Conservative dentistry

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Biologic Consideration of Enamel and its Clinical Significance in Practice of Operative Dentistry

Morphologic & Histologic Review:

Enamel is formed by cells called ameloblast. Enamel provides a hard, durable shape for the functions of teeth & a protective cap for the vital tissues of dentin & pulp. Both color & form contribute to the esthetic appearance of enamel. Enamel is incapable of repairing itself once destroyed because the ameloblast cell degenerates after formation of enamel rod.

Permeability: at maturity, enamel is about 90% inorganic hydroxyapatite mineral by volume. Enamel also contains a small amount of organic matrix & 4% to 12% water which is contained in the intercrystalline spaces & in a network of micropores opening to the external surface. The micropores 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.

Enamel is soluble when exposed to an acid medium, the solubility of surface enamel is decreased when fluorides are present during enamel formation or are 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 and diagnosis:

The dentist must pay close attention to the surface characteristics of enamel for evidence of pathologic or traumatic conditions.

Key diagnostic sings include:

1. Color changes associated with demineralization:

Enamel 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.** Subsurface enamel 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 enamel at the DEJ, subsurface cavitation imparts a blue or gray color to the overlying enamel.

2.Cavitation: the dentin is affected until enamel breaks away to create **a** cavity, **a restoration must then be placed.** If untreated the cavitation expands to compromise the structural strength of the crown & microorganisms infiltrate into deep dentin to affect the vitality of the tooth. When the carious lesion extends gingival to CEJ as in root caries, isolation, access & gingival tissue response complicate the restorative procedure..

3. Wear: 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 is demonstrated when rounded cuspal contacts are ground to flat facets. Depending on factors such as bruxism, malocclusion, age & diet cusps may be completely lost & enamel abraded away so that dentin is exposed. So cavity outline form should be designed so that the margins of restorative materials avoid critical high stress areas of occlusal contact.

4. Faults & fissures: a deep fissure is 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 cavitations 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.

Crystal Structure & Enamel Rods:

Structurally enamel is composed of millions of enamel rods or prisms. The rods are densely packed & have a wavy course & each extends from the DEJ to the external surface of the tooth.

Enamel is the hardest substance of **human** body, enamel is very brittle, and so it requires a base of dentin to withstand the masticatory stress. Enamel rods that fail to possess a dentin base because of caries or improper cavity design are easily fractured away from neighboring rods.

The structural components of enamel prisms are millions of small elongated apatite crystal which are tightly packed in a distinct pattern of orientation that gives strength & structural identity to the enamel prisms. An organic matrix or prism sheath surrounds individual crystal. The spacing & orientation of the crystals & the amount of organic matrix make the enamel rod boundary & the central core differentially soluble when exposed for a short time to weak acids.

The acid- treated enamel surface has an irregular and pitted surface with numerous microscopic undercuts, the etched enamel has a higher surface energy, so resin monomer flows into & adheres to the etched depressions to polymerize & form retentive resin tags. Because there are (30,000) to (40,000) E. rods/ mm^2 & acid etch penetration increases the bondable surface area to (10) to (20) folds, micromechanical bonding of resin restorative materials to E. is significant.

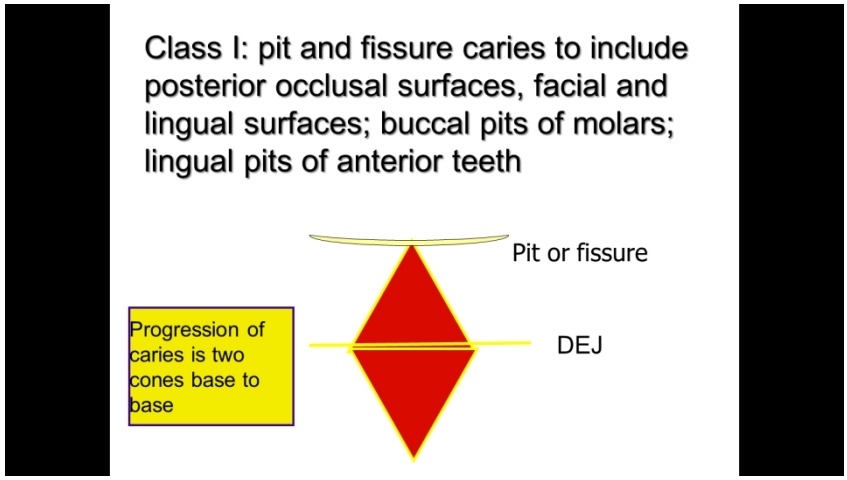
Acid-etch modification of E. for restoration retention provides conservative, reliable, alternative to traditional surgical methods of tooth preparation& restorations. (retentive grooves, pins, extension for prevention...).

Loss of enamel rods that form the cavity wall of cavomargin of dental restorations creates a gap defect, leakage of bacteria & their products that may lead to secondary caries. Therefore a basic principle of cavity wall preparation is to bevel or parallel the direction of E. rods & avoid undercutting them.

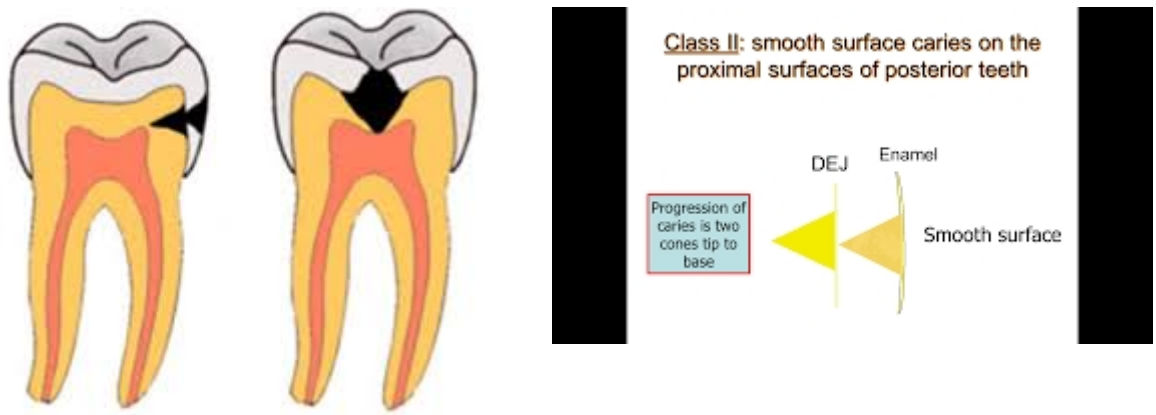
Clinical sites for caries initiation:

There are three distinctly different clinical sites on teeth where cariogenic plaque may originate:

1. Pits & fissures of enamel: this is the most susceptible site. The path of carious lesion is roughly parallel to the long axis of enamel rods; the entry site may appear much smaller than the actual lesion making clinical diagnosis difficult. In cross section the appearance of the lesion is an inverted V with a wide area of involvement at DEJ. Pit & fissure caries are most commonly found on the occlusal surface of posterior teeth, lingual surface of maxillary anterior teeth & buccal & lingual pits of molars.



2. Smooth enamel surface: plaque usually develops on the smooth surface that is near the gingival or under proximal contacts; the path of ingress of the lesion is roughly parallel to the lone axis of the enamel rods in the region. Across section shows a V shape with wide area of origin & the apex of the V directed towards the DEJ. It includes proximal caries & lesions on other smooth surfaces. After caries extension to the DEJ there is both lateral spread along the junction & extension pulpally. A cross section of caries in dentin is always V shaped with its base at the DEJ and the apex directed pulpally.



3- Root surface: the cementum covering the root surface is extremely thin and provides little resistance to caries attack Root lesions have less well defined margins tend to be U-shaped in cross section.