residual blood
- Debris under fixed or removable appliances
- Ulcers
- Dry mouth
- Putrefaction of postnasal mucus drip stagnating on the tongue
- The microorganisms implicated are predominantly gram-negative anaerobes and includes:
  - Porphyromonas gingivalis
  - Prevotella intermedia
  - Fusibacterium nucleatum
  - Bacteroides forsythus
  - Treponema denticola
- Recently gram-positive bacteria have been implicated because they can denude the available glycoprotein's of their sugar chains, enabling the anaerobic gram-negative proteolytic bacteria to breakdown the denuded protein resulting in the production of gases which include:
  - Volatile sulphur compound (methyl mercaptan, hydrogen sulphide).

  - Diamines (putrescine and cadaverine).
  - Short-chain fatty acid (butyric, valeric and propionic).
- In absence of oral infection systemic causes for oral malodor are:
  - Starvation
  - Drugs
  - Systemic disease.

Psychogenic Malodor

This is a group of patients who are by no means has any evidence of halitosis which can be detected even with objective testing and the halitosis may be attributable to a form of delusion of mono-symptomatic hypochondriasis (self-halitosis; halitophobia). Such patients rarely wish to visit a psychological specialist because they fail to recognize their own psychological condition and may have latent psychosomatic tendencies. Medical help may be required to manage these and other patients with the systemic background to their complaint.

Many of these patients will adopt a behavior to minimize their perceived problem, i.e.

- Using chewing gum, mints, mouthwash or sprays.
- Covering their mouth while talking.
- Avoiding or keeping a distance from other people.
- Cleaning their tongue.
- Avoiding social situations.

EXAMINATION

Physical Examination

A thorough physical examination of the head and neck area must be performed as a bad breath can arise from other regions apart from oral cavity. If a strong smell arises from nasally expired air, it indicates that the lesions or disease of nasopharynx, nose or sinuses whereas a more noxious odor from orally expired air indicates an oral, oropharyngeal, hypopharyngeal or rarely a gastric source.

Extraoral Examination

A thorough extraoral examination may eliminate considerations. Some extraoral causes of bad breath or degenerative process of lip tissue can cause the patient to become aware of malodor.

Infections or tumors in the oropharynx may produce enlarged lymph nodes. Salivary gland swelling can result in xerostomia or drainage of purulent material. The patency of nasal passage can be established by having the patient pinch one nostril closed and blow through the other nostril.

A stronger smell from the nasally expired air can indicate lesion of disease of nasopharynx which is also a common site of bacterial overgrowth contributing to bad breath.

Chronic sinusitis, tonsillitis, choanal atresia and nasal foreign bodies have been reported to cause halitosis. Malodor can be an early symptom of nasal tumors. Vomiting or eructation (belching) can also result in bad breath.

Schiff Mann has stated that specific odors can be associated with infections, nutritional and mental diseases.

Intraoral Examination

Intraoral examination consists of assessment of all abnormal findings of soft tissue, hard tissue and supporting structures such as dental decay, periodontal disease, lesions present on buccal mucosa, labial mucosa, tongue, palate, vestibule and floor of the mouth of bacterial, viral and fungal origin which can contribute in the production of the malodor due to putrefaction of bacteria.

Radiographs may be necessary to evaluate carious extension in the pulp of the teeth. Carious sites are sites for food retention and putrefaction. Restorations, crowns, appliances, fixed and removable prosthesis should be examined carefully to rule out food stagnation.

Examination of tongue may reveal a potential source of bad breath. Tongue appears to be a principle contributor to oral malodor in periodontally healthy children as the surface anatomy of tongue provides an ideal environment for collection of desquamated cells, food debris and bacterial growth capable of malodor production.

Such patients have highly exaggerated fear that they suffer from bad breath, which might be a delusion associated with mono-symptomatic hypochondrial psychosis. All the above mentioned investigations may reveal the factors causing halitosis but as such are not the test to measure halitosis. Self assessed questionnaire is needed for the diagnosis of halitophobia. Medical referral is indicated when a non-physical cause for perceived oral malodor is suspected.

Halitosis has got a multi-factorial etiology. It can be caused either by local factors or systemic factors. Therefore the treatment approach should be primarily based on elimination of etiological factors. Since halitosis is mainly caused by local factors, removal of these factors should be the first step in treating halitosis especially when the medical history is essentially negative. The eradication of periodontal pockets, improvement of oral hygiene, restoration of carious lesion and open contacts between the teeth, prophylaxis through thorough polishing and scaling, extractions of unrestorable teeth, and correction of any other defects that could minimize the accumulation and putrefaction of food debris, elimanating stagnated saliva and degradation of breakdown of protein byproducts would solve most cases of halitosis.

Also the best way to treat bad breath is to motivate patients to practice good oral hygiene and to ensure that their dentition is properly maintained.

Oral hygiene maintenance includes.

Periodic and professional maintenance of oral hygiene with particular attention to potential foci of microbial colonization. Since oral diseases are a major cause of bad breath, prompt diagnosis and treatment that includes continuous home oral hygiene measures with frequent recalls and maintenance.

Mechanical Methods

- *Tooth brushing*: Brushing with toothpaste may reduce malodor for as long as two hours. Sodium bicarbonate dentifrice appears to be superior to fluoride dentifrice for the reduction of volatile sulphur compounds levels.

- *Flossing*: It is one of the most effective day to day home treatments that do not involve antimicrobial agents. However the level of patient compliance in daily flossing is low but once the connection is made between flossing and fresh breath, compliance improves. Having patient smell the floss after each use is a good way to illustrate the importance of regular flossing in breath odor. Other interdental cleaners (such as tooth picks) also can be effective in identifying and cleaning sites of odor production.
- *Tongue cleaning*: Gentle but effective deep tongue cleaning should become a part of the daily hygiene routine. To prevent tongue odor, the tongue should be cleaned in a gentle but thorough manner. The patient should be reminded that the posterior portion of the tongue is the least accessible but usually smells the worst.

Chemical Methods

Mouth rinses may be indicated in those patients who even after maintaining good oral hygiene continue to have malodor. Many commercially available mouth rinses claim to effectively eliminate malodor, but most of them have a masking effect. Other products have antibacterial mechanisms, but many of these rinses have insufficient strength to control odor for longer than few hours. This in turn, enhances the ability of the mouthwash to physically remove oral microorganisms in addition to inhibiting those that remain behind.

Mouth rinses are more effective if used directly prior to bedtime, since residue of mouth rinse may remain in the mouth for a longer period and can help off set the lack of salivary flow during the night.

Mouth rinses may also help in the initial diagnosis of bad breath. Reduction in oral malodor following several uses of a potential antibacterial mouth rinse is a good indicator of oral environment. The best mouth rinse is not produced commercially, but by our own salivary glands. Bad breath levels often correspond with reduced salivary flow. Morning breath (so called "jungle breath") results from build up of microbes and volatile microbial products during the night when salivation is reduced.

Thus any regimen, which promotes saliva flow especially in conjunction with mastication, will often reduce bad breath. Patients may be encouraged to chew fibrous vegetables during the day, or sugarless gum. Patients should also be encouraged to drink ample amount of water. If any systemic cause is suspected, it should be investigated thoroughly and appropriate surgical or medical referral should be done.

Masking substances may be of some help. Peppermint follows the same metabolic route as garlic, hence can mask its odor. Oil of winter green is some what less potent but also helpful. There are also over the counter cures present which are in the form of mints, chewing gums, sprays and mouth rinses.

Patients suffering from halitophobia should be provided psychiatric treatment. They should be counseled and eliminate their belief that they suffer from bad breath so that they can face the people more comfortably (Table 8.2). The

Table 8:2: Prevention of halitosis

- Take regular meals
- Brush your teeth after eating
- Eat a good breakfast, as its common on awakening
- Avoid any odoriferous food such as onions, garlic and spices
- Treat any identifiable cause
- Avoid habits that may worsen breath odor
- Keep oral hygiene regular and good
  - Tooth brushing
  - Flossing
  - Prophylaxis
  - Rinsing twice daily with chlorhexidine mouthwash
- Keep your mouth as moist as possible
- Clean your tongue before going to bed
- If you have dentures or removable appliance, keep them clean

patient's response to the therapy, if there is no response to therapy in a complaint of patient, re-evaluate the working diagnosis. Although consultation and treatment may result in dramatic reduction in bad breath, patients may find it difficult to sense the improvement themselves. This problem can be addressed with the help of the confidant, who can help monitor changes over time. Cases in which the odor (or the subjective complaint) persists, the patient should be referred to a physician for further assessment.

CHAPTER 9

Oral Ulcers

Effective treatment of ulcer depends on correct diagnosis. Most of the ulcers are painful.

Ulcers can be divided into:

1. Oral ulcerations with acute onset
   - Viral infections
   - Bacterial infections
2. Chronic or recurrent course
   - Autoimmune diseases
   - Genetic diseases
   - Granulomatous diseases
   - Malignancy.

Localized Bacterial Infections

Most of the ulcers are due to periapical and periodontal abscess. There may develop pathological sinus. Opening of sinus forms as an epithelial lined drainage from periapical or periodontal abscess. Thus sinus opening is associated with edematous enlargement, erythema and white/yellow center.

Traumatic Ulcer/Decubitus Ulcer (Fig. 9.1)

It results due to physical injury, which may occur in various forms such as biting the mucosa, denture irritation, tooth

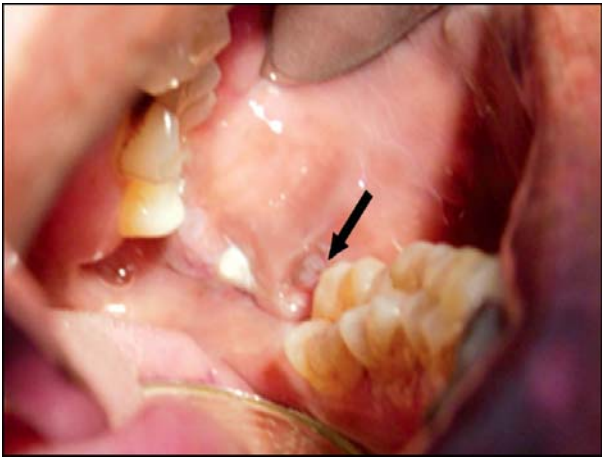


Fig. 9.1: Constant cheek biting habit result in the traumatic ulcer of buccal mucosa

brush injury or an injury to the mucosa by an external stimulant. Ulcer is solitary covered by a white or tan fibrin clot and generally located at lateral border. It is generally seen on the lateral border of the tongue, buccal mucosa, lips and occasionally on the palate. It can be painful. Recurrent trauma can make it firm and elevated with rolled borders. After removing the cause ulcer may heal within 1-2 days.

Factitial Injuries

These are accidental, self induced injuries based on ones habit such as lip biting, cheek biting also referred as ***pathomimia mucosae oris***. These injuries constitute a group of lesions, related only to a manner to which they

are produced. They lack any particular anatomy, etiology and microscopic similarities to other lesions.

Sore spots: These are traumatic ulcers caused by denture irritation. The lesion generally develops within a day or two after the insertion of a new denture. The reason behind this could be the over- extension of the appliance or the high spots on the inner aspect of the denture. The ulcers are small, painful, with an irregular shape covered by a grey necrotic membrane and surrounded by an inflammatory halo.

Correction of underlying cause, i.e. relief of flanges and reduction of high spots will be the treatment of choice.

Denture sore mouth/denture stomatitis is a condition that occurs in a patient using dentures. The condition is non-allergic and can be caused by *Candidia albicans,* however, it has also been associated with sweat retention syndrome. The mucosa below the denture appears red, swollen, smooth or granular and painful. Multiple pinpoint foci of hyperemia can be seen usually involving maxilla. The lesion is restricted only to the area in contact with the denture and is extremely painful.

TUBERCULOUS ULCER

It is caused by *Mycobacterium tuberculosis*. It is an aerobic, slender, non-motile, non-spore forming, rod shaped organism. Most of people get infection in first year of life. Primary infection usually takes place in lungs. Hemoptysis, abundant sputum and pleuritic pain are very common.

Tuberculous lesions of the oral cavity are secondary to pulmonary infections. Important oral lesions are tuber-

culous ulcers, tuberculous gingivitis, tuberculous osteomyelitis and tuberculosis of salivary gland. Tuberculous infections may produce nodules, vesicles, fissures, plaques and granuloma. For tongue ulcers it is the most common site of occurrence. On lateral borders these appear either single or multiple lesions. On palate these may appear as small ulcer or granuloma.

Lesions often produced are small, granulating ulcers at the mucocutaneous junction. Gingival lesions of tuberculosis usually produce granulating ulcers or erosive lesions with gingival hyperplasia.

Salivary gland itself may be involved or it may affect the intra or periglandular lymph nodes. There may be swelling or abscess formation of gland. Chronic osteomyelitis of maxilla and mandible may occur in case of tuberculosis. There will be pain, swelling or sinus formation.

PRIMARY SYPHILIS

It can be acquired or secondary:

Primary syphilis The characteristic primary lesion of syphilis is called '**chancre**' and is a solitary, painless, indurated, ulcerated or eroded lesion. To start, it develops as a dull red macule which becomes eroded. Regional lymph nodes are enlarged, painless and are rubbery.

Secondary syphilis usually appears in about 6-8 weeks after the appearance of primary chancre. It is characterized by skin and mucosal lesions. There will be generalized lymphadenopathy. Skin lesions may be macular, papular, follicular or lenticular. Coin like lesions are common on

face. Areas of hyper-pigmentation can be seen on soles and palms. Lymph nodes are painless, discrete and not fixed to the surroundings. With or even without treatment lesions heal within 2-4 weeks.

Tertiary syphilis develops after 5-10 years of primary lesion affecting every organ of the body. It mainly affects CNS and CVS. Typical lesion is '**gumma**'. It is a localized chronic granulomatous lesion having either nodular or ulcerated surface. Ulcer is punched out vertical walls. Base is dull, red granulomatous with irregular outline. Skin lesions leave tissue paper like scars. Pregnant lady may deliver a child with congenital syphilis. Bone lesions may result in osteomyelitis and destruction of joints.

ORAL MANIFESTATIONS OF SYPHILIS

Primary syphilis 'Chancre' occurs on lips, tongue, palate, gingiva and tonsils within 3 weeks. In males upper lip is involved and in female's lower lip is involved. Tongue ulcer is seen on lateral border of anterior two-third of it. Chancres can be painful if secondary infection develops. Lymph nodes are developed bilaterally.

Secondary syphilis These lesions are mucocutaneous patches are that flat, multiple, irregular or circular round erosions. Snail track like ulcers are seen. It is contagious. Tongue becomes fissured. Oral condylomata are round, white velvety lesions.

Tertiary syphilis Lesions are seen on soft and hard palate. These start as firm, pale, nodular masses. Ulcers are single and painless. Necrosis and continuous sloughing may result in perforation of palate.

HISTOPLASMOSIS

It is caused by fungus histoplasma capsulatum. Oral involvement is secondary to pulmonary involvement. Oral lesion may be papule, nodule or an ulcer. Ulcer grows slowly. Cervical lymph nodes are enlarged and firm diagnosis is confirmed by culture of infected tissue.

Treatment includes ketoconazole for 6-12 months.

BLASTOMYCOSIS

It is a fungal infection due to blastomyces dermatidis. It may cause shortness of breath, weight loss and blood tinged sputum. X-ray chest may show chest lesion. Oral lesion is a nonspecific painless verrucous ulcer with undurated borders. Hard nodules with radiolucent jaw lesions may also be seen. Diagnosis is made on biopsy.

MUCOMYCOSIS

It is caused by saprophytic fungus. Fungus is generally non-pathogenic. Fungus invades arteries resulting in thrombosis and ischemia. Fungus may spread from oral and nasal area to brain causing death. In oral cavity it results in ulceration of palate resulting necrosis. Ulcers may develop on gingivae, lip and alveolar ridge. Treatment includes surgical debridement of an infected area with Amphotericin – B for 3 months.

PRIMARY HERPETIC GINGIVOSTOMATITIS

Both herpes simplex virus I and II can infect.

Clinical Features

- Abrupt onset of fever, malaise and lymphadenopathy of cervical chain of glands
- Vesicles rupture soon forming painful ulcers
- Lesions are highly infectious
- Gingiva is erythematous, ulcerated and enlarged
- Lesions resolve automatically within 10-14 days

Acyclovir is useful in early limiting of the disease.

ERYTHEMA MULTIFORMAE

It is a non-viral condition. It can affect skin and sub-mucosa separately or in combination. In skin central vesicle is surrounded by concentric erythematous skin colored rings. Ulcers are painful and most commonly located on lips, tongue and buccal mucosa.

EPIDERMOLYSIS BULLOSA (FIGS 9.2 TO 9.4)

It is a genetic skin disease which causes oral ulceration. Ulcers develop recurrently. Skin lesions are bullae which rupture to give ulcers. Oral ulcers vary in frequency and severity. Complications include ankyloglossia. These cannot be cured or effectively controlled. Only supportive line of treatment is given.

APHTHOUS STOMATITIS

It is an idiopathic, non-infections inflammatory disease. Ulcers develop recurrently on non-keratinized oral mucosa.

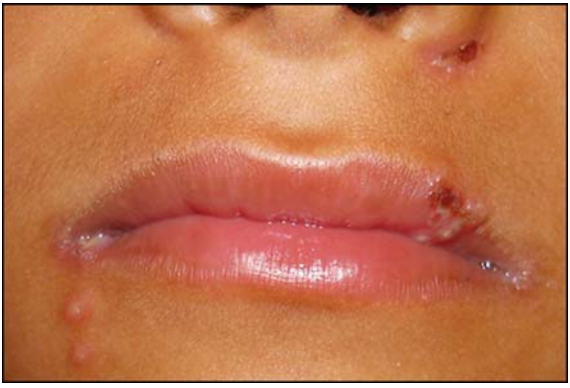


Fig. 9.2: Extraoral ulcers involving lips, angles of the mouth and nose

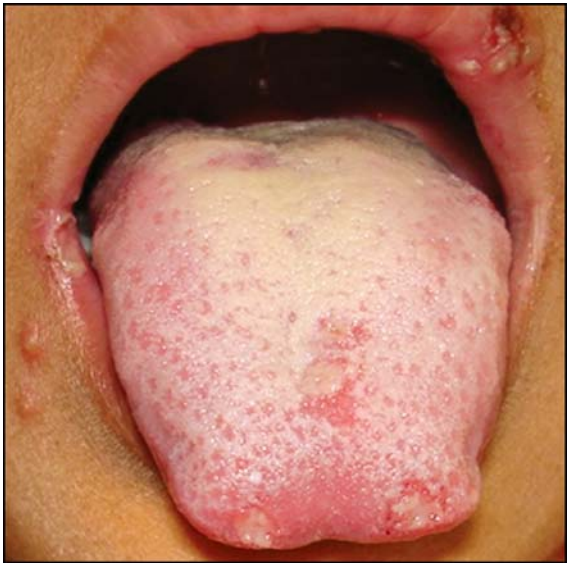


Fig. 9.3: Intraoral lesions involving bifid tongue and ulcers

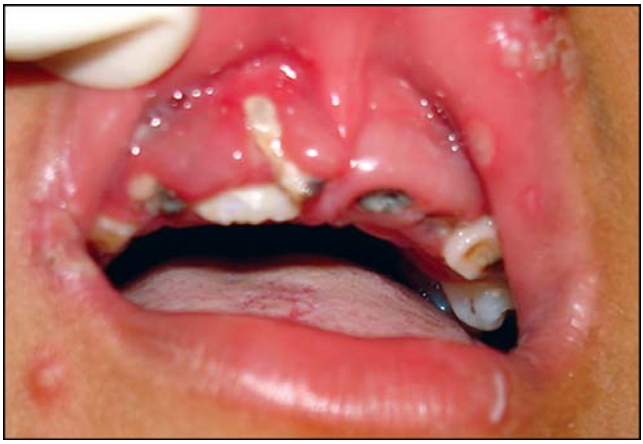


Fig. 9.4: Involvement of labial mucosa, gingiva

There are 3 types of ulcers:

- Minor aphthous stomatitis produces small ulcers which heal within 10 days.
- Major aphthous ulcers produces larger ulcers and takes longer time to heal. It heals by scarring and at least one ulcer is always present.
- Herpetiform aphthae presents multiple painful ulcers of 1 to 20 cm affecting non-keratinized area.

Ulcers are superficial with an erythematous halo. These are non-indurated and painful.

REITER'S SYNDROME

It is a immune mediated disease. It produces:

- Oral aphthous like ulcers
- Polyarthritis
- Uretheritis
- Conjunctivitis.

BEHÇET'S SYNDROME

It is an idiopathic disorder characterized by oral, ocular and genital lesions. Oral features includes aphthous like ulcers. Systemic corticosteroids help.

CICATRICIAL PEMPHIGOID

It is an autoimmune disease and is also known as benign mucous membrane pemphigoid. Mucous membranes including oral and conjunctival surfaces develop lesions. There develops transient vesicles and bullae formation which on rupture, gives ulcers. Ulcers of cicatricial pemphigoid tend to heal more with scarring. Oral lesions can be widespread.

Long-term management requires local and systemic corticosteroid.

NOMA

It is a rare form of gangrene localized to orofacial region. Tissue necrosis progresses after infection with anaerobic bacteria.

It produces non-specific ulcer of buccal or alveolar mucosa. Ulcers fail to heal producing necrosis. Broad spectrum antibiotics may be help.

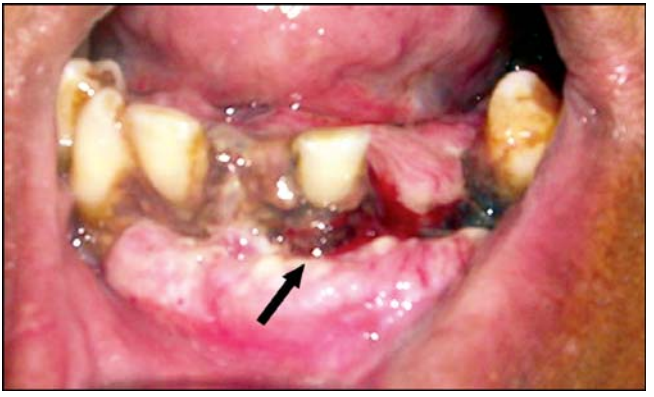


Fig. 9.5: Non-healing ulcer of gingivolabial sulcus

MALIGNANT ULCER (FIG. 9.5)

An ulcer which is not healing for more than a month, malignancy may be ruled out. Carcinoma produces:

- White areas of epithelial thickening with red foci of epithelial thinning.
- Destructive process is caused by growth of tumor.
- Appearance is heterogeneous.

CHAPTER 10

Radiolucencies of Jaw

There are many radiolucencies seen in mandible and maxilla. Appearance may differ according to age and view of films.

Mandibular Foramen

It is usually situated just above the midpoint in the medial surface of ramus. It receives inferior dental nerve and artery. It may be identified on panoramic film. It is up to 1 cm in diameter.

Mandibular Canal

It can be seen on panoromic or periapical view of molar region. It has thin radiopaque lines around it. There is great variation between patient due to width and prominence of mandibular canal. Its course may vary and its obliteration may be suggestive of an underlying pathology.

Mental Foramen

It permits the exit of mental branches of mandibular artery and nerve. It lies near premolar apices.

Lingual Foramen

It is located well below central incisors. It is surrounded by prominent radiopaque ring of cortical bone. It is about 1 or 2 mm in diameter.

Submandibular Fossa

It is a concave area on the lingual side of mandible below molar area. It accommodates submandibular salivary gland.

Intramaxillary Suture (Fig. 10.1)

It can be identified as a thin vertical radiolucency in the midline between central incisors.

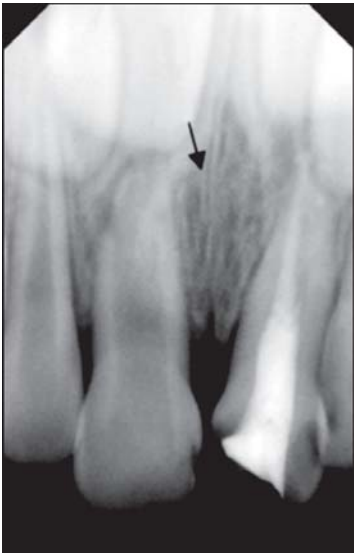


Fig. 10.1: Thin radiolucency in the midline can be identifies as intramxillary suture

Nasolacrimal Duct

It is a duct on each side and is enclosed in such a way that in one periapical film it is hardly seen.

Maxillary Sinus (Fig. 10.2)

It is seen as a well defined radiolucency with thin sharp radiopaque borders.

Greater Palatine Foramen (Figs 10.3A and B)

It is present bilaterally and on each side it is a round or ovoid ill defined radiolucency over apices of maxillary second and third molars.

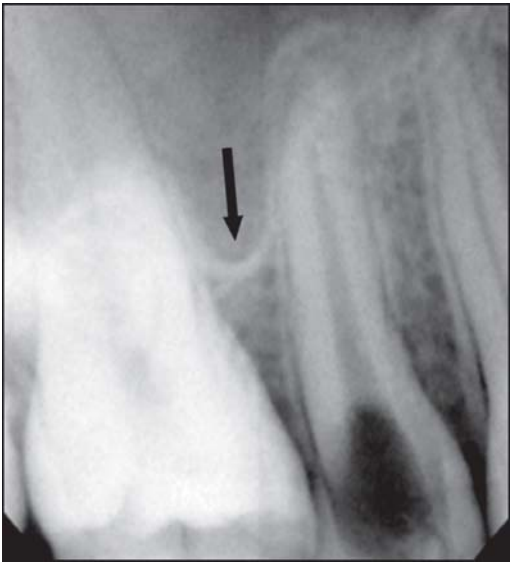
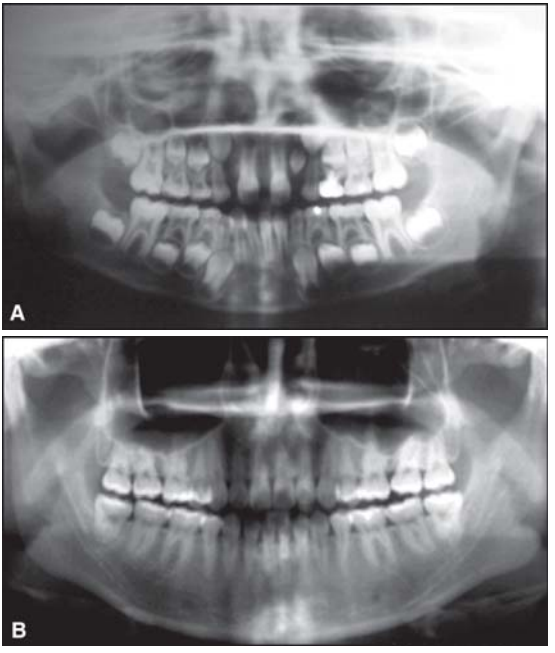


Fig. 10.2: Sharp radiopaque borders of maxillary sinus



Figs 10.3A and B: Panoramic film showing normal radiolucencies of jaw in a child and an adult

Nutrient Canal

It appears as a line of ribbon like radiolucencies of fairly uniform width found between the tooth.

PERIAPICAL RADIOLUCENCIES

Periapical Cyst (Fig. 10.4)

The periapical cyst is the commonest pathological radiolucency. It is a well circumscribed radiolucency,

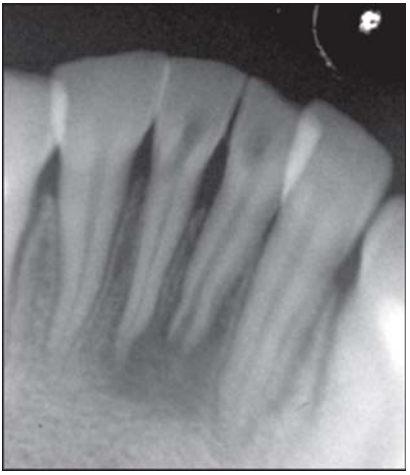


Fig. 10.4: Periapical cyst

round in shape and surrounding the apex of tooth. It may or may not have a thin radiopaque border. Few granulomas becomes larger than 2.5 cms in diameter. Tooth is always asymptomatic. Crown may exhibit darker color due to non vital pulp.

Radicular Cyst (Fig. 10.5)

It constitutes about 35% to 40% of all periapical radiolucent lesions. All radicular cysts develop in pre-existing periapical granulomas. An untreated cyst may slowly enlarge and cause expansion of cortical plates. The wall may also vary in thickness.

Cholesteatoma

It does not develop very frequently. It starts with formation of a few localized cholesterol crystals which forms due to

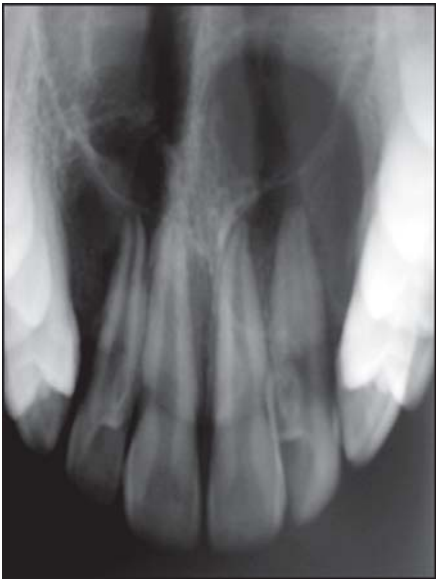


Fig. 10.5: Radicular cyst

fatty degeneration in a dental granuloma. Occasionally the radiolucent image of cholesteatoma appears somewhat less distinct and cloudy because of increased density of accumulated cholesterol crystal.

Osteomyelitis

Sometimes periapical abscess may develop into an osteomyelitis. It involves all the three layers of periosteum; Osteitis is a more localized condition. In maxilla rarely osteomyelitis develops due to rich blood supply. The

concerned tooth will contain a non-vital pulp, may be sensitive to percussion and may be having an associated abscess. Borders of radiolucency will be poorly defined and irregular. The bony course of draining tract traversing the body of jaw bone is seen as radiolucent band. It extends from periapical radiolucency through the cortical plate beneath the sinus opening in skin. The course will be longer and deeper. Sequestrum will show radiopacity in the radiolucency.

Periapical Cementomas

These include periapical cementomas, ossifying fibromas and arise from elements in the periodontal ligaments. In the early stage, these develop as rounded radiolucencies having well defined border. Mostly females are affected. Lesions are multiple and asymptomatic.

Traumatic Bone Cyst

It may be a false cyst without having epithelial lining. There may not be a history of trauma and lesion may be found on routine X-ray. Mandible is involved more than maxilla. The premolar - molar is the most common site involved. Tooth involved are vital and lamina dura is intact. Traumatic bone cyst may be found under the age of 25 years.

In radiographs, traumatic bone cyst is a well defined radiolucency above mandible. Aspiration will not be useful. The pulps of associated teeth are vital in traumatic bone.

Non-radicular Cyst

It may project over the apices of teeth. Incisive canal cyst is 2 cm cyst like radiolucency over the apex of a vital maxillary incisor and can be seen away from apex by changing the angle of X-ray tube. Midpalate cyst is at the periapex of a maxillary first molar on a periapical film.

Malignant Tumors

These may be found as a single periapical radiolucency mimicking benign lesion. Most common malignancies are squamous cell carcinoma, osteolytic sarcoma, melanoma, fibrosarcoma and multiple myeloma. Secondary metastasizing tumors to the jaws include malignant tumors of lungs, breast, GIT, prostate, thyroid and kidneys.

Malignant periapical radiolucencies may be indicated by one of the following images:

- Poorly defined periapical radiolucency
- Well defined radiolucency
- Large, well defined, irregular radiolucent tumor

Pain may or may not be a prominent feature. Expansion of the jaw is a feature in advanced lesion.

PERICORONAL RADIOLUCENCIES

Follicular Space (Fig. 10.6)

Radiographically it appears as a homogeneous radiolucent halo. It has a thin outer radiopaque border. This halo appears as a space, merges with periodontal ligament space.

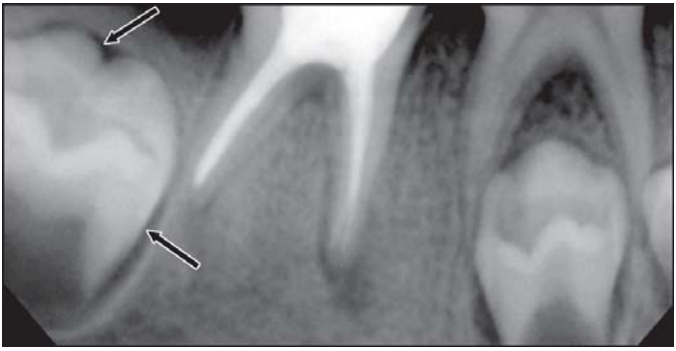


Fig. 10.6: Arrows indicating follicular space of erupting permanent first molar

Dentigerous Cyst

It is the most common odontogenic cyst. It has a lumen lined with epithelium. Most commonly affected tooth is mandibular premolar and maxillary third molar.

Multiple dentigerous cysts may be accompanied with basal cell nevus syndrome or cleidocranial dysostosis. Cyst varies from 2 cm in diameter to large expansion of jaws. Cysts usually yield a straw colored thin liquid. Cyst is painless and causes delayed eruption of tooth. Painless cyst is indicative of some infection. It generally does not expand so much so that it may press sensory nerve. Pain is localized but may be referred to any part of face.

Unicystic Ameloblastoma

There will be localized thinning and haziness of radiopaque rim. It generally occurs before the age of 30. Lesion slowly enlarges due to expansion of cortex. On palpation it is hard and bony.

Calcifying Odotogenic Cyst

It appears as a completely radiolucent cyst like lesion.

Adenoameloblastoma

It arises from residual odontogenic epithelium.

i. In early stage tumor is completely radiolucent. It may be solid or may contain large cystic areas. Minute calcifications are also seen under microscope.
ii. In advanced stage sufficient calcification occurs to produce clusters of radiopaque foci with the radiolucency.

Majority occurs in unerrupted tooth or in the walls of dentigerous cyst. Mostly involved teeth are maxillary canine, lateral incisors, mandibular premolar. Slow growth expands the cortical plates and produces a clinical signs of swelling and asymmetry is seen. Soft tissues are not involved.

Ameloblastic Fibroma

It is a true mixed odontogenic tumor, containing nests and strands of ameloblastic epithelium. Calcified dental structures are not seen. There will be well defined unilocular radiolucency. Multilocular lesions may occur. Vast majority occurs in mandible and highest incidence is of premolar region. Tumor is often associated with unerrupted teeth.

Solitary Cyst like Radiolucencies not Necessarily Contacting Teeth

These are dark radiographic images that are approximately circular in outline, smooth with well defined borders.

Maxillary Sinus

It is an out pouching of maxillary sinus. X-rays in some position may show a connection between such an out pouching and larger maxillary sinus.

Early Stage of Tooth Crypts (Fig. 10.7)

In early stage of development it will be round, smooth and well defined with radiopaque rim similar to a cyst. If no calcification is seen up to 1 year discontinuity in bone is likely a primordial cyst.

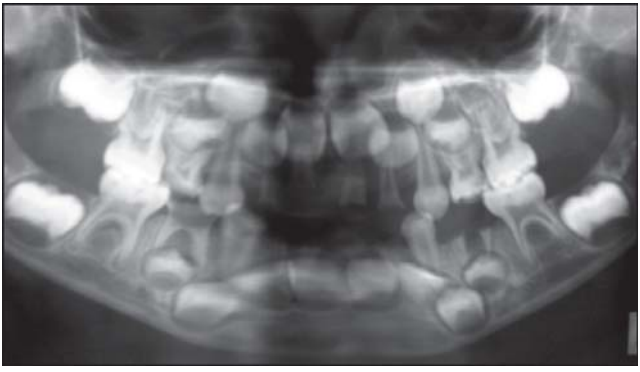


Fig. 10.7: Early tooth crypts

Post Extraction Socket

Sometimes after an extraction radiographically the tooth socket will look like a cyst like radiolucency. Sockets may remain uncalcified for years.

Residual Cyst

It is a radicular, lateral or dentigerous cyst remained after its associated tooth has been lost. It appears as a round radiolucency with well defined borders. Cyst may enlarge in elliptical shape and borders will be accentuated by a thin radiopaque line. It occurs mostly in alveolar process and body of jaw bones.

Traumatic Bone Cyst

Mostly occurs in inferior portion of alveolar process adjacent to apices of teeth. On early radiographs it may be seen as separate from apex of tooth. The cause of traumatic bone cyst is not very clear. There are two schools of thoughts, some think that it develops due to trauma induced from intra-bony hematoma. While others think that it is a burned out cyst. These are mostly found on routine radiographs. Mandible is involved in majority of cases. It is painless. Paresthesia is not a feature. It does not measure more than 3 cm.

Primordial Cyst

It may result due to cystic degeneration of cyst. X-ray is nonspecific showing only a cyst like radiolucency. There may be a missing permanent tooth. It has no sexual predilection. Third molar is the most common site of involvement.

Stafne's Cyst

It is a developmental bone defect of the mandible. The radiolucency may vary in size from 1-2 cm in diameter and can be oval in shape. It may be completely enclosed by bone. It is asymptomatic and is located generally at the angle of mandible. It does not contact the apices of molars.

Odontogenic Keratocyst

Diagnosis depends on its microscopic features and is dependent of its location. Radiographically it is very difficult to diagnose it. Sometimes its lumen is densely filled with keratin showing hazy radiolucent image. On occasion it may show multilocular appearance.

Symptoms are of bony cysts. It develops during second and third decades.

Ameloblastoma

It is an odontogenic tumor, a locally malignant lesion arising from ameloblasts. It is painless and slow growing. It can cause migration and loosen the tooth. It develops resorption and paresthesia of lip. It may extend into the cortical plates or may even erode them invading by the adjacent tissues. It can develop either unilocular or multilocular cyst. Multilocular may appear either into soap bubble or honeycomb type radiographically.

Multilocular Radiolucencies

Theses are produced by multiple adjacent frequently coalescing and overlapping compartments in bone. Mostly these occur in maxilla. The term soap bubble, honeycombs and tennis racket are frequently used.

Multilocular Cyst

It is the most frequently encountered entity in jaws. It is always of soap bubble type and a true cyst of jaws. Small cyst is usually asymptomatic; it increases slowly and may cause displacement of teeth. Root may be resorbed. If the overlying bone becomes thin it may produce crackling sound. Later covering plate may be destroyed. It may contain thin, straw colored fluid.

Central Giant Cell Granuloma

It may occur as a solitary cyst like radiolucency but as it grows larger it develops to give a soap bubble appearance.

Hyperparathyroidism

Lesions are similar to giant cell granuloma. It may be unilocular, bilocular or multilocular found in primary, secondary or tertiary hyperthyroidism.

Cherubism

It occurs between the ages of 2 and 20 years. It geneally develops bilaterally and involves mandible rami. Painless swelling develops on face. A few posterior teeth may be missing.

Odontogenic Myxoma

It is a benign tumor of bone developing only in jaws. It contains myxomatous tissue. X-ray may show unilocular cyst like radiolucency, pericoronal radiolucency and a mixed radiolucent radiopaque image. Tennis racket

appearance is the commonest. Very few tumors cross the midline. It generally develops between the age group of 25-35 years. Tumor develops slowly as painless expansion of jaw. Rarely there may be numbness of lips. Tumor may cause facial asymmetry. Sometimes it may perforate the cortices. Skin over tumor looks normal.

Odontogenic Keratocyst

It shows multilocular cysts and recurrence after operation is common.

Metastatic Tumor of Jaw

Carcinoma of jaws, breast, kidney, GIT, testis and thyroid gland and metastasis in jaw. It spreads through the arterial blood. Mandible is more commonly involved than maxilla. It may give different X-ray picture.

i. Solitary well defined radiolucency
ii. Solitary poorly defined radiolucency
iii. Multiple, separate, poorly defined radiolucency.
iv. Multiple punched out radiolucencies like multiple myeloma
v. Irregular salt and pepper appearance
vi. Solitary radiopaque area.

Symptoms are similar to primary malignant jaw tumor. Advanced tumors often involve inferior dental canal. Anaesthesia of lip develops. An enlarging lesion may erode rapidly through cortical plates without expanding them. Then it invades the surrounding tissues fixed to jaw bone. Pain is very frequent and is of short duration.

Central Hemangioma of Bone

It is a benign tumor. It may be congenital or traumatic in origin. X-ray may show various forms

i. Multilocular appearance
ii. Linear trabaculae radiating from center.
iii. Cyst like radiolucency, empty cavity and hyperostotic border.

There develops slow asymmetry of jaw. Numbness, tenderness or pain may be felt. Solitary tumor is more common. There may be local hemorrhage.

Solitary Radiolucencies with Ragged and Poorly Defined Borders

Main causes of such lesions are:

i. Inflammation of bone
ii. Fibrous dysplasia
iii. Osteolytic malignancy

Chronic Alveolar Abscess

It is a local mild inflammation of infection. It occurs around the roots of tooth. It may result due to pulpitis. Sinus may be present and pass through alveolar bone to open into mucosa.

Chronic Osteomyelitis

There may be clinically apparent pus. Inflammation may involve any soft part or the marrow. Haversian canal and periosteum – separately or in combination. *Staphylococci* and *Streptococci* are the most frequent causative organisms.

Chronic osteomyelitis may produce different X-ray images:

- A radiolucency with ragged borders
- A radiolucency containing one or more radiopaque foci
- Salt and pepper appearance
- Dense radiopacity.

Complete radiolucent type of bony lesion results from a chronic osteomyelitis has irregular shapes with ragged and poorly defined borders. Often the surrounding bony borders are denser than adjacent.

Mandible is more frequently involved than maxilla being rich in blood supply.

Squamous Cell Carcinoma

It is an intra-oral white lesion and is most common to develop. All squamous cell carcinoma don't invade and destroy bone. Carcinomas originating on or near the crest of mandibular ridge or on the posterior hard palate are the tumors to cause destruction of bone. Such tumors can be of two types peripheral or mucosal.

In peripheral type, patient may complain of worsening of oral ulcer. Person may be an alcoholic or heavy smoker. Other complaints are foul odor and bad taste. There may even be anesthesia of lip. While in central type patient will complain of pain and swelling. There may be anesthesia also. In peripheral type lytic effects of two types are seen.

- Semicircular or saucer shaped erosion into bony surface with ill defined borders
- Mandibular lesion with advanced horizontal resorption.

Tooth may be migrated, loose and resorbed. Regional lymph glands will be enlarged. Glands will be enlarged, painless and very firm. Central squamous cell carcinoma is rare and appears as rounded radiolucency. To start border is smooth and regular later on wall may be destroyed with ragged definitions.

Fibrous Dysplasia

It is lesion of bone developed as a result of abnormal proliferation of fibrous tissue. As lesion matures it destroys trabaculae. Radiographs in early stage will show radiolucent lesion. Intermediate lesion will be smoky, mottled or hazy pattern. Intense aggregations will be distributed throughout lesion. In mature stage, picture will be of salt and pepper type, ground glass or orange peel appearance. It may result in irregularity of bone without pain. There may be malocclusion.

Malignant Salivary Gland Tumor

Their incidence is much slow. Peripheral malignant salivary gland tumors can occur anywhere in lining of oral cavity but seldom in gingiva. Central variety is still rare. It generally develops between the age group 40-70 years of age. It grows slowly. When tumor infiltrates bone it produces radiolucency.

Osteogenic Sarcoma

It is most commonly found in jaw bones. It may show:

i. Completely radiolucent
ii. Radiolucent
iii. Radiopaque areas.

Radiolucent and radiopaque areas may show sun burst appearance. Peak age of incidence is around 25. Jaw lesions have fewer tendencies to metastasize hence better prognosis.

CHAPTER 11

Diseases of Jaw

Fibrous Dysplasia of Bones

It is an idiopathic condition. Normal part of bone is replaced by abnormal fibrous connective tissue. Bone is transformed lastly in lamellar dense bone.

Exact etiology of bone tumor is not known. It can be due to developmental error. It develops in younger age group specially in females. Maxilla is more commonly affected than mandible. Multiple jaw lesions are noted.

Clinical Features

- It is a slow growing, painless lesion which generally develops on one side
- Marked swelling of cheek develops
- Facial asymmetry is seen
- Expansion and gradual distortion is seen
- There will be disturbance in eruption of teeth
- Some lesions may be more aggressive and may undergo malignancy.

Radiological Features

- Disease produces multilocular or unilocular areas
- Bone consists of faint trabaculae
- There will be extension and distortion of cortical plates

- Margin of lesion are ill defined
- Erupted and unerupted both types of lesions are seen.

PAGET'S DISEASE

It shows irregular phases of abruption and resorption of bone. Disease can occur at any age. It has 3 phases:

- Initial osteolytic phase
- Mixed osteolysis and osteogenetic phase
- Predominantly sclerotic phase.

Clinical Features

- There will be bilaterally symmetrical swelling of the involved bone
- Bony deformity of face is developed
- Some may develop deafness, headache, blindness and facial paralysis
- Gait will be of waddling type
- Skull will be enlarged, legs will bow
- Loosening of teeth is noted and lips may become incompetent
- Post-extraction hemorrhage occurs
- Pathological fractures are seen
- A few patients may develop osteogenic sarcoma.

Radiological Features

- Radiolucent areas
- Cotton–wool appearance will develop
- Teeth may develop
  - Hypercementosis
  - Loss of lamina dura

- Obliteration of periodontal ligament space
- Root resorption may take place.

A few cases may transform into malignancy.

OSTEOGENSIS IMPERFECTA

It develops due to defective matrix formation and lack of mineralization. Fetal collagen is not able to transform into mature collagen. There are four types. Two are autosomal recessive and the rest two are autosomal dominant type.

i. *Moderate and deforming type* – These are associated with deciduous teeth and blue sclera. Child may have abnormal shape of skull, joint dislocations and defective heart valve.
ii. *Mild not deforming type* – Patient otherwise is clinically normal. Person has blue sclera with dentinogenesis imperfecta.
iii. *Neonatal lethal type* – It is a serious form of disease. There are multiple fractures of bone.
iv. *Severe non-lethal type* – Patient develops fracture with slight trauma. Fractures heel early but deformity remains.

Radiological Features

- Extremities are short and deformed
- Cysts like radiolucencies are seen
- Teeth show short roots and bulbous crown.

Once disease is fully developed no improvement is expected.

CLEIDOCRANIAL DYSPLASIA

It is a hereditary disorder. There is an abnormal growth of bones of face, skull and clavicles. There is failure of tooth eruption. There is autosomal dominant trait. There develops spontaneous mutations.

Clinical Features

- There is hypermobility of shoulder joint
- Frontal bossing is seen
- Entire mid-face is underdeveloped
- Nose is wide, flat without bridge
- Multiple unerupted and impacted teeth, supernumerary teeth are seen
- Clavicles are hypoplastic.

Radiological Features

- Multiple wormin bones can be seen
- Open sutures are noted
- Roots of teeth are thin and lack cellular cementum
- Maxillary sinus is underdeveloped

Radiation therapy may prevent the progress of disease.

HYPOPLASIA OF MANDIBULAR CONDYLE

It may be congenital or due to fracture/infective disease.

Clinical Features

- Deviation of mandible to the affected side
- Deficiency in mastication
- Midline shift of dentition on to the affected side.

Hyperplasia of mandibular condyle is a rare disease. In this condition chin will be deviated toward the opposite side. Treatment requires surgical intervention.

Rheumatoid Mandibular Joint

It may result in progressive destruction of joint structure.

Clinical Features

- Most female patients of 30-40 years of age are affected.
- Fever, weight loss, malaise and raised ESR.
- Restricted jaw movements.

Radiological Features

- Irregularity of condyle
- Widening of TM joint space
- Flattening of condyle

Cortisone and antibiotics help.

MYOFACIAL PAIN DYSFUNCTION SYNDROME

There is a pain and limited movements of TMJ. It may be caused by spasm of muscles of mastication and fatigue. Habitual grinding of teeth may also result in this syndrome.

Clinical Features

- Generally females suffer between the age group of 30-40 years.
- Pain in temporo mandibular joint.
- Muscle tenderness.
- Limitation of movements.
- Sometimes clicking sound will be produced.

Analgesics, muscle relaxants and tranquilizers will help.

CHAPTER 12

Diseases of Salivary Glands

Salivary glands comprises of three paired major glands:

- Parotid glands
- Submandibular glands
- Sublingual salivary glands.

Besides these there are about 300 minor glands. That keep oral cavity moist. Dysfunction of salivary glands not only affects the composition and secretion of saliva but also the oral cavity inviting various types of diseases and associated problems.

Different Lesions of Salivary Glands

- Infective
  - Acute and chronic infections
  - Viral sialadenitis
  - Mumps
- Immune medicated
  - Sjögren's syndrome
  - Mikulicz's disease
- Neoplasms
  - Pleomorphic adenoma
  - Mixed tumor
  - Adenolymphoma
  - Epidermoid carcinoma

  - Fibroma
  - Fibrosarcoma
  - Hemoangioma
  - Lymphoma
- Reactive lesions
  - Mucus retention cyst
  - Sialolithiasis
  - Post-radiation sialadenitis.
- Developmental anomaly
  - Agenesis
  - Hypoplasia
  - Atresia
  - Ectopia
  - Diverticulae

Aplasia

Total aplasia is not very common. It may develop with Melkersson-Rosenthal syndrome and may manifest as facial paralysis, fissured tongue and orofacial granuloma. It may result in case of xerostomia and in absence of saliva, multiple caries may develop.

Hypoplasia

It is associated with hereditary ectodermal dysplasia.

In some cases hypertrophy may be associated with mucoviscoidosis.

Ectopic Salivary Glands

Stafne's bone presents the growth of salivary gland tissue at an angle of mandible. It appears well circumscribed.

Cysts of Salivary Glands

These cysts are known as mucoceles. These are of two types:

i. Mucous retention cyst (Fig. 12.4)
ii. Mucous extravasation cyst (Fig. 12.3)

These develop as a result of obstruction to the duct of the minor salivary gland. Saliva is accumulated in the duct causing its expansion. While mucous extravasation cyst develops after damage to the salivary gland and saliva leaks out into the connective tissue. Mucoceles can occur at any age and lower lip is the commonest site. Superficial lesions occur as small, vesicle like raised areas. Such swellings often recur (Figs 12.1 and 12.2).

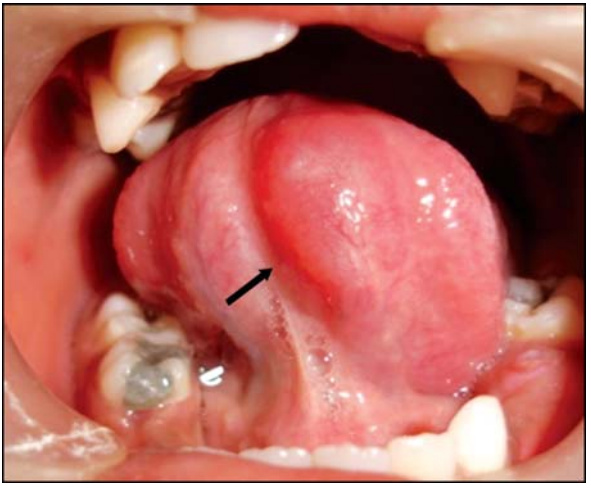


Fig. 12.1: Lingual mucocele

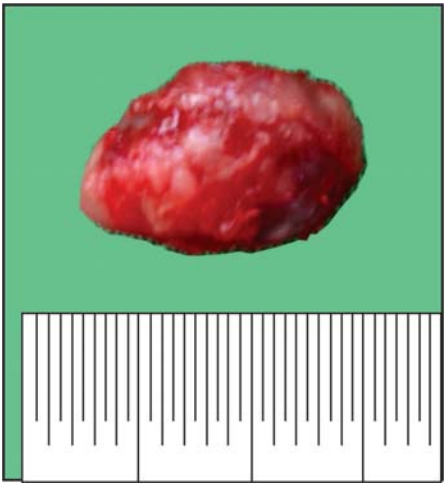


Fig. 12.2: Post-surgical picture of injected impression material depicting size of the mucocele

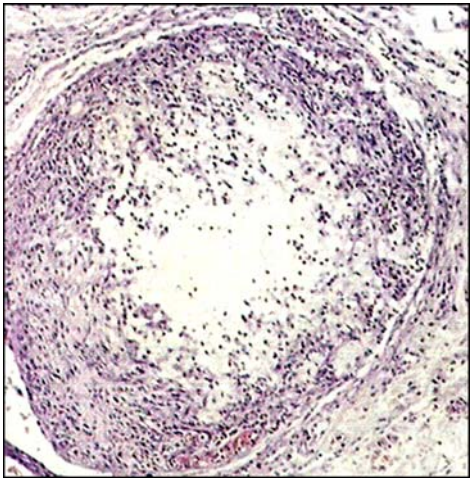


Fig. 12.3: Histological section showing extravasation cyst of a salivary gland

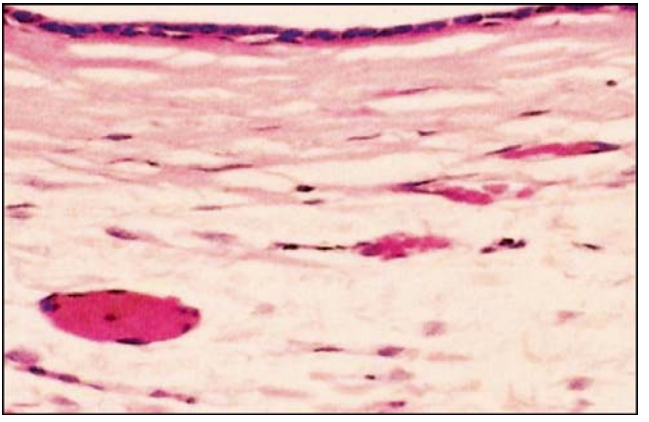


Fig. 12.4: Histological section showing retention cyst of a salivary gland

Sialolithiasis

In this type of condition there is one or more calcified stones in the gland or in the duct. Nidus of stone is made up of mucin, bacterial protein and desquamated epithelial cells. Later on lamellar crystallizations occur. Ring per ring stone grows in size. Stone formation is more common in submandibular gland and its duct as saliva of submandibular duct is more viscous. Calcium levels are also high in it. Stone generally doesn't produce any symptoms and may be diagnosed on routine examination. Some patients may develop pain, discomfort and swelling during meals. Pain can be severe like stabbing with swelling. Affected gland may become enlarged and firm.

If swelling is persistent it may lead to chronic sclerosing sialadenitis. If multiple stones are formed in all branches then it may result in complete calcification of gland.

Involvement of submandibular gland generally produces unilateral glandular enlargement. Swelling is firm and tender. In chronic cases sinus and ulcers may be formed, followed by on complete loss of secretion of saliva. Parotid stone often causes firm swelling over the ramus of the mandible. Swelling exaggerates during the intake of meals.

Chronic Sclerosing Sialadenitis

It is a chronic inflammation of salivary gland tissue. It results in degeneration and subsequent replacement of acini by fibrous tissue. Trauma, infection and autoimmune all may cause it. Major or minor gland all may be affected. Affected gland is enlarged and remains freely movable. More the fibrosis develops the more firm it becomes. Once the acini are lost the gland parenchyma undergoes progressive sclerosis. Then sialodectomy will be the treatment of choice.

Necrotizing Sialometaplasia (Fig. 12.5)

It is a benign, inflammatory reaction of salivary gland tissue, which is developed most likely due to local ischemia. There is development necrosis of minor salivary glands. It develops due to infarction of tissues. Necrotizing sialometaplasia develops as one or two deep seated punched out ulcerations on the hard or soft tissues. Ulcers measures 2-3 cm in diameter. At the base a few grey granular lobules are present. Some patients may complaint of numbness or a burning type pain in that area. It occurs most commonly in men than women during the 4th and 5th decade of life, occurring most commonly on palate, followed by buccal

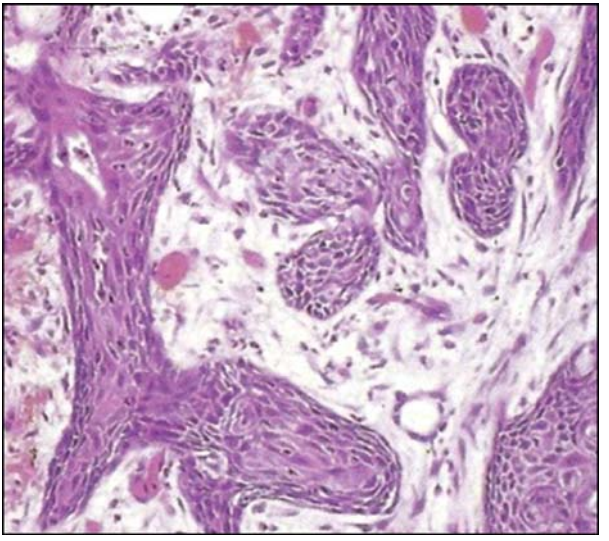


Fig. 12.5: Necrotizing sialometaplasia

mucosa, lip and retromolar area. The lesion is essentially self-limiting and heals by secondary intention within 1-3 months. Recurrence is generally not encountered.

Acute Bacterial Sialadenitis

It is caused by *Streptococcus pyogenes* and *Staphylococcus aureus*. Bacteria reach by stensen's duct. Diabetes, Sjogren's syndrome is the causative factors. Clinical features include sudden onset of painful swelling in the preauricular region. One or both sided parotid glands may be involved. Fever, weakness and redness of skin over parotid are seen. There may be difficulty in swallowing. Antibiotics are helpful.

Chronic Bacterial Sialadenitis

It is a non specific inflammatory disease of salivary gland generally after obstruction of duct. Usually parotid gland of one side is affected. There may develop recurrent, tender swelling. Salivary flow is decreased.

Viral Infection

Mumps/ Epidemic parotitis is an acute contagious viral infection caused by a paramyxovirus which is characterized by unilateral or bilateral swelling of parotid gland. It may complicate gonads, CNS, pancreas and myocardium.

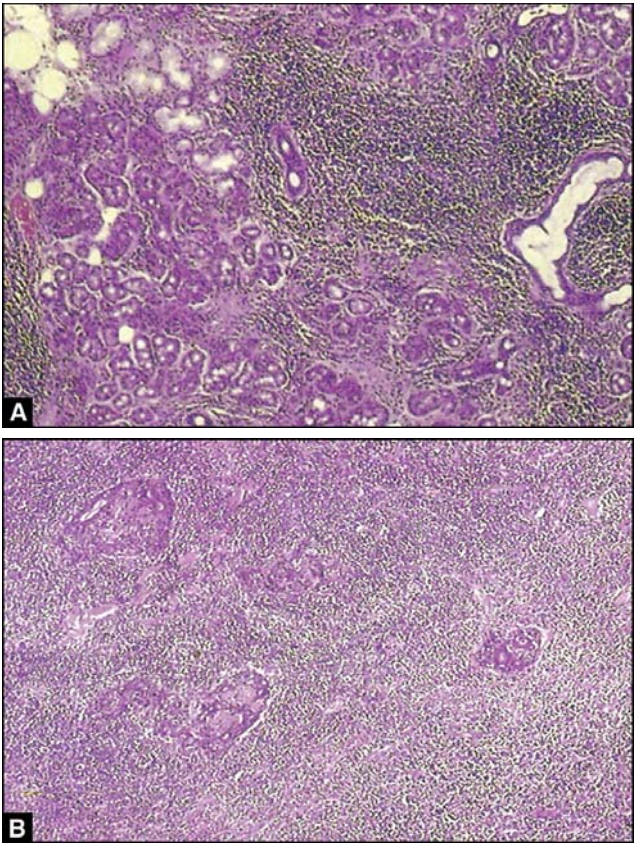
Clinical features include sudden fever, malaise, anorexia, pain below the ear and pain upon mastication. Initially one sided parotid gland is involved then second is involved. Parotid gland enlarges for 2-3 days and return to normal within seven days. Rarely submandibular and sublingual gland may also be involved. Most of the causes are self-limiting within a week. In some children meningitis or encephalitis may develop. Antibiotics and cortisone avoid complications.

Allergic Sialadenitis

It is a non-neoplastic, non-inflammatory enlargement of salivary gland. Enlargement is bilateral and painless. Periauricular portion of enlargement is noted. Salivary potassium content is enlarged and salivary sodium content is reduced.

Sjögren's Syndrome/Sicca Syndrome (Figs 12.6A and B)

It is a condition originally described as a triad of kerato-conjunctivitis sicca, xerostomia and rheumatoid arthritis.



Figs 12.6A and B: Histological section showing Sjögren's syndrome

It is a multisystem immune mediated chronic inflammatory disease. There develops lymphocytic infiltration with acinar destruction of glands. In primary Sjögren's syndrome only salivary and lacrimal glands are involved. Where the mouth

and eyes becomes dry. In secondary Sjögren's syndrome along with above symptoms, rheumatoid arthritis may also develop. Some may develop arthalgia. Patient may feels tired. Xerostomia results in difficult swallowing and talking. Taste sensations may also be distorted. Mucosa becomes red and patchy. Dorsum of tongue becomes red and atrophic. There may be fissuring. Parotid salivary glands is enlarged and mainly affected.

Ptylism

It is an increased salivation of mouth which can be due to

- Abnormal neurosecretary stimulation
- Aphthous ulcer
- Psychological factor
- Idiopathic.

  No treatment is required.

Neoplasm of Salivary Gland

More than 50% minor salivary gland neoplasms occur in palate, 20% in upper lip. Lower lip is less involved.

Pleomorphic Adenoma/Mixed Tumor (Fig. 12.7)

It is a benign mixed tumor of gland. It can occur at any age. It accounts for more than 50% of neoplasm of parotid gland. It is a slow growing, well delineated, exophytic growth. Neoplasm is solitary but in some cases there may be multiple recurrent lesions. Neoplasm is rubbery and painless. Overlying mucosa may remain intact.

In lip, pleomorphic adenoma presents small, painless, well defined, movable nodular lesion.

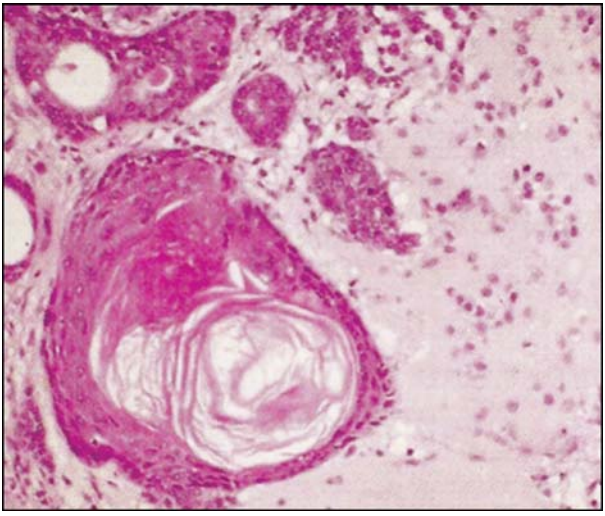


Fig. 12.7: Histologic section of pleomorphic adenoma – showing sheets of epithelial cells which have form ducts

Clinical Features (Fig. 12.8)

- More frequent in women than men; patient in 4th-6th decade are more affected
- Well circumscribed
- Lobulated
- Globular mass surrounded by capsule
- Cut surface is not smooth and shows cystic and hemorrhagic areas
- Pleomorphic adenoma of parotid gland does not show any fixation to the underlying tissue
- The skin seldom ulcerates even though these tumor reach the fantastic size

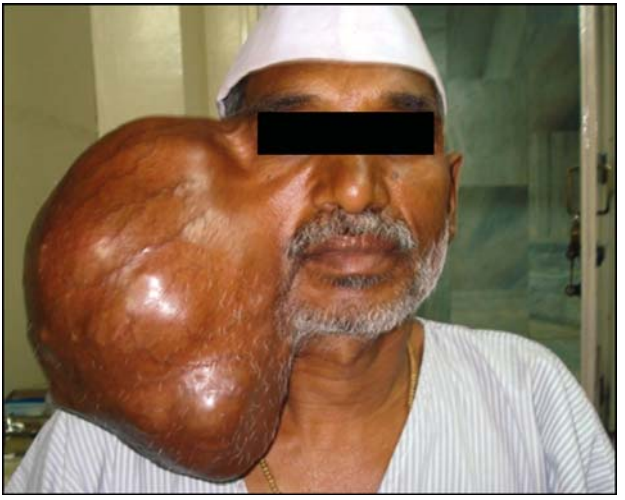


Fig. 12.8: Pleomorphic adenoma of parotid gland

- Presence of local discomfort
- Intraoral lesion seldom attains size greater than 1-2 cm in diameter.

Adenolymphoma

It is a benign salivary gland neoplasm. It consists of cystic spaces with intraluminar projections. It contains lot of lymphoid tissue. It may develop due to proliferation of ectopic salivary gland tissue. Some think that it is a hemartous growth than a true neoplastic lesion. It comprises 20% of all parotid tumors.

Clinical Features

- It is well encapsulated and movable
- Is it a slow growing well circumscribed soft and painless
- It can grow up to 2-4 cm in diameter
- It gives a compressible and doughy feeling on palpation
- On cut surface confluent cystic spaces are seen and chocolate colored fluid comes out. Dense fibrous capsule surrounds it.

  Simple surgical excision is the treatment of choice.

Malignant Salivary Gland Neoplasm

It is not a very common tumor. Benign pleomorphic adenoma changes into malignancy. Some tumors may be malignant from the very beginning and develops very fast in six months.

- There will be severe pain and paralysis of facial nerve
- It is fixed to bone or muscle
- Lymph glands are enlarged.

Extensive surgery followed by radiotherapy is the treatment of choice.

Adenocystic Carcinoma/Cylindroma/Adenoid Cystic Basal Cell Carcinoma (Fig. 12.9)

It is a malignant neoplasm arising from glandular epithelium. It is a most common malignant tumor of parotid, submandibular salivary gland. Accessory glands of palate and tongue can also be involved. Occuring most commonly during the 5th-6th decade of life. Parotid tumour produces a subcutaneous mass anterior to or below the external ear. Pain is very common feature in this tumor. There is fixation and induration of tumor. Submandibular gland tumors

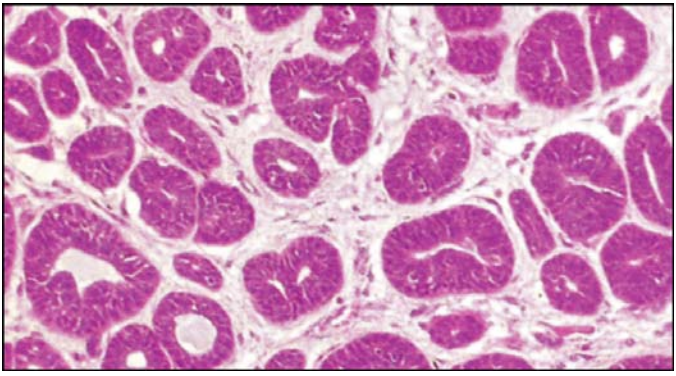


Fig. 12.9: Low power histological section of adenoid cystic carcinoma

becomes quite large. While palatal lesions are often accompanied by delayed healing of socket once the tooth has been extracted. If greater palatine nerve is involved; palatal parasthesia will develop. Treatment involves excision followed by radiotherapy because tumor cells are radiosensitive.

Mucoepidermoid Tumor (Fig. 12.10)

It is an unusual type of malignant salivary gland neoplasm with varying degree of aggressiveness. Tumor generally involves parotid gland. As the name suggests the tumor is composed of both mucus secreting and epidermoid type of cells in various proportions.

Clinical Features

- It is similar to pleomorphic adenoma
- Equal predilection for males and females

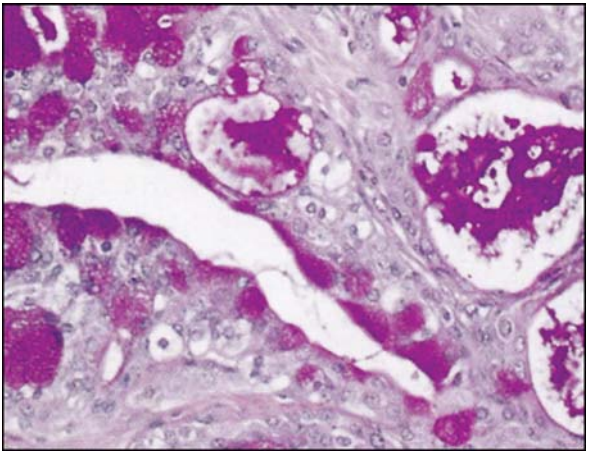


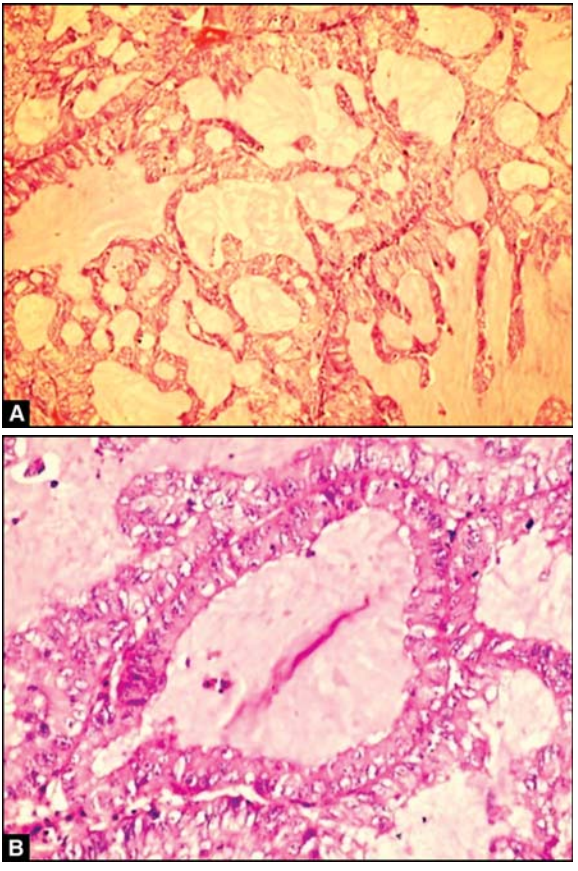
Fig. 12.10: Mucoepidermoid carcinoma/ tumor

- It is a slow growing, painless swelling of low grade malignancy
- It has cystic feeling
- Radiographs will show unilocular or multilocular radiolucent areas in jaw
- Parotid tumor shows relatively, focal nodular swelling
- Swelling is mobile because they have the tendency to develop cystic areas
- Facial nerve paralysis is frequent in parotid tumor
- Distant metastasis is common
- Low grade tumor shows fluctuation
- High grade tumors are fixed to the adjacent areas, grow rapidly and produces early pain
- In a few cases tumor may be fast growing with ulceration, hemorrhage and parasthesia.

Treatment is wide surgical excision followed by radiotherapy.

Adenocarcinoma (Figs 12.11A and B)

It occurs more commonly in relation to minor salivary glands. The tumor is slow growing with no surface



Figs 12.11A and B: Adenocarcinoma of palate in a 50 yr old patient

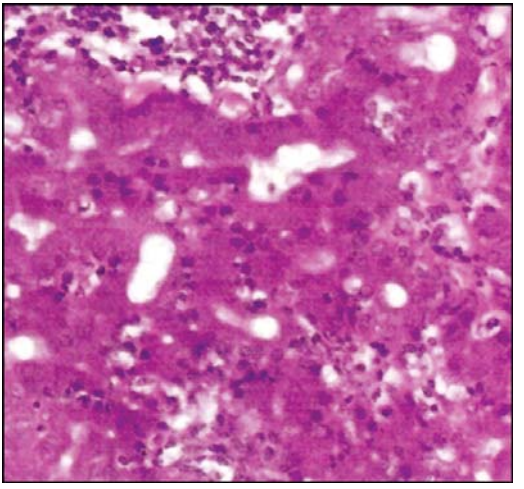


Fig. 12.12: Acinic cell carcinoma

ulceration. It is a painless mass. Later on tumor grows faster. Swelling becomes painful. Ulceration and loss of sensation develops. Under microscope one can see numerous proliferating malignant ductal epithelical cells. There will be areas of hemorrhage and necrosis.

Acinic Cell Tumor (Fig. 12.12)

These are not common neoplasm of salivary gland. Parotid is affected and is not frequently seen in intraoral sites.

Clinical Features

- Size of lesion is about 3 cm
- Overlying skin is intact
- Lesion is well defined and slow growing
- Cystic spaces makes it fluctuant
- Rarely lip or cheek may be involved.

CHAPTER 13

Disorders of Taste

There can be varied disorders of taste:

- Complete loss of taste
- Diminished taste sensitivity
- Persistent bad taste
- Altered taste.

It may be caused by:

1. Damage of taste nerves
   - Viral/bacterial infection
     - Otitis media
     - Diptheretic neuritis of facial nerve
     - Tumours of 7th and 9th cranial nerves
   - Surgical trauma to:
     - Chorda tympanic
     - Facial nerve
     - Glossopharangeal.
2. Loss of taste buds:
   - Glossitis
   - Lichen planus
   - Leukoplakia
   - Therapeutic radiation
   - Penicillamine.
3. Transport disorders:
   - Salivary hypofunction
   - Xerostomia
   - Therapeutic radiation

- Blocking of palatal receptors by dental prosthesis
- Blocking of taste bud pores by bacteria.

4. Metabolic disorders:
   - Diabetes mellitus
   - Hypothyroidism
   - Adrenal insufficiency
   - Hepatic disease
   - Therapeutic radiation.
5. Lesions affecting central pathways of taste:
   - Cerebrovascular lesion
   - Neurodegenerative disease
   - Epilepsy
   - Head trauma.

NON NEUROPATHIC ORIGIN

Substances with unpleasant or unusual look are produced in mouth due to bacterial fermentation in dental plaque.

It may be caused due to:

- Blocking of palatal taste receptors
- Abnormal oral secretions
- Abnormal secretions of salivary gland.

Complete loss of taste generally doesn't occur but loss of taste for particular quality, i.e. sweet or sour may occur.

NON PATHOGENIC DYSGEUSIA OF CENTRAL ORIGIN

Gustatory branches of 7th, 9th and 10th cranial nerves involve synapses at three levels

- Medulla
- Thalamus
- Cerebral cortex.

At the levels of medulla small lesions of thalamus and medulla may be affected with taste loss. Person may loose sensations of both taste and odour. In some patients dysgeusic symptoms may be dramatic after brain trauma/surgery, seizure and cerebrovascular accident.

ANOMALIES OF TASTE RECEPTORS

It can be congenital or acquired. Tongue may not have vallate and fungi form papillae. In aglycogeusia, congenital abnormality of taste buds is noted. There is no taste for sugar, due to deficiency of vitamin B complex it may also result in taste blindness. Long term use of corticosteroid may also cause loss of taste perception.

Dysguesia due to chemotherapeutic agents can result due to interference of replacement of taste buds. Zinc deficiency may also lead to blunting of taste sensitivity.

LESIONS OF LINGUAL NERVE

As nerve carries both general sensory and gustatory fibres to the anterior third of tongue, various sensation of pain, burning and numbness of that area are accompanied with change in taste on damage of sensory fibres.

LESIONS OF GLOSSOPHARANGEAL NERVE

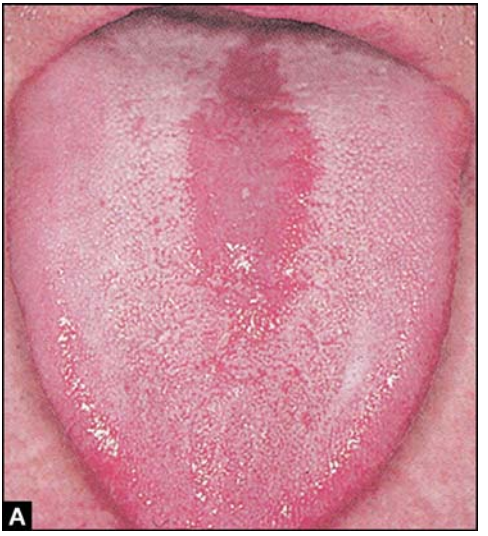
Removal of pharyngeal tumours may cause lesions of glossopharangeal nerve. Transient dysgeusia is restricted to one side of posterior 2/3rd of tongue. An extra cranial lesion of vagus nerve doesn't cause such lesions. Intra-cranial lesions of 7th, 9th and 10th nerve due to infection or neoplasm may result in dysgeusia.

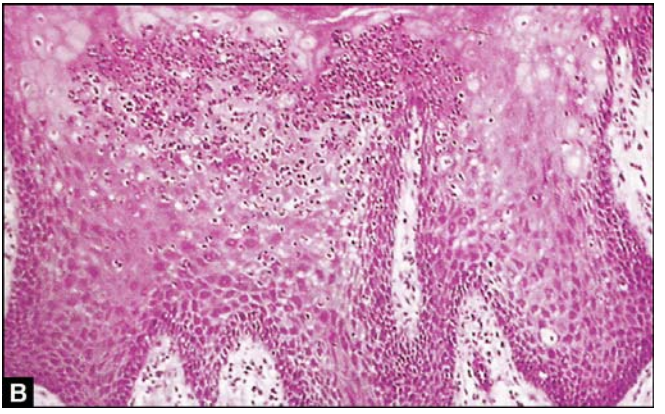
CHAPTER 14

Diseases of Tongue

Geographic tongue/Migratory glossitis (Figs 14.1A and B) refers to irregularly shaped, reddish areas of depapillation. There will be thinning of dorsal tongue epithelium. There is spontaneous development and regeneration of affected area. There may be associated fissured tongue, although this may be a coincidental finding. Etiology of geographic tongue is not clear. An immunologic reaction is suggested. No inheritance pattern is noted. The disease is asymptomatic but some may complaint of burning, pain and stinging. Clinically irregularly shaped red patches with white patterns look like a map. Red patches are smaller to start surrounded by a white rim. Red patches go on enlarging and regressing and pattern goes on changing every week. No sex predilection is found. The central portion of lesion sometimes appears short, while the border may be outlined by thin, yellowish white line or band. Desquamated areas are located in one area for a short while, heal and then reappear in another area thus giving the name migratory glossitis.

Coated/hairy tongue *is an unusual condition characterized by hypertrophy of filliform papillae of tongue (Fig. 14.2).* Normally keratinized surface layers of filliform papillae are continuously desquamated due to friction of food and





Figs 14.1A and B: Clinical and histological picture showing geographic tongue

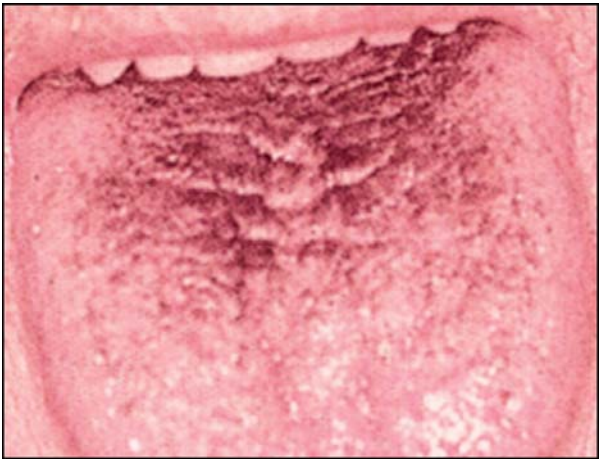


Fig. 14.2: Hairy tongue

anterior upper teeth. These are replaced by new epithelial cells from below. When tongue movements becomes restricted during illness, the papilla enlarges and become heavily coated. The color of papilla varies from yellowish white-brownish black depending upon the type of stains the tongue is exposed to. Longer papilla entangles food particles of different colors. Tobacco smoke colors it black. Mid dorsum is first to be affected. Dehydration and terminally ill patients also develop thick coatings. Nicotinamide deficiency has produce black hairy tongue in experimental animals. Excessive exposure of radiation to head and neck area and systemic antibiotics may also produce hairy tongue, because the condition is benign, the treatment is also empirical, in such cases, thorough scrapping and cleaning of tongue is advised to promote desquamation and removal of debris.

Thrush

There is formation of pearly white pin head sized flecks scattered all over dorsal surface consisting of large number of yeasts pseudomyelia. Constant use of corticosteroids and cholinergic drugs may result in the development of thrush.

White Sponge Nevus (Fig. 14.3)

It is an inherited anomaly. Mucosa is involved by white spongy plaques without keratosis. It is an autosomal dominant condition. Numerous pedigrees of families may show this condition.

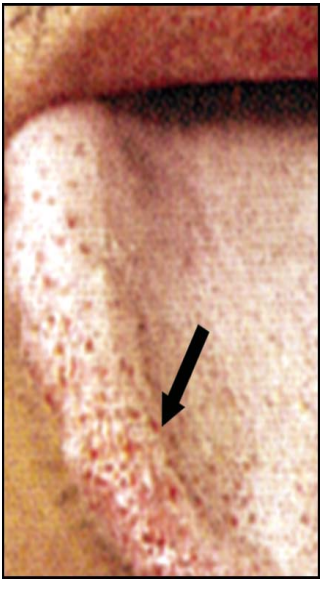


Fig. 14.3: White spongy nevus

Pachyonychia Congentia

There is congenital gross thickening of finger and toe nails. Corneal dystrophy, thickening of tympanic membrane and mental retardation are reported. Dorsum of tongue becomes thickened and grayish white. Cheeks may also be involved on occasion. Frequent oral aphthous ulceration may be seen.

Lichen Planus

There are three basic types: keratosis, erosions and bulla formation. Psychogenic problems play an etiological role. During deep emotional problems remissions and exacerbations are seen. It may be associated with diabetes. Lesions may transform into malignancy. Five different varieties of lichen planus are seen reticular, erosive, atrophic, papular and bullous.

Leukoplakia

It is clinical diagnosis. There are two etiological factors:

- Those caused by smoking
- Those associated with chronic Candidiasis.

Clinical Features (Fig. 14.4)

- It has three main clinical forms
- Homogeneous leukoplakia – It is a localized lesion or extensive white patch which presents consistent pattern.

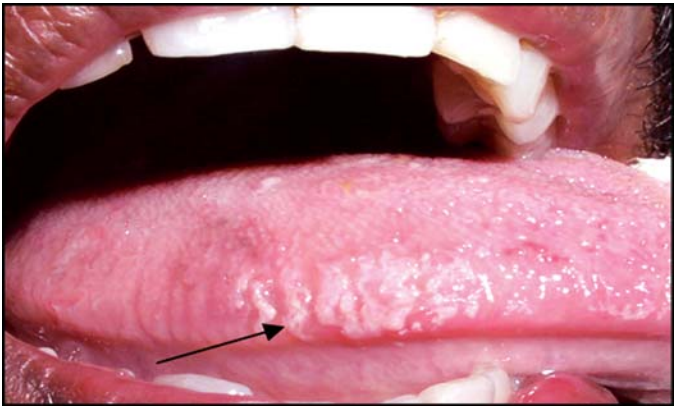


Fig. 14.4: Leukoplakia on the lateral borders of tongue

- Nodular leukoplakia – It refers to a mixed red and white lesion in which small keratotic nodules are scattered over a patch of atrophic mucosa. Their transformations to malignancy are higher.
- Verrucous leukoplakia – Oral white lesion with multiple papillary projections.

Diagnosis is confirmed by biopsy which will show cellular dysplasia.

Depapillation (Fig. 14.5) Generally occurs on the anterior 2/3rd of the tongue. Diabetes, Candidiasis, trauma, nutritional deficiency and medication may cause it. Long term Xerostomia can also result in it.

- Chronic trauma – localized areas of atrophy are seen in areas of jagged teeth or rough margins of restorations. Papillary regeneration may take place around these areas.

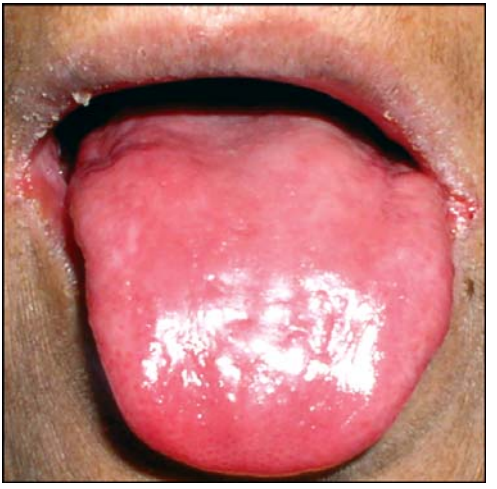


Fig. 14.5: Depapillation of tongue

- Nutritional deficiency – Redness, loss of papillae and painful swelling of tongue is found in vitamin B complex deficiency. Iron deficiency may also cause it.
- Sideropenic anemia also results in atrophic glossitis and angular chelitis. Person may develop dysphagia. Lips may become narrow and thin along with dry skin and brittle nails.

Peripheral Vascular Disease

Decreased nutritional status and vascular changes of dorsal capillary plexus or lingual vessel may result in atrophic glossitis.

Chronic Candidiasis and Median Rhomboid Glossitis

Chronic Candidiasis may result in central atrophy of dorsum. In median rhomboid glossitis, rounded lozenge shaped raised areas are seen in midline.

Tertiary Syphilis

The tongue is tertiary syphilis may present as a gumma formation or diffuse granulomatous lesions. Tongue may show non ulcerating irregular indurations. To start tongue is enlarged and later on it shrinks.

Pigmentation of Tongue

Endogenous pigmentation is not identifiable but jaundice may give yellowish appearance. Exogenous pigmentation is caused by microbial growth and food debris. Certain drugs also exhibit colors to tongue. Certain anti hypertensive and antiviral drugs also stain tongue.

Ulcers of Tongue

- Carcinoma – There may be foul smell because sloughing ulcer may be heavily infected. Person may feel pain in early stage and will not be able to protrude tongue.
- Epithelioma develops in the side of tongue. Ulcer is deep, foul and sloughy. Edges will be raised and everted.
- Squamous cell carcinoma of tongue is most common. 65% lesions develop on anterior 2/3rd of tongue. Local pain, pain on swallowing and swelling in neck are the initial symptoms (Fig. 14.6).

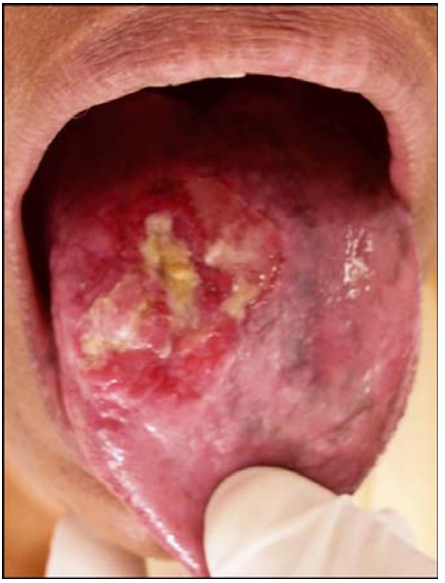


Fig. 14.6: Non healing ulcer of tongue

- In scirrhous carcinoma there will be minimal ulceration of mucous membrane. Affected part is shriveled up.
- In papillomatous type multiple ulcerations are uncommon.
- In all these conditions ulcer is hard and resistant to treat. Actually ulcer of a tongue of more than 3 weeks duration should always be suspected.

Syphilitic Ulcer

Syphilitic ulcer is not seen early because it starts as a simple pimple which later on ulcerates and becomes indurated. Tertiary ulcers are superficial or deep. Ulcers are shallow,

often irregular and are associated with chronic glossitis. Deep gumma starts as hard swelling on the substance of tongue.

Tuberculous Ulcer

Causative organism is tubercle bacilli. Ulcer develops on the tip or on the side of anterior half. Outline of ulcer is irregular. Edges are thin and undermined. Base is sloughy, nodular or caseous. In some persons tongue swells and becomes woody.

Dental Ulcer

It is due to repeated small injuries from a sharp edge of a decayed tooth. It develops on the lateral border. Ulcer is small, superficial and not indurated. It should heal within 3 weeks.

Ulcerative Stomatitis

It develops due to decayed teeth, alkalies or acid. These form vesicles which rupture giving rise to ulcer.

Cretinism

There is retardation of dental development due to delay in the formation of dental buds. Tongue is thickened. Patient is having dull looking face and slow pulse.

Acromegaly

There is osseous hyperplasia of frontal ridges while the lower jaw is usually enlarged in all directions. Forehead becomes wrinkled with massive nose. Thick upper lip and

heavy chin can be seen. Lower teeth are unduly wide apart and may project some distance in front of upper teeth. There develops many fissures on tongue. Tongue may swell.

PAINFUL TONGUE

Pain of the Surface of Tongue

- Pain may be there even without bite for instance. After general anesthesia patient may complain of soreness of tongue due to application of forceps.
- Injury by a tooth or dental plate may cause a local pain upon the side of the tongue. If antibiotics are taken for long, it may cause diffuse soreness of tongue. Tongue may also become inflamed and painful due to pemphigus vulgaris.
- Carcinoma of tongue to start is painless and becomes painful as it involves deep structures. Pain often radiates to ear that is being supplied by lingual branch of trigeminal nerve.

Pain Underneath Tongue

Calculus in submandibular salivary gland is not always painful. Injury to lingual foramen may cause visible abrasion or ulcer. Injured end will be painful. Foreign body in tongue like fish bone may be a cause of pain.

CHAPTER 15

Diseases of Paranasal Sinuses

Maxillary sinus disease is most concerned to a dentist in practice.

Clinical Features

Common clinical features include:

- Feeling of heaviness over maxillary area
- Pain on movement of head
- Sensitivity to teeth on percussion.

Sinusitis

It is a generalized inflammation of paranasal sinuses mucosa. Cause may be allergic, viral or bacterial. It causes blockage of drainage and thus retention of sinus secretion. It may be caused by extension of dental infection. Sinusitis is divided into three types:

- Acute sinusitis—it is of less than two weeks
- Subacute sinusitis—up to three months
- Chronic—when it exists for more than three months.

Clinical Features

- Presence of common cold
- Nasal discharge, allergic rhinitis
- Pain and tenderness over the sinus involved

- Pain may be referred to premolar and molar teeth
- Fever, chills and malaise.

Radiological Features

- Secretions reduce air and make sinus radiopaque
- Mucosal thickening of floor and later on may involve whole sinus
- Thickened mucosa may be uniform or polypoid
- Air fluid level may also be present due to accumulation of secretions. It is horizontal and straight
- Chronic sinusitis may result in opacification of sinus.

Empyema

It is a cavity filled with pus. It appears more radiopaque.

Mucositis

Mucosal lining is composed of respiratory epithelium. It is about 1 mm thick. Normally it is not seen on radiograph. When seen, mostly it is an incidental finding. Radiographically, it is seen as a thick band, paralleling around the bony wall of sinus.

Polyps

Thickened mucus membrane of chronically inflamed sinus undergoes irregular folds known as polyps. It may cause destruction of bone. Multiple polyps are known as polyposis.

Radiological Features

- Polyp occurs with a thick mucus membrane lining
- While in retention pseudocyst, mucus membrane lining is not apparent.

Mucocele

It is an expanding destructive lesion due to blocked sinus ostium. Sinus wall may be thinned out or it may even be destroyed. When mucocele is infected, it is known as mucopyocele.

Clinical Features

- It results in radiating pain
- Sensation of fullness in cheek
- Swelling over antrum
- If lesion expands inferiorly, tooth may become loose
- If it expands to orbit, diplopia may be caused.

Radiological Features

- 80% of mucocele occurs in ethmoidal sinus
- Shape of sinus becomes more circular
- It becomes uniformly radiopaque
- Bones and septa may be destroyed and thinned out
- If mucocele is with maxillary antrum, teeth may be displaced
- Roots of teeth may be resorbed
- If frontal sinus is involved, the inter sinus septum may be displaced.

- When ethmoidal sinus is involved, contents of orbit may be displaced
- Large odontogenic cyst displacing maxillary antral flow may mimic a mucocele.

Antrolith

- It occurs within the maxillary sinus
- It results due to deposition of calcium carbonate, calcium phosphate and magnesium.

Clinical Features

- Small antrolith's are symptom free
- If bigger, blood stained discharge may be present
- There may be nasal pain and facial pain as well.

Radiological Features (Figs 15.1 to 15.6)

- Mostly occur in maxillary sinus
- Internal density may be homogenous or hetrogenous.

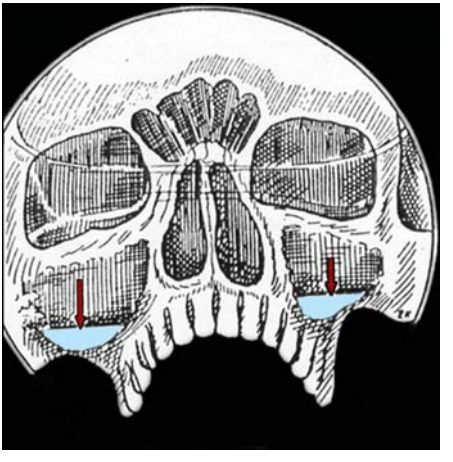


Fig. 15.1: Air fluid level sinusitis hemorrhage

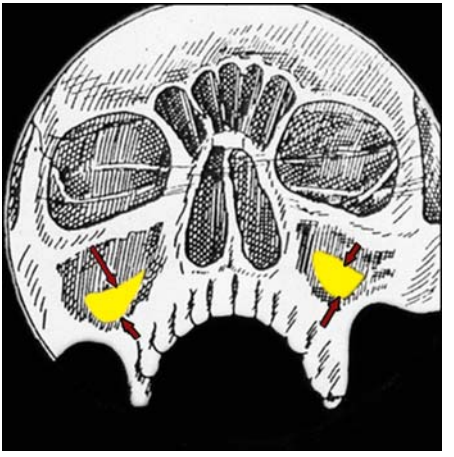


Fig. 15.2: Mucoperiosteal thickening of maxillary antra; the thickened mucosal line runs parallel to antral wall and is straight: allergic thickening is often scallaoped in appearance

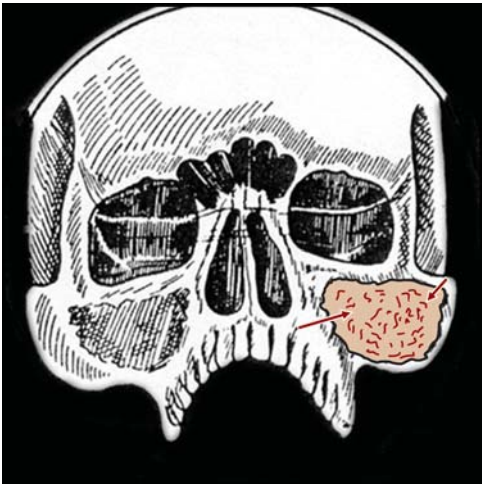


Fig. 15.3: Carcinoma of left maxillary sinus

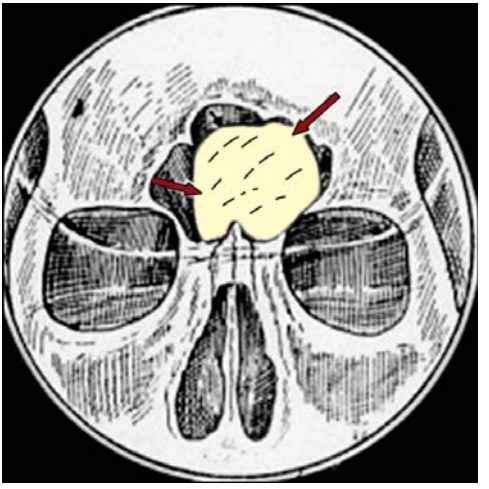


Fig. 15.4: Pressure atrophy of surrounding bone caused by a radiolucent expanding, sharply demarcated lesion

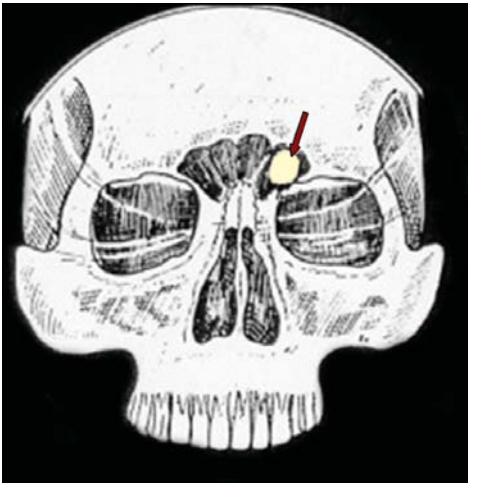


Fig. 15.5: Osteoma frontal sinus

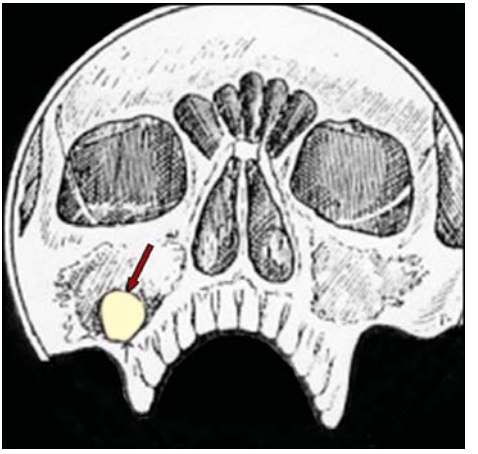


Fig. 15.6: Polypoid filling defect in maxillary antrum, allergic in origin

CHAPTER

16 Endocrine Disorders Affecting Oral Cavity

Hyperpituitarism (Fig. 16.1)

Pituitary gland lies within the sella tursica at the base of the brain. It has three distinct lobes. Hyperpituitarism result due to hyperfunction of the anterior lobe of pituitary gland producing growth hormone.

Before closing of epiphysis, gigantism occurs and after closure, acromegaly. In hyperpitutarism, bone overgrowth

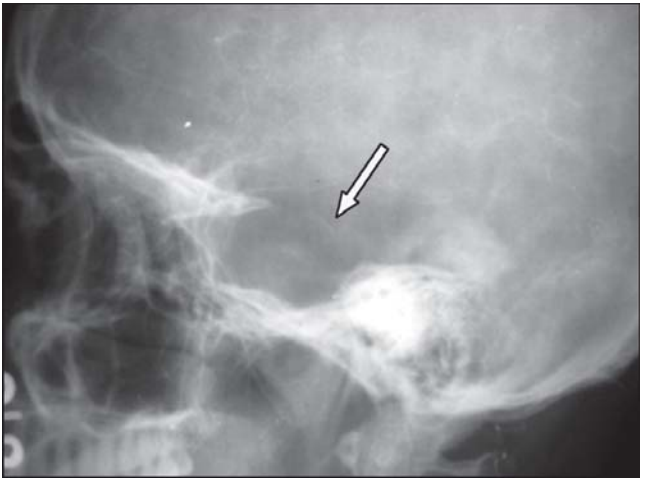


Fig. 16.1: Irregular enlargement of pituitary fossa

and thickening of soft tissue causes a coarsening of facial features. Head and feet become large with clubbing of toes and fingers. There will be enlargement of sella tursica, paranasal sinuses and thickening of outer table of skull.

Angle between ramus and body of mandible is widened. There is enlargement of inferior dental canal. Radiograph will show increased tooth size specially root due to secondary cemental hyperplasia.

ORAL MANIFESTATIONS

- Mandibular condylar growth is very prominent
- Overgrowth of mandible leading to prognathism
- Lips become thick like Negro's
- Spaced dentition
- Body and root may be longer than normal.

Hypopituitarism

It may be congenital or due to destructive disease. Space occupying lesions like craniopharyngioma, adenomas may result in Hypopituitarism.

CLINICAL FEATURES

- There is symmetrical underdevelopment but in some cases there may be disproportionate length of long bones
- Hypoglycemia may develop due to growth hormone and cortisol deficiency
- Onset of puberty is delayed
- Patient becomes lethargic, fat mass is increased
- Skull and facial bones are small.

ORAL MANIFESTATIONS

- Tooth eruption is hampered
- Overcrowding of teeth due to underdevelopment of alveolar arch
- Delayed exfoliation of deciduous teeth resulting in delayed eruption of permanent teeth.

Hyperthyroidism

It is also known as thyrotoxicosis, due to overproduction of thyroxine. It may be caused by Graves' disease (Figs 16.2A and B).

CLINICAL FEATURES

- Enlarged thyroid
- Asymmetrical and nodular enlargement.
- Thyroid may be tender

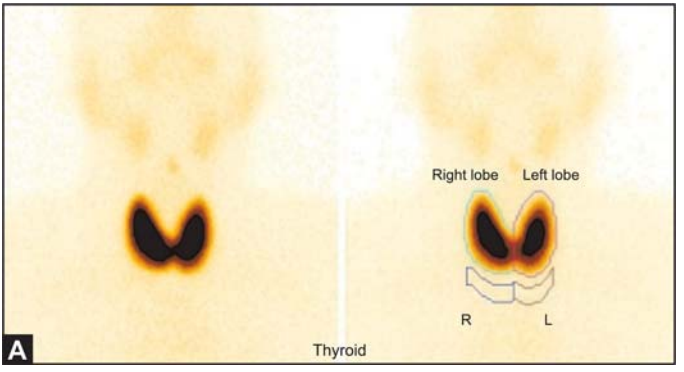


Fig. 16:2A: Graves' disease

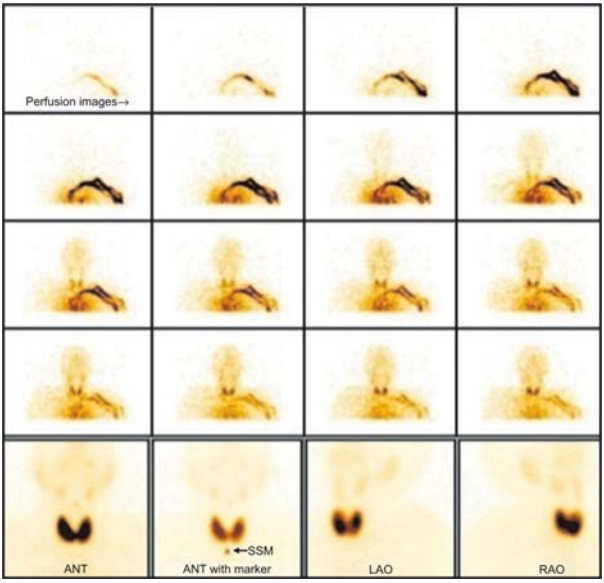


Fig. 16.2B: Sequential images showing Increased vascularity to thyroid gland

- Enlarged liver and spleen
- Nervousness, muscle weakness and fine tremors
- Cardiac palpitation, irregular heart beat and excessive perspiration
- Tachycardia and increased pulse pressure
- Ankle edema and systolic hypertension
- Amenorrhea, infertility, decreased libido and impotence
- There may be lymphadenopathy and osteoporosis.

ORAL MANIFESTATIONS

- There may be alveolar resorption
- Trabaculae may be of greater density
- Premature loss of primary teeth
- Early eruption of permanent teeth
- Early jaw development
- Generalized decrease in bone density
- Loss of edentulous alveolar bone.

Hypothyroidism

In this condition, secretion of thyroid is diminished. It may be due to atrophy of thyroid gland or failure of thyrotropic function of pituitary gland.

It may lead to three types:

- Cretinism- hormone failure occurs in infancy
- Juvenile myxedema occurs in childhood
- Myxedema occurs after puberty.

CLINICAL FEATURES

- Cretinism and myxedema may be present at birth
- Constipation and hoarse cry
- Delayed fusion of epiphysis
- Hair becomes dry and sparse.

MYXEDEMA

- Early symptoms include weakness, cold intolerance, lethargy and dry skin
- In late stage there is slowing of motor and intellectual activity

- Patient may gain weight
- Peripheral edema, decreased taste, and sense of smell
- Development of Muscle cramps
- Dull, expressionless face, sparse hair
- Facial pallor, puffiness of face and eyes.

CRETINISM

- Delayed development of teeth
- Enamel hypoplasia
- Abnormal dentin formation
- Overdeveloped maxilla
- Underdeveloped mandible
- Enlarged tongue
- Face becomes wider and lips may become puffy and thickened.

Hypoparathyroidism

There is insufficient secretion of parathyroid hormone. It may result from:

- Autoimmune destruction
- Surgical damage to parathyroid gland
- Parathyroid damage from radioactive iodine I-131.

Clinical Features

- Stiffness in hands, feet, and lips
- Parasthesia around mouth
- Tingling sensation in fingers and toes
- Anxiety and depression
- Intelligence is lowered.

ORAL MANIFESTATIONS

- Delayed eruption of teeth
- Hypoplasia of enamel
- Chronic candidiasis is present
- Altered taste
- Angular cheliosis
- Diffuse enlargement of parotid.

Hyperparathyroidism (Figs 16.3A and B)

There is an excess of circulating parathyroid hormone. Bone and kidney are the target organs. Serum calcium level is elevated.

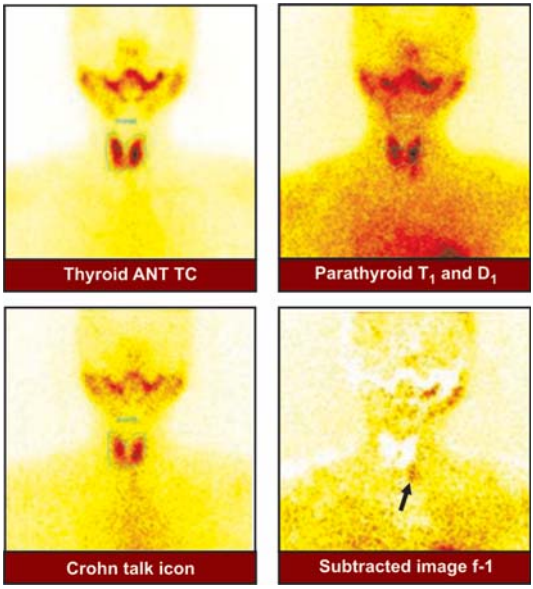


Fig. 16.3A: Technetium thallium scan showing left inferior parathyroid adenoma

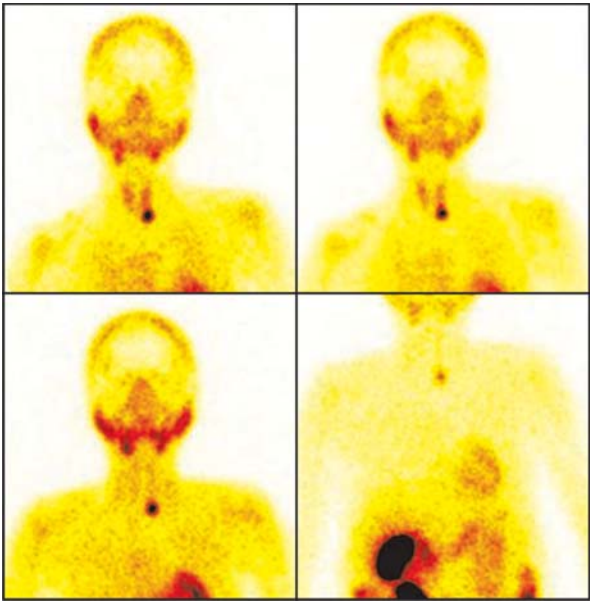


Fig. 16.3B: 99 mTc–MIBI scan showing left inferior parathyroid adenoma

Clinical Features

- Emotional instability
- Painful bones and joints
- Crampy abdomen
- Bowing of long bones
- Pigeon chest
- Collapse vertebra
- Muscle weakness, fatigue, polyuria and polydipsia.

RADIOLOGICAL FEATURES

- Ground glass appearance
- Moth eaten appearance with varying intensity
- Punctate and nodular calcification may develop in kidney and joints
- Pepper pot skull due to osteopenia
- Teeth may become mobile and migrate
- Loss of lamina dura which may be complete or partial.

Diabetes Mellitus

There is hyperglycemia. It is caused by disorder of carbohydrate metabolism due to deficiency of insulin and other factors such as genetic, autoimmune and pancreatic dysfunction.

CLINICAL FEATURES

- Polyphagia—excessive hunger
- Polyuria—an excessive urine passage
- Polydypsia—an excessive intake of fluid
- Visual difficulty
- Atherosclerosis and stroke
- Recurrent infections
- Blood pressure may be elevated
- Nephropathy.

ORAL MANIFESTATION

- Patients are more prone to periodontal disease
- There may be bleeding on probing

- Patient may develop fulminating periodontitis
- More severe and rapid alveolar resorption of bone
- It may result in median rhomboid glossitis
- *Candida albicans* due to impaired glucose level
- Trigeminal nerve may be involved
- Increased caries activity
- There is delay in healing of oral wound and infections
- Tongue is dry, fissured and atrophy of lingual papillae occurs.

Addison's Disease

It was first described by Addison in 1855. It may be caused by tuberculosis, metastatic carcinoma, hemorrhage and hypoplasia.

CLINICAL FEATURES

- General debilitation, feeble heart and postural hypertension
- Bronzing of skin and pigmentation of oral mucosa
- There is reduced resistance to infection and stress
- Muscle weakness.

ORAL MANIFESTATIONS

- Deep chocolate pigmentation of oral mucosa
- Buccal mucosa and angle of mouth is also involved
- Biopsy will show acanthosis with silver positive granules.

Cushing's Syndrome

It develops due to excess secretion of glucocorticoids by adrenal glands. It may be caused by:

- Exogenous corticosteroids
- Adrenal adenoma
- Carcinomas.

CLINICAL FEATURES

- Rapid development of obesity and moon face
- Weakness, distal extremities are thinner
- Diabetes or hypertension is found
- Menstrual irregularity
- Hirsuitism
- Hypertension.

ORAL MANIFESTATIONS

- Skeletal and dental age is retarded
- Jaw becomes osteoporotic.

CHAPTER 17

White and Red Lesions

These look whiter than the surrounding tissue. These are raised and roughened areas. There are many causative factors.

Non-keratotic and Keratotic White Lesions

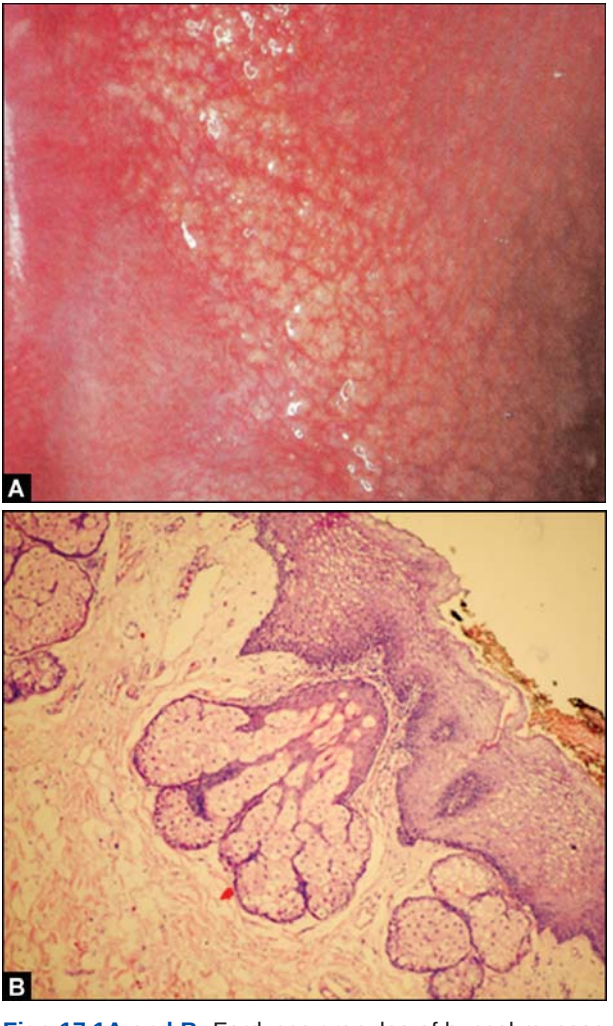
Non-keratotic lesions are easily dislodged with rubbing or at the most by scrapping. Those which resist scrapping involves mucosal epithelium increasing thickness of keratinized layers and are known as keratotic.

Leukoedema

In this condition, buccal mucosa becomes grayish white, whereas the softness and flexibility remains intact. Lesions can be scrapped temporarily but it develops again very quickly. Epitheliums in these areas are thicker.

Fordyce Granules

Oral mucosa contains number of sebaceous glands in vermilion border of lip and buccal mucosa. Histologically these are similar to skin sweat glands. These have no specific function. The number of these glands increases with age. If it occurs on lips and considered disfiguring, it may be removed surgically (Figs 17.1A and B).



Figs 17.1A and B: Fordyces granules of buccal mucosa clinical and histological view

Non-keratotic White Lesions (Fig. 17.2)

Several white lesions may be developed due to masticatory trauma. Unintentional biting of cheek, tongue or lip mucosa may produce ulcers. Tobacco induced keratosis may also develop. Habitual biting lesions are superficial and feel rough and poorly outlined.

Burns of Oral Mucosa

Burn is a frequent cause of non-keratotic white lesion. White color is due to pseudomembrane inflammatory exudates. Normally, saliva protects the mucous membrane. Hence in xerostomic condition injury becomes severe even those caused by hot cigarette smoke, ingestion of hot food and tea, etc. causes mild thermal burns in anterior third of tongue. A severe burn in central area may be caused by hot sticky pizza, holding dry CO_2 snow. Amongst drugs aspirin

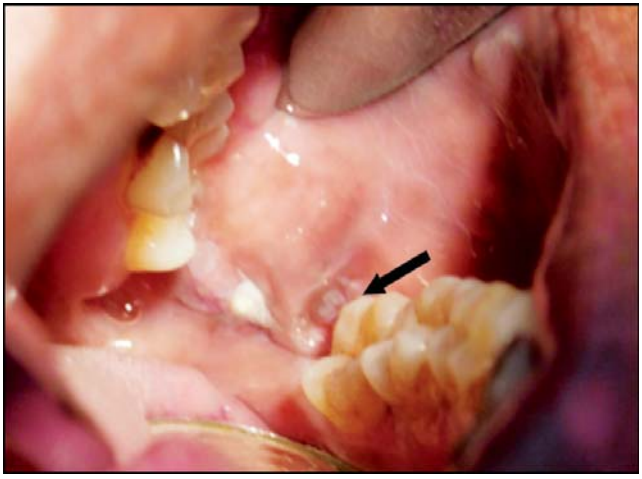


Fig. 17.2: Cheek biting resulting in non-keratotic white lesion

containing compounds may cause burn of oral mucosa. Application of 70% ethyl alcohol to dry mucosa area result in sloughing.

Uremic Stomatitis

BUN level above 50 mg/dl with renal failure may result in extensive pseudomembranous white lesions. Chemical burn results due to increased ammonia levels.

Candidiasis

It is associated with both non-keratotic and keratotic oral white lesions.

Acute Atrophic Candidiasis (Fig. 17.3)

It includes antibiotic sore mouth, raw painful mucosa with pseudomembranous white lesions. Tongue becomes

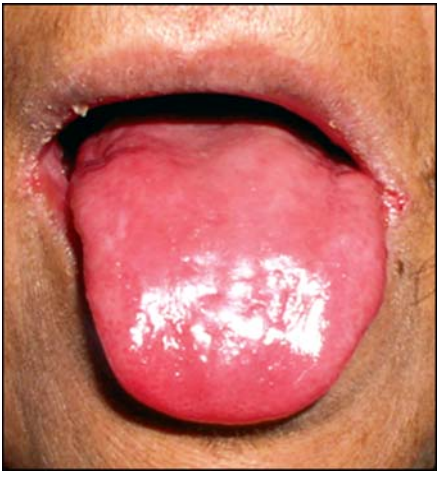


Fig. 17.3: Atrophic tongue and angular cheilitis due to candidial infection

smooth and depapillated. Antibiotic sore throat develops oral burning, bad taste and sore throat. Person may develop angular chelitis and thrush. White flecks of thrush can be removed leaving patchy red excoriation.

Denture Sore Mouth (Fig. 17.4)

It is a form of chronic atrophic candidiasis. There develops a diffuse inflammation area which may start bleeding on slight pressure. Poorly fitting denture results in this condition. There are mainly 3 types of it.

i. Diffuse erythema
ii. Localized simple pinpoint erythema
iii. Granular type.

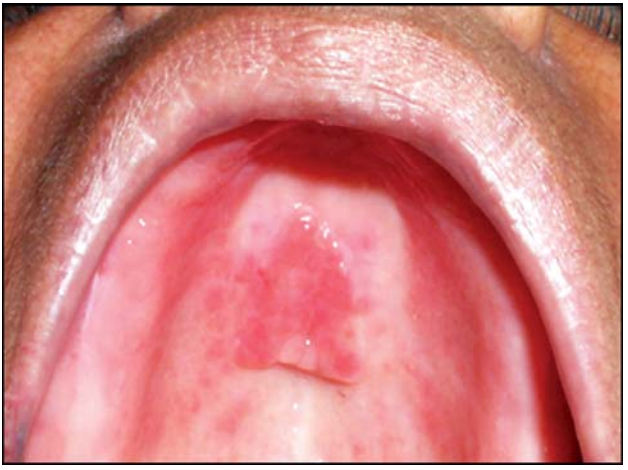


Fig. 17.4: Denture sore mouth due to ill fitting denture

Angular Cheilitis (Fig. 17.5)

Actually it is a clinical diagnosis for majority of lesions affecting lip commissures. B-complex deficiency, anemia may cause it. There may be associated denture stomatitis. Cheilo candidiasis is more extensive and often desquamative lesions affecting full lip instead of angles only.

Chronic Hyperplastic Candidiasis (Fig. 17.6)

Histologically lesions are quite different from those of thrush and atrophic candidiasis. Candida leukoplakia is an extremely chronic form and one finds white firm,

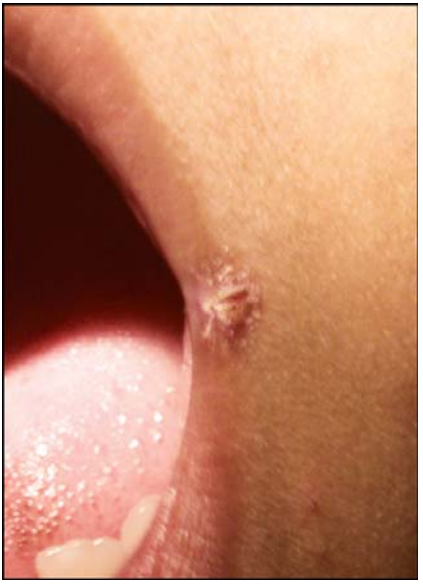


Fig. 17.5: Angular cheilitis

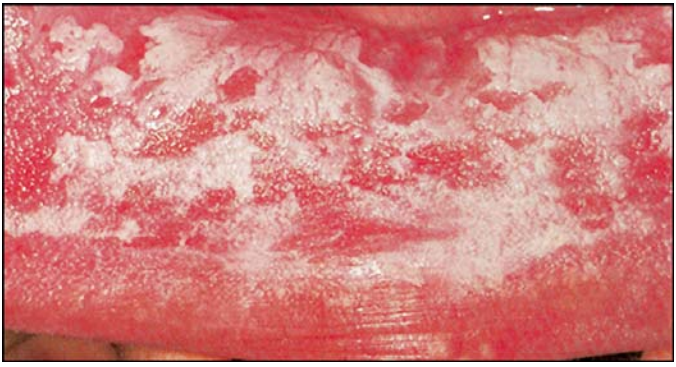


Fig. 17.6: Leukoplakia of labial mucosa

leathery plaques on lips, cheeks and tongue. Oral Candida leukoplakia is a characteristic of speckled lesions.

Thrush

It is known as pseudomembranous candidiasis. Actually it is a superficial infection of upper layers of mucosal epithelium. It forms a patchy white plaques or flecks on mucosal surface. Once you remove this area of erythema, shallow ulceration is seen. Antifungal drugs are helpful.

In infants lesions are soft, white or bluish. It is adherent to oral mucosa. Intraoral lesions are painless and being removed with difficulty, it leaves a raw bleeding surface. Any mucosal area of mouth may be involved. Constitutional symptoms are not present but in adults rapid onset of a bad taste may develop. Some may feel burning of mouth also.

Causative organisms are yeast like fungus causing thrush. It occurs in both yeast and mycelia forms of oral cavity and infected tissue. Candidate species are normal inhabitants of the oral flora. Concentration is 200-500 cells per ml of saliva. Carrier state is more in diabetics. The wearing of removal prosthetic appliances may also become asymptomatic carrier.

Predisposing factors include:

- Post administration of antibiotics
- Ill-fitting dentures
- Long-term consumption of cortisone
- Pregnancy and old age
- AIDS and low immunity
- Xerostomia.

Stomatitis

It is also known as leukokeratosis. Smoker's patch develops on palate in heavy smokers. Lesions are limited to smoke area. To start with, mucosa is reddened but becomes grayish white. It is thick and fissured.

Histologically epithelium shows acanthosis and hyperkeratosis. There is chronic inflammation in the sub-epithelial connective tissue. If smoker stops smoking, changes are reversible. Lesion has no precancerous potential.

Frictional Keratosis

Due to some local irritant an isolated area of thickened whitish oral mucosa develops. Histologically these show varied hyperkeratosis and acanthosis. Broken and rough

edges may cause the lesion. Majority of lesions will be reduced, if irritant is removed.

Focal Epithelial Hyperplasia

Lesion occurs primarily in lips and cheek. It is 0.1 to 0.4 cm. Flat, raised, whitish plaques are seen. Histologically local acanthosis is seen. Dyskeratosis is not present. Lesions may regress suddenly.

Geographic Tongue

It shows annular, serpiginous lesion of tongue and oral mucosa with slightly depressed atrophic center. Borders are white and raised.

White Sponge Nevus

It is also known as Cannon's disease. It is an autosomal dominant condition that affects only oral mucosa. Buccal mucosa is the site of lesions. Lesions are asymptomatic. Friction from mastication may strip off the keratotic layer. Cytological study will show empty epithelial cells. Centrally placed are pyknotic nuclei. There is no evidence of lesions being transformed into malignancy.

Hereditary Benign Intraepithelial Dyskeratosis

It is also known as Witkop – von Sallman syndrome. It is an autosomal dominant trait. Patient shows oral mucosal thickening plaques occur on bulbar conjunctiva. Histologically peculiar intraepithelial dyskeratosis in addition to acanthosis is seen. Cell with in cell phenomenon occurs.

Pachyonychia Congenita

It refers to oral leukokeratosis as well as striking nail changes. Nail lesions develop soon after birth and nails become thick and hardened with brownish material at nail bed. Paronychial inflammation is common. It affects primarily the dorsum of tongue. Frequent oral aphthous ulceration may develop.

Porokeratosis

Plaques are surrounded by raised border of epidermal proliferation. Plaques are produced by mutant clones of epidermal cells. On sections oral lesions show characteristic cornoid lamella.

Acrodermatitis Enteropathica

It is transmitted as an autosomal recessive character. It may be due to zinc deficiency. Zinc supplements will be treatment of choice. Person develops skin lesions, hair loss, nail changes and diarrhea. Retarted body growth and mental changes also occur.

Buccal mucosa, palate gingival and tonsils may show red and white spots. Erosions and ulcers are noted. Halitosis is severe.

Dystrophic Epidermolysis Bullosa (Fig. 17.7)

Trauma produces extensive bulla formation and desquamation. Hands, feets, esophagus and oral cavity are involved. Oral mucosa becomes thick, gray and inelastic. Lesions are smooth. Buccal and lingual sulci become

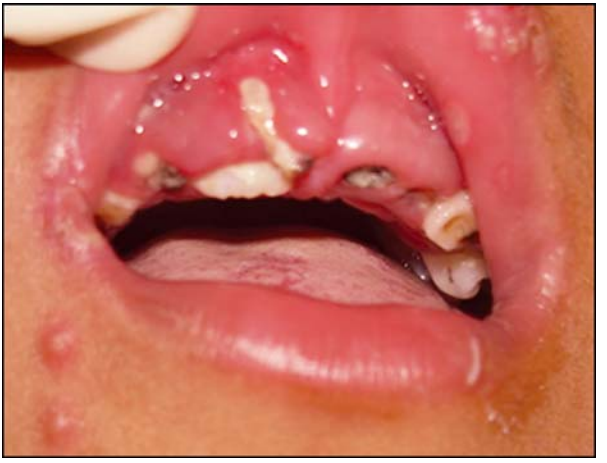


Fig. 17.7: Epidermolysis bullosa

obliterated. Scarring develops. Lips may become immobile. Patient may be dwarf. There may be associated conjunctival scarring, laryngeal stenosis and hoarseness of voice.

Keratosis Follicularis

It is rare and inherited as an autosomal dominant condition.

In less severely affected cases intraoral lesions are papular. It is referred as warty dyskeratoma. In severe cases dermal inflammatory exudates and a tendency to cobblestone changes is seen.

Some persons may develop psychological problem and mental disability.

Hereditary Mucoepithelial Dysplasia

It is an autosomal dominant disorder. Whole of the oral mucosa is involved and oral lesion is a fiery red. Gingival and hard palate are commonly involved. Fissured tongue is seen.

PRECANCEROUS LESIONS

Leukoplakia

It may be localized or diffuse and is of 3 forms:

i. Homogeneous
ii. Verrucous
iii. Nodular.

- Homogeneous leukoplakia is a localized lesion. If extensive, it is of consistent pattern. It may appear as wrinkled or papillomatous as well.
- Verrucous leukoplakia is an oral white lesion in which surface is broken up by multiple papillary projections.
- Nodular granular is hairy mixed white and red lesion. In it lesions are scattered over an atrophic patch of mucosa.

Leukoplakia can be developed on any part of oral cavity but is more common on buccal mucosa and gingiva. 50% lesions affect cheeks. Tobacco, alcohol, mechanical irritants are causative factors. If floor of mouth is involved then chances of transforming into malignancy are more. Microscopically one finds cellular dysplasia. Benign leukoplakia shows hyperkeratosis and chronic inflammatory cell infiltration. Cytological study is of little

importance. Topically applied toluidine blue shows the areas which are more likely to show carcinomatous changes. Biopsy is to be done from that area.

Surgical excision with or without grafting remains the standard treatment for leukoplakia.

Erythroplakia

It is reddened velvety textured mucosa. It is less common than white lesion leukoplakia. Several clinical variant have been described. It develops in elderly age group. Biopsy is needed. After removing the irritant toluidine blue staining should be done. A lesion that stains are likely to be precancerons in nature.

Carcinoma *In Situ*

Severe dysplastic changes in white lesion are an indication of precancerous state. More severe grade of dysplasia is carcinoma *in situ*.

Lesions of carcinoma *in situ* are to be treated vigorously with local excision. Wider surrounding area should also be removed, regional nodes are generally not removed. Majority of these lesions occur in floor of mouth.

Bowen's Disease

It is localized, intraepidermal squamous cell carcinoma. It may develop into invasive carcinoma over years. Clinically it is a slowly enlarging erythematous patch. Histologically epithelial cells are seen in complete disarray. Many will be of hyperchromatic nuclei.

This disease may develop in genital mucosa. Erythroplakia and Browen's disease both are hairy red patches.

Oral Submucous Fibrosis

It is a slow growing disease in which fibrous bands are formed. Movement of tongue may be restricted. There may be burning sensation. Etiology of disease is not known.

Discoid Lupus Erythematosis

Mixed red and white lesions are more common. A radiating pattern of very delicate white lines is seen. Alternate red, white and red zones provide a characteristic appearance. Histological changes consist of hyperorthokeratosis with keratotic plugs.

Most of the intraoral lesions in DLE occur on the cheeks with the gingival tissues and border of lip. Hot and spicy food will produce burning sensation. It responds to cortisone well.

Lichen Planus

It is a common dermatosis developing on mucus membrane. Causative factor of lichen planus is immunologically induced degeneration of basal layer of epithelium.

Generally it is a bullous form of disease and a painless lesion, the reticular form is slightly elevated, fine, whitish line. Lines are fine radiating of lacing in nature. Cheeks and tongue are generally affected.

- In the papular form lesion is less than 1 mm in size. It is whitish elevated lesion.
- Bullous lichen planus is rare. It presents coexisting bullous pemphigoid.

- Atrophic lichen planus is inflamed area covered by red, thinned epithelium.
- Erosive lesion is a complicated atrophic process when thin epithelium is ulcerated.
- Main histological features of lichen planus are:
  - Areas of hyperparakeratosis
  - Liquefaction degeneration
  - Dense epithelial band of lymphocyte damage of basal cell layer is the diagnostic point.

The lesions of oral lichen planus appears, regresses and reappear in unpredictable fashion. Lesions are too diffuse to remove surgically. Antihistamine rinses and corticosteroids are useful. In resistant cases intralesional injections of trimcinolne acetanide are useful. Retinoids may be given in addition.

Lichenoid Reaction

These reactions are to show degeneration of basal epidermal cells. In addition to lichen planus it includes erythema multiforme, dermatomyositis and lupus erythematosis.

Diseases showing lichenoid reactions

Site of reaction	*Disease*
Skin	Lichen nidus
	Lupus erythematosus
	Lichen striatus
	Lichenoid photodermatitis
Skin and oral mucosa	Lichen planus
	Lupus erthematosus
	Secondary syphilis

Drugs which may result in lichenoid reaction:

- Antihypertensives
  - ACE inhibitors
  - Chlorthiazide
  - Methyl dopa
  - Preactolol
- Antiarthritics
  - Colloid gold
- Anxiolytic
  - Lorazam
- Oral hypoglycemic agents
  - Diabenese
  - Tobulamide
- Dental casting alloys
- Antiparasites
  - Antimony
  - Organic arrencals
  - Choloroquin
- Antibiotics
  - Streptomycin
  - Tetracyclin.

CHAPTER 18

Benign Neoplasm of Oral Cavity

Neoplasm is an uncontrolled growth due to irregular growth of its cells. A malignant tumor arising from epithelial tissue is known as carcinoma, from gland is known as adenoma, from cartilaginous tumor it is called as 'chondroma'.

Spread

- Local invasion is when tumor penetrates into adjoining tissue due to rapid growth.
- Metastasis can be defined when tumor cells spread away from primary location. Spread may be through blood or lymph.

Table 18.1: Differences between benign and malignant tumors

Feature	*Benign*	*Malignant*
Rate of growth	Slow	Fast growing
Pain	Absent	Terminal stage is painful
Hemorrhage	Not common	Common
Size	Small	Large
Ulceration	Absent	Present
Cell uniformity	Regular	Irregular
Tissue architecture	Intact	Mostly lost
Capsule	Usually present	Absent
Necrotic area	Absent	Present
Prognosis	Good	Poor

Benign Neoplasm of Epithelial Tissue Origin

Kertoacanthoma

It is a benign endophytic epithelial tissue neoplasm. It develops over skin surface of lips near vermilion border.

Clinical Features

- It starts as a small bud like growth and grows fast reaching to the size of 1-2 cm in 6-8 weeks.
- It is a small, well circumscribed, elevated, umbilicated lesion with a central depression.
- Lesion is single, painful with lymphadenopathy.
- With in 6-8 weeks it regresses automatically. Hence, it is known as self healing cancer otherwise surgical excision may be done.

Papilloma

It is a common benign neoplasm arising from epithelial tissue. There develops a exophytic papillary growth of stratified squamous epithelium.

Clinical Features

- Slow growing, small, painless, cauliflower like growth with many finger like projections. Size varies from a few mm to 1 cm.
- It is pedunculated, ovoid swelling, white in color.
- Multiple papilloma of oral cavity is known as papillomatosis.
- It is not a premalignant condition. Surgical excision along with base is advised. Recurrence is very rarely seen.

Intradermal Nevus

It is congenital development of tumor like malformation of the skin/mucus membrance. Function of nevus cells is to produce melanin.

Clinical Features

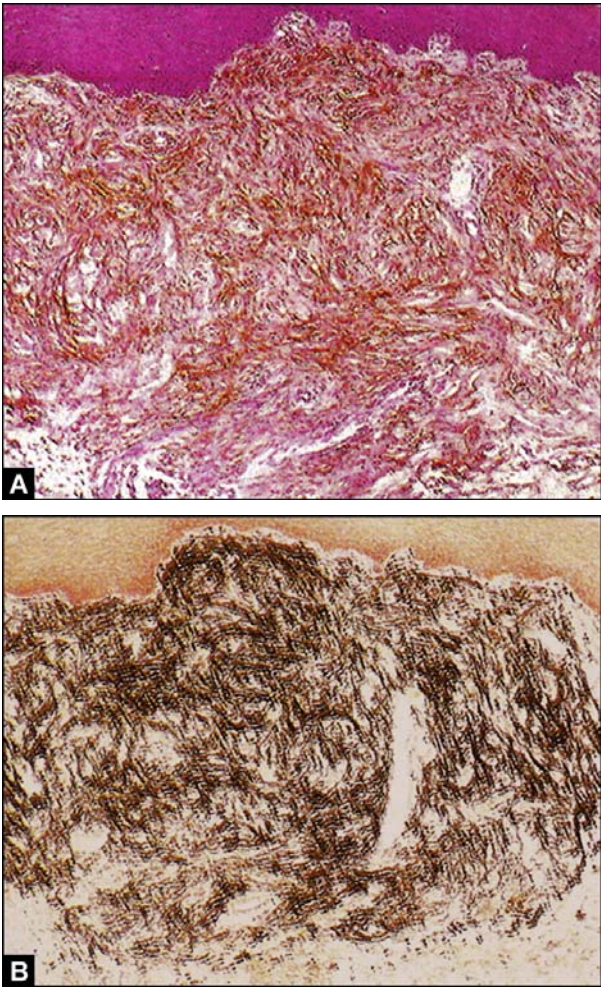
- These are commonly known as mole of dark brown color. It is slightly raised.
- Intraoral lesions are commonly seen over hard palate or gingival. These grow slowly.
- Microscopically nevus reveals cluster of nevus cells confined within connective tissue. No mitotic activity is seen.
- These generally don't require any treatment.

Blue Nevus (Figs 18.1A and B)

It is common pigmented lesion of oral cavity.

Clinical Features

- These are commonly seen on the mucosal surfaces of hard palate.
- It is a dome shaped, dark blue papule.
- Cells of blue nevus elongated, bipolar and spindle shaped.
- Few pigmented macrophages may be present.
- These don't change into malignancy and hence, surgical resection serves the purpose.
- Compound nevus presents the combined features of intradermal nevus and junctional nevus.



Figs 18.1A and B: Blue nevus

BENIGN NEOPLASM OF BONE

Osteoma

These are benign neoplasm. It may consists either mature compact bone or cancellous bone. Jaw bones are often affected.

Clinical Features

- Patient is mostly young male.
- It causes swelling of bone of about 1 cm in diameter.
- Lesion is not painful.
- It causes expansion of cortical plates of bone. Teeth may be displaced.
- Osteomas of maxillary antrum predisposes to sinusitis.
- On X-ray cortical bone is sclerotic, periosteal layer is more active.
- Surgical excision is needed.
- Osteoid osteomas and osteoblastomas are having similar findings.

BENIGN NEOPLASM OF CARTILAGE TISSUE

Chondroma

It is a benign neoplasm of cartilaginous origin. It affects the anterior part of maxilla.

Clinical Features

- It is a slow growing, painless bony and hard swelling
- Expansion and distortion of cortical plate occurs
- Most lesions are painful

- Covering epithelium is smooth
- Chondromas are mostly single tumor
- Histological study shows that lesion consists of well defined lobules of hyaline cartilage containing mature chondrocytes. Surgical excision helps.

Chondroblastoma

It develops in younger age group.
Mandibular condyle is affected more.

Clinical Features

- It is a large, bony hard lesion
- There may be bulging of jaw bones
- Displacement of regional teeth occurs
- It may be tender

Surgical resection is the treatment of choice.

CHAPTER 19

Malignant Neoplasm of Epithelial Tissue

Squamous Cell Carcinoma (Figs 19.1 and 19.2)

It is derived from stratified squamous epithelium; commonest sites involved are lower lip, lateral borders of tongue and floor of mouth. Tobacco, betal nut chewing, alcohol, AIDS, lichen planus are the important causative factors.

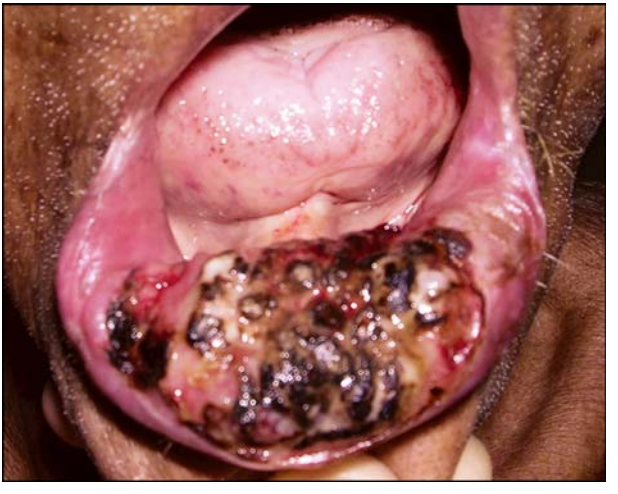


Fig. 19.1: Carcinoma of lower lip

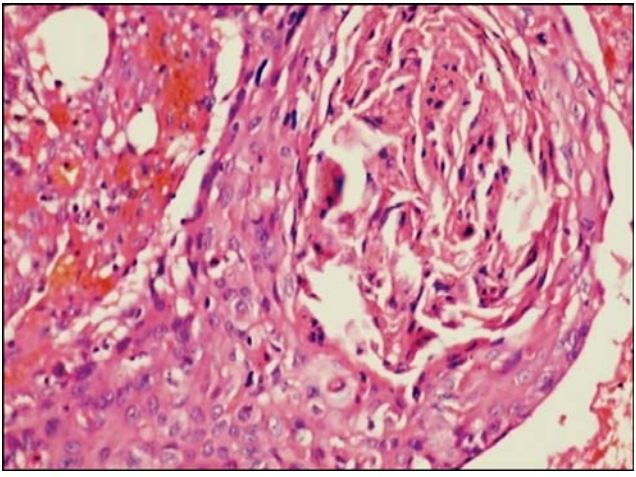


Fig. 19.2: Keratinization seen in squamous cell carcinoma

Clinical Features

- To start it may appear as extended oral leukoplakia and erythroplakia.
- It may be symptomless, white or red vegetated patch.
- It is painless in nature.
- Advanced lesion is fast growing irregular and large tumor mass.
- Ulcer has indurated borders with everted margin.
- Secondary infection may result in pain. It bleeds on touching.
- Tumor is fixed to tongue.
- Regional lymph nodes are tender, fixed and enlarged.
- Untreated case may destroy oral tissue and extend through cheek. Patient may not be able to eat food as pathological fracture of jaws may take place.

- Such cancer may also be present in intestine and lung.
- In an oral cavity itself two or more separate malignant lesions may be seen.
- Tongue lesions are more vascular and start bleeding even on slightest touch. Tongue may be fixed to floor of the mouth and patient may be unable to talk or swallow.
- Histologically lip carcinoma is well differentiated. If treated before metastasis, prognosis is good.
- Radiographically bone involved shows large, irregular and ill defined radiolucent area and appears as of 'moth eaten' type.

In addition to histopathology, exfoliative cytology is helpful. Toludine blue test is needed to detect dysplastic lesions.

Staging of Tumor

T = Primary tumor N = Regional lymph node
M = Metastasis
T_0 - No evidence of tumor
T_1S - Carcinoma in situ
T_1 - Tumor 2 cm or less
T_2 - Tumor 2-4 cm in size
T_3 - Tumor size greater than 4 cm
T_4 - Tumor invades surrounding structures.
N - Regional lymph node
N_o - No palpable lymph gland
N_1 - Palpable ipsilateral node
N_2 - Suspicious palpable contralateral node
N_3 - Palpable, large fixed node
M - Distant metastasis
M_o - No distant metastasis
M_1 - Clinical or imaging evidence of metastasis

Basal Cell Carcinoma

It is a locally aggressive malignant neoplasm. It does not metastasize. It arises from basal layer of epidermis or from hair follicle. Direct strong sunlight is the causative factor. Common site involved includes upper lip, nasolabial folds, cheek, forehead, etc. It does not arise from oral mucous membrane.

Clinical Features

- It starts as a slow growing small nodule.
- It develops into central crusted ulcer with an elevated but smooth rolled border.
- It develops as a sole lesion.
- If not treated it invades adjacent structures
- There is basaloid cell's proliferation but cells doesn't show any mitosis.
- In some cases squamous cell may occur in tumor.

Surgical treatment followed by radiotherapy gives good results.

Malignant Melanoma

These develop from melanocytes of skin or mucous membrane. These are most aggressive and deadly tumors. Commonest sites involved are hard palate and gingiva.

Clinical Features

- Most of the lesions are pigmented and are dark brown in color
- It starts as macular pigmented focal lesions.

- Initially it grows very fast into large, painful diffuse mass.
- Surface ulceration is very common.
- Small satellite lesions may develop at periphery.
- There is widespread spread of tumor cells in lung, liver, bone, and brain.

Radical surgery and prophylactic neck dissection is advised. Still survival chances are sleak.

Verrucous Carcinoma (Fig. 19.3)

It is diffused; non metastasing, well differentiated malignant neoplasm. It mostly develops in tobacco chewing patients.

Clinical Features

- Commonest site involved is gingiva, alveolar mucosa and buccal mucosa.

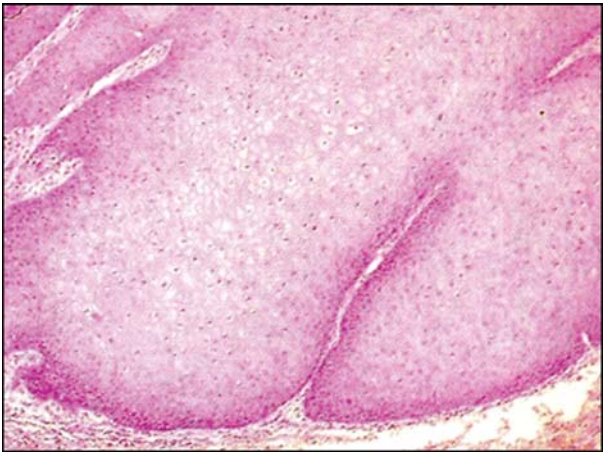


Fig. 19.3: Verrucous carcinoma showing multiple deep clefts

- Surface of the lesion shows multiple deep clefts in between
- Lesion may be single or multiple involving different parts of buccal mucosa.
- Pain may develop making chewing difficult.
- It becomes rapidly fixed to underlying bone.
- Regional lymph nodes are often enlarged.
- Malignant epithelial cells are usually well differentiated.

Spindle Cell Carcinoma

It is an unusual form of poorly differentiated squamous cell carcinoma consisting of spindle shaped epithelial cells.

Clinical Features

- Lesion may cause pain, ulceration and swelling. There is fleshy but polypoid growth pattern.
- Histologically tumor cells often exhibit marked nuclear hyperchromatism. There is a minimum degree of epithelial dysplasia with little or no keratin formation.
- This tumor is less aggressive, hence wider surgical excision serve the purpose.

Giant Cell Fibroma (Fig. 19.4)

It develops from fibrous connective tissue, mandibular or maxillary gingiva is mostly affected.

Clinical Features

- Lesion is pedunculated.
- It has papillary or warty surface
- Some may be painless, smooth nodular growth
- Histologically multiple multinucleated giant cells.

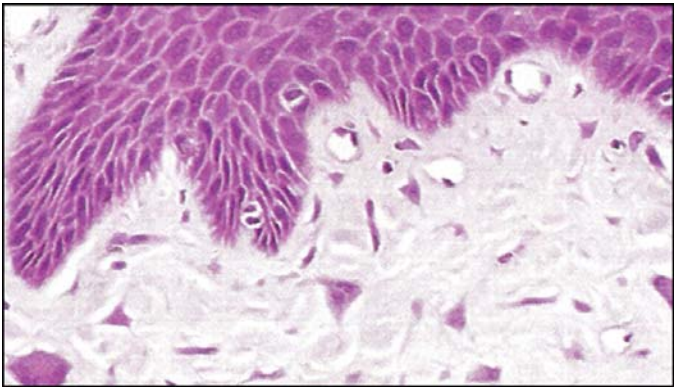


Fig. 19.4: Giant cell fibroma

- Nuclei are large of giant cells. Numerous small capillaries are noted.
- Surgical excision is the line of treatment.

Desmoplastic Fibroma

These are benign neoplasm arising from mesenchymal tissues of jaw bone. It develops in younger age group. Mandible is affected more.

Clinical Features

- These are generally asymptomatic neoplasm.
- Neither teeth nor lesion is tender
  Treatment includes local excision and curettage.

Central Ossifying Fibroma

It is a central ossifying fibroma. It represents a well demarcated expansile jaw lesion. It is well encapsulated.

Children and young adults are affected.

Clinical Features

- There is usually a single lesion
- There is localized, painless, non tendered, bony hard swelling in jaw
- Tumor is slow growing but may disfigure the face
- In some cases displacement of teeth may be seen due to expansion and distortion of cortical plates
- Histopathologically highly cellular fibroblastic stroma with 'whorled pattern' collagen fibers may be noted.

Central Giant Cell Granuloma (Fig. 19.5)

It is a benign intraosseous destructive giant cell lesion. Generally anterior part of jaw bone is affected. It is a reactive lesion.

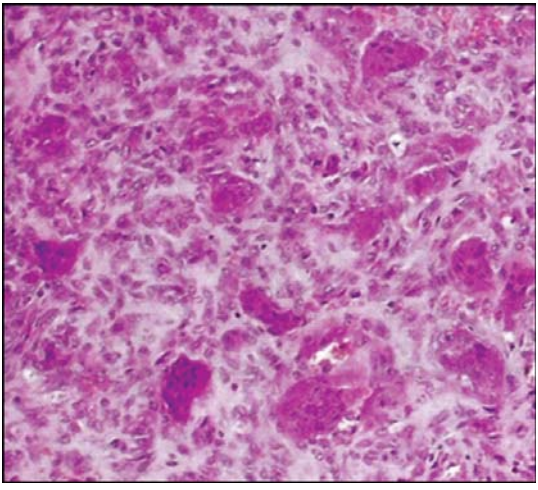


Fig. 19.5: Central giant cell granuloma

Clinical Features

- It affects mandible more than maxilla.
- Some lesions may cross the mid line.
- It is a small, slow enlarging, and bony hard swelling of jaw.
- Lesion may cause perforation of cortical plate.
- Teeth in affected region are always vital.
- Radiographically lesion will slow multilocular radiolucent areas in jaw and it gives a soap bubble appearance.
- When unilocular it will show a drop shaped appearance.
- Histologically central giant cell granuloma shows multinucleated giant cells. Several areas of hemorrhage and hemosiderin pigmentation are also seen around these blood capillaries.

MYXOMA (FIG. 19.6)

These resemble primitive mesenchyme and are true neoplasm. Oral submucosal area, salivary gland and jaw bones are commonly affected.

Clinical Features

- It is a slow growing invasive tumor that sometime reaches large dimensions.
- Jaw is distended.
- Radiographs shows a soap bubble appearance.
- Being locally aggressive radical surgery is advised.

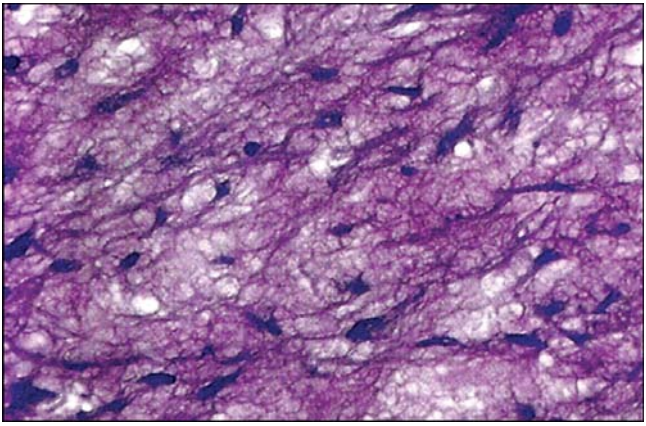


Fig. 19.6: Myxoma

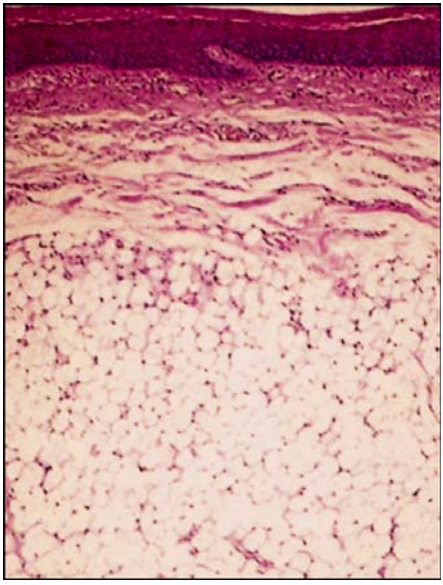


Fig. 19.7: Lipoma

LIPOMA (FIG. 19.7)

It is a tumor of adipose tissue. It is composed of mature fat cells. Cheek, tongue and salivary glands are frequently involved.

Clinical Features

- These are painless lesions with cystic feeling.
- These are yellowish in color with smooth surface.
- There is no surrounding capsule.
  Simple surgical excision helps

HEMANGIOMA

These are proliferative lesions of vascular tissue. It may be by birth. In oral cavity these frequently occur.

Clinical Features

- These are raised, red/blue/purple lesions
- Lesions are raised, but are easily blanched on pressure
- These are soft and pulsatile.
- Capillary hemangioma
  - These are characterized by small, numerous, endothelial lined pyogenic granuloma.
- Cavernous hemangioma
  - It reveals large, irregularly shaped, dilated endothelial lined sinuses.
  - Such blood pooled sinuses are communicating with each other.
  - These lack muscular coat.

Local exicision is the treatment. In case of large lesion sclerosing agents may be given earlier to reduce the size.

Lymphangioma

These are benign neoplasms due to proliferation of lymphatic vessels. Most of lesions are present at birth. Mostly tongue is involved.

Clinical Features

- These are painless, nodules or vesicle like lesions over oral mucosa
- Color of lesion is lighter than surrounding mucosa
- On palpation 'crepitation' sound is produced
- These regress automatically
- Lymphangioma of tongue produces macroglossia.

BENIGN NEOPLASM OF MUSCLES

Leiomyoma

It is a benign neoplasm of smooth muscle cells. These generally occur in tongue. Palate and buccal mucosa may also be involved.

Clinical Features

- These grow slowly and are painless
- Sub mucosal nodules may be seen
- These are firm and yellow in color
- Lesion can be multi nodular
- These are composed of spindle shaped smooth muscle cells.

Surgical excision including surrounding normal tissue is practiced.

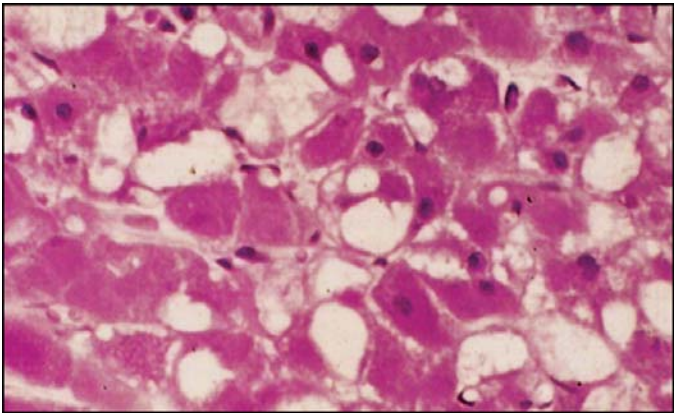


Fig. 19.8: Rhabdomyoma

Rhabdomyoma (Fig. 19.8)

These are benign neoplasm of striated muscles. Tongue and floor of mouth are usually involved.

- It is a slow growing
- It is well circumscribed, painless mass.
- Lesion is often deep seated.

TUMORS OF NEURAL TISSUES

Schwannoma

These are derived from shwann cells.

Intraorally, dorsum of tongue is most favored location. Buccal mucosa, floor of mouth, gingiva, and lips can also be involved.

Clinical Features

- It clinically presents as a slowly enlarging lesion.
- It is painless, nodule of different size.
- Sometime small, lobulated, firm growth may occur in gingiva.
- It has a true capsule.
- Surgical excision is the line of treatment.

Neurofibroma

It arises from perineural fibroblasts. It is a dominant hereditary condition with overgrowth of nerve sheath.

Clinical Features

- These present as small and asymptomatic nodules.
- These are submucosal with multi lobulated surface.
- These are painless in nature.
- On skin these are soft and firm freely mobile nodules.
- Nodular lesions vary in size and number varies widely.
- Radiographically it is a well dermarcated unilocular or multilocular radiolucent areas are seen.

MALIGNANT NEOPLASM OF ORAL CAVITY

Osteosarcoma (Fig. 19.9)

It is a very common neoplasm arising from bone. According to the site tumor has been divided into:

- Periosteal sarcoma
- Medullary osteosarcoma
- Periosteal osteosarcoma
- Soft tissue osteosarcoma.

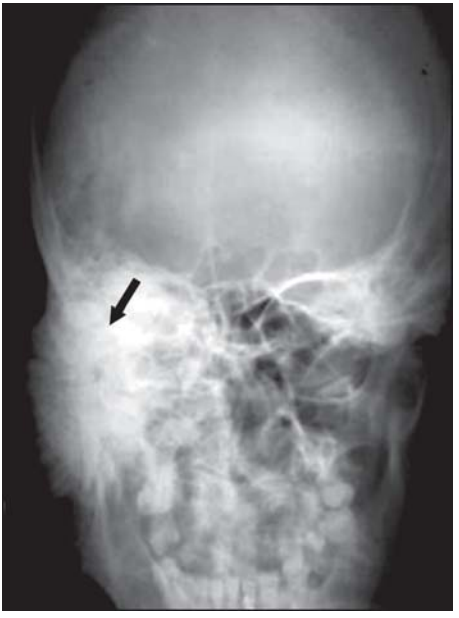


Fig. 19.9: Osteosarcoma of maxilla

Maxilla is affected more than mandible.

Clinical Features

- It s a fast growing
- It develops as a painful swelling of jaw
- Expansion and distortion of cortical plates may takes place
- Severe deformity of face leads to the displacement of teeth
- Overlying skin becomes red and inflamed
- Ulceration, hemorrhage and pathological fracture takes place.

Radiological Findings

- In osteolytic type it presents irregular, radiolucent areas giving rise to a moth eaten appearance
- Expansion, destruction and perforation of cortical plates is noted
- Teeth are displaced in position.

Non-Hodgkin's Lymphoma

It occurs more commonly than Hodgkin's lymphoma.

Clinical Features

- Patient may develop, fever, night sweats, malaise with weight loss.
- Oral lesion is fast growing, painful and firm.
- There may be ulceration and bleeding.
- Multiple lymph nodes are firm and rubbery.
- Non-Hodgkin's lymphoma is also noted in AIDS patients.
- Swelling of gingiva and palate are common.

Radiological Findings

- Floating teeth in radiolucent area may be seen.
- There will be diffuse, large, irregular areas of radiolucency with expansion and destruction.

Chemotherapy followed by radiotherapy will be the treatment of choice.

Hodgkin's Lymphoma

It is not a common disease. Males between the age of 15 and 35 years are affected.

Clinical Features

- Generalized lymphadenopathy is the hallmark of the disease
- Enlarged lymph nodes are firm, and rubbery in consistency
- Fever, weight loss, pain abdomen are the prominent features
- There may be persistent cough due to pressure on trachea
- Liver and spleen enlarged
- Oral lesion includes submucosal swelling with ulceration. Pain is present
- Lesion may be fixed to underlying bone.
  Chemotherapy with radiotherapy helps.

Multiple Myeloma

It is a group of malignant disease of plasma cells. It infiltrates bone and soft tissue. Bones anywhere in the body can be affected.

Clinical Features

- Deep bone pain is an early feature
- Pain increases with movement. Numbness of chin and lips may develop.
- Jaw swelling and pain may be confused with toothache.
- Jaw swelling, crackling or pathological fracture may develop.
- Extraction of teeth results in severe hemorrhage.
- Radiographs will show numerous punched out areas of radiolucency with peripheral bone reaction.
  Chemotherapy is given but disease is fatal.

Rhabdomyosarcoma

It develops from striated muscle cells. Most of tumors develop from childhood. Tongue and palate are usually affected.

Clinical Features

- Its a rapidly growing lesion
- It causes swelling, pain and extensive tissue damage.
- These are fixed, ulcerated and hard.

It has a poor prognosis even after surgery/chemotherapy followed by radiotherapy.

Neurogenic Sarcoma

It is a malignant tumor of Schwann cells. It has a poor prognosis.

Clinical Features

- These are rapidly growing and painful
- It is an exophytic mass
- Lips may be anesthetized
- Expansion of mandible occurs
- Lesions doesn't move and are fixed to underlying tissue
- Nuclei are often pleomorphic and hyper chromatic.

Neoplastic cells spread along the affected nerve.

Metastatic Tumors of Jaw

Primary tumors of bronchus, kidney, colon, and prostate metastasize to jaw. Mandible is more affected.

Clinical Features

- Tooth mobility
- Pain and swelling
- Expansion of cortical plates.

Radiographs shows osteolytic/osteoblastic areas.

Fibrosarcoma

These develop from fibroblast cells.

Clinical Features

- It commonly arises from cheek, tongue, gingiva and floor of mouth
- Among the jaw lesions mandible is most commonly involved
- To start with, the lesion is asymptomatic
- Soon it enlarges into a large, painful fleshy mass
- Lesion is firm and indurated
- These are not capsulated tumors
- Radiographs shows sharply defined radiolucent area.

Radical surgery chemotherapy is the treatment of choice.

Hemangioendothelioma

It is a malignant angiomatous neoplasm of mesenchymal tissue. It is more common in younger age group. Oral lesions are not very common.

Clinical Features

- It is a fast enlarging
- Localized, nodular, painful swelling

- Surface ulceration is present
- Teeth becomes mobile and bleed on pressure
- Jaw shows destruction of the lesion is expansible with cortical expansion.

  Surgical excision and radiotherapy helps.

Kaposi's Sarcoma

It is a malignant neoplasm arising from endothelial cells of capillaries.

Clinical Features

- It affects skin, lymph nodes, bone and viscera
- With AIDS lesions are multicentric
- Homosexuals are affected more easily
- It has three states:
  i. *Patch stage*—It is the initial stage having red, pink, or purple. These develop over oral mucosa.
  ii. *Plaque stage*—Patch converts into plaque. Lesion is large and raised.
  iii. *Nodular stage*—There develops multiple nodular lesions on the skin.

Being multicentric surgery is not feasible. Radiotherapy and chemotherapy is given as a treatment of choice.

Ewing's Sarcoma

It is highly malignant. It arises from endothelial cells of blood vessels within the bone.

Clinical Features

- It develops between 5 and 25 years
- Mandible is affected more than maxilla
- Person develops fever, leukocytosis ESR is raised
- Jaw bone is expanded
- Mobility of tooth is a common feature
- Neoplasm develop surface ulcer.

Radiological Features

- Expansion, distortion of cortical bones
- There is radiolucent area with ill defined margins
- Periosteum shows lamellar layering.

Radiotherapy and chemotherapy is the line of treatment.

Chondrosarcoma

It is a malignant neoplasm of bone. In this condition abnormal cartilage tissue is formed but no osteoid bone is produced. Peak age is 30-40 years. Males are more affected. In mandible it develops in posterior region.

Clinical Features

- It produces a painless swelling of jaw
- Later on it becomes painful and tender
- It is very fast enlarging tumor, causing severe expansion of bone.

Radiological Features

- It shows moth eaten radiolucent area in bone with ill defined borders
- Blotchy areas of radiopacities are also found

Even wide surgical excision gives poor prognosis.

CHAPTER 20

Sequel of Radiation on Oral Tissues

Oral Mucousal Membrane

The mucus membrane shows areas of redness and inflammation. Later it breaks down to form white to yellow pseudo membrane. As mucositis is very severe, topical anesthetics will be required. Secondary infection of *Candida albicans* may develop. After treatment, it may take about two months to heal. Then mucus membrane is atrophied, becomes thin and avascular. Ulcers can result from a denture sore.

Salivary Glands

Salivary glands are more sensitive. These develop due to progressive loss of secretion of saliva. Mouth becomes dry and tender. Swallowing becomes painful and difficult. Saliva looses its lubricating power. The pH value of saliva becomes acidic and the buffering capacity of saliva falls. In progressive irradiation, glands show fibrosis, loss of fine vasculature and concomitant parenchymal degeneration.

Taste Buds

These are also very sensitive to radiation. Radiation leads to the degeneration of normal histological architecture of taste buds. Also there is a loss of acquity of taste during

the third week of radiation. Bitter and acid flavors are more severely affected when posterior tongue is irradiated and salt/sweet when anterior 2/3rd is irradiated. After 4 months of irradiation, taste sense starts improving.

Teeth

During development, radiation retards growth of teeth. Before calcification, it may destroy tooth bud. After calcification, it may inhibit cellular differentiation. It may result in defect of permanent dentition, or dwarfing of tooth. Postradiation adult teeth are resistant to the effect of radiation. It has no effect on crystalline structure of enamel, dentin or cementum. Due to decrease in saliva the development of radiation caries can be seen.

Radiation Caries

It is a rampant form of dental decay due to change in salivary glands and saliva, reduced flow, decreased pH, increased viscosity and reduced buffering capacity.

Caries are of three types:

- Dark pigmentation of entire crown
- Widespread superficial lesion attacking buccal, occlusal, incisal and palatal surfaces
- Loss of crown.

Effects on Jaw

Radiation brings changes in:

- Vasculatures of periosteum and cortical bone
- It destroys osteoblastic cells more than the osteoclastic cells

- Marrow tissue becomes hypovascular, hypoxic and hypocellular
- Mineralization may be reduced making bones brittle
- May result in osteoradionecrosis.

CHAPTER 21

Chronic Orofacial Nerve Pain

It is a sudden paroxysmal pain along the distribution of one or more cranial nerve. Neuralgias of craniofacial region include:

1. Trigeminal neuralgia.
2. Glossopharyngeal neuralgia.
3. Occipital neuralgia.
4. Geniculate neuralgia.
5. Acute herpes zoaster.

Trigeminal Neuralgia

Primary is idiopathic while secondary may result due to trauma. Idiopathic neuralgia involves maxillary and mandibular branch. Due to stimulation of trigger zones recurrent shock like unilateral pain develops for few seconds to few minutes.

Such trigger zones lie in the same dermatome. Normally after each episode there is a refractive period. During this period even stimulation of trigger zone will not produce pain. No sensory loss is detected. No specific cause is known. Progressive degeneration and demyelination of trigeminal ganglion is seen.

Secondary trigeminal neuralgia can result due to intra cranial tumor, tumor vascular malformation, CNS lesions involving trigeminal pathways.

Trigeminal neuralgia due to multiple sclerosis may have bilateral facial pain. There may be hypoaesthesia too.

In both cases carbamazepine and GABA are used. Phenytoin in doses of 400 mg/day reduces the dose of carbamazepine.

Glossopharyngeal Neuralgia

It is a neuralgia of 9th cranial nerve. Its pain is paroxysmal but less severe than trigeminal nerve pain. Location of trigger zone and pain sensation follows distribution of the nerve. It includes posterior tongue, pharynx and infra-auricular area.

Pain is triggered by chewing and swallowing because all this stimulate pharyngeal mucosa.

Glassopharyngeal neuralgia may be associated with trigeminal neuralgia. Sometimes it may be associated with vagal symptoms.

Intra or extra cranial tumors may compress cranial nerve. Treatment is similar to trigeminal neuralgia.

Occipital Neuralgia

It is a neuralgia in the distribution of sensory branches of cervical plexus in neck. Trauma, neoplasm or infection is the most important cause.

Geniculate Neuralgia

It is characterized by pain in ear and anterior tongue. Location of pain is similar to sensory distribution of the

nerve, i.e. external auditory canal, posterior auricular region and the soft palate. If motor root is also involved then facial palsy may develop. Pain is not too severe.

Short course of high dose steroid therapy is beneficial.

Acute Herpes Zoaster

The neurotropic virus travels along the involved nerve. It causes neuritis and vesicular disease of skin and mucous membrane. Eruption is located to a single dermatome.

High dose antiviral therapy in acute phase of herpes zoster improves the rate of healing. Intensity of pain is also reduced.

Post herpetic neuralgia may continue in old persons.

CHAPTER 22

Fever

If one can diagnose the cause of fever and pain, fifty percent of the patients in OPD can be treated well. It is also true that number of patients don't feel that they are sick even although they are having fever as in case of early tuberculosis. While some fevers give lot of toxemia and patient looks ill from a distance as in typhoid. As opposed to it in case of malaria person feels sick when he is having rigor otherwise he remains well.

Fever with Rigor

Many patients come with rigors. Common conditions in which fever occurs with rigors are malaria, kala azar, amoebic hepatitis, liver abscess, pyelitis, influenza, filariasis, lung abscess, dengue and pyocele. All septic conditions like tonsillitis, sinusitis, lobar pneumonia or bronchopneumonia can result in rigors.

Malaria

It has three stages:

- Cold stage
- Hot stage
- Sweating stage.

Cold stage lasts 15 minutes to 2 hours and is followed by hot stage in which fever may go very high. On an average, hot stage lasts for 4-8 hours. It is followed by sweating stage which lasts for 1 to 4 hours.

Patient may have fever, vomiting and excessive thirst. Patient feels exhausted. In malignant malaria, fever may be accompanied by sever headache, diarrhea and toxemia. There may be successive bouts of fever followed by severe cachexia. Splenic enlargement will be there.

Kala Azar

It is characterized by fever, hepatic and splenic enlargement. Fever is having double daily remission. There is dusky pigmentation which is noted on feet, hands and abdomen. There will be generalized enlargement of glands. Blood shows leukopenia and Leishman-Donovan bodies. Specific complement fixation test will become positive.

Pyelitis

It is more commonly knows as pyelonephritis. There may be high fever and acute severe rigor. Several bouts of fever may come in a day. There will be frequency, urgency and burning micturation. Pain may be radiated to lumbar region. Kidney area becomes tender. Urine will show pus cells, RBC's and albumin. *E. coli* is the causative factor.

Influenza

It starts suddenly with all signs of fever, cough and running nose like common cold. Fever is often associated with chills or rigor like malaria. Body ache is too much. Several strains

of influenza have been discovered. There is no specific test to diagnose it.

Suppurations

Common suppuration are lung abscess, brain abscess, pyocele and perpural sepsis. Similarly common infection can also give rise to rigors such as tonsillitis, sinusitis bronchiectasis.

Brain Abscess

Brain abscess may extend from face or middle ear. There will be localized signs of irritation of brain. Cerebral malaria can also give similar symptoms but in brain abscess total white cell count and pus cells in CSF will help in diagnosis.

Lung Abscess

Lung abscess is generally a complication of lobar pneumonia. There will be high fever with rigor, pain chest and bronchial breathing. Radiograph will help in reaching a diagnosis.

Fevers with Rashes

There are many fevers with rashes. The rashes of mucous membrane appear early in invasive stage in measles and scarlet fever and chicken pox.

Rashes may be prodromal in certain diseases. Measles is an acute specific infection. It is characterized by Koplik's spots in the mouth. Rash over chest and face generally appears on 4th day. There may be fever, severe common cold, conjunctivitis and photophobia.

Buccal mucosa shows generalized congestion. Koplik's spots start to develop on 2nd day. These are like table salts crystals. Typical rash of measles consists of fine, discrete, brick red macules or maculo papular rash. Rash generally starts from behind the ear, then to face and chest etc. The initial papules coalesce to form blotches and later on whole body may show generalized erythema.

First batch of rashes disappear and next batch starts appearing. Respiratory infection and eye symptoms persist throughout mucous membrane of the body and also may have secondary infections. Laryngitis, pneumonia, bronchopneumonia all may follow. Otitis media is commonly noted. Leukocyte count will be low.

German Measles

German measles is an acute specific fever characterized by suboccipital and posterior cervical glandular enlargement. Macular rash starts on first day. Fever is moderate, with malaise. Rash consists of small, pink, discrete macule. Occasionally patient may complain of joint pain. There is typical enlargement of lymph gland and mild coryza.

Chicken Pox

Chicken pox is mild but is highly infectious. Rash shows centripetal distribution. Fever is mild and develops along with rash. Disease may start with exanthem on buccal mucosa and soft palate. Rashes appear as crops on face, scalp and trunk. Extremities are less involved. Rash is macular or maculopapular which within a few hours changes into vesicular. One can see all the stages at one

point of time. Rash goes on appearing for 3 to 5 days and then goes to crust formation. Most of the scrubs fall in 2 weeks. Generally it does not leave permanent scars. Mortality is lower but eyes require special care.

Scarlet Fever

Scarlet fever is not common in our country. It is caused by hemolytic streptococcus. Throat is generally involved. Throat becomes red and there is moderate edema of pharynx. Typical strawberry tongue is seen. Tonsils are enlarged and show patchy exudate. Cervical group of glands become tender. After 24 hours rash tends to spreads all over. Rash is smooth with minute punctate spot on axilla, groin and dorsum of feet. Rash disappears in 2 to 5 days.

Typhoid

Typhoid is caused by *Salmonella Typhi*. The infection gets localized to small intestine. To start person may have headache, general bodyache and malaise. Fever rises in step ladder fashion. Temperature increases every day especially in the evening. In morning fever comes down but never touches normal. It happens for 7 to 10 days. During the second stage patient usually looks very ill. Tongue is coated, with clean tip. There will be relative bradycardia. In the third week patient starts looking better. During acute stage, the organisms would be positive in first week. Urine, stool culture becomes positive by second week. Widal test is positive during second week.

Fever with Membrane Formation in Throat

Diphtheria

It is an acute infection caused by *cornyebacterium diphtheria*. It is a disease of childhood. After a short incubation period of 4 days disease starts with malaise, loss of appetite, sore throat and fever. Fever may be very high. On examination one finds uniform grayish or yellowish white membrane covering tonsil. It may extend to pharynx and soft palate. Membrane is adherent to underlying mucosa. Cervical glands are enlarged. In severe cases membrane may show hemorrhagic areas and there may be bleeding. Neurological complications include palatal paralysis, ocular paralysis and peripheral neuritis within 6-8 weeks but all three are reversible. Throat swab will confirm the diagnosis.

Thrush

It is an infection of oral cavity due to over growth of fungus *Candida Albicans*. Creamish white curd like patches are seen in mouth. On scrapping a raw bleeding surface is left. Lesions may be found over buccal mucosa, tongue or fauces.

Infectious Mononucleosis

The symptoms include: fever, sore throat, malaise and glandular enlargement. Facues may show exudates. Spleen is enlarged. Hepatitis may be seen. Liver may be enlarged. In severe cases myocarditis, neuritis and encephalitis may develop.

Agranulocytosis

In this condition granular white blood cells disappear from circulation. It develops due to idiosyncrasy towards certain drugs like sulphonamides, phenylbutazone and phenothiazines. Person will develop fever, rigor and extreme weakness. Throat shows brownish grey or white exudates and along with ulceration of tonsils and buccal mucosa. Lymph glands are enlarged.

Vincent's angina is an ulcerative infection of mouth and throat. There is an acute inflammation of mouth and throat with enlarged lymph glands, pain and fever. Streptococcal sore throat also produces exudative membrane. Throat becomes sore and extremely painful. Pharynx may show edema and reddening.

Fever with Pain

Sinusitis

In frontal sinusitis pain may be severe. There will be local tenderness of area. Radiograph helps in reaching the diagnosis.

Otitis Media

It is often associated with chronic throat condition in children. There will be severe, acute, agonizing pain of severity. Measles may result in it.

Lobar Pneumonia/Dry Pleurisy

Illness starts with severe pain in chest localized on base, of lung with fever and cough. Fever is continuous and cough is non productive. Breathing becomes painful. Then

sputum becomes rusty. Dry pleurisy is mostly tubercular in origin. It is accompanied with severe pain in chest, fever and cough. Pleural rub can be found. X-ray of chest may prove helpful.

Amoebic Hepatitis

It gives fairly severe abdominal pain in right hypochondrium. Similarly infective hepatitis may also give rise to pain.

Abdominal TB

It develops gradually. Low fever develops in the evening. Abdomen shows tenderness all over. Similarly renal tuberculosis patient may complain of burning and frequency of urine.

Osteomyelitis

Pyogenic or tubercular is responsible for causing high fever and local pain in affected area. There will be high fever, pain and raised WBC count.

Arthritis

Various types of arthritis are associated with fever and local pain. ESR will be raised.

CHAPTER 23

Cheilitis

It is also known as chapped lips. It may develop due to exposure of sun or atopic dermatitis.

Clinical features include:

- Redness
- Cracking
- Crusting
- Mild swelling

Lower lip is often more involved. Lip biting may develop marked crusting, edema and hemorrhage.

GRANULOMATOUS CHEILITIS

It is also known as Miescher's syndrome of unknown etiology. Young females are more involved. Histologically, there is focal non caseating granuloma formation. There will be epitheloid cells with Langhans type of giant cells.

Clinical Features

- Lower lip is diffusely swelled up.
- There may be fever, headache and visual disturbances.
- Some may develop scales, fissures and pustules.
- Swelling is non pitting.

- Swelling may be hard as rubber.
- Regional lymph nodes may be enlarged.
- It may be associated with Melkeroson-Rosenthal syndrome consisting of fissured tongue and facial paralysis.

Differential diagnosis includes angioedema and sarcoidosis. Angioedema appears suddenly. History of recurrence is there. Sarcoidosis will be excluded by absence of any other manifestation and by a negative Kveim test.

It is managed by repeated injection of corticosteroid every few weeks and cheiloplasty.

GLANDULAR CHEILITIS

It is not very common and lower lip becomes enlarged, everted and firm. Use of tobacco and constant exposure of sun may result in it. Emotional disturbance may also result in glandular cheilitis.

There are three types:

1. Simple—these are multiple, pin headed and painless lesions.
2. Superficial suppurative type—there will be painless swelling, crusting, induration and superficial ulceration of lip.
3. Deep suppurative type—there is deep seated infection with abscess and fistula

Clinical Features

- It is more common in adults
- Lower lip is more involved

- Mucosal surface shows numerous dilated salivary duct orifices surrounded by a red macular area. Person notes a mucus secretion at the orifice of duct
- In severe form, lip is considerably and permanently enlarged and develops pain and tenderness
- Volkmann's cheilitis is a severe form and epidermoid carcinoma may be associated
- Vermiliionectomy is the line of treatment.

ANGULAR CHEILITIS

It is also known as angular cheliosis. It may be caused by Candida Albicans, streptococci or staphylococci. Riboflavin folate and iron deficiency may cause it. General protein deficiency can also result in it. Recurrent trauma from dental flossing may also be implicated.

CHAPTER 24

Vitamins and Oral Lesions

Vitamins

These are chemical compounds made of carbon, hydrogen and O_2. Vitamins are soluble in either water or fat. Solubility affects their absorption, storage and excretion.Vitamins do not provide energy but are required for the metabolism of food. Vitamins cannot be synthesized by the body, if any then quantity is not sufficient, hence are to be supplied from outside. Body can make Vitamin D, Vitamin A and niacin if the required precursors are available. Microorganisms of GI tract can synthesize Vitamin K and B_{12} but not to the requirement of body's need.

Provitamins

These are vitamin related compounds that can be converted to active vitamins in body such as tryptophan can be converted to vitamin niacin, carotene to Vitamin A and cholesterol to Vitamin D.

Antivitamins

These are substances known as vitamin antagonists, which block the synthesis or metabolism of vitamins. Drug methotrexate is antagonist of folic acid; antibiotics interfere with vitamin K synthesis.

Avitaminosis

It means without vitamins.

Hypervitaminosis

An excess of specific vitamins accumulated in the body to the level of toxicity. Commonest is Vitamin A and Vitamin D.

Mega dose: It is an amount at least 10 times greater than RDA. In mega doses, vitamins act as drugs.

FAT SOLUBLE VITAMINS

- These are not excreted when consumed in excess; rather these are stored in liver and adipose tissue
- These can be toxic when consumed in large doses over a long time specially Vitamin A and Vitamin D
- Fat soluble vitamin deficiency symptoms are slow to draw
- Body can store these vitamins, so need not to consume daily
- These are not easily destroyed by heat/cooking
- These are to be attached to protein carriers because these are not soluble in water (blood)
- These are absorbed into the lymphatic system with fat
- Deficiency of fat soluble vitamins occurs in mal-absorption syndromes, pancreatic and biliary diseases
- Fat soluble vitamins include Vitamin A, D, E and K.

WATER SOLUBLE VITAMINS

- Water soluble vitamins are filtered through the kidneys and excreted in urine when consumed in excess
- These are generally nontoxic and to be supplied daily
- Body reserve of these vitamins are minimal, hence toxicity does not develop
- Water soluble vitamins include Vitamin C and B complex including thiamine, niacin, riboflavin, pyridoxine, biotin, cynacobalamine and folate.

Each of the above mentioned vitamins has a specific function and deficiency leads to specific deficiency disease. Minimum quantity of each vitamin has been determined to keep the body healthy.

VITAMIN A

Even though vitamin A was not discovered until 1913, cod liver has been in use for centuries. It was chemically synthesized in 1930. Retinol is found only in foods of animal origin. Herbivorous obtain the vitamins from its precursor i.e. carotenoid pigment. Conversion of beta-carotene into retinol in small intestine is only 30 percent. Absorption of both retinal and carotene is facilitated by bile salts. Vitamin E prevents the destruction of Vitamin A in the body.

FUNCTIONS

- It is essential for skeletal growth
- It is anti-infective, protects body from microbes

- It plays role in immunological defence mechanism of body
- It plays a critical role in normal vision.

SOURCES (TABLE 24.1)

- Liver is the richest source.
- Yellow coloured carrots, pumpkin, papaya and mango are rich sources of Vitamin A.
- Carotene is converted into retinol mainly in walls of intestine.

Daily requirement is 750 microgram. 90% of it is stored in liver. 50% of it is lost in storage. 15-20% of it is lost at room temperature for 24 hrs.

DEFICIENCY

- Night blindness occurs due to lack of photosensitive pigment.

Table 24.1: Important sources of vitamin A

Source	*Microgram/ 100gm*
Cod liver oil	10,000
Liver	6000
Butter	720
Egg whole	300
Carrot	325
Mango ripe	375
Milk whole	50
Fish fatty	50

- Gray, triangular elevated area on conjunctiva called Bitot's spot.
- Xeropthalmia: the sebaceous glands and hair follicles of skin and tear glands of eye becomes blocked with horny plug of keratin, so their secretion is diminished. Lack of tears produces xerophthalmia.
- Keratomalacia: It consists of softening of entire thickness of a part or whole of cornea. It has to be treated on emergency. It is the major cause blindness in India.

ORAL MANIFESTATIONS

- Gingivitis
- Mucosal atrophy
- Scaling of lips.

VITAMIN D

It is intimately connected with the metabolism of calcium and phosphorous. It promotes the absorption of calcium and phosphorous. It promotes the absorption of calcium from intestine and helps in mineralization of bones. It was synthesized in 1935. Exposing the skin to the sun enables the ultraviolet rays to react on the skin's surface oil to produce Vitamin D. The result is not achieved unless you "strip off", since an intervening layer of clothing or of glass impedes the production of Vitamin D. Vitamin D is found in small quantities and in a few animal foods. Foods of plant origin do not contain Vitamin D (Table 24.2).

Table 24.2: Important sources of vitamin D

Food	*Microgm/100gm*
Shark liver oil	30-100
Cod liver oil	200-500
Butter	0.5-1.5
Milk whole	0.1
Fish	5-3

DAILY REQUIREMENTS

- Infants and children-10 microgram (400 IU)
- Pregnant women/Lactating mothers-10 microgram (400 IU)
- Adults—25 microgram (100 IU).

DEFICIENCY

- **Rickets:** The infant with rickets often receiving sufficient calories may appear well nourished but will be restless, fretful and pale with flabby muscles. Sweating of head is common. Abdomen distends. Development of child is delayed.
- Radiological features:
  - Enlargement of epiphysis at lower end of radius
  - Beading of costochondral junctions known as ricketary rosary
  - Bossing of parietal and delayed closure of anterior fontanel
  - Knock knee or bow legs
  - Therapeutic dose is 25 to 125 micrograms daily.

- **Osteomalacia:** It is the counter part of rickets in adults. Skeletal pain is often present and persistent. Bone tenderness and muscular weakness are often present. Patient may find it difficult to climb stairs or getting out of chair. A waddling gait is not unusual. Osteoporosis which is atrophy of bone occurs and is due to defective formation of bone matrix. Bones becomes soft.
- Radiological features:
  - Rarefaction of bones
  - Translucent zones known as Looser's zone.

Oral Manifestations

- Dental pain and swelling caused by pulpitis and dental abscess
- Large pulp chambers and elongated pulp horns
- Delayed eruption of teeth
- Abnormal dental calcification
- Minimal caries
- Increased radiolucency of jaws and reduced density of lamina dura.

Vitamin C

Ascorbic acid is a white crystalline, odourless compound readily soluble in water. It is a strong reducing agent. It is comparatively stable in acid medium but is destroyed by action of heat and catalysts such as copper. Smoking destroys Vitamin C and it has been claimed that each cigarette smoked uses up to 25 microgram of Vitamin C which is equivalent of one orange.

Functions

- It performs number of important functions in body.
- It is a part of cementing material which holds the body cells in place.
- It helps the body to build resistance to infection.
- It helps in absorption of calcium and iron.
- Deficiency of it results in delayed healing of wound.

Sources (Figs 24.1 and 24.2)

Animal food is a poor source of Vitamin C. Amla is one of the richest source of Vitamin C. Lemon and oranges are good source of it. Guava and drumsticks have high ascorbic acid content. Leafy vegetables such as cabbage contain lesser quantities. Sprouting converts part of carbohydrate and pulses into Vitamin C (Table 24.3).

Fig. 24.1: Source of vitamin C

Fig. 24.2: Sources of vitamin C

Table 24.3: Sources of vitamin c mg/100gm

Food	*Vitamin C*
Amla	600
Guava	210-300
Lime	60
Orange	30
Tomato	27
Spinach	28
Capsicum	180
Cabbage	124
Cholai	93
Potatoes	17
Onion	11

Recommended daily requirement is 50 mg.

Fig. 24.3: Amla-the richest source of vitamin C

Deficiency

- The general deficiency of Vitamin C is petechial or massive haemorrhages into skin, muscles and joints
- Resorption of bone leads to osteoporosity
- There may be petechiae and echymoses in oral mucous membrane
- Hyperaemia, edema and enlargement of gingiva are seen
- Spontaneous bleeding of gingiva is seen
- Loosening of teeth is noted
- Delayed wound healing.

Thiamine (Vitamin B1)

It was the first of the Vitamin B complex discovered. It is destroyed by refining, exposure to alkalis such as baking

powder. Up to 50% is lost in cooking water and 15% during baking. Toasting of bread destroys further 15% for thick slice or up to 30% for thin slice. Recommended daily intake is 0.4 mg/1000 Kcal or about 1.2 mg in adult man. Limited amount is synthesized by microorganisms in GIT. Normally, about 25-35 mg is stored in the body.

SOURCES (TABLE 24.4)

Important sources are dried yeast, unmilled cereals, pulses, seeds and groundnuts (Fig. 24.4).

DEFICIENCY

Wet Beri Beri

Edema is the most notable feature. Palpitation is marked and there may be pain in the legs after walking due to accumulation of lactic acid. Neck vein becomes prominent.

Fig. 24.4: Sprouted bengal gram

Thiamine content mg/100 gm:

Table 24.4: Important sources of vitamin B_1

Food	*Mg%*
Wheat whole	0.45
Rice home pounded	0.21
Rice milled	0.06
Almonds	0.24
Milk (cow)	0.05
Egg (hen)	0.10
Mutton	0.18
Liver sheep	0.36

Dry Beri Beri

Essential feature is a polyneuropathy. Muscles become weak and wasted and walking becomes difficult. Chronic patients may develop edema.

Infantile Beri Beri

It starts from 2nd to 5th month and is rapidly fatal. Infant suddenly becomes cyanosed with dyspnoea and tachycardia and dies within 24-48 hrs.

Clinical and Oral Manifestations

- Muscle weakness
- Mental changes
- Sore mouth and tongue
- Cracked corner
- Anaemia.

RIBOFLAVIN (VITAMIN B_2)

It is found in eggs and green leaves. It is a yellow green fluorescent compound soluble in water. It is stable to boiling in acid and is decomposed by heat and exposure to light.

Liver, meat, milk, egg and growing leafy vegetables are its good source (Table 24.5). Cereals and pulses are not rich in it. Milling of grain removes it. Germination increases its content.

Table 24.5: Important sources of vitamin B_2

Diet Sources	*mg/100 gm*
Liver	1.70
Milk	0.19
Egg	0.40
Meat	0.14
Pulses	0.21
Leafy vegetables	0.15
Cereals	0.10
Milled cereals	0.03

Daily requirements is 1.7 mg in adult

Oral Manifestations

- Changes are seen in tongue and lips
- Tongue becomes red and inflamed, loses its papillae, and may appear cyanotic; due to cyanosis it is known

as **Magenta Glossitis.** Sometimes swelling of tongue may also appear

- Ulcers may appear on tongue and buccal mucosa
- Lips show fissures, cracks, they become painful, maceration and scaling began to appear.

Therapeutic dose is 5 mg three times daily – urine becomes fluorescent green in color.

Pyridoxine (Vitamine B_6)

It was discovered by Gyorgyi in 1936. It existed in three forms i.e. Pyridoxine, Pyridoxal and Pyridoxamine. It plays an important role in metabolism of amino acid. Daily requirements depends upon consumption of total quantity of protein.

Symptoms include:

- Angular chelitis
- Glossitis
- Stomatitis
- Atrophy of papillae on dorsum of tongue.

CYANOCOBALMINE (VITAMIN B_{12})

It is a red crystalline substance containing the metal cobalt, it is necessary for the synthesis of DNA. It is not present in food of vegetable origin. It is only available in food of animal origin. It is synthesized by bacteria in human colon but is not being able to absorb. This is relatively heat stable and little is lost in cooking. Daily requirement is 0.5 to 2 microgram.

B_6 content of food, microgram/100 gram.

Table 24.6: Important sources of vitamin B_{12}

Food	*µgm/100gm*
Liver	120
Mutton	10.5
Fish	23.5
Egg	10.4
Cow milk	0.60
Skimmed milk powder	3.0

Fig. 24.5: Maize

Generally, its deficiency is not seen except in mal-absorption syndrome, post gastrectomy or Crohn's disease. It develops neurological symptoms in 10% of cases of pernicious anaemia. Subacute degeneration of spinal cord may occur. Deficiency of it may lead to sore tongue with or without depapillation.

CHAPTER 25

Oral Manifestations of Bleeding Disorders

Iron Deficiency Anemia

It occurs due to:

1. Inadequate absorption of iron.
2. Excessive loss of iron.

It is a chronic microcytic hypochromic type of anemia and exists most commonly.

It may cause pale skin and generalized weakness. Some may develop splitting and cracking of hair. Palpitation, dizziness and sensitivity to cold may develop.

Oral Manifestations

- Pallor of oral mucosa and gingiva
- Atrophic mucositis
- Loss of tongue papillae
- Glossitis
- Angular chelitis.

Total RBC count is low. Stool should be examined for occult blood. Ferrous sulfate in sufficient quantity helps.

Hemolytic Anemia

It occurs due to increased hemolysis in body and becomes difficult for bone marrow to compensate. Hereditary causes

include thalassemia, hereditary spherocytosis, sickle cell anemia, acquired autoimmune hemolysis and erythroblastosis fetalis.

Certain chemical agents can also damage to RBC's.

Clinical Features

- Pallor
- Weakness and fatigability
- Jaundice
- Dyspnea may also occur.

Oral Manifestations

- Pallor of oral mucosa
- Discoloration of teeth in the event of erythroblastosis.

Blood hemoglobin level goes down to 10 mg/dL and average survival of RBC comes down to 12 days instead of 120 days. Reticulocyte count is raised.

Treatment includes blood transfusion in severe cases, splenectomy in cases of spherocytosis and administration of folic acid.

Thalassemia (Fig. 25.1)

It is a group of chronic hemolytic anemia. It produces thin, fragile erythrocytes. In this condition, there is insufficient synthesis of alpha and beta chains of hemoglobin.

Clinical Features

- It is detected in first two years of life
- There may be jaundice, fever with chills

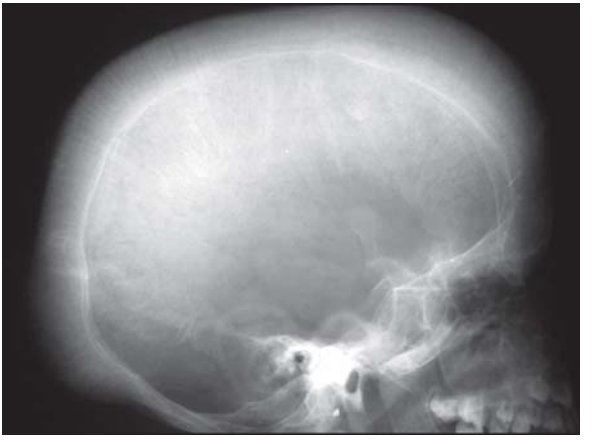


Fig. 25.1: Hair on end appearance

- Marked anemia and malaise
- Spleen is enlarged
- Mongoloid face
- Death often occurs during puberty.

Oral Manifestations

- Spacing of anterior maxillary teeth
- Pallor of oral mucosa
- Bimaxillary protrusion
- Prominent malar bones
- Delayed pneumatization of sinuses
- Discoloration of teeth due to iron.

Radiological Features

- Skull bones will show "hair on end" appearance
- Jaw bones may show "salt and pepper effect".

Pernicious Anemia

It is due to impaired RBC maturation due to lack of $B_{12,}$ it is a chronic, progressive, megaloblastic anemia of adults.

Clinical Features

- It shows large RBC'S
- Hypochlorhydria
- Generalized weakness
- Anorexia
- Weight loss
- Dyspnea
- Skin becomes smooth, dry and yellow
- Neurological symptoms include tingling sensation in hand and feet
- Depression and irritability is seen in a few people.

Oral Manifestations

- Beefy red tongue, areas of patchy ulceration may be noted
- Tongue may be fissured or lobulated. In some cases, it may become bald
- A few patients may develop macular lesions in entire mucosa and may develop burning sensation
- There may be hyperpigmentation of oral mucosa.

Erythroblastosis Fetalis

It is actually a congenital anemia due to Rh incompatibility. It results due to the destruction of fetal RBC.

Oral Manifestations

- Discoloration of deciduous tooth
- Enamel hypoplasia
- Defect in tooth crowns also known as Rh hump.

Polycythemia Vera

It is a chronic disease with proliferation of RBC's. Disease has a gradual onset. Patient complains of headache, dizziness, tinnitus and pruritus. Some feel fatigue, impaired mental ability and visual disturbance. Face becomes reddish purple.

The RBC count goes as high as 8-12 million/mm^3 of blood. Total blood volume rises and blood viscosity increases. Platelet count is increased. Serum uric acid level goes up very high.

Complications may lead to myocardial and cerebral infarctions. Patients may develop thrombophlebitis. Over production of uric acid produces gout. Acute leukemia is the terminal stage of polycythemia.

Leukemia

It is a malignant disease with increased proliferation of WBC's at the cost of other cells. It is more common in younger age groups. Exact cause is not known. But exposure of high doses of radiation therapy, phenyl butazone, chloramphenicol may lead to it.

Some families have more incidence of leukemia. These have been classified into acute and chronic.

Clinical Features

- Generally either children or old people are affected
- Persistent fever of unknown origin
- Spontaneous bleeding from gingiva
- Multiple large ulcers may develop on mucosa
- Candidiasis, histoplasmosis and HSV infections are common in leukemic patients
- Teeth may start loosening, alveolar destruction may take place
- Leukemic cell infiltration may cause atypical dental pain
- There may be prolonged bleeding after extraction.

Agranulocytopenia

There is marked reduction in granular leukocytosis. Toxic effect of certain drugs like chloramphenicol, phenyl butazone may result in it. Long term administration of analgesics, diuretics and anticoagulants may cause it. Ionizing radiation, TB, malaria and typhoid may also induce agranulocytosis.

Clinical Features

- Fever, generally starts with chills and sore throat
- Skin becomes pale
- Jaundice may develop
- Regional lymphadenitis may develop
- Urinary tract infection may develop
- Rectal ulcerations are common
- Severe leukopenia develops
- Neutrophil count comes down to 0-2%.

Oral Manifestations

- Agranulocytic angina develops showing necrotizing ulceration over gingiva, soft palate and cheeks
- Excessive salivation is noted
- Ulcers develop which are covered by yellow/gray membrane
- Ulcers may start bleeding
- Halitosis develops
- Opportunistic fungal infections may develop.

Purpura

It is the extravasations of small amount of blood into mucous membrane.

Clinical Features

- It generally develops in young adults
- Bleeding spots on skin or mucosal surfaces
- Epistaxis, hematuria and hematemesis are common
- Intracranial hemorrhage may produce hemiplegia
- Bleeding in joints may limit its movements
- Spleen is not enlarged.

Oral Manifestations

- Gingival bleeding
- Palatal ecchymosis is seen
- Bleeding in temperomandibular joint result in TMJ pain
- Trismus may develop
- Muscle bleeding may result in limited closing and opening of mouth

  Steroid therapy and repeated transfusions help.

Hemophilia

It is a fatal inherited bleeding disorder. There is reduction in clotting factor. It is a sex linked recessive trait.

It is of two types:

- Hemophilia A, classic hemophilia
- Hemophilia B, christmas type

In hemophilia A, factor VIII is missing and in hemophilia B, factor IX is missing.

Clinical Features

- Serious bleeding may occur from umbilical cord
- Easy bruising and prolonged bleeding
- Spontaneous bleeding in subcutaneous tissue
- Fatal epistaxis may develop rarely
- Gastric bleeding from ulcer may develop
- Spontaneous intracranial hemorrhage may develop.

Oral Manifestations

- Gingival tissue may bleed after extraction
- Recurrent subperiosteal hematoma of jaw may develop
- High caries index
- Severe periodontal disease.

CHAPTER 26

Oral Implications of Medication

Certain oral medications result in side effects producing oral manifestations in general.

- Caries:
  - Prolonged use of sugar containing drugs may result in early childhood caries. Certain cough syrups and lozenges to allure children contain more amount of sugar, which consequently leads to the formation of acidic pH thus making the tooth substance more prone to decalcification.
- Allergy:
  - Oral anti-diabetic drugs may give rise to white patches similar to lichen planus.
- Change in oral flora:
  - Oropharyngeal thrush is an occasional side effect of corticosteroids. Prolonged use of antibiotics may colonize resistant microorganisms.
- Delayed healing:
  - Topical steroids may delay surgical sites or trauma. Prolonged use of acidic medication, mouth washes may cause erosion of teeth.
- Irritation:
  - Aspirin tablets if kept long at the site of cavity leads to white patches and ulceration
  - Pottasium chloride tablets irritate oral mucosa
  - Oil of clove if spilled results in burning of mucosa.

- Tooth discoloration:
  - Ferric oxide gives black stain to the tooth
  - Chlorhexidine mouthwash stains teeth brown
  - Tetracycline stains teeth
  - These were the local effects but certain drugs may cause systemic effects also.
- Exfoliative Stomatitis:
  - Gold, barbiturates and phenylbutazone may cause it
  - These can be fatal in some cases
  - There will be widespread erosion of oral mucosa.
- Erythema multiforme:
  - Penicillin, phenytoin and chlorpropamide may cause swollen lips
  - Oral mucosa bleeds and becomes crusted
  - Severe conjunctivitis may occur. Target shaped lesions may develop.
- Angioedema:
  - Penicillin, aspirin and ACE inhibitor may cause the condition
  - Laryngeal edema may even result in death
  - Anaphylactic shock may be accompanied.
- Bad Breath:
  - Smoking, alcoholism may result in typical breath
  - Dry mouth may also cause foul smell
  - Chloral hydrate and dimethyl sulphoxide may cause bad breath
  - Airway infections are also a common cause.
- Aphthous Ulceration:
  - Folate deficiency and microcytic anemia may cause it.

- Hyper salivation:
  - Clozapine results in excessive production of saliva.
- Fluorosis:
  - Excessive ingestion of fluoride may result in motlling and areas of hypoplasia and pitting.
- Oral pigmentation:
  - Tetracycline gives brown color to the teeth
  - Topical antibiotics and antiseptic mouthwashes color dorsum of tongue black
  - Lead and mercury may discolor the gingiva.
- Pain in salivary glands:
  - Antihypertensives may cause it.
- Change in taste:
  - Metronidazole may result in metallic taste.
- Dry mouth:
  - Excessive use of diuretics and tricyclic anti-depressants may cause dry mouth
  - It may develop caries and poor oral hygiene.

CHAPTER 27

Oral Changes in Old Age

Age brings lot of changes in mouth anatomically as well as physiologically. Certain diseases affecting old age also affects oral cavity.

Oral Mucosa

Histologically certain changes take place in oral mucosa.

- Thinning of epithelium
- Decreased cellular proliferation
- Loss of submucosal elastin and fat.

Clinical Changes

- Dry, thin, smooth mucosal surfaces
- Loss of elasticity
- Loss of stippling
- Trauma to oral mucosa develops due to continuous use of prosthesis
- Delayed wound healing and regeneration of tissue.

Oral Mucosal Diseases

Many adults have oral pigmented conditions:

- Lingual varicosities (Fig. 27.1)
- Melanotic macules.

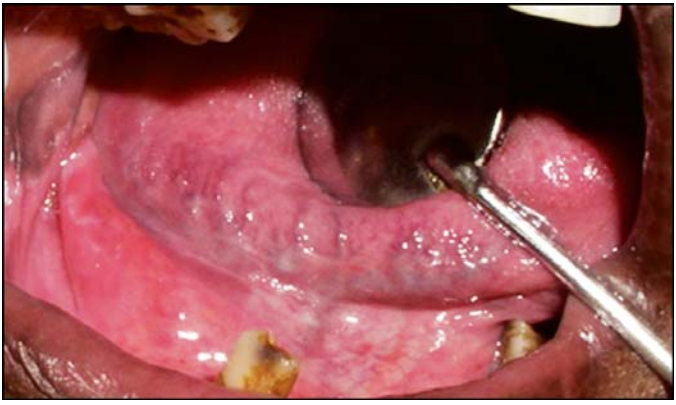


Fig. 27.1: Lingual varicosities

Soft Tissue Conditions

- Fordyces granules
- Fibromas.

Tongue condition includes

- Black hairy tongue
- Geographic tongue
- Atrophy of filliform (Fig. 27.2)
- Fissured, enlarged and coated tongue
- Shiny tongue due to anaemia.

Ulcerative Mucosal Condition

- Denture-related irritation.

Oral Vesicobullous Diseases

- Lichen planus
- Pemphigus vulgaris

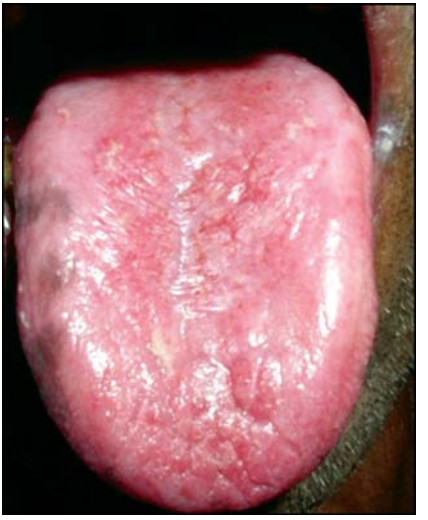


Fig. 27.2: Atrophy of filliform papillae

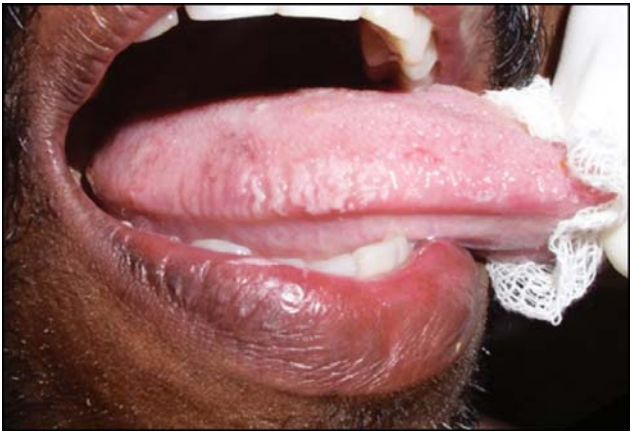


Fig. 27.3: Premalignant condition—leukoplakia

- The most common premalignant oral lesion is leukoplakia (Fig. 27.3)
- Ninty percent of all oral cancers are squamous cell carcinomas.

Dentition

- External tooth changes include yellowish brown colour
- Appearance of small polished facet on cusp tip/ridge or a slight flattening of an incisal edge
- Loss of enamel due to attrition leads to gradual reduction in cuspal heights (Figs 27.4 and 27.5)
- Severe enamel wear will expose underlying dentin producing sclerotic and secondary dentin and this helps as an aid to protect pulp from further injury

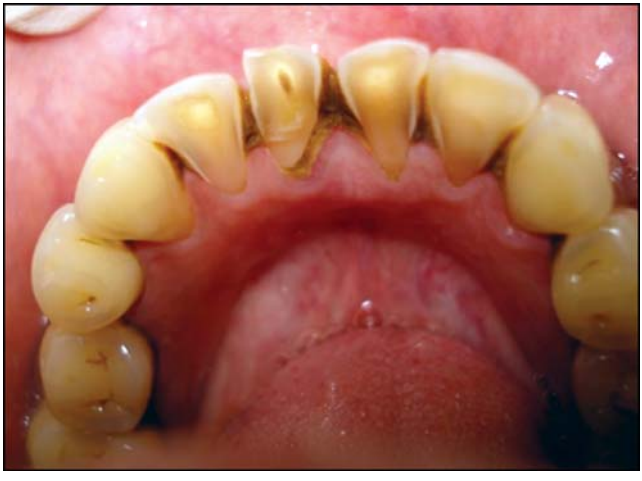


Fig. 27.4: Severe attrition leading to exposed root canals and change in color

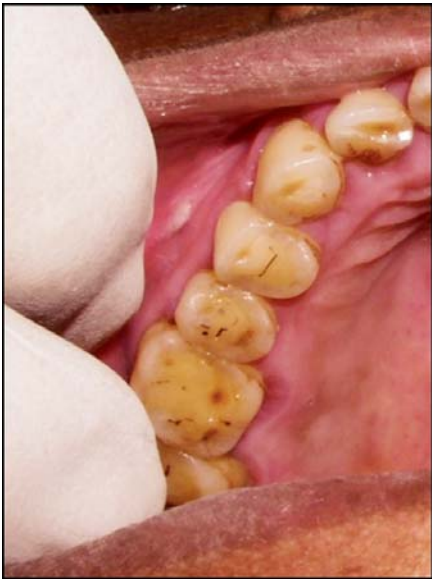


Fig. 27.5: Attrition of posterior teeth

- Dentin undergoes reduction in thermal and osmotic sensitivity
- Pathologic wearing due to abnormal mechanical process or related to a habitual opening of bobby pins which may result in notching of incisal edge of maxillary central incisor
- Cementum thickness and pulp dimension are reduced
- There is formation of dead tracts in dentin
- Formation of pulp stones with the coronal and radicular portion of the teeth

- Occurrence of internal/external resorption due to continuous irritation, trauma, or persistent infections
- Root surfaces caries are increased due to gingival recession
- Tooth remains susceptible to new decay as well as recurrent caries around restoration due to gingival recession
- Dental plaque remains primary source of micro-organisms
- Since cementum is less mineralized than enamel, it remains more susceptible to decay
- Coronal caries are quite prevalent among older persons
- Enamel lesions present clinically as discoloured defects on occlusal surfaces
- As age increases, deposition of secondary and reparative dentin occurs which increases caries resistance and decreases dental sensitivity.

Peridontium (Figs 27.6 and 27.7)

- With age, gingival recession, loss of periodontal attachment and loss of alveolar bone occurs
- There will be increased periodontal pocket depth
- Severe osteoporosis reduces the mineral content of jaw with tooth loss
- Several drugs are associated with gingival overgrowth.
- Gingivitis and periodontitis is much more likely to develop in older patients
- Dental plaque, gingival bleeding and calculus accumulation develop on consumption of a softer diet
- Formation of horizontal and angular bone defects

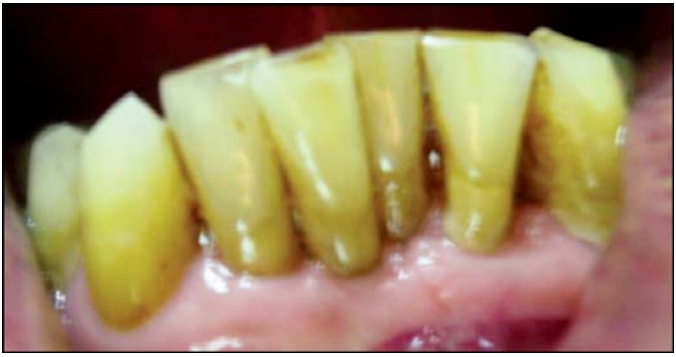


Fig. 27.6: Gingival recession of lower anteriors

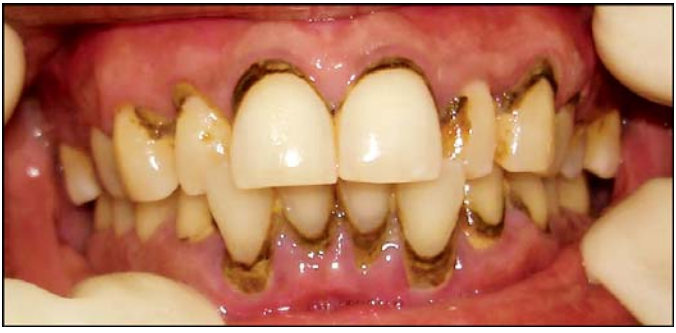


Fig. 27.7: Accumulation of subgingival calculus

- Gingival hyperplasia is associated with the use of cyclosporine, phenytoin and calcium channel blockers. Even controlled diabetes will result in rapid periodontal breakdown due to impaired leukocyte function. Exposed cemental surfaces are more susceptible to root surface caries and loosening the teeth.

Salivary Glands

- Diminished salivary flow may cause dental caries, sensory disturbances, denture irritation and difficulty in chewing and swallowing. But no significant age-related decrease in salivary flow or its constituent in particular is seen
- Histologically there is increase in connective tissue and adipose deposition and decrease in acinar cells
- Alzheimer's disease, diabetes and dehydration can result in salivary gland hypofunction
- Antidepressants, antihypertensive, antiparkinsonian drugs, antihistamines and antipsychotics have been reported to cause xerostomia
- Extraoral manifestations of salivary glands dysfunctions include candidiasis in labial commissures and dry cracked lips.

Taste and Smell

- It plays a great role in quality of life
- The olfactory bulb and peripheral receptors are sensitive to trauma, toxins and medicines
- Many disease of old age have been associated with smell and taste function
- Taste conditions may be due to oral conditions, salivary gland hypofunctions, gingivitis, halitosis, abscess and cancerous lesions
- Parkinson's disease, diabetes, GIT ulcers impair the sense of taste and odors.

Mastication and Swallowing

- Decreased salivary output will adversely affect swallowing in older people
- Neurological and muscular disorders, dementia and depression will result in dysphagia
- Patients with cerebral palsy
- Lip weakness results in drooling from lips. It delays initiation of the oral phase of swallow
- Tongue weakness impairs formation of food bolus. It can result in pneumonia.

Edentulousness

- Taste loss is directly linked to dental caries and periodontal diseases, but it may also be caused by diabetes mellitus and osteoporosis
- Diminished oral motor function may result in masticatory muscle atrophy.

Oral Facial Pain

- Oral, systemic, psychological and behavioural problems are more likely to contribute orofacial pain
- There is a decrease in sensitivity to painful stimulation as person gets old.

CHAPTER 28

Syndromes of Oral Cavity

Stevens-Johnson Syndrome

Erythema multiforme is classified Stevens-Johnson syndrome when the vesicles and bullae involve skin, mouth, eyes and genitals. It may be secondary to a drug reaction. It can slough skin and mucosa. Electrolyte imbalance may develop.

Von-Sallman Syndrome

It is a hereditary benign intraepithelial dyskeratosis. It is a rare autosomal dominant trait. Patient shows oral mucosal thickening with superficial gelatinous looking plaques on bulbar conjunctiva. Cytological scrapings show 'cells within cells'.

Jadassohn-Lewandowsky Syndrome

In it there is congenital gross thickening of finger and toe nails. Along with it leukokeratosis is noted. Nail lesion is noted just after birth with a horny brownish material at nail bed. Oral leukokeratosis affects dorsum of tongue which becomes thickened and grayish white. Histologically, these are similar to white sponge nevus. Frequent oral aphthous ulceration may develop.

Dyskeratosis Congenital (Zinsser-Engman-Cole Syndrome)

It is a rare X-linked disorder characterized leading to atrophic, leukoplakic oral mucosa. Tongue and cheek are adversely affected. Oral lesions start before the age of 10 as vesicles and white necrotic patches with candida, ulcerations and erythroplakic changes.

Greincspan's Syndrome

There is a triad of lichen plannus, diabetes mellitus and hypertension. It may predispose to sqaumous cell carcinoma.

Cushing's Syndrome

In two adrenal hyperactivity is seen. Patient may be hypertensive and hypoglycemic and show moon face. Gingiva, palate and buccal mucosa may be blotchy due to melanin granules.

Peutz-Jeghers Syndrome

There is an oral pigmentation. Multiple, focal, melanotic brown macules are concentrated on lips. Macules may go upto 3-5 cm in size. Anterior tongue may be involved. Histologically, these show basilar melanogenesis without melanocytic proliferation.

Albright's Syndrome

It is like fibrous dysplasia of bone. There is replacement of spongy bone by a peculiar fibrous tissue. Radiographs will show radiopacity and radiolucency, some like compact

bone other like cystic bone. The cause of endocrine manifestation in Albright's syndrome is not known. Transformation to malignancy occurs. Radiotherapy is not advised.

Gardner's Syndrome

There is carcinomatous transformation of adenomatous intestinal polyps. There may be osteomas of jaw and accompanying cysts are indictors of Gardner's syndrome and bowel should be examined.

Nevoid Basal Cell Carcinoma Syndrome

Individuals parent with jaw cysts, facies, calcification, bifid ribs and skin lesions. Multiple pink to brown papules appear on face. In 50% cases pitting of soles and palms is seen.

Cowden's Syndrome

There is hamartomas involvement of many organs with a potential of neoplastic transformation. It is inherited as autosomal dominant character. Multiple cysts over lips and gingivae are seen. Tongue is also fissured. Hemangiomas and neuromas are also seen.

Rutherford Syndrome

There are congenitally enlarged gingivae, delayed tooth eruption and superior corneal opacities are curtain like.

Patient may have mental retardation, aggressive behavior and dentigerous cysts.

Zimmermann-Lebarnd Syndrome

Person develops gingival fibromatosis with defects of ear, nose, bones and terminal phalanges. Joints become hyperexible. Spleen is enlarged.

Goltz-Gorlin Syndrome

Patient will develop gingival and other mucosal papillomatous lesions. Lips and teeth develop defects. Associated features include poikiloderma, syndactly and adactyly.

Romon Syndrome

Patient develops gingival and alveolar enlargement, micro -ophthalmia. Cornea becomes cloudy. Hypopigmentation results with athetosis. Mental retardation is rare.

Lysosomal Storage Syndrome

Development of childhood gingival enlargement, widened alveolar ridges and widely spaced teeth certain visceras are enlarged.

Sturge-Weber Syndrome

Person develops orofacial and meningeal angiomatosis with secondary mental deficiency. There may be seizures, hemiplegia and mild to severe enlargement of gingiva. Hyperplastic vascular gingiva blanch on pressure. Bony hemangiomas and delayed tooth eruption may be noted.

Acanthosis Nigricans Syndrome

There is formation of gingival papillomatosis with perioro-facial mucosal and skin lesions. Gastric adenocarcinoma may develop.

Neck-Tongue Syndrome

It is characterized by unilateral upper nuchal or occipital pain with or without numbness in these areas. There is simultaneous numbness of tongue on the same side.

Burning Mouth Syndrome

Painful, burning sensations localized in the tongue or affecting other areas of oral mucosa. The pain of burning tongue is rated similar to that of toothache. Tongue temperature is decreased. Some evidence suggests the existence of oral somatosensory and special sensory deficits.

Sjögren's Syndrome

It is an autoimmune disorder of exocrine glands which may be associated with neuropathy and lymphoproliferative disorder. Lacrimal and salivary glands are affected. Histological hallmark is focal lymphocytic infiltration of exocrine glands. Epstein-Barr or a type A retrovirus is the causative organism. Xerostomia is a major complaint in most patients with parotid gland enlargement. Enlargement of submandibular gland may be there. There may be symptoms of rheumatoid arthritis. Kidney involvement is noted.

Uremic Syndrome

Early symptom of kidney failure is decreased creatinine clearance. As disease progresses glomerular filtration rate falls and blood urea nitrogen rises. Patient may develop

impaired ability to concentrate urine, nocturia and mild anaemia. Advanced uremia is associated with pericarditis, pericardial effusion and neuropathies.

AIDS

Acute infection occurs 3 to 6 weeks after initial contact with HIV. Nonspecific syndrome occurs during which joints pain, gastrointestinal symptoms and a macular rash predominate. These self-limiting symptoms persist for 2-3 weeks. Eight to twelve weeks after infection antibody to HIV can be detected in serum. After acute infection person enters the symptomatic phase of the disease. The average time from infection to development of disease is 8 to 10 years. During this period T_4 lymphocytes comes down from 800 mm^3 to 50 mm^3.

Oral manifestations in HIV infected patients are:

- Oral candidiasis occurs in 30 to 90% cases
- Persistent ulcers of mouth in AIDS should be biopsied for deep fungal infections
- Hairy leukoplaia has been noted in HIV positive homosexuals
- Recurrent herpes simplex virus progresses to large chronic oral lesions
- Ulcers are surrounded by raised white border. Lesions may coexist with genital and anal lesions
- Painful oral ulcers caused by cytomegalovirus may be seen
- Kaposi's sarcoma is the most common oral neoplasm in AIDS. Early lesions look like hemangiomas, flat or raised discolorations
- Generalized gingivitis and periodontitis.

Chédiak-Higashi Syndrome

It is a congential autosomal recessive defect. Defect is of granule containing cells as granulocytes and melanocytes. Hypopigmentation will be seen on skin and hair. Some may develop neuropathy. Child dies before the age of 10 due to recurrent infection. Lymph nodes spleen, liver and bone marrow are infiltrated with lymphohistiocyte cell. Gingival and periodontal findings are common. Early loss of teeth is noted.

Guillain-Barré Syndrome

It is caused by progressive demyelinating neuropathy. It is a autoimmune following a nonspecific disease. Impaired swallowing or paraesthesias of mouth and face is the early sign. There may be ascending anaesthesia and paralysis of legs and trunk. In severe case respiration is compromised.

Sweat Retention Syndrome

There is an extravasation of sweat in the tissue with subsequent inflammation, keratin plug formation in sweat glands.

Nursing Bottle Syndrome

This occurs in babies on bottle feeding containing more of sugar. It results in multiple numbers of caries in many teeth mostly the lower one.

Miescher's Syndrome

There develops diffuse swelling of lip. On vermillion border scaling, fissuring, vesicle or pustule formation may take place.

Cracked Tooth Syndrome

Development of a crack in a restored or an unrestored tooth due to excessive occlusal forces is one of the main cause. There is sharp pain on biting. Pain is similar to that of trigeminal neuralgia. Radiographs are unable to show cracked tooth.

Meckel's Syndrome

Midline clefting of tongue is a feature of this syndrome. Tongue is also thrown into multiple nodular or papillomatous projections.

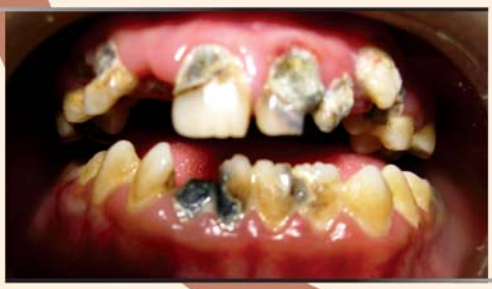
Maffucci's Syndrome

Development of multiple hemangiomas of skin and oral mucosa. Multiple chondromas of jaw bone is seen. They represent calcification of organized blood clots in dilated vascular spaces.

SECTION

2

Causes of Dental Signs and Symptoms



Anatomic Periapical Radiolucencies (Fig. 1)

- Maxillary sinus
- Nasolacrimal duct
- Incisive foramina
- Greater palatine foramen
- Dental papillae
- Naris
- Mental foramen
- Submandibular fossa
- Mandibular canal.

Anatomic Radiopacities of Mandible

- External oblique ridge
- Internal oblique ridge
- Mental ridge
- Mylohyoid ridge

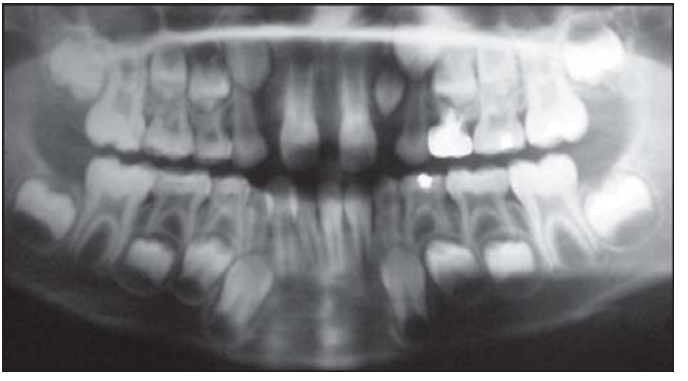


Fig. 1: OPG showing radiopacities and radiolucencies

- Genial tubercles
- Condylar process.

Anatomic Radiopacities of Maxilla

- Anterior nasal spine
- Posterior nasal spine
- Canine eminence
- Coronoid process
- Maxillary tuberosity
- Walls and floor of maxillary sinus.

Bad Taste (Fig. 2)

- Poor oral hygiene
- Dental caries
- Periodontal disease

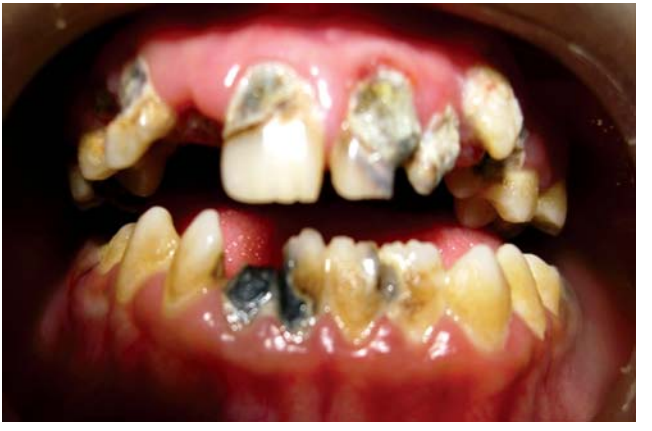


Fig. 2: Poor oral hygiene, caries can cause bad taste

- ANUG
- Age changes
- Drugs
- Diabetes
- Uremia
- Intraoral malignancies.

Bilateral Parotid and Submandibular Swelling

- Sjögrens syndrome
- Mikulicz's disease
- Parotitis
- Metabolic sialdenosis
- Tuberculosis.

Tumors of the Jaw—Benign (Fig. 3)

- Giant cell tumor
- Giant cell granuloma

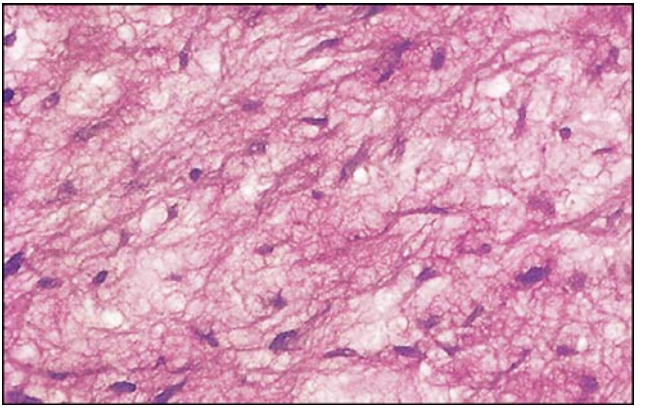


Fig. 3: Benign tumor of the jaw-myxoma

- Chondroma
- Myxoma
- Osteoblastoma
- Osteoma
- Fibroma
- Hemangioma.

Benign Tumors of Oral Soft Tissues

Epithelial Tumors

- Verrucus vulgaris
- Condyloma acuminatum
- Papilloma
- Keratoacanthoma.

Mesenchymal Tumors (Fig. 4)

- Periapical fibroma
- Irritation fibroma

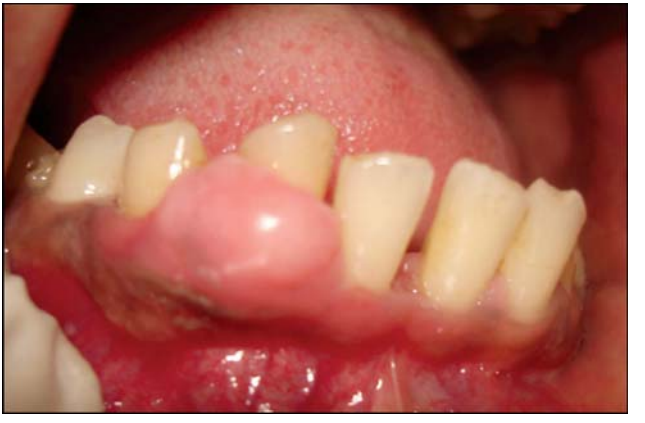


Fig. 4: Irritation fibroma-benign tumour of soft tissue

- Epulis fissuratum
- Myxoma
- Peripheral giant cell granuloma
- Traumatic granuloma
- Hemangioma
- Lymphangioma
- Pyogenic granuloma
- Cystic hygroma
- Rhabdomyoma
- Neuroma
- Neurofibroma
- Lipoma
- Hamartoma.

Bleeding Gums (Fig. 5)

- Local conditions
- Gingivitis

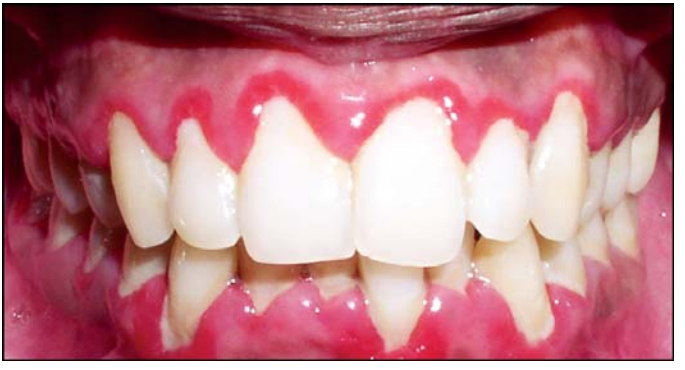


Fig. 5: Gingivitis and gingival recession

- Periodontitis
- Injury
- Gingival recession
- Lesions such as:
  1. Epulis
  2. Stomatitis
  3. Papilloma.

Systemic Diseases

- Scurvy
- Acute leukemia
- Bleeding diasthesis
- Thrombocytopenia
- Platelet dysfunction.

Halitosis

Due to local factors of pathologic condition:

- Poor oral hygiene
- Extensive caries
- Gingivitis
- Periodontitis
- Open contact allowing food impaction
- Vincent's disease
- Hairy or coated tongue
- Infectious malignant neoplasm's of the oral and pharyngeal cavities
- Healing extraction wounds
- Necrotic tissue from ulceration
- Dentigerous cyst

- Increase in gram negative filamentous organisms
- Increase in plaque pH to 7.2, formation of indoles and amines in the oral cavity.

Due to local factors of non-pathologic origin:
- Stagnation of saliva associated with food debris
- Dentures
- Excessive smoking
- Diet and dietary habits
- Endodontic patients.

Due to systemic factors of pathologic origin:
- Diabetes mellitus
- Liver failure
- Acute rheumatic fever
- Lung abscess, tuberculosis, bronchiectasis
- Uremia, kidney failure
- Blood dyscrasias
- Syphilis
- Scurvy
- Non-lipid Reticuloendotheliosis disorder e.g. eosinophilic granuloma
- Letterer-Siwe disease and Hand-Schuller-Christian disease.
- Inborn errors of metabolism e.g. Trimethylaminuria
- Cancer
- Acidosis.

Due to systemic factors of non-pathologic origin:
- Metabolites from ingested food that are excreted through the lungs
- A vegetarian has fewer tendencies to produce halitosis than an excessive meat eater

- Garlic, onion, leeks, alcohol etc impart odors to the breath by being absorbed into the circulatory system.
- Excessive alcohol drinking leads to alteration of the microbial flora and causes proliferation of odor fermenting organisms
- Young children aged 2 to 5 years may have a sweet fetid mouth odor due to their tonsil crypts lodging food and bacteria
- Vincent's disease and its odor are most frequently seen in teenagers
- Some patients with dysmenorrhea suffer from a mousy odor resembling decayed clotted blood
- From time to time, one may experience hunger odor with hunger sensations. It might be due to putrefaction of pancreatic juices in the stomach and such an odor cannot be eliminated even with tooth brushing.

Brown Lesions on Lips

- Nevus
- Melanoma
- Labial melanotic macule
- Albright's syndrome
- Kaposi's sarcoma
- Hemochromatosis
- Addison's disease.

Burning Sensations in Tongue (Fig. 6)

- Idiopathic burning tongue
- Geographic tongue
- Xerostomia

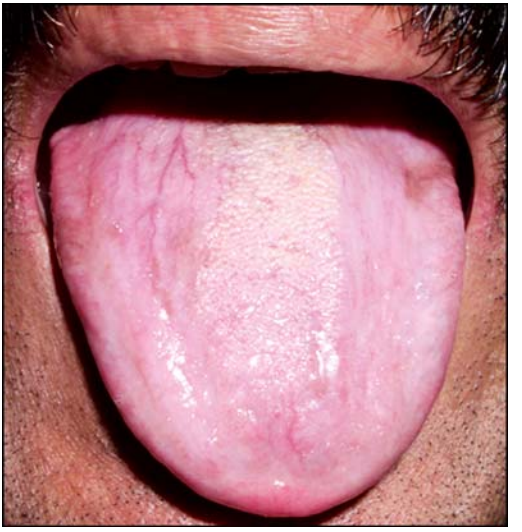


Fig. 6: Idiopathic burning tongue

- Anemia
- Achlorhydria
- Vitamin B deficiency
- Multiple sclerosis
- Psychosis
- Neurosis.

Calculus Formation

- Non-calcareous calculi
- Idiopathic
- Gout
- Low urinary pH
- Cystinuria

- Increased phosphate dissociation
- Malignant tumors
- Hyperuricosuria
  - Increased saturation of monosodium urate
  - Urate-induced crystallization
  - Hypercalciuria
  - Increased saturation of calcium oxalate and calcium phosphate
  - Spontaneous precipitation of calcium salts
  - Hypocitraturia- renal tubular acidosis
  - Increased saturation of calcium salts.

Xerostomia

- Local inflammation
- Fibrosis of salivary glands
- Drugs
- Atropine
- Other cholinergic drugs
- Antihistamines
- Tranquilizers
- Autoimmune diseases
- Postradiation changes
- Chemotherapy
- Psychosis.

Soft Tissue Growth of Oral Cavity

- Eruption cyst
- Gingival cyst
- Mucocele
- Epidermoid cyst

- Ranula
- Lymphangioma
- Cystic hygroma.

Cutaneous Fistulas and Sinuses

- Chronic osteomyelitis
- Extraoral draining cyst
- Orocutaneous fistula
- Pustules.

Cysts of Soft Tissues (Fig. 7)

- Mucocele
- Ranula
- Mucous cyst
- Epidermoid cyst

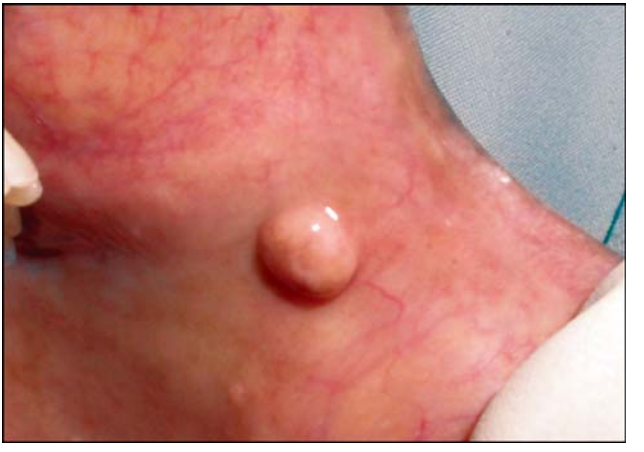


Fig. 7: Mucocele of lower lip

- Gingival cyst
- Branchial cyst
- Thyroglossal cyst
- Nasoalveolar cyst.

Delayed Tooth Eruption

- Delayed growth
- Space loss
- Supernumerary tooth (Fig. 8)
- Ankylosis
- Cysts and Tumor
- Odontomas
- Sclerosed bone
- Anodontia
- Cleidocranial dyostosis
- Hypothyroidism.

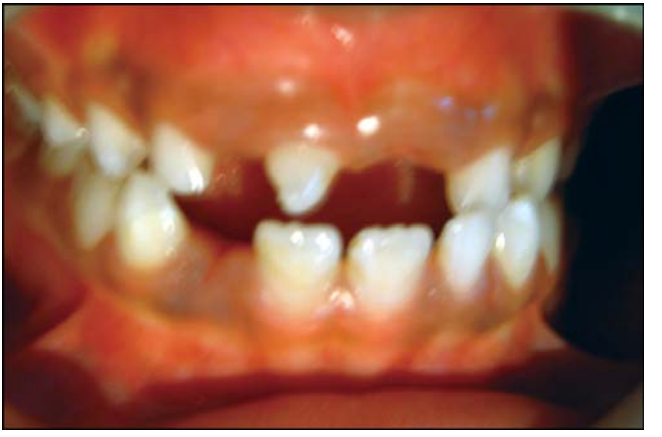


Fig. 8: Supernumerary tooth

Developmental Disturbances Affecting Skull, Jaw

- Cleidocranial dysostosis
- Cleidocranial dysplasia
- Craniofacial dysostosis
- Mandibulofacial dysostosis
- Micrognathia.

Developmental Disturbances Affecting Teeth

- Ectodermal dysplasia
- Anodontia
- Hypodontia (Fig. 9)
- Microdontia
- Accessory teeth
- Supernumerary teeth
- Natal and neonatal teeth.

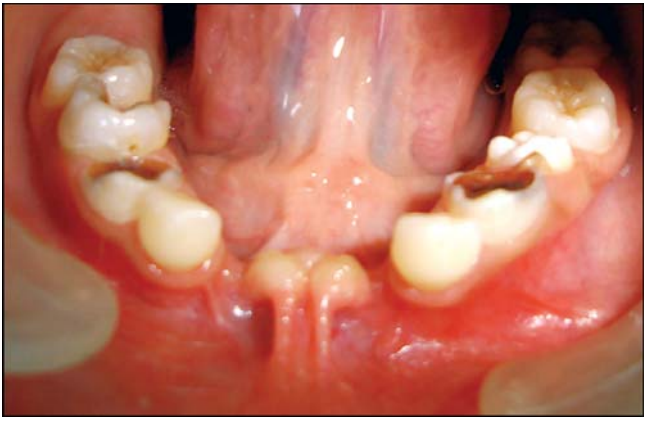


Fig. 9: Congenitally missing anterior teeth-hypodontia

Diffuse Facial Swelling (Fig. 10)

- Ludwigs angina
- Cellulitis
- Emphysema
- Cherubism
- Angioneurotic edema
- Facial hemiatrophy
- Cushing's syndrome
- Fibrous dysplasia.

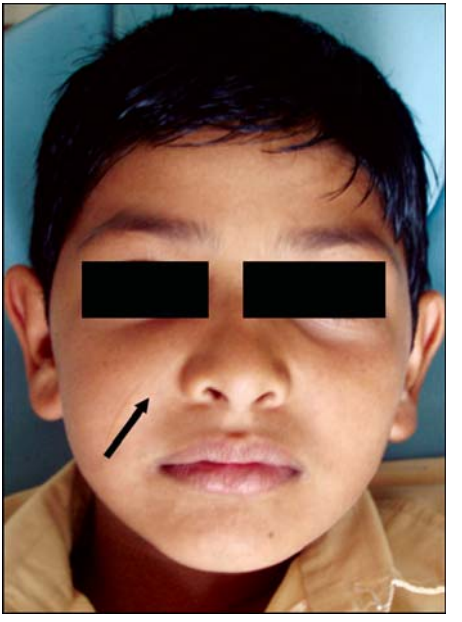


Fig. 10: Diffuse facial swelling involving buccal and infra orbital space

Diseases of Maxillary Sinus

- Cysts of maxillary sinus
- Maxillary sinusitis
- Antral polyp
- Extrinsic bony cyst
- Foreign bodies
- Oroantral fistula.

Taste Disorder

Distorted taste:

- Drugs
- Post burn.

Metabolic disorder:

- Hypothyroidism
- Adrenal cortical insufficiency
- Hypovitaminosis
- Psychosis
- Dyspepsia

Loss or reduction of taste:

- Damage to chorda tympanii
- Damage to facial nerve (taste of 2/3 anterior tongue is affected)
- Lesions of lingual nerve
- Lesions of glossopharyngeal nerve
- Glossitis

Local conditions:

- Caries
- Epithelioma
- Stomatitis
- Gingivitis.

Disturbances during Formation of Hard Dental Tissues (Fig. 11)

- Amelogenesis imperfecta
- Dentinogenesis imperfecta
- Enamel hypoplasia
- Dentinal dysplasia
- Shell teeth
- Enamel pearls
- Cemental hypoplasia
- Pigmentation of enamel
- Dens evaginatus
- Dens invaginatus
- Taurodontism
- Dilacerations
- Talons cusp
- Germination
- Fusion (Fig. 12).

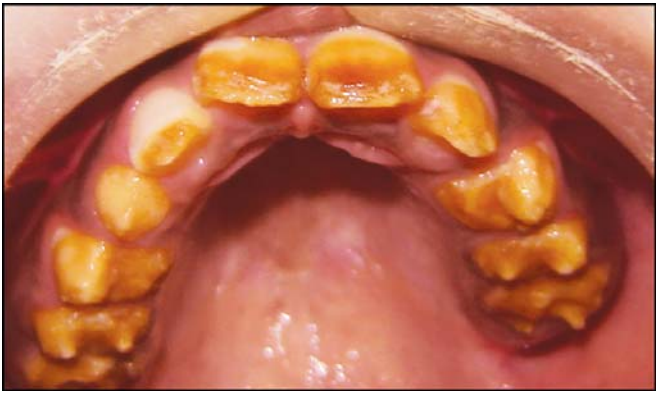


Fig. 11: Amelogenesis imperfecta

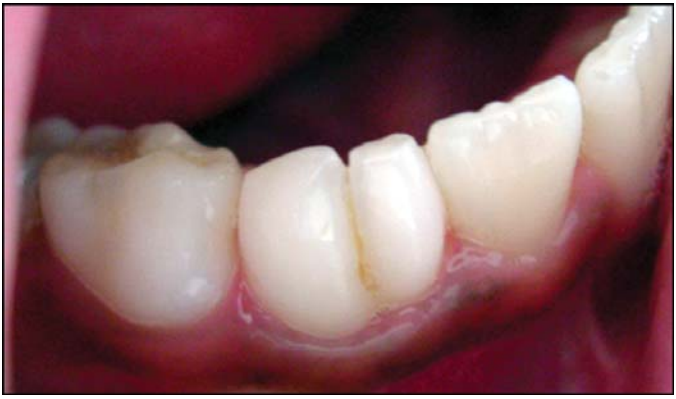


Fig. 12: Fusion of lateral incisor and canine

Drugs causing Lymphadenopathy

- Penicillin
- Streptomycin
- Heparin
- INH
- Viomycin
- Hydantoin
- Phenylbutazone
- Phenacetin
- Iron dextran
- Follicular hormone
- Antitoxins
- Sulphonamides.

Dry Mouth

- Local inflammation
- Post radiation changes

- Infection and fibrosis of salivary gland
- Autoimmune disease
- Chemotherapy
- Psychosis
- Drugs.

Yellow Conditions of Oral Mucosa

- Fordyce's granules (Figs 13A and B)
- Lipoma
- Carotenemia
- Lymphoepithelial cyst
- Abscess
- Superficial nodules of tonsillar tissue.

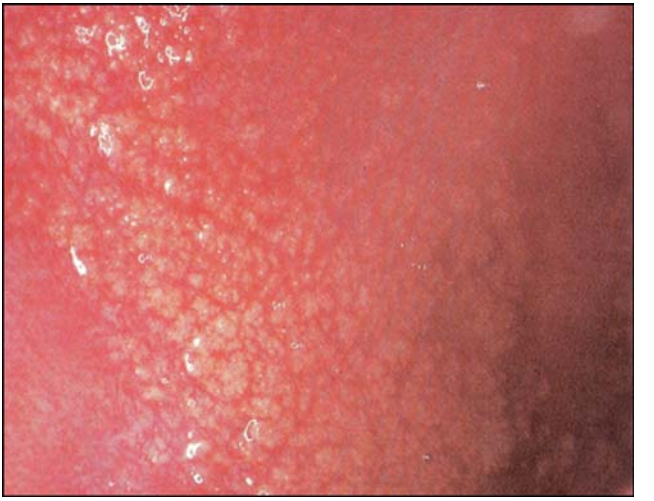


Fig. 13A

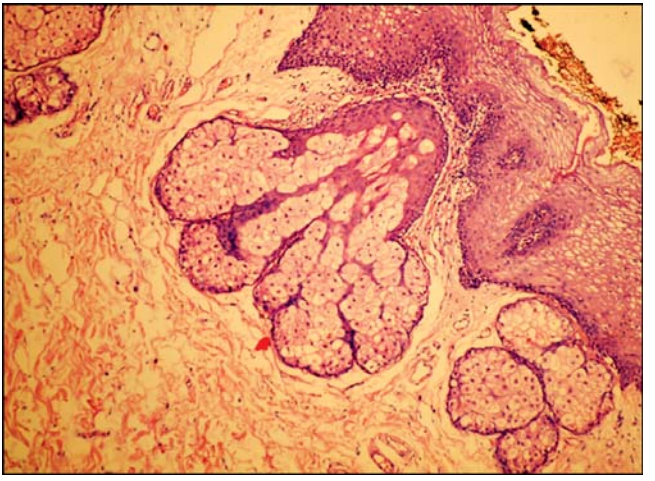


Fig. 13B

Figs 13A and B: Fordyce's spots ectopic sebaceous granules on buccal mucosa

Elevated Lesions on Lip

- Fibroma
- Lipoma
- Mucocele
- Hemangioma
- Angioedema
- Salivary gland tumor
- Plasma cell cheilitis
- Cheilitis granuloma.

Exophytic Anatomic Structures

- Buccal fat pads
- Foliate papillae (Fig. 14)
- Accessory tonsillar tissue

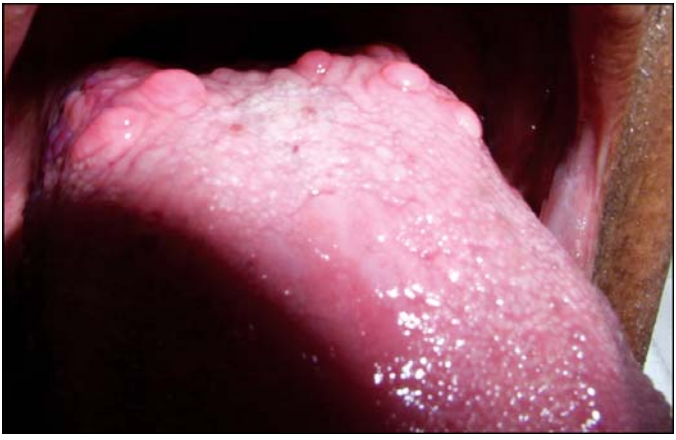


Fig. 14: Exophytic foliate papillae

- Genial tubercles
- Palatal rugae
- Lingual tonsillar tissue
- Palatine tonsils.

Salivary Gland Pain

Drugs

- Vinca alkaloids
- Cytotoxic drugs
- Antihypertensive drugs.

Common causes

- Mumps
- Stones.

Other causes
- Malignant tumors
- Acute sialadenitis
- Recurrent sialadenitis.

Facial Nerve Palsy

Infections
- Cholesteatoma
- Otitis media.

Trauma
- Idiopathic injury
- Birth trauma
- Temporal bone fracture.

Tumors

Tumors of facial nerve:
- Neurilemmoma
- Schawanoma

Idiopathic
- Bell's palsy

Tumors of cerebellopontine angle
- Meningioma
- Acoustic neurinoma
- Neurofibroma
- Tumor of parotid.

Projected Radiopacities of Tooth

- Foreign bodies
- Calcified lymph nodes
- Rhinoliths

- Pulpal calcification
- Ectopic calcifications
- Pathologic soft tissue masses
- Tonsilolith
- Sialoliths.

False Periapical Radiopacities

- Supernumerary teeth (Fig. 15)
- Impacted teeth (Fig. 16)
- Tori
- Compound odontoma
- Retained root tips

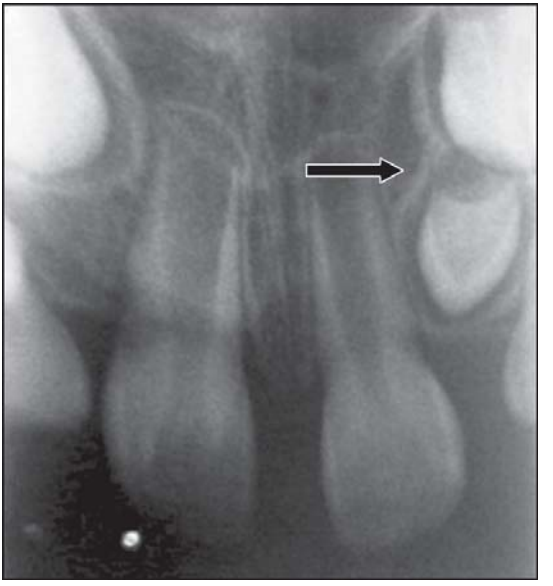


Fig. 15: Supernumerary teeth

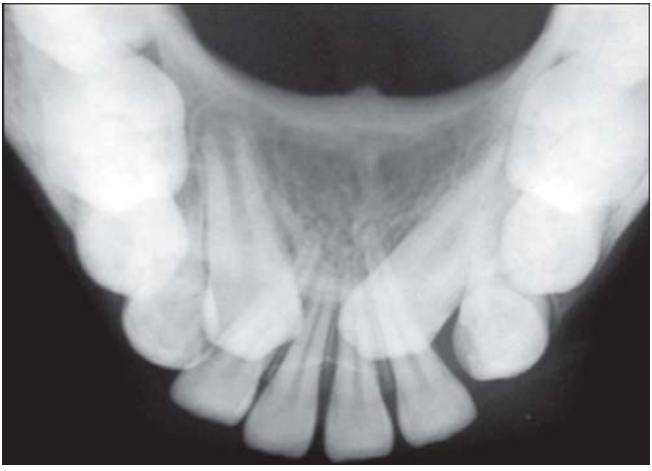


Fig. 16: Impacted teeth

- Phleboliths
- Foreign bodies
- Sialoliths.

Nonhemorrhagic Soft Tissue Growth of Oral Cavity

- Irritation fibroma (Fig. 17)
- Peripheral fibroma
- Myxoma
- Torus
- Neurofibroma
- Fibromatosis
- Tumors of salivary gland
- Lipoma.

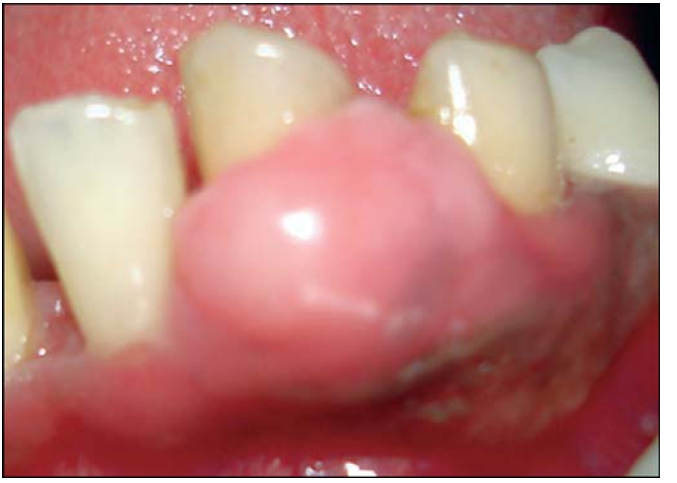


Fig. 17: Irritation fibroma

Flushing of Face

Alcohol

- Chlorpropamide
- Menopause
- Allergic reactions
- Drugs
- Nitrites
- Nicotinic acid
- Calcium channel blockers
- TRH injection
- Carcinoid syndrome
- Morphine injection
- Parkinsonism
- Neoplasia

- Pancreatic tumors
- Medullary cancer thyroid
- Psychological
- Neurological
- Anxiety.

General Brownish, Bluish or Black Condition

- Cyanosis
- Wharton's disease
- Addison's disease
- Hemochromatosis
- Pellagra
- Sprue
- Chloroquine therapy
- Dermatomyositis.

Generalized Radiopacities

- Diffuse cementosis
- Paget's disease
- Osteopetrosis
- Polyostotic fibrous dysplasia.

Generalized Rarefaction of Jaw Bones

- Hyperparathyroidism
  - Primary
  - Secondary
  - Osteoporosis
  - Drug induced
  - Malnutritional state
  - Postmenopausal

- Thyrotoxic
- Cushing's syndrome
- Osteomalacia
- Multiple myeloma
- Paget's disease
- Leukemia.

Generalized Red Conditions and Multiple Ulcerations

- Recurrent aphthous stomatitis
- Lichen planus
- Epidermolysis bullae (Fig. 18)
- Erythema multiforme
- Psoriasis
- Desquamative gingivitis
- Pemphigus

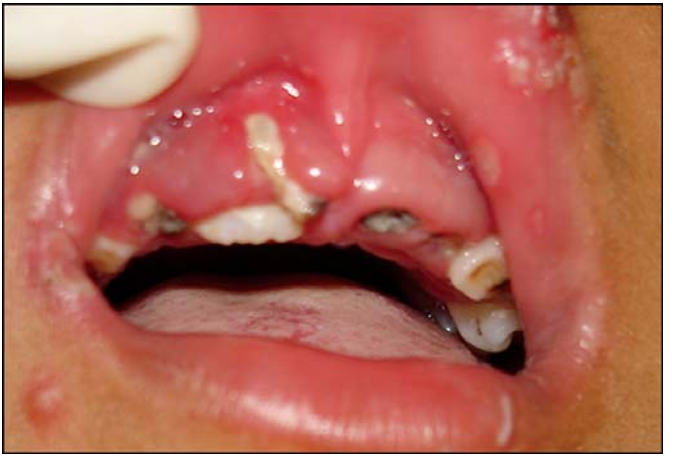


Fig. 18: Multiple ulceration of epidermolysis bullosa

- Diabetic ulcerations
- Measles
- Leukemia
- Ulcerative colitis.

Gray/Black Oral Pigmentation

- Graphite tattoo
- Hairy tongue (Fig. 19)
- Amalgam tattoo
- Heavy metal consumption

Headache of Dental Origin

- Trigeminal neuralgia due to dental infection
- TM joint disorder
- Bruxism
- Myofunctional pain dysfunction syndrome.

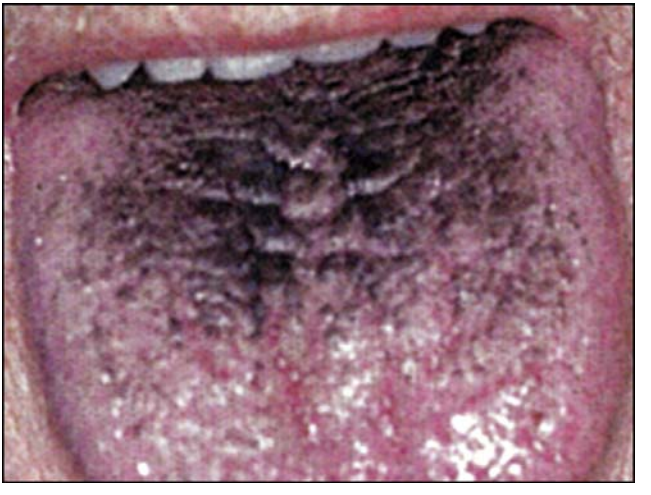


Fig. 19: Black hairy tongue

Headache due to Infections

Bacterial Origin

- Meningitis
- Pneumonia
- Typhoid
- Encephalitis

Protozoal Origin

- Malaria

Viral Origin

- Post-herpetic
- Poliomyelitis
- Rabies
- Dengue

Intraoral Bleeding

- Gingivitis
- Traumatic incident
- Allergies
- Inflammatory hyperplasia
- Hemangioma

Persistent Oral Ulcer

- Constipation
- Stress
- Vitamin B deficiency
- Major aphthous ulcer
- Squamous cell carcinoma

- Gumma
- Keratoacanthoma
- Ulcer secondary to systemic disease
- Traumatized tumor
- Secondary to underlying habit

Pits of Oral Cavity

- Congenital lip pit (Fig. 20)
- Fovea palantine
- Postsurgical pit
- Commissural lip pit.

Intraoral Brownish, Bluish or Black Conditions

- Amalgam tattoo
- Mucocele
- Melanoplakia
- Hemangioma

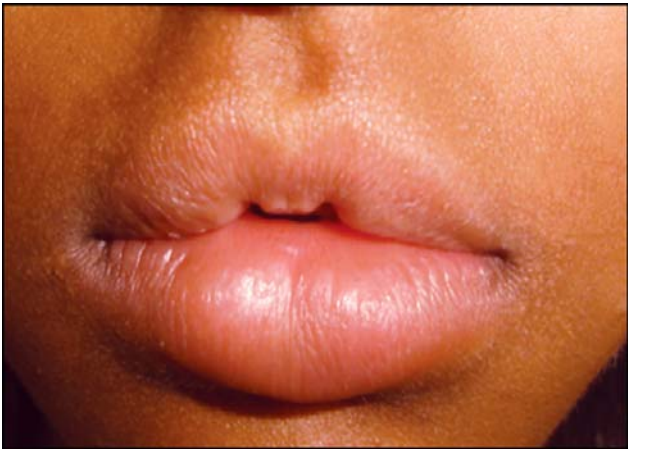


Fig. 20: Congenital lip pits

- Ranula
- Black hairy tongue
- Lupus erythematous
- Nevus
- Melanoma.

Labial/Buccal Mucosa and Vestibular Lesions

- Recurrent aphthous ulcer
- Traumatic ulcers
- Cheek bites
- Epulis fissuratum
- Amalgam tattoo
- Fordyce's granules
- Lichen planus
- Leukoderma
- Leukoplakia (Fig. 21)

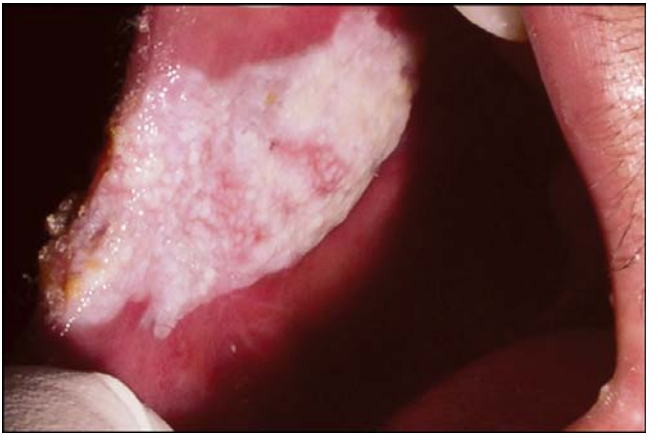


Fig. 21: Leukoplakia of buccal mucosa

- Hemangioma
- Salivary gland neoplasm.

Intraoral Sinuses and Fistulas

- Draining alveolar abscess
- Oroantral fistula
- Draining cyst
- Draining mucocele
- Patent nasopalantine duct.

Intraoral Soft Tissue Swelling

- Fibromatosis gingiva
- Hyperplastic gingiva
- Leukemia
- Hyperplastic gingivitis (Fig. 22)

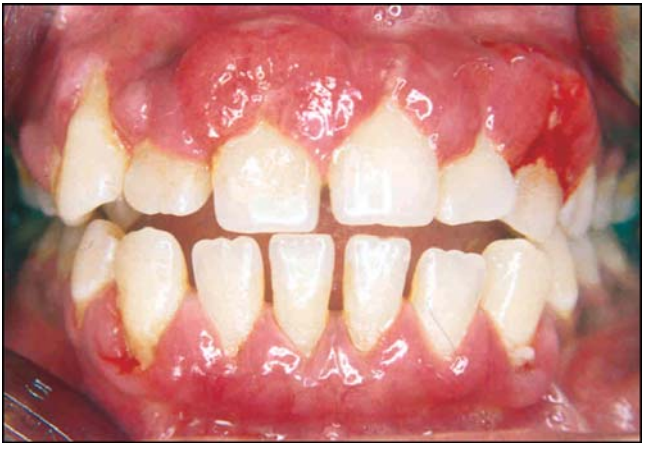


Fig. 22: Hyperplastic gingiva

- Localized gingival tumefactions pyogenic granuloma
- Peripheral fibroma
- Giant cell fibroma
- Gingival cyst
- Eruption cyst
- Congenital epulis
- Squamous cell carcinoma.

Cystic Lesions of Jaw

- Dentigerous cyst
- Radicular cyst (Fig. 23)
- Nasopalantine cyst
- Keratocyst

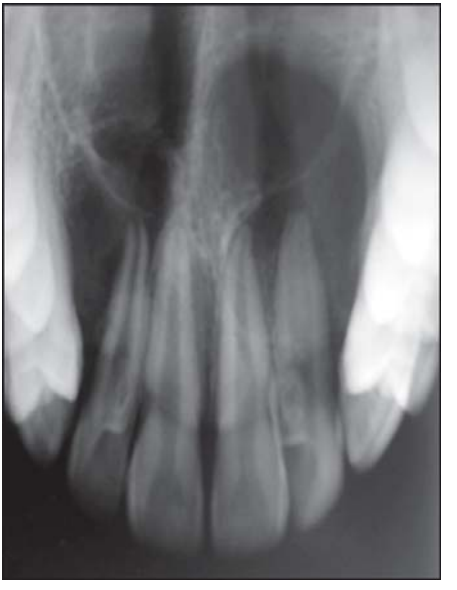


Fig. 23: Radicular cyst

- Aneurysmal bone cyst
- Solitary bone cyst.

Giant Cell Lesions of Jaw

- Osteoclastoma
- Hyperparathyroidism
- Giant cell reparative granuloma
- Endosteal hemangioma
- Aneurysmal bone cyst.

Keratotic White Lesions

- Benign migratory glossitis (Fig. 24)
- White hairy tongue
- Lichen planus

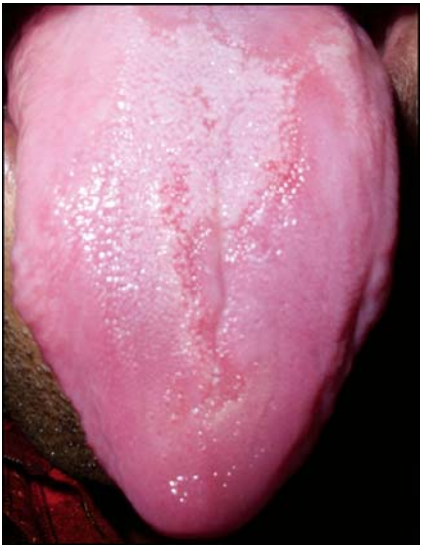


Fig. 24: Migratory glossitis

- Leukoderma
- Lichenoid drug
- Leukoplakia
- Papilloma
- Submucous fibrosis
- Hypovitaminosis A
- Scleroderma
- Koplick's spot
- Lupus erythematosus
- Verruciform xanthoma

Lesions around Crown of Impacted Tooth

- Dentigerous cyst
- Eruption cyst
- Ameloblastoma (Fig. 25)
- Odontogenic fibroma
- Adenoameloblastoma.

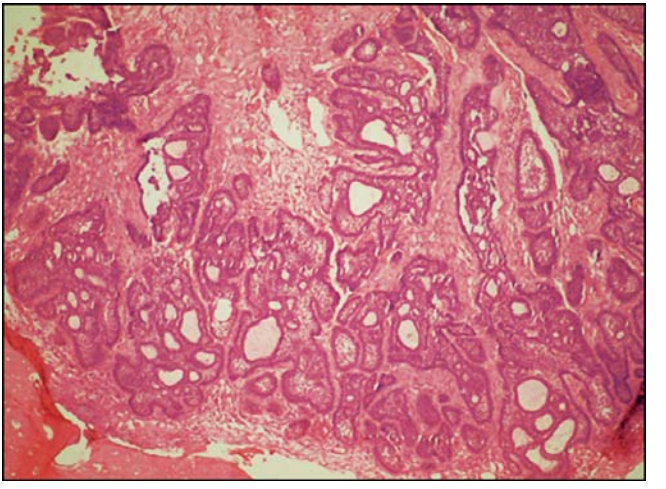


Fig. 25: Ameloblastoma

Midline Lesions of Maxilla

- Incisive canal cyst
- Median palatine cyst
- Globulomaxillary cyst
- Nasoalveolar cyst
- Cyst of palatine papilla
- Median alveolar cyst.

Lesions of Facial Skin

Premalignant and Malignant Lesions

- Bowen's disease
- Keratoacanthoma
- Squamous cell carcinoma
- Acinic keratosis
- Basal cell carcinoma.

Benign Epidermal

- Appendages
- Trichoepithelioma
- Senile sebaceous hyperplasia
- Syringoma.

Miscellaneous Tumor like

- Epidermal cysts
- Seborrheic lesions.

Vascular Tumors

- Nevus flammeus
- Juvenile hemangioma

- Angiosarcoma
- Pyogenic granuloma.

Miscellaneous Tumors of Melanocystic System

- Malignant melanoma
  - Intradermal nevus
  - Junctional nevus
  - Compound nevus
  - Blue nevus.

Lesions of Hard Dental Tissues

- Caries
- Erosion
- Abrasion
- Attrition (Fig. 26)
- Root resorption
- Hypercementosis.

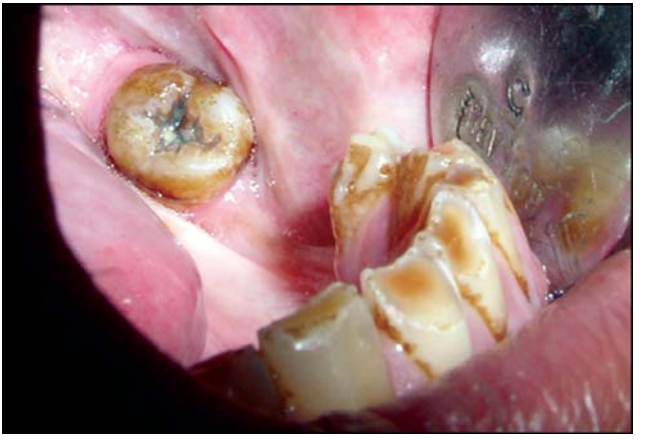


Fig. 26: Severe attrition of lower incisors

Lesions of Lips

- Congenital
- Cleft lip
- Double lip
- Erythema multiforme
- Elevated lesions
- Commissural pits
- Lower lip sinuses
- Ulcerative lesions
- Traumatic ulcers
- Keratoacanthoma
- Aphthous ulcers
- Fibromas
- Mucocele
- Angioedema
- Lipoma
- Cheilitis granuloma
- Salivary gland tumor
- Hemangioma.

Lesions over Dorsal and Lateral Surfaces of Tongue

- Fissures
- Traumatic ulcer
- Bald tongue-vitamin deficiency
- Recurrent aphthous ulcers
- Hemangioma
- Lichen planus
- Hairy tongue
- Median rhomboid glossitis
- Migrating glossitis

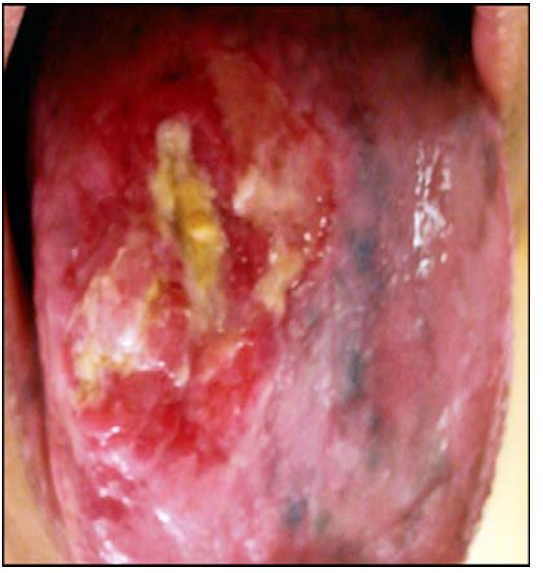


Fig. 27: Squamous cell carcinoma of tongue

- Hypothyroidism
- Neurofibromatosis
- Hemangioma
- Cancer tongue (Fig. 27).

Lesions over Ventral Surface of Tongue

- Ankyloglossia (Fig. 28)
- Traumatic ulcer
- Lingual varices
- Mucous retention cyst
- Squamous cell carcinoma
- Benign mesenchymal tumors.

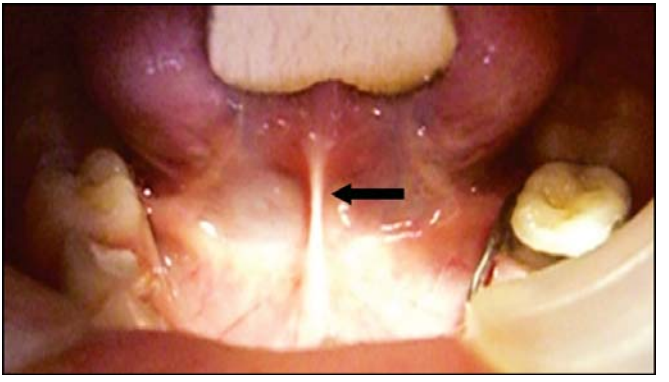


Fig. 28: Ankyloglossia

Mobile Tooth (Fig. 29)

- Trauma
- Normal resorption of deciduous teeth
- Pulpoperiapical lesion

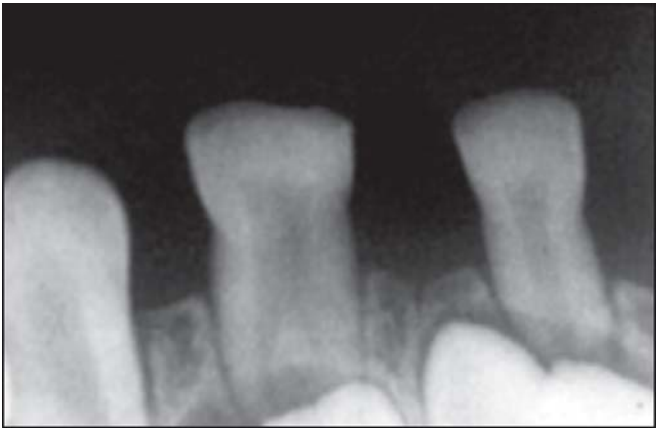


Fig. 29: Normal resorption of primary tooth

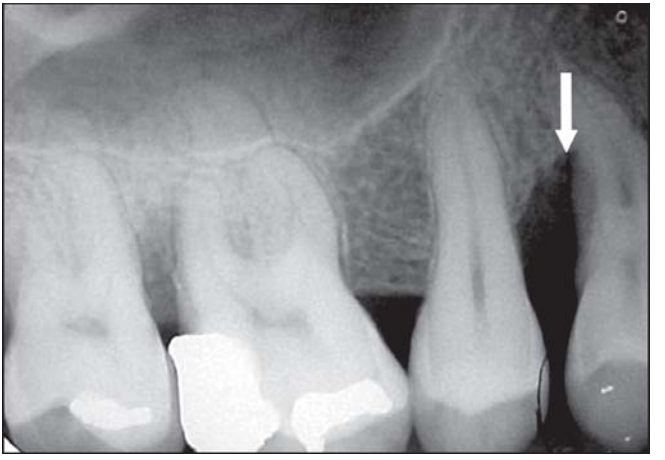


Fig. 30: Bone resorption due to periodontitis

- Malignant tumor
- Histiocytosis
- Hypophosphatasia
- Root absorption
- Trauma from occlusion
- Bone resorption (Fig. 30)
- Fracture
- Advanced periodontal diseases.

Lumps in Tongue

Localized

- Congenital
- Hamartomas
- Lingual thyroid
- Inflammatory

Postoperative infection insect bite
- Traumatic
  Edema hematoma
- Neoplastic
  Papilloma carcinoma granular cell tumor
- Others
  Foreign body cyst.

Malformations Affecting Soft Tissue

- Cleft lip
- Macrostomia
- Microstomia
- Macrocheilia
- Double lip
- Macroglossia
- Lingual thyroid
- Ankyloglossia
- Cleft tongue (Fig. 31)
- Thyroglossal duct cyst
- Branchial cyst
- Fissured tongue
- Dermoid cyst
- White sponge nevus
- Branchial sinus.

Malformations Affecting Teeth

- Ectodermal dysplasia
- Anodontia
- Accessory teeth
- Supernumerary teeth (Fig. 32)

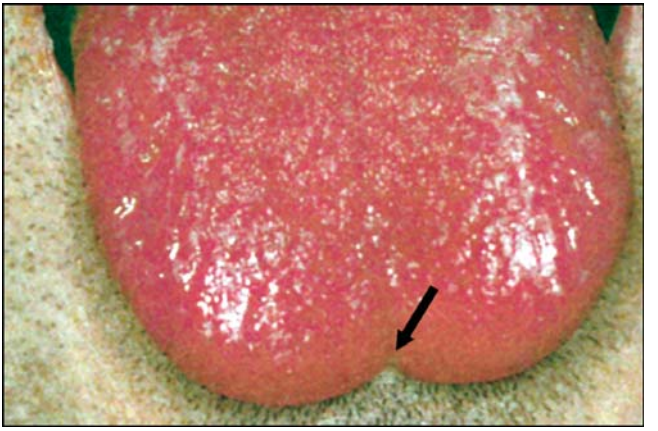


Fig. 31: Cleft/bifid tongue

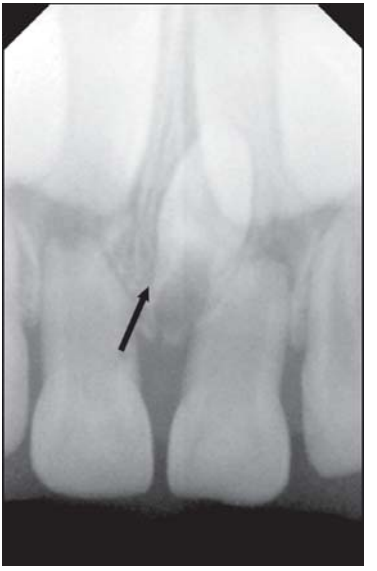


Fig. 32: Supernumerary teeth

- Predeciduous dentition
- Hutchinson's incisors
- Mulberry molars
- Macrodontia
- Microdontia
- Gemination
- Dentinogenesis imperfecta
- Amelogenesis imperfecta
- Shell teeth
- Enamel hypocalcifications.

Malignant Tumor of Jaw

- Ewing's tumor
- Osteosarcoma
- Metastatic tumor
- Chondrosarcoma
- Histiocytic lymphoma
- Sarcoma.

Mandibular Joint Clicking

- Chronic arthritis of TM joint
- Abnormalities of intra-articular disk
- Tear of meniscus
- Laxity of meniscus
- Following trauma
- Malposition of teeth
- Tooth extraction.

Mass in Neck

- Benign lymphoid hyperplasia
- Acute lymphadenitis

- Fibrosed lymph nodes
- Sebaceous cysts
- Space abscess
- Salivary gland inflammation
- Lipoma
- Solitary gland tumors
- Thyroid gland enlargements
- Lymphomas
- Thyroglossal cysts
- Cystic hygroma.

Midline Neck Swelling

- Goiter
- Thyroid neoplasia
- Dermoid cyst
- Thyroglossal tract cyst.

Mixed Lesions of Jaw

- Cystic odontoma
- Cementoma
- Osteogenic sarcoma
- Chondrosarcoma
- Ossifying fibroma
- Paget's disease
- Metastasis
- Chronic sclerosing osteomyelitis
- Condensing osteitis.

Mixed Lesions of Teeth

- Pericoronal mixed lesions
  - Calcifying odontogenic cyst

  - Odontoma
  - Adenomatoid odontogenic tumor
  - Ameloblastic fibrodontoma
  - Odontogenic fibroma
  - Calcifying epithelial odontogenic tumor
- Periapical mixed lesions
  - Tooth root with osteitis
  - Calcifying crown of developing tooth
  - Periapical cementoma
  - Rarefying and condensing osteitis
  - Cementifying/ossifying fibroma.

Multilocular Radiolucencies of Oral Cavity

- Ameloblastoma
- Multilocular cyst
- Cherubism
- Odontogenic myxoma
- Metastatic tumor of jaws
- Central giant cell granuloma
- Central hemangioma of bone
- Aneurysmal bone cyst.

Multiple Exophytic Oral Lesion

- Amyloidosis
- Cysticercosis
- Histiocytosis-X
- Kaposi's sarcoma
- Acanthosis nigricans
- Calcinosis
- Giant cell fibroma

- Multiple lipoma
- Phenytoin hyperplasia
- Xanthema disseminatum
- Multiple exostosis
- Condylomatalata
- Crohn's disease.

Multiple Separate Radiolucent Lesions of Jaw

- Multiple myeloma
- Eosinophilic granuloma
- Cherubism
- Metastatic tumor
- Hyperparathyroidism.

Multiple Separate Radiopacities

- Tori and exostosis
- Cysticercosis
- Multiple retained roots
- Multiple mature cementoma
- Multiple socket sclerosis
- Multiple impacted tooth.

Multiple Separate Well-defined Radiolucencies

- Multiple myeloma
- Basal cell nevus syndrome
- Multiple cyst/granuloma
- Histiocytosis-X
- Metastatic carcinoma.

Multiple Well-defined Radiolucencies

- Multiple myeloma
- Anatomic variations
- Metastatic carcinoma
- Basal cell nevus syndrome
- Multiple cystic or granuloma.

Myofacial Pain Dysfunction

- Stress
- Trauma to TM joint
- Over closure of mouth
- Poorly fitting dentures
- Abnormal chewing habits
- Improper occlussion.

Nonkeratotic White Oral Lesions

- Habitual cheek and lip biting
- Uremic stomatitis
- Burns of oral mucosa.

Normal Radiolucencies of Mandible

- Mandibular foramen
- Mandibular canal
- Mental foramen
- Lingual foramen
- Mental fossa
- Midline symphysis
- Submandibular fossa.

Normal Radiolucencies of Maxilla

- Nasolacrimal duct
- Incisive foramen
- Intermaxillary sutures
- Maxillary sinus.

Odontogenic Tumors of Jaw

- Epithelial tumors
  - Ameloblastoma
  - Unicystic ameloblastoma
  - Odontogenic adenamatoid tumor
  - Melanoameloblastoma
  - Acanthomatous ameloblastoma
- Mesenchymal tumors
  - Cementoma
  - Benign cementoblastoma
  - Odontogenic myxoma
  - Cementifying fibroma
  - Odontogenic fibroma
  - Dentinoma
- Mixed tumors
  - Ameloblastic odontoma
- Ameloblastic fibroma
- Odontoma
- Granular cell ameloblastic fibroma
- Ameloblastic fibrodontia
- Rare tumors
  - Extraosseous odontogenic tumor
  - Ameloblastic fibrosarcoma
  - Granular cell ameloblastoma

- Squamous odontogenic tumor
- Calcifying epithelial odontogenic tumor.

Oral Bleeding

- Gingivitis
- Traumatic injuries (Fig. 33)
- Allergies
- Ulcers
- Pulpal exposure
- Inflammatory hyperplasia
- Deficiencies in hemostasis
- Vitamin C deficiency.

Oral Blue/Purple Vascular Lesions

- Hereditary hemorrhagic telangiectasia
- Varices

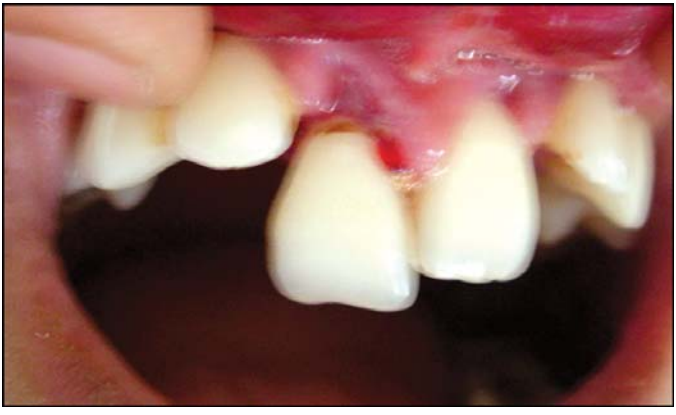


Fig. 33: Avulsed tooth due to traumatic injury

- Kaposi's sarcoma
- Hemangioma
- Angiosarcoma.

Oral Burning Sensation of Tongue

- Psychosis
- Viral infection
- Idiopathic burning tongue
- Neurosis
- Geographic tongue
- Fissured tongue
- Anemia
- Multiple sclerosis
- Vitamin deficiency.

Oral Candidiasis

- Acute-Thrush
- Chronic-Chronic atrophic candidiasis
- Denture sore mouth
- Angular cheilitis
- Median rhomboid glossits.

Oral Inflammatory Hyperplasia

- Fibroma
- Pulp polyp
- Pyogenic granuloma
- Myxofibroma
- Hormonal tumor
- Epulis fissuratum.

Oral Multilocular Radiolucencies

- Cherubism
- Central giant cell granuloma
- Odontogenic Keratocyst
- Odontogenic myxoma
- Central hemangioma
- Metastatic tumor of jaws.

Oral Radiolucency with Ragged and Ill-defined Borders

- Fibrous dysplasia
- Chronic osteitis
- Chronic osteomyelitis
- Squamous cell carcinoma
- Osteogenic sarcoma
- Chondrosarcoma
- Metastatic tumor of jaw
- Hematopoietic bone marrow defect.

Oral Tumors

- Odontogenic origin
- Non-Odontogenic origin
  - Primary
  - Secondary
- Extension of malignant tumors
- Bone lesions not of osseous origin
  - Fibroma
  - Inclusions of salivary tissue
- Salivary tumors and other swellings of glands
- Epthelial lesions

- Benign
- Malignant
- Ulceration
  - Warty
  - Nodular thickening
  - Leukoplakia with fissuring.

Oral Ulcers

Single ulcer
- Histoplasmosis
- Blastomycosis.

Recurring Oral Ulcer

- Recurrent aphthous stomatitis
- Recurrent herpes simplex
- Behçet's disease.

Acute Multiple Lesions

- Vericella zoster virus infection
- Herpes virus infection
- Allergic stomatitis
- Erythema multiforme.

Chronic Multiple Lesions

- Pemphigus
- Bullous pemphigoid
- Erosive and bullous lichen planus.

Osteomyelitis

Direct inoculation
- Foreign body in wound

- Hematoma
- Prosthetic joint
- Traumatic tissue necrosis by hematogenous route
- Bacteremia
- Septicemia
- Pyemia
- Boil/abscess or an occult focus.

Contiguous Spread

- Diabetic ulcers
- Neuropathic ulcers
- Ischemic ulcers

Palatal Swelling (Fig. 34)

- Palatal abscess/cyst
- Torus palatinus
- Pleomorphic adenoma

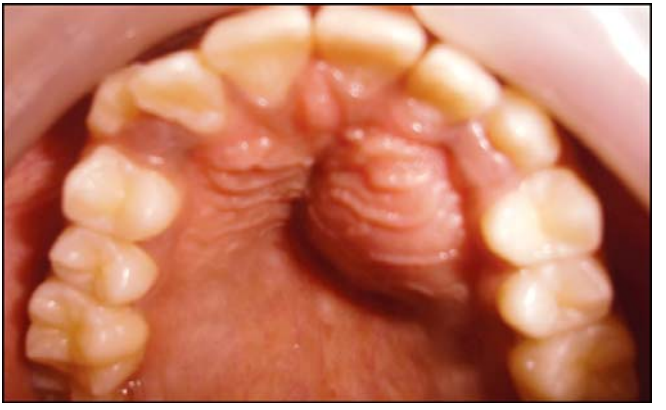


Fig. 34: Radicular cyst of the palate

- Adenoid cyst carcinoma
- Mucoepidemoid carcinoma
- Adenomatous hyperplasia.

Periapical Mixed Lesions

- Tooth root with rarefying osteitis
- Rarefying and condensing osteitis
- Calcifying crown of developing tooth
- Periapical cementoma
- Ossifying fibroma.

Periconal Radiolucencies

- Dentigerous cyst
- Ameloblastoma
- Ameloblastic fibroma
- Unicystic ameloblastoma
- Calcifying odontogenic cyst.

Persistent Anosmia (Abnormality of Smell)

- Paralysis of 5th nerve
- Polyp
- Adenoids
- Tumor
- Partial or full blockade of nose
- Basal meningitis
- Deflected septum
- Hypertrophic rhinitis
- Diseases of olfactory mucous membrane
- Atropic rhinitis
- Tumor frontal lobe
- Abnormalities of olfactory nerve.

SECTION

3

Differentiating Tables

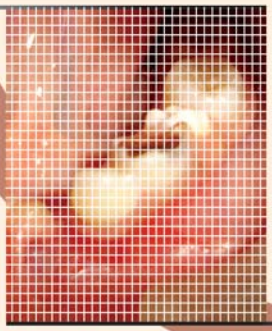


Table 1: Acute herpetic gingivostomatitis and acute necrotizing ulcerative gingivitis

Acute herpetic gingivostomatitis	*Acute necrotizing ulcerative gingivitis*
Duration of 7 to 10 days	No definite duration
Specific viral etiology	Interaction between host, bacteria and fusospirochetes
Vesicles rupture leaving depressed oval or spherical ulcer	Punched-out gingival margin. Pseudomembrane peels off
Occurs more freely in children	Relatively uncommon in children
An acute episode results in some degree of immunity	No immunity demonstrated
Diffuse erythema and vesicular eruption	Necrotizing condition
Contagious	Not proved

Table 2: Acute necrotizing gingivitis and primary herpetic gingivostomatitis

	Myth	*Fact*
Occurrence	Rare in children	Most frequently seen in children
Sites	Preferentially, interdentally penetrates deeply and spreads laterally	No definite intraoral predilection. Diffusely spreads over entire gingiva. Lesions are seen upon the tongue and oral mucosa
Fetor	Characteristic fetor	Fetor may be present but not characteristic of disease
Fever/malaise	May occur	Fever is high and marked
Erythema	Linear erythema	Diffuse erythema
Lesions	Necrotic ulcers	Round, grayish small vesicles which disrupt and leave small ulcers

Table 3: Acute necrotizing ulcerative gingivitis and secondary stage syphilis

Acute necrotizing ulcerative gingivitis	*Secondary stage of syphilis (mucous patch)*
It is a painful condition	Minimal pain
Affects marginal gingiva	Rarely affects marginal gingiva
Easy to remove membrane	Membrane cannot be removed
Immunity not conferred	Immunity not conferred
Marginal gingiva affected	Any part of mouth affected
Contagiousness not sure	Only direct contact will communicate disease

Table 4: Acute necrotizing ulcerative gingivitis/desquamative gingivitis and chronic destructive periodontal diseases

Acute necrotizing ulcerative gingivitis	*Desquamative gingivitis*	*Chronic destructive periodontal disease*
Bacterial smear shows fusospirochetal complex	Bacterial smears reveals numerous epithelial cells, a few bacterial forms	Bacterial smears are variable
It is painful	May or may not be painful	Painless
Acute onset	Chronic onset	Chronic onset
Marginal gingiva affected	Other areas of oral mucosa	Marginal gingiva affected
Characteristic fetid odor	None	Some odor present but not fetid
Papillary and marginal necrotic lesions	Papilloma does not undergo necrosis	Papillae do not undergo notable necrosis

Table 5: Ameloblastoma and adenomatoid odontogenic tumor

	Ameloblastoma	*Adenomatoid odontogenic tumor*
Incidence	1% of all oral tumors and cysts	100 reported cases
Age of occurrence	20-49 years	11-19 years
Sex incidence	Male and female equal	Females > Males
Location	Mandible > maxilla	Maxilla > Mandible (cuspid area)
Signs and symptoms	Destructively invasive "locally malignant" swelling with few true metastases	Expands cortical bone but is not invasive
Radiographic appearance	Multilocular radiolucency	Radiolucency (1-2 cm diameter) with small masses of calcification
Treatment	Surgical resection with adequate margins (Block excision)	Curettage

Table 6: Syndromes associated with oral lesions

Syndrome	*Oral lesions*
Tuberous sebaceum	Adenoma sebaceum, gingival lesions, enamel hypoplasia and cranial defects.
Peutz-Jeghers syndrome	Pigmented macules of lips and oral mucosa
Albright's syndrome	Solitary or multiple foci of fibrous dysplasia of jaw bones and oral pigmentation (rarely)
Acanthosis nigricans	Perioral and oral-mucosal papillomatosis with areas of black pigmentation
Paget's Disease	Localized or generalized bony jaw growths, hypercementosis
Cowden's Syndrome	Papillomatosis of lips, gingiva, palate, pharynx, fauces and fissured tongue
Vonghausen's neurofibromatosis	Intraoral neuro (especially of tongue) leading to macroglossia Rarely, intrabony neurofibromas of jaws
Gardner's syndrome	Multiple osteomas of cranial and facial skeleton (especially frontal bone, mandible, and maxilla) Compound odontomas and hypercementosis
Nevoid basal cell carcinoma syndrome	Multiple jaw cysts (simple, primordial, and odontogenic keratocysts) Dialceration of teeth adjacent to cysts. Facial abnormalities (frontal bossing, sunken eyes, and wide nasal bridge; mild mandibular prognathism.
Multiple mucosal (multiple endocrine neopiasia type III)	Neuromas of lips, tongue and buccal mucosa (oral cavity most common site) Thick or "bumpy" lips prognathism (infrequent)

Table 7: Categories of tooth fracture

	Fractured cusp	*Split tooth*	*Cracked tooth*	*Vertical root fracture*
Location	Crown and cervical margin of root	Crown and root extension to surface	Crown and root extension (depth varies)	Root only
Direction	Mesiodistal and faciolingual	Mesiodistal	Mesiodistal	Faciolingual
Origination	Occlusal surface	Occlusal surface	Occlusal surface	Root (any level)
Etiologies	Undermined cusp and damaging habits	Damaging habits, weakened tooth structure	Damaging habits, weakened tooth structure	Wedging posts, obturation forces, excessive root dentin removal and trauma
Symptoms	Sharp pain on mastication and exposure to cold	Pain on mastication	Cracked tooth syndrome, or highly variable	None to slight
Signs	Not significant	Separable segments, periodontal abscess	Variable	Variable
Prevention	Place conservative class II restorations, onlays, posterior composite restoration	Obliterate deleterious habits, coronal protection (onlay cusps). Removal of excessive occlusal forces	Obliterate deleterious habits (ice chewing, etc.) coronal protection (onlay cusps)	Prevention of trauma, Minimize root dentin removal, avoid wedging posts, reduce condensation forces

Table 8: Chronic mandibular hypomobilities

	Contracture of elevator muscle	*Capsular fibrosis*	*Ankylosis*
History	Prior protracted restriction of opening of jaw Prior muscle trauma or inflammation	Prior trauma, surgery, capsular inflammation	Prior trauma or infection
Masticatory pain	None, unless injured by excessive movement	None, unless injured by excessive movement	None, unless injured by excessive movement
Restriction of movement	Extracapsular restriction of condylar movement	Capsular restriction of condylar movement	Intracapsular restriction of condylar movement
Interference during movement	None	None	None
Radiographic confirmation	Positive for extracapsular restraint of condyle	Positive for capsular restraint of condyle	Positive for intracapsular restraint of condyle

Table 9: Deciduous teeth

Upper	*Lower*
Longer	Shorter
Crown is wider mesiodistally	Crown is narrow mesiodistally
Crown is blunt	Crown is more pointed
Cingulum prominent	Cingulum is more prominent
Molar has 3 roots—mesiobuccal, distobuccal and palatal	Molars has 2 roots—mesial and distal
Wider labiolingually	Narrower labiolingually

Table 10: Deciduous and permanent teeth

Deciduous teeth	*Permanent teeth*
Total 20 teeth	Total 32 teeth
2 incisors + 1 canine	2 incisors + 1 canine
2 molars	3 molars
No premolars	2 premolars
Smaller teeth	Larger than deciduous
Cusps are more pointed and crowns are bulbous	Crowns are blunt.
Contact areas are smaller	Contact areas are broader
Enamel is less translucent	Enamel is more translucent
White color	Color is yellowish white
Roots are shorter delicate	Roots are longer and strong
Roots are more flared	
Dentin is less thick	Dentin is thicker.
Pulp cavity is larger	Pulp cavity is smaller
Pulp horns arise high	Pulp horns are lower
Enamel is less calcified and shows more attrition	Enamel is less permeable more calcified.
Set perpendicularly in jaw	Placed obliquely in jaws
Eruption starts at 6 months	Eruption starts at 6 years

Table 11: Dental calculus

Characteristic	*Supragingival calculus*	*Subgingival calculus*
Color	White, creamy-yellow, gray May be stained by tobacco, food, or other pigments Slight deposits may be invisible until dried with compressed air	Light to dark brown, dark green, or black Stains Derived from blood pigments from diseased pocket
Shape	Amorphous, bulky Gross deposits may: • Form interproximal bridge between adjacent teeth • Extend over the margin of the gingiva Shape of calculus mass is determined by the anatomy of the teeth, contour of gingival margin, and pressure of the tongue, lips, cheeks	Flattened to conform with pressure from the gingival pocket wall Combinations of the following calculus formations occur • Crusty, spiny, or nodular • Ledge or ring-like formations • Thin, smooth veneers • Finger and fern-like formations Individual calculus islands
Consistency and texture	Moderately hard Newer deposits less dense and hard Porous surface covered with plaque	Brittle, flint-like Harder and more dense than supra-marginal calculus Newest deposits near bottom of pocket are less dense and hard. Surface covered with plaque

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Characteristic	*Subgingival calculus*	*Supragingival calculus*
Size and quality	Quantity has direct relationship to: • Personal oral care procedures and plaque control measures • Physical character of diet • Individual tendencies • Function and use Increased amount in tobacco smokers	Related to pocket depth Increased amount with age because of accumulation Quantity is related to personal care, diet, and individual tendency as it is with supramarginal. Submarginal is primarily related to the development and progression of periodontal disease
Distribution on individual tooth	Above margin of gingiva may cover a large portion of the visible clinical crown, or may form fine thin line near gingival margin	Below margin of gingiva. Extends to bottom of the pocket and follows contour of junctional epithelium. With gingival recession, submarginal calculus may become supramarginal and become covered with typical supra-marginal calculus.
Distribution on teeth	Symmetrical arrangement on teeth except when influenced by: • Malpositioned teeth • Unilateral hypofunction • Inconsistent personal care • Abrasion from food occurs with or without associated submarginal deposits Location related to openings of the salivary gland ducts: • Buccal of maxillary molars • Lingual mandibular anterior teeth	May be generalized or localized on a few teeth. Heaviest on proximal surfaces, lightest on facials. Occurs with or without associated supramarginal deposits

Table 12: Drugs causing oral lesions

Manifestation	*Group of drugs*
Gingival hyperplasia	Phenytoin, nifedipine, estrogen, phenobarbitol, cyclosporin A
Black tongue	Chlorhexidine, iron preparation, corticosteroids, penicillin
Tooth discoloration	Chlorhexidine, ferric ammonium chloride, corticosteroids, tetracycline
Pigmentation	Contraceptives, antimalarials, gold, silver
Drug enanthemas	Analgesics, antibiotics, quinine, sulfonamides
Lichenoid reaction	Antimalarials, beta-receptors, gold compounds
Macular enanthemas, ulcerative stomatitis	Analgesics, barbiturates, chloramphenicol, chlorpromazine, hydantoin, phenacetin, salicylates, indomethacin
Mucosal edema	Anticonvulsants, analgesics, local anesthetics, penicillin, sulfonamides

Table 13: Facial pain

	Atypical facial pain	*Idiopathic trigeminal neuralgia*	*Migrainous neuralgia*
Character associated features	Dull ± Depression	Lancinating. No neurological sign	Boring lacrimation conjunctival congestion
Age sex site	30 to 50 years Females + ± Bilateral maxilla	> 50 Females + Unilateral mandibular or maxilla	30 to 50 Males + Retrorbital
Precipitating factors	None	± trigger zones	± Stress and alcohol
Duration of episode	Continual	Brief	Few hours
Treatment	Anti-depressant	Carbamazepine	Ergot derivatives

Table 14: Gingiva

	Normal appearance	*Pathological changes*
Surface texture	Free gingiva: smooth Attached gingiva: stippled	Acute condition: loss of stippling, with smooth, shiny gingiva Chronic: hard, firm, with stippling, sometimes heavier than normal
Position	Fully erupted tooth: margin is 1-2 mm. Above CEJ, at or slightly below the enamel contour.	Enlarged gingiva: margin is higher on the tooth, above normal, pocket deepened. Recession : margin is more apical, root surface is exposed
Bleeding	No bleeding upon probing	Spontaneous bleeding Bleeding on probing: Bleeding near margin in acute condition; bleeding deep in pocket in chronic condition.
Exudate	No exudate on pressure	White fluid, pus, visible on digital pressure.Amount not related to pocket depth
Position of junctional epithelium	In fully erupted tooth the JE is at the CEJ During eruption: along the enamel surface	Position, determined by use of probe, is on the root surface
Mucogingival lines	Makes clear demarcation between the pink, stipped, attached gingiva and the darker alveolar mucosa with	No attached gingiva Color changes may extend full height of the gingiva; mucogingival line obliterated

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	Normal appearance	*Pathological changes*
	smooth shiny surface and thin keratinization	Probing reveals that the bottom of the pocket extends into the alveolar mucosa Frenal pull may displace the gingival margin from the tooth
Shape	Marginal gingiva: Knife-edge, flat Follows a curved line about the tooth Papillae: Normal contact, papilla is pointed and pyramidal; fills the inter-proximal area created • Shape (diastema) between teeth; gingiva is flat or saddle-shaped	Marginal gingiva; Rounded rolled bulbous Papillae : Bulbous flattened blunted
Consistency	Firm, resilient Attached gingiva is firmly bound down	Soft, spongy, dents occurs readily when pressed with probe associated with red color, smooth shiny surface, loss of stippling, bleeding on probing
Color	Uniformly pale pink Variation in pigmentation related to complexion, race	Acute: bright red Chronic: bluish pink, bluish redpink Attached gingiva: color change may extend to the mucogingival line
Size	Flat, not enlarged Fits snugly around the tooth	Enlarged

Table 15: Histologic features of oral lesions

Disease	*Gross features*	*Epithelial changes*	*Fluid content*
Bullous pemphigoid	Epithelial separation from underlying connective tissue	Minor degeneration alteration	Fibrinous exudate
Cicatricial	Epithelial separation from underlying connective tissue	Basal layer may remain intact	Clear fluid scattered vessels
Pemphigus	Epithelial cells are separate from basal cell layer	Disappearance of intercellular bridge	Edema, poly-morphonuclear leukocytosis
Lichen planus	Subepithelial vessel	Parakeratosis	Edema with inflammatory cell
Desquamative gingivitis	Thin epithelium	Atrophy disrupted basal layer	Epithelial intercellular edema

Table 16: Identification of deciduous teeth

Tooth	*Maxillary arch*	*Mandibular arch*	*How to tell (right from left) Maxillary teeth*	*Mandibular teeth*
Central	• Large cingulum • Long bulky root • Root bends facially in apical one third	• Small crown • Long, thin and straight root	• More cervical distal than mesial contact • More rounded distoincisal angle • Crown outline flat on mesial side, round on distal side • More mesial cervical curvature	
Lateral incisor	• Narrow and oblong • Asymmetrical crown • Root bends facially in apical one-third	• Same shape as appears • Smaller cingulum • Less acute mesio-incisal angle • No labial root bend	• Flat mesial crown outline • Rounded distal crown outline • Distal contact more cervical than mesial • Acute mesioincisal angle • More mesial cervical curvature Cingulum slightly to distal	• More rounded • Distoincisal angle • Distal contact • More cervical than mesial distal crown bulge • Cingulum slightly distal

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Tooth	*Maxillary arch*	*Mandibular arch*	*How to tell (right from left) Maxillary teeth*	*Mandibular teeth*
Canine	• Diamond crown, • Sharp centered cusp • Rounded contacts in middle of tooth • Longer, steeper mesial cusp arm • Flat labial cervical line • Cingulum centered • Root bends facially in apical one-third	• Longer, narrower less symmetrical crown • Cusp tip toward mesial • Short mesial cusp ridge • Distally located • Cingulum contacts in incisal one-third • No labial root end	• Longer mesial cusp slope • Mesial contact more cervical than distal • More mesial cervical curvature • Deeper and narrower distal than mesial fossa	• Shorter mesial • Slope flat mesial crown outline • Distal contact more cervical than mesial • Cingulum toward distal
First molars	• 3 roots—MB, DB, L Unique crown shape • 3 cusps—MB (verylarge) DB, ML	• 2 roots—mesial and distal • 4 cusps—MB, DB, ML,DL • Unique crown shape	• Crown longer on mesial than distal • Crown wider mesially than distally (F-L)	• Crown longer on mesial than distal • Crown wider distally than mesially (F-L) • Mesial cervical crown bulge

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Tooth	*Maxillary arch*	*Mandibular arch*	*How to tell (right from left) Maxillary teeth*	*Mandibular teeth*
	• Crown oblong faciolingually tapering toward lingual • Crown wider mesially than distally (F-L) • H-shape occlusal grooves • Crown exactly like small, short permanent maxillary first molar	• Crown very wide mesiodistally and narrow faciolingually • Crown wider distally than mesially (F-L) • Well developed high mesial marginal ridge • Crown closely resembles permanent mandibular first molar	• Mesial cervical crown bulge • Distal marginal ridge more cervical than mesial • Distobuccal root is smallest and shortest	• Larger distal than mesial fossa pointed mesiolingual cusp • Distal marginal ridge more cervical than mesial • Longer, wider (F-L) mesial root Crown longer on mesial than distal • Mesial cervical crown bulge identify and position distal cusp (fifth cusp) • Distal marginal ridge more cervical than mesial longer, wider (F-L) mesial root

Table 17: Infectious diseases: systemic manifestation and their oral manifestations

Disease	*Other systemic manifestations*	*Oral manifestations*
AIDS	Pneumonia, kaposi's sarcoma, lymphoma	Herpes simplex candidiasis,Hairy leukoplakia,Periodontal disease, ulcerations, Kaposi's sarcoma, cervical lymph-adenopathy
Chickenpox	Rash, papule, regional lymph nodes enlarged, mild fever	Cervical lymphadenopathy
Diphtheria	Tonsillar or pharyngeal exudate cervical glands++ myocarditis	Tonsillar exudate palatal palsy
Hepatitis erysipelas Herpes simplex	Jaundice, malaise, pale stools, dark urine rash, fever, cervical glands ++	Oral ulceration ,Tonsillitis, Gingivostomatitis
Herpes zoster	Rash similar to chickenpox in affected dermatone	Oral ulceration
Measles	Rash, fever, acute respiratory symptom	Koplik's spots, pharyngitis
Mumps	Fever, malaise, parotitis	Sialadenitis, trismus
Pertusis	Cough, fever	Ulceration of lingual frenum
Polio	Paralysis	Ulceration of oral cavity

Table 18: Inflammatory disorders of the joint

	Synovitis and capsulitis	*Retrodiskitis*	*Inflammatory arthritis*
History	Prior trauma, inflammation of disc ligaments or temporomandibular ligament. Prior capsule fibrosis, periarticular inflammation	Prior recent trauma Activated pre-existent non-anchored posterior overclosure	Prior trauma, infection, illness progressive degenerative disorder, involvement of other joints, or arthropathy
Masticatory pain	Capsular type arthralgia, continuous but variable, increases with stretching of capsule. Palpable tenderness over joint proper. Secondary pains may occur	Retrodiskal type arthralgia Intermittent, occurs with maximum intercuspation Reduced by biting against a separator	Arthropathy type arthralgia continuous but variable. Relates to functional demands. Usually accompanied by capsulitis Secondary pain may occur
Restriction of movement	Capsular type restrained joint movement. Variable in degree, up to complete immobilization. Extracapsular restraint due to secondary spasm	None	If any, due to capsulitis, pre-existent disorder or arthropathy, or secondary myospasm activity
Acute malocclusion	If any, due to accumulation of intracapsular fluid	Posterior disocclusion with premature contact of contralateral anteriors	If any, due to intracapsular fluid, preexistent disorder or arthropathy, or secondary lateral pterygoid spasm

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	Synovitis and capsulitis	*Retrodiskitis*	*Inflammatory arthritis*
Radiographic confirmation	Positive for location of restricted movement and gross malocclusion	Positive for gross malocclusion	Positive for location of restricted movement, gross malocclusion, change in subarticular bone

Table 19: Major and minor aphthous ulcers

	Major	*Minor*
Size	> 1 cm	< 1 cm
Duration	Upto 30 days	Heal within 10 days
Scarring	Yes	No
Numbness	Upto 10	1-5
Palatal and pharyngeal	Yes	No
Associated disorder	Behcet's	No

Table 20: Mandibular first, second and third molars

First molar	*Second molar*	*Third molar*
Occlusal outline pentagonal/hexagonal	Rectangular	Ovoid
Generally 5 cusps are present	Four cusps are present	Four cusps and one buccal groove
Two buccal grooves	There is one buccal groove	
Main occlusal grooves	Main grooves form a ++ pattern	Pattern variable
Mesial root is broad	Mesial root is not as broad	Mesial root is not as broad
Roots divergent	Roots are more or less parallel	Mesial roots are curved distally

Table 21: Mandibular central incisors and Mandibular lateral incisors

Mandibular central incisors	*Mandibular lateral incisors*
Bilaterally symmetrical	Bilaterally symmetrical
Mesioincisal and distoincisal are sharp at right angles	Distoincisal angle is pointed
Incisal edge is at right angle to labiolingual bisecting line	Incisal edge is twisted on crown
Mesiodistally narrower	Mesiodistally wider

Table 22: Maxillary and mandibular canines

Labial view(Maxillary canine)	• More acute cusp angle on uppers • Crown of uppers is more squatty (short and wide) • Both contact points are nearer to the level of cusp tip on lowers • Length of cusp slopes (mesial is shorter, much shorter on lowers), opposite in mandibular canines • Extramesial bulge to crown beyond root on uppers • Continuous mesial crown root outline on lowers • Labial ridge more pronounced on uppers

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Proximal view	• Straighter, shorter root and more blunt root tip on lowers • More pointed root tip and distal bend in apical one third on uppers • Less prominent cingulum on lowers • Location of cusp tip to root axis line—labial on uppers lingual on lowers • Labial crest of curvature closer to cervical line on lowers • Slope of incisal ridges—located toward labial and downward on lowers, toward lingual and upward on uppers (due to attrition)
Incisal view	• Asymmetrical crown outline on uppers—more symmetrical and oblong faciolingually on lowers • Location of cusp and cusp ridges—labial to center uppers; lingual to or on center for lowers • Greater faciolingual bulk compared to mesiodistal width on lowers • Cingulum centered on uppers, off to distal on lowers • Attrition on cusp ridges of lowers but on lingual surfaces of uppers • Mesiodistal direction of cusp ridges—straight on uppers; bent slightly toward lingual on lowers • More bulky cusp tip on uppers due to heavy lingual ridge

Table 23: Maxillary first, second and third molar

First molar	*Second molar*	*Third molar*
Largest in series	Smaller and weaker than first molar	Smallest and weakest ++ in series
Cusps of Carabelli may be present	Carabelli's cusp absent	Carabelli's cusp absent
Occlusal outline is square	Romboid	Outline triangular or heart shaped
Buccal cusps are equal in height	Smaller	Distobuccal cusp is much shorter
Oblique ridge is prominent	Oblique ridge is smaller	Oblique ridge barely visible
Roots are strong	Roots are slender	Roots often fused

Table 24: Mucosal lesions of tongue

Conditions	*Causes*
Coated tongue	Chronic GIT disorders, oral thrush, febrile disease, smoking , hypovitaminosis
Superficial changes	Fissured tongue, geographic tongue, black hairy tongue
Smooth gray tongue	Vitamin A deficiency, lichen planus, glossitis, progressive systemic scleroderma
Smooth red tongue	Scarlet fever, asthma, hepatic cirrhosis, pernicious anemia, iron deficiency anemia, malnutrition, hypertension, right heart failures

Table 25: Oral pain

	Character of pain	*Pain* *Pain intensified by*	*Pain intensity*
Pulpal	Explosive, intermittent, throbbing, boring	Hot, cold, sometimes on chewing	Usually severe, may be mild to moderate
Periradicular	For hours on same level, deep boring	Chewing	Moderate to severe
Periodontal	For hours at same level, boring	Chewing	Moderate to severe
Gingival	Pressing, annoying	Food impaction, brushing	Mild to severe
Mucosal	Burning, sharp	Sour, sharp and hot food	Mild to moderate

Table 26: Orofacial pain syndromes

	Superficial pain	*Deep pain*	*Neurogenous pain*
Systemic effect of pain	Stimulating	Depressing	Stimulating
Subject's ability to localize the pain	Excellent	Poor	Excellent
Relationship between site of pain and its true source	Same	May or may not be the same	Not the same (except traumatic neuroma)
Effects that may accompany pain	None	Effects of central excitation	Direct neurologic effects
Effect of topical anesthetic applied to site of pain	Arrest the pain	None (except accessible visceral mucosa)	None (except accessible superficial triggers)

Table 27: Permanent filling materials

Filling	*Uses*	*Advantages*	*Disadvantages*
Amalgam	Permanent filling for posterior teeth	Simple technique Rapid set Strength Prevents microleakage	Lining usually required Delayed expansion
Composite	Restorations, Restoration of fractured incisors, malformed teeth Direct bonding of orthodontic brackets Post and core Splinting Space maintainers	Simple technique Strength and permanence Suitable for large and complicated restorations Undercut or acid etch retention Aesthetics Surface glazing	No strength for posterior tooth, polymerization shrinkage, technique sensitive procedure
Gold	Permanent filling or crown for back teeth	Much stronger than amalgam	Involves more time and expense than any other filling
Glassionomer cement	Permanent filling in primary teeth Cervical cavities in all teeth Deciduous teeth, Cavity lining Dentin substitute Adhesive cement Fissure sealant Cementation of crown and space maintainers PRR	Chemical bond to tooth Cariostatic Non-irritant Low thermal diffusivity Biocompatible	Opaque for front teeth in young patients insufficient strength for posterior tooth

Table 28: Permanent mandibular and maxillary incisors

Maxillary incisors	*Mandibular incisors*
Four upper incisors forming fixed cutting edge	Four lower incisors forms moving cutting edge. Lingual fossa is shallow
Lingual fossa is deeper	These are narrowest
More convex	Facial surfaces are less convex
Wider mesiodistally than labiolingually	Crowns are wider labiolingually. Roots are rounded or triangular
Roots are oval	These are first permanent teeth to appear
These erupt after lower incisors	Very few variation
Variations are numerous	Mesiodistal and buccolingual diameter is same
Crown is wider mesiodistally	

Table 29: Second premolar and first premolar

First premolar	*Second premolar*
Relatively less stronger	Stronger and more ragged
Two roots	One root only
Occlusal crown outline is hexagonal	Occlusal crown outline is ovoid or rectangular
Two root canals	Single root canal
Marginal ridges are narrower and not so strong	Marginal ridges are wider
Mesial marginal groove	There is no mesial marginal groove
Occlusal outline is diamond shaped	Occlusal outline is squarish or triangular
Buccal cusp is prominent and lingual cusp is rudimentary	Both cusps are equal in size
Transverse ridge is common	No transverse ridge is noted
Occlusal surface slopes lingually	Occlusal surface is horizontal
Buccal crown is bilaterally asymmetrical	Buccal crown is bilaterally symmetrical
Lingual surface is narrower than buccal	Lingual surface is not much narrower as compared to buccal

Table 30: Sequence of tooth eruption of course, many variations occur, but the usual order of appearance of the teeth in the oral cavity is as follows

Primary teeth	*Months*	*Permanent teeth*	*Years*
Mandibular central	6	Mandibular molars	5-3/4
Mandibular lateral	7	Maxillary first molars	6
		Mandibular central	6
Maxillary central	7½	Maxillary central and	7-8
Maxillary lateral	9	Mandibular lateral	
Mandibular first molar	12	Maxillary lateral	8-9
		Manibular canine	9-10
Maxillary first molar	14	Maxillary first premolar	10-11
Mandibular canine	16	Maxillary second premolar and mandibular first premolar	10-12
Maxillary canine	18	Mandibular second premolar and maxillary canine	11-12
Mandibular second molar	20	Mandibular second molar	11-13
Maxillary second molar	24	Maxillary second molar	12-13
		Third molars	17-21

Table 31: Temporary fillings

Fillings	*Uses*	*Advantages*	*Disadvantages*
Zinc oxide and eugenol cement	1 Temporary filling 2 Lining deep cavities 3 Sedative dressing for dry socket 4 Gingivectomy 5 Obturating material for primary teeth	1 Economical 2 Easily available 3 Can be used in deep cavities 4 Rate of resorption is closer to the primary tooth	1 Soft 2 Slow-setting 3 Irritant to soft tissue
Gutta-percha	1 Permanent filling for root canals 2 Vitality tests 3 Temporary obturator for cleft palates and cyst cavities 4 Lining for gunning splints	1 Inert 2. No mixing	1 Too soft 2. Periapically can act as a foreign body
Zinc phosphate cement	1 Thick mix : cavity lining: temporary filling 2 Thin mix: adhesive cement for inlays and crowns	1 Hard 2 Rapid set 3 Adhesive	1 Irritant in deep cavities
Polycarboxylate	1 Thick mix: cavity lining : temporary filling 2 Thin mix: adhesive cement for inlays, crowns and orthodontic bands	1 Less irritant 2 More adhesive	1 May be difficult to manipulate

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Fillings	*Uses*	*Advantages*	*Disadvantages*
Calcium hydroxide	1 Cavity lining 2 Endodontic treatment 3 Antibacterial 4 Pulp capping agent 5 Root induction procedures 6 Intracanal medicament	1 Non-irritant 2 Can be used in deepest cavities 3 Compatible with all materials 4 Restorable 5 Can be used in periapical areas	Sublining only, in deep cavities under metal fillings Contraindicated in primary dentition as it causes internal resorption

Table 32: Upper central incisors and upper lateral incisors

Upper central incisors	*Upper lateral incisors*
Labial surface is flat	Labial surface is rounded
Mesioincisal angle is sharp	Mesioincisal angle is rounded
Mesial profile is straight	Mesial profile is rounded
Moderately deep. Marginal ridges moderately prominent	Lingual fossa is deep
Mesial contact point is in lower third	Mesial and distal contact points are in middle third

Table 33: Maxillary molars and mandibular molars

Maxillary molars	*Mandibular molars*
Crown is broader buccolingually	Crown is broader mesiodistally
These have three roots	These have two roots
There are 3 large cusps and one small	There are 4 large cusps and one small
Two buccal cusps are unequal in size	Two buccal cusps are of equal size Two lingual cusps are of equal size

Table 34: Differential diagnosis of pain

	Musculoskeletal pain	*Odontogenous pain*	*Vascular pain*
Local etiologic factors chiefly responsible for the syndrome	Functional abuse of muscles and joints	Oral environmental conditions	None (except cranial arteritis)
Local factors that initiate or aggravate the pain	Manual palpation or functional manipulation	Noxious oral stimuli	None (except cranial arteritis)
Dysfunction that may accompany the pain	Dysfunction due to muscular or joint causes	Difficulty in mastication	None
Temporal behavior of the pain	No time frame (unless due to inflammation)	Pulpal to periodontal sequence-inflammatory curve	Periodic, recurrent (except cranial arteritis)

Table 35: Epilepsy and syncope

	Epilepsy	*Syncope*
Pulse	Difficulty in palpation	Slow
Color	Cyanosed	Pale and sweating
Convulsions	Usual	Uncommon
Urinary incontinence	Yes	Yes if bladder is full
Tongue biting	Yes	No
Duration	Minutes or hours	Brief
Warming	None	Visual loss, noises in ear
Provocation	None/may be alcohol/exertion	Posture/coughing/stress/ hypoglycemia

Table 36: Facial signs suggestive of disease

1	*Facial appearance*	*Disease*
	Hippocratic facies (shunken cheeks, pinched, nose, cold ears	Gastric ulcer diseases, tetanus acromegaly, nephrosis
	Risus sardonicus	
	Coarse features	
	Bloated face	
2	*Different colors of face*	
	Red face dark red to purple	Diabetes mellitus, essential hypertension
	Brown red skin color,	Alcoholism febrile disease
	Redden cheeks, Pallor	Dermatomyositis, hemosiderosis, mitral stenosis, anemia, shock
	Gray color, Jaundice	acute internal hemorrhage, renal disease, malignancy,
	Straw colored skin	Cirrhosis of liver, vitamin A deficiency, hepatobiliary disease, hemolytic disease, Pernicious anemia
3	Diffuse facial edema	Heart failure, nephritis, nephrotic syndrome Drug side effect. Hypoproteinemia
4	Pigmentary changes	Diabetes mellitus, Addison's disease, hyperthyroidism, ovarian tumors, arsenic poisoning

Table 37: Identifying features of categories of temporomandibular disorders

	Acute muscle disorders	*Disk-interference disorders*	*Inflammatory disorders*	*Chronic mandibular disorders*	*Growth disorders*
History	Sudden onset Changeable Recurrent	Insidious onset Persistent Progressive	Prior trauma, non-inflammatory joint disorder, illness	Prior trauma or infection Protracted course	Insidious structural change precedes symptoms
Masticatory pain	Dominates complaint. Myalgia relates to muscle action. Continuous but variable, may see secondary effects	If any, arthralgia of disk attachment type; intermittent	Dominates complaint. Arthralgia of inflammatory type relates to joint function. Continuous but variable; may see secondary effects	None, unless subjected to abusive movement	If any, due to progressive structural dysfunction
Restriction of movement	If any, extracapsular due to shortened, rigid, or swollen elevator muscle	If any, intracapsular, due to jamming or dislocation of disk	If any, due to capsulitis, disk jamming preexistent	Dominates complaints Due to ankylosis fibrous, or	If any, due to progressive structural dysfunction

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	Acute muscle disorders	*Disk-interference disorders*	*Inflammatory disorders*	*Chronic mandibular disorders*	*Growth disorders*
			arthropathy, extra-articular disease or secondary spasm	contracture of elevator muscle	
Interference during movement	If any, to increased interarticular pressure or lateral pterygoid spasm	Dominates complaints Abnormal sensations, noises and movements due to disc interference	If any, due to disruption of disk-condyle complex function	None	None, due to distruption of disk-condyle complex function
Acute malocclusion	If any, due chiefly to lateral pterygoid spasm	If any, due to fracture of dislocation of disc	If any, due to intracapsular fluid, retrodiskal swelling or bone loss	None	If any, due to rapid osseous change

Table 38: Anatomical differences of primary and permanent dentition

		Deciduous	*Permanent*
1.	Number of teeth	20 in number	32 in number
2.	Members of dentition	8 Incisors, 4 canines, 8 molars	8 incisors, 4 canines, 8 permolars and 12 molars
3.	Size	Smaller in size	Larger in size
4.	Color	Whiter and more opaque	Yellowish white and more translucent
5.	Contour	Has a more pronounced cervical bulge	Has a less pronounced and tapering cervical margin
6.	Cusps and Fossae	In newly erupted teeth, more pointed cups with deeper fossae	Less pointed cups with shallower fossae
7.	Roots	Shorter roots	Longer roots
8.	Root trunk	Definite root trunk absent	Definite root trunk present
9.	Root divergence	The roots of molars are more divergent [to accommodate developing premolars]	Roots of permanent molars not so divergent
10.	Root – Crown ratio	Roots of anterior deciduous teeth are very much longer compared to their crown length	Root length in permanent teeth not so long compared to their crown height. [Exception being the canine]

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		Deciduous	*Permanent*
11.	Shape of the root	Flatter	Rounded
12.	Cementoenamel junction	It is less sinous. Enamel end abruptly at the cemento enamel junction producing a bell shaped crown with a constricted neck	More sinuous
13.	Pulp chamber	Larger than in permanent teeth with correspondingly less depth of dentine	Smaller than in the deciduous with correspondingly more depth of dentine
14.	Pulp horn	More prominent and at higher level	Less prominent and low level

Table 39: Histological differences of primary and permanent dentition

		Deciduous	*Permanent*
1.	Thickness of enamel and dentine	The thickness of enamel and dentine is approximately half that of the permanent teeth	Twice that of the deciduous teeth
2.	Uniformity of enamel thickness	Though the enamel is thinner as compared to the permanent teeth, it has a more or less uniform thickness throughout	Though thicker as compared to deciduous teeth the thickness is not uniform. The enamel is very thin at the cervical region
3.	Direction of enamel rods	The rods in the cervical third do not inclined apically	The rods in the cervical third inclined apically
4.	Neonatal lines	A define neonatal line is present in all the deciduous teeth	Present only in the permanent first molar
5.	Interglobular	A submantle interglobular dentine layer is absent	Present
6.	Direction of dentinal tubules	Tubules are more horizontally placed at the	Just the reverse

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		Deciduous	*Permanent*
		cementoenamel junction so that the incremental pattern which runs at right angles to the dentinal tubules does not present the sharply S shaped appearance seen in permanent teeth	
7.	DEJ	Smooth	Scalloped
8.	Striae of Retzius	Less prominent in number and pigmentation	More prominent in number and darker

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