Operative dentistry

Lec.1

Carious lesion: is tooth demineralization as a result of carious process.

Factors affect the caries process

- Oral hygiene: careful mechanical cleaning of teeth distrupts the biofilm and leaves a clean enamel surface. The cleaning process does not destroy most of oral bacteria, but merely removes them from tooth surface. Large numbers of these bacteria subsequently are removed from the oral cavity during rinsing and swallowing after flossing and brushing, but sufficient numbers remain to recolonize teeth.
- Saliva: it plays a key role as a natural anti-caries agent. Many medications are capable of reducing salivary flow and increasing caries risk (for examples patients receiving therapeutic radiation in the head and neck area will suffer from near-total destruction of the teeth in few months after radiation treatment because the salivary gland becomes fibrotic and produce little or no saliva(xerostomia).Salivary protective mechanisms that maintain the normal oral flora and tooth surface integrity include bacterial clearance, direct antibacterial activity, buffers and remineralization.

- Diet : high frequency exposure of fermentable carbohydrates such as sucrose may be the most important factor in producing caries lesions by starting a series of changes in the local tooth environment that promotes the growth of highly acidogenic bacteria and the acid leads to caries development.

Clinical characteristics of caries

Caries is a product of disequilibrium between demineralization and remineralization. The cavitation of the tooth surface produces a synergistic acceleration of the growth of cariogenic biofilm. This situation results in a rapid and progressive destruction of the tooth structure. When enamel caries penetrates to DEJ,rapid lateral expansion of caries occurs to dentin.

Enamel structure

Enamel is highly calcified and hardest tissue in the body, Crystalline in nature, Insensitive—no nerves, Acid-soluble—will demineralize at a pH of 5.5 and lower, Cannot be renewed, Darkens with age as enamel is lost, Fluoride and saliva can help with remineralization.

Structurally enamel is composed of millions of E. rods or prisms are densely packed and have a wavy course. The structural component of E. prisms are millions of apatite crystals which are tightly packed. Enamel rods appear wavy in section of enamel as they extend from the DEJ to the enamel.

Starting at 1 mm from CEJ the rods run occlusally or anisole at 60 degree inclination and progressively incline approaching the marginal ridges and cusp tips where the rods are parallel to the long axis of the crown. In the cervical region the rods are oriented slightly in apical direction. Understanding enamel orientation is very important in restorative dentistry, because enamel unsupported by underlying dentin is prone to fracture

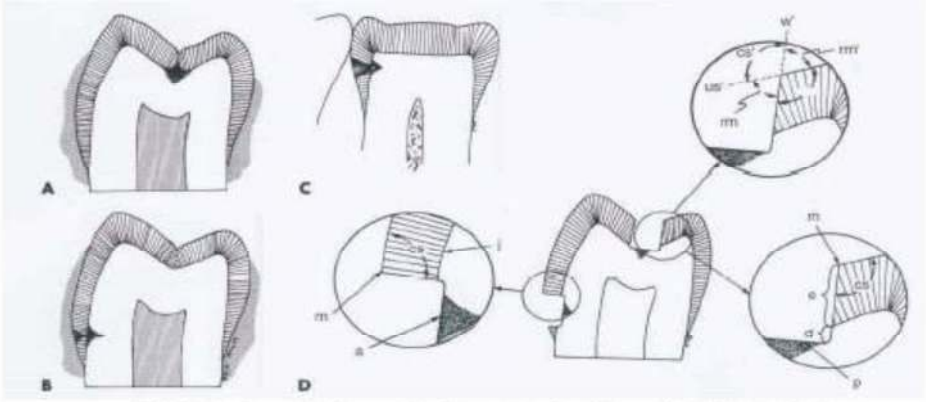


FIG. 6-2 Graphic example of cones of caries (decay) in pit and fissure of tooth (A) and on facial (B) and proximal (C) surfaces when caries has penetrated approximately same depth into dentin. Note differences in loss of enamel on external surfaces. D, Sectional view of initial stage of tooth preparations for lesions in A and B showing cavosurface angle (cs); axial wall (a); pulpal wall (p); enamel wall (e); dentinal wall (d); margin (m); and dentinoenamel junction (j). Note in upper exploded view that cavosurface angle (cs) can be visualized by imaginary projections (w') of the preparation wall and (us') of the unprepared surface contiguous with margin, forming angle cs'. Angles (cs) and (cs') are equal because opposite angles formed at the intersection of two straight lines are equal. Likewise, minimal restorative material angle (rm) is equal to angle (rm').

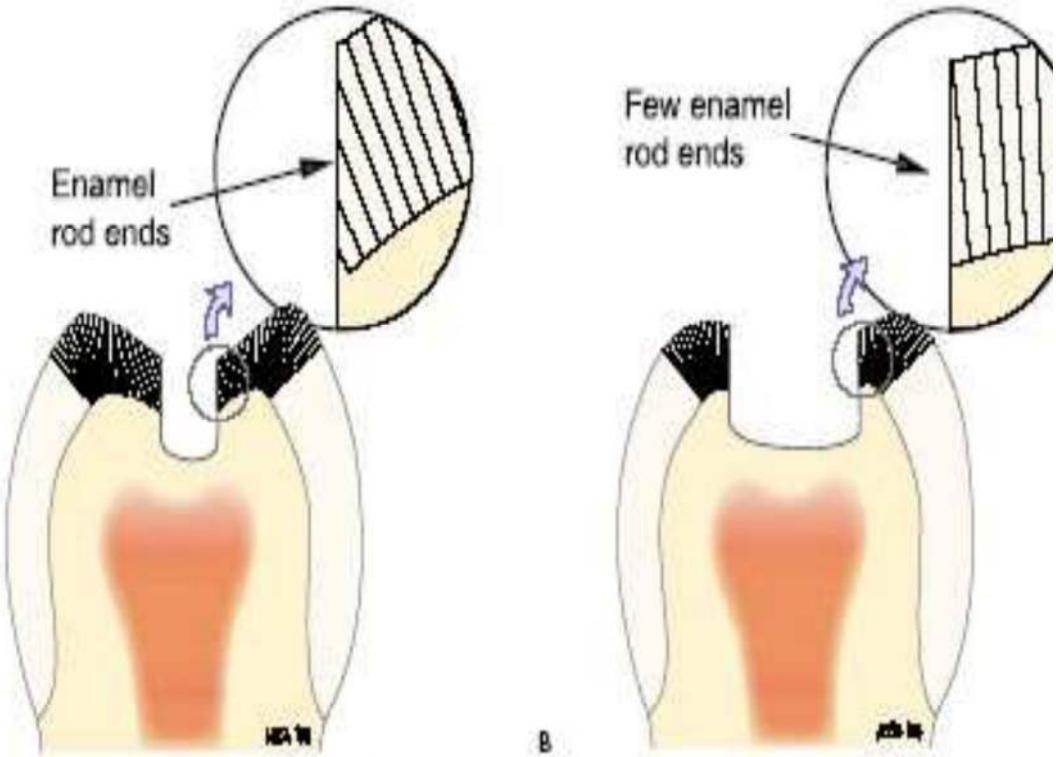


Figure 11–9. *A*, Diagram of proximal view of a Class I composite restoration preparation using a standard amalgam-ty
degree exit. This preparation is appropriate when only small lesions extend into dentin. Note that enamel rod ends are e
owing to the angle of the cusps. *B*, Diagram of a larger Class I restoration on a worn tooth. Note that few rod ends are ex

The earliest lesions is a white spot which is partially or totally disappear visually when the enamel is hydrated in which care must be exercised in distinguishing it from white spots of hypo calcified enamel. The surface texture of non-cavitated lesion is unaltered and is undetectable by tactile examination, But more advanced lesion develops a rough surface that is softer than normal enamel. Softened chalky enamel that can be Chipped away with explorer is a sign of active caries ,but sometimes misuse of an explorer can cause actual cavitation in non- cavitated areas. Non-cavitated enamel can be seen on radiograph as a faint radiolucency limited to superficial enamel.

Permeability

at maturity, enamel is about 90% inorganic hydroxyapatite mineral by volume. E. also contains a small amount of organic matrix & 4% to 12% water, which is contained in the intercrystalline spaces & in a network of micro pores opening to the external surface. The micro pores form a dynamic connection between the oral cavity & the systemic pulpal & dentinal tubule fluids.

Various fluids, ions & low molecular weight substance can diffuse through the semipermeable enamel. Therefore, the dynamics of acid demineralization, caries, remineralization, fluoride uptake are not limited to the surface but are active in three dimensions.

Solubility

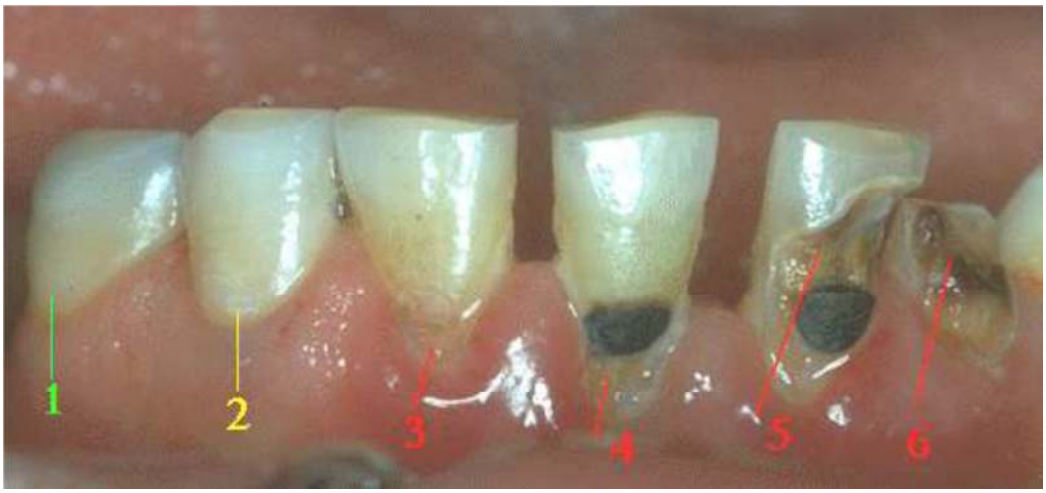
Enamel is soluble when exposed to an acid medium, the solubility of surface enamel decreased when fluorides are present during enamel formation or topically applied to enamel surface. Fluoride additions can affect the chemical and physical properties of the apatite mineral and influence the hardness, chemical reactivity and stability of enamel by lowering acid solubility, decreasing the rate of demineralization and enhancing the rate of remineralization

Clinical Appearance & Diagnosis:

Key diagnostic signs includes:-

1-Color changes associated with demineralization which includes

E. is relatively **translucent;** its **color** is primarily **a function of its thickness** & the color of underlying dentin. The **thickness** is more at the cusps tips & incisal edges & decreases below deep fissures & become thin cervically at the junction with cementum. **Color changes** related to E.demineralization & caries are critical diagnostic observation. Subsurface E. porosity from carious demineralization is manifested clinically by a milky white opacity called **(white spot lesion);** when located on smooth surfaces. In later stages of caries, internal demineralization of E. at the DEJ, subsurface cavitation imparts **a blue or gray color** to the overlying enamel.



2-Cavitations

The dentin affected until enamel breaks away to create a cavity, then a restoration must be place. If untreated the cavitation expands to compromise the structural strength of the crown and microorganisms infiltrate into deep dentin to affect the vitality of the tooth.

3-Wear:depending on many factors such as bruxism,malocclusion,age and diet. Enamel may be completely lost &abraded away so that dentin is exposed. Enamel is as hard as steel, however enamel will wear because of attrition or frictional contact against opposing enamel or harder restorative materials such as porcelain.Heavy occlusal wear demonstrated when rounded cuspal contacts are ground to flat facets.Depending on factors such as bruxism, malocclusion, age and diet; cusps may be completely lost & enamel abraded away so that the dentin is exposed.

4-Faults& Fissures.

A deep fissure formed by incomplete fusion of lobes of cuspal enamel in the developing tooth. The resulting narrow clefts provide protected .area for acidogenic bacteria

Pits & fissures defects are eight times more vulnerable to caries than .are smooth surfaces

Careful observation of enamel surrounding fissures for evidence of demineralization or cavitation is necessary to determine the need for restorative intervention.

5-Cracks:
Pronounced cracks that extend from developmental grooves across marginal ridges to axial walls or from the margins of large restorations may cause cuspal fracture. When this crack extends through dentin or when the patient has pain when chewing؛ the tooth requires a restoration that provide complete cuspal coverage.

Clinical sites for caries initiation
1-Pits&fissures : their shapes contribute to their high susceptibility to caries. The long, narrow fissure prevents adequate biofilm removal. Some pits and fissures end blindly, others open near the dentin, and others penetrate entirely through the enamel. The caries expands as it penetrates into the enamel. The entry site may appear much smaller than the actual lesion making clinical diagnosis difficult.Caries lesions develop from attack on their walls. Progression of the dissolution of the walls of a pit and fissure lesion is similar to that of smooth surface lesion because a wide area of surface attack extends inward, paralleling the enamel rods. A lesion originating in a pit or fissure affects a greater area of DEJ than does a comparable smooth surface lesion.in cross section the caries appear an inverted V with narrow entrance and progressively wider area of involvement closer to DEJ

2-smooth enamel surfaces: that are near the gingiva or under proximal contacts. They have a broad area of origin and a conical, or pointed extension toward the DEJ. The path of ingress of the lesion is roughly parallel to the long axis of enamel rods in the region. A cross section shows a V-shape with wide origin and apex toward DEJ. After caries penetrates the DEJ, softening of dentin spreads rapidly laterally and pulpally.

3-root surface: it has less well-defined margins, tend to be U- shaped in cross section and progress more rapidly because of the lack of protection from enamel covering. In recent years, the prevalence of root

caries has increased significantly because of the increasing number of older persons who retain more teeth, experience gingival recession.