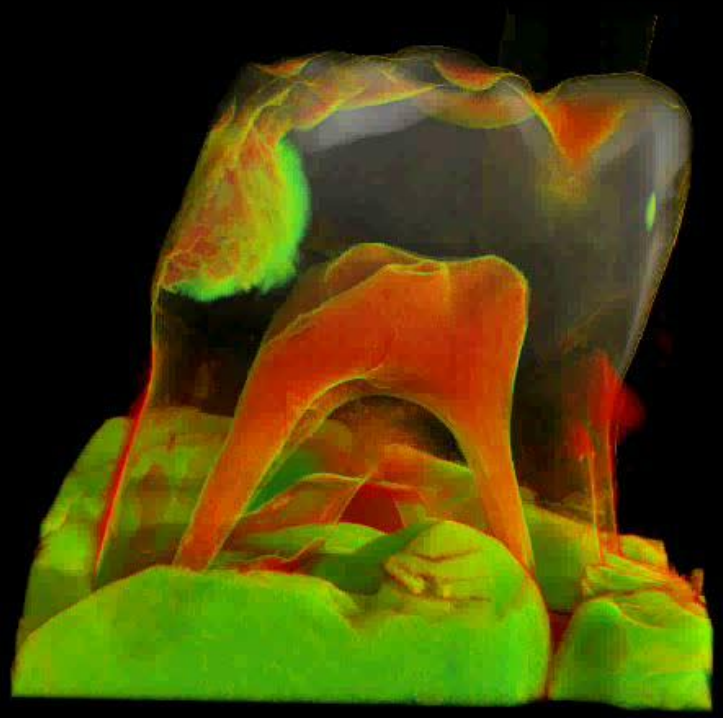

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Media centre

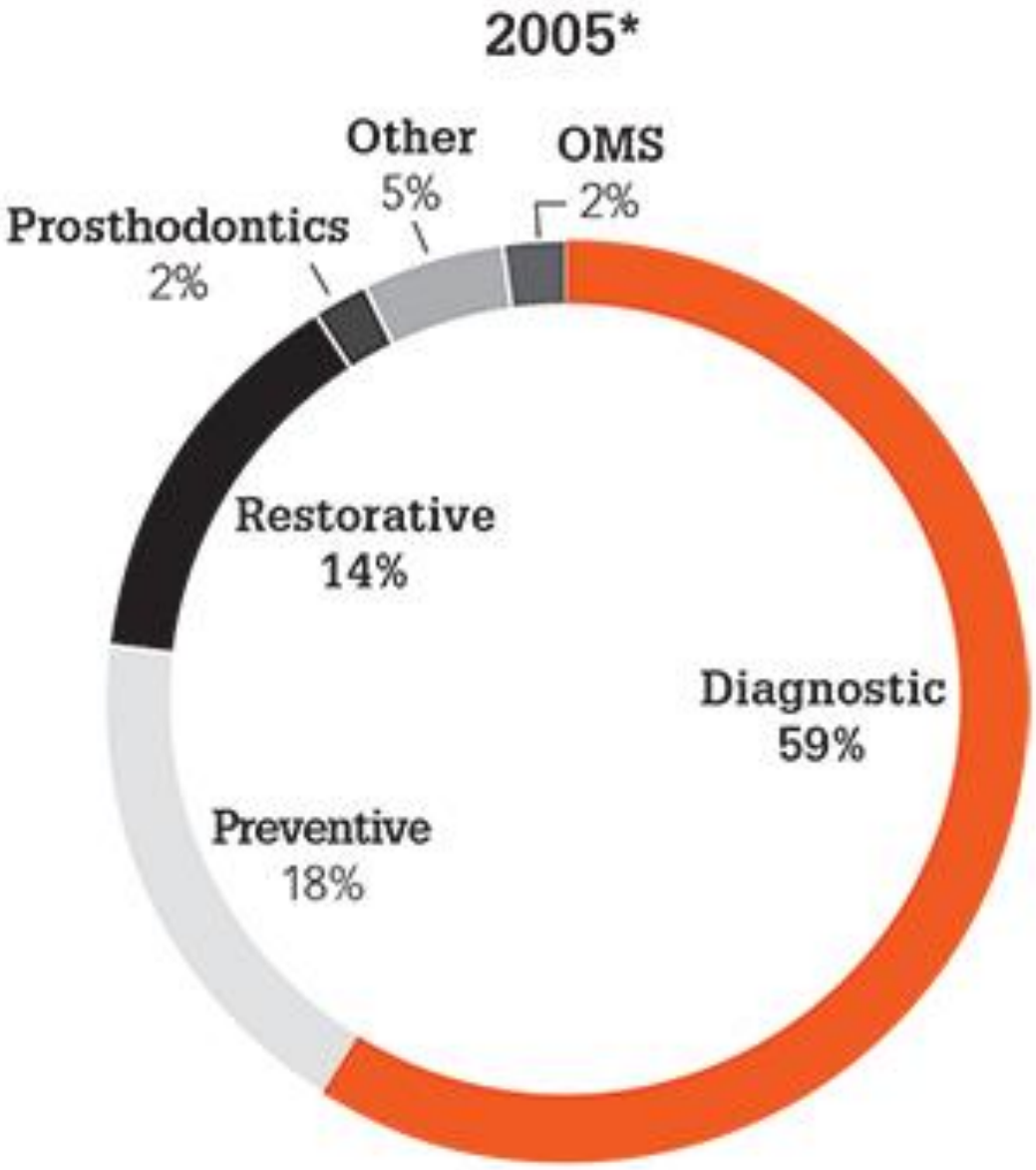
Oral health

Fact sheet N°318
April 2012

Key facts

- Worldwide, 60–90% of school children and nearly 100% of adults have dental cavities.
- Dental cavities can be prevented by maintaining a constant low level of fluoride in the oral cavity.
- Severe periodontal (gum) disease, which may result in tooth loss, is found in 15–20% of middle-aged (35-44 years) adults.

Procedures completed in the dental office



* General dentists

DENTAL CARIES

Contents

- Introduction
- Dental caries
  - Definition
  - Classification
  - Etiology
  - Pathogenesis
- Theories
  - The legends of worms
  - Humors
  - Vital theory
  - Chemical theory

Contents

-Parasitic or septic theory

-Chemoparasitic theory

-Proteolytic theory

-Proteolysis-chelation theory

-Phosphate sequestration theory

- Role of Diet in Dental Caries
- Prevention

-Wait and watch approach

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- Sealants

- Lasers

- Antimicrobials

- Xylitol
- Replacement therapy
- Diet modification
- Air abrasion
- Caries infiltration

- Conclusions
- References

Introduction

Dental caries

Definition:

Dental caries is an irreversible microbial disease of the calcified tissues of the teeth, characterized by demineralization of inorganic portion and destruction of the organic substance of the tooth, which often leads to cavitation.

(Shafer, fifth edition)

DENTAL CARIES

Cariology

Discipline of the study of dental caries

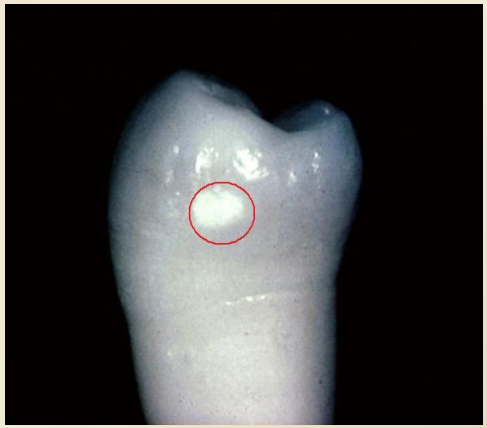
Dental Caries

disease that is characterised by the localized destruction of susceptible dental hard tissue by acidic by-products from bacterial fermentation of dietary carbohydrates

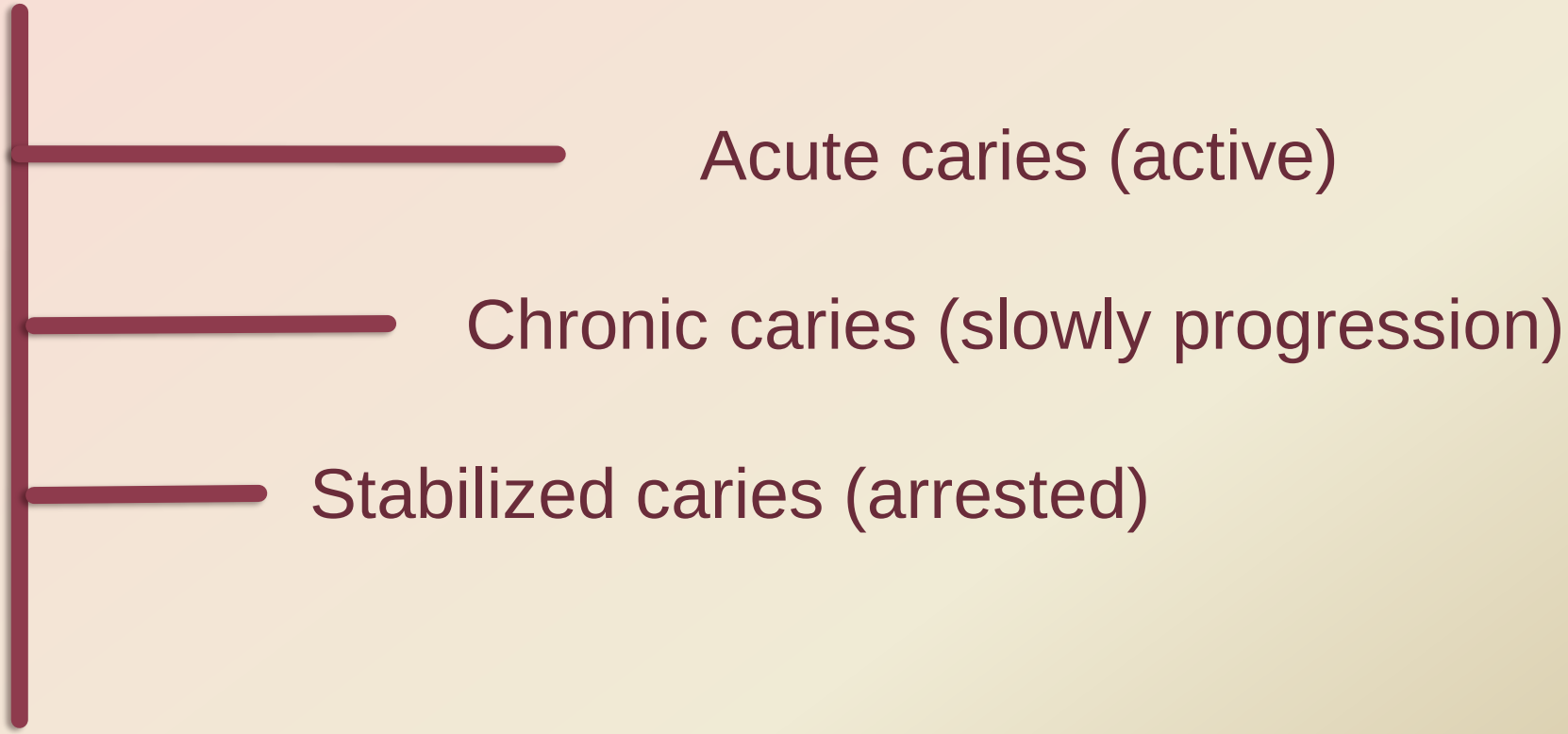
Classification

According to the stage of lesion progression

- Non cavitated lesion (incipient lesion)
- Cavitated lesion



According to the severity of the disease



- Acute dental caries:

Caries which runs a rapid clinical course and results in early pulp involvement by the carious process. It occurs most frequently in children and young adults.

- Chronic dental caries:

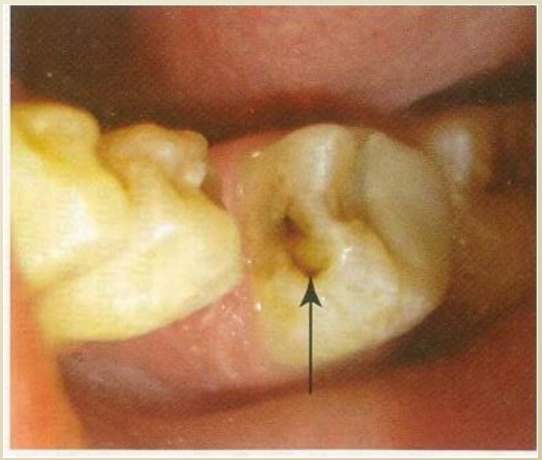
Caries that progresses slowly and tends to involve the pulp much later than the acute caries. It is most common in adults.

Arrested caries

- Caries which becomes static and does not show any tendency for further progression.

Forms of arrested caries:

- Large open cavities - Eburnation of dentin
- In proximal surfaces of teeth in which the adjacent tooth has been extracted.
- Caries arrest following application of stannous fluoride.



According to clinical manifestation

- White spot lesion *macula caroisa*
- Superficial caries *caries superficialis*
- Medium caries *caries media*
- Deep caries *caries profunda*
- Secondary caries *caries secundaria*
- Deep complicated caries caries profunda complicata

According to anatomical depth of the defect

- Enamel caries
  - Incipient caries
  - Linear enamel caries (odontoclasia)
- Dentin caries
- Cementum caries

Linear enamel caries

Definition:

- A typical form of dental caries that has been observed in the primary dentition of children, in Latin America and Asian countries.

- Lesions occur on labial surfaces of anterior teeth, in the region of neonatal line.

Synonym:

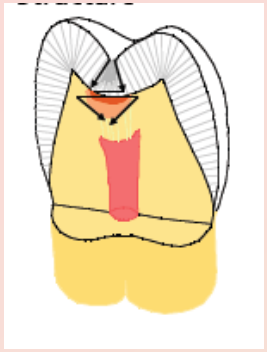
- Odontoclasia (Linear enamel caries in Far East)

According to location of the lesion

- Occlusal surfaces (pit and fissure carie

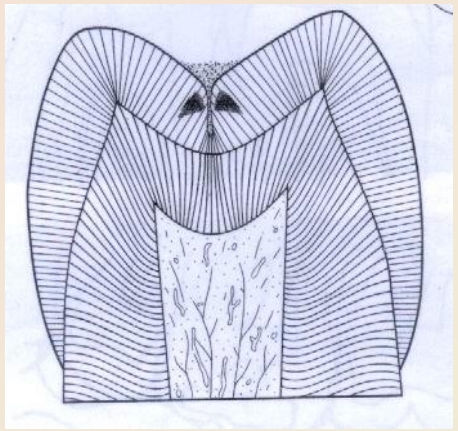
- Smooth surfaces
  - Buccal and lingual surface caries
  - Interproximal caries

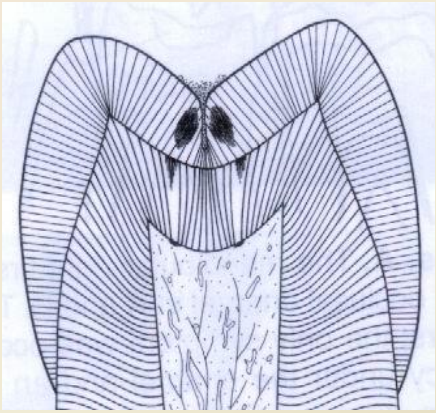
- Root caries

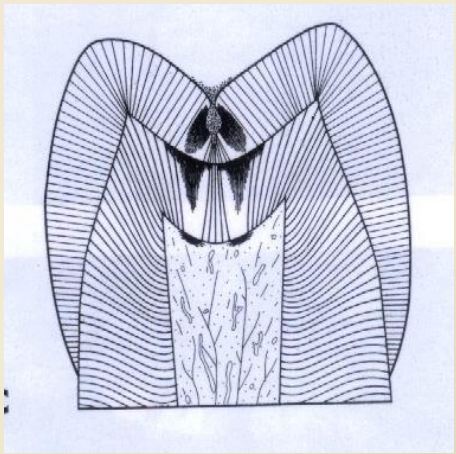


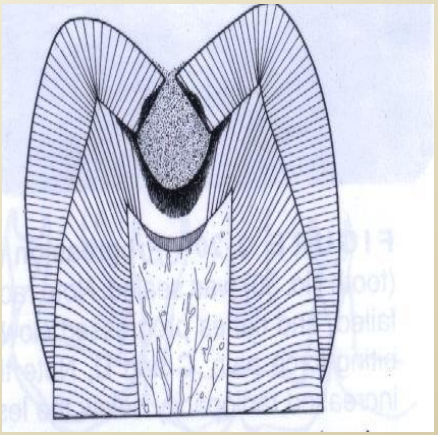
Pits and Fissures

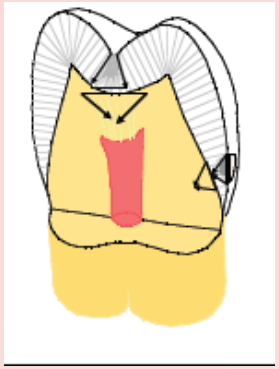
- Lateral walls affected
- Follow enamel rods
- Narrow entrance
- Wide DEJ involvement
- Inverted V







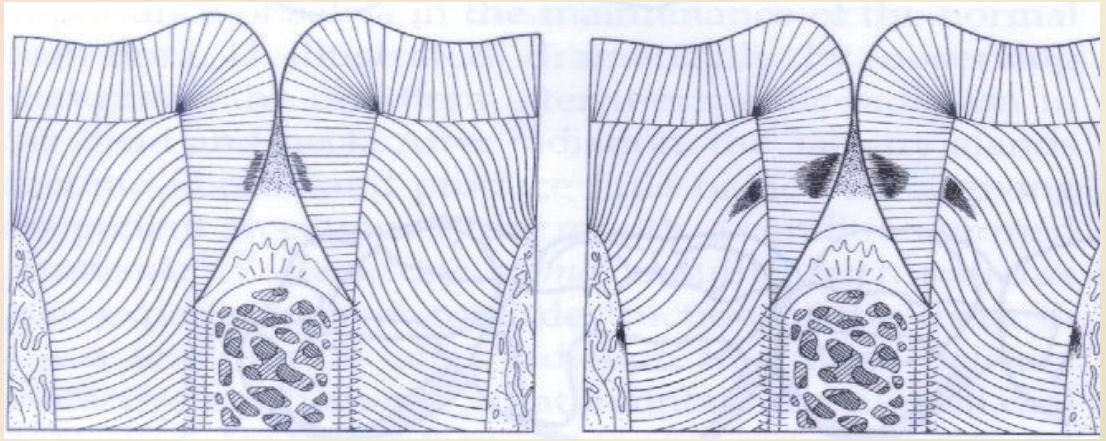


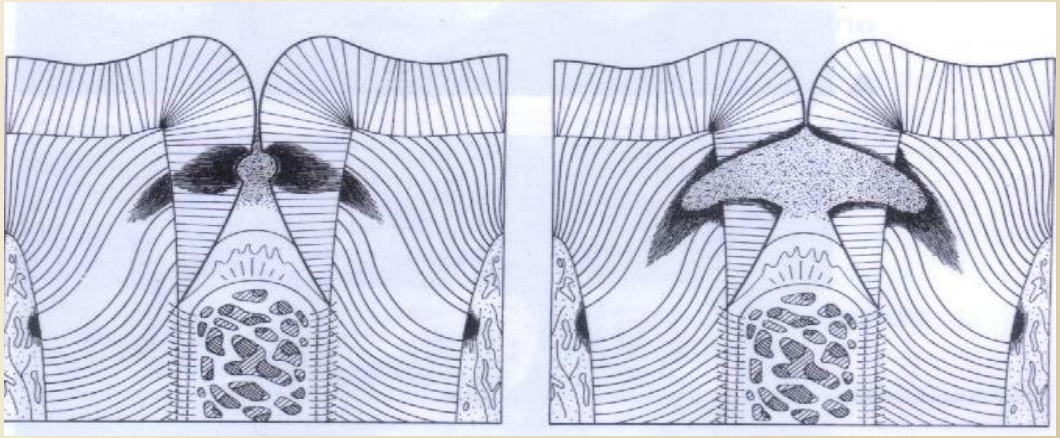


Smooth surface caries

- Wide area surface involved
- Narrow area as it approaches DEJ
- V shaped

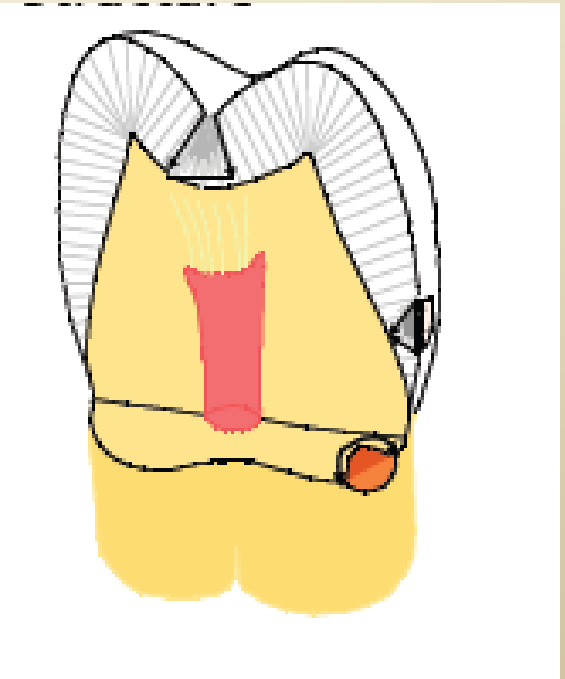
  Apex towards DEJ





Root caries

- U shaped
- More Rapid progress
- Aging population



Based on the initiation

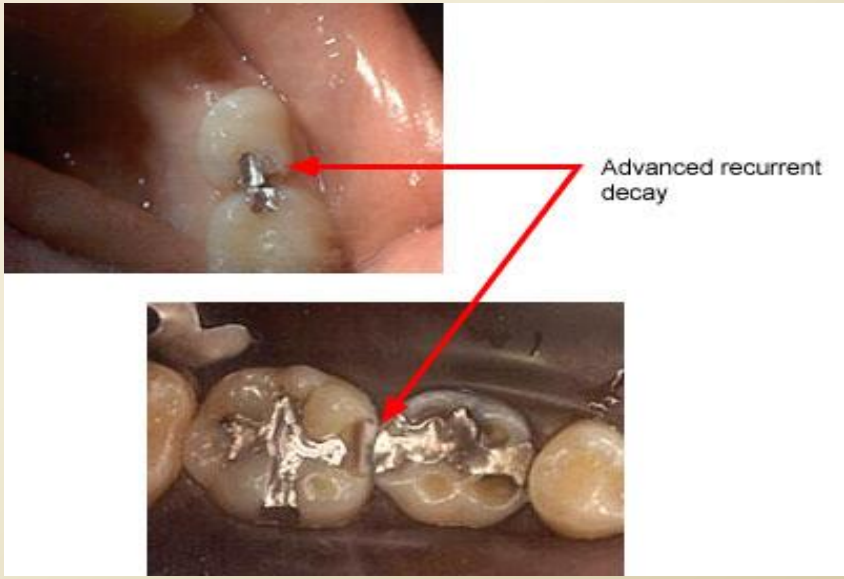
Initial/ primary

Recurrent/ secondary

Recurrent caries

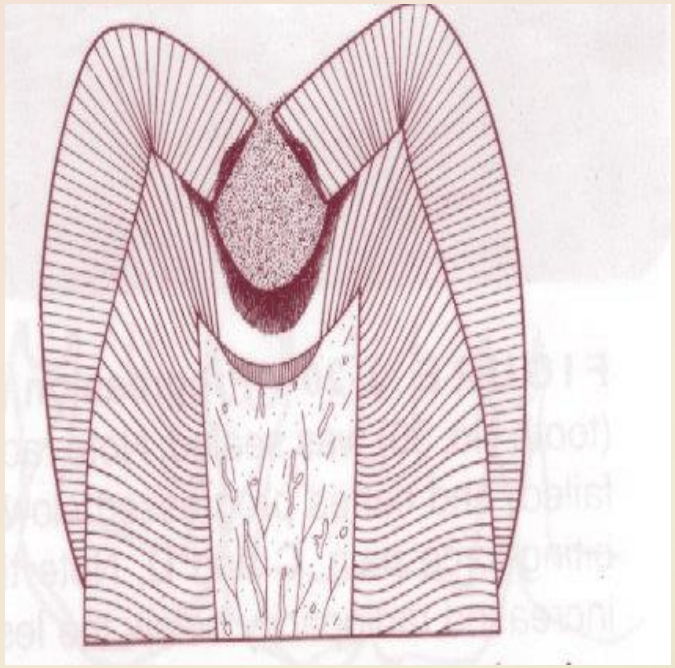
- Type of caries which occurs in the immediate vicinity of a restoration.

- Causes
  - Inadequate extension of original restoration
  - Poor adaptation of restoration

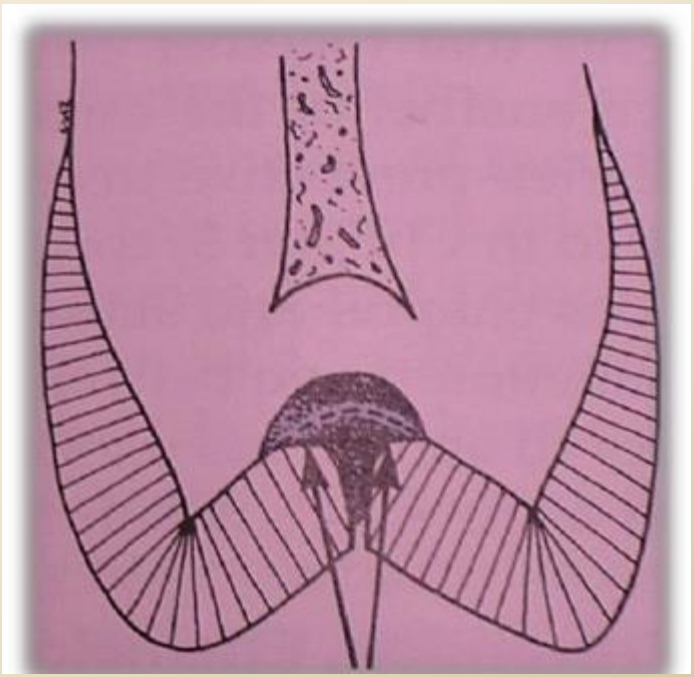


Based on the pathway of caries spread

Forward caries



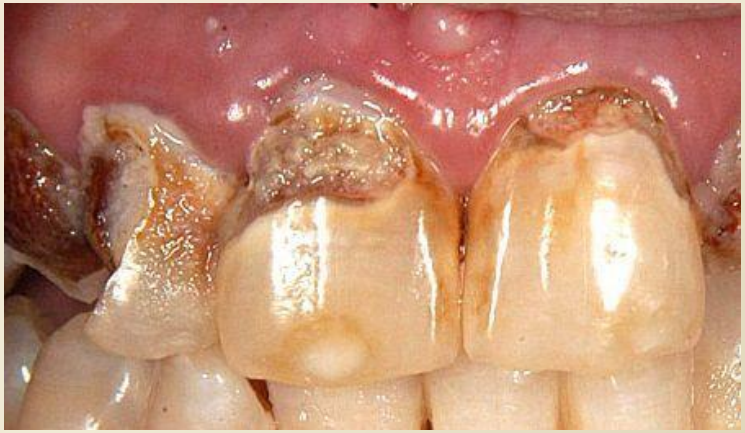
Backward caries



Based on the number of tooth surfaces involved

Simple

Compound



Complex

Based on chronology

- Early childhood caries
- Adolescent caries
- Adult caries

Early childhood caries

Definition:

Presence of at least **one decayed** (non-cavitated or cavitated lesion), **missing** (due to caries) or **filled** surface in any primary tooth (dmft >0) in a child aged **71 months or less** (before the age of 6 years)

(AAPD, 2005)

- **Infancy or Soother caries** - Rapidly progressing type of dental caries affecting primary teeth of children, usually during first two years of life.

- **Adolescent caries** – Acute exacerbation in caries rate at 11-18 years of age.

Black' s Classification

Class 1 lesion

Class 2 lesion

Class 3 lesion

Class 4 lesion

Class 5 lesion

Class 6 lesion

WHO classification

According to this classification, the shape and depth of the carious lesions can be scored on a four point scale

D1 – clinically detectable enamel lesions with intact surfaces (non cavitated)

D2 – clinically detectable cavities limited to the enamel

D3 – clinically detectable lesions in the dentin (with and without cavitation of dentin)

D4 – lesions into pulp

Other types of caries

- Rampant caries
- Radiation caries
- Hidden caries

Rampant caries

Definition:

Suddenly appearing, widespread, rapidly burrowing type of dental caries resulting in early pulpal involvement and affecting those teeth that are usually regarded as immune to ordinary decay.

(Massler, 1945)

Synonyms:

Early childhood caries

Maternally derived streptococcus mutans disease

Radiation caries

- Development of rampant caries in patients undergoing radiation therapy in the head and neck region is radiation caries.

Consequences of irradiation:

- Xerostomia
- Increase in viscosity of saliva
- Low pH

} Causes of radiation caries

Hidden caries

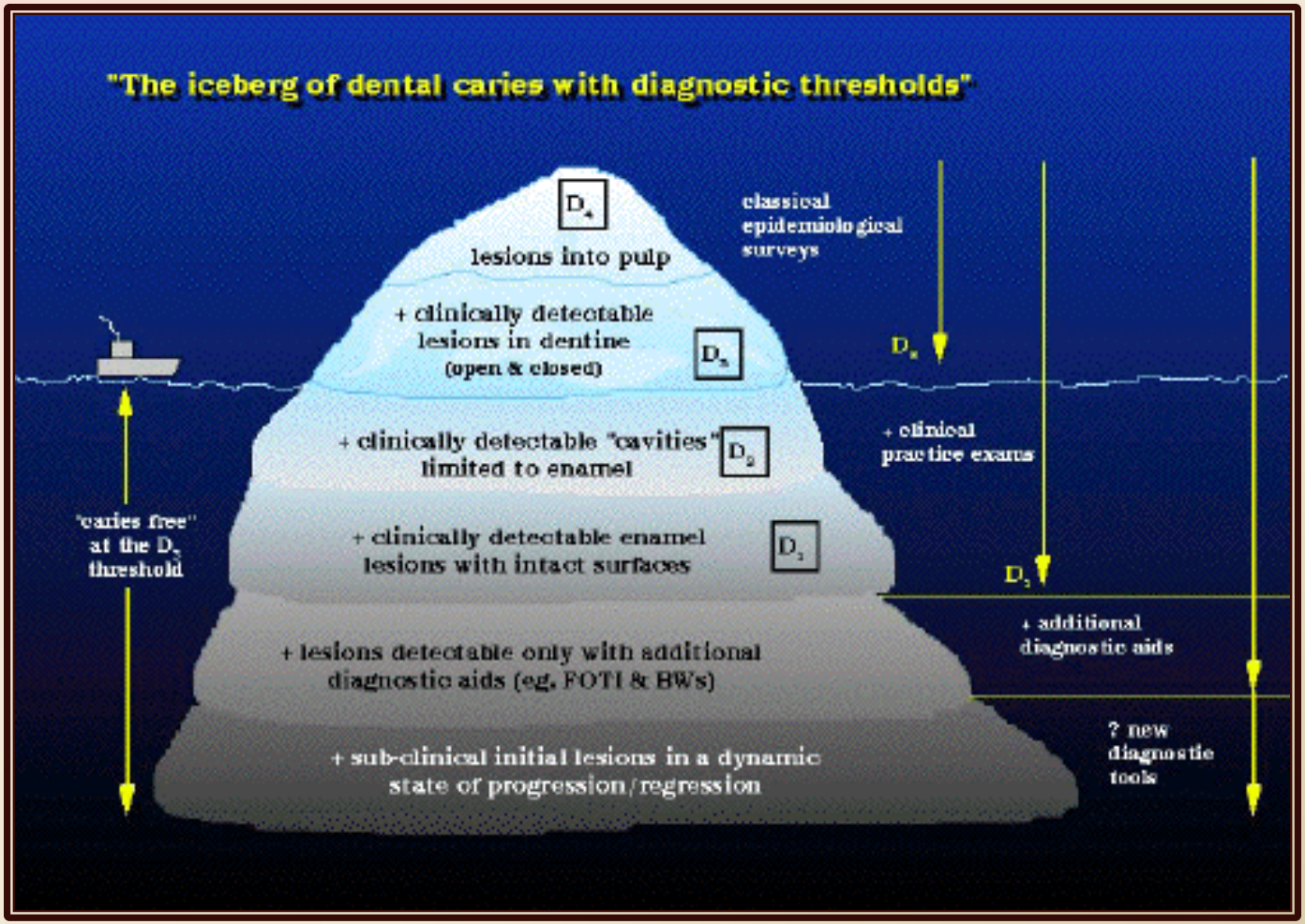
Synonyms:

Covert caries
Occult caries
Fluoride syndrome, fluoride bombs

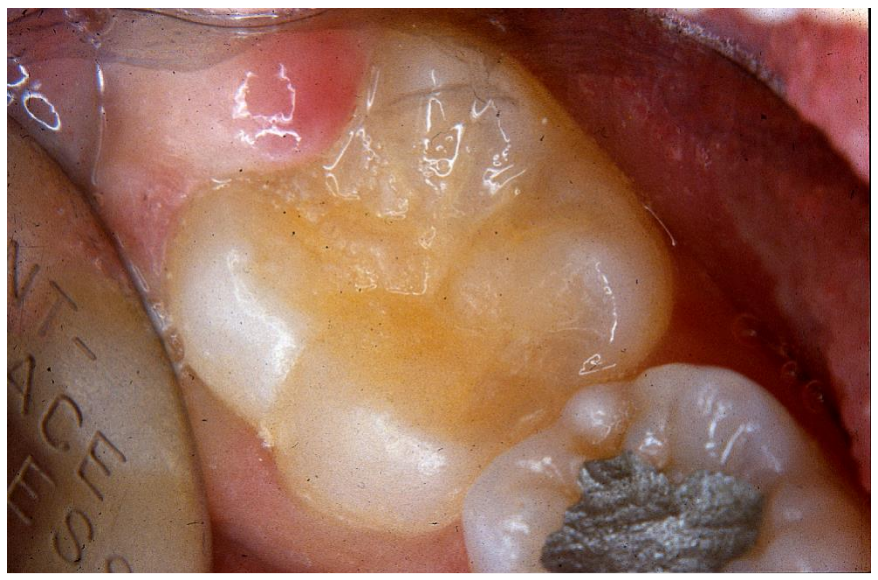
Definition:

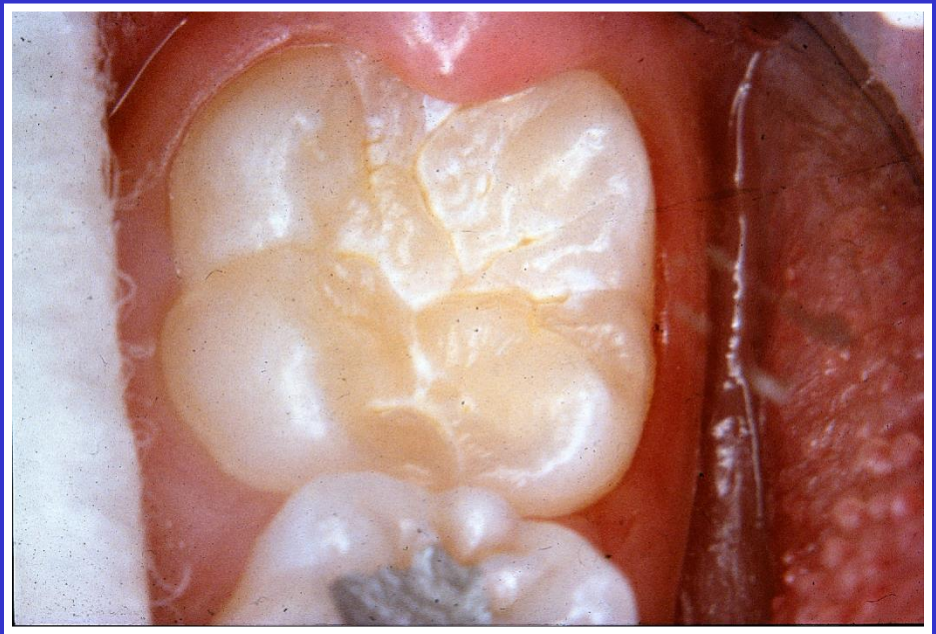
Caries lesions in dentin that are missed on a visual examination but are large enough to be detected radiographically.

A NEW CLASSIFICATION



In the ICDAS-system caries is scored after cleaning of the teeth



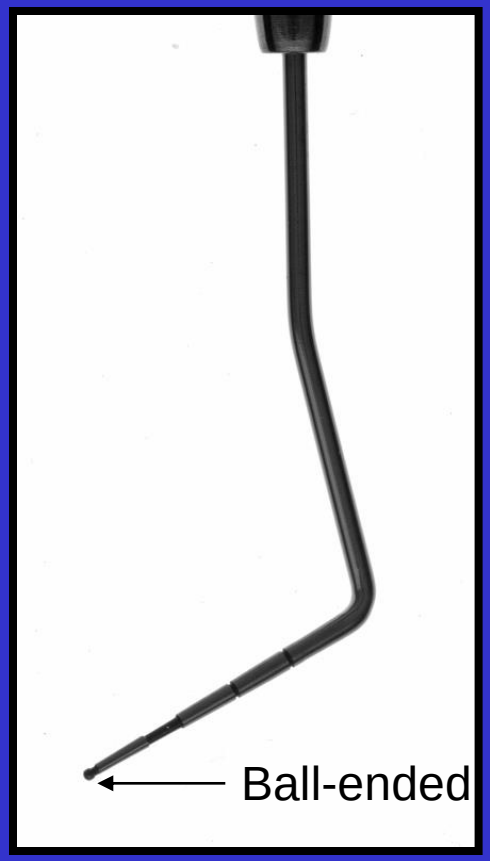


Otherwise caries will be underscored

Carvalho et al., 1989

Probes?

In the ICDAS-system perio-probes are used to feel with



Explorers are not recommended as they may produce traumatic defects.

Ekstrand et al., 1987

1.The next slides illustrate the ICDAS-detection criteria

A description of the criteria is given in the protocol which can be found on http://www.icdas.org/

The classification system operates with 7 scores.

- Score 0=sound
- Scores 1-2 involve different stages of white or brown spot lesions
- Score 3= Lesions with microcavities
- Score 4= Shadowed lesions
- Scores 5-6 involve cavitated (into the dentine) lesions

Detection system: Each of the 7 scores are illustrated with an example

SOUND	OPACITY with air-drying: WHITE, BROWN	OPACITY without air-drying: WHITE, BROWN	SURFACE INTEGRITY LOSS	UNDERLYING GREY SHADOW	DISTINCT CAVITY	EXTENSIVE CAVITY
Score 0	Scores 1	Scores 2	Score 3	Score 4	Score 5	Score 6

Sound

Ekstrand et al., (1997) modified by ICDAS (Ann Arbor), 2002 and again in 2004 (Baltimore)

The detection system

- The scores were devised related to the fact that each scoring represents a severity stage (depth). Members of the ICDAS group demonstrated a close relationship between the scores and histological depth
- The histological classification system used are based on the criteria on the next slide

The histological classification system (Ekstrand et al., 1997)

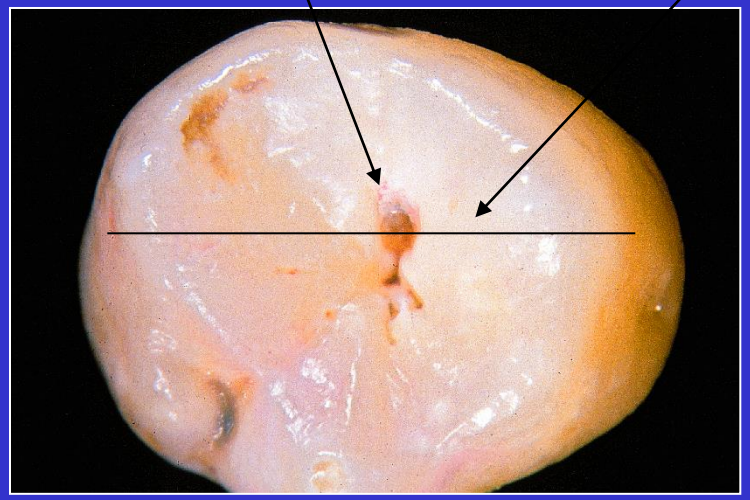
- 0= no enamel demineralisation or a narrow surface zone of opacity (edge phenomenon)
- 1= enamel demineralisation limited to the outer 50% of the enamel layer
- 2=Demineralisation involving between 50% of the enamel and 1/3 of the dentine
- 3= Demineralisation involving the middle 1/3 of the dentine
- 4=Demineralisation involving the inner 1/3 of the dentine

Method for examining relationship between visual appearence (ICDAS) and histological changes (Step 1-3)

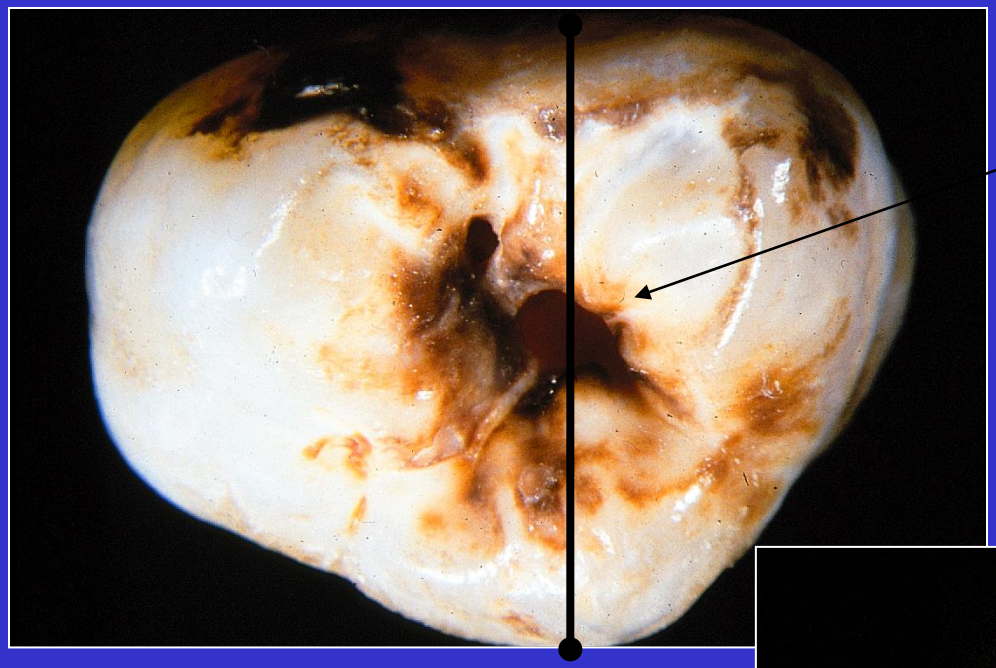
1. step
Visual appearance
ICDAS score 3= microcavity

2. step
Sectioning

3. step
Histological evaluation

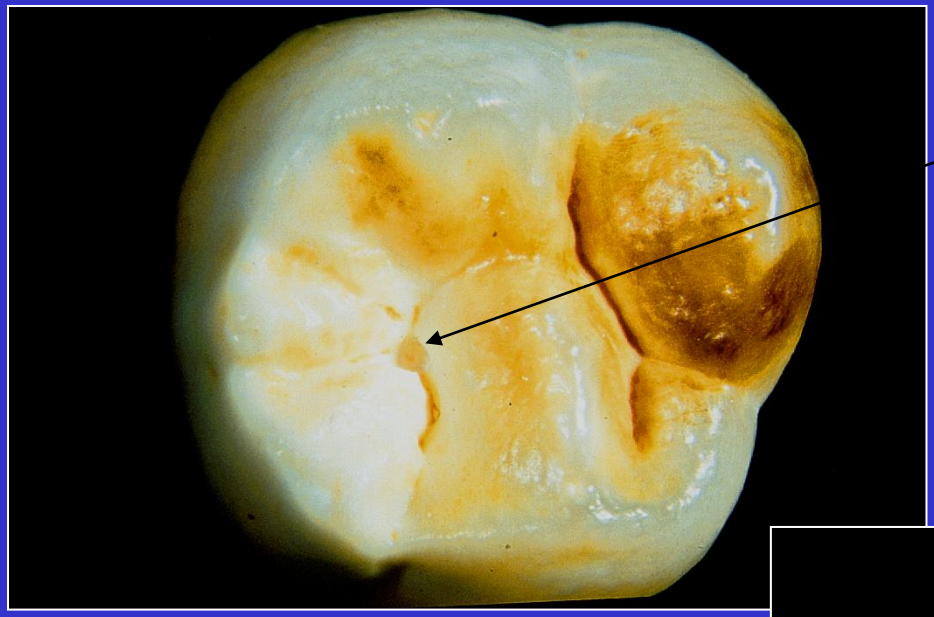


0= no demineralization
1= demineralization limited to outer ½ enamel
2= demineralization in inner ½ enamel to outer 1/3 dentine
3= demineralization middle 1/3 of the dentine
4= demineralization inner 1/3 of the dentine



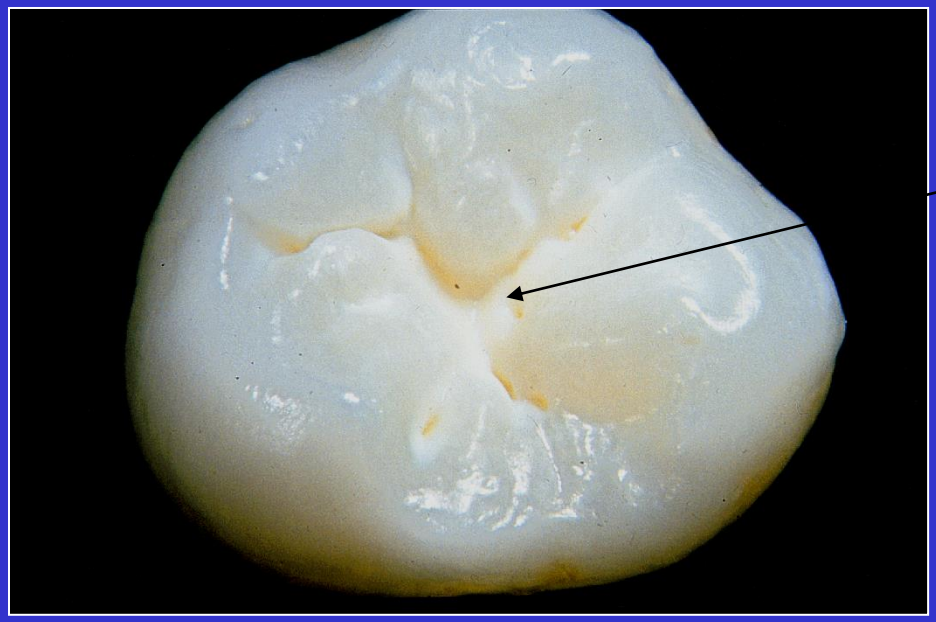
ICDAS score 5

Histological score 4



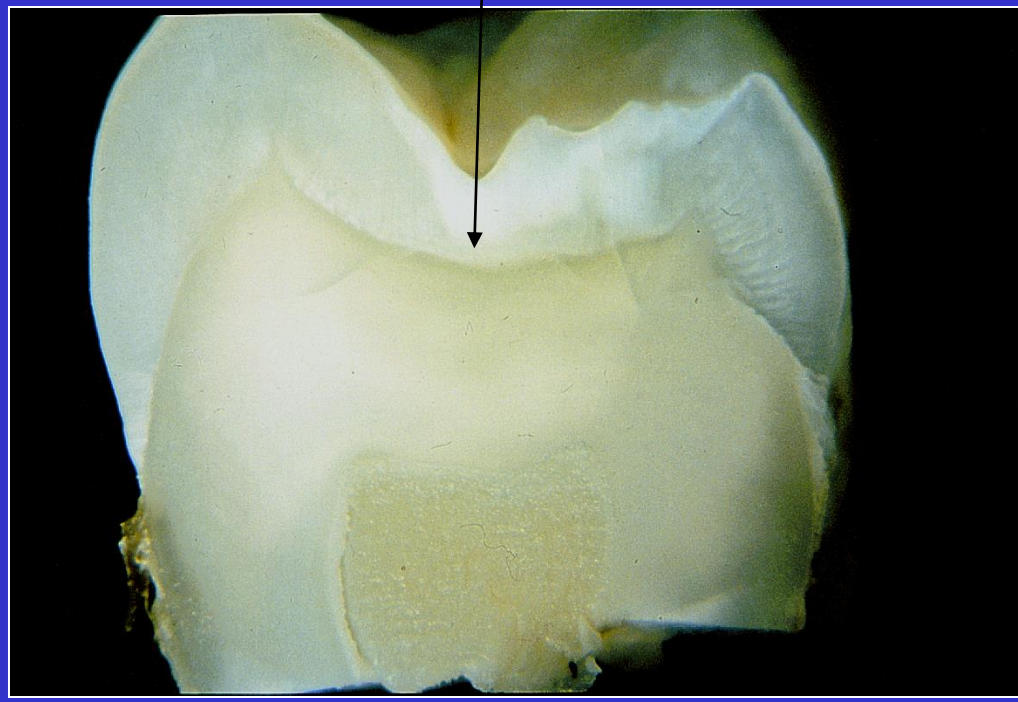
ICDAS score 3

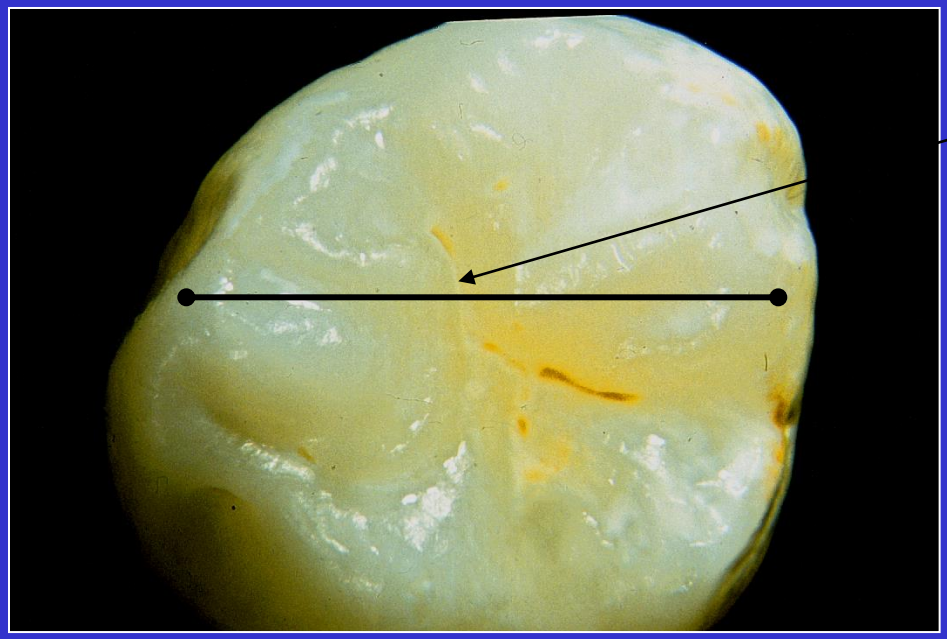
Histological score 3



ICDAS score 2

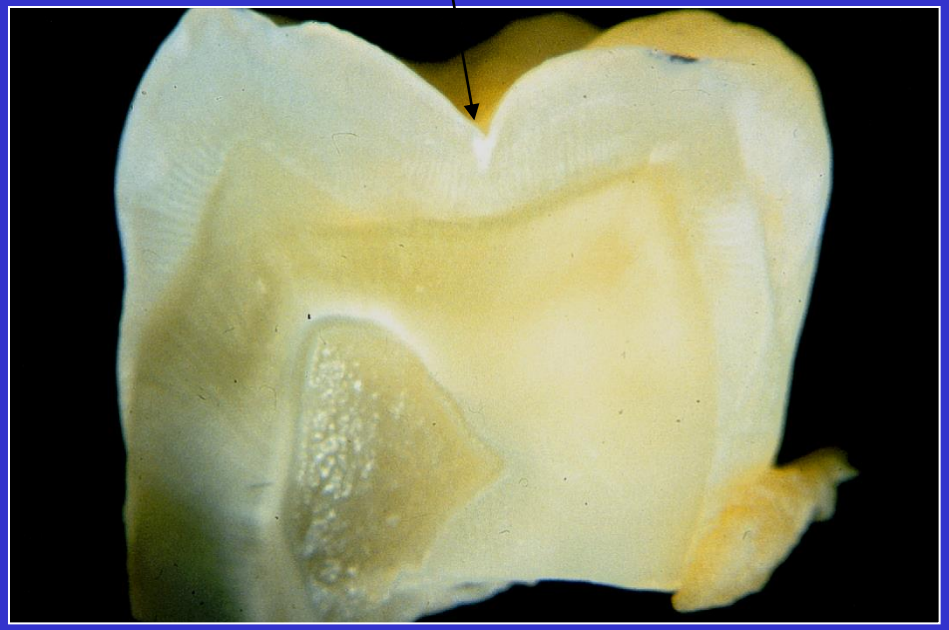
Histological score 2

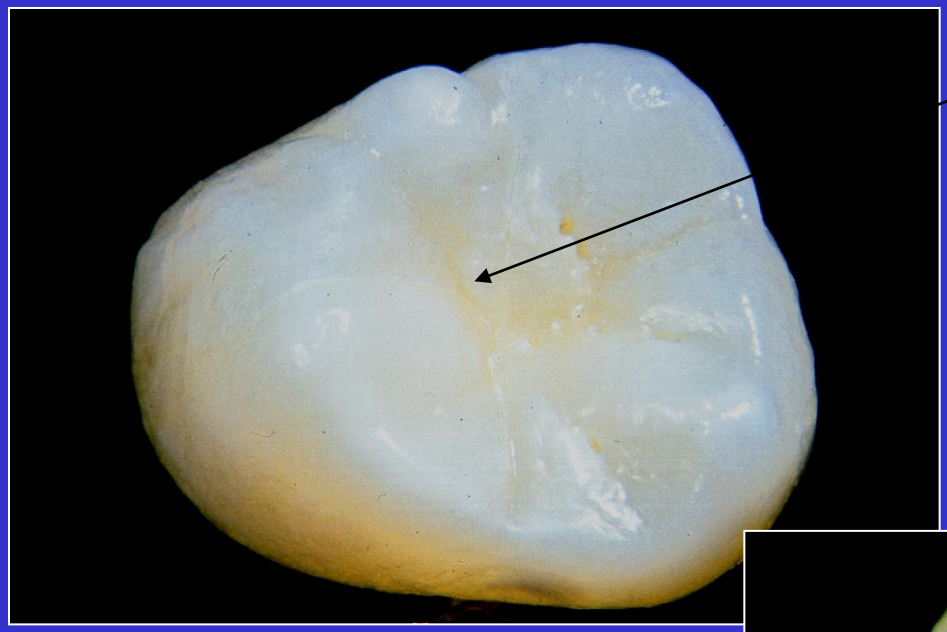




ICDAS score 1

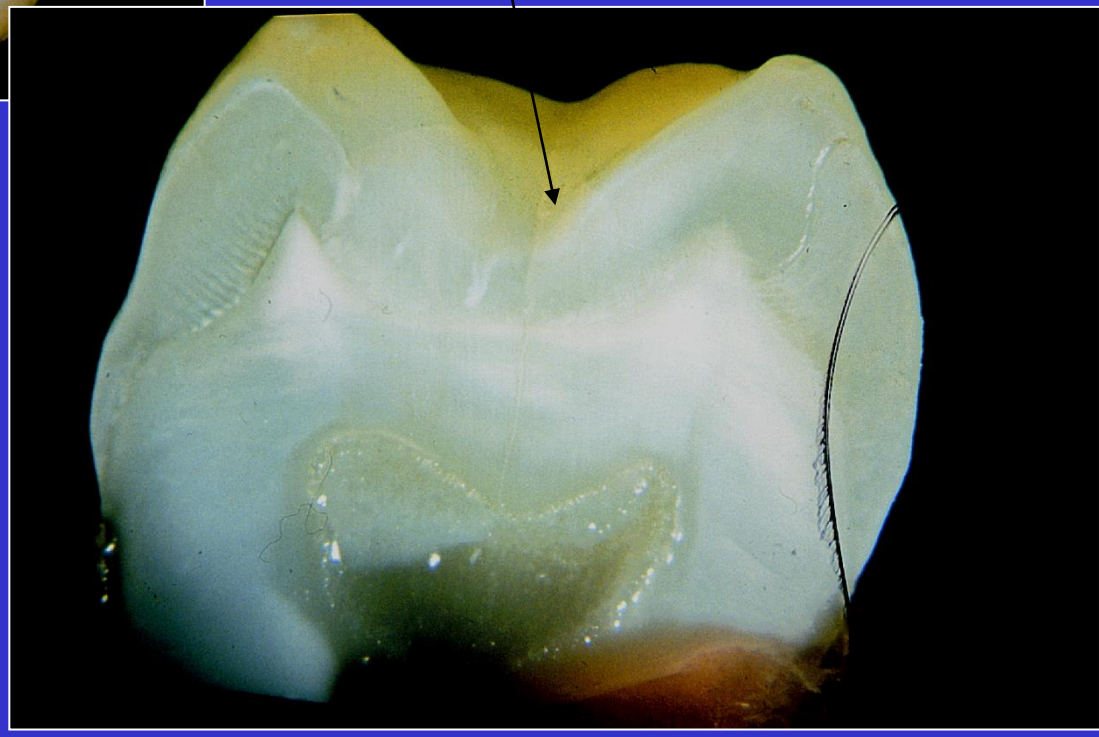
Histological score 1





ICDAS score 0

Histological score 0

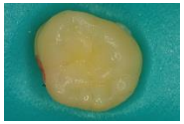


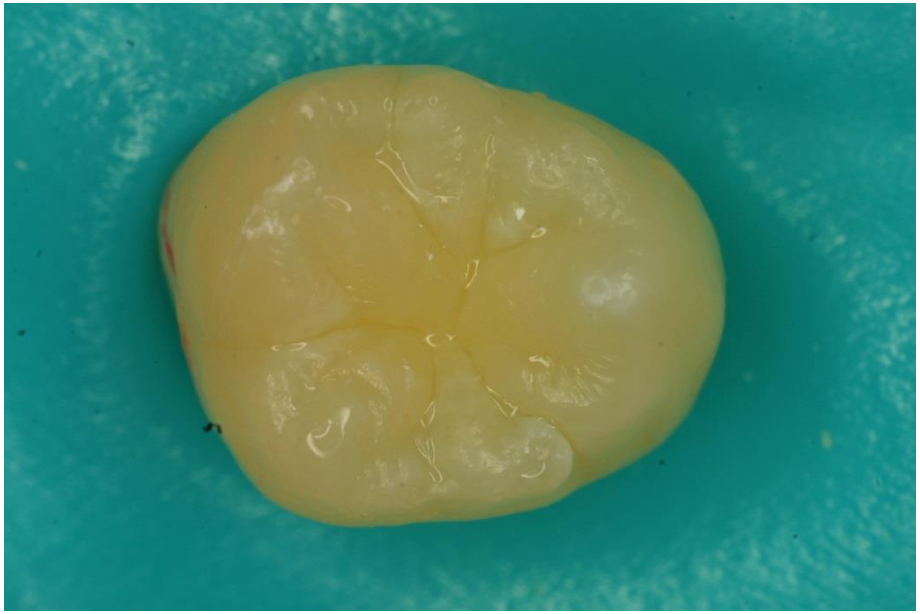
Always

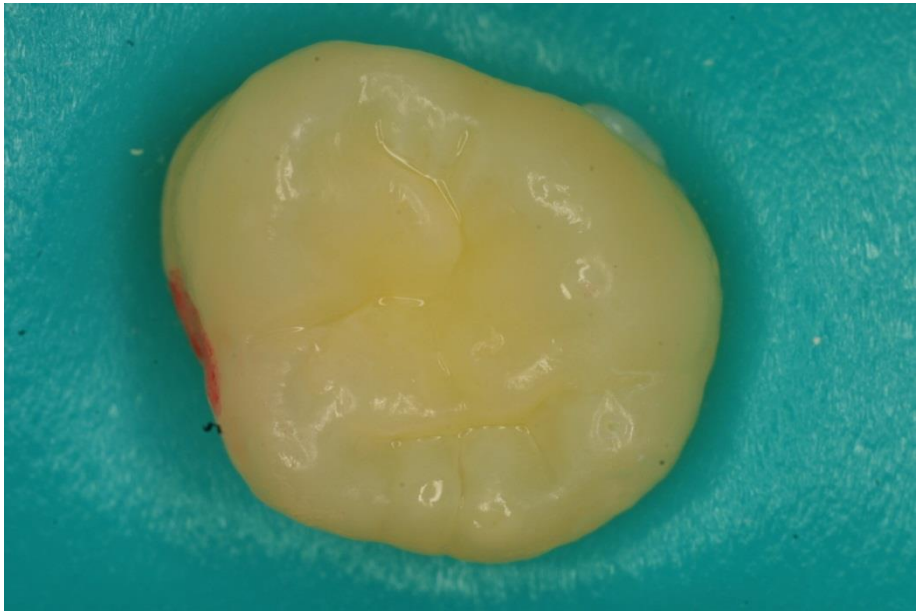
- In doubt score low

Score 0: Nothing

Magnification 1X

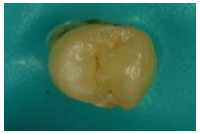


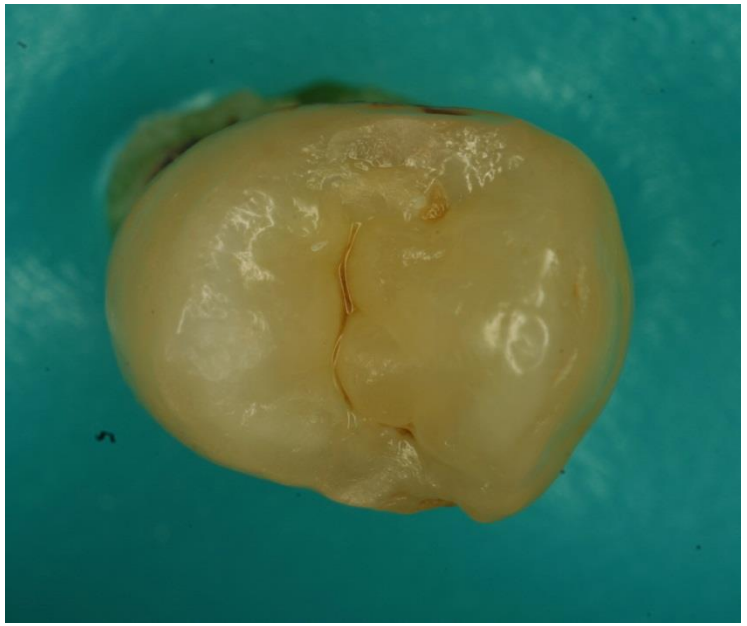




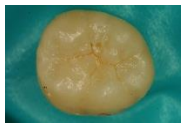
Magnification 5X

Score 0:Brown discoloration confined to fissure and generalized

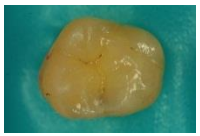




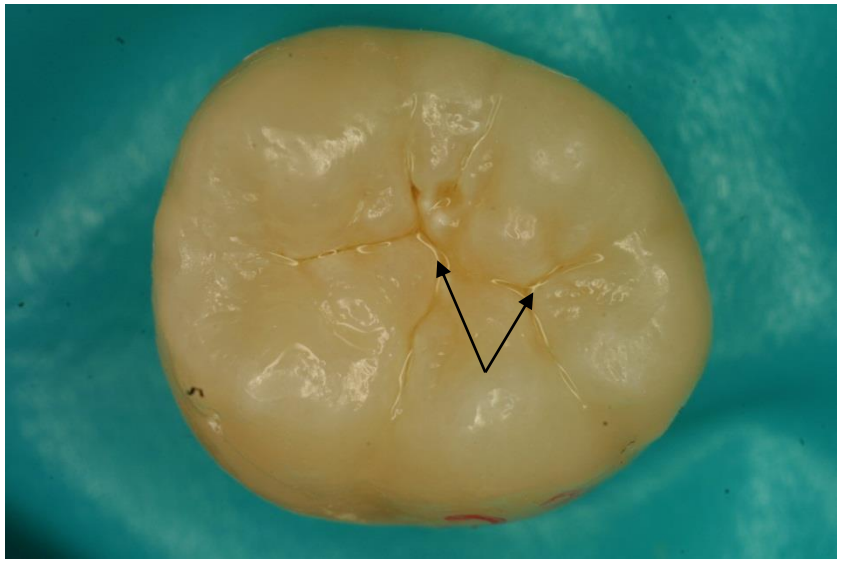
Score 1: White opacity or brown discoloration confined to fissure and in isolation

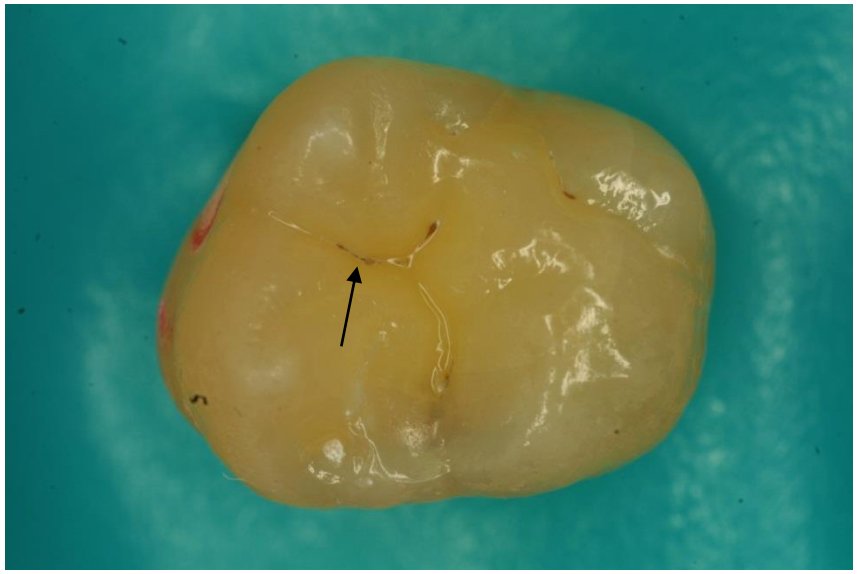


1W

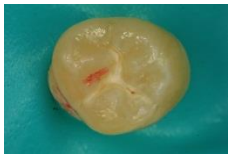


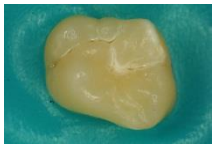
1B

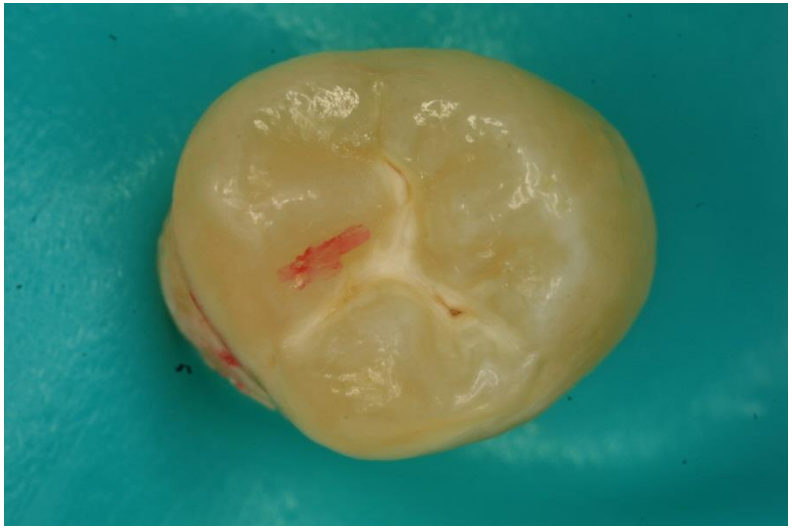


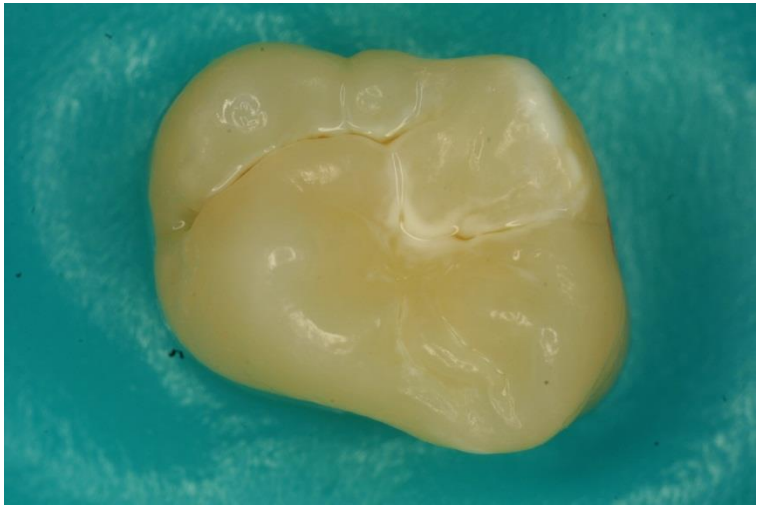


Score 2: White opacity beyoung fissure (no loss of surface integrity)

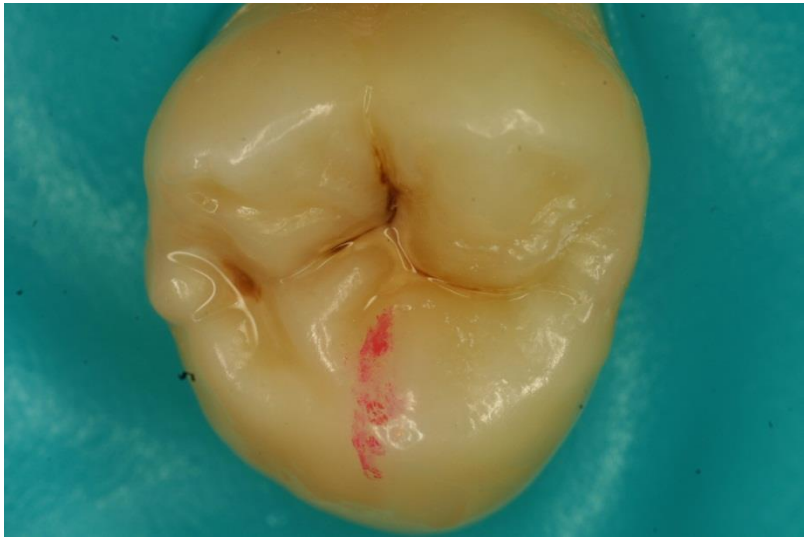






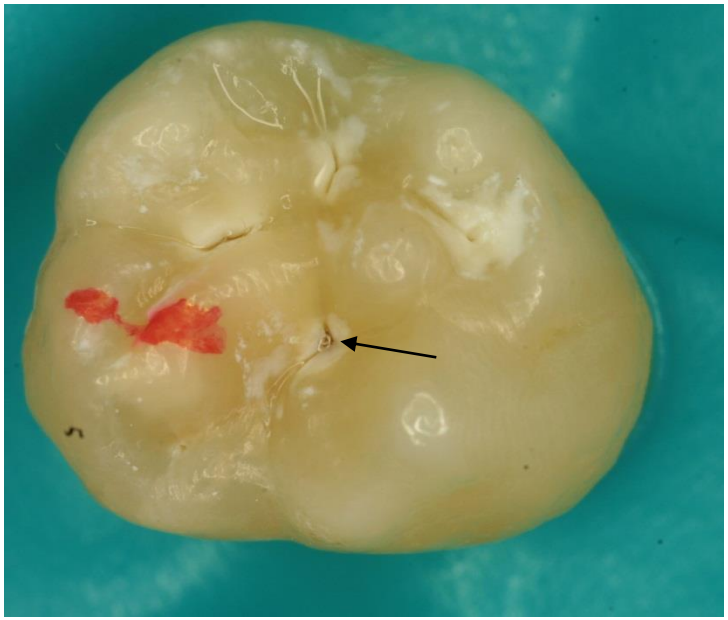


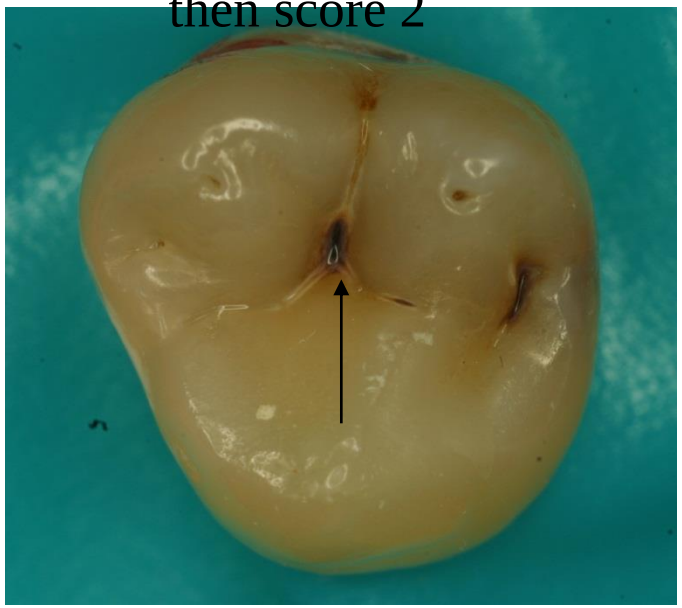
Score 2: Brown discoloration beyoung fissure (no loss of surface integrity)



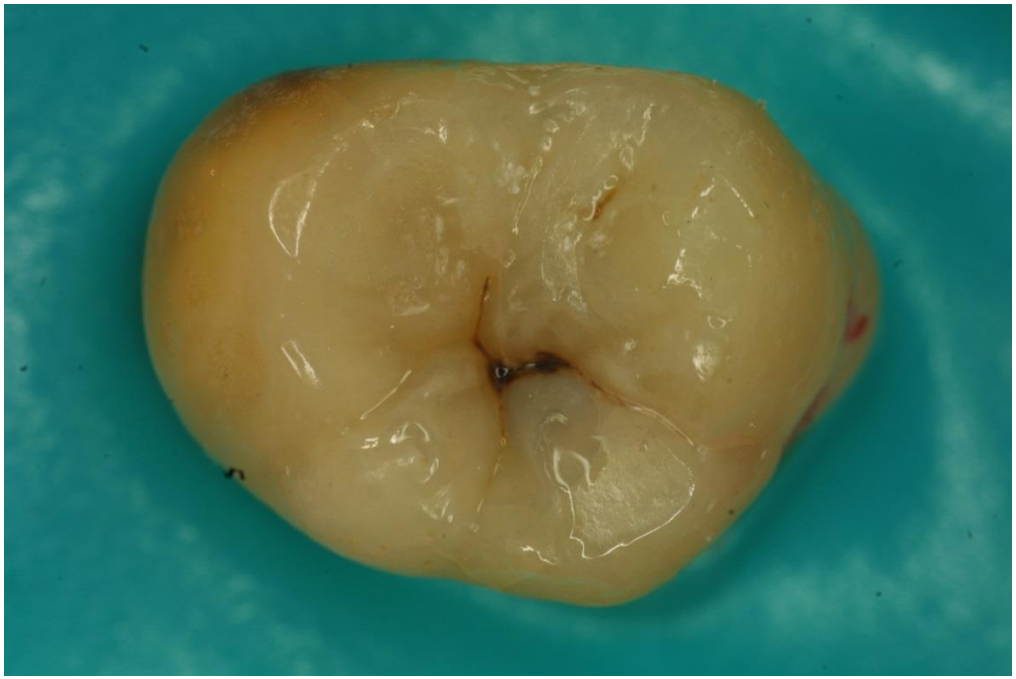
Score 3: Microcavities in white spots and in brown spots

In doubt of shadow (score 4), then score 3, in doubt then score 2

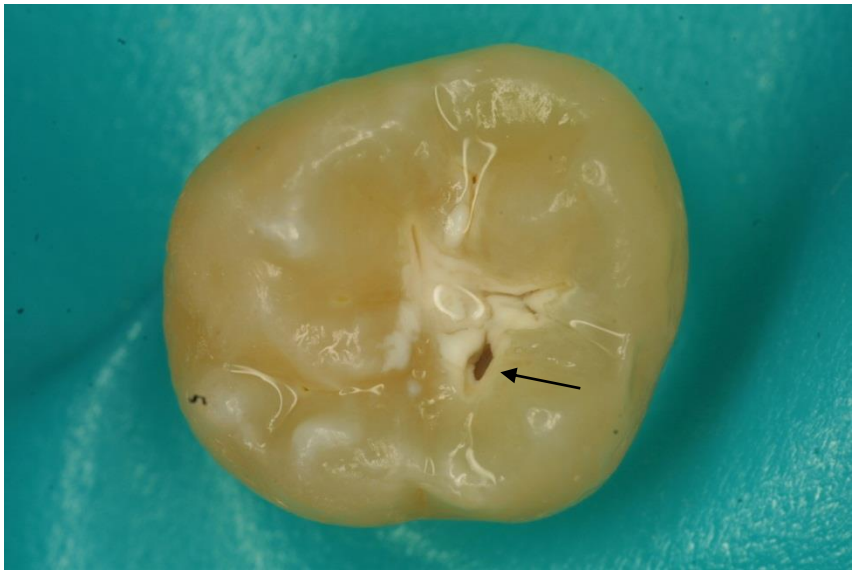


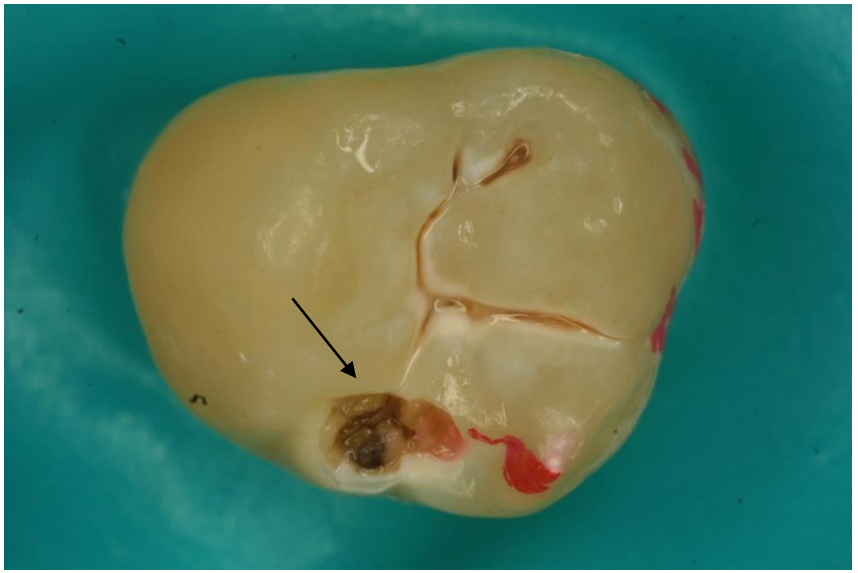


Score 4: Under lying shadow of dentine no loss of surface integrity

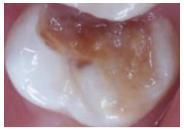


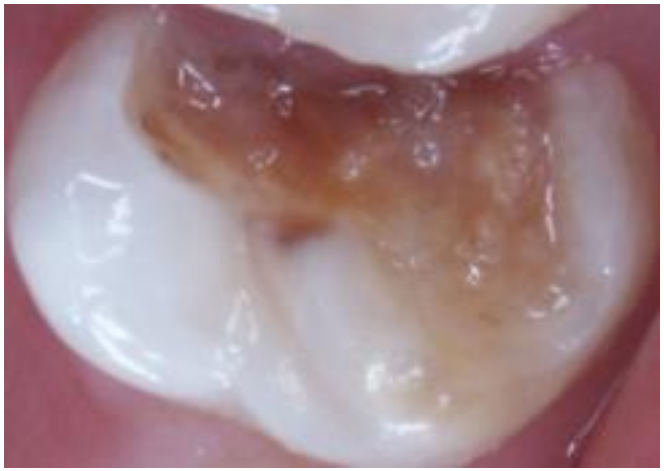
Score 5: Distinct cavitation exposing dentine





Score 6: Extensive cavity- dentine clearly visible





MANAGEMENT

of dental caries

CAMBRA (caries management by risk assessment)

Part of an overall approach to prevent and treat the caries infection and is composed of the following sections:

- Caries disease indicators
- Caries risk factors
- Caries Protective Factors

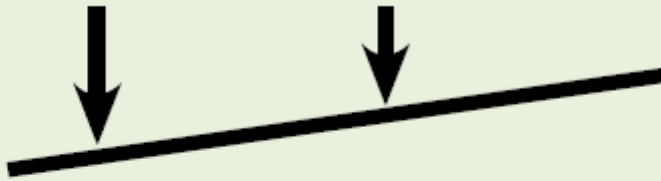
Caries Risk Assessment Form — Children Age 6 and Over/Adults

Patient Name: _____ Chart #: _____ Date: _____

Assessment Date: Is this (please circle) base line or recall

Disease Indicators (Any one "YES" signifies likely "High Risk" and to do a bacteria test)**	YES = CIRCLE	YES = CIRCLE	YES = CIRCLE
Visible cavities or radiographic penetration of the dentin	YES		
Radiographic approximal enamel lesions (not in dentin)	YES		
White spots on smooth surfaces	YES		
Restorations last 3 years	YES		
Risk Factors (Biological predisposing factors)		YES	
MS and LB both medium or high (by culture**)		YES	
Visible heavy plaque on teeth		YES	
Frequent snack (> 3x daily between meals)		YES	
Deep pits and fissures		YES	
Recreational drug use		YES	
Inadequate saliva flow by observation or measurement (**If measured, note the flow rate below)		YES	
Saliva reducing factors (medications/radiation/systemic)		YES	
Exposed roots		YES	
Orthodontic appliances		YES	

Protective Factors			
Lives/work/school fluoridated community			
Fluoride toothpaste at least once daily			
Fluoride toothpaste at least 2x daily			
Fluoride mouthrinse (0.05% NaF) daily			
5,000 ppm F fluoride toothpaste daily			
Fluoride varnish in last 6 months			
Office F topical in last 6 months			
Chlorhexidine prescribed/used one week each of last 6 months			
Xylitol gum/lozenges 4x daily last 6 months			
Calcium and phosphate paste during last 6 months			
Adequate saliva flow (> 1 ml/min stimulated)			
****Bacteria/Saliva Test Results: MS: LB: Flow Rate: ml/min. Date:**			



VISUALIZE CARIES BALANCE
(Use circled indicators/factors above)
(EXTREME RISK = HIGH RISK + SEVERE SALIVARY GLAND HYPOFUNCTION)
CARIES RISK ASSESSMENT (CIRCLE): EXTREME HIGH MODERATE LOW

Doctor signature/#: _____ Date: _____

From: Featherstone JD, Domejean-Orliaguet S, Jenson L, Wolff M, Young DA. Caries risk assessment in practice for age 6 through adult. J Calif De
2007;35(10):703-713. Reprinted with permission from the California Dental Association.

CAMBRA

Low risk – no dental lesions, no visible plaque, optimal fluoride, regular dental care

Moderate risk – dental lesion in previous 12 months, visible plaque, suboptimal fluoride, irregular dental care

High risk – one or more cavitated lesions, visible plaque, suboptimal fluoride, no dental care, high bacterial challenge, impaired saliva, medications, frequent snacking

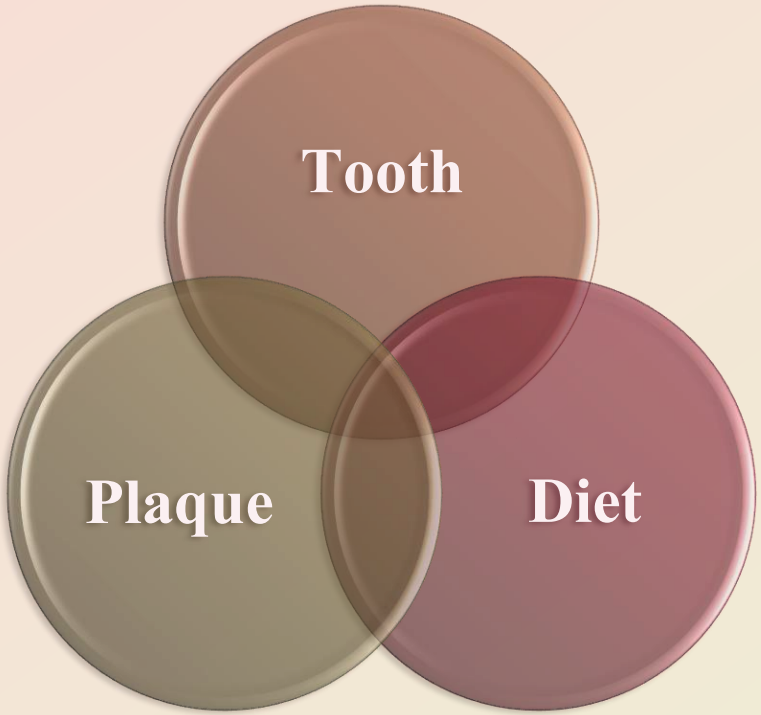
Extreme risk – high risk patient with special needs or severe hyposalivation

Caries Management by Risk Assessment
Clinical Guidelines for Patients Age 6 and Older

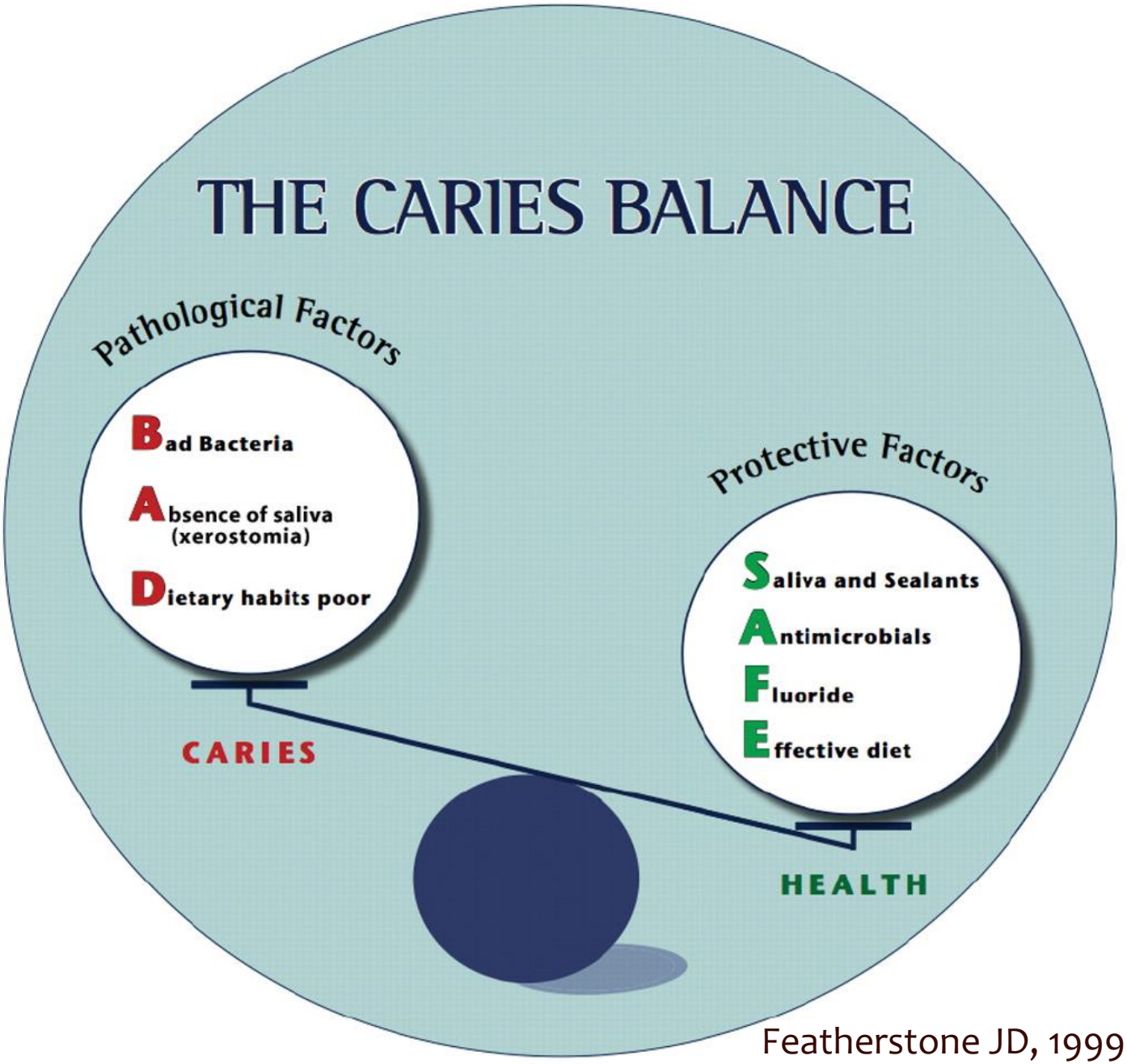
Risk Level ### ***	Frequency of Radiographs	Frequency of Caries Recall Exams	Saliva Test (Saliva Flow & Bacterial Culture)	Antibacterials Chlorhexidine Xylitol ****	Fluoride	pH Control	Calcium Phosphate Topical Supplements	Sealants (Resin-based or Glass Ionomer)
Low risk	Bitewing radiographs every 24-36 months	Every 6-12 months to re-evaluate caries risk	May be done as a base line reference for new patients	Per saliva test if done	OTC fluoride-containing toothpaste twice daily, after breakfast and at bedtime. Optional: NaF varnish if excessive root exposure or sensitivity	Not required	Not required Optional: for excessive root exposure or sensitivity	Optional or as per ICDAS sealant protocol (TABLE 2)
Moderate risk	Bitewing radiographs every 18-24 months	Every 4-6 months to re-evaluate caries risk	May be done as a base line reference for new patients or if there is suspicion of high bacterial challenge and to assess efficacy and patient cooperation	Per saliva test if done Xylitol (6-10 grams/day) gum or candies. Two tabs of gum or two candies four times daily	OTC fluoride-containing toothpaste twice daily plus: 0.05% NaF rinse daily. Initially, 1-2 app of NaF varnish; 1 app at 4-6 month recall	Not required	Not required Optional: for excessive root exposure or sensitivity	As per ICDAS sealant protocol (TABLE 2)
High risk*	Bitewing radiographs every 6-18 months or until no cavitated lesions are evident	Every 3-4 months to re-evaluate caries risk and apply fluoride varnish	Saliva flow test and bacterial culture initially and at every caries recall appt. to assess efficacy and patient cooperation	Chlorhexidine gluconate 0.12% 10 ml rinse for one minute daily for one week each month. Xylitol (6-10 grams/day) gum or candies. Two tabs of gum or two candies four times daily	1.1% NaF toothpaste twice daily instead of regular fluoride toothpaste. Optional: 0.2% NaF rinse daily (1 bottle) then OTC 0.05% NaF rinse 2X daily. Initially, 1-3 app of NaF varnish; 1 app at 3-4 month recall	Not required	Optional: Apply calcium/ phosphate paste several times daily	As per ICDAS sealant protocol (TABLE 2)
Extreme risk** (High risk plus dry mouth or special needs)	Bitewing radiographs every 6 months or until no cavitated lesions are evident	Every 3 months to re-evaluate caries risk and apply fluoride varnish.	Saliva flow test and bacterial culture initially and at every caries recall appt. to assess efficacy and patient cooperation	Chlorhexidine 0.12% (preferably CHX in water base rinse) 10 ml rinse for one minute daily for one week each month. Xylitol (6-10 grams/day) gum or candies. Two tabs of gum or two candies four times daily	1.1% NaF toothpaste twice daily instead of regular fluoride toothpaste. OTC 0.05% NaF rinse when mouth feels dry, after snacking, breakfast, and lunch. Initially, 1-3 app. NaF varnish; 1 app at 3 month	Acid-neutralizing rinses as needed if mouth feels dry, after snacking, bedtime and after breakfast. Baking soda gum as needed	Required Apply calcium/ phosphate paste twice daily	As per ICDAS sealant protocol (TABLE 2)

Etiology of dental caries

Keyes circle, 1963



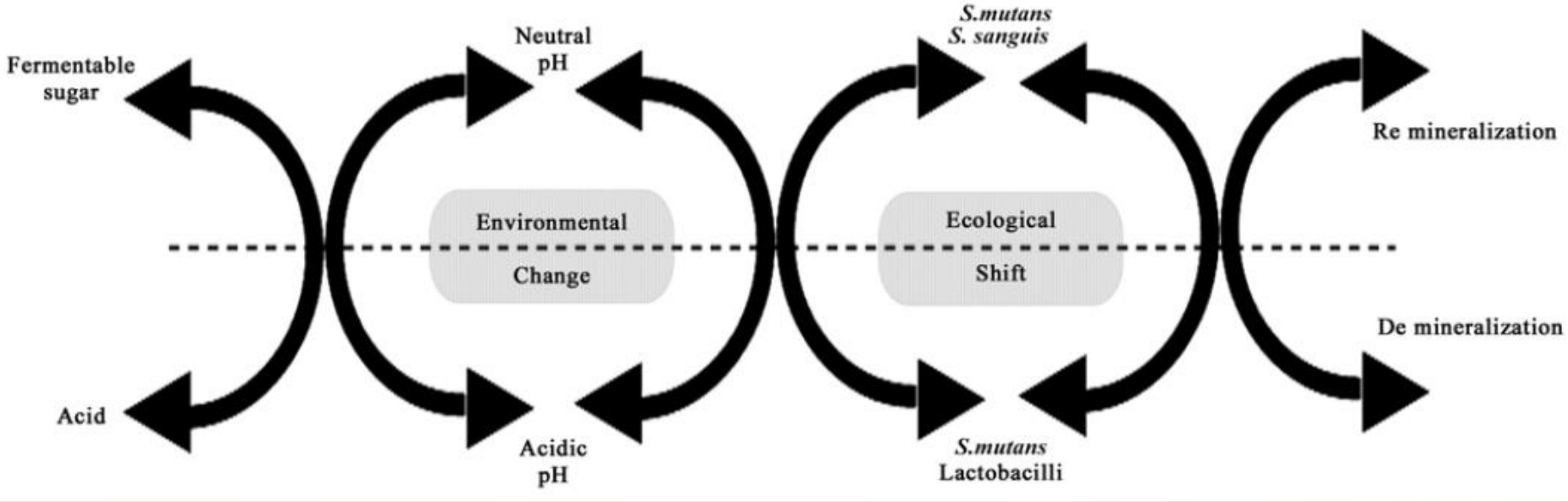
Newbrun, 1983



Featherstone JD, 1999

Pathogenesis of dental caries

Ecological Plaque Hypothesis



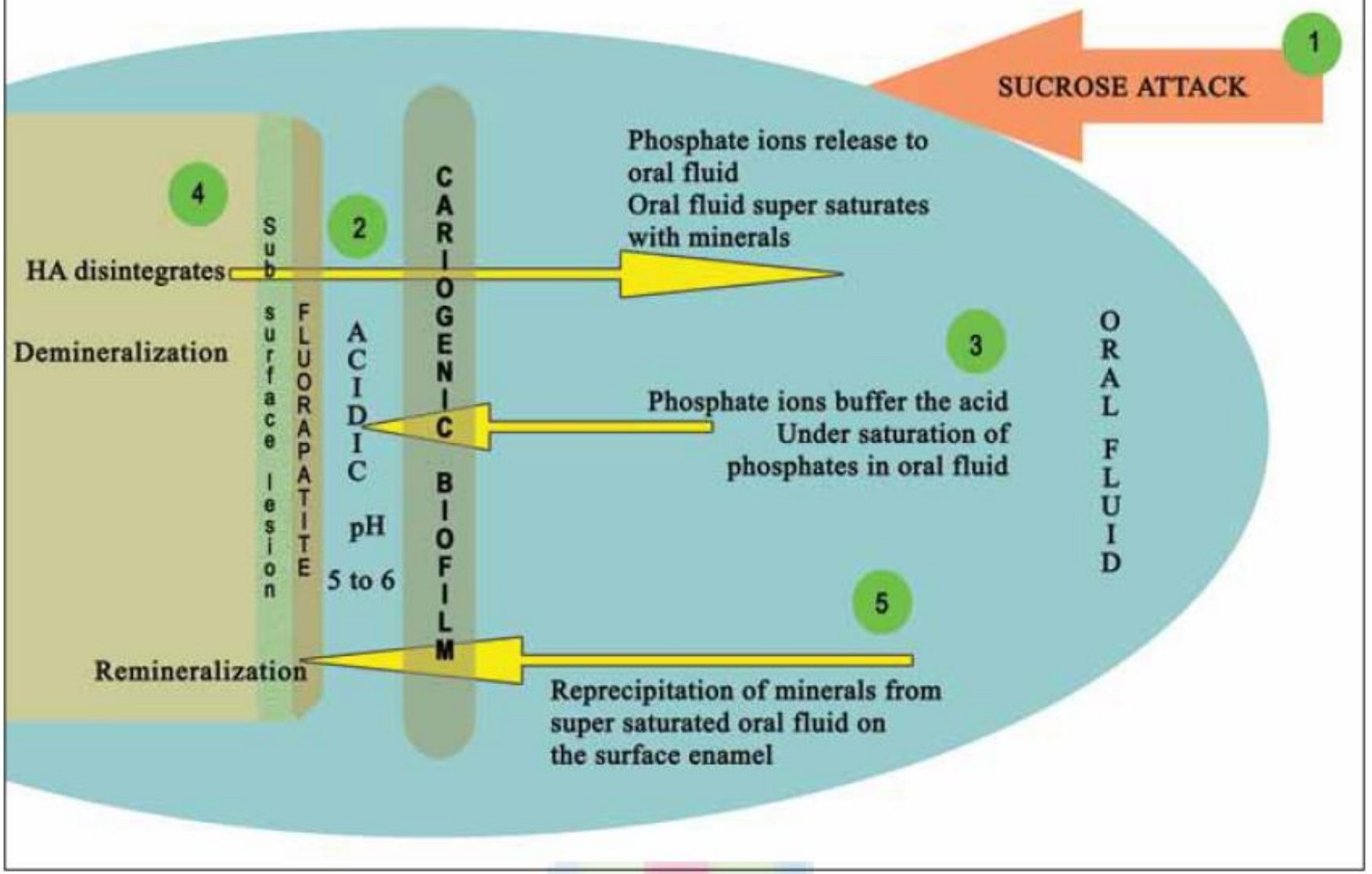


Figure 5: Demineralization of hydroxyapatite (HA) and remineralization with fluorapatite (FA) - depicted as stage 1-5

Stage 1: Fermentable sucrose intake.

Stage 2: Microbes in cariogenic plaque metabolise them releasing acid in the biofilm-tooth interface. The pH in the interface drops below the critical pH of HA

Stage 3: Phosphate ions from oral fluid buffer the acidic ions resulting in undersaturation.

Stage 4: HA disintegrates to release the phosphate ions back to the oral fluid till it supersaturates-Demineralization

Stage 5: Supersaturated oral fluid re precipitates the minerals onto the disintegrated enamel. If fluoride also deposits FA is formed on the superficial layer - Remineralization. Sub surface demineralization remains.

Chemical composition of enamel

- Enamel is composed of long thin hydroxyapatite crystals surrounded by a matrix of water and organic material

- Enamel apatite contains **2-4% carbonate**
- Carbonated – apatite crystals are incorporated into the water/protein/lipid superstructure which occupies about 15% by volume of enamel

- Protein and lipid with a large proportion of water form the diffusion channels.

Caries process

In the presence of suitable carbohydrate substrate, organic acids are produced by plaque bacteria.

↓

Diffusion of acids into the enamel

↓

Dissociation of acids into H^+ ions

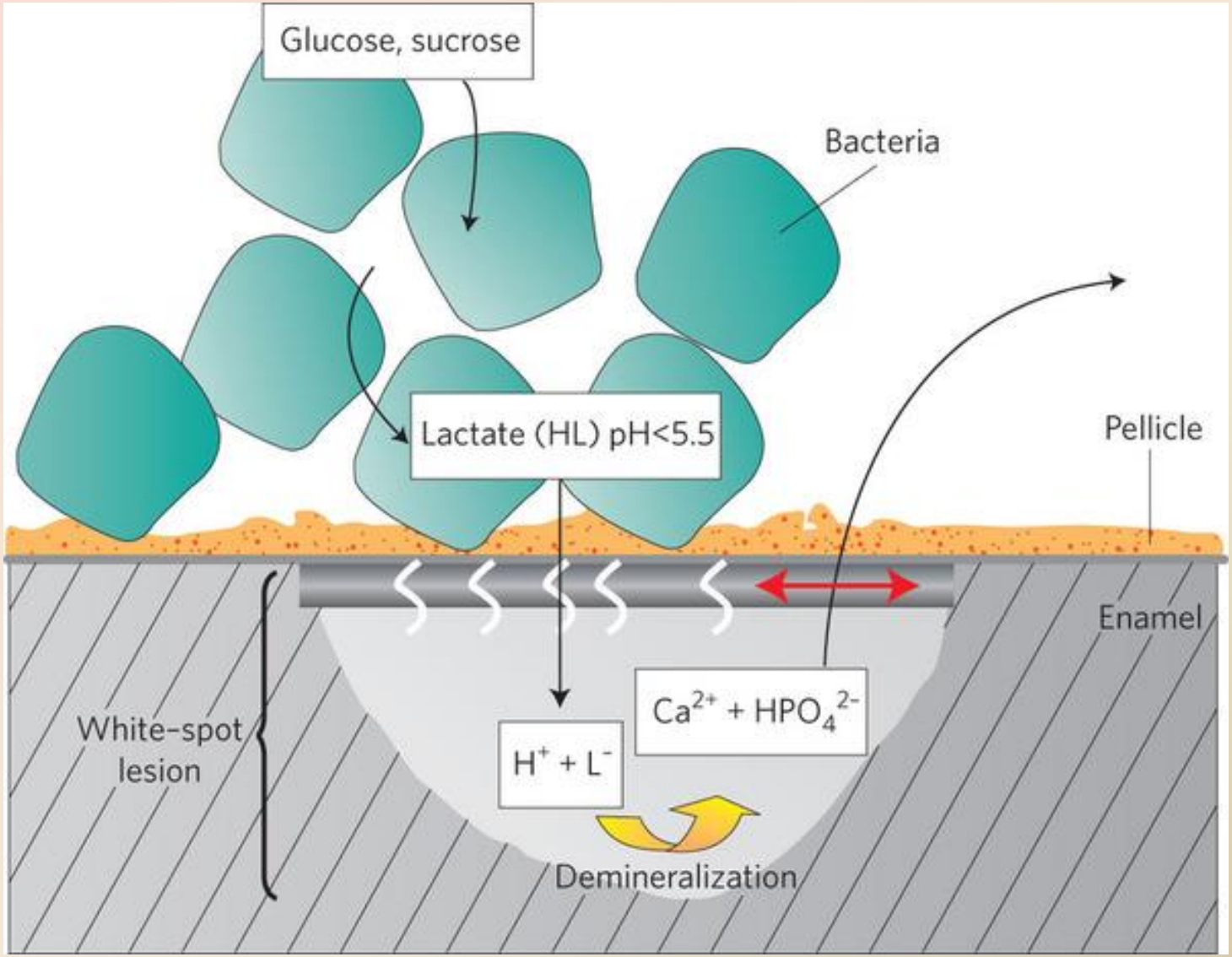
↓

H^+ ions attack the apatite crystals at vulnerable points where carbonate is present

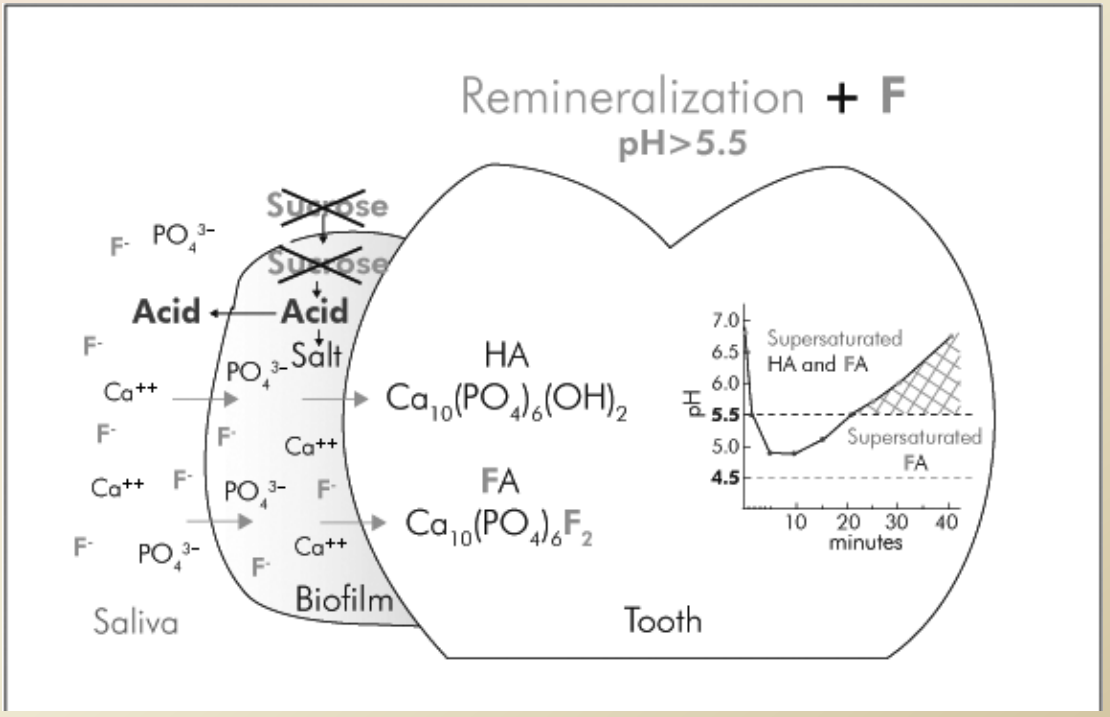
↓

Removal of calcium, hydroxyl, phosphate, carbonate ions through the diffusion channels (demineralization)

Caries process

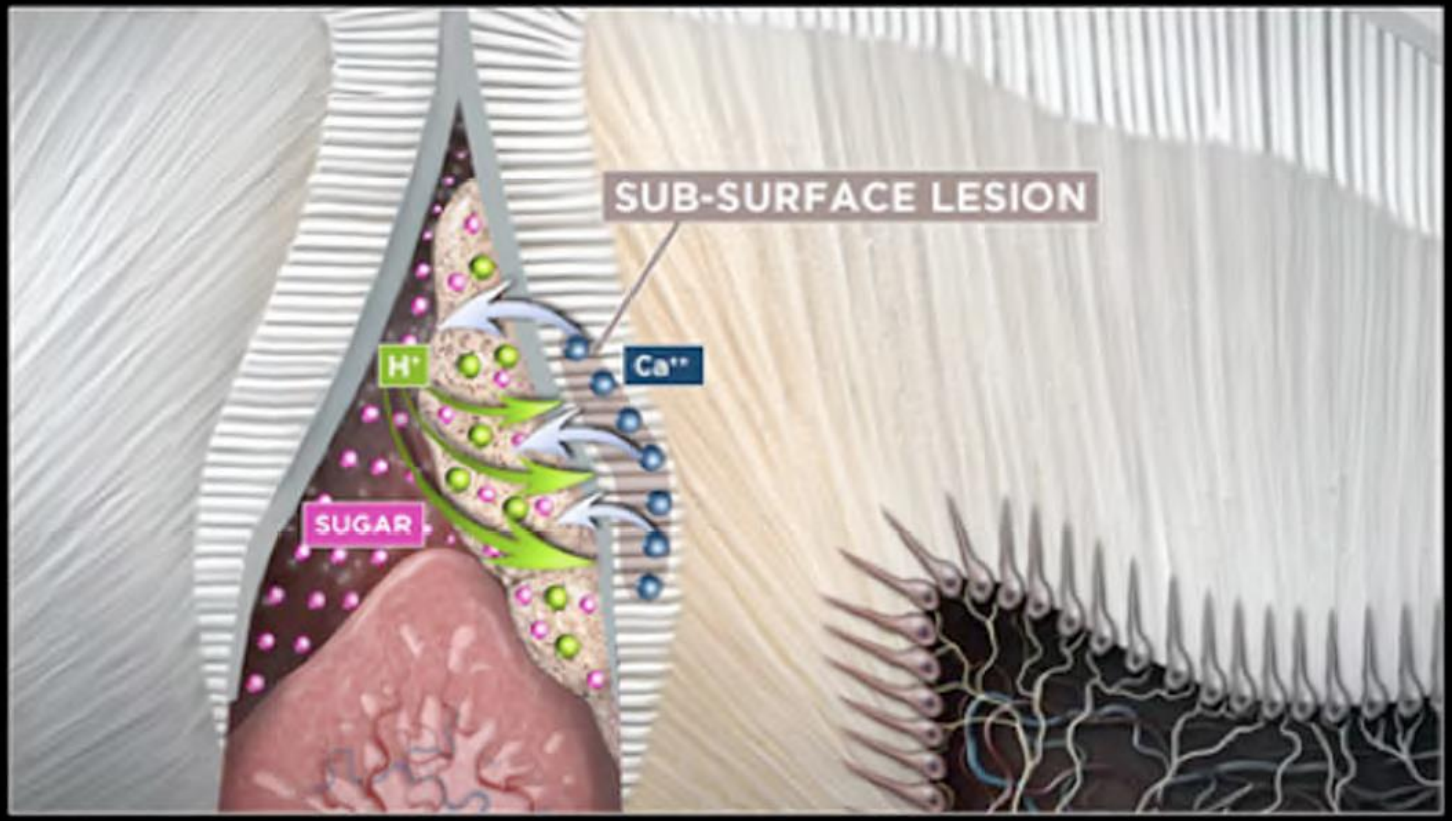


- Increase in the concentration of calcium and phosphate ions – remineralization on the surface of existing crystals or formation of new crystals.



CARIES LESION INITIATION AND PROGRESSION

FERMENTATION PRODUCES ACID LEADING TO DEMINERALIZATION



- Fermentation of sugars lowers biofilm pH
- Lower pH causes shift in biofilm ecology
- *S mutans* and *Lactobacilli* proliferate
- More acid production leads to demineralization and the sub-surface lesion

Initiation of dental caries

Dental caries develops where microbial deposits are allowed to form biofilms that are not frequently removed or disturbed by mechanical wear

After one week, no changes can be seen macroscopically even after careful air-drying procedure.

At the ultrastructure level, there is direct dissolution of the outer enamel surface.
The intercrystalline spaces are wider

Further increase in enamel porosity occurs by preferential removal of mineral from the tissue deep to the outer surface.

After 3 and 4 weeks, the outermost surface exhibits complete dissolution of thin perikymata and more marked dissolution corresponding to larger developmental irregularities such as pits of Tomes' processes and focal holes.

Intercrystalline spaces of the entire involved enamel surface are enlarged and hence contribute to the overall increase in porosity of enamel.

After 14 days with completely undisturbed plaque, the enamel changes are visible after air-drying as whitish opaque changes.

From this stage, the clinical changes can be readily seen even without air-drying, the more extensive loss of mineral beneath the outer surface constantly increasing.

After 4 weeks, an active enamel lesion, the white spot lesion has a characteristic chalky surface.

Increase in internal enamel porosity due to demineralization causes loss of translucency and this makes the enamel more opaque.

Enamel loses its shiny appearance because the irregular surface generated by the erosion of the very outermost surface gives rise to diffuse reflection of light.

Due to surface erosion, small scratches can be appreciated with a probe in the surface of active lesions

Theories of Dental Caries

The Legend of Worms

Acc to ancient sumerian text, dates back from 5000 BC, toothache was caused bya worm that drank the blood of the teeth and fed on roots of the jaws. The idea was almost universal at one time, as evidenced by the writings of Homer. Guy de Cahuliac (1300-1368), the greatest surgeon of middle ages, believed that worms caused dental decay

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 imbalance of these humors.

Theories of Dental Caries

Vital theory

Regarded dental caries as originating within the tooth itself, analogous to bone gangrene. This theory, proposed at the end of the eighteenth century. A clinically well known type of caries is characterized by extensive penetration into the dentin, and even into the pulp, but with barely detectable catch in the fissure.

Chemical theory

Parmly (1819) proposed that an unidentified 'chemical agent' was responsible for caries. He stated that caries began on locations where food putrified and acquired sufficient dissolving power to produce the disease chemically. Support came from Robertson (1835) and Regnart (1938) who carried out experiments with different dilutions of inorganic acids and found that they corroded enamel and dentin.

Parasitic or septic theory

In 1843, Erdl described filamentous parasites in the “surface membrane” of teeth. Shortly thereafter, Ficinus, observed filamentous microorganisms, which he called denticole, in material taken from carious cavities that cause decomposition of enamel and dentin.

Theories of Dental Caries

Chemo-parasitic theory

Willoughby D. Miller (1853-1907) proposed that caries is caused by acids produced by microorganisms of the mouth. In the series of experiments, he demonstrated the following facts-

- Acid was present within the deep carious lesions.
- Different kinds of foods mixed with saliva and incubated at 37 C could decalcify the entire crown of a tooth.
- Several types of mouth bacteria 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 micrococci) invade carious dentin.

Theories of Dental Caries

Proteolytic theory

Gottlieb (1944) maintained that the initial action was due to proteolytic enzymes attacking the lamellae, rod sheaths, tufts and walls of dentinal tubules.

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 favouring their solution by acidogenic bacteria.

Pincus (1949) contended that proteolytic organisms first attacked the protein elements. He suggested that sulfatases of gram negative bacteria hydrolyzed mucoitin sulfate of enamel or chondroitin sulfate of dentin and produced sulfuric acid that combine with the calcium of mineral phase.

Theories of Dental Caries

Proteolysis-chelation theory

This theory considers dental caries to be a bacterial destruction of teeth where the initial attack is essentially on organic components of enamel. The breakdown products of this organic matter have chelating properties and thereby dissolve the minerals in the enamel. Thus, both the organic and inorganic constituents of enamel are simultaneously demolished.

According to this theory, decalcification is mediated by a variety of complexing agents, such as acid anions, amines, amino acids, peptides, polyphosphates and carbohydrates derivatives. These substances are microbial breakdown products of either the organic components of enamel and dentin, or of food that is ingested and diffuses through the plaque.

Theories of Dental Caries

Phosphate sequestration theory

Luoma showed that inorganic phosphate was taken up by plaque bacteria during metabolism of carbohydrates. It has been postulated that a steady state equilibrium exists between the inorganic phosphate of saliva and the mineral phase of enamel.

Acc to theory, as bacteria take up phosphate, inorganic phosphate must be removed from the enamel.

Role of Diet in Dental Caries

Diet is “the customary allowance of food and drink taken by any person from day to day”

Sucrose in Dental Caries

SUCROSE-ARCH CRIMINAL (NEWBRUN 1969)

- Effect on plaque
- substrate for cariogenic microflora

➢ Sucrose → polymers → bulk of plaque

↓

attachment of bacteria

➢ High free energy → high specificity of enzymes

SUGARS – THE ARCH CRIMINAL (zero 2004)

Cariogenicity of Foods

- Based on acidogenic potential

  Raw vegetables<nuts<milk<corn chips<fresh fruit<ice cream<French fries<dried fruit.

- Retention

  High sugar foods- caramel, chocolate bars

  Sucrose+ cooked starch

Cariogenecity- food composition, texture, solubility, retentiveness, and rate of salivary clearance than sucrose alone

Fat and Dental Caries

- Coating of tooth surface - oily substance - less retention
- Prevent fermentable sugar substrate from being reduced to acid.
- Fatty acids = interfere with growth and metabolism of cariogenic bacteria.
- Dietary fat = amount of dietary fermentable carbohydrate necessary for organic acid formation

Trace elements and Dental Caries

Minerals that may inhibit or promote caries :Navia 1970

- Strongly cariostatic : Fl , P.
- Mildly cariostatic : Mb , Sr, Ca, Bo, Li , Au , Cu.
- Promoting elements : Se, Mg , Cd , Pl , Pb , Si.
- Caries inert : Ba, Al , Ni ,Fe , Ti.
- Doubtful : Co , Mn , Sn , Zn , Br, I.

Novel Carbohydrates and their role

- **Artificially produced polymers of glucose**
  - Glucose Syrup & Maltodextrins
- **Oligosaccharides of glucose, fructose and galactose**
- **Many resist digestion and pass on to large intestine (PROBIOTIC)**
- **Isomaltooligosaccarides prevent glucans synthesis by bacteria**
- **Fructooligosaccharides- are as acidogenic as sucrose.**

Sugar substitutes and alternative sweeteners

- **Most important role :**Sugar free confectionery, chewing gums soft drinks, table top sweeteners and in liquid oral medicines.

- **Non sugar sweeteners :**

1. **Bulk sweeteners or Caloric sweeteners.**
   Ex: Polyalcohol (Sorbitol , Xylitol), Starch hydroylsates

2. **Intense sweeteners or Non caloric sweeteners.**
   Ex: Aspartams, Saccharine, Cyclamate, Some Plant sources.

- Bulk sweeteners :

❖ Chemically similar to sugars.

❖ Add volume and sweetness to a product.

❖ 0.5 to 1.0 times as sweet as sucrose.

❖ Have an energy value (Kilocalories).

❖ Naturally found in foods.

- Sorbitol

- Prepared from glucose by hydrogenation.
- One half as sweet as sucrose .
- Slowly and incompletely absorbed from the intestine : result in osmotic diarrhoea.
- Microbial Metabolism of Sorbitol.

  Most oral microorganisms lack the enzymatic makeup to utilize Sorbitol.

Xylitol

- Absorption slow and incomplete .
- Used in Diabetics.
- Metabolism by Oral Microorganisms :Human oral microorganisms do not have enzymes to utilize xylitol.

Starch hydroylsates

- Lycasin :Hydrogenated glucose syrup produced from starches .
- Caloric value – Similar to other carbohydrates.

- Intense sweeteners

- Not chemically related to sugars.
- Added in very small quantities and not volume.
- 100 to 1000 times sweeter than sucrose.
- Negligible energy value (Kilocalories)
- Low caloric sweeteners are used in:

Gelatin desserts puddings

Desert toppings

Soft drinks

Chewing gums

Medicinal preparations

Dentifrices and mouth washes

Aspartame

- 180-200 times as sweet as sucrose.
- Composed 2 amino acids : L-aspartic acid and Methyl ester of L-phenylalanine.
- Reduces caries – Limiting the amount or frequency of fermentable sugar in the diet.

Saccharine

- Pharmacologically inert and is stable.
- Widely used in – diets , soft drinks , dietic food , mouth washes, medicinal preparations , sweeteners for table use.

Cyclamate

- Organic sweetener .
- Economical.

Other sweeteners derived from plant sources

- 500-3000 times as sweet as sucrose.
  Ex: Monelin, Licorice, Dihydrochalcone, Miraculin

Sugar Substitutes in Pharmaceutical Preparations

- Xylitol, Mannitol , Sorbitol , Lycasin.

Food Additives

- May decrease the local caries challenge or conversely enhance the local natural defense mechanisms .
  Ex : Presence of Ca and P in Saliva

Dietary Recommendations

- **Restrict the number of eating times to three main meals.**
- **Avoid carbohydrate (sugars) snacks in between meals.**
- **Take low carbohydrate and high protein snacks and fibrous fruits in between meals, if required.**
- **Eliminate eating sticky sweets like chocolates, toffees, candies, cake, and pastries, if not completely then as much as possible.**

Dietary Recommendations

- Increase eating of high protein food like meat, fish, milk, egg, pulses and beans.

- Restrict carbohydrate eating so that they only provide between 30 to 50 percent of total calories requirement of the body.

- Eat firm detersive food like raw vegetables and fruits which will reduce dental plaque formation and increase salivary flow.

- Fluoride
  - If present ,Free sugars – 15 to 20 Kg/person/year (40-55 g/day).
  - If Absent, Free sugars – below 15Kg/person/year (40g/day).

CLINICAL FEATURES

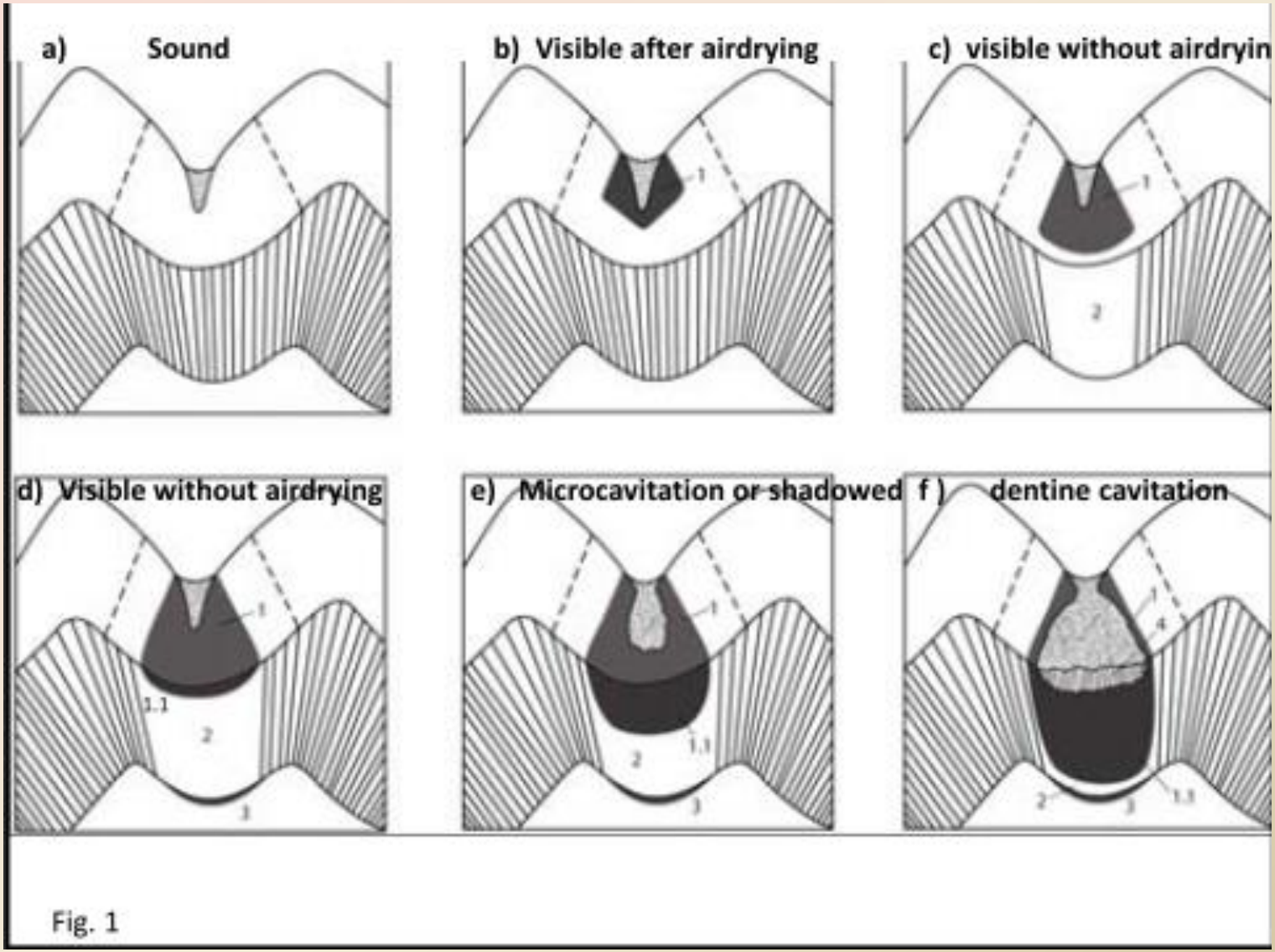


Fig. 1

CLINICAL FEATURES

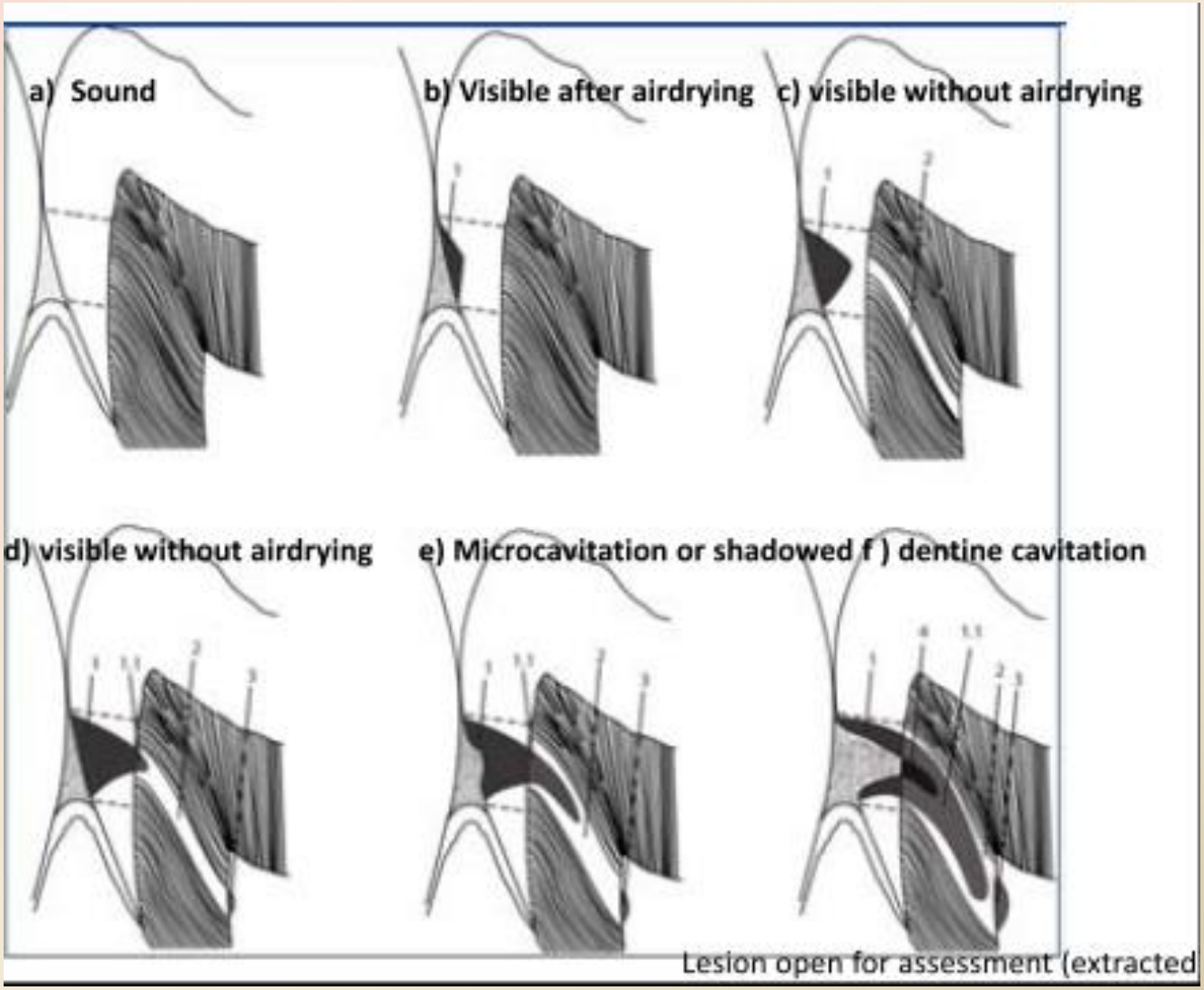


Table 6: The pathoanatomical features of the enamel and dentin in active and arrested lesions[60]

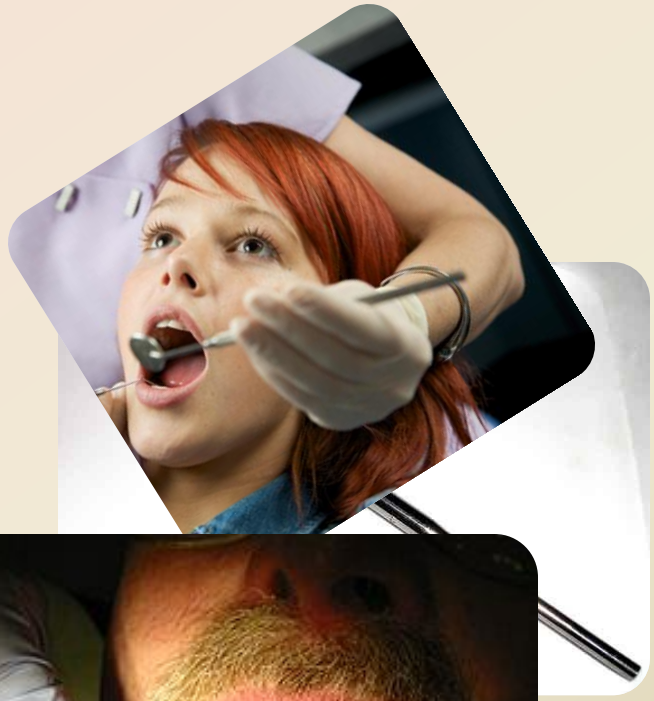
	Visual	Tactile
Enamel		
Active	The lesion is whitish/ yellowish; the lesion is chalky (lack of luster); the lesion can be cavitated or not	The lesion feels rough to probing; probing might or might not find cavity
Arrested	The lesion is more yellowish/ brownish than whitish; the lesion is more shiny than matte; the lesion can be cavitated or not	The lesion feels more smooth than rough; probing might or might not find a cavity
Coronal dentine		
Active	The lesion may manifest itself as a shadow below the intact but demineralized enamel; if a cavity extends into the dentine, the dentine appears yellowish/brownish	Dentine soft to probing
Arrested	The lesion may manifest itself as a shadow below the intact but demineralized enamel; if a cavity extends into the dentine, the dentine appears brownish	Harder than at the active lesion but not as hard as sound dentine
Root dentine		
Active	Yellowish/brownish	Soft/leathery
Arrested	Brownish/blackish	Harder but not as hard as sound root dentine

PROCEDURE FOR DIAGNOSING CARIES

DIAGNOSTIC METHODS

VISUAL EXAMINATION

TACTILE EVIDENCE



PROCEDURE FOR DIAGNOSING CARIES

- Lussi A. Validity of diagnostic and treatment decision of fissure caries. Caries Res 1991;25:296-303

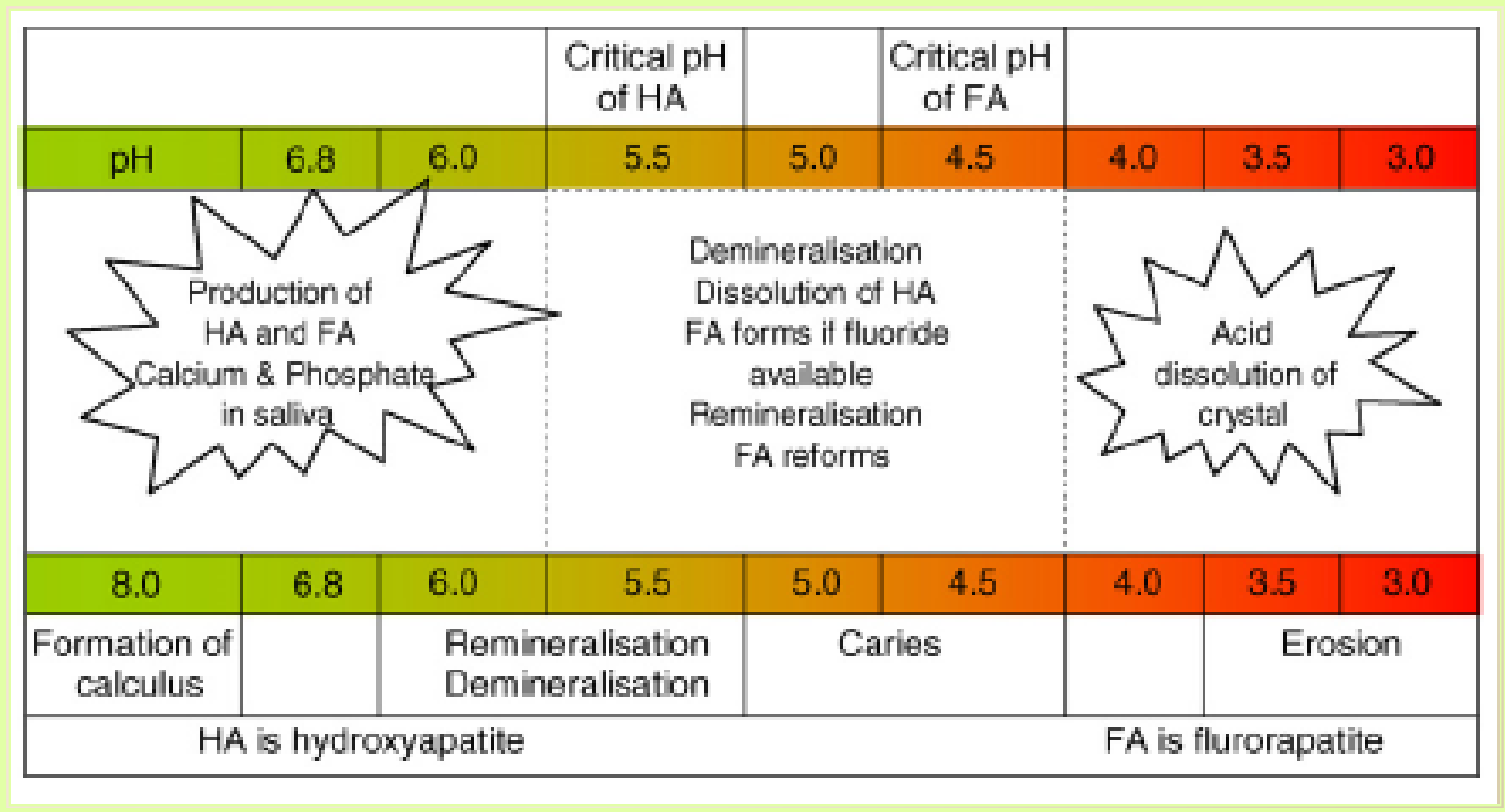
- 73% for both techniques

- Explorer→ Overtreatment
- Visual → Under-treatment

- Bergman and Linden 1969
- National institute of health consensus development 2001

- Periodontal probe (Ekstrand and collagues 2007)

PROCEDURE FOR DIAGNOSING CARIES



TOOLS FOR DIAGNOSING CARIES

Radiographic methods

Electrical resistance

Fiberoptic transillumination

DIFOTI

Laser autofluorescence

Quantitative laser fluorescence

Infrared fluorescence

Ultraviolet illumination

Endoscope/Videoscope

Ultrasonic detection

Optical coherence tomography

Terahertz imaging

Magnetic resonance micro-imaging

Dye penetration method

PROBLEMS ASSOCIATED

Overlapping
Occlusal caries → Imperceptible
Caries or Resorption or Wear facet or…..??
Two dimensional
Buccal or lingual caries
Cervical burnout

1. Conventional radiography
2. Xeroradiography
3. Digital imaging
4. Computer image analysis
5. Subtraction radiography

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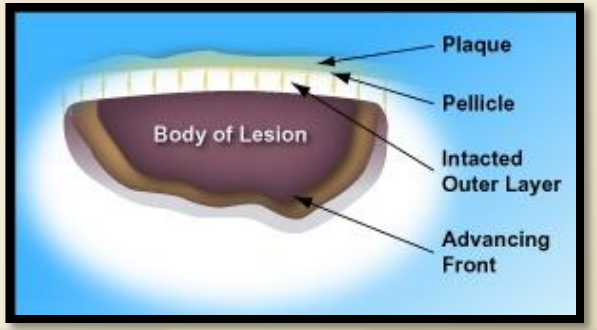

40-50%
(early and associates)

Bitewings- 215%!

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Radiographic caries scoring by Haugejorden and slack- progression of approximal caries-

C1- Enamel caries- < ½ way through enamel

C2- Enamel caries- > 1/2 way but not involving DEJ

C3- Caries of enamel and dentin – at/ through DEJ in dentin extending < ½ way to the pulp

C4- Caries of enamel and dentin – in dentin extending > ½ way to the pulp

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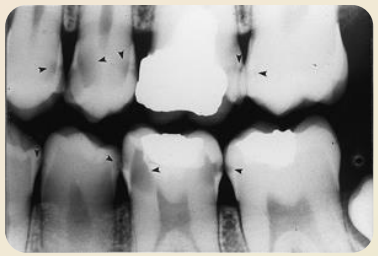
Conventional radiography

Paralleling technique

IOPAR



BITEWING

Radiographic appearance of caries

1. Occlusal caries: In Enamel ?????
   In Dentin
   Buccal or lingual caries
2. Interproximal caries
   Actual depth of lesion
   White chalky appearance
   Triangles in Enamel & Dentin
   Bitewing is better ?????
3. Root caries
   Within 2mm of CEJ
   Ill defined saucer like appearance
4. Secondary caries
   Radiograph ????
   Restorative material
   Cervical burnout & Bonding agent

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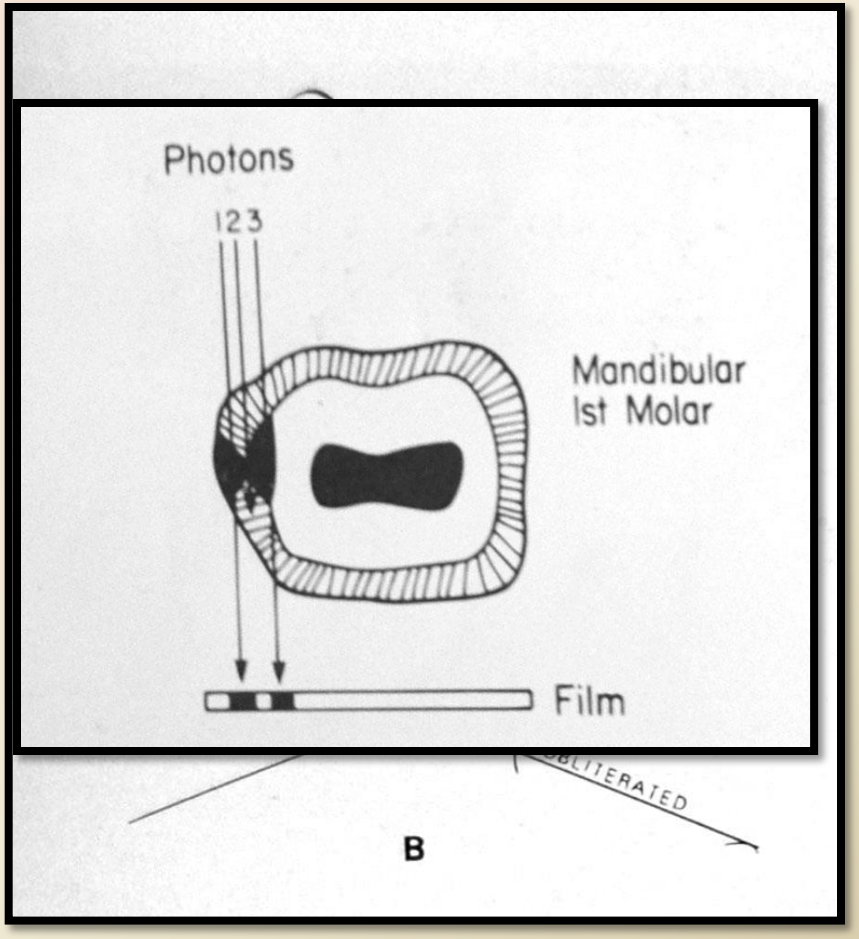
X- ray Angulation



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Xeroradiography

Al plate coated with Selenium particles

Electrostatic charge

Selective discharge of particles → Latent image

Advantage

Edge enhancement
No dark room required
More diagnostic
Radiation

Disadvantage

Discomfort to patient
Develop within 15 min

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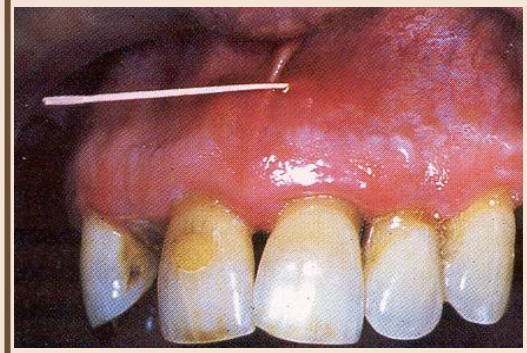
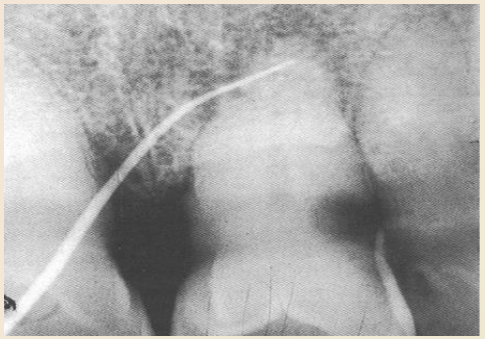
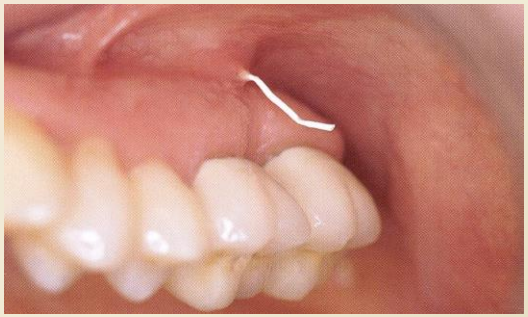
Sinus tracing







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DIGITAL IMAGING

Pixels

ADVANTAGES

Non film receptors

Darkroom is not required, instant images viewed

Types:

1. Digital image receptor (DIR)
2. Video camera for forming digital images of a radiograph

The quality of image is consistent

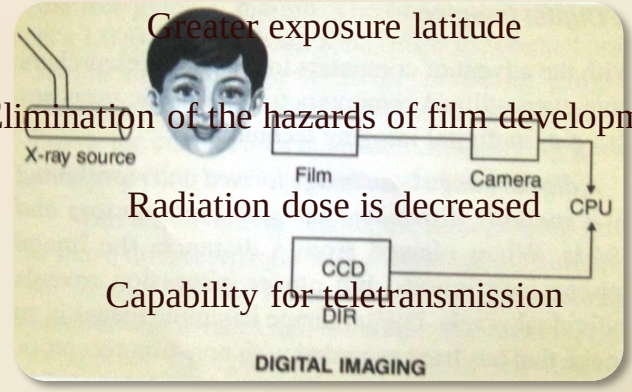
Super resolution

Greater exposure latitude

Elimination of the hazards of film development

Radiation dose is decreased

Capability for teletransmission



CCD: Oxides like silicon coated with X-ray sensitive phosphorous

COMPUTER IMAGE ANALYSIS

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SUBTRACTION RADIOGRAPHY

Reduction of Structured noise

Picture of radiograph using High quality camera

↓

Digitizer

↓

2 standardized radiographs of identical exposure geometry
(1^{st} → Reference image)

USES

Approximal caries
Recurrent caries
Remineralization and Demineralization patterns
Progression of periodontal disease
90% accuracy → 5% mineral loss of bone
0.12 mm thickness of bone

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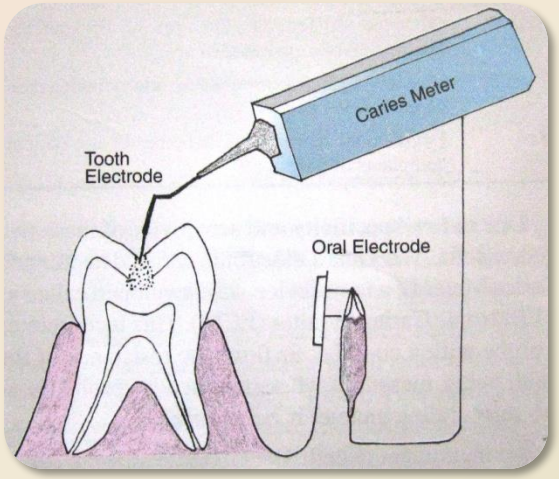
Electric Conductivity is directly proportional to amount of demineralization



Sensitivity →74.8
Specificity → 87.6
Reproducibility → Good to Excellent

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Impedance values		
Color	Impedence values	Indication
Green	600 K Ohms	Healthy tooth
Yellow	Between 250 and 600 k ohms	Enamel caries
Orange	Between 15 and 250 k ohms	Caries extending into dentin
Red	Below 15 k Ohms	Caries extending to tooth pulp exposed to bacterial infection

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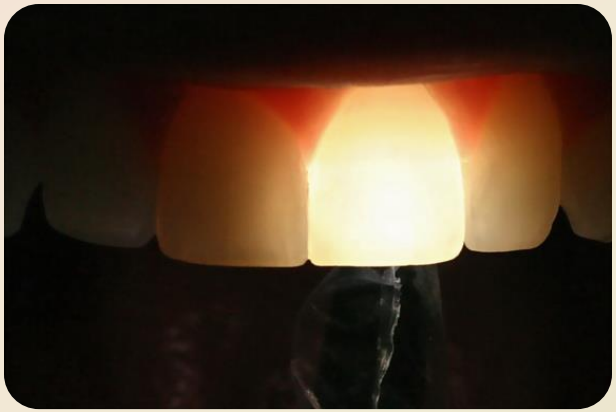
A carious lesion has a lowered index of light transmission, an area of caries appears as a darkened shadow that follows the spread of decay through the dentine



Advantages

No hazards of radiations

Simple and comfortable for the patients

Time consumption

Disadvantages

Permanent records are difficult to maintain as can be kept in radiographs.

It is subjected to intra and inter observer variations.

Difficult to locate the probe in certain areas

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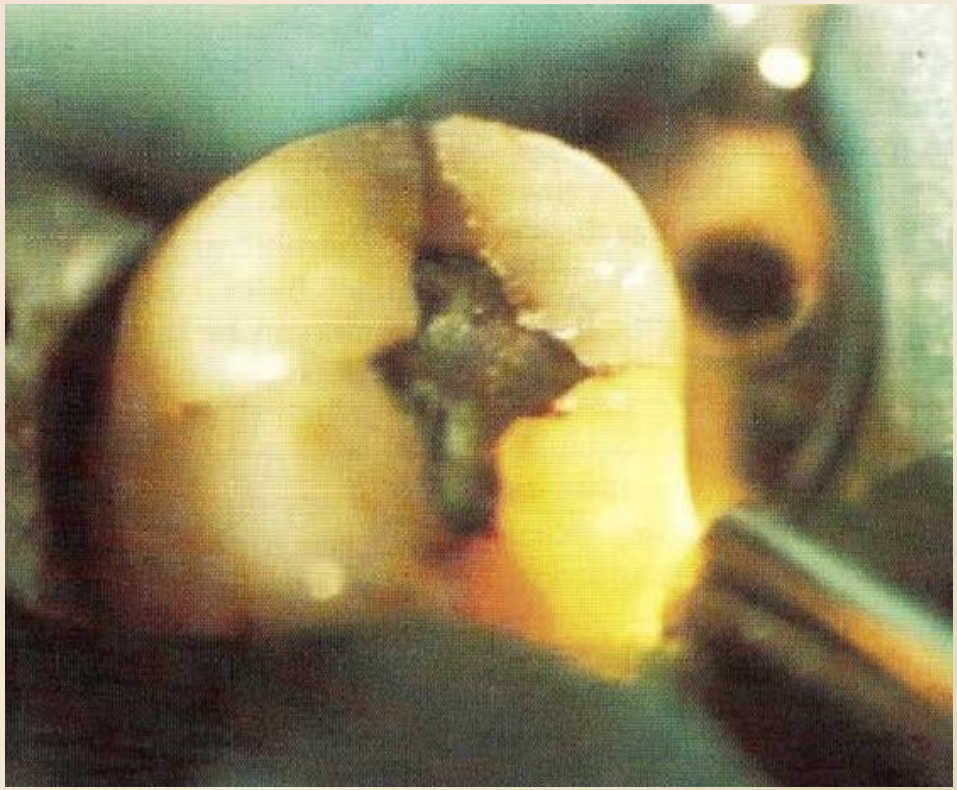
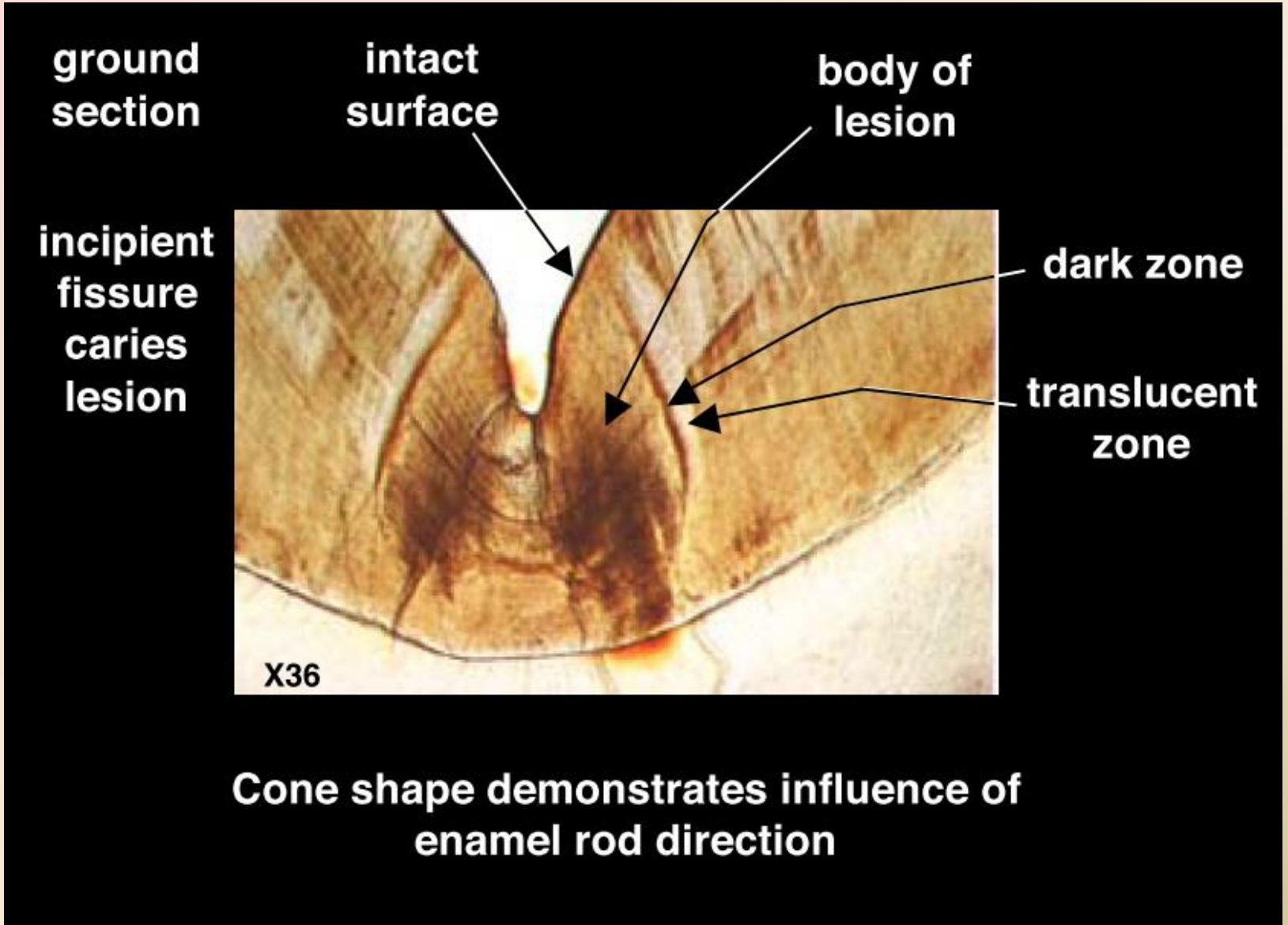


HISTOPATHOLOGY OF ENAMEL CARIES



ZONES OF ENAMEL CARIES

SURFACE ZONE

BODY OF THE LESION

DARK ZONE

TRANSLUCENT ZONE

HISTOPATHOLOGY OF CARIES

ground section

dentin caries lesion

4) surface zone of decomposed dentin

3) decalcified matrix with bacteria in tubules

2) zone of sclerotic dentin where mineral fills tubules & optical distinction between tubules & matrix lost

1) dead tracts: tubules containing degenerating odontoblastic processes

ZONES OF DENTINAL CARIES

NORMAL DENTIN

SUBTRANSPARENT DENTIN

TRANSPARENT DENTIN

TURBID DENTIN

INFECTED DENTIN

ICDAS (INTERNATIONAL CARIES DETECTION AND ASSESSMENT SYSTEM)

Carious Lesion Codes
0 = Sound tooth surface, no or slight change after prolonged air drying
1 = First visual change in enamel seen after prolonged air drying
2 = Distinct visual changes in enamel
3 = Localize enamel breakdown, no dentin involvement
4 = Underlying dark shadow from dentin (not cavitated into dentin)
5 = Distinct cavity with visible dentin
6 = Extensive distinct cavity with visible dentin

Prevention of dental caries

Initial lesions can be treated with preventive measures to arrest the process and remineralize the lost tissues

In contrast to the traditional management of dental caries based on surgical restoration of tooth damage alone, **current management strategies** explore treating dental caries based on an **individual risk assessment** of the patient

Caries assessment tool (CAT)

Caries-risk Indicators	Low Risk	Moderate Risk	High Risk
Clinical conditions	■ No carious teeth in past 24 mos.	■ Carious teeth in past 24 mos.	■ Carious teeth in past 12 mos.
	■ No enamel demineralization	■ 1 area of enamel demineralization	■ More than 1 area enamel ■ Demineralization (enamel caries "white-spot lesion")
	■ No visible plaque; no gingivitis	■ Gingivitis	■ Visible plaque on anterior (front) teeth
			■ Radiographic enamel caries
			■ High titers of mutans Streptococci
			■ Wearing dental or orthodontic appliances
			■ Enamel hypoplasia

Caries-risk Indicators	Low Risk	Moderate Risk	High Risk
Environmental characteristics	■ Optimal systemic and topical fluoride exposure	■ Suboptimal systemic fluoride exposure with optimal topical exposure	■ Suboptimal topical fluoride exposure
	■ Consumption of simple sugars or foods strongly associated with caries initiation primarily at meal times.	■ Occasional (i.e., 1-2) between-meal exposures to simple sugars or foods strongly associated with caries	■ Frequent (i.e., 3 or more) between meal exposures to simple sugars or foods strongly associated with caries.
	■ High caregiver socioeconomic status	■ Midlevel caregiver socioeconomic status (i.e. eligible for school lunch program or SCHIP)	■ Low-level caregiver socioeconomic status (i.e., eligible for Medicaid)
	■ Regular use of dental care in an established dental home	■ Irregular use of dental services	■ No usual source of dental care ■ Active caries present in the mother
General health conditions			■ Children with special health care needs ■ Conditions impairing saliva composition / flow

Risk Category

■ **High Risk:** The presence of a single risk indicator in any area of the "high-risk" category is sufficient to classify a child as being at "high risk".

■ **Moderate Risk:** The presence of at least 1 "moderate risk" indicator and no "high risk" indicators present results in a "moderate risk" classification.

■ **Low Risk:** The child does not have "moderate risk" or "high risk" indicators.

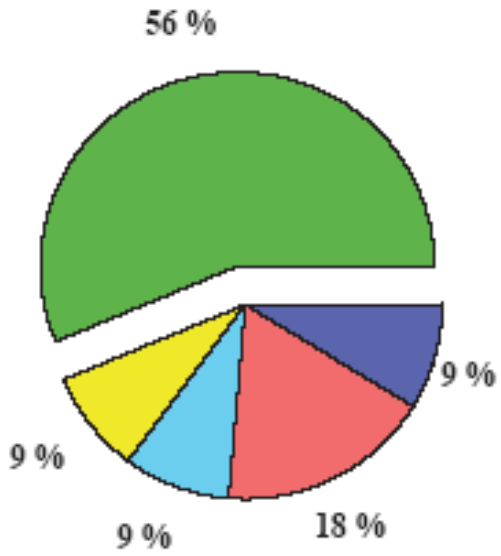
*AAPD, Council on Clinical Affairs, www.aapd.org

Cariogram

Cariogram

Name:
Ident.No.:
Date:
Examiner:

Country/area: Standard set
Group: Standard set



Actual chance to avoid new cavities	56 %
Diet	9 %
Bacteria	18 %
Susceptibility	9 %
Circumstances	9 %

Caries experience	3
Related diseases	0
Diet, contents	1
Diet, frequency	1
Plaque amount	2
Mutans streptococci	2
Fluoride program	2
Saliva secretion	0
Buffer capacity	0
Clin. judgement	1

Fig. 1. Example of the Cariogram® caries risk diagram for one individual in the sample .

Bratthall D, 1996

No Treatment and Watch

- Incipient caries are difficult to detect and it is equally
  as difficult to decide how to treat them.

- One treatment option is to not treat the lesion surgically but to treat the factors that caused the lesion and to observe its progression or reversal.

Classification

At tooth level:

- Fluorides
- Restorative materials
- Slow release fluoride device:

  Copolymer membrane with fluoride

  Glass beads

  Mixture of hydroxyapatite with sodium fluoride

Dental caries: a complete change over, Part III- changeover in the treatment decisions and treatments. Carounanidy U, Satyanarayanan R. J Cons Dent 2010; 13(4): 209- 217

Classification

- Topical application of Calcium and Phosphate complexes:
  - **CPP-ACP**
  - **Bioactive glass**
- Laser activated fluoride (LAF)
- Restoration of incipient lesions

Dental caries: a complete change over, Part III- changeover in the treatment decisions and treatments. Carounanidy U, Satyanarayanan R. J Cons Dent 2010; 13(4): 209- 217

Classification

At the microbial level

- Removal of thick biofilm – mechanical and chemical

- Mechanical blocking of susceptible tooth sites by sealants:
  - -Pit and fissure sealants
  - -Sealing the proximal lesions

- Antimicrobials

- Replacement therapy

Classification

At the salivary level

- Increase salivary flow
  Xylitol chewing gums

At the dietary level

- Dietary counseling

Fluorides

- Optimal level in drinking water 1 ppm (supplemental fluoride if lower)
- Toothpaste (0.1%F / daily or more)
- High F toothpaste (Rx 0.5%F /daily or more)
- Stannous fluoride gel (Rx 0.1%F /daily or more)
- Fluoride rinses
  - High dose/low frequency (0.1%F / weekly)
  - Low dose/high frequency (0.02%F /daily or more)

Fluorides

- Topical fluorides – more effective than systemic fluorides

- Fluoridated water (if locally available) and fluoride containing dentrifices - most common approaches to applying topical fluoride

- Professional and prescription fluoride treatments – gels and foams (maximum of 5000 ppm), rinses (223ppm) and varnishes (23000ppm)

Antimicrobial effect of Fluoride

- Inhibits glycolysis and glucose uptake – bacteriostatic and bacteriocidal
- Reduces ability to make extracellular matrix and reduces adhesion
- Acts as catalyst for remineralization
- Incorporated into mineral making it acid resistant

Remineralization

- Treating incipient caries lesions with fluoride is the hallmark of non-invasive remineralization therapy employed by dentists.

- The effectiveness of the fluoride ion to incorporate into a lesion of demineralized enamel hydroxyapatite, promote the establishment of sound fluoridated apatite, and ultimately reverse and arrest an area of incipient caries has been demonstrated in several studies (Biesbrock, *et al., 1998).*

Remineralization

- *Cariogenic priming*

- Incipient lesions in tooth pits and fissures respond less favourably to fluoride therapy than lesions isolated to smooth surfaces.

Theories surrounding the inhibition of fluoride's action in pits and fissures focuses on the significantly decreased substantivity and ability for fluoride ion to access and concentrate in aforementioned areas (Ogaard, 1990).

Remineralization

- Fluoride varn[illegible]apy can be increasingly

A nine month study assessed the effect of fluoride varnish (duraphat)on early enamel carious lesions in primary teeth. Children between the ages of 3 and 5 years were selected and randomized into varnish and control groups.

The experimental group received varnish at baseline and four months.

Control group – no intervention

Results:- significantly more inactivated lesion in the varnish group at 9 months

- Autio-Gold JT, Courts F., 2001

Slow release fluoride devices

- Copolymer membrane
- Glass beads
- Combination of NaF and hydroxyapatite

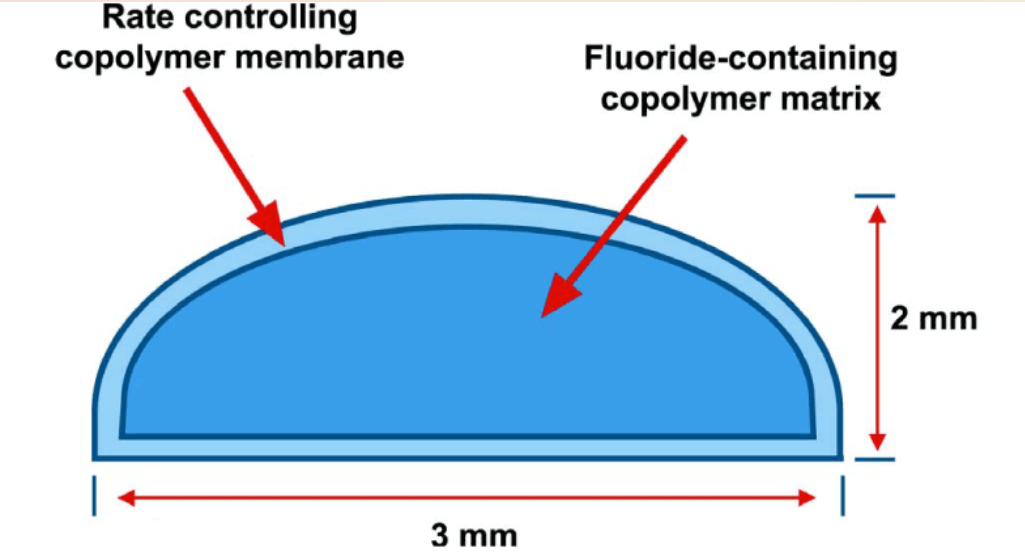


FIGURE 1- Schematic cross-sectional view of the copolymer device, which originally had 8 mm in length, 3 mm in width, and 2 mm in tickness. Modified from Mirth, et al. (1982)

FIGURE 5- Latest glass device and bracket attached to upper first permanent molar tooth

CPP-ACP

In a 24 month clinical trial, progression and regression of enamel approximal caries were investigated after 2720 subjects randomly chewed gums with or without 54 mg of CPP-ACP

Results: for subjects chewing the CPP- ACP gums, the odds of a surface experiencing caries progression were 18% less than those of a surface experiencing caries progression for subjects chewing the control gum

- Morgan MV, Adams GG, Bailey DL, et al. 2008

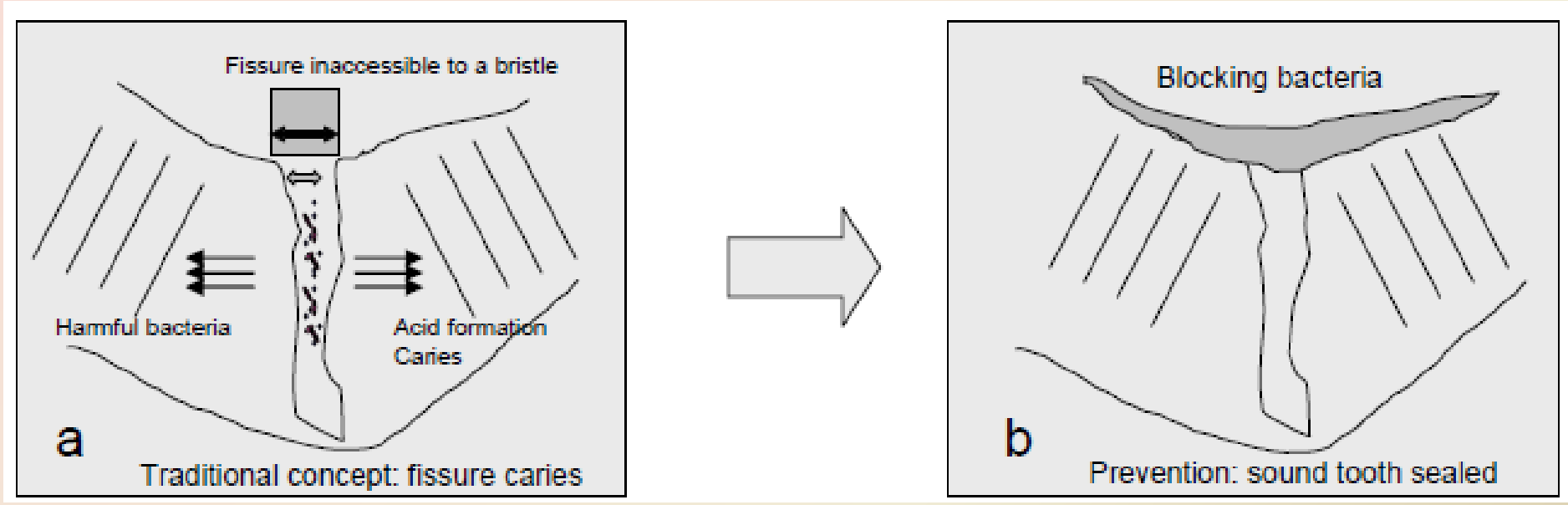
Laser activated fluoride

- The beam changes the structure of hydroxyapatite to ceramic like material – acid resistant
- Greatest protection - combination of laser irradiation of enamel in combination with topical fluoride application
- Reduce the critical pH at which enamel dissolution occurs from 5.5 to 4.31.
- This critical pH is further reduced in the presence of fluoride in concentrations as low as 0.01 ppm.

Sealants

- Sealants used to occlude pits and fissures
- Eliminate the geometry that harbours debris and bacteria
- Eliminates energy (sucrose) supply to existing bacteria

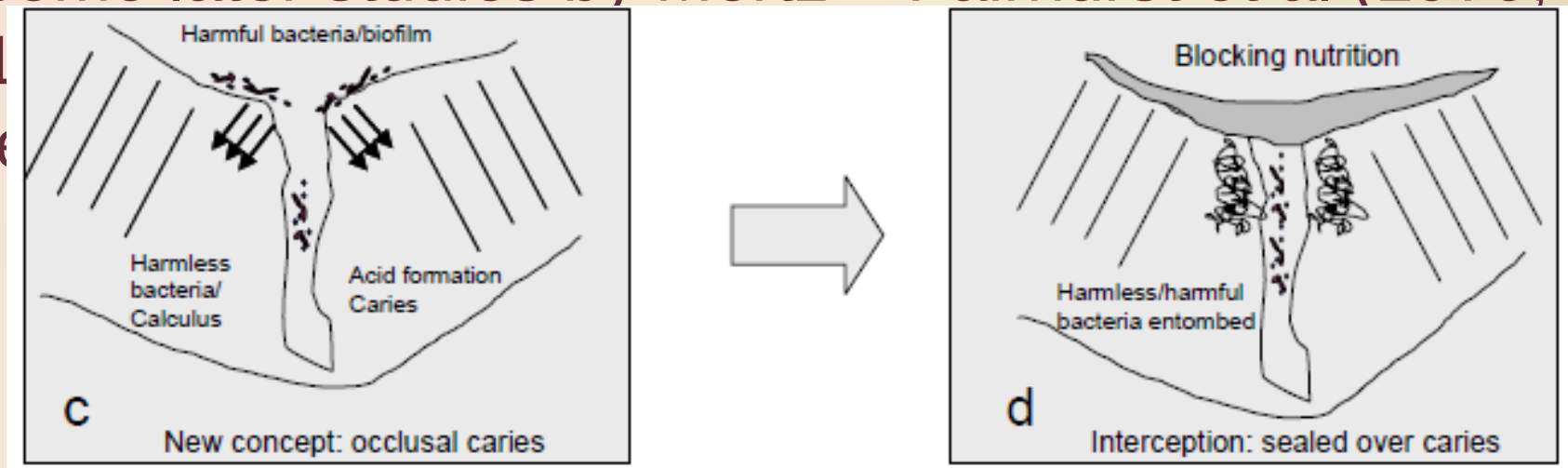
Sealants


Fissure inaccessible to a bristle
Harmful bacteria
Acid formation
Caries
a
Traditional concept: fissure caries
Blocking bacteria
b
Prevention: sound tooth sealed

Sealants

- Given that sealants occluded the fissures, the question
  about whether caries could progress or not beneath a
  sealant soon arose.

- The studies of Handelman (1972,1973, 1976) and some later studies by Mertz – Fairhurst et al (1979, 1
  le



Sealants

- When a sealant is placed over an incipient carious lesion, meaning a stained fissure in which softness at the base could be detected but where cavitation had not yet occurred, caries does not progress provided the sealant remains intact.

- Sealants are retained on the carious teeth just as well as on sound teeth, and neither lesion depth nor microbiologic counts progress under an intact sealant.

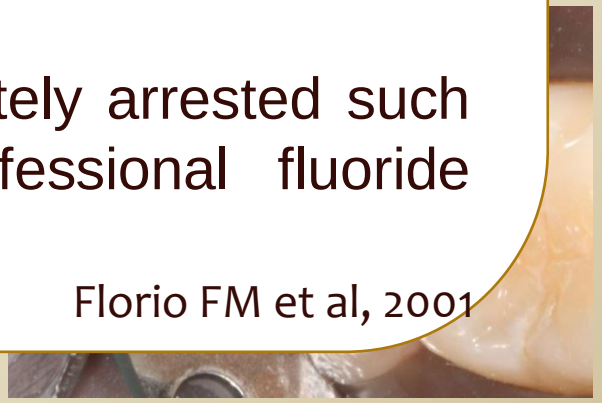
Sealants

- A 12 month study – to evaluate non invasive treatment on occlusal caries lesions
  Children at the age of 6 were randomized into two test groups and one control group

  Test group 1 – **fluoride leaching resin modified glass ionomer sealants**
  Test group 2 – **professional fluoride varnish (2.26% NaF Duraphat) every 6 months**
  All groups including the control were instructed to rinse with 0.2% NaF weekly and given oral hygiene instructions

  Results:- indicated that sealants completely arrested such enamel lesions compared to the professional fluoride varnish and control

  Florio FM et al, 2001



Sealing proximal lesions

Split mouth study – Danish and Columbian young adults
Included approximal lesions with radiolucencies in enamel or in the outer third of the dentin – sealed

After 18 months – sealing of the lesion was more effective for preventing lesion progression than flossing as 22% of the test lesion had progressed compared to 47% of the control lesions ($p<0.05$)

Even after 3-4 years – 22% of the test lesions had progressed compared to 68% of the control lesions

- Martingnon et al, 2006

Approximal sealing – polyurethane tapes

2 year study – patch sealed initial approximal lesions were compared with lesions treated by flossing at home and by fluoride application.

The sealants showed good retention, marginal adaptation and colour

In both groups, **most lesions were stable** and there were no significant difference in the numbers regressing and progressing

- Alkilzy et al, 2009

Sealants

- Tw
  c
- G

pro

- C
- R

A study comparing the effects of resin based sealants, Fluoride containing sealants, fluoride varnish and a glass ionomer cement – stabilizing or reversing incipient caries

Conclusion –

GIC was the most effective in reducing carious areas and the most efficient at inhibiting new caries lesions and demineralization of intact enamel adjacent to sites where it was placed

Trairatvorakul C, Kladkaew S, Songsiripradabboon S. Active management of incipient caries and choice of materials. *J Dent Res.* 2008:87(3):228-232.

Restoration of Incipient Lesions

An experienced clinician - judge patient factors and make a decision as to whether sufficient indications exist for restoration

Indications for restorations

•Poor oral hygiene •H/o caries and numerous restorations on the contralateral surface or throughout the mouth	•Change in color – transilluminated •Radiographic evidence – extends to the dentinoenamel junction	•Low frequency of routine dental care •Degree of caries susceptibility •Age

Blocking plaque

- Reducing the plaque mass is important in increasing the pH and facilitating remineralization – mechanical and chemical methods

- There is good evidence that an early lesion can be reversed with plaque removal (Wilding and Solomon,1996)

- Inhibition of glucosyltransferase, GTF - reducing sticky glucans formation.

- Interfering with these molecules prevents bacterial adhesion and co-aggregation.

Chlorhexidine

- Broad spectrum antiseptic
-

10 % coating was used – 25% reduction in caries in an adult risk population

- Athena S. Papas, 2010

A combination of chlorhexidine and 0.05% NaF rinse was administered to the irradiated patient with low salivary flow. Results – possible remineralization of incipient lesions

No randomized clinical trials have shown that chlorhexidine is directly responsible for reducing incipient caries

- Katz S., 1982

Other antimicrobial agents

- 10% povidone iodine

- 0.10% Sodium hypochlorite – anti microbial rinses

Replacement therapy

- A subtler form of antibacterial therapy.
  - consists of either replacing Streptococci mutans with a more benign counterpart or a mutant form of mutans - - **lacks the lactate dehydrogenase gene.**

- **Another approach** - transferring the arginine deminase gene from Streptococci sanguis, which is responsible for base production, into Streptococci mutans to counteract its acid production.

Xylitol

- Not met[illegible] the oral bacteria

24 month prospective cohort study – effect of chewing xylitol gum on incidence and progression of dental caries was tested in a sample of 274 children, aged 8 and 9 years of low SES and high caries rate

Test groups – 15% and 65% xylitol gums, 3 times/ day
Control group – no gum

Results for the two gum chewing groups were similar- had beneficial effect on caries process for all types of tooth surfaces, especially for bucco-lingual surfaces

- Kandelman D, Gagnon G., 1990

- [illegible]ns

Saliva

- Adding peptides to saliva augments and enhances the protective qualities of saliva by mimicking mucins.
- These are currently in clinical trial and would be very valuable for patients with xerostomia.
- These peptides would most likely be added to mouthrinses and dentifrices because frequent applications would be necessary for successful saliva enhancement (Mandel, 1996).

Diet modification

- Diet counseling is the major part
- Decreasing sugar consumption between meals
- Including preservatives in food with enhanced antibacterial activity
- Addition of inhibitors of demineralization into food e.g. phosphates.
- More frequent use of protective foods such chocolates (containing polyphenols), oat, pecan hulls, cheese and other milk products is beneficial.

Air Abrasion

- **One method for the treatment of incipient caries is to remove it with air abrasion.**

Air abrasion also has been recommended to aid in the diagnosis of questionable pit and fissure lesions (Hamilton, 2002.)

- Air abrasion was actually develop 1940s by Dr. Robert B. Black

Air Abrasion

- A powerful, focused stream of 27 micron aluminium oxide (Al_2O_3) particles - propelled by compressed air or bottled carbon dioxide or nitrogen gas.

- The abrasive particles strike the tooth with high velocity and remove small amounts of tooth structure.

Air Abrasion

- When dealing with possible incipient caries, air abrasion can be used to explore that possibility.
- The clinical applications - removal of organic debris from pit and fissures, if incipient caries are found under this debris the abrasives can be continued until sound tissue is reached.
- When caries are limited to the enamel then a sealant or flowable resin based composite can be placed.

Air Abrasion

- Should a suspected incipient lesion be explored and found to not be carious, then the air abraded surface will be etched and have a sealant applied.

- Air abrasion has been found to enhance enamel bonding as long as an acid etchant is still used (White et al., 2000).

Air Abrasion

Drawbacks:-

- The lack of high-speed suction
  - the use of air- propelled microparticles made a mess and posed an aspirational or respiratory hazard to both the patient and the practitioner.

- Retention specific preparations involving well - defined walls and margins could not be achieved with air abrasion.

Caries infiltration

- **Novel technology for arresting dental caries – uses**

  low viscous resin

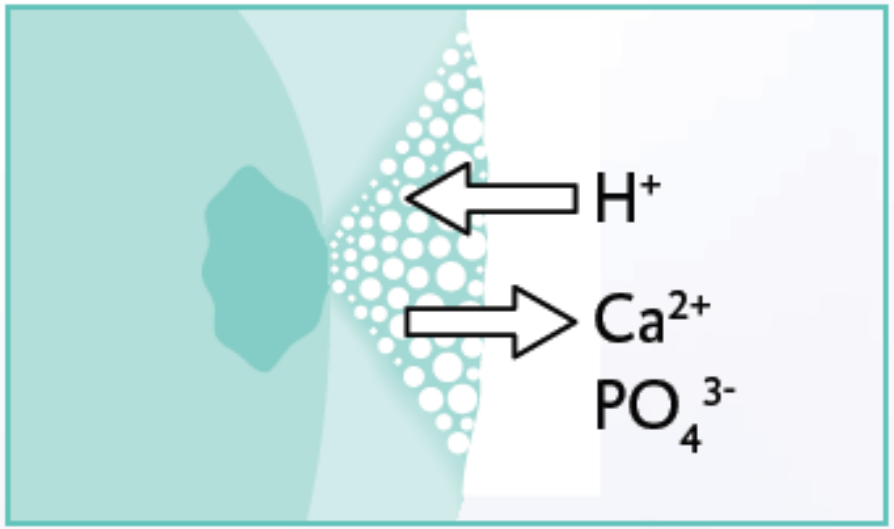
- A new intermediary treatment option between prevention and restorative therapies
- Microinvasive approach-smooth surface and proximal carious lesions.
- Works by capillary action – has high penetration coefficient

Caries infiltration

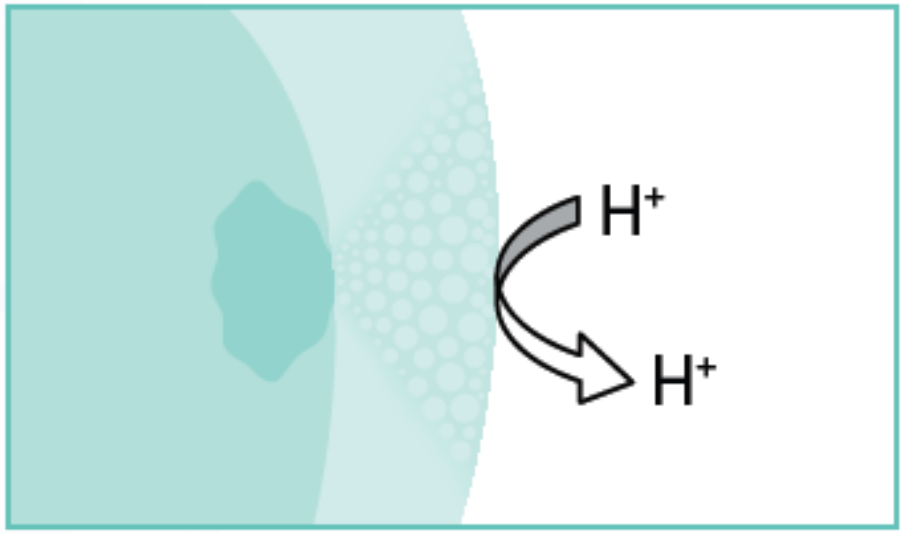
Clinical data on primary teeth (12 month follow up) and permanent teeth (18 month follow up) revealed a significantly reduced caries progression for infiltrated lesions (31%, 11%) compared with lesions that were only subjected to fluoride applications or to oral hygiene (61%, 38%)

- Paris et al, 2009

Caries infiltration



Cariogenic acids demineralize the enamel.



Icon blocks the diffusion paths.

Caries infiltration

- For proximal lesions, the affected teeth are slightly separated with dental wedges
- The surface area of the lesion is eroded with 15% HCL gel
- Pore system is then dried with ethanol
- Icon is then applied onto the lesion body.
- Excess material is then removed and the material is lightly cured
- Total treatment time per lesion – about 15 minutes

Benefits of caries infiltration

- Enables immediate treatment of lesions
- Ends wait and see approach
- Arrests caries progress without unnecessary loss of healthy tooth structure
- Cosmetic treatment of cariogenic white spots in one patient visit
- No drilling or anesthesia required
- Prolonged life expectancy of tooth
- Not just minimally invasive dentistry.. Micro-invasive!

Catch the caries ‘Young’

- Instead of the ‘drill and fill’ technique followed after the tooth is cavitated – lets prevent the lesion from cavitation

- It is a cost effective, tooth conserving and patient friendly method

‘Lets hear the cries of the tooth
And wash their tears with the hands of prevention’

Conclusions

- The more we can educate ourselves as operators of the many interventional methods to reverse the caries cycle and promote remineralization, the more tooth structure we will conserve, disease we will prevent, and patient gratification we may enjoy.

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Thank You