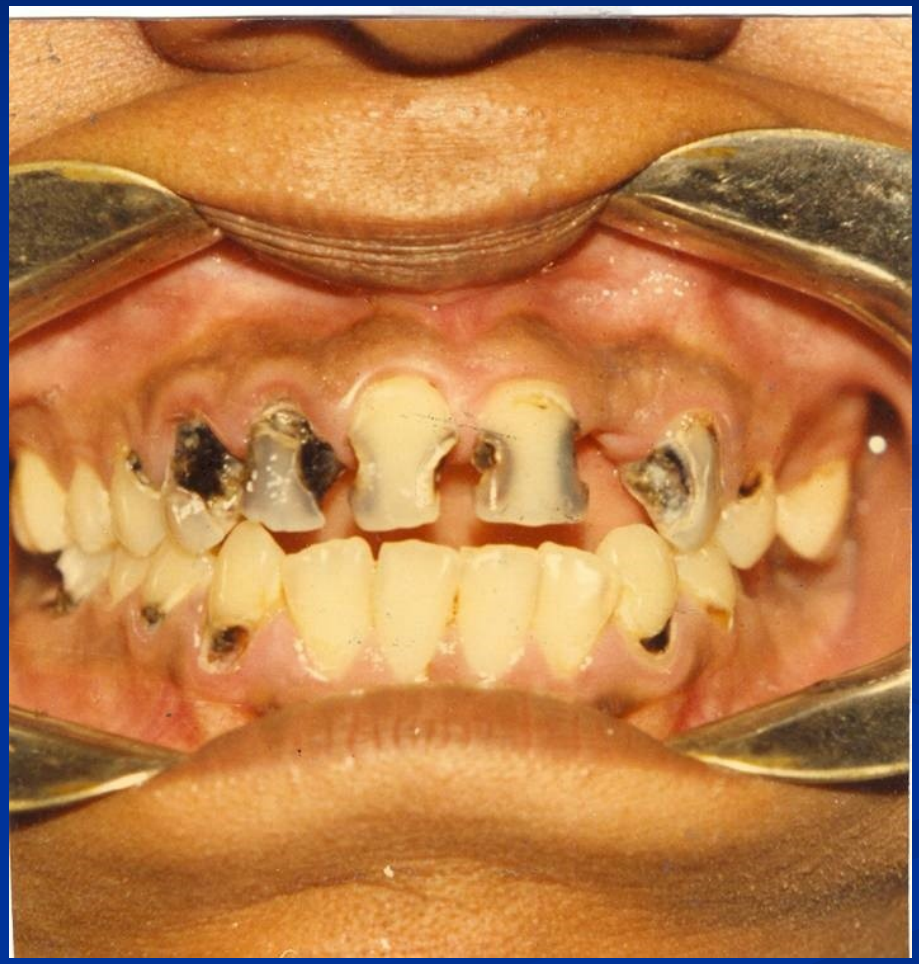
Dental caries

V. Žampachová



Pathologic loss of tooth structure

- non-bacterial (mechanical – abrasion, attrition; non-bacterial chemical – erosion; pathological resorption, etc.)
- bacterial disease associated – dental caries

Dental caries is a sugar-dependent infectious disease.

Dental caries

- Multifactorial dynamic process, partialy reversible
- Involves the interaction of inborn or acquired host factors (tooth surface, saliva, acquired pellicle), diet, dental plaque (biofilm).
- Caries does not occur in the absence of either plaque or dietary fermentable carbohydrates.
- Dental caries can be modified by protective factors.

Etiology

- Classic triad of essential factors necessary to development of a carious lesion. All must be present for caries to occur: bacteria, degradable carbohydrate and susceptible tooth structure.
- Time factor important

Dental caries - etiology

- bacterial plaque biofilm
- cariogenic bacteria
- plaque biofilm stagnation sites
- susceptible tooth surfaces
- fermentable bacterial substrate (sugar)
- time

Dental caries - etiology

- possible interventions at any level
  - reduced sugar intake
  - avoid frequent sugar intake (snack)
  - stimulate sugar clearance incl. salivary flow
  - reduction of Str. mutans
  - reduction of susceptible teeth surface
    - fluoridation
    - prevention during posteruptive maturation
    - remineralising solutions
    - proper restoration, fissure sealing

Multifactorial process

Disease factors

Genetic ↔ Environmental

↕

Biological

Social

Behavioral

Psychological

Dental caries incidence

- changes in the prevalence of dental caries (↓ of deciduous t. caries, ↓ of smooth surface caries, ↑ of root caries)
- changes in the distribution and pattern of the disease in the population
- improved diagnosis of noncavitated, incipient lesions and treatment for prevention and arrest of such lesions
- restorations: repair the tooth structure, possible caries stop, but commonly limited life span

Teeth and caries

- Tooth factors: location, morphology, composition, ultra-structure, post-eruptive age of the tooth.
- Teeth have a high resistance to caries (low caries prevalence in primitive humans)
- Modern humans have restricted this natural resistance by modifying our diets.

Structural resistance

- Larger and more uniform crystals ↓ the specific surface area and reactivity (solubility).
- More closely packed crystals ↓ the space for water and diffusion pathways between crystals.
- Spaces between crystals → enamel a microporous material. Water - diffusion channel for acids to attack the crystals.

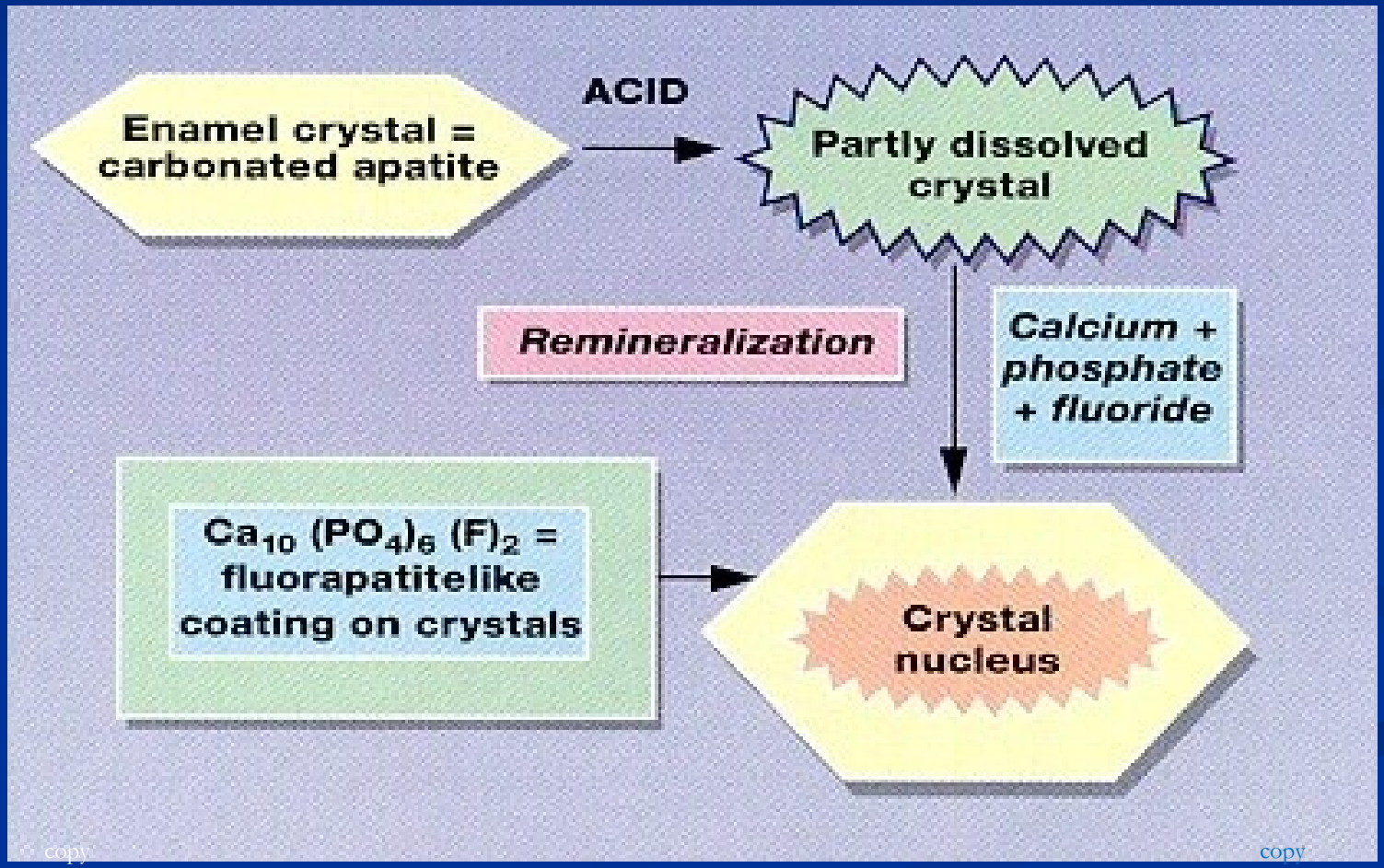
Posteruptive maturation

- Caries susceptibility greatest immediately subsequent to eruption, tends to decrease with age.
- Post-eruptive maturation process with changes in the composition of the surface enamel.
- Related to the demineralization-remineralization dynamics.

Demineralization : remineralization

- Dynamic equilibrium state of the enamel surface with its local oral environment (plaque fluid and saliva), constant movement of ions in and out.
- Drop of the pH of plaque → the mineral phase of enamel begins to dissolve.
- Critical point between 5.0 and 6.0

Demineralization : remineralization



Demineralization : remineralization

Protective factors	Pathological factors
Saliva – flow + components	Reduced saliva functions
Proteins, antibacterial agents	Bacteria: str. mutans, lactobacilli
Fluoride, calcium, phosphate	Diet: carbohydrates + frequency
↓	↓

Δ

no caries **caries**

Demineralization

- **De/remineralization process -** the more soluble carbonate-rich apatite lost, replaced by apatite lower in carbonate and higher in fluoride (if fluoride present in the oral environment).
- Reprecipitated crystals - larger than the original crystals, creating hypermineralized areas of enamel.
- Response of the enamel - ↓susceptibility to caries occuring with age.
- The effectiveness of fluoride in caries prevention can be largely attributed to its ability to enhance the remineralization process.

Demineralization : remineralization

- Demineralization of relatively unaffected surface zone ↑ rate of the progress of the lesion.
- If the surface layer above a lesion can be „strengthened“ through fluorides or mineralizing solutions, the lesion can become arrested, and the process reversed.
- If the surface area is plaque-free, then the saliva itself, (supersaturated with calcium and phosphate) can remineralize the initial lesion as well.
- The average time from the stage of a white spot lesion to clinically detectable caries - approximately two years.
- A high frequency of exposure to sucrose – acceleration of demineralization;
- Exposure to fluorides favors remineralization.

Etiology of caries

- Decalcification by bacterial acid followed by destruction of all other tooth tissues
- Bacteria, dental plaque
- Role of carbohydrates

No theory is universally accepted

- Acidogenic theory
- Proteolysis chelation theory
- Proteolytic theory

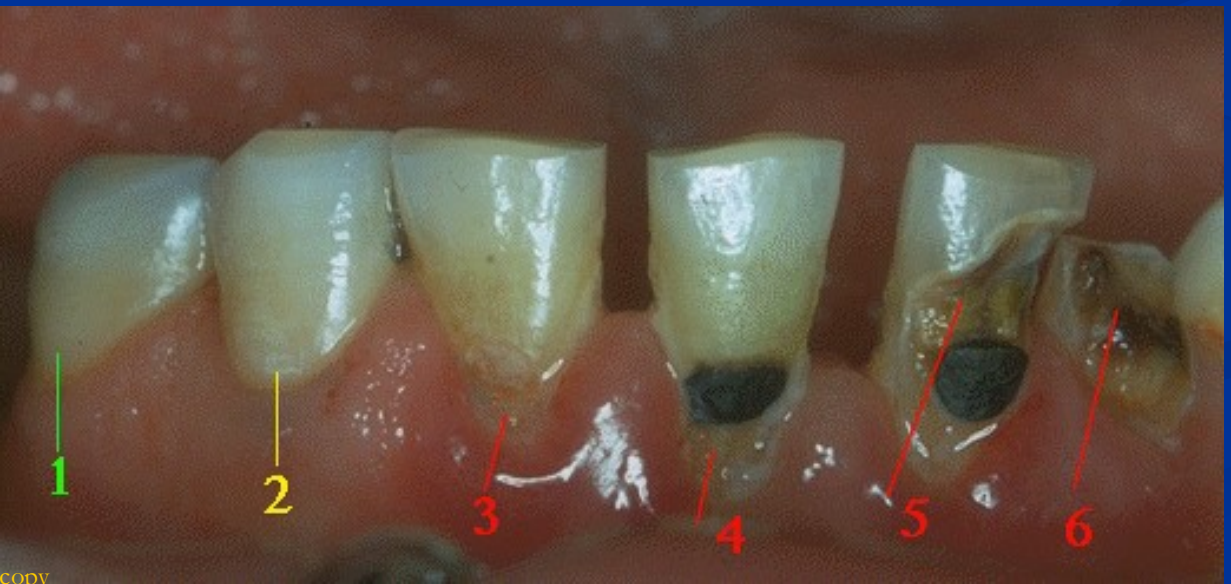
Acidogenic theory

Dental caries is a sugar-dependent infectious disease.

- Acid produced from metabolism of carbohydrate by plaque bacteria → drop in pH at the tooth surface.
- In response, calcium and phosphate ions diffuse out of enamel → demineralization.
- This process is reversed when the pH rises again.
- Caries is a dynamic process characterized by episodic demineralization and remineralization occurring over time.
- Predominant destruction → disintegration of the mineral component → cavitation.

Proteolytic theory

In addition to acid, proteolytic substances produced by plaque bacteria break down the organic portion of enamel and dentin



Evolution of caries

Proteolysis chelation theory

- Bacterial attack on enamel is initiated by keratinolytic bacteria causing breakdown of enamel protein, mostly keratin
- Organic and inorganic portion of enamel undergoes demineralization by formation of calcium chelates, even at neutral pH
- Mucopolysaccharides may act as chelators

Dental plaque

Yellowish white soft, amorphous material deposited on tooth surface

Formation

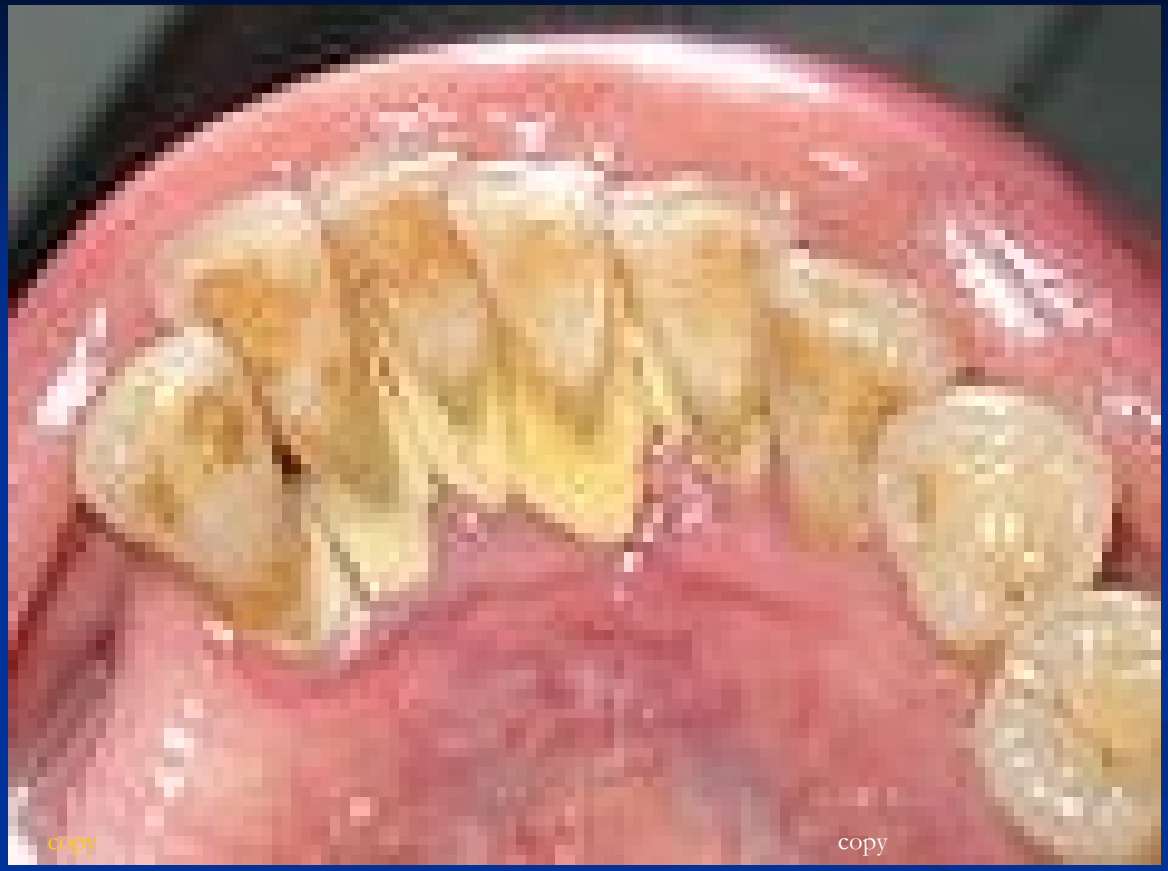
Adherent layer of mucinous material from saliva.

Colonisation of this layer by diffusion of microorganisms. Production of polysaccharides - glukans

Within 48 hrs the whole layer owergrown by microorganisms.

Dental plaque

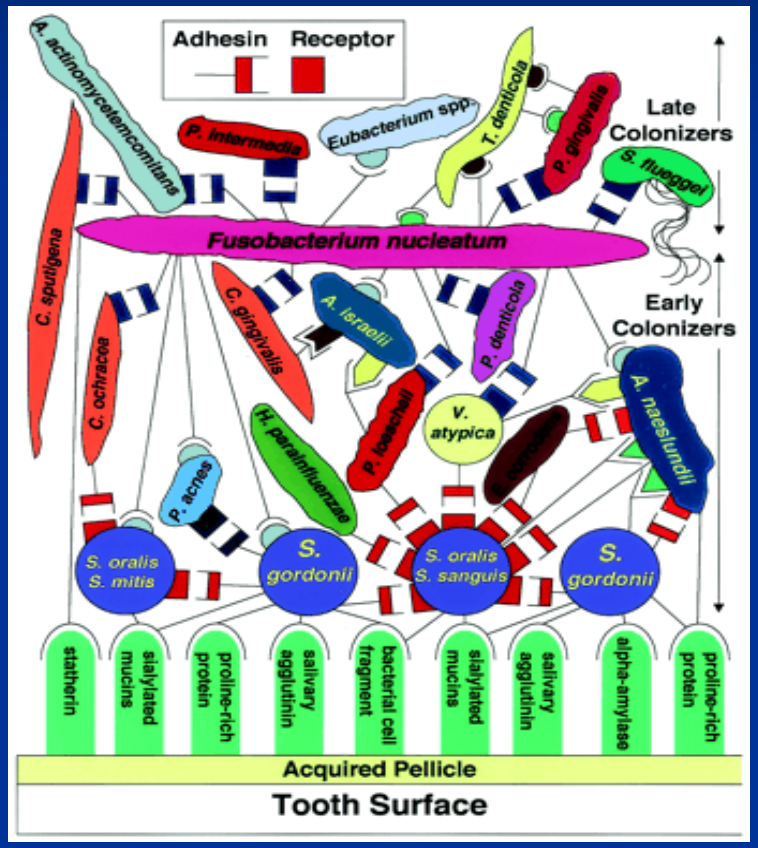
- A number of endogenous oral microorganisms in dental plaque can contribute to the caries process:
  - *mutans streptococci* (*S. mutans*, and *S. sobrinus)*
  - *S. sanguis* and *salivarius*, and other non-mutans species
  - *Lactobacilli species*
  - *Actinomyces* species
  - yeast
- Even in a caries free mouth, 1 ml of saliva contains 10-100,000 endogenous microorganisms.

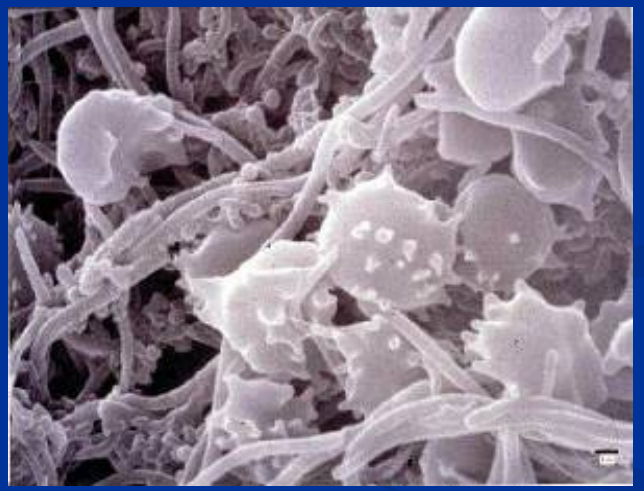


Dental plaque

Heavy staining and calculus deposits exhibited on the lingual surface of the mandibular anterior teeth, along the gumline.

Dental plaque





(Kolenbrander, *Microbiol Mol Biol Rev*, 2002)

Properties of cariogenic bacteria

- acidogenic
- able to produce low pH (at least pH 5) to start decalcification
- acidoresistant, acid production at any pH
- possibility of attachment - to adhere to smooth tooth surface
- production of adhesive/sticky insoluble polysaccharides – glukans
- survival in the mixed bacterial ecosystem - competition

Dental plaque

- Initial colonization of the plaque biofilm on a tooth surface is predominately S. *sanguins and S. salivarius.*
- Shortly after initial adherence to the tooth, *Streptococcus mutans* a major component of the biofilm.
- *Streptococcus mutans* highly probably the most virulent of the organisms participating in dental caries.

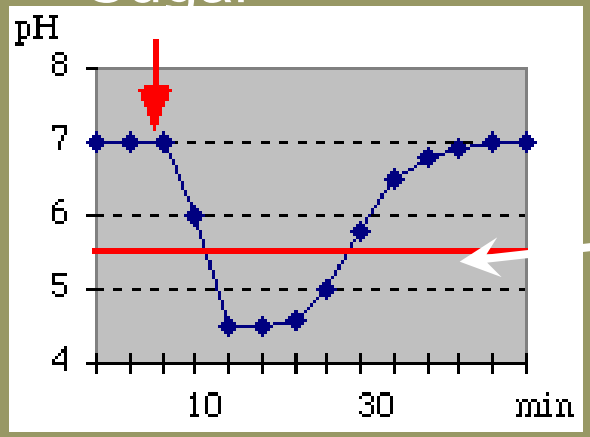
Dental plaque

- Maturation of the plaque - a shift from a predominating aerobic Gram positive cocci to anaerobic Gram negative rods.
- With progress to cavitation (particularly advancing into the dentin) *lactobacilli* favored in sheltered, highly acidic environment.
- The process of enamel demineralization and eventual cavitation related to bacterial succession, one organism initiating the plaque, while subsequently another organism takes over.

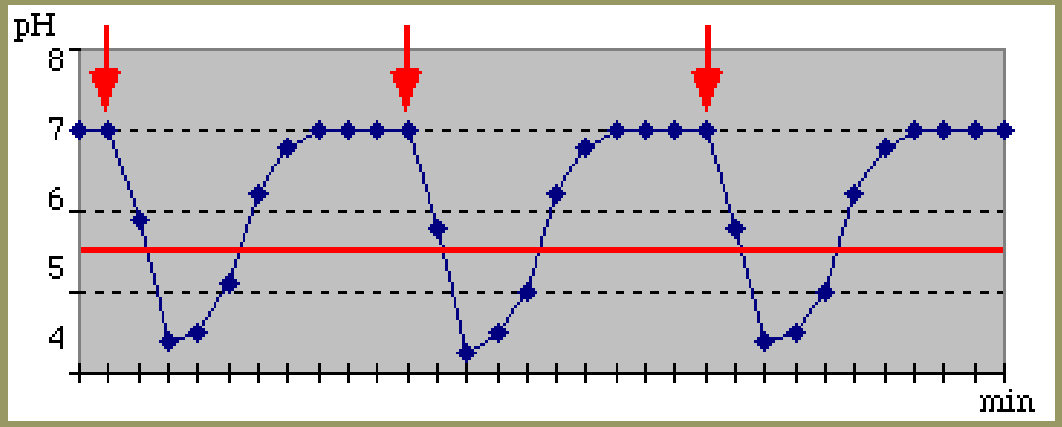
Dental plaque pH

- The pH of dental plaque normally close to neutrality.
- After ingestion of a fermentable carbohydrate (sucrose, etc.) the plaque bacteria produce acids → drop in the pH level.
- pH levels lower than 5.5 can initiate demineralization (after a sucrose rinse, the pH value can fall to as low as 4.0).
- Low pH levels→ calcium and phosphate ions begin to dissolve out of the enamel, so as long as the environment remains sufficiently acidic.

Sugar



Demineralization occurs



Properties of bacteria

Ability to produce acid by fermentation of sugars

Ability to polymerise sugars into long chain polysaccharides → plaque adheres firmly to the tooth surface, bacteria adhere to each other

Lactic acid (mainly), acetic acid

Cariogenicity of microbes

- *Streptococcus mutans/sobrinus*
  - Cariogenic properties
  - Major source of demineralization
    - Highly acidogenic
    - Extracellular polysaccharide

      from sucrose – insoluble, + reserve energy source
    - Adheres to pellicle
      - So do most oral streptococci
    - Transmisible - mother/caregiver to child
      - So are all oral bacteria
  - Microcolonies - localized zones of high acidity in protected sites
    - Occlusal pits and fissures; interproximal contacts

Microbes as risk factors

- Necessary, but not sufficient
- High *S. mutans* levels in saliva/plaque ↑ the risk
  - Longitudinal studies
    - Most people who get new lesions will have „high“ levels **BUT**
    - Many people with „high“ levels won't get new lesions
- The majority of oral streptococci belong to non-mutans species
  - *S. mutans* - a minority streptococcus - not a good competitor
  - High % of acidogenic non-mutans = increased risk?
  - Low % of acidogenic non-mutans = decreased risk?
- Other species may moderate risk
  - *Veillonella* may be related to lower lactate levels

Antimicrobial strategies

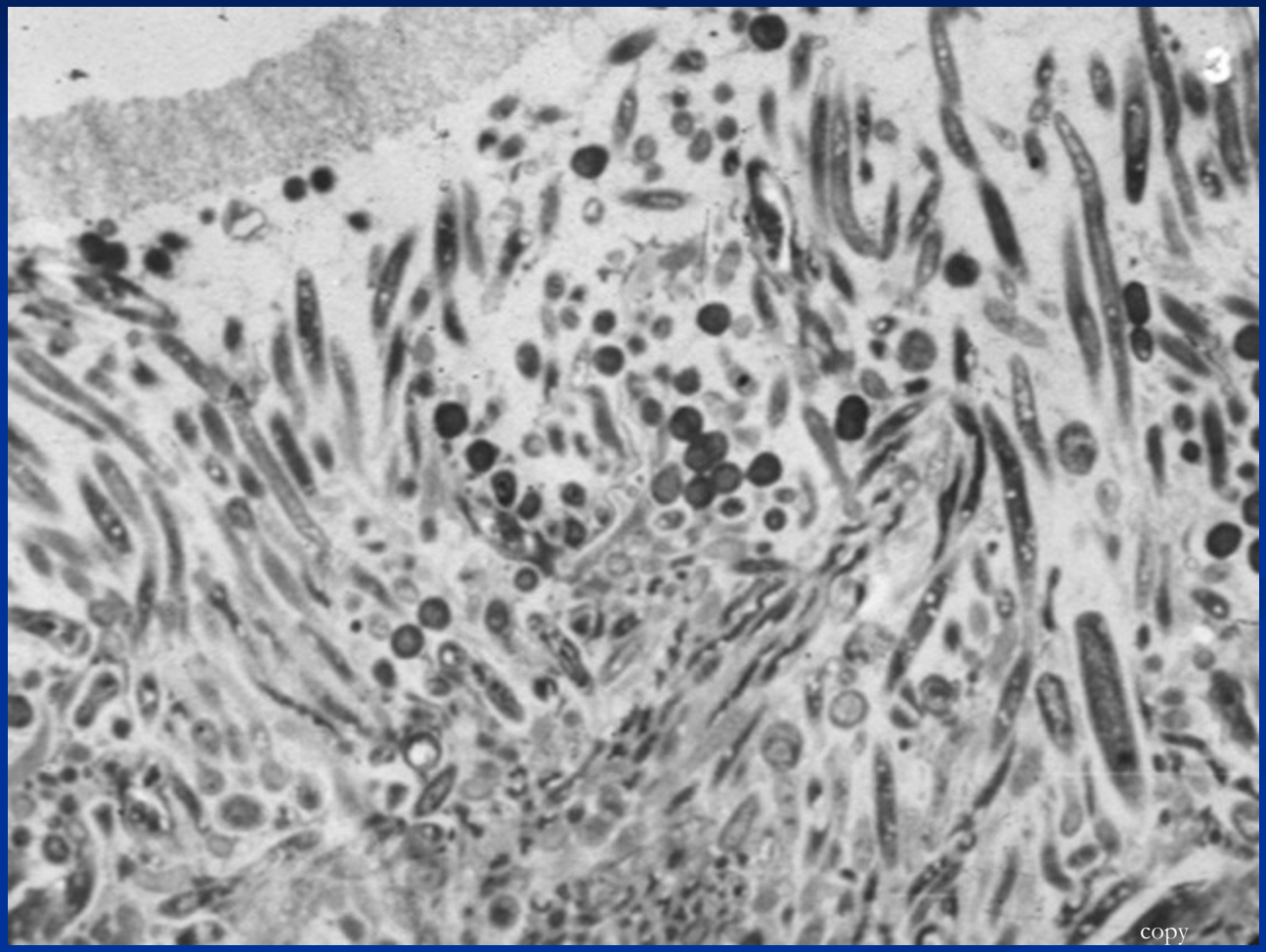
- Targeted attacks on mutans streptococci
  - Fundamental concept - *S. mutans* is the main demineralizer
  - Caries vaccines - results not impressive
    - Secretory immune system (S-IgA) is tolerant of oral microbes
  - Topical antibodies - results not impressive
  - Antimicrobial peptides combine *S. mutans* pheromones
- Broad-spectrum attempts to eliminate/limit biofilm
  - Allows for the possibility of other acidogenic species
  - Systemic antibiotics (fungal overgrowth)
  - Chlorhexidine rinses or varnishes (recolonization from reservoirs)
  - Quorum sensing inhibitors
- Replacement with „probiotics“, natural or genetically engineered
- All approaches have limitations, possible risks

Plaque composition

Other than organisms:

- Inorganic: calcium, sodium, potassium, phosphorus
- Organic: proteins, lipids, reactive inflammatory and other cells.

Dental plaque (EM)

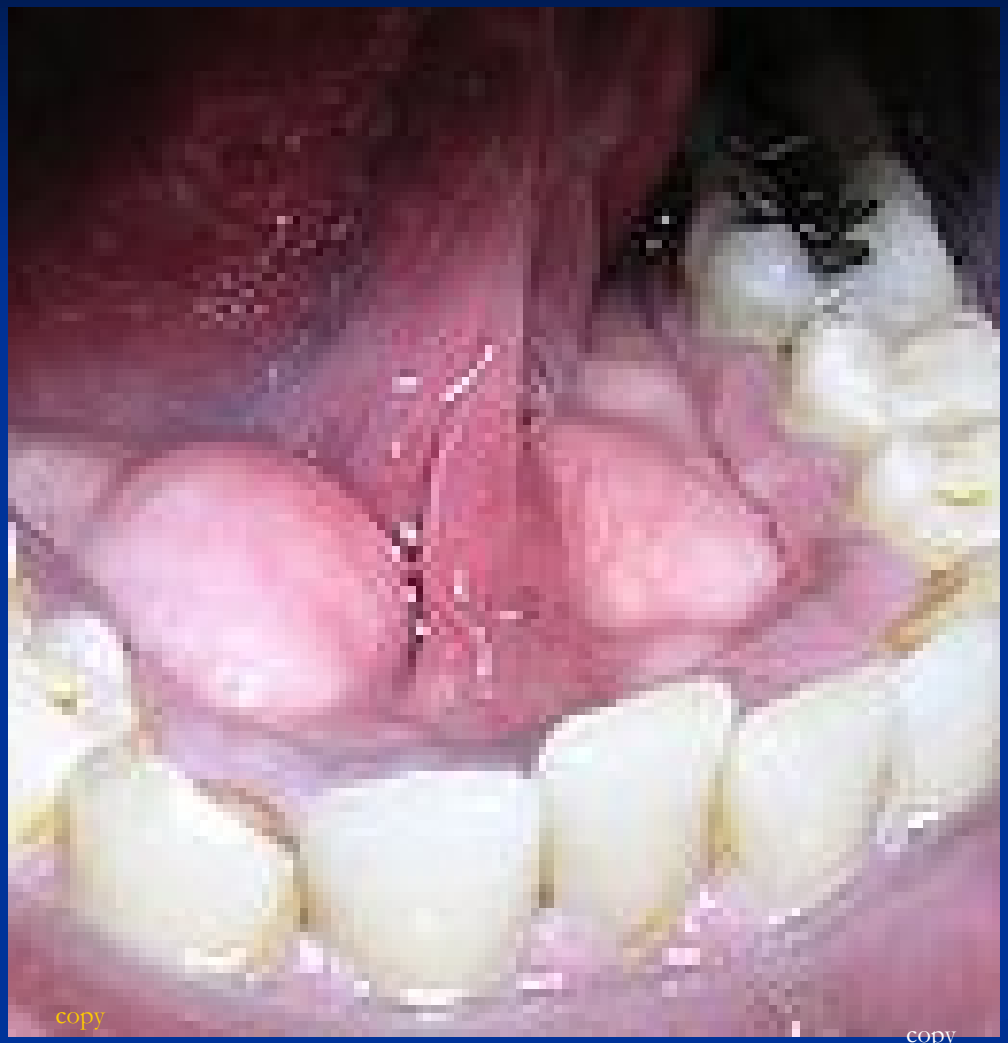


Growth of the plaque

- Multiplication of existing bacteria
- Addition of new bacteria
- Accumulation of metabolic products of bacteria
- Food debris from diet

Dental plaque

- Acids released from dental plaque lead to demineralization of the adjacent tooth surface, and consequently to dental caries.
- Saliva unable to penetrate the build-up of plaque and thus cannot act to neutralize the acid produced by the bacteria and remineralize the tooth surface.
- Cause of irritation of the gums around the teeth → possible gingivitis, periodontal disease and tooth loss.
- Plaque - eventually mineralized → calculus (tartar).



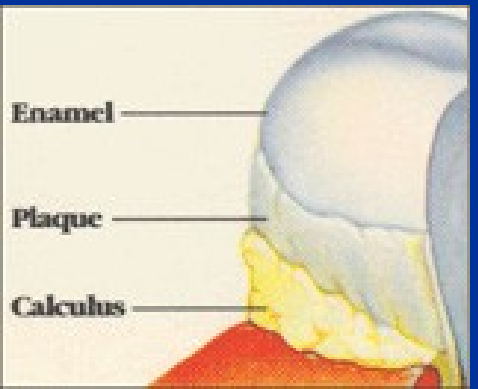
Inadequate removal of plaque caused a build up of calculus (dark yellow color) near the gums on almost all the teeth.

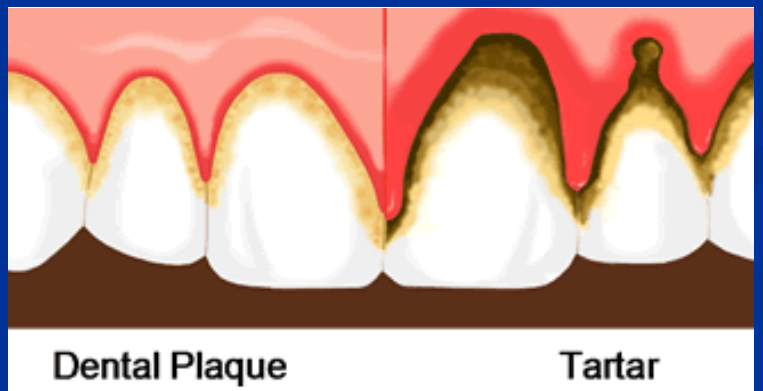
Dental calculus (tartar)

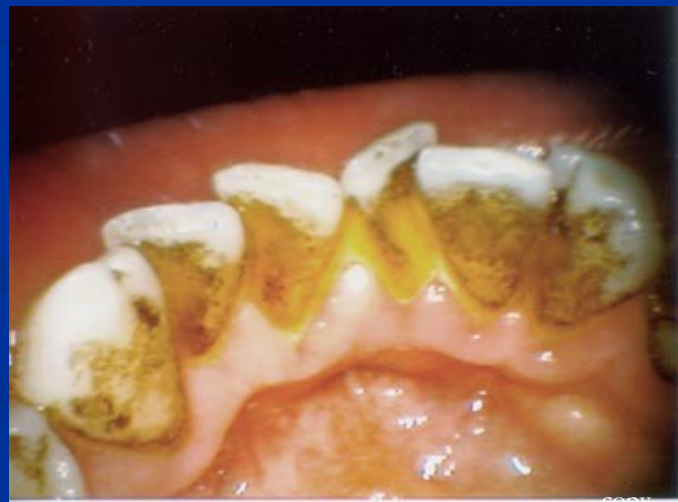
- Hard deposit formed on the tooth (due to mineralisation of dental plaque)
- Plaque converted to calculus in 50 – 60 days

Classification

- Supragingival – coronal to gingival margin
- Subgingival – below the crest of gingival margin







Composition:

- 70-90% inorganic material
- 10-30% organic material

Calculus formation can result in a number of clinical manifestations:

including foetor ex ore (bad breath)

receding gums,

chronic gingival inflamation.

Calculus

Supragingival calculus:

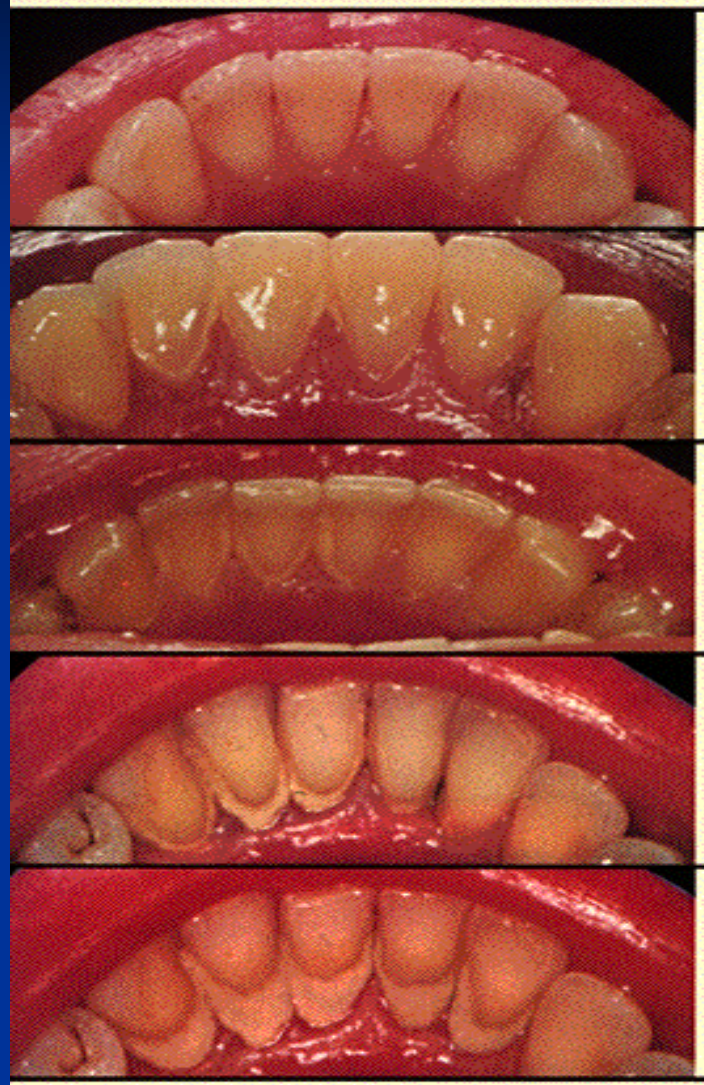
Colour: yellowish to white, blackish

- Consistency: clay-like
- Maximum in upper buccal region of molar teeth, lingual and interproximal surface of lower to anterior teeth.

Subgingival calculus:

- Dense brown to greyish black in colour

CALCULUS GRADING SCALE



0 No calculus

1 **TRACE**
Trace levels of calculus at gingival margin or between teeth

2 **SLIGHT**
Calculus deposits 1 mm or less

3 **MODERATE**
Calculus deposits 1 to 2 mm, but covering less than one third of the tooth surface

4 **HEAVY**
Calculus deposits greater than 2 mm, may extend over soft tissues, or may bridge teeth

Plaque prevention

1. Mechanical – brushing, flossing

- Teeth brush twice daily using a fluoride-based toothpaste.
- Teeth floss daily, or use of an interdental cleaner.

2. Chemical – Mouth wash

3. Food intake –

- Coarse, dry (Avoid 3s sweet, sticky, soft)
- Balanced diet.
- Avoidance of tobacco products.
- Limit the number of snacks throughout the day.

4. Gingival massage

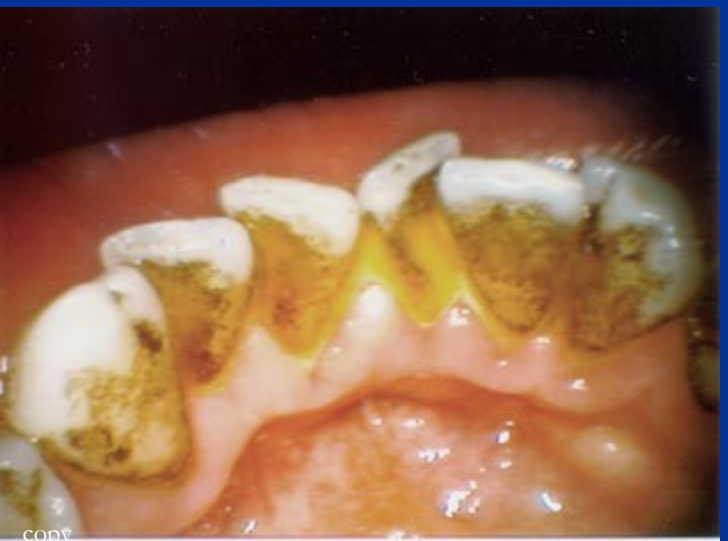
Calculus

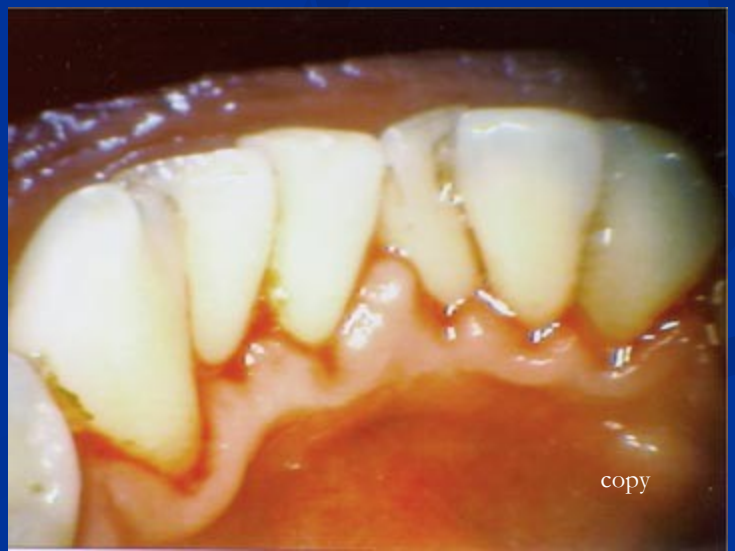
Treatment

- Scaling:

  Manual

  Ultrasonic scaling





Host factors - teeth

- Genetics (twin studies)
  - Occlusal morphology
    - Predisposing
      - Complexity (e.g. buccal pits)
    - Simplicity may be protective

http://www.zahntechnik-online.de

- Environment (diet, prevention)
  - Resistance to demineralization
  - Replacement ions in hydroxyapatite
    - Fluoride, strontium - protective
    - Selenium - predisposing

Genetics

- Genetic factors relate to:
  - tooth composition and structure
  - tooth morphology
  - arch form
  - tooth alignment
  - saliva flow rate and composition
  - oral physiology
  - endogenous microflora
  - food preferences
  - personality traits

Saliva

- Salivary flow rate and composition
- Salivary tooth protection mechanisms:
  - mechanical cleansing action,
  - dilution and buffering plaque acids
  - anti-microbial properties
  - source of inorganic and organic components that inhibit tooth demineralization and assist in the remineralization and repair process.

- Reduction or loss of salivary function associated with dramatic increases in caries activity (rampant caries in xerostomia).

Host factors - normal saliva

- Variation in flow rate
  - High flow rate - protective; low (normal) flow rate - predisposing
  - Not considered a major risk factor by itself
- Variation in salivary buffering capacity

High basic components - protective; not considered a major risk factor by itself

- Variation in antimicrobial protein concentrations
  - S-IgA, peroxidase, lysozyme, lactoferrin and others
  - Expectation: High - protective; Low - predisposing
  - Studies results are inconsistent, sometimes contradictory

Host factors - no saliva

- Xerostomia due to radiation therapy or Sjogren's syndrome
  - Very high *S. mutans* levels + rampant caries
  - Decay in unusual sites in multiple teeth

Acquired pellicle

- The acquired pellicle - acellular, essentially bacteria-free organic film of mucopolysaccharides deposited on teeth
- Critical position between the enamel surface and the dental plaque biofilm
- The pellicle formed mainly by selective adsorption of salivary glycoproteins and proteins with a high affinity for the enamel surface, rapid adsorption to a clean enamel surface.
- The pellicle adheres to the enamel and acts as a diffusion barrier to protect the enamel from acid exposures of short duration, as in ingestion of acidic foods.

Acquired pellicle

- If removed (by dental polishing) the pellicle requires a maturation period (7 days) before it becomes maximally protective against acids.

- The use of abrasive toothpastes and whitening products removes the pellicle, and can have an adverse effect on exposed tooth surfaces in increasing the probability of loss of tooth enamel by demineralization.

Diet

- The frequency of eating fermentable carbohydrates has been strongly associated with dental caries.
- Factors associated with diet and dental caries include the relative retentiveness of the food; the presence of protective factors in food, such as calcium, phosphate, and fluoride, and the type of carbohydrate.
- Complex carbohydrates (starches) are less cariogenic than simple carbohydrates (sucrose, glucose, and fructose).

Dynamic nature of caries

- The earliest macroscopic evidence of caries of a smooth enamel surface - a small opaque white region - white spot lesion.

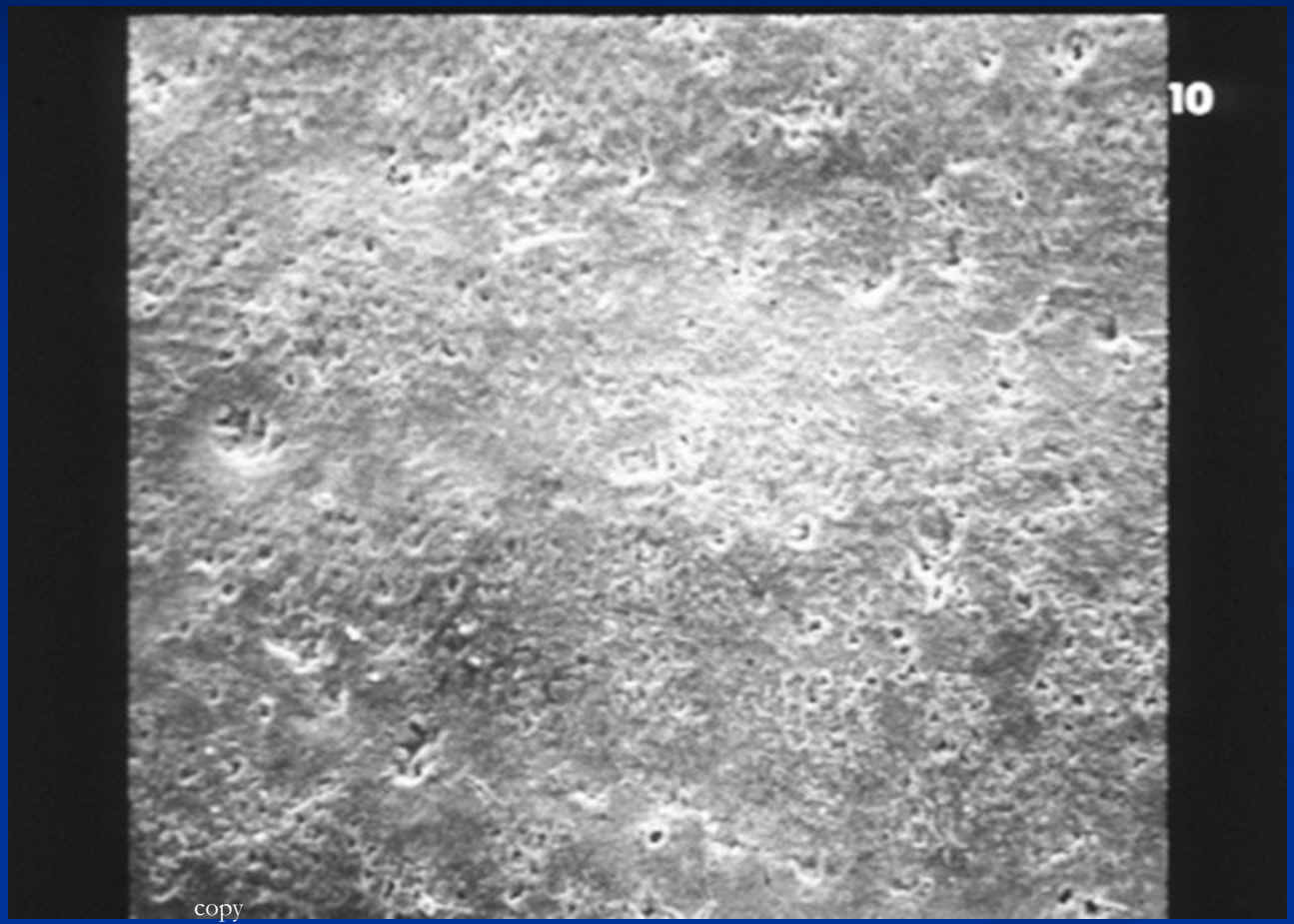
- Its presence - indication for a localized decrease in mineral content of the enamel, although the surface still hard when examined with a dental explorer.

White spot lesion

- The appearance of the white spot lesion in the scanning electron microscope:

small pits representing accentuation of prism outline as the earliest stage of enamel decay.

White spot lesion - EM



Enamel caries

The white spot lesion: conical shape with the base towards the outer enamel surface and the apex towards the amelodentinal junction.

Caries spreads in zones:

- Surface zone
- Body of the lesion
- Dark zone
- Translucent zone

White spot lesion

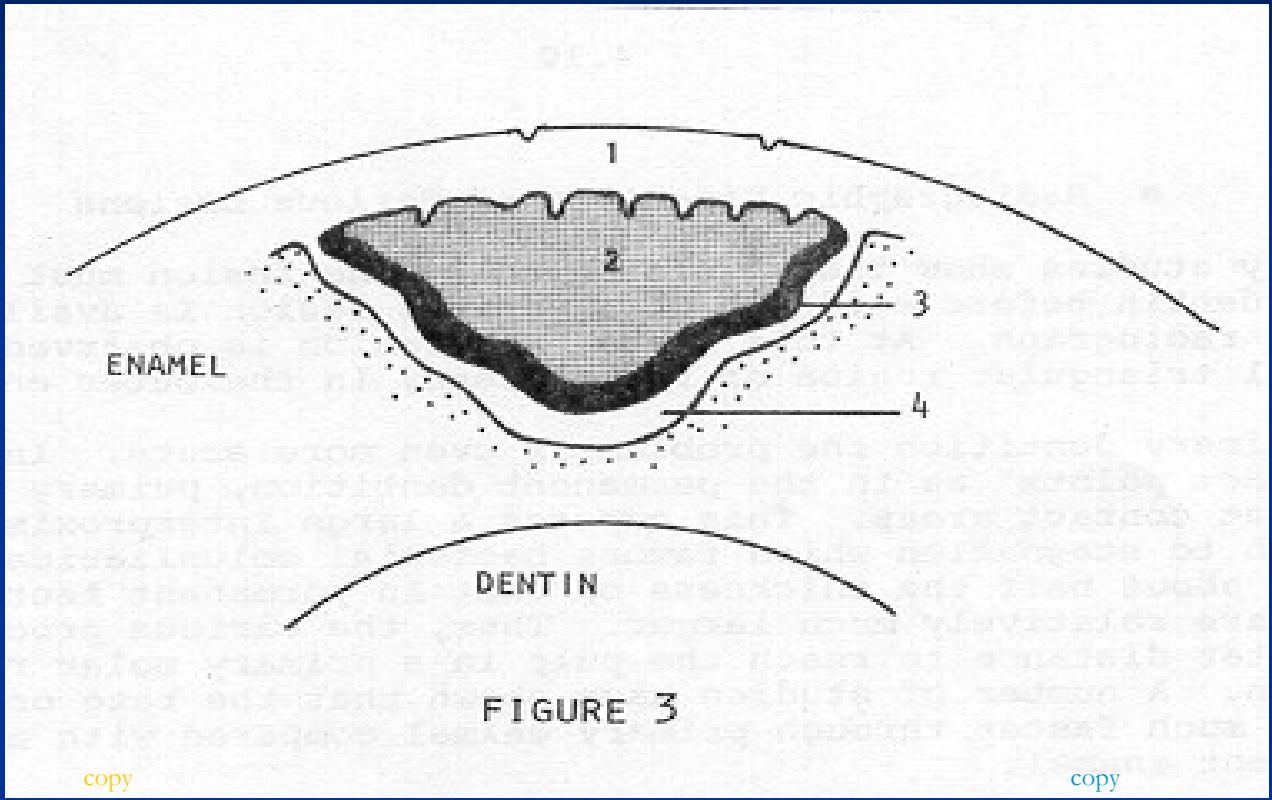


FIGURE 3

1. SURFACE ZONE

Initial lesion of caries, most of the demineralization begins to occur at a subsurface level, leaving the surface zone relatively unaffected.

Surface zone

- ~40 micrometer thick
- relatively normal: maximum remineralization from the inorganic components of both the plaque and saliva.

Subsurface

- Theory: minerals dissolved from this subsurface zone pumped toward the surface → remineralization of the surface zone by precipitation of minerals
- The surface layer of enamel also more mineralized than the subsurface layer, possibly more resistant to acid attack.
- The surface layer relatively unaffected, but more porous in comparison with the unaffected surface.

White spot lesion

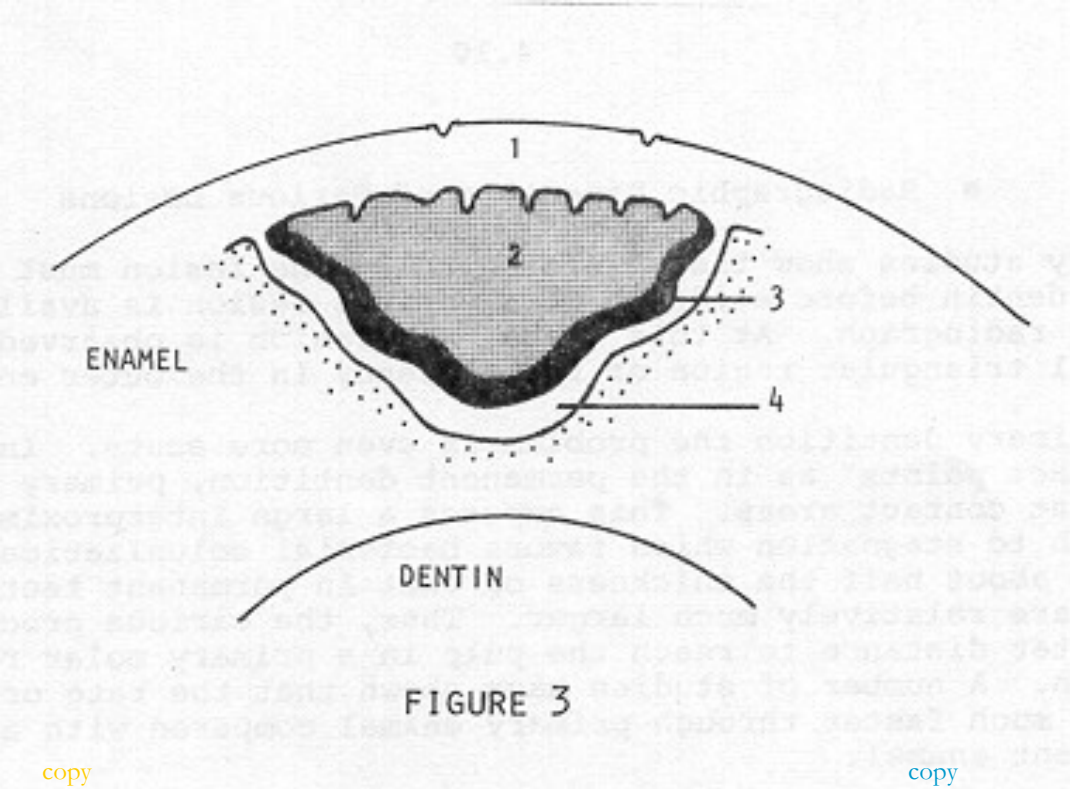


FIGURE 3

2. BODY OF THE LESION

The largest portion of carious enamel in the white spot lesion.

Loss of ~ 1/4 of its original mineral content.

Body of the lesion

- Pore volume of 5-25%
- Apatite crystals larger than the normal enamel.
- Effort for the remineralization, but by the further attack → further dissolution of the mineral
- The zone of maximum demineralization.

White spot lesion

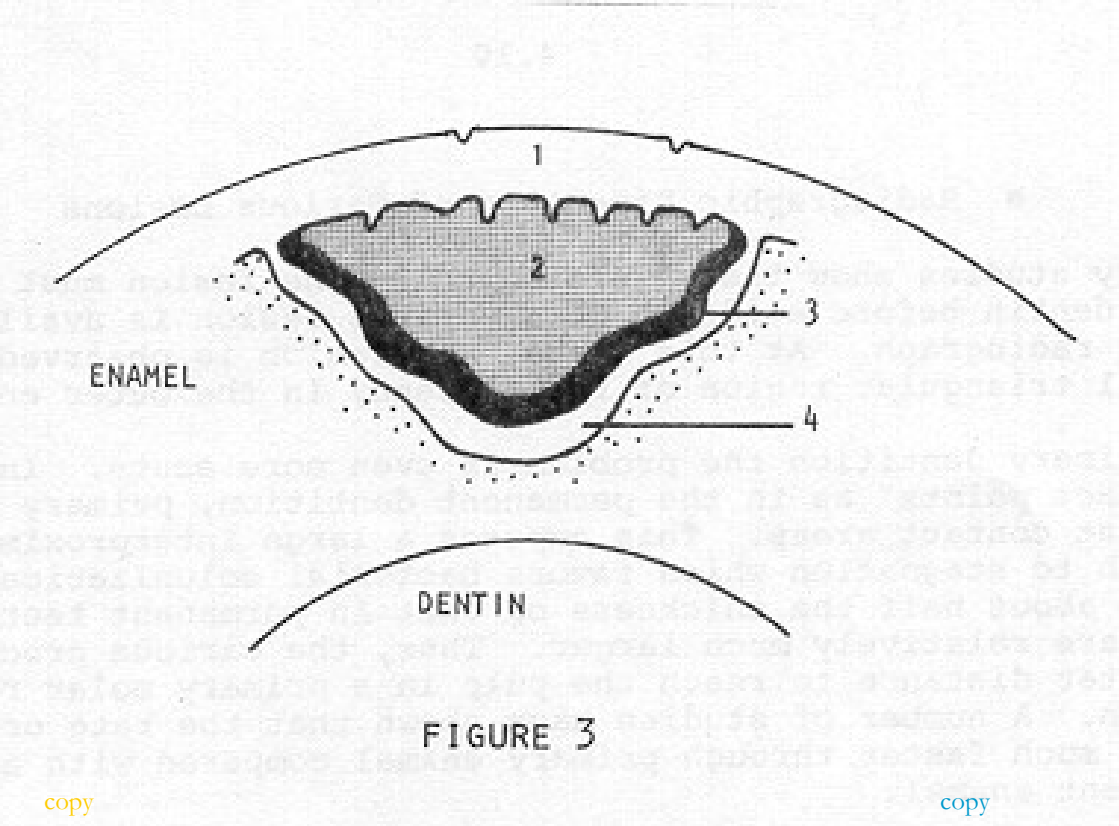


FIGURE 3

3. DARK ZONE

Porous zone +

mineral loss of about 6%.

Dark zone

- Pore volume 2-4%
- Some pores larger, some smaller than in the translucent zone suggesting that some remineralization has occurred.
- In rapidly advancing lesion narrow dark zone.
- Previously liberated salts redeposited here

White spot lesion

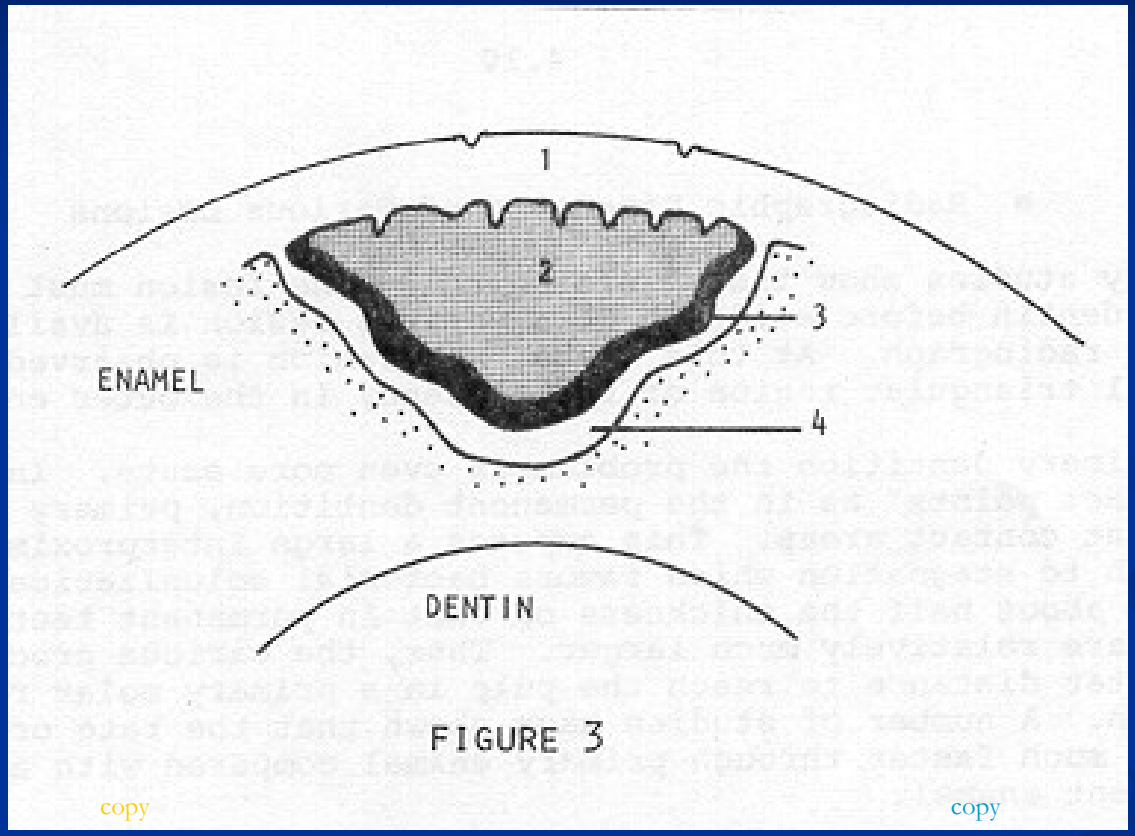


FIGURE 3

4. TRANSLUCENT ZONE

The advancing front of the enamel lesion. More porous than the sound enamel but in sum less porous than the dark zone.

Translucent zone

Zone of intial demineralization

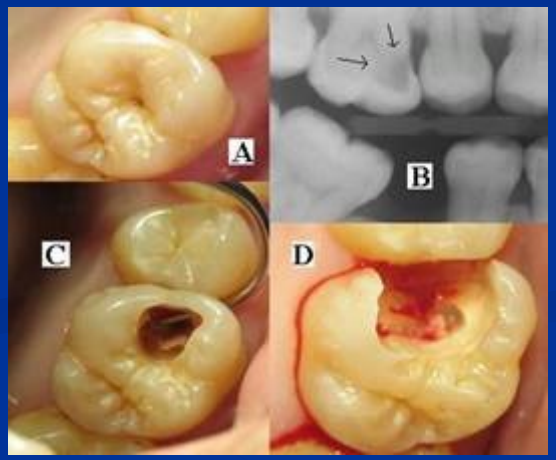
- Pore volume 1%
- Pores larger than in the normal enamel.
- ↓ magnesium and carbonate content in comparison to normal enamel

Zones of enamel caries

copy

The five stages of caries development[1,2]

1. Initial subsurface demineralization — *Reversible lesion*
2. Extension of demineralized zone towards dentine
3. Collapse of surface layer to form cavity — *Irreversible lesion*
4. Extension of caries lesion into dentine
5. Extension of caries into pulp — *Possible formation of apical abscess*



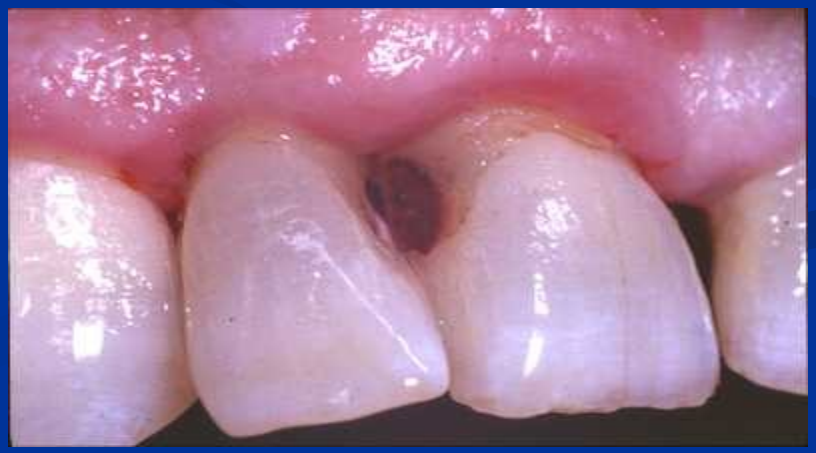
1. Collins WJN, et al. A Handbook for Dental Hygienists. 3rd edition. Oxford: Wright, 1992.
2. Clarkson BH, et al. Caries Res 1991;25:166-173.

Enamel caries

- Begins as discrete lesions in the enamel of specific sites (reservoirs)

Occlusal pits and fissures of molars and premolars

Interproximal contacts between adjacent teeth

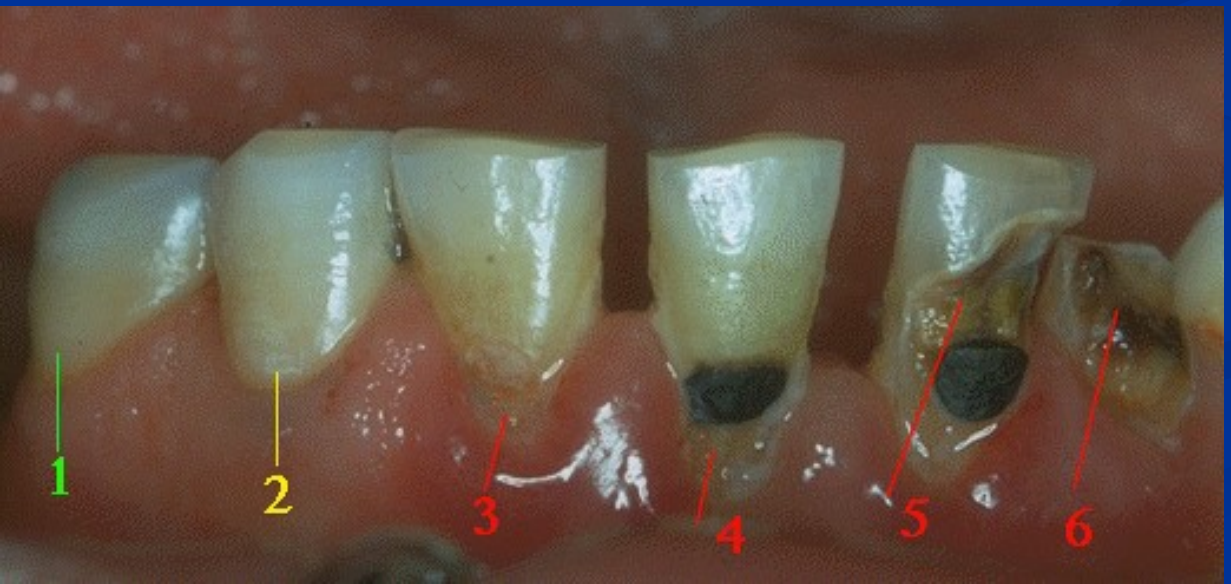


Enamel caries

The initial lesion is visible as a white spot, due to demineralization of the prisms in a sub-surface layer, with the surface enamel remaining more mineralized.

With continued acid attack the surface changes from being smooth to rough, and may become stained.

As the lesion progresses, pitting and eventually cavitation occur.



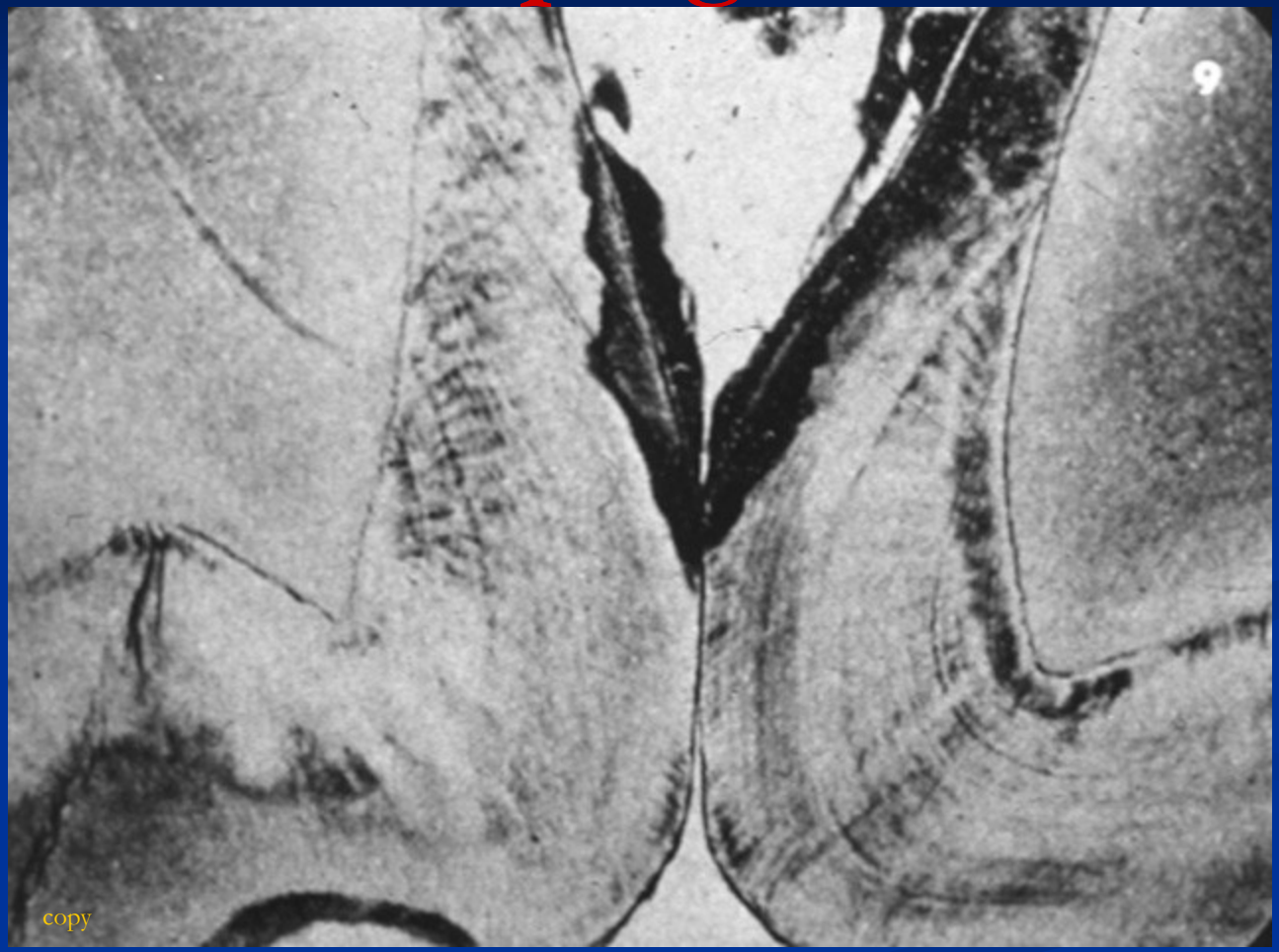
Caries progression

- Even if the enamel surface clinically intact when the lesion reaches the enamel-dentin junction, acids may diffuse into the dentin via carious enamel (together with other clinical stimuli), possible dentin and pulp response.
- Lateral spread along the enamel-dentin junction → a broad-based lesion following the curvature of the dentinal tubules - its narrow apex approaches the pulp.
- In the dentin: a zone of sclerosis walling off the lesion from the surrounding normal dentin.
- The pulp reacts to the advancing lesion by laying down a region of reparative dentin.

Caries progression

- The body of the dentinal lesion may at first be uninfected (bacteria not able to gain access until a cavitation forms in the surface enamel).
- At this stage - if preventive measures instituted - the lesion can remain static or regress.
- After cavitation of the enamel lesion bacteria can penetrate into the tissue - ↑ rate of progression of the dentin lesion
- Proteolytic enzymes of the bacteria destroy the organic collagenous matrix of the enamel and dentin → characteristic dental cavity.

Caries progression

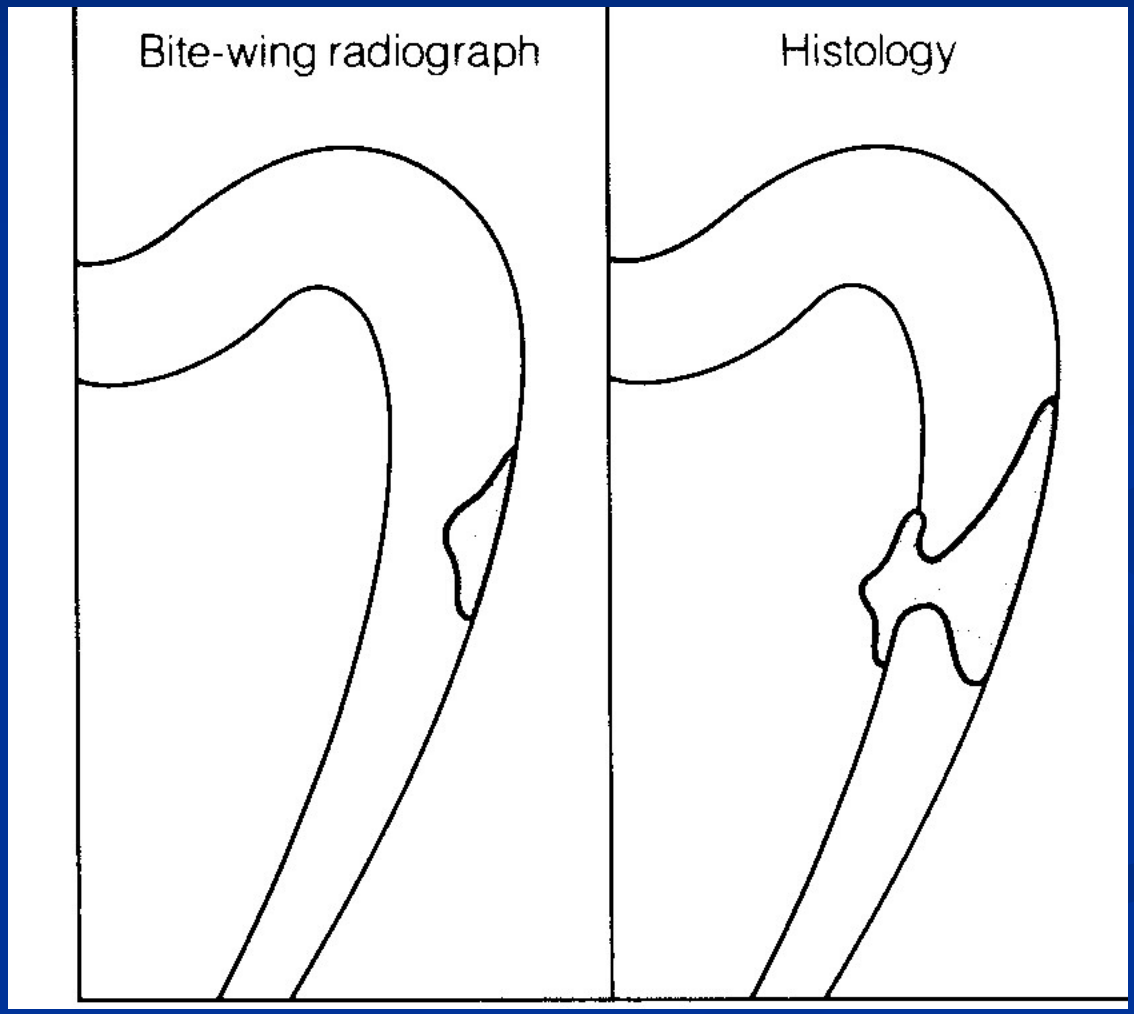


Implications for radiographic diagnosis

- Laboratory studies demonstrate that histologically the lesion must penetrate just into the dentin before evidence of a carious lesion is observed on a routine bite-wing radiograph

- At this stage the lesion is observed on the radiograph as a small triangular region of radiolucency in the outer enamel.

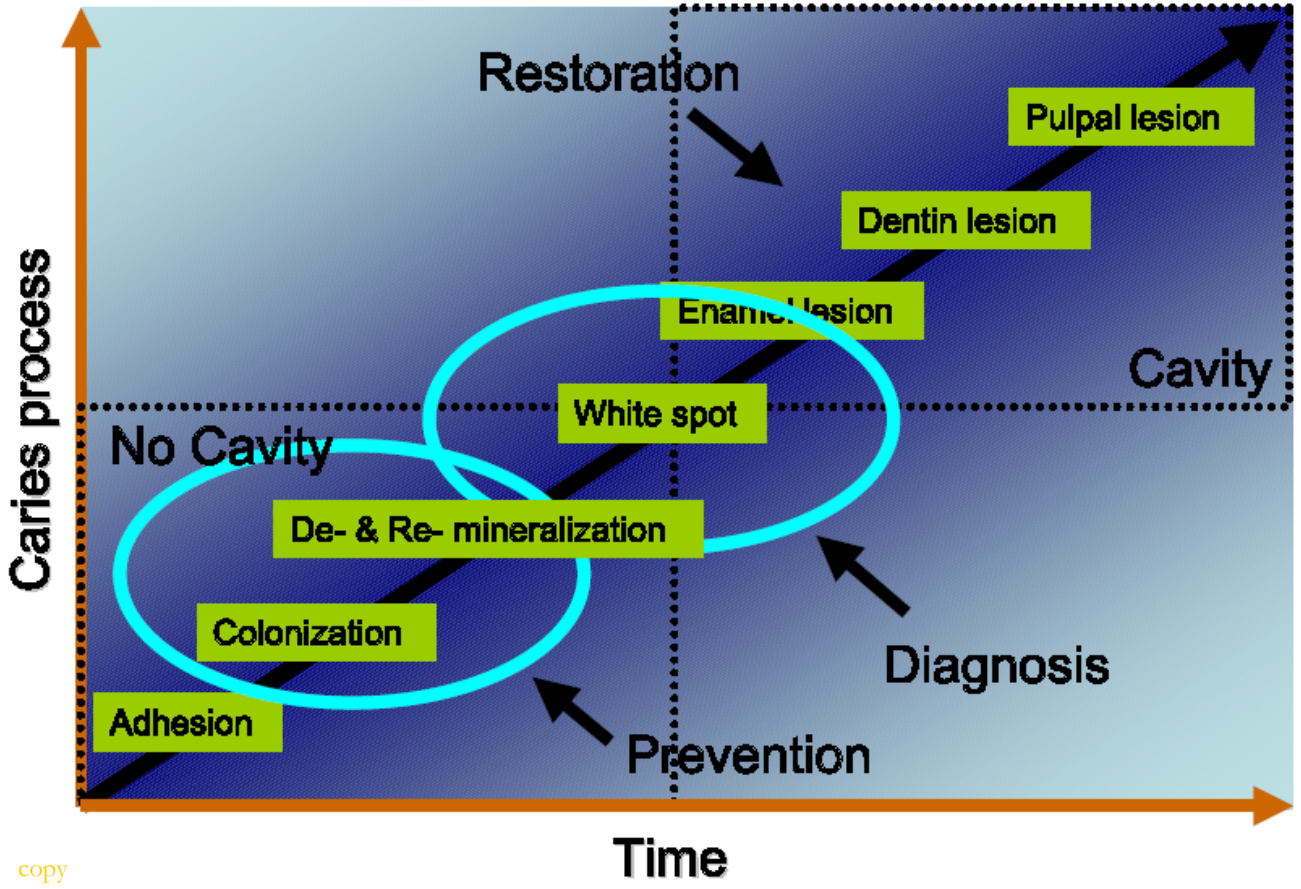
Radiograph versus histology

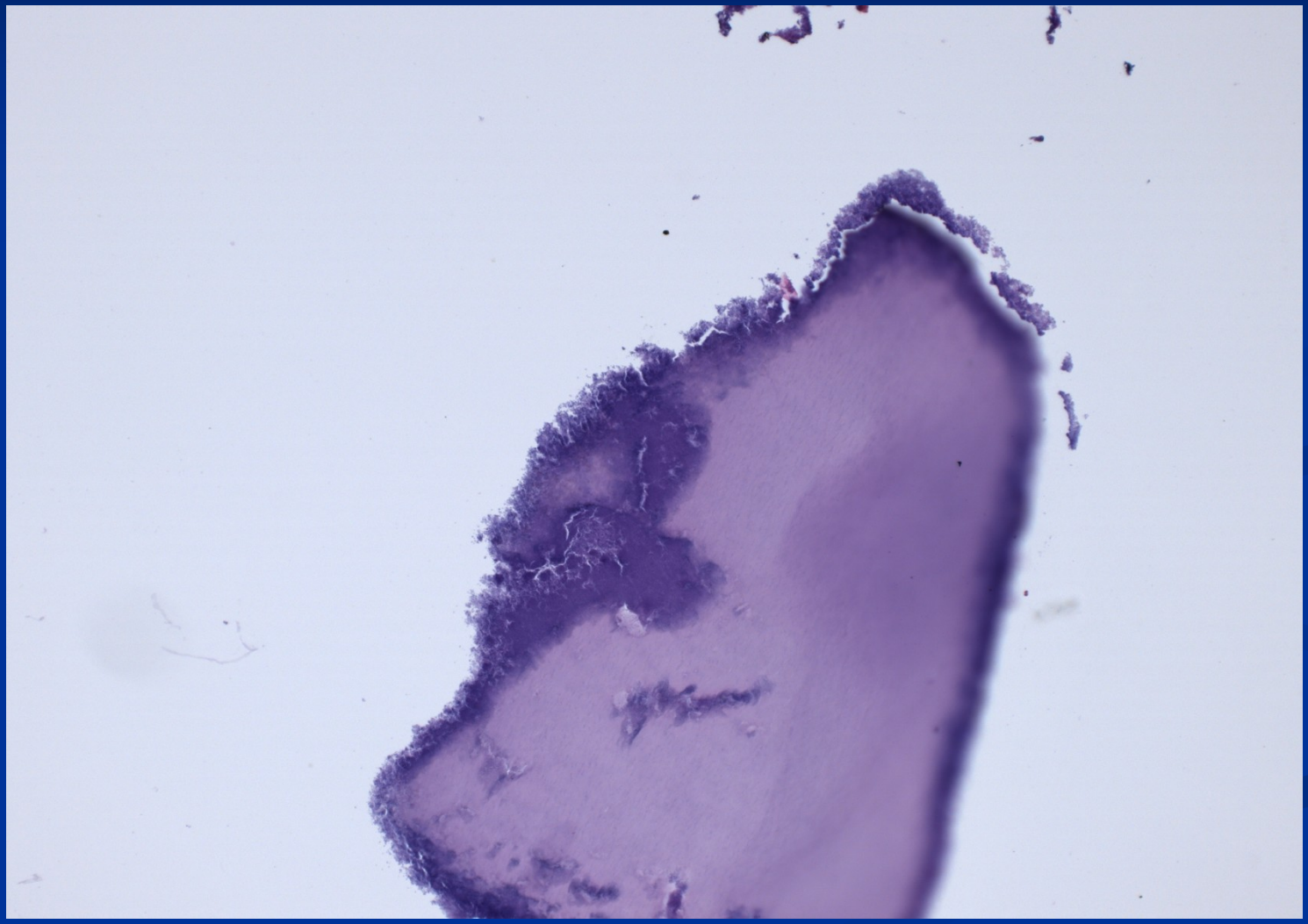


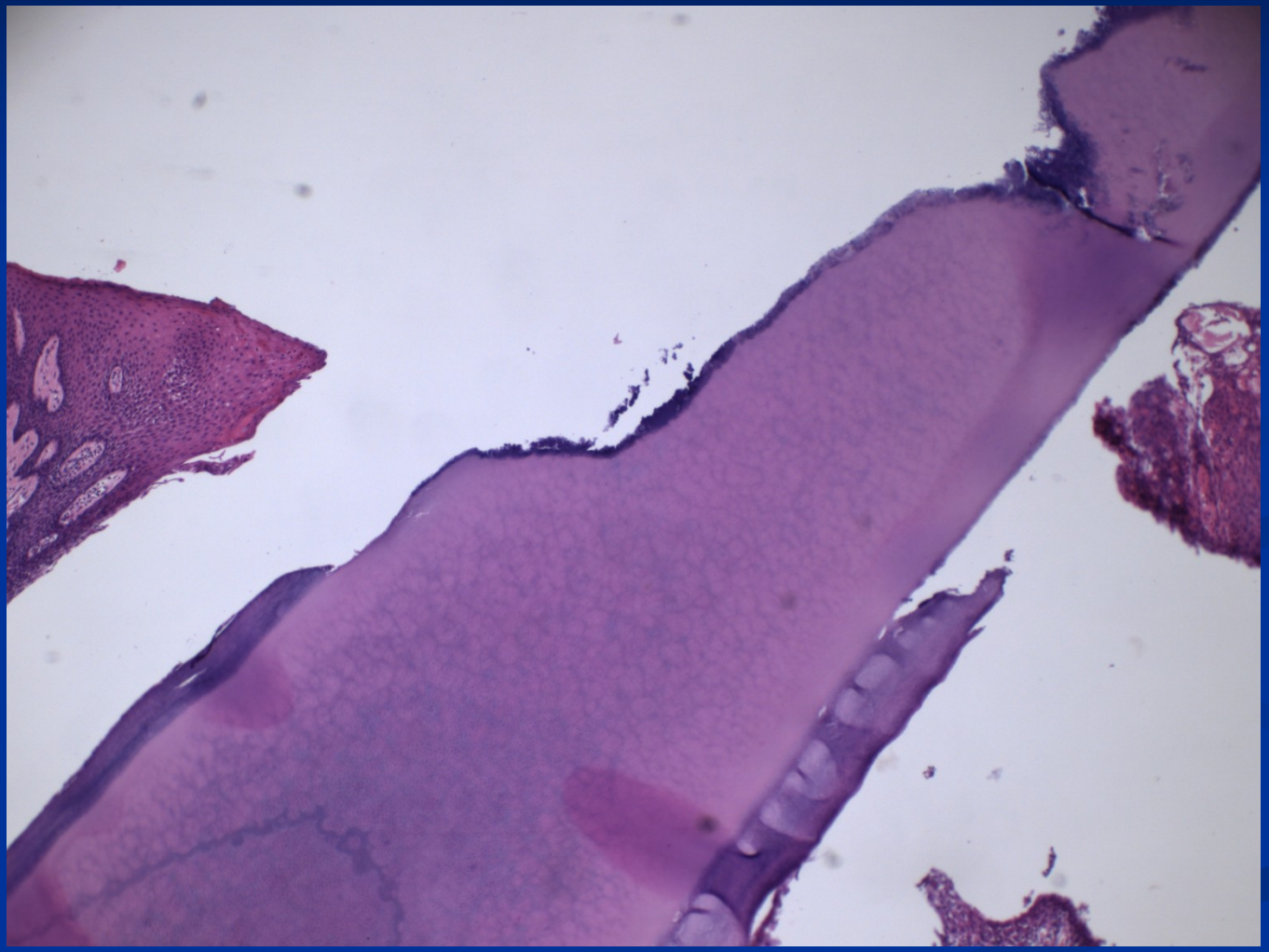
Clinical implications for caries diagnosis

- A carious lesion which could not be detected by explorer or X-ray has already penetrated halfway through the enamel.
- A lesion which can be observed on a bite-wing radiograph has probably already advanced into the dentin (especially in the primary dentition).

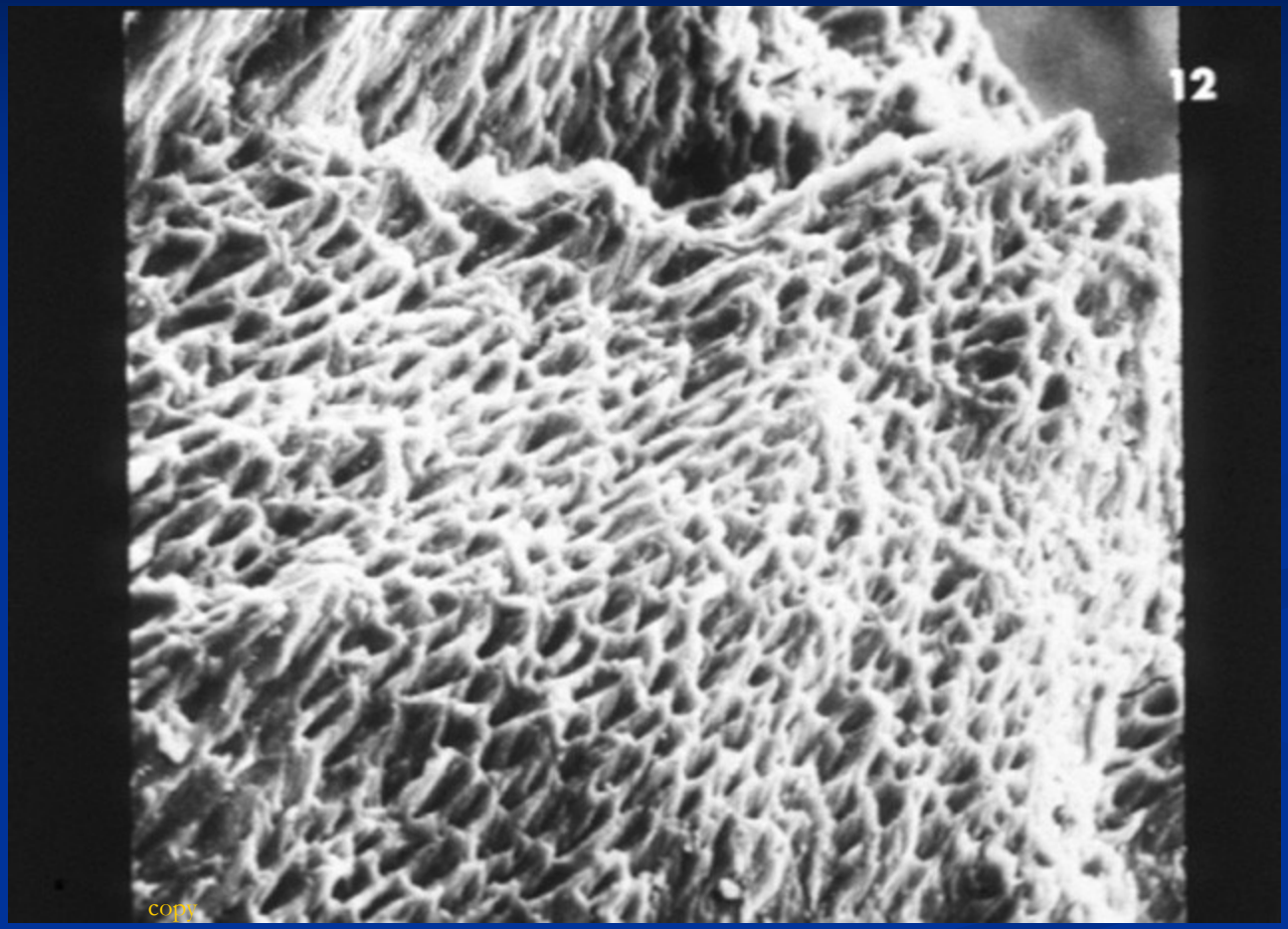
The Caries Evolution







Demineralized enamel



Enamel + dentin caries

- Section of enamel and early dentinal caries. Although the outer surface of the enamel looks intact, the dentinal process (arrow) has started.

Dentinal caries

- Differs from the enamel caries - a living tissue responding in a unique way
- Dentin with a high organic component consisting predominantly of collagen
- A defense mechanism activated in the dentin caries by pulp-predentin complex.
- Caries spread much faster in this zone - more porous + dentinal tubules.
- Bacterial strains able to release large amount of proteolytic enzymes causing damage to dentin.

Dentinal caries

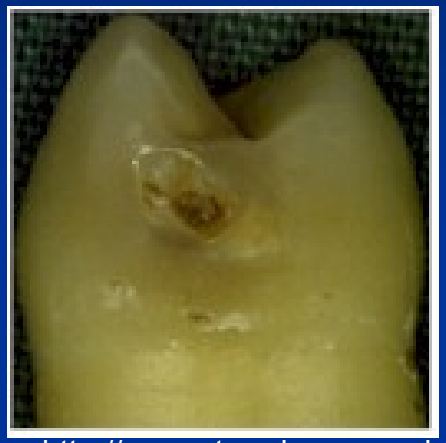
Defense reaction of the pulpodentinal complex

- Sclerosis
- Reactionary dentine formation
- Sealing of the dead tracts

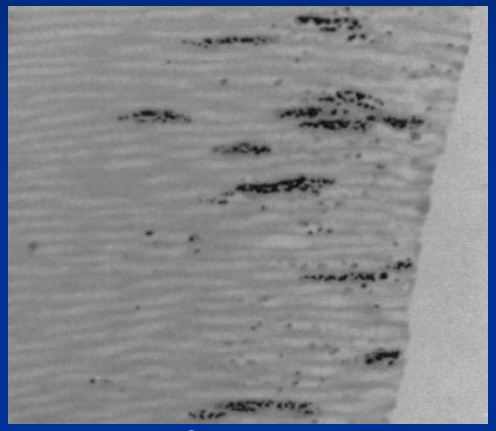
Carious destruction

- Demineralization
- Proteolysis

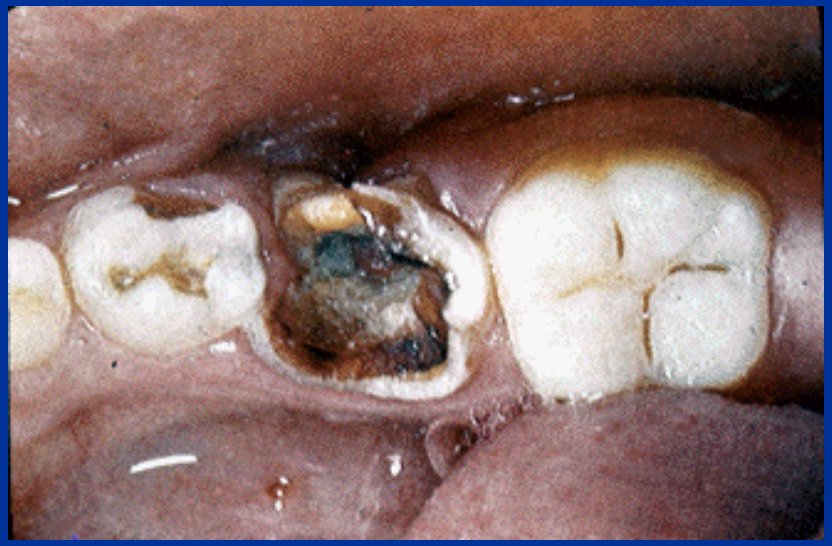
Dentinal caries



http://www.st-andrews.ac.uk/~amc/research/medical.htm



Love et al. Infect. Immun. 68:1359



http://www.dent.umich.edu/research/loeschelabs

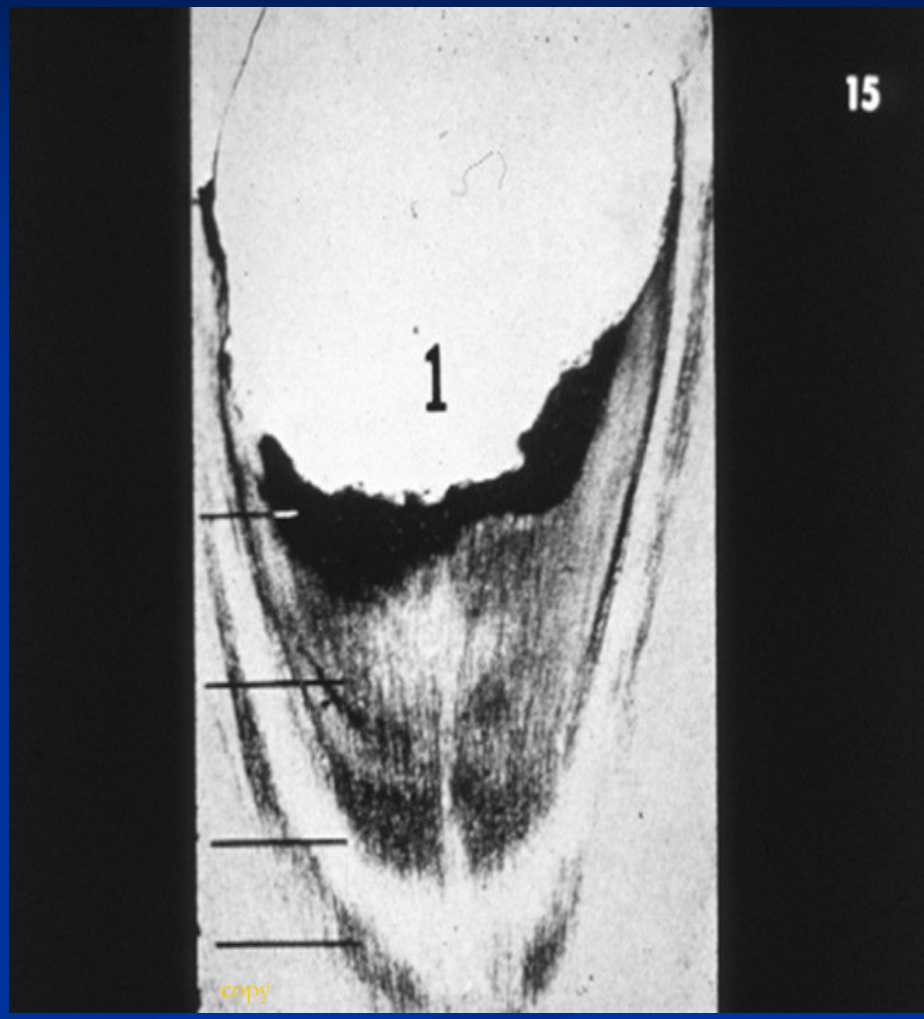
- Cavitation
- Demineralization + proteolysis
- Bacteria move down tubules
- Pulpal involvement
- Major damage if unchecked

Can be arrested, but generally must be restored

Dentinal caries

5 bands: outside (1)

- zone of necrotic dentin
- zone of infected dentinal tubules
- zone of transparent dentin or sclerotic dentin
- zone of fatty degeneration of tubules
- area of intact dentin.



Dentinal caries

zone of necrotic dentin

proteolysis, liquefaction

foci parallel with tubules

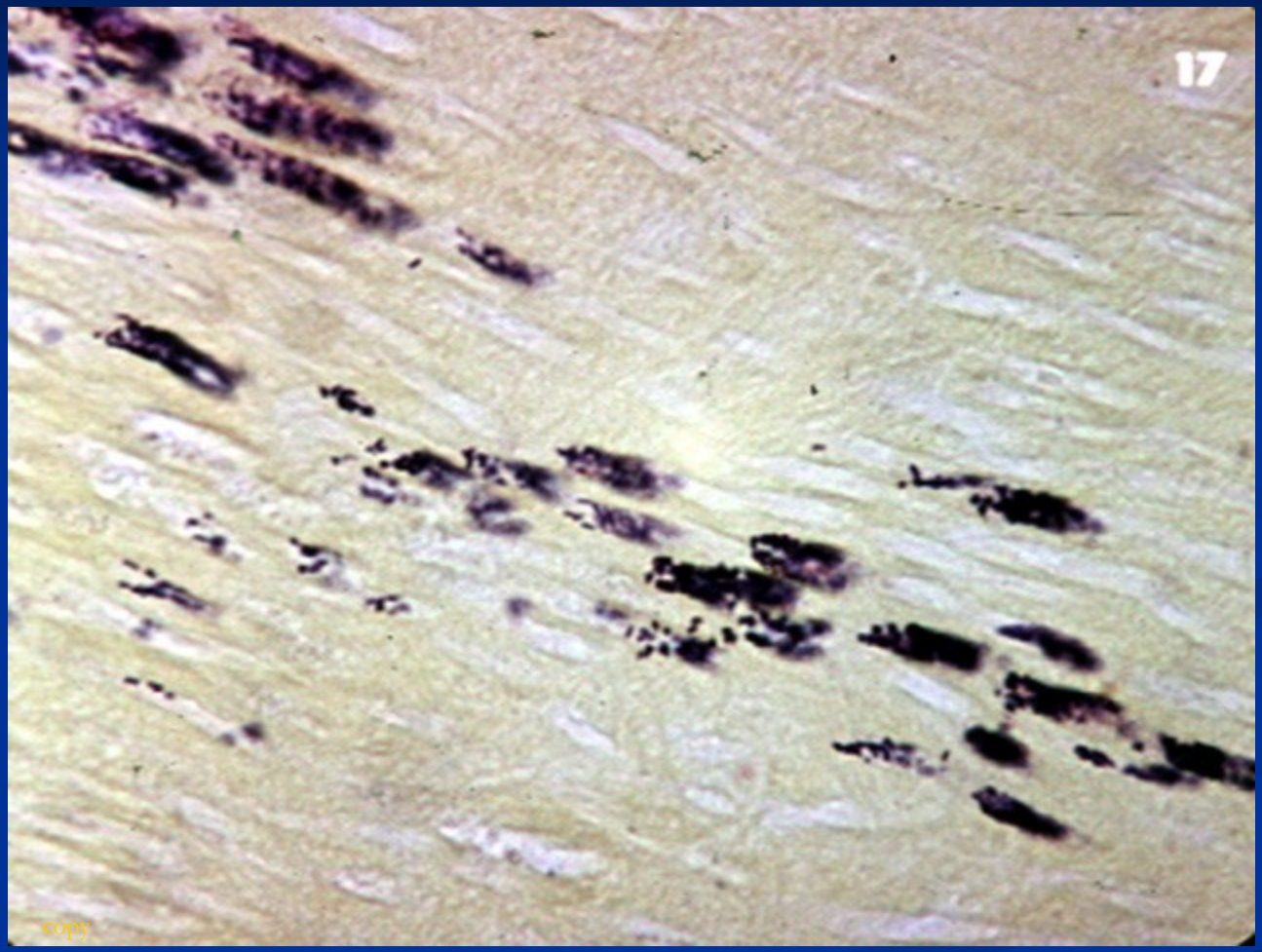
Dentinal caries

Zone of bacterial invasion

- acidogenic microorganisms (lactobacilli)
- acidogenic and proteolytic microorganisms

Infected tubules

Bacteria in dentinal tubules

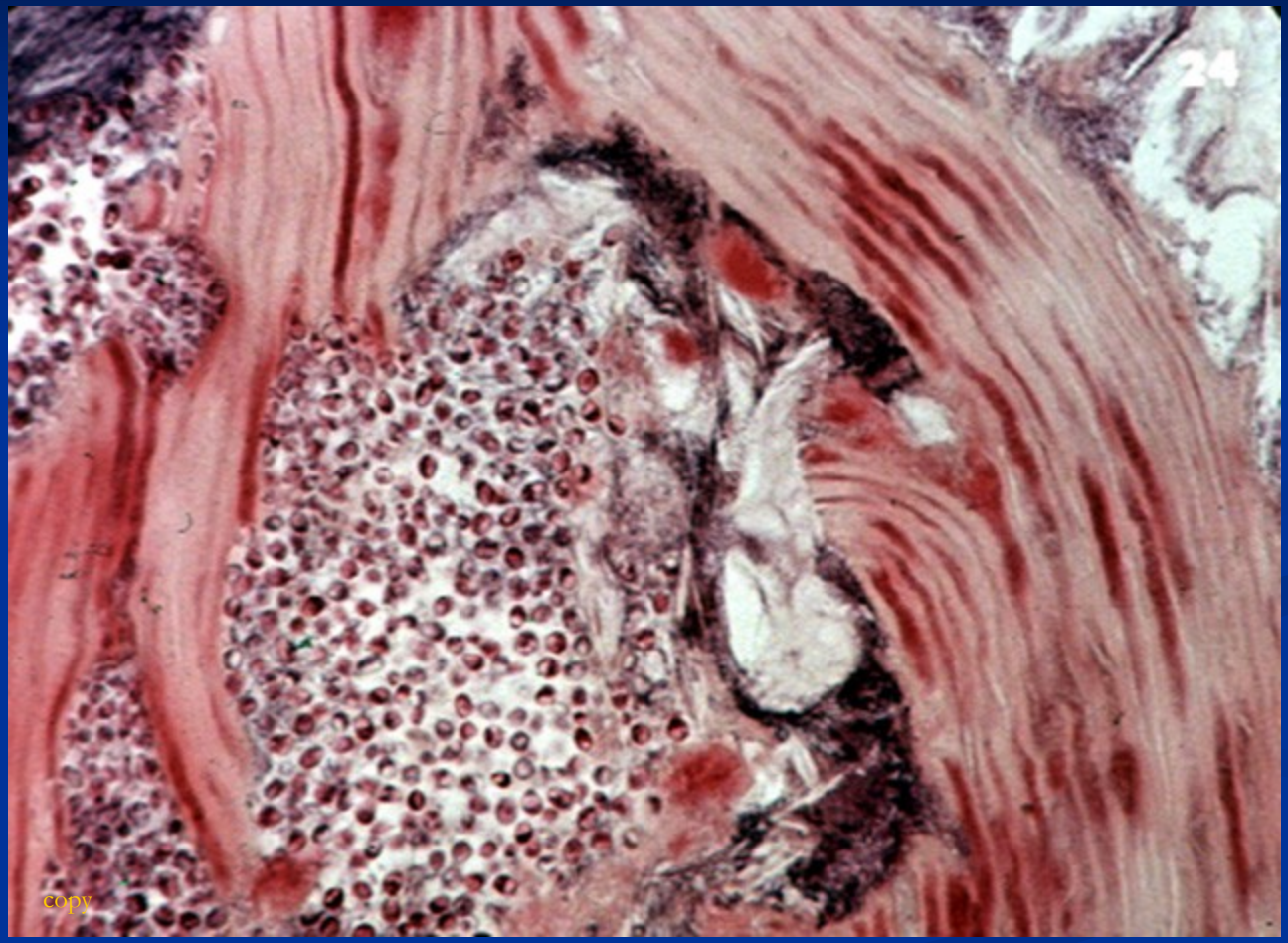


Dentinal caries

- **Clefting**
- Horizontal clefting occurs at right angles to the dentinal tubules. The process of beading, coalescence, and clefting typifies the mode of progression of dentinal caries.

Dentinal caries

Dentinal caries



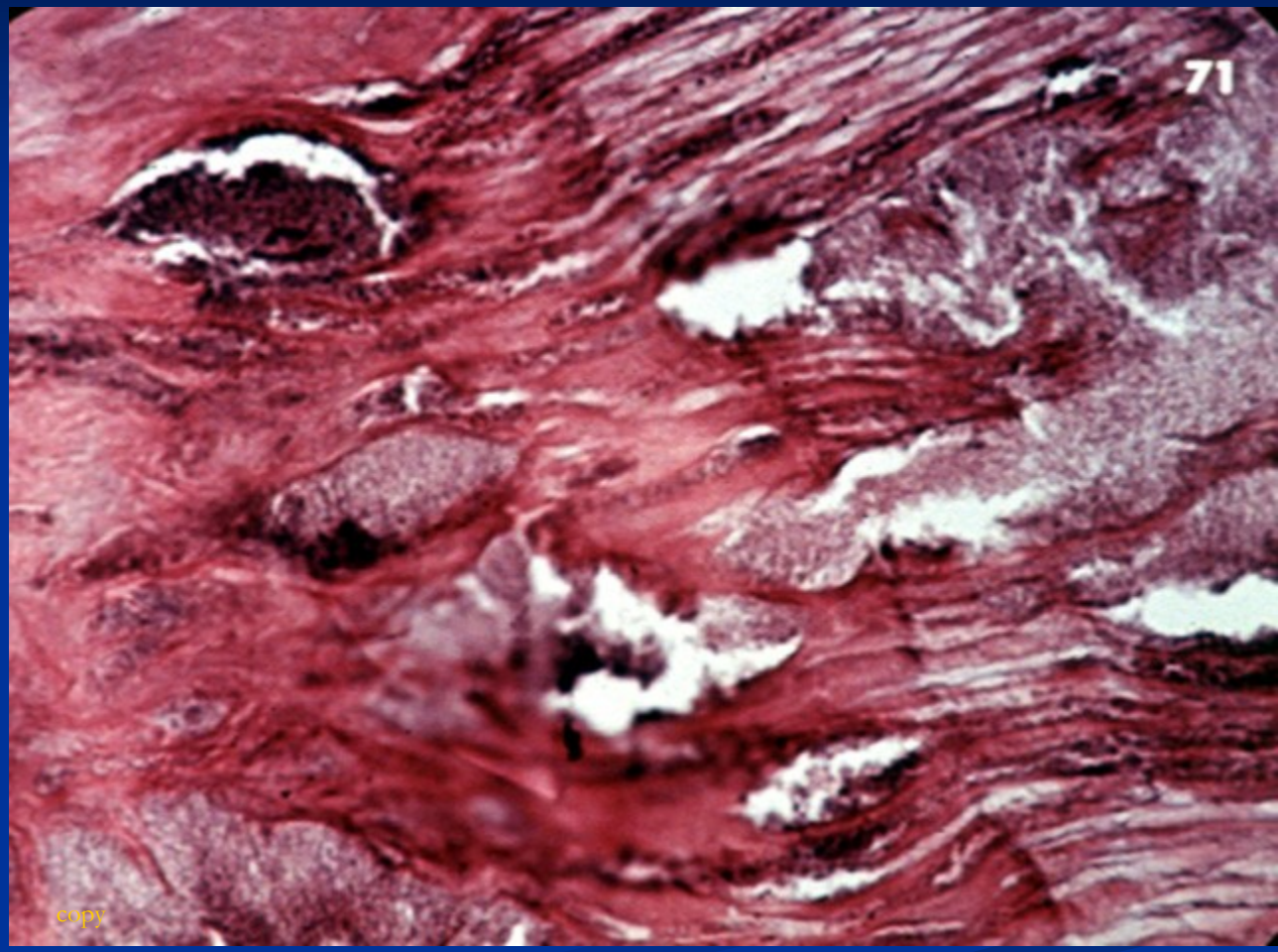
Dentinal caries

Zone of demineralization

- diffusion of acids
- softened dentin
- usually direct progression into zone of bacterial invasion
- may be brownish

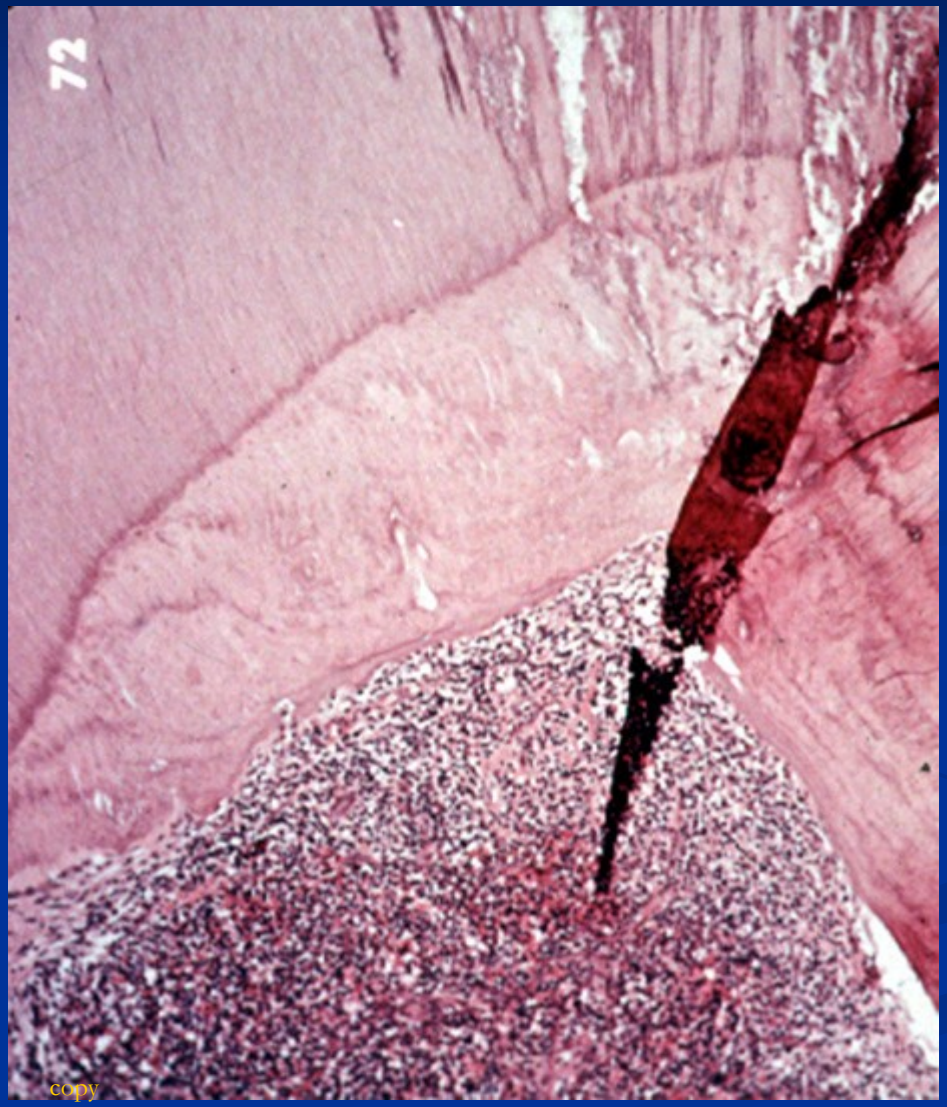
Dentinal caries

Beading, coalescence, and clefting in severely decayed dentin.



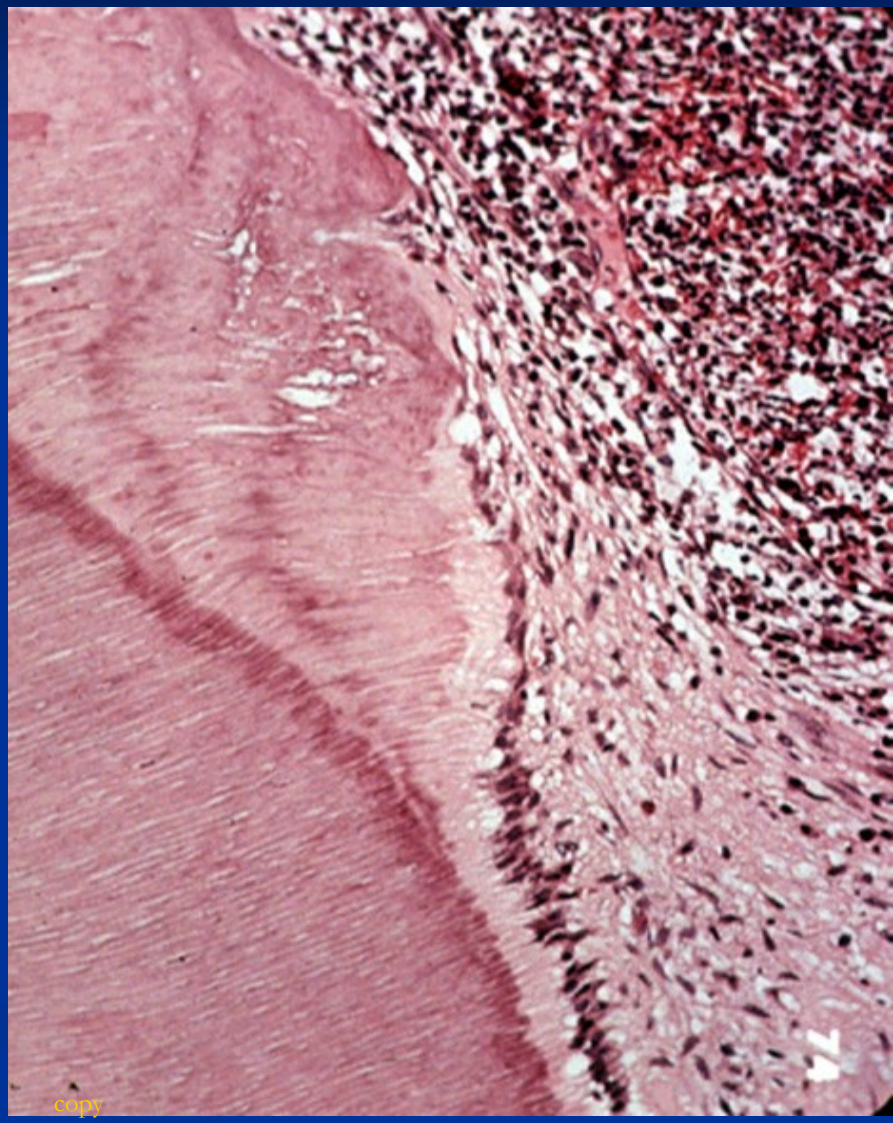
Dentinal caries

Extension of decay process to the pulp with reparative dentin and severe pulpal inflammation.



Dentinal caries

Edge of the intensive inflammation with reparative dentin and maintenance of part of the odontoblastic layer adjacent to the uninflamed pulp.



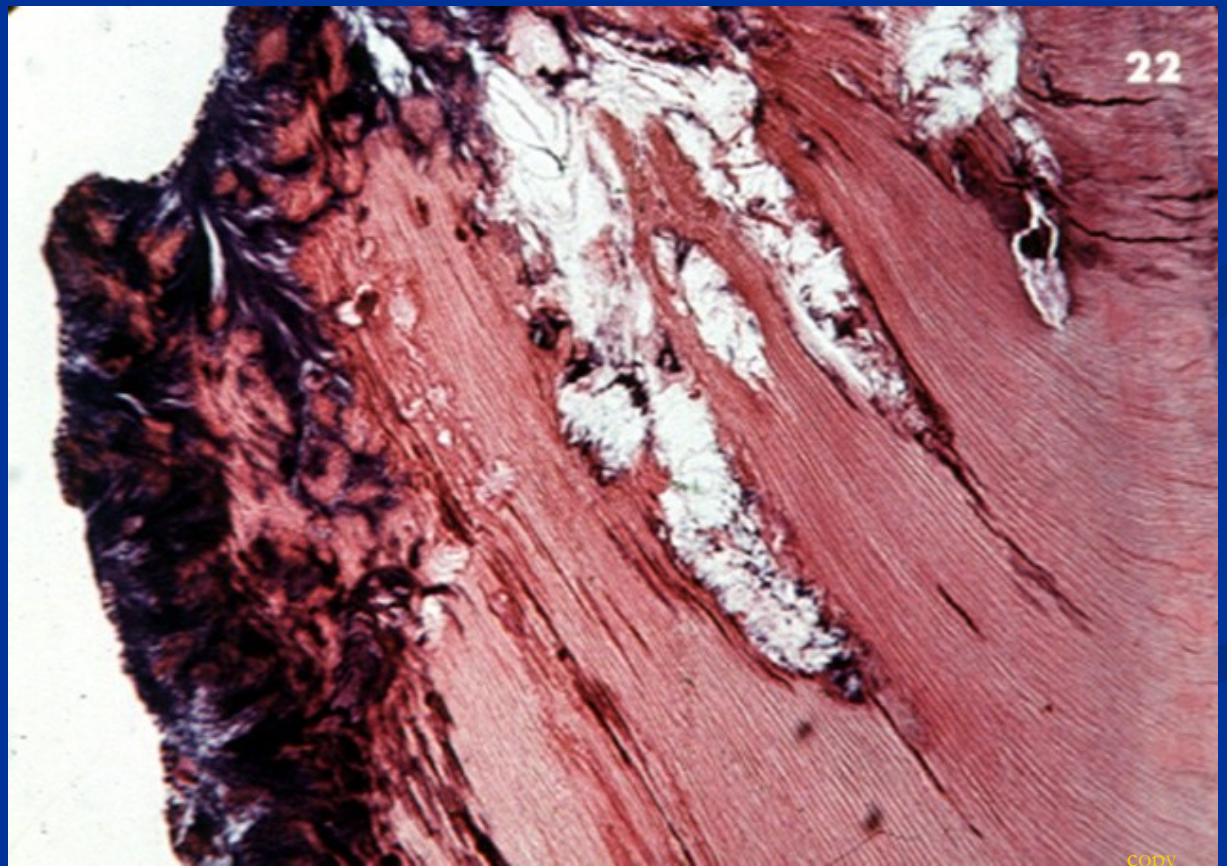
Dentinal caries

Zone of sclerosis

- translucent zone
- odontoblastic reaction with mineralization - ↑ mineral content
- dead tracts – loss of odontoblasts, contain air, possible access of bacteria – sealing with hyaline calcified matter, produced by pulpal cells

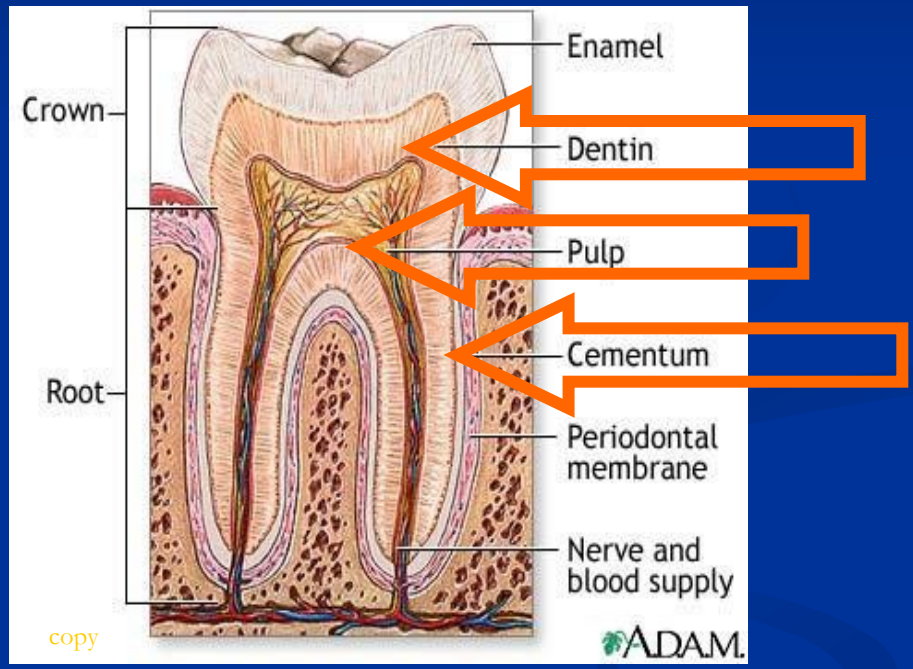
Advanced dentinal caries

- extreme breakdown of dentin with large clefts appearing both vertically and horizontally and with bacteria proceeding down the tubules.



Root surface caries

- Mechanism the same as enamel caries
- Bacterial enzymes break down dentin in the root



Root caries

Initial phase

- gingival recession (abrasion, aging, periodontal disease)

Phase II

- begins apically to the cemento-enamel junction
- few clinical symptoms
- brownish color, softening

Root caries

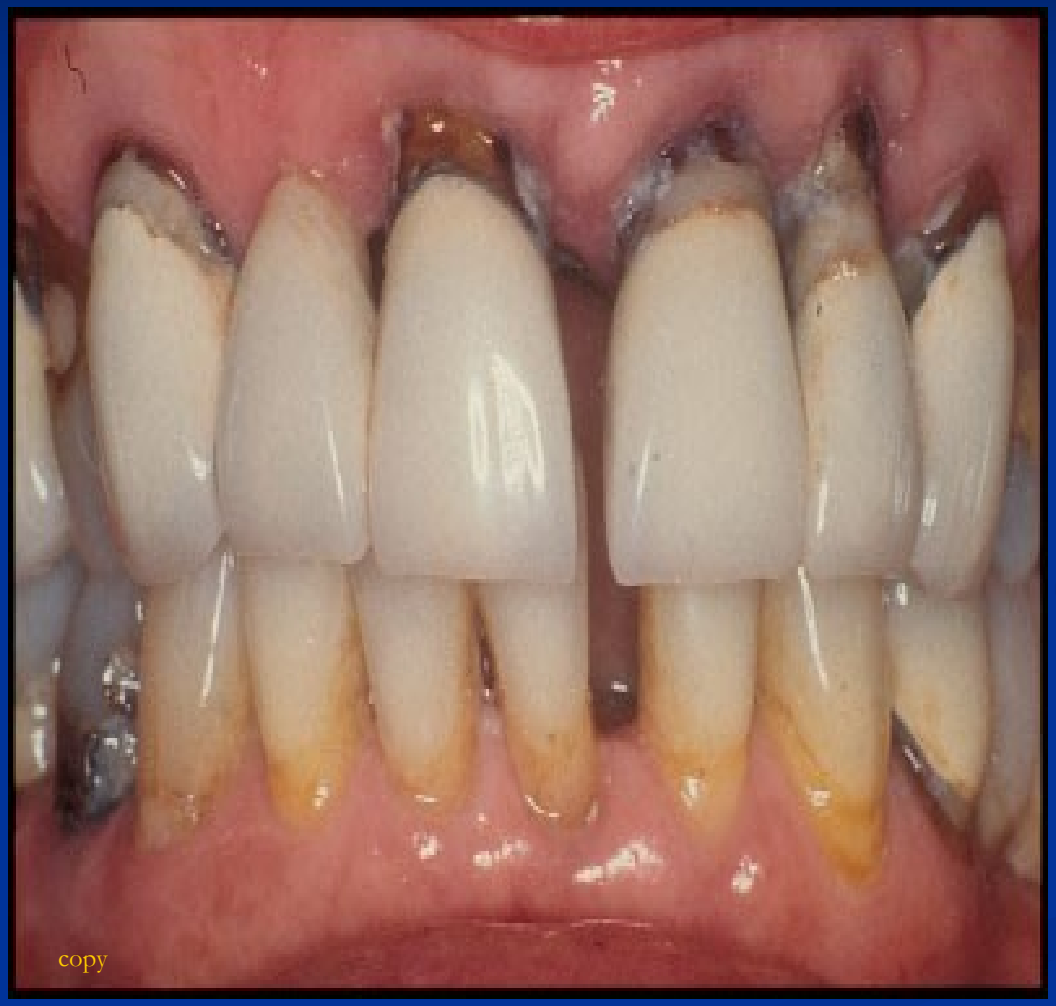
- cementum - primary tissue affected in the root caries
- root exposed to the oral environment as a result of the periodontal disease, followed by the bacterial colonization.
- superficial hypermineralisation
- subsurface demineralization of the cementum and the chain of events similar in the dentine as in the crown portion.

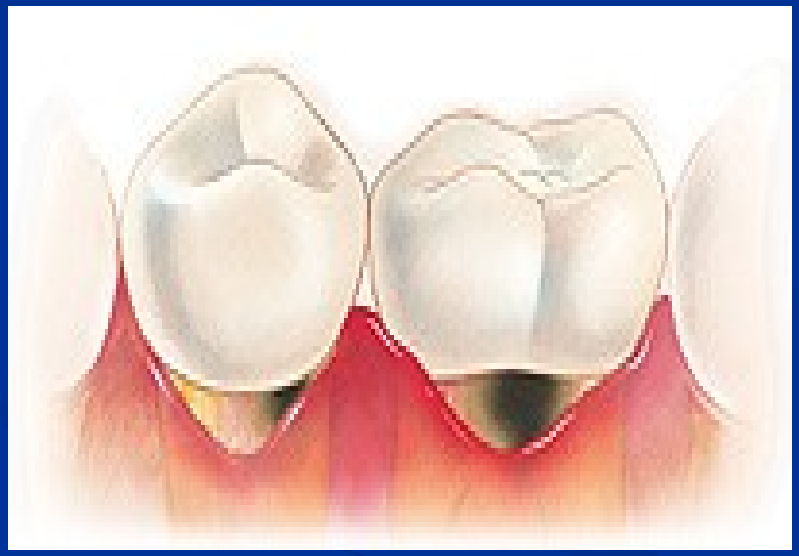
Root caries

Root surface caries:

- 45% minerals (x 88% in coronal c.)
- Plaque + recession related
- Acid dissolves surface – contour changes
- Once collagen destroyed, remineralization not possible (x coronal c. – possible until cavitation)

Root caries



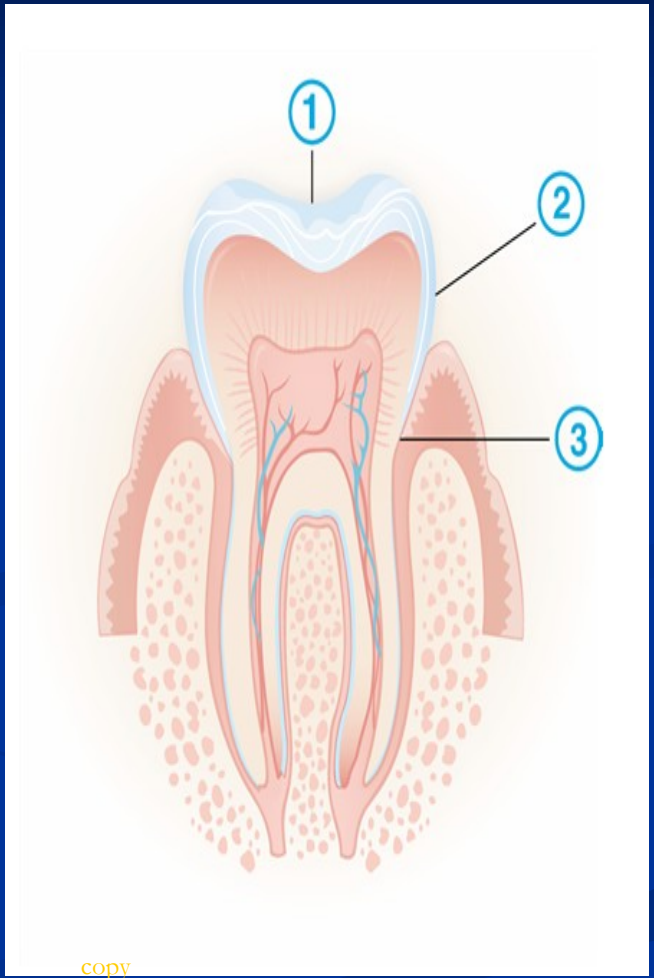


Pathology of caries

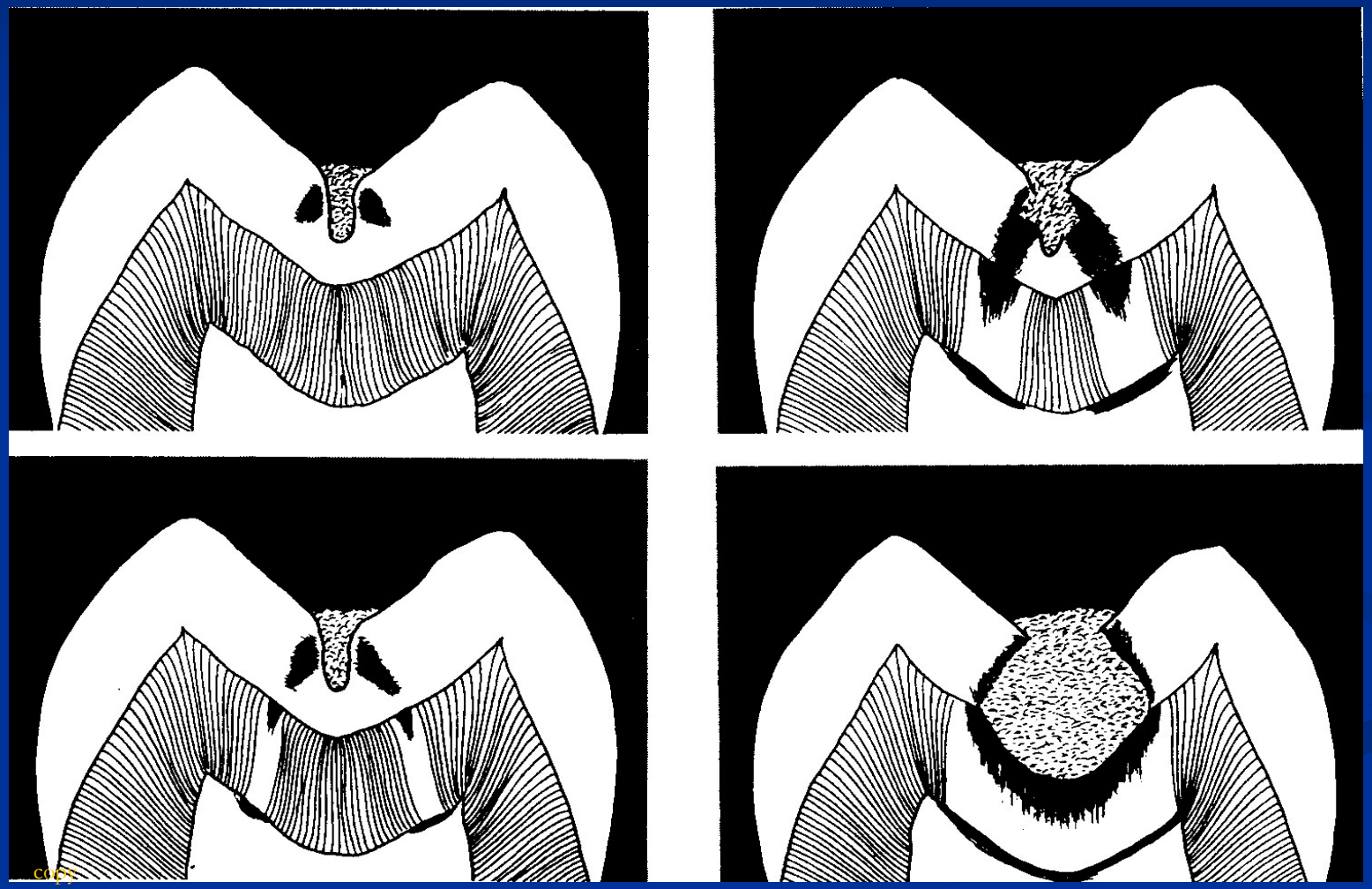
- **Classification by site of attack**
  - pit (fissure) caries
  - smooth surface caries
  - cemental (root) caries
  - recurrent caries
- **Classification by rate of attack**
  - acute (rampant) caries
  - slowly progressive, chronic caries
  - arrested caries

Caries sites

1. Pit-and-fissure caries initially in the fissures of the teeth, but can spread into the dentine
2. Smooth-surface caries most common on interdental surfaces, but can occur on any smooth surface of the tooth
3. Root caries attack the cementum and dentine, exposed as gums recede.



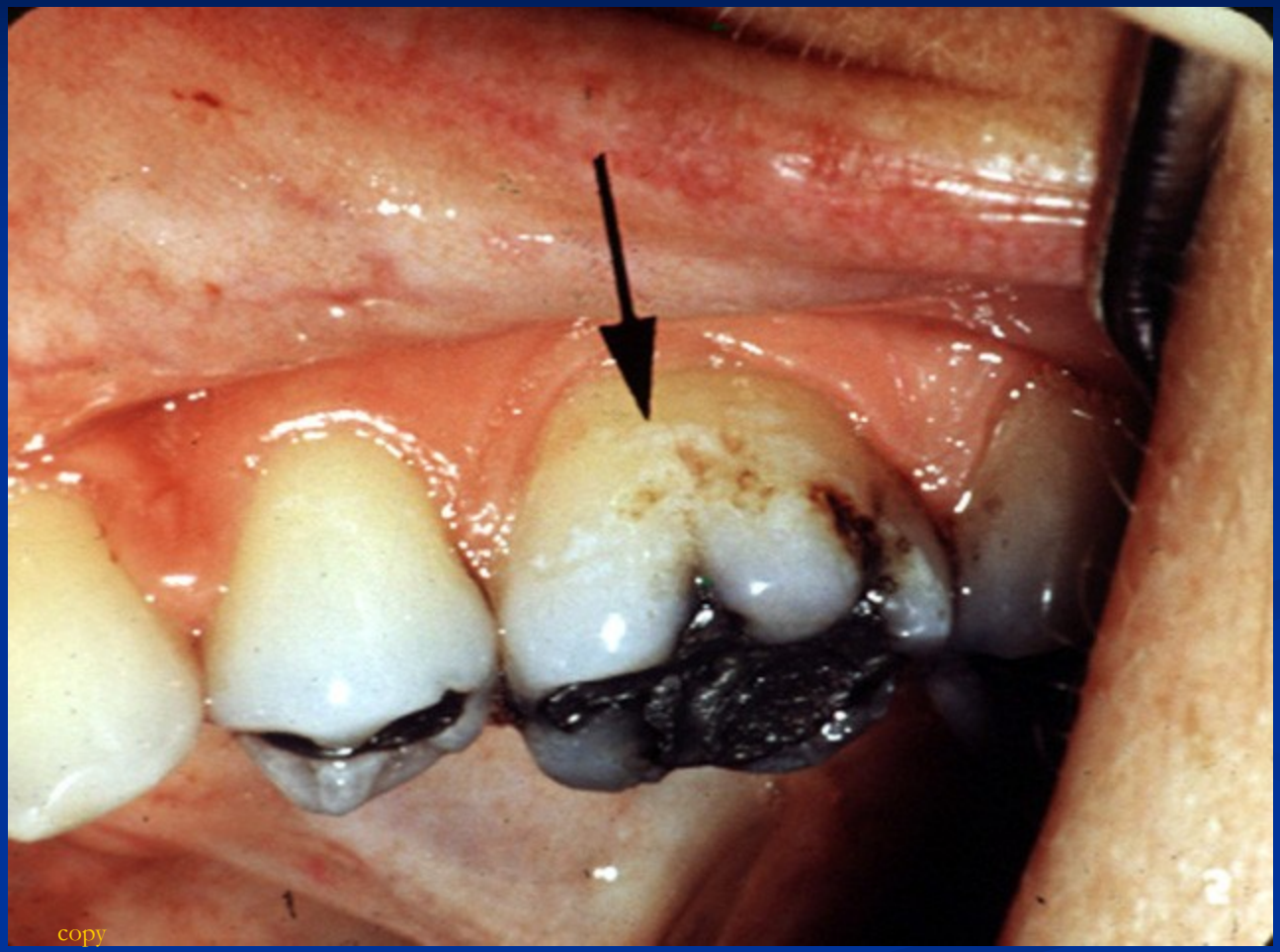
Caries progression in a fissure



Smooth surface enamel caries

- Enamel caries on the smooth surface: a focally brown or white spot lesion
- Radiographically undetectable because of the intact surface.

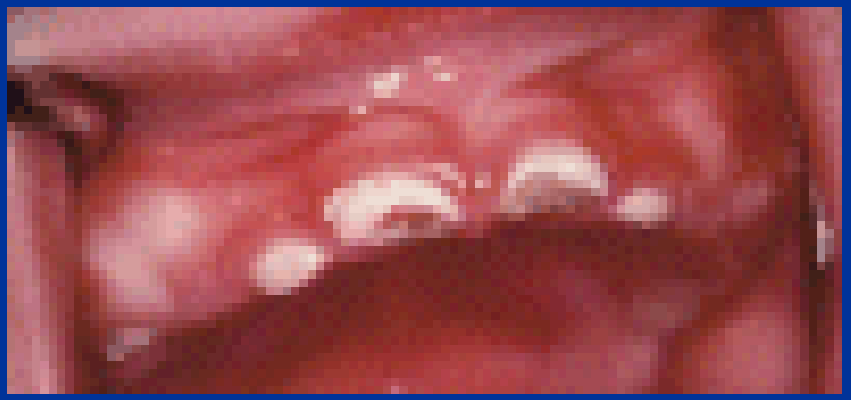
Smooth surface enamel caries



Primary teeth

- Primary molars with broad, flat contact areas in contrast to the contact „points“ in the permanent dentition.
- Exposition of a large interproximal area of primary teeth to stagnation, favoring bacterial colonization.
- Primary enamel thickness ~one-half that of permanent enamel, the pulp chamber is relatively larger.
- The rate of progression of a lesion through primary enamel is much faster compared with an equal distance through permanent enamel.

Early childhood caries



(Douglass et al., *Am Fam Physician*, 2004)

Early childhood caries (ECC)

- Also called Baby Bottle Tooth Decay (BBTD)
- Associated with dietary carbohydrates
- Risk factors
  - Frequent sugar consumption
    - Foods/fluids sweetened with fermentable carbohydrates over extended period
  - Exposure to environmental tobacco smoke

Arrested caries

Enamel

- non-cavitary lesion accessible to plaque control
- remineralization

Dentin

- early dentinal sclerosis (not in acute caries)
- lateral spread of caries possible, undermining, removal of softened structures
- leaves brown-black hypermineralized dentine