

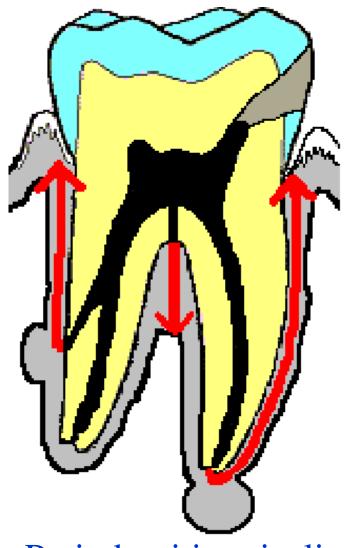


# PERIODONTOLOGY

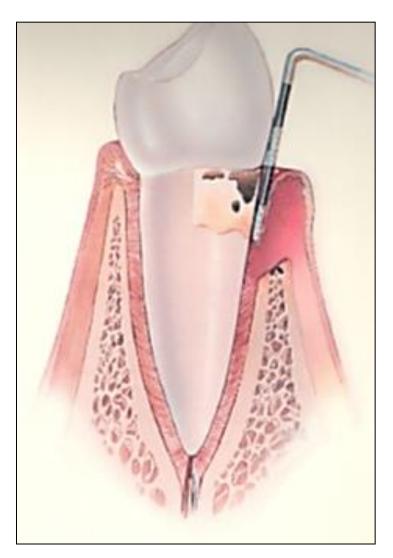
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Do not confuse it, these are two completely different diseases !!!



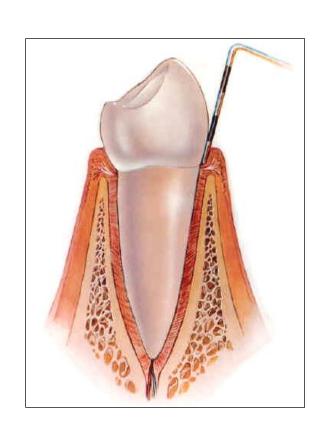
Periodontitis apicalis
- result of caries



Periodontitis marginalis
- see below

# Periodontium - the tooth supporting structures

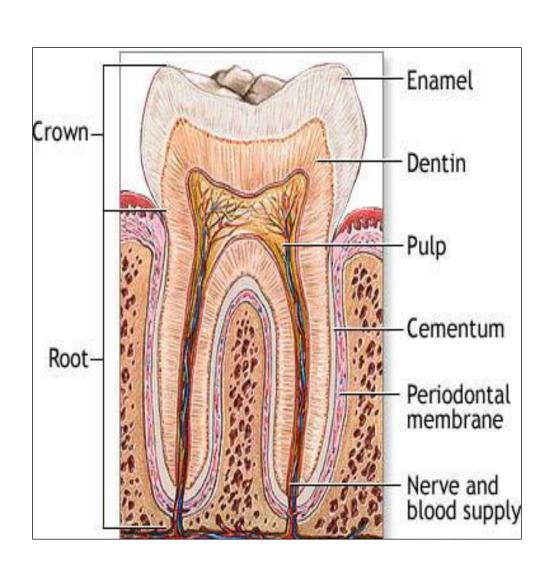
- complex highly specialized shock-absorbing pressure senzing system
- consist of four interrelated tissues supporting the teeth
- covering tissues
- supporting tissues

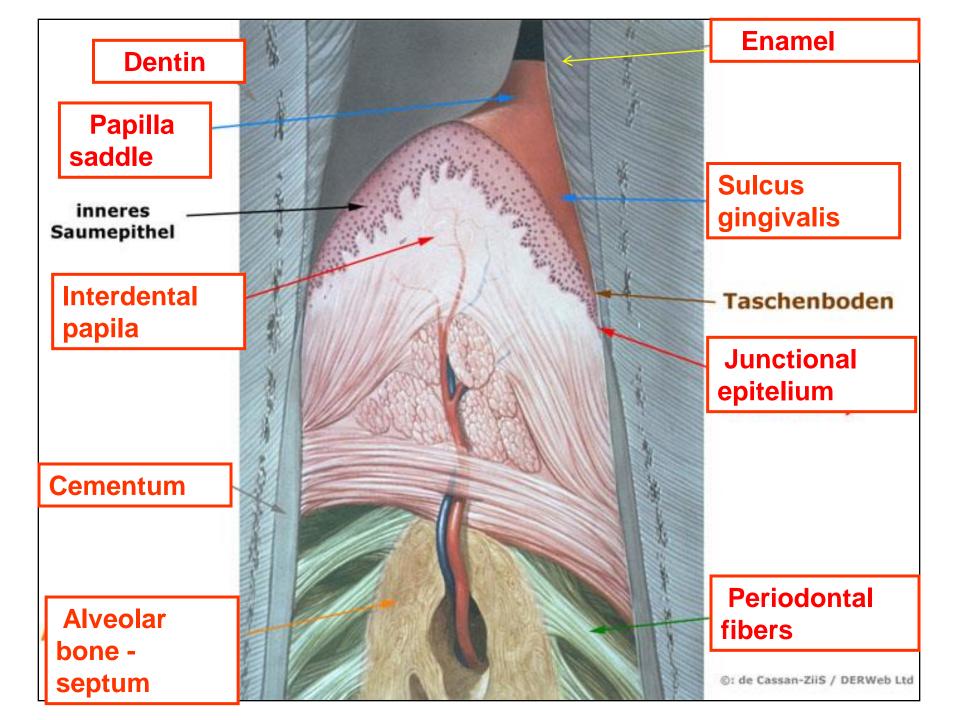


## Periodontal tissue

- Gingiva
- Periodontal membrane with periodontal ligaments (fibers)
- Root cementum

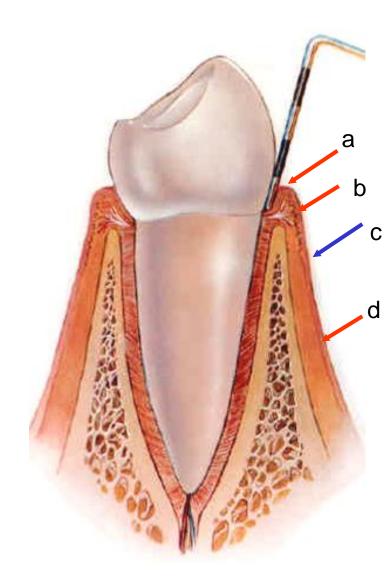
 Alveolar bone (tooth socket)

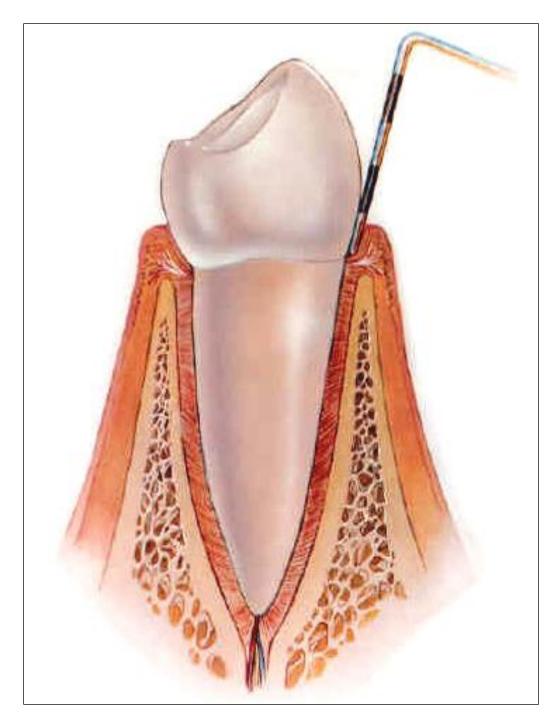




#### GINGIVA

- is one portion of the oral mucosa
- covers limbal part of the alveol
- Free gingiva (a b)
   forms a rim around a tooth neck
- Sulcus gingivalis gingival fluid
- Dentogingival junction
- Interdental papila fills the interdental space
- Attached gingiva (b c) firmly attached to the subgingival structures
- Mucogingival line c
- Alveolar mucous d





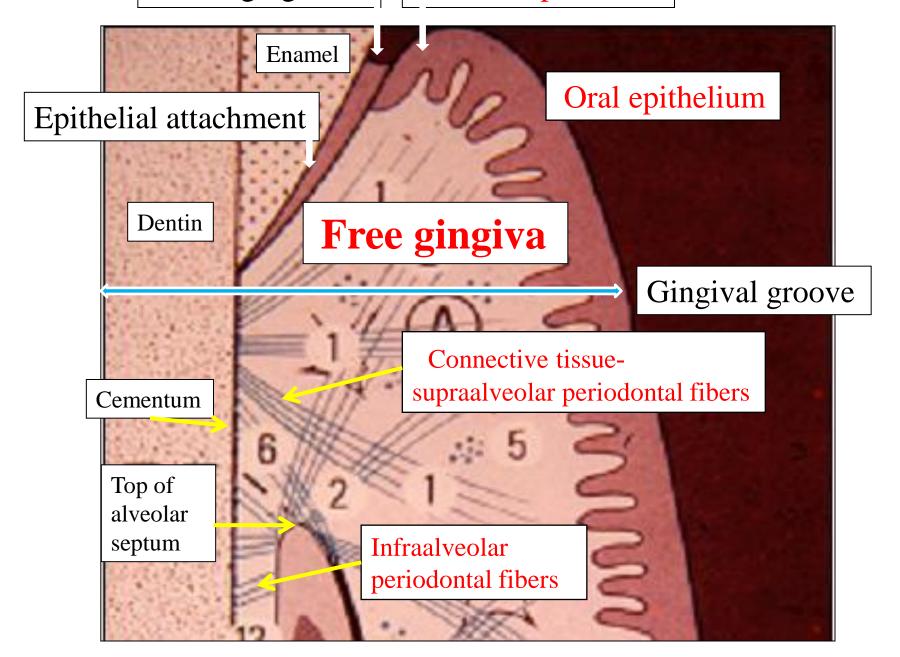
### Healthy gingiva

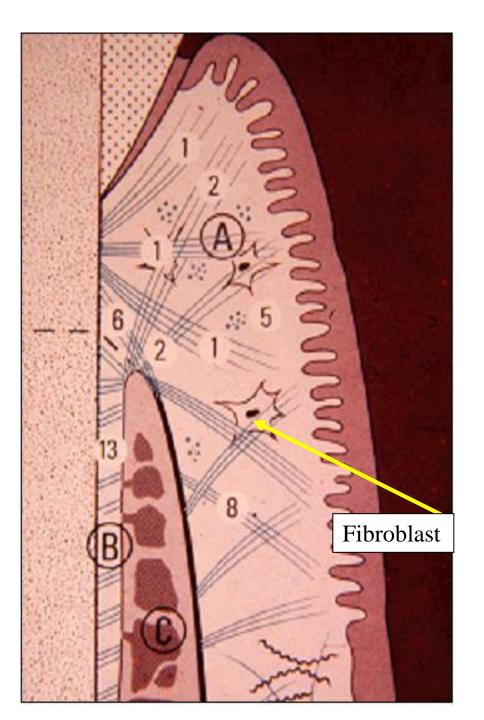
- white/pink color
- stiff consistency
- stippling
- relativly firm
- no bleeding



Sulcus gingivalis

Sulcular epithelium

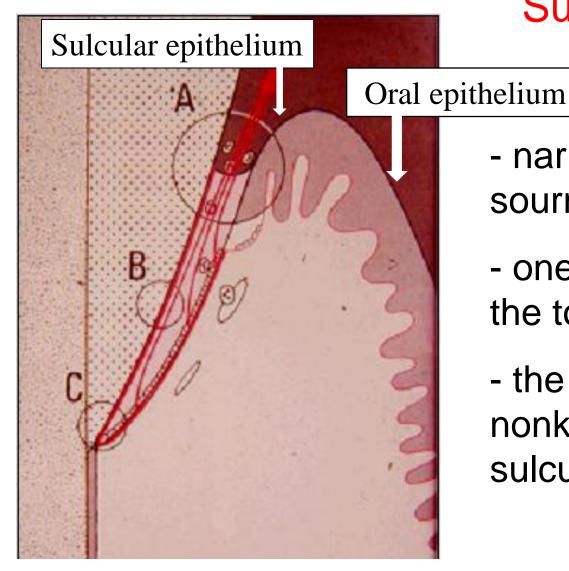




A – Gingival fibers
supraalveolar part of
periodontium – supracrestal
attachment

- fix a gingiva to the bone base, to the tooth root
- provide turgor of gingiva and its firm adherence to the tooth surface
- composed from collagen (that are produced by fibroblasts)

### A - sulcus gingivalis 0,5-1mm

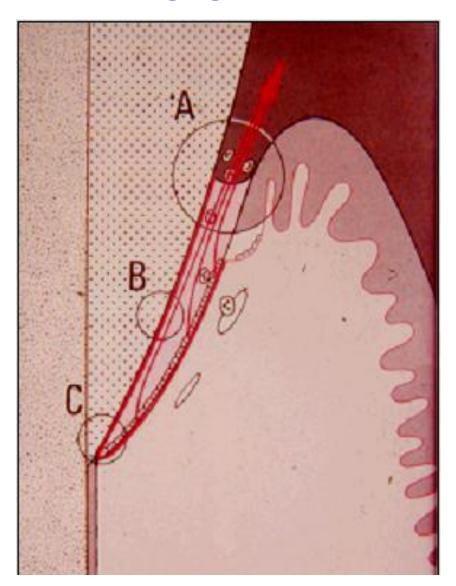


### Sulcus gingivalis

- narrow groove sourrounding the tooth
- one wall is made up of the tooth structure
- the other wall is the nonkeratinized oral sulcular epithelium

B,C - junctional epitelium 2mm

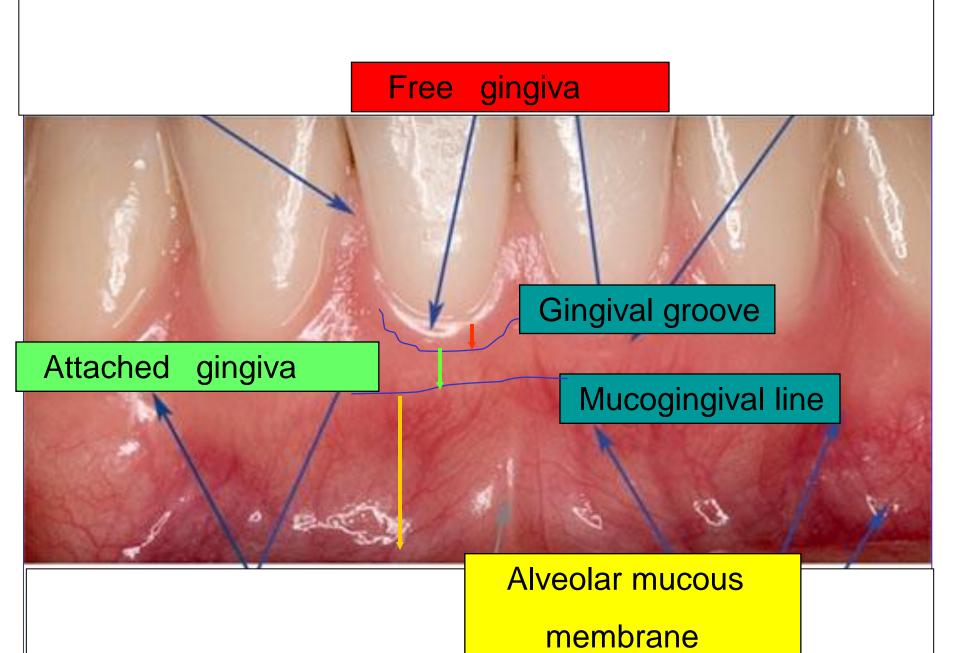
### A - sulcus gingivalis 0,5-1mm



B,C - junctional epitelium 1-2mm

### Junctional epitelium

- (≈ dentogingival junction, epithelial attachment)
- surrounds the neck of each tooth
- conection between soft and hard tissue, formed by cells
- basal layer and 10-20 suprabasal cell layers
- cells quickly regenerate 4-6 days (oral epithelium 6-12 days)
- undifferentiated,
   nonkeratinized epithelium
- attached by hemidesmosomes



### On the palatal aspect

- the mucogingival line is absent
- gingiva is a part of keratinized mucosa
- rugae palatinae





Width of attached gingiva varies from tooth to tooth and the width is individual (healthy minimum is 1-2 mm)

# Gingival biotype - thick, medium, thin



Sufficient width of attached gingiva and thick gingival biotype



Thin gingival phenotype, gingival recession at the tooth 41 - consequence of piercing



There is insufficient width of the attached gingiva at the tooth 41 (and 42) and gingival recession (exposed neck)



Attached gingiva is completely absent at lower canines, there is insufficient width of the attached gingiva in lateral incisors - association with gingival recessions





### Shallow vestibulum

- (≈ insufficient width of the attached gingiva)
- pulls the marginal gingiva
- gingiva is chronically irritated→ inflammation
- worsens hygienic condition
- gingival recessions

Lip frenula attachment

Fyziological ————



Gingival



Papilllary



Frenulum passing through the papila

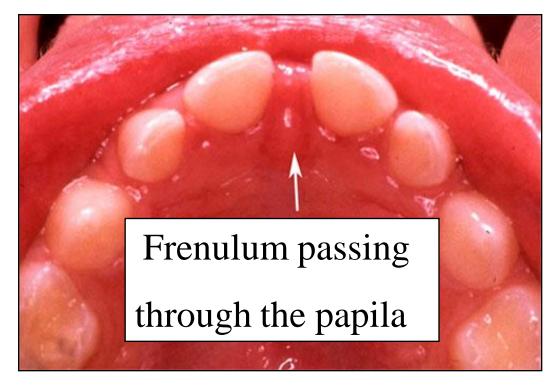


# Lip frenula with "high attachment"



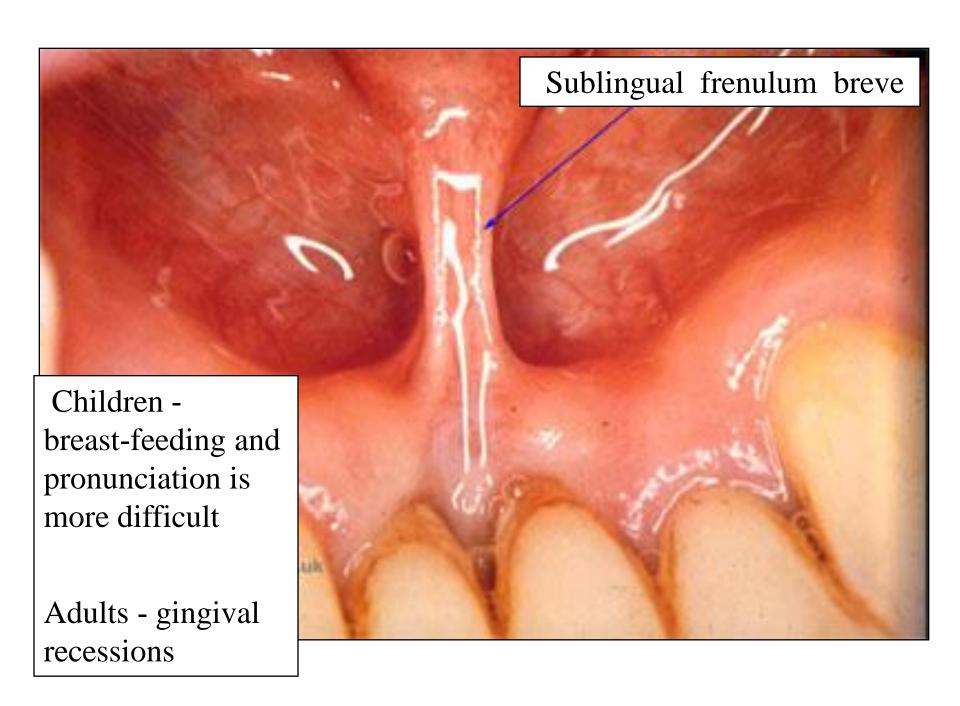


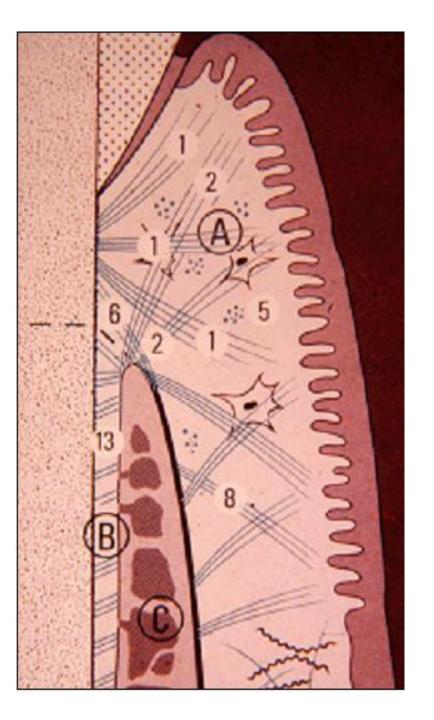
- pulls upon the gingival margin and the interdental papilla
  - worsens hygienic condition
  - gingiva is chronically iritated and inflammed
  - it can cause locallized recession







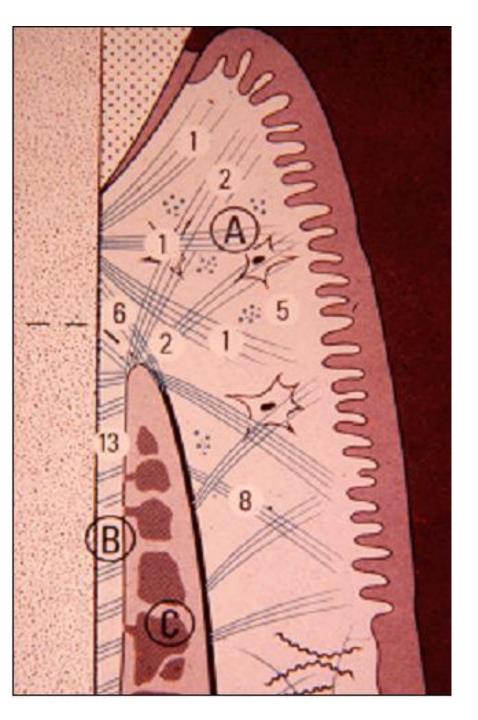




# Periodontal membrane with periodontal ligaments

B – Periodontal fibers infraalveolar part connects the tooth to the alveolar bone

- composed from collagen (produced by fibroblasts)
- very adaptable and able to react to the physiological forces
- high rate of remodeling and turnover



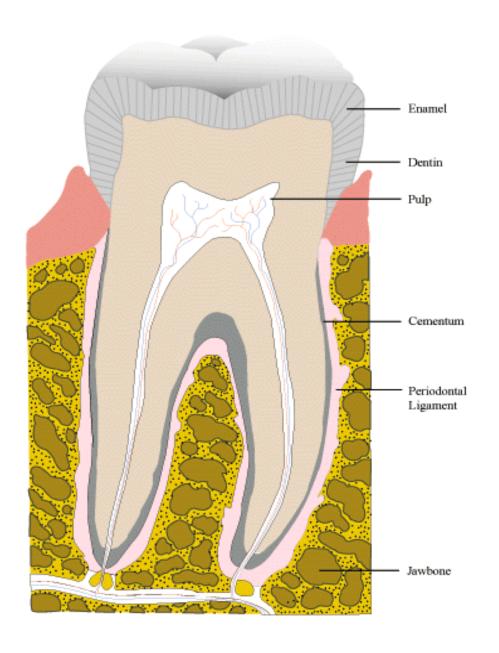
### Periodontal fibers

- Ligaments lead from lamina dura of an alveolus to the cement of a root's surface
- Tooth is set in an alveolus and may move slightly upon a mastication load
- Chewing pressure distributes on the whole bone socket without overloading



### Plexus gingivalis

- vascular plexus, has rich blood supply
- enable high metabolismus of cells and tissue
- hydraulic pressure distribution of chewing pressure
- forms a source of immunocompetent cells to migrate from this location to the surrounding tissue



#### Root cementum

- covers the root surface
- thickness 0,2 1 mm
- avascular
- histologically more types

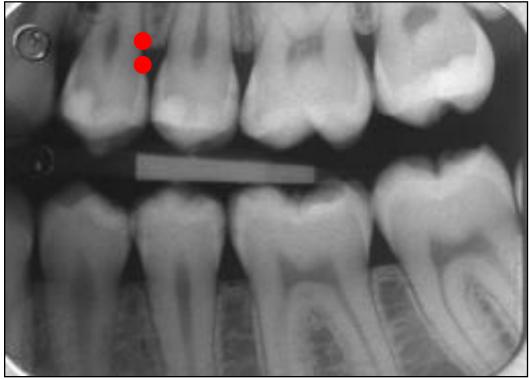
- Alveolar bone tooth socket, interdental septa, interradicular septa
- spongious bone
- compact bone (lamina corticalis)
  in the radiograph linea corticalis lamina dura
  in the region of root apex lamina cribrosa

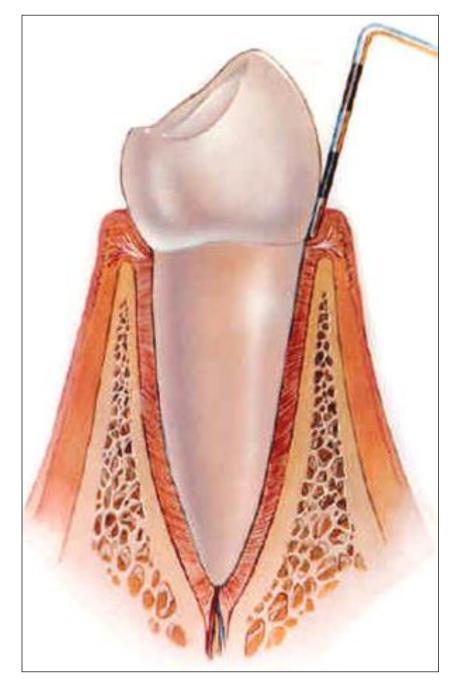


### Healthy periodontium

- lamina dura is present
- distance cementoenamel junction margin of alveolar bone is 1-2 mm





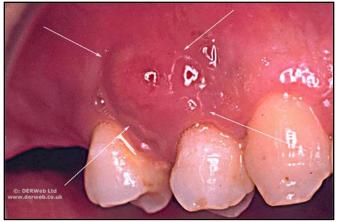


## Healthy periodontal tissue









Healthy gums?









# Etiology of inflammatory perio diseases – gingivitis, periodontitis

- Exogenous factors
  - dental microbial plaque (main factor)
  - other local (irritant) factors: calculus and other plaque retention factors, articulation, orthoanomalies, soft tissue anomalies...
  - smoking
- Endogenous factors (systemic)
  - immunity
  - systemic diseases (diabetes mellitus, ...)

# Calculus - calcified dental plaque

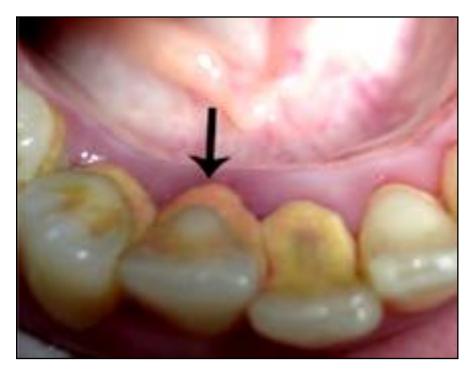
- calculus is always covered by an unmineralized layer of bacterial plaque
- good place for plaque accumulation
- reservoir and retention web for bacteria and endotoxins





# Calculus - calcified dental plaque

- Calculus is formed by the deposition of calcium and phosphate salts in bacterial plaque
- salts are present in saliva, in crevicular fluid





# Calculus - calcified dental plaque

- inorganic compounds (40% 80%), as well as proteins and carbohydrates, microorganisms
- the mineralization starts in centers intracellulary in bacterial colonies or extracellulary from matrix with crystallization nuclei (different crystals of calcium phosphate)
- time required for the formation of calculus is individually variable
- rate of formation depends on the quality of saliva and on the level of OH

# Origin of minerals and Location

- Supragingival calculus
- source saliva
- excretion ducts of the major salivary glands
- on the lingual surfaces of the mandibular incisors
  - on the buccal surfaces of maxillary molars

- Subgingival calculus
- source sulcular fluid

- on the root surfaces below the gingival margin
- can extend deep into periodontal pockets

- Subgingival calculus
- on the root surfaces below the gingival margin
- can extend deep into periodontal pockets





#### Local plaque retention factors

- Locations where there is a worse approach to teeth brushing and thus more plaque accumulation
- Dental calculus (plaque carrier)
- Faulty restorations
  - overhanging fillings
  - non-fitting crowns
- Orthoanomalies (crowded teeth...)
- Orthodontic appliances, dentures





#### Local plaque retention factors

- Anatomical deviations of mucous membranes
  - lip frenula with high attachment
  - shallow vestibulum, gingival recessions



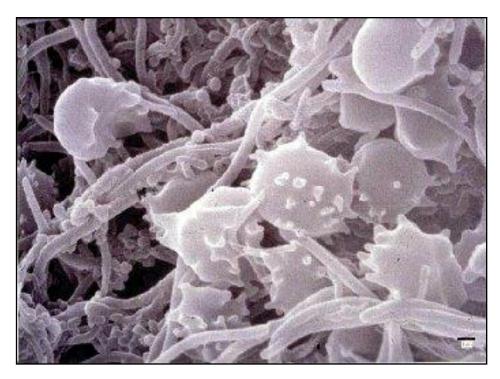
- soft deposits (bacterial mass) that form the biofilm adhering to the tooth and other intraoral surfaces
- may be removed by mechanical means only

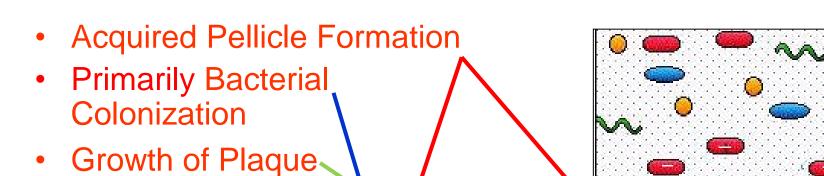


- Composed of bacteria in a matrix
- Microorganisms (75 %) a their products
- Matrix (25%)
  - bacterial (extracellular polysacharids) and salivary origin (salivary glycoproteins and mucopolysacharids)
  - calcium, phosphates (mineralization of plaque)

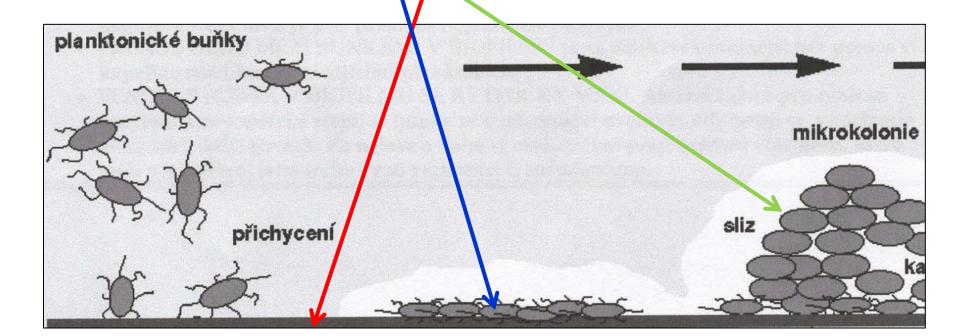
Composition and formation rate depends on

- quality of OH
- quality of saliva
- food, smoking
- immunity





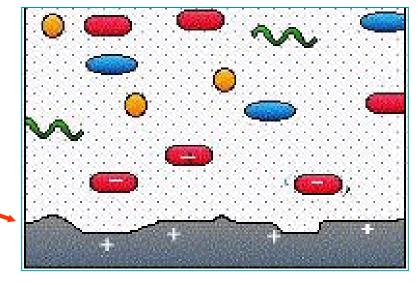
Maturation of Plaque



- Acquired Pellicle Formation
  - minutes, 1-2 microns thick
  - amorfous film from salivary glycoproteins

- increases the efficiency of bacterial

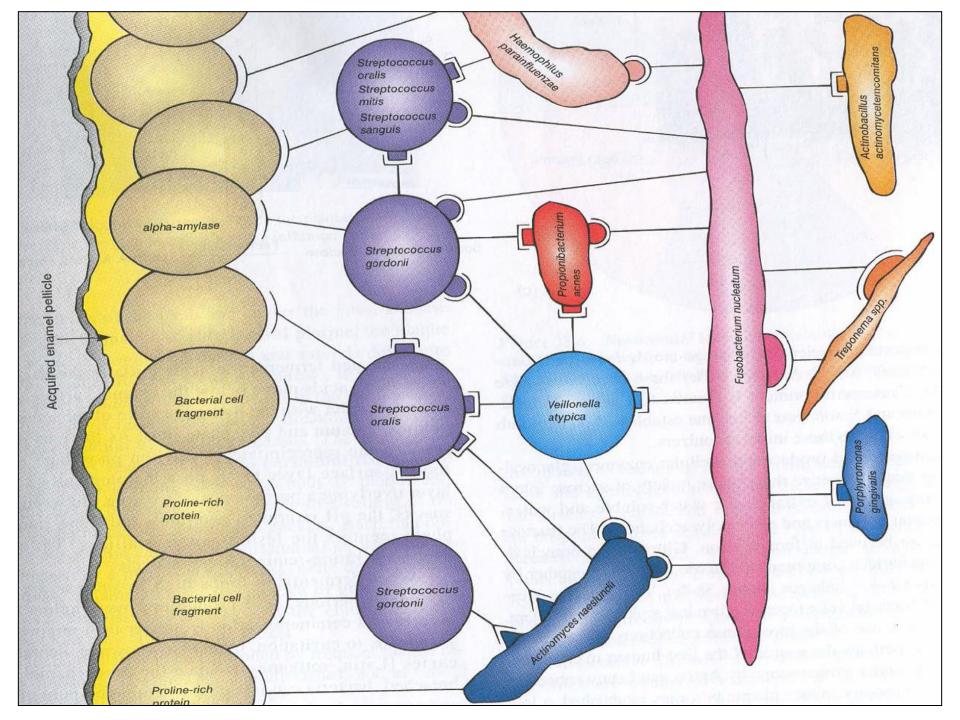
adhesion

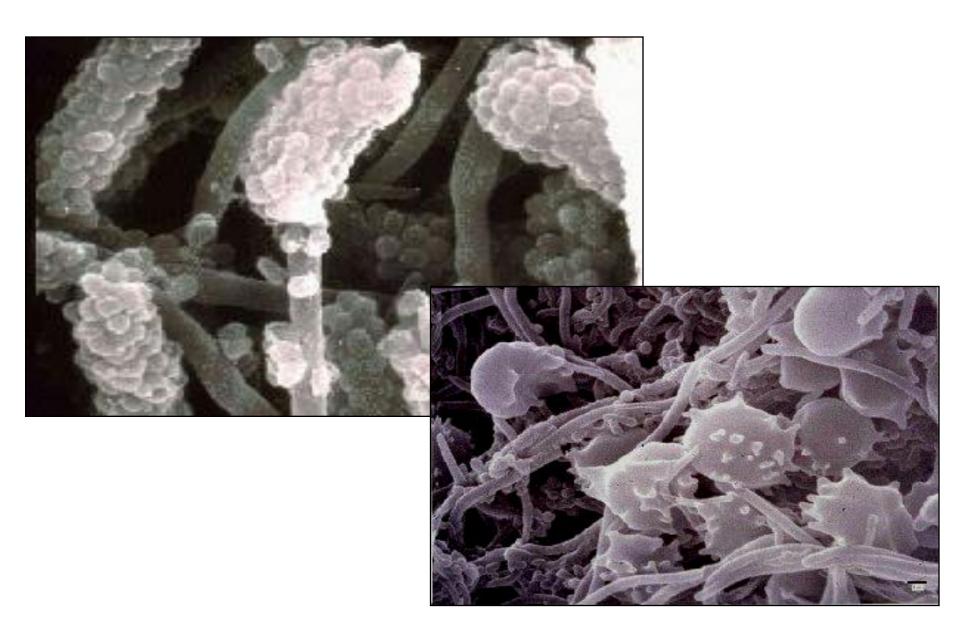


- Primarily Bacterial Colonization
- bacterial adhesion by single microorganisms
- extracellular polymeric substances and fimbriae, enable them to attach rapidly upon contact
- become established within 24 hours
- G+ aerobs, cocci (Streptococcus sanguis), G+ rods, G+ fillaments (Actinomyces sp.)
- immature plaque less adherent

- Growth of Plaque in next few days
- bacterial mass increases in quantity due to adhesion of new bacteria (surface receptors on G+ cocci and rods allow adherence of G-(Fusobacterium nucleatum) and synthesis of extracellular polymers
- multiplication of adhering bacteria and growth of extracellular matrix
- increasing of thickness diffusion is more difficult poor diffusion of oxygen - anaerobic conditions
- G- cocci, G+ G- rods and filaments (fusobacteria), aerobs and anaerobs

- Maturation of Plaque
  - formation of more complex and mature biofilm
- stable bacterial biofilm
- different morphotypes cocci, motile rods, spirochetes (filamentous organisms predominate)
   multiplication of bacteria, new bacterial species
- mature plaque very pathogenic





- microbial community
- coexistence of different populations in the biofilm
- bacteria communicate in different ways (coagregation, adherence, provid nutrients, exchange of genetic material)
- this symbiosis gives new features and greatly increases the resistance of dental plaque
- can be removed by mechanical means only

# Bacterial complexes as described by Socransky et al :

- 5 complexes:
- Primary colonizers:

Streptococcus species Actinomyces odontolyticus

Secondary colonizers:

- Elkenela corrodens
- A.a comitans serotype a
- Capnocytophaga species

- Fusobacterium
- Prevotella intermedia
- Camplylobacter sp.

- Porphyromonas gingivalis
- Tannerella forsythia
- Treponema denticola

- perio pathogens

# Perio patogens



- Porphyromonas PG gingivalis
- Tannerella forsythia TF
- Treponema denticola
- Prevotella intermedia
- Fusobacterium nucleatum
- Peptostreptococus micros

capable of invading the soft tissues of the gingiva





- coronar
- fissural



supragingival – in gingival region



- subgingival
  - 1 in sulcus gingivalis of healthy periodontium
  - 2 in periodontal pocket

### Subgingival plaque (sulcus × pocket)

 adherent plaque (enamel, root surface) composition in gingival sulcus resembles the supragingival plaque (G+ and Gcocci, Actinomyces sp., rods and filaments), can become mineralized

 non adherent plaque - freely moving, G - anaerobs (motile and nonmotile rods), larger number of spirochets, no intermicrobial matrix, important role in the progression of periodontitis, bacterial invasion (AAC, PG, TF)

#### **DENTAL** microbial PLAQUE - pathogenity

#### Supragingival plaque – consequences:

- bacteria in dental plaque produce acids → caries
- mineralization → supragingival dental calculus
- increase amount and source of bacterias in oral cavity

# DENTAL microbial PLAQUE – pathogenity for perio tissue

#### 1 Direct effect

a / bacterial products

- enzymes (proteases, collagenases, hyalouronidases)
- endotoxins (lipopolysaccharides of the bacterial wall)
- exotoxins (leucotoxin AAC)
- indole, skatol, ammonia, hydrogen sulphide
- b / invasion of microorganisms into perio tissue (AAC, PG, TF)

#### **DENTAL** microbial PLAQUE - pathogenity

#### 2 Indirect effect

- via inflammatory mediators (IL –1,6, TNF, PGE)
- bacterial antigens sensitize cells of the immune system and activate the immune response of the host
- there is hypersecretion of inflammatory mediators (especially by macrophages), which activate osteoclasts, resulting in bone resorption

# Pathogenity of plaque

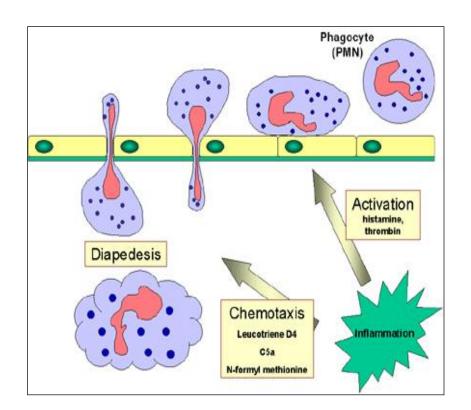
- depends on
- amount and composition of plaque
- virulence of microorganisms
- ability to invade tissues (direct invasion true infection)
- immunity reaction of organism accumulation of plaque along the gingival margin leads to inflammatory reaction of the soft tissue

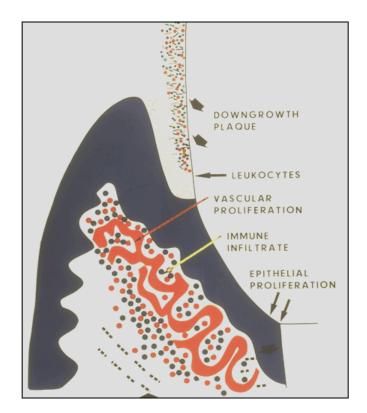
#### Host defence reaction

- Acute non-specific host response first and rapid reaction
- non-specific immune cells (macrophage, neutrophil, or dendritic cell) function in the first line of defense against infection or injury
- cells of the innate immune system do not have specific responses and respond to each foreign invader using the same mechanism
- Specific (acquired, adaptive) immunity reaction
- this type of immunity creates immunological memory after an initial response to a specific pathogen
- leads to an enhanced response to subsequent encounters with that pathogen. This process of acquired immunity is the basis of vaccination
- includes both humoral immunity components and cellmediated immunity components

# Acute nonspecific host response

 Inflammation - PolyMorphoNuclearLeucocytes chemotaxis, diapedesis, adherence to bacteria, phagocytosis, microbicidal activity, complement system





# Specific immunity reaction

- immunological memory after an initial response to a specific pathogen
- the immune system responds to antigens by producing cells that directly attack the pathogen, or by producing special proteins called antibodies
- defense cells recognize antigen
- Lymfocytes
- T cells cell mediated reaction
- B cells antibody response upon contact with antigen; they differentiate into plasma cells (that produce antibody)

#### Dental biofilm and systemic diseases

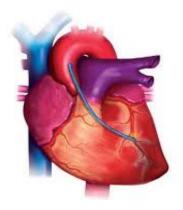
- Inflammation is not only a local matter of the periodontal tissue, but bacteria and inflammatory mediators enter the systemic circulation
- Bacteremia
- Inflammatory mediators in systemic circulation (interleukins, TNF alpha, prostaglandins...)

see http://www.efp.org/newsupdate/oral-health-and-general-health

#### Dental biofilm and systemic diseases

- 9th European Workshop in Periodontology "Periodontitis and Systemic Diseases" 2012
- The Perio-Diabetes Workshop 2017 (organised jointly by the European Federation of Periodontology (EFP) and the International Diabetes Federation (IDF)
- See below https://www.efp.org/perioworkshop/workshop-2012/index.html





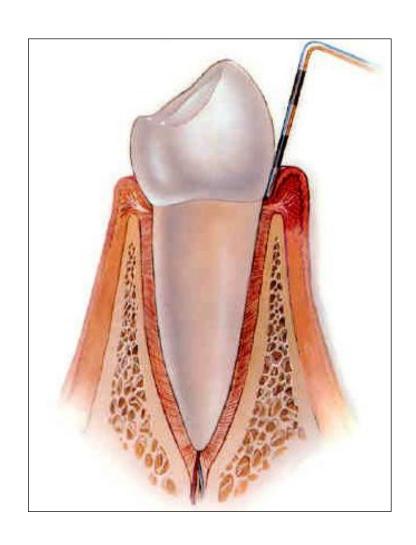




# Gingivitis - symptoms

- gingival bleeding
- redness to livid colour
- swelling
- gingiva loses its turgor
- tenderness or pain

- inflammation of the gingival tissue surrounding a tooth
- reversible





Gingivitis showing edema, redness and bleeding on probing







# **Gingivitis**

- 1. Plaque Associated Gingivitis (90 95%)
- Gingivitis induced with plaque only
  - modified by local factors
- Gingivitis modified by systemic factors
  - hormone induced gingivitis (puberty, pregnancy)
  - DM
  - medications
  - malnutrition (vit C)

#### 1/ Gingivitis induced with plaque only

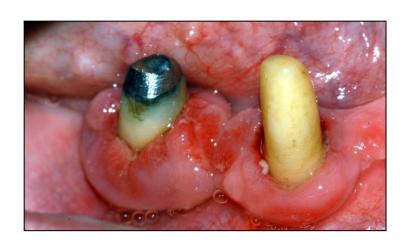
with plaque only/+ local factors





(overhanging fillings, crowns, bridges, mouth breathing, crowding of teeth, fixed ortho apliances, soft tissue anomalies,...)





#### 2/ Gingivitis modified by systemic factors

hormone induced gingivitis - puberty, pregnancy





#### 2/ Gingivitis modified by systemic factors

diabetes mellitus



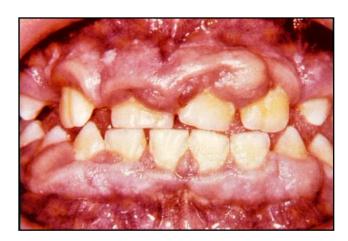
# 3/ Gingivitis modified by medication – hyperplastic gingivitis





- Calcium channel blockers (nifedipin)
- Hydantoins (fenytoin)
- Cyklosporin





## 4/ Gingivitis modified by nutrition

Avitaminosis C (scurvy)





### Acute necrotizing ulcerative gingivitis

- · Painful, rapidly progressive inflammation of gingiva
- Spirochets, fusiform bacterias
- Starts usually without general symptomes
- Gingivitis + crateriform ulcerations of ID papillae, necrosis at ID papillae





# 2. Non - Plaque - Associated Gingival Diseases (5 – 10%)

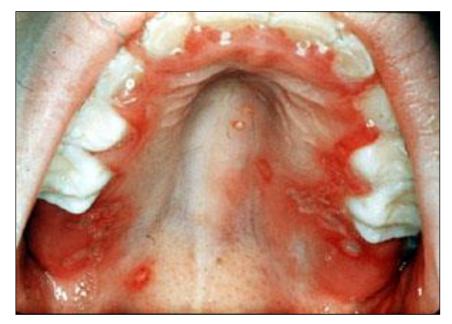
- Bacterial, Viral or Fungal associated lesions
- Hereditary Gingival Diseases
   Gingival fibromatosis
- Allergic reactions of the gingiva
- Traumatic lesions of the gingiva

#### 2. Non - Plaque - Associated Gingival Diseases



Primary Herpetic Gingivostomatitis







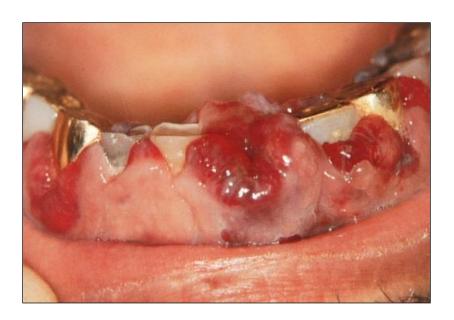


#### Desquamative gingivitis

- pemphigus
- pemphigoid
- lichen planus



## Overgrowth of gingiva (hyperplastic gingivitis) in acute leucemia





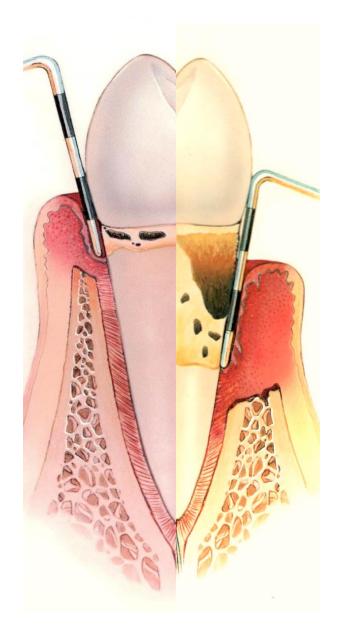
- there is a multiplication of leukemic cells in the gingiva
- gingival ulcerations



#### 2/ Periodontitis

Inflammation of the perio apparatus of the tooth

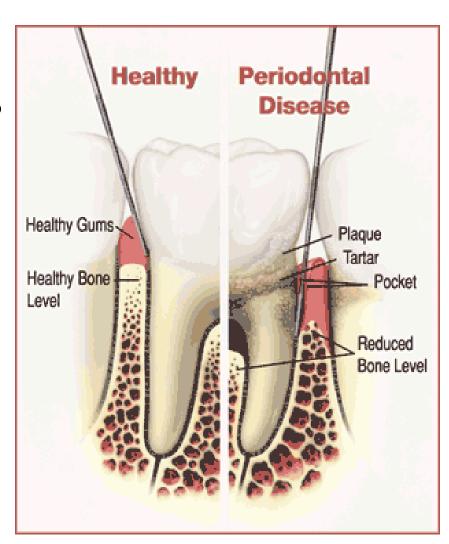
- inflammatory destruction of the junctional epithelium and gingival ligaments
- periodontal pocket formation
- resorption of alveolar bone
- irreversible

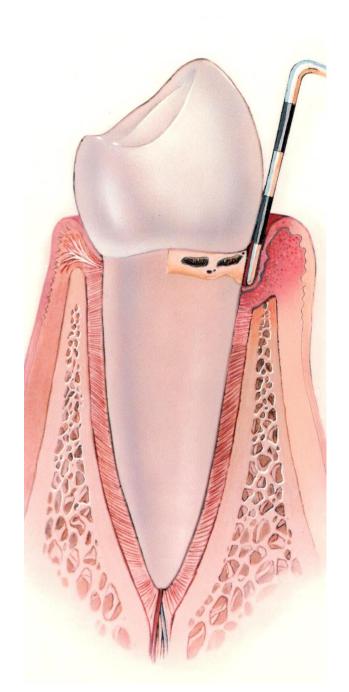


#### **Periodontitis**

 inflammation of gingival tissue extends into the junctional epitelium

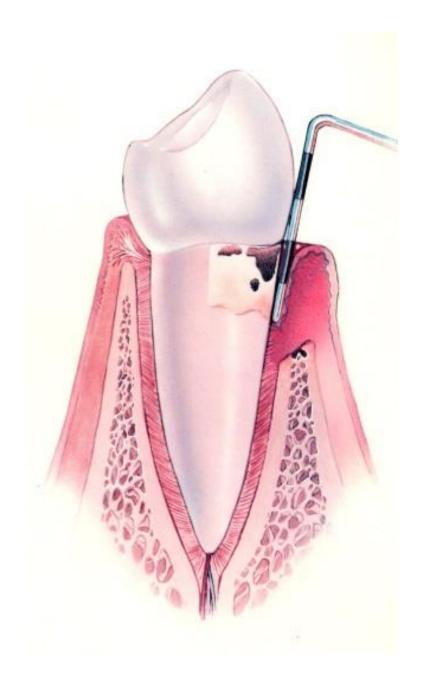
- attachment damage
- loss of alveolar bone periodontal pocket
- irreversible





#### Initial periodontitis

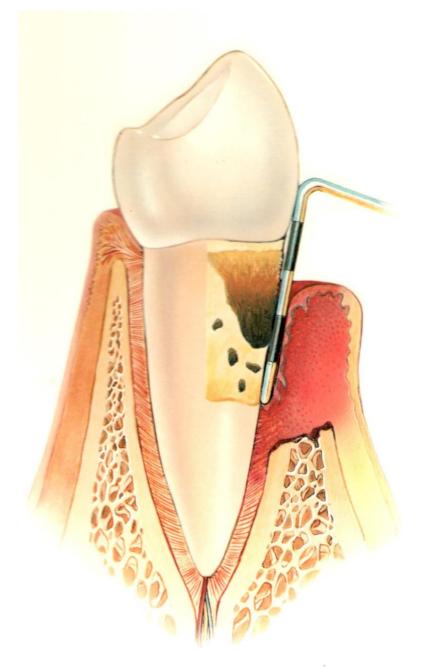
- Symptoms are mild
- gum bleeding after irritation
- gingival edema
- redness of the gingiva
- probing up to 6 mm
- mild bone resorption



### Moderate periodontitis

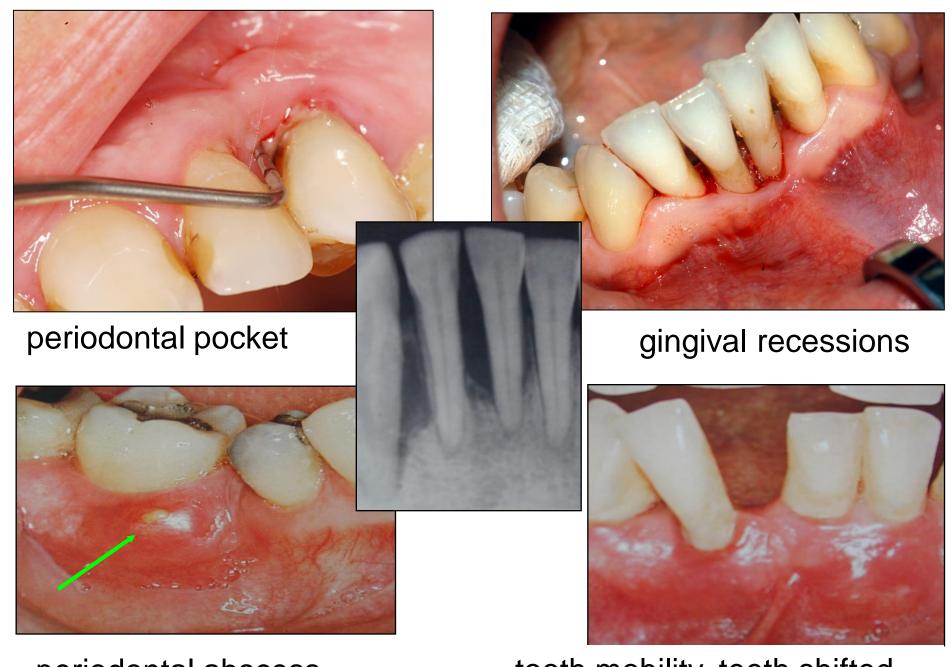






#### Advanced periodontitis

- deep periodontal pockets over 6 mm
- periodontal abscess, pus between teeth and gums
- tooth mobility, teeth tend to shift or lose itself
- significant bone resorption
- bad breath



periodontal abscess

tooth mobility, teeth shifted

#### Periodontitis - classification

Older classification

Chronic periodontitis (adults, slower progression)

Aggressive periodontitis (beginning in younger age, rapid progression, significant proportion of AAC)

New classification (from 2018)

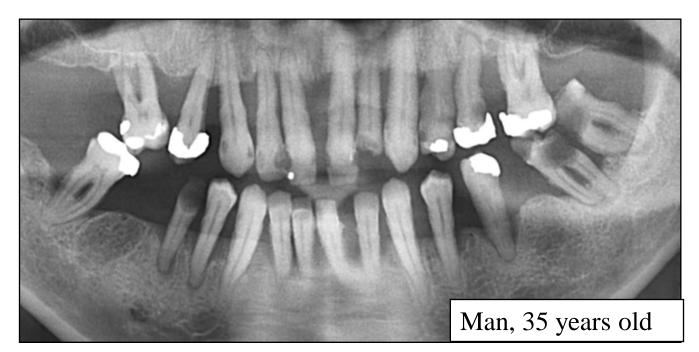
Stage I - IV (depending on attachment loss)

Grades A - C (slow, medium, fast progress)

#### Periodontitis

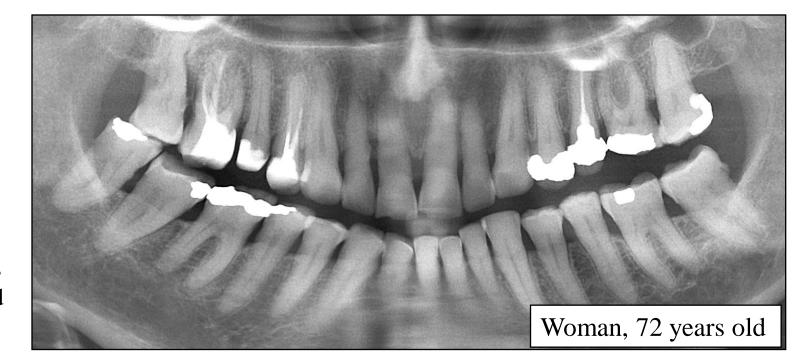
```
Chronic periodontitis (90 %)
localized / generalized
Aggressive periodontitis (5-10 %)
localized / generalized
```

Periodontitis – as a manifestation of systemic disease associated with genetic disorders (rare cases)



Agressive periodontitis

- generalized
- IV/C



Chronic periodontitis

- generalized
- III/A

- Chronic periodontitis
  - adults
  - polymicrobial infection
  - continous and slowly progression
  - horizontal resorption



#### Agressive periodontitis

- early onset
- rapid attachment loss and bone destruction
- possible familial aggregation of disease
- except for periodontal disease, patients are systemically healthy
- tissue destruction is greater than would be expected given the level of local factors including bacterial plaque

- Agressive periodontitis
  - elevated levels of Actinobacillus actinomycetemcomitans (AAC) Porphyromonas gingivalis (PG)
  - phagocyte abnormalities and increased production of prostaglandin E<sub>2</sub> and interleukin-1β





# Periodontitis – as a manifestation of systemic disease

- associated with genetic disorders
- Papillon Lefevre syndrom
  - uncommon inherited autosomal recessive disease
- hyperkeratosis of the palms and soles (knee, elbows)
- advanced periodontitis (both deciduous and permanent dentitions)
- at 20% immunologic defects







80%

**Gingivitis** 

Mild or moderate chronical periodontitis



Prevalence of perio diseases 90-95%



10%

Healthy

10%

Agressive advanced periodontitis

## Periodontal therapy

- inseparable part of dental therapy
- the goal is to eliminate the etiologic factor
- decrease the level of pathogenic microorganisms
- eliminate inflammation and periodontal pocket
- Gingivitis ? Type?
- Periodontitis? Chr Agr?

Gingival recessions?

## Periodontal therapy

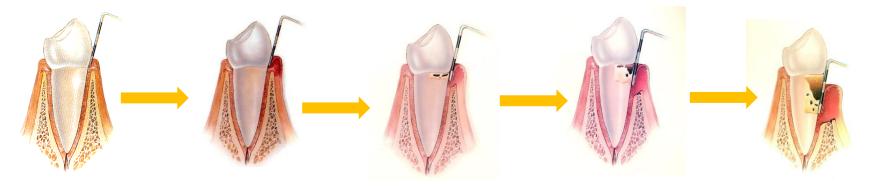
- Preliminary phase
- 1 Initial phase of periodontal therapy
- Evaluation of response
- 2 Surgical phase
- Evaluation of response
- 3 Restorative phase
- Evaluation of response
- 4 Maintenance phase (supportive therapy, recall)

#### 1- Initial phase of periodontal therapy

- Plaque control oral hygiene (motivating and instructing the patient, control)
- Elimination of plaque retentive areas and Correction of irritating factors - removing all local irritants that may cause gingival inflammation)
  - reduction of naturaly occuring plaque retentive areas
  - removal of iatrogenic irritants
- Supragingival calculus removal
- Subgingival scaling and root planinig
- Antimicrobial therapy
- Temporary restorative treatment and prosthetics (caries, root canal therapy, occlusal therapy, splinting)

# 1a - Motivation, education, oral hygiene instruction



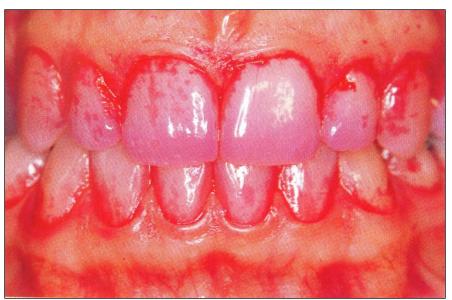












Special tablets or rinses can colour plaque



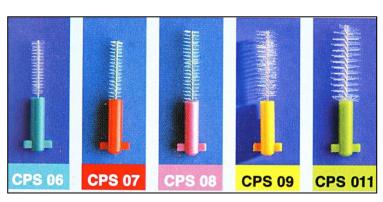


## Oral hygiene products

- Toothbrush
- Single toothbrush
- Dental floss (waxed, unwaxed, tape, flossholder)
- Interdental cleaners (correct size !)
- Toothpaste (fluorid, antimicrobial agents,

anticalculus agents)

- Oral irrigators
- Mouth rinses



#### **Toothbrush**

- short head
- soft, medium, multitufted, straight bristels



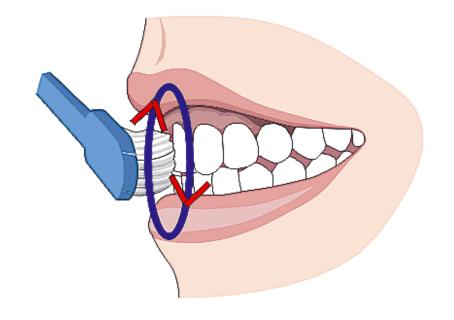


Fones method – for children

Charters method

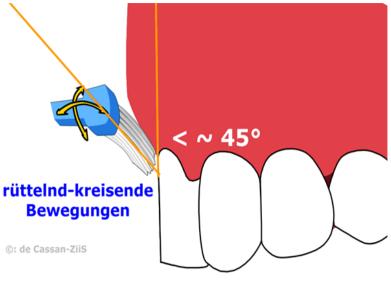
Stilmann method

Bass method

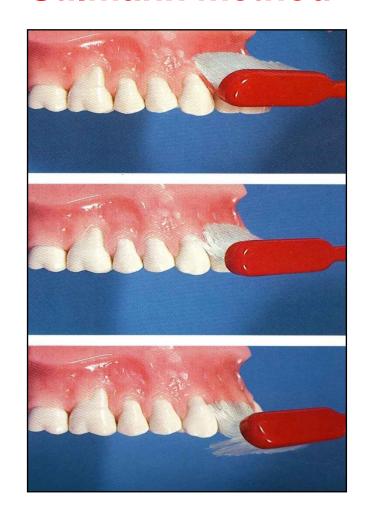


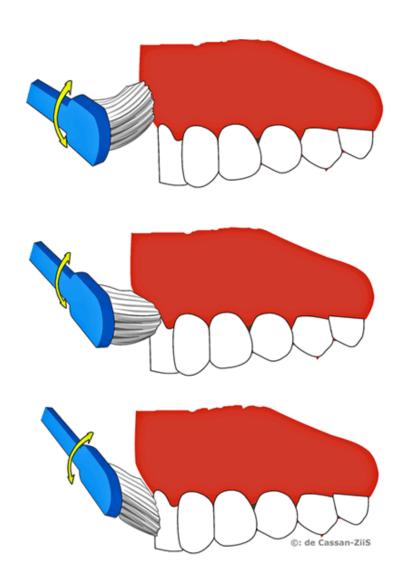
Charters method





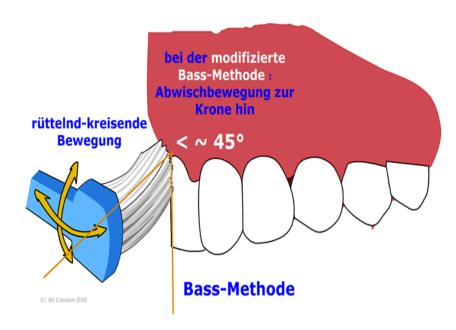
#### Stilmann method

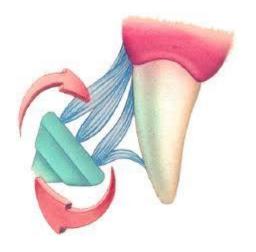




Bass method









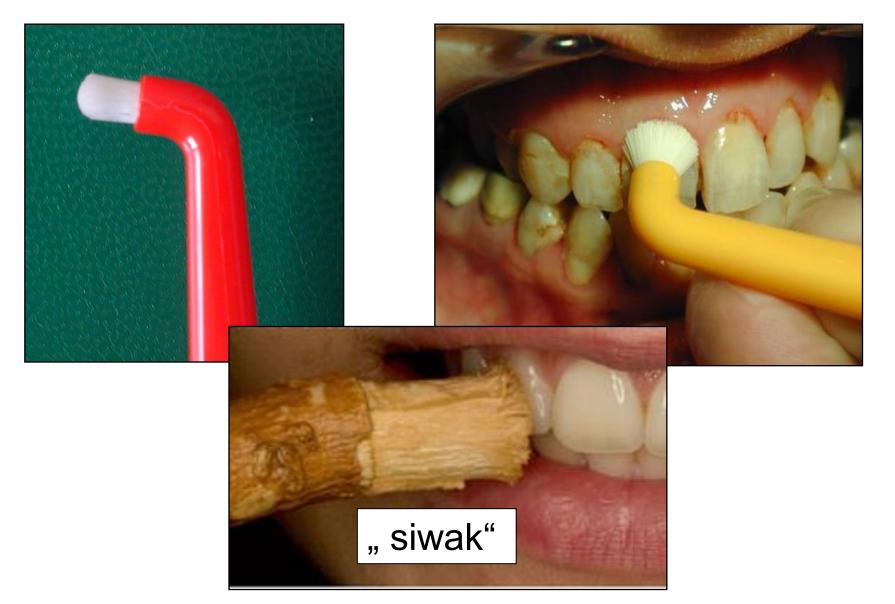
#### Pay attention !!!

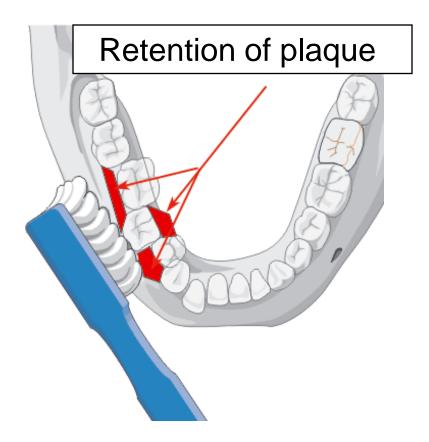
- Horizontal toothbrushing
- Hard bristels
- Toothbrushing too frequently



- abrasion of the tooth structure
- gingival recession (root exposure, hypersensitivity)

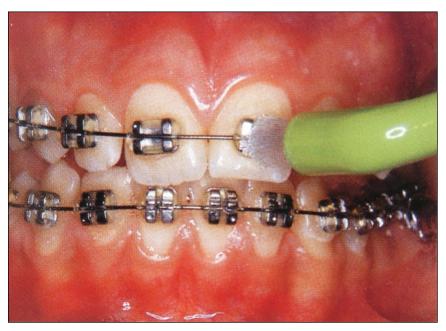
## Single toothbrush





- Crowded teeth
- Orthodontic apliances (braces)
- Implants

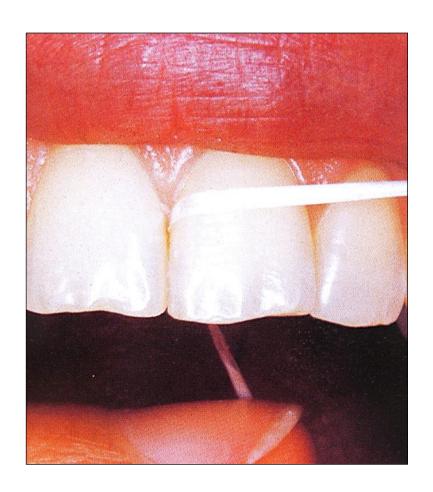


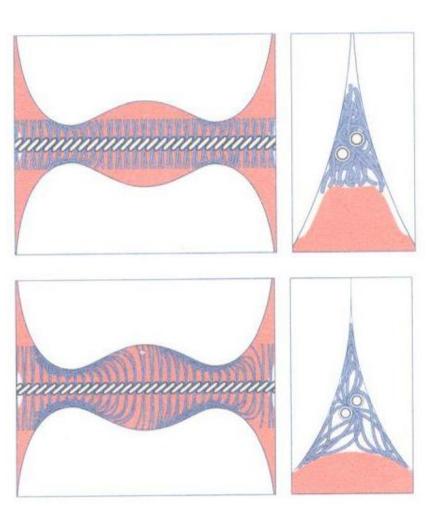


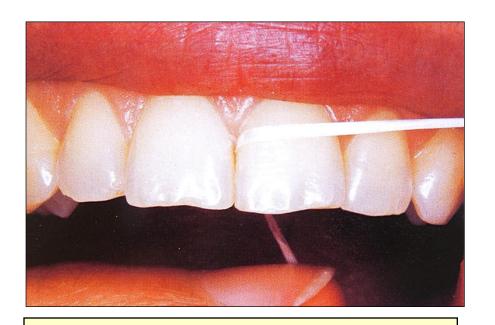
## Interdental hygiene

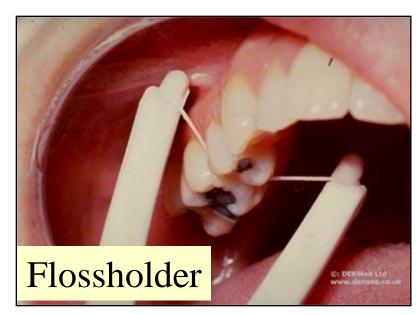
Dental floss

Interdental cleaners

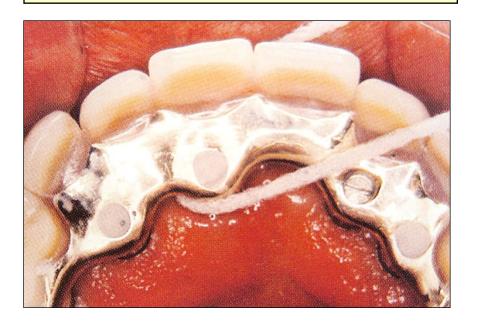


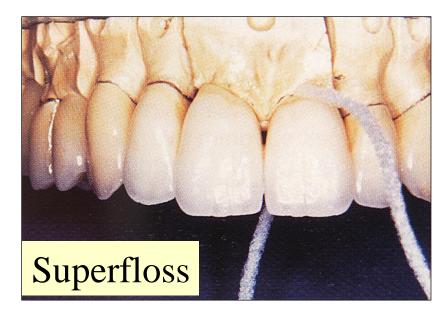






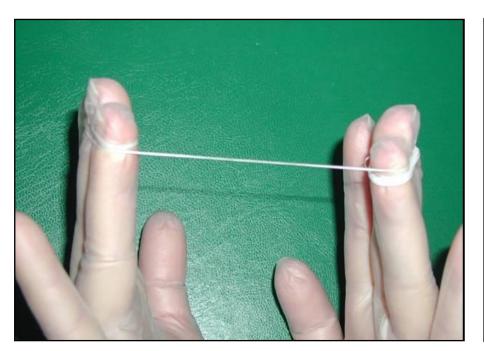
Floss - waxed, unwaxed

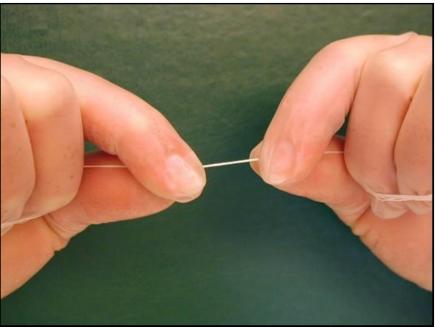




- break off about 15 20 cm
- roll floss around middle fingers

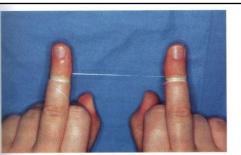
- Flossing
- hold the floss between thumbs and index finger
- guide floss between your teeth- curve it into a C shape against one tooth
- slide it into the space between the gum and the tooth
- move the floss up and down
- do not forget back side of last tooth











Obr. 9a "Upevnění" dentální nitě namotáním na prostředníky obou rukou.



Obr. 9b Zbylé konce se uchopí palci a ukazováky obou rukou.



Obr. 10 Dentální nit musí být napjata kolem každé čištěné proximální plochy.

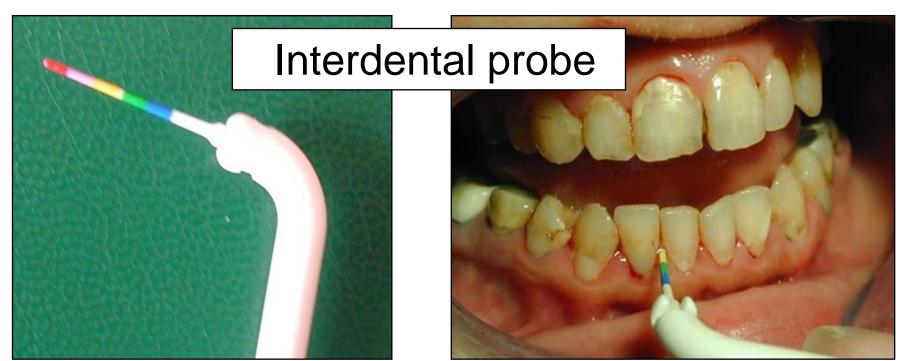


Obr. 11a a b Rozsah pohybu dentální nitě ve vertikálním směru.















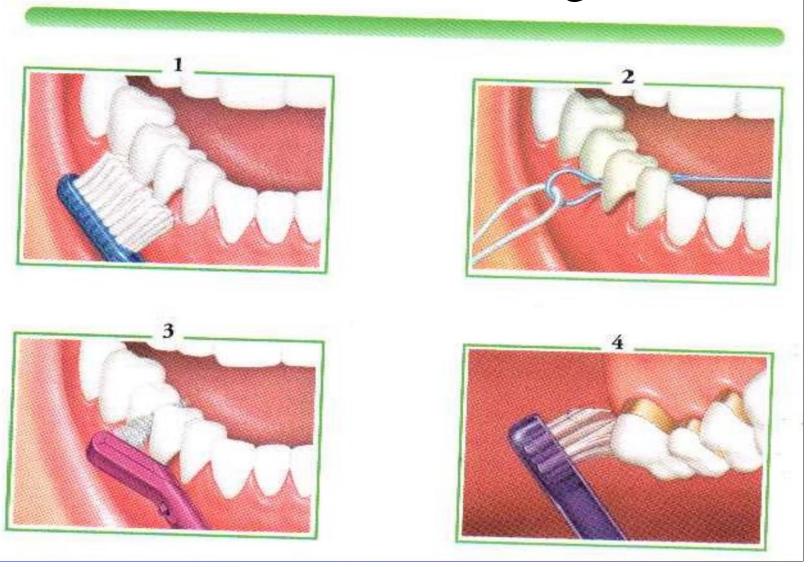




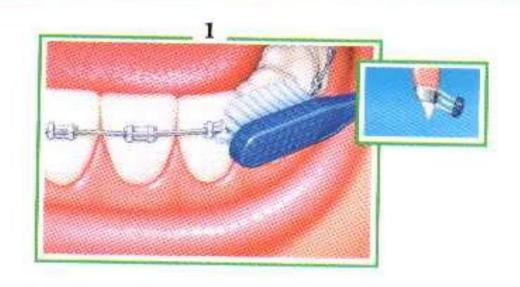


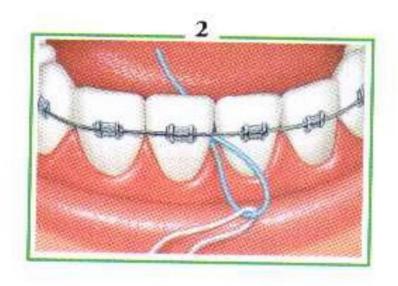


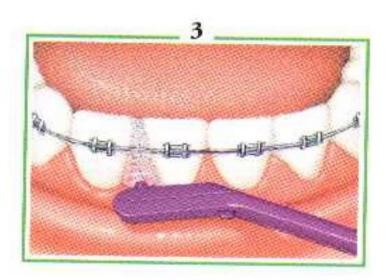
## How to clean bridges



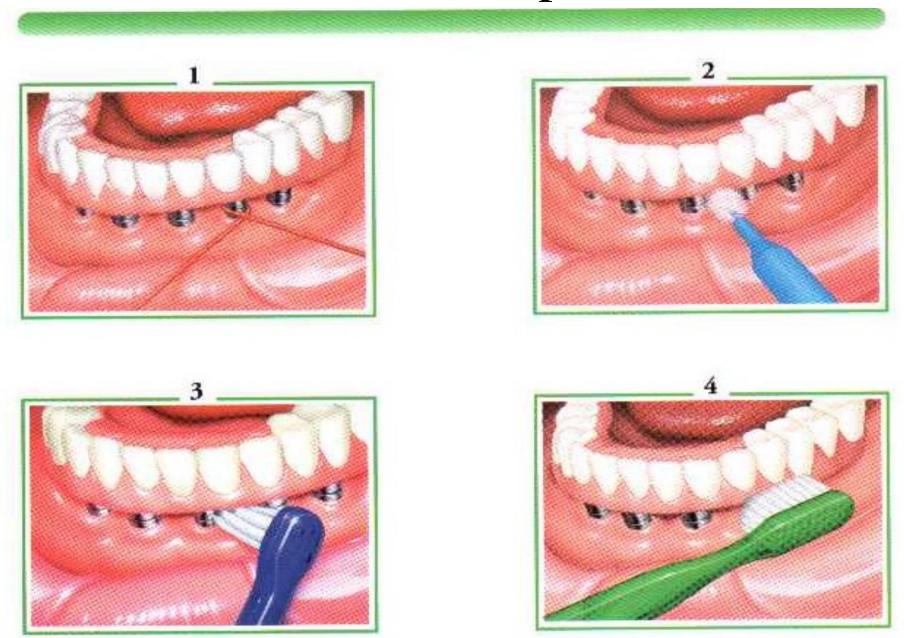
### How to clean orthodontic appliances







## How to clean implants



# Toothpaste

- Abrasive polishing compounds (improve mechanical cleaning)
- Fluorid compounds (NaF, Aminofluorid)
- Antimicrobial compounds (triclosan, CPC, CHX)
- Anticalculus compounds

### Mouthwash - antiseptic agents

Chlorhexidin (0.06 – 0.2 %) gold standard



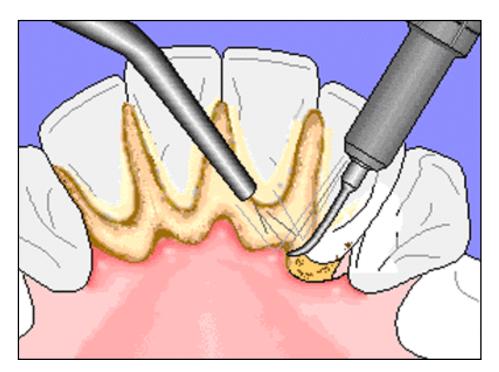
- 0,2% CHX max 2 weeks; 0,12% 0,1% 0,06%
- Unpleasant side effects
  - stainig of teeth and tongue
  - taste disturbances
  - mucosal desquamation

It is not necessary to rinse daily with antiseptic mouthwash, we use it at some stage of periodontal treatment

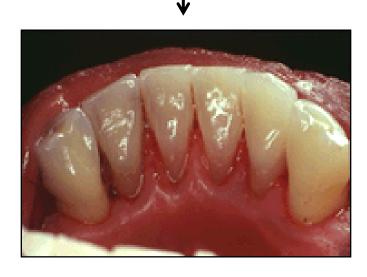
- Adjunct during initial therapy
- Desinfection of oral cavity before dental treatment
- In handicapped patients
- Periodontal surgery

# Initial phase

- 1b Supragingival removal of the calculus and bacteria (hand and sonic /ultrasonic instruments)
  - recontouring defective restorations and crowns









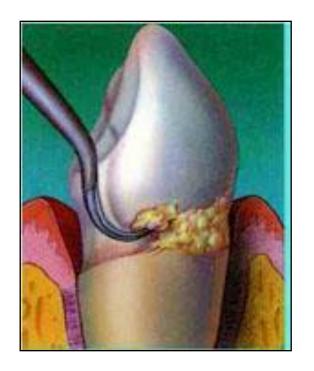
Scalers – hand instruments

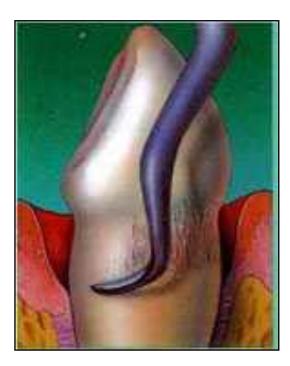


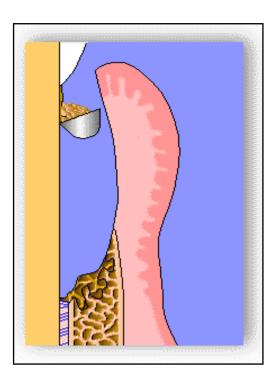


# Initial phase

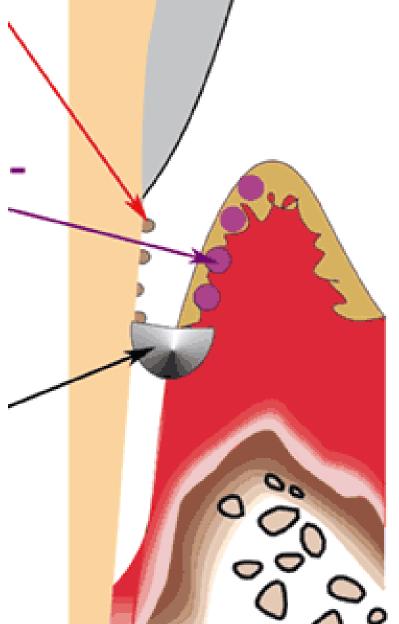
- 1c Subgingival treatment of perio pocket scaling and root planinig (SRP)
- removal of subgingival plaque and callculus
- smoothing of the dental root surface







#### Gracey curettes – scaling and root planinig





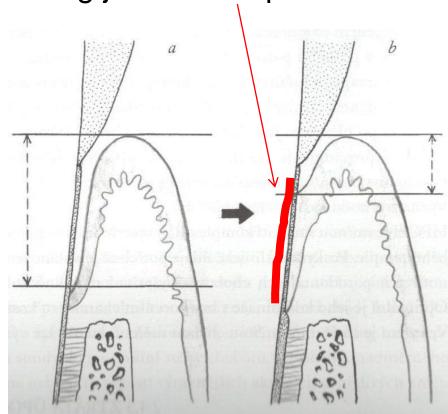


# Results of scaling and root planing

- debridement of bacteria and calculus
- removal of infected cementum and dentin
- a shift in the microbial population

# Healing after scaling and root planing – by repar (scar healing)

- epithelial and connective tissue
- long junctional epithelium



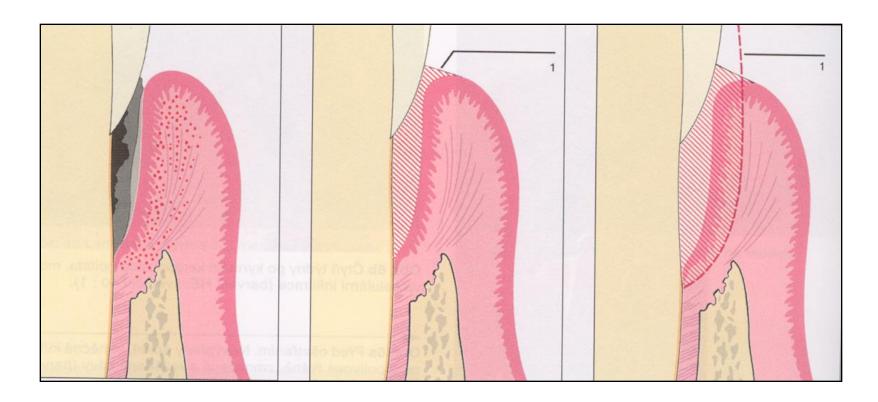
#### 1d - Antimicrobial prophylaxis

Local - oral rinse (usually with CHX)
 reducing growth of plaque, CHX is not able
 to destroy existing plaque !!!

 Systemic antibiotics treatment (against anaerobes - metronidazol, amoxycilin, tetracyclin) if needed (aggressive periodontitis), always at the same time as treatment of perio pockets (SRP)

# 2 - Surgical phase

- Elimination of periodontal pockets
- Better gingival configuration
  - better conditions for oral hygiene







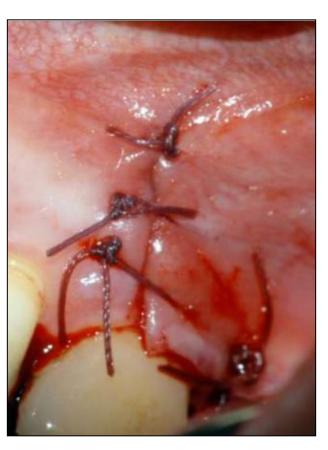
# 2a/ Flap surgery in case of unsuccessful conservative therapy

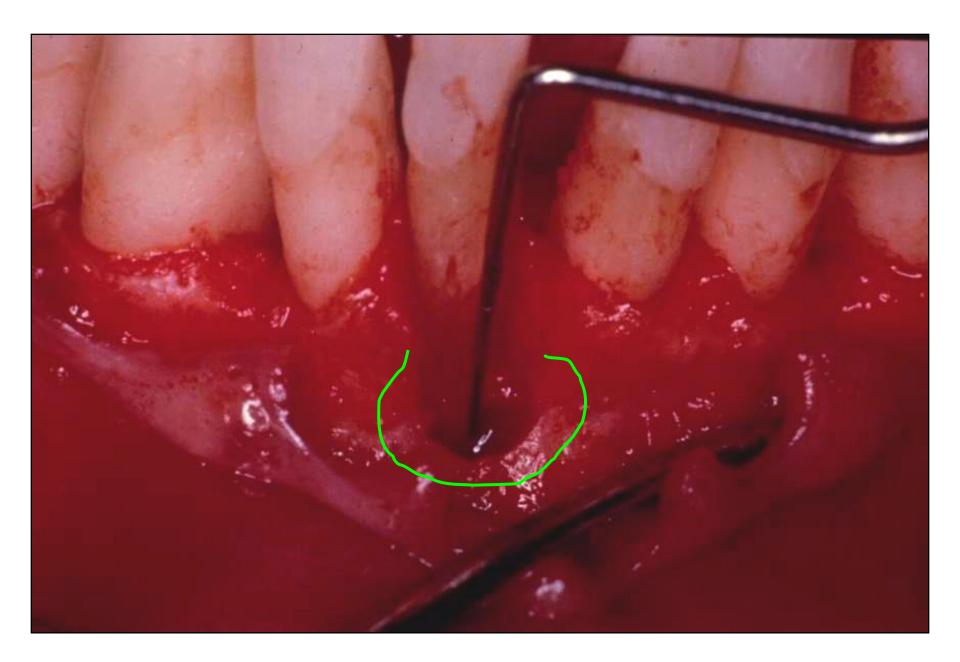
- treatment of a defect under direct visual control
- surgical exposure of the root by opening gingival flap
- removal of residual subgingival plaque and callculus
- smoothing of the dental root surface
- open operation field
- the flap is raised and then fixed

### Flap surgery – open flap curettage









### 2b/ Mucogingival surgery

- Periodontal-prosthetic corrections Crown lengthening Socket preservation • Ridge augmentation • Esthetic surgical corrections • Coverage of the denuded root surfaces • Reconstruction of papillae
  - Esthetic surgical correction around implants •

Upper frenulectomy in connection with ortotreatment of diastema





Enlargement of attached gingiva with epithelial graft from palate



# Clean teeth are the main condition of healthy teeth and periodontium

