Dermatologie en orale aandoeningen? Jazeker!

De mond toegangspoort tot ons lichaam.

Dirk Verbist
Liesbet Matthijs
Stijn Vervaeke

Tandarts en huisarts: een l.a.t.-relatie.

- Toespraak van prof. De Maeseneer ter gelegenheid van de promotie tandartsen 2007

- Op zich is het zeer merkwaardig dat wij in de voorbije decennia zo “apart” zijn gaan leven.
- Als huisarts worden wij als laagdrempelige hulpverlener op de eerste lijn, regelmatig geconfronteerd met mondproblemen, al dan niet door de patiënt als “reden tot contact” geformuleerd.

Tandarts en huisarts: een l.a.t.-relatie

- De inspectie van farynx en tonsillen voor de frequente klacht “keelpijn” voert de blik van de arts ”in het voorbijgaan” immers door de mondholte: gebit, tandvlees, en tong passeren de revue.
- Als huisarts zien we daarbij verschillende zaken: verkleuring, irritatie, cariës, tandsteen…
- In eerste instantie zijn we hierbij geïnteresseerd in wat normaal versus afwijkend is, maar vooral willen we weten wat “pluis” en “niet pluis” is.

Tandarts en huisarts: een l.a.t.-relatie.

- Niet louter de klinische observatie, maar ook de vaststelling gedurende de laatste 10 jaar dat ontstekingen in de mondholte een weerslag hebben op de algemene gezondheid, vormt een motivatie voor meer samenwerking.
- Het staat tegenwoordig vast dat deze ontstekingen mede verantwoordelijk zijn voor arteriosclerose en de daaruit volgende hart- of herseninfarcten en dat ze ook de frequentie van vroeggeboorten en laag geboortegewicht doen toenemen.

Tandarts en huisarts: een l.a.t.-relatie.

- Er zijn ook verbanden met gewrichts- en longaandoeningen.
- Deze algemene gezondheidsimplicaties wijzen erop dat er naast het behoud van een gezond en functioneel gebit ook andere redenen bestaan om aan de mond de noodzakelijke zorg te besteden.

programma

1 Anatomie van de orale weefsels en zijn varianten

2 Mucosapathologie

3 Plaquegerelateerde parodontale infecties

4 Interactie mongezondheid – algemene gezondheid

5 Gebruik van medicatie in de parodontologie

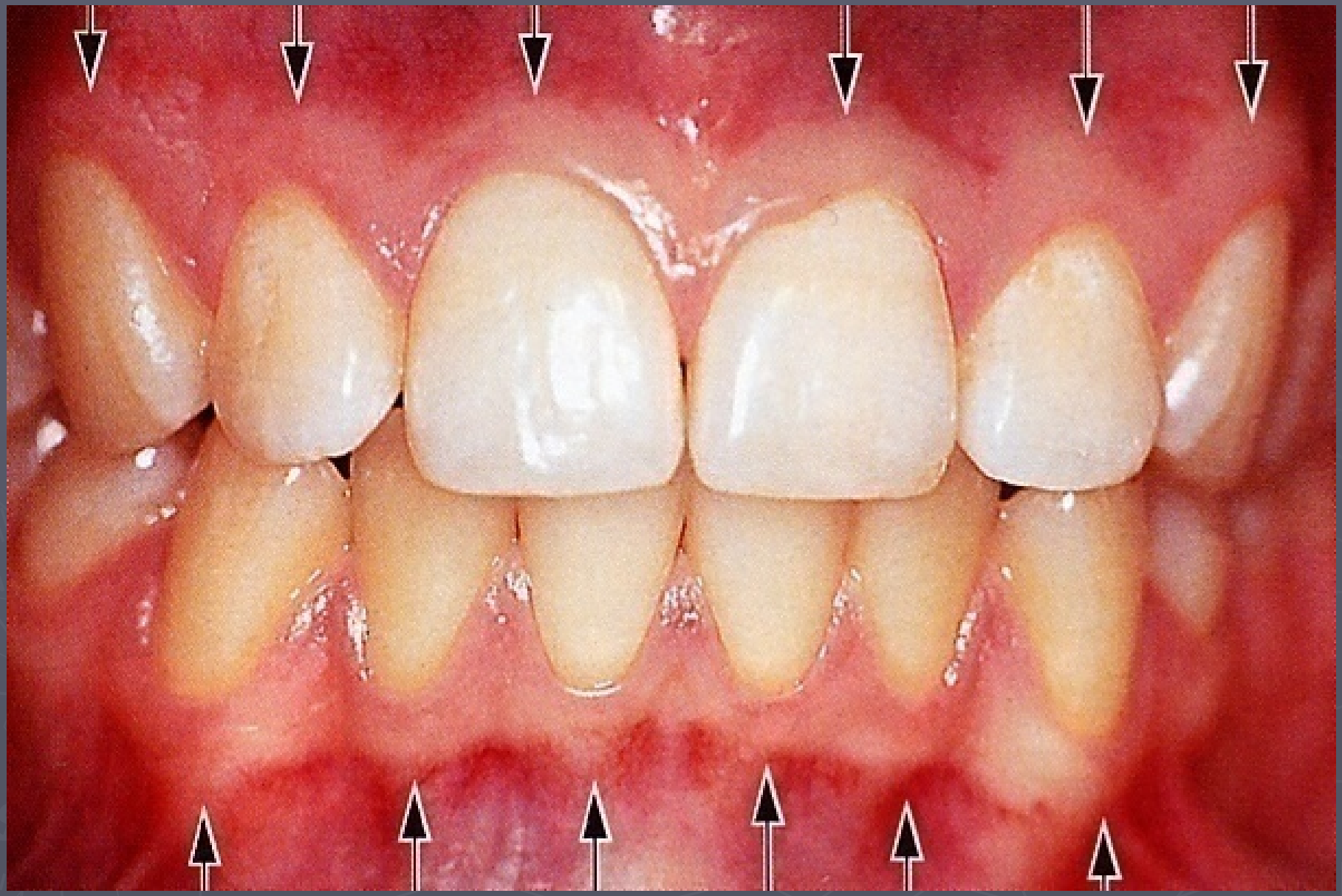
Invloed van medicatie op het parodontium

6 Vragenkwartiertje

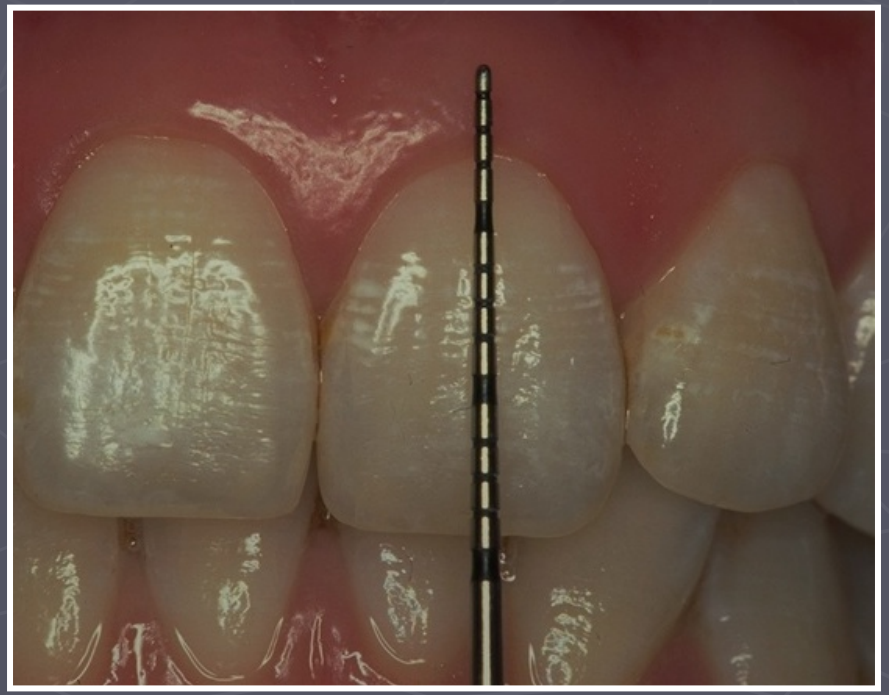
Oral mucosa

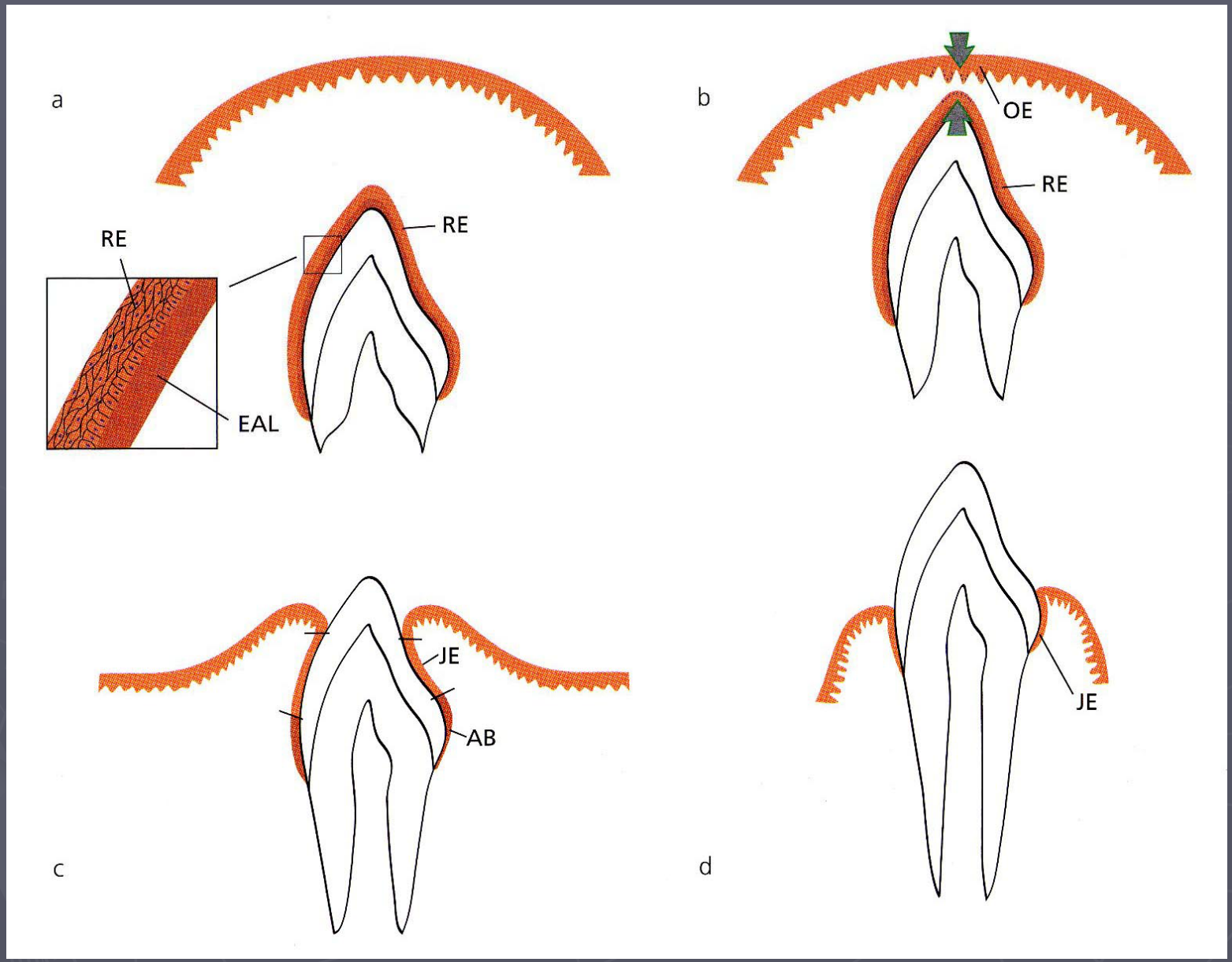
3 types

- masticatory mucosa : gingiva
  hard palate
- specialized mucosa : dorsum tongue
- lining mucosa : remainig part between skin of the lips and the soft palate and pharynx



papil

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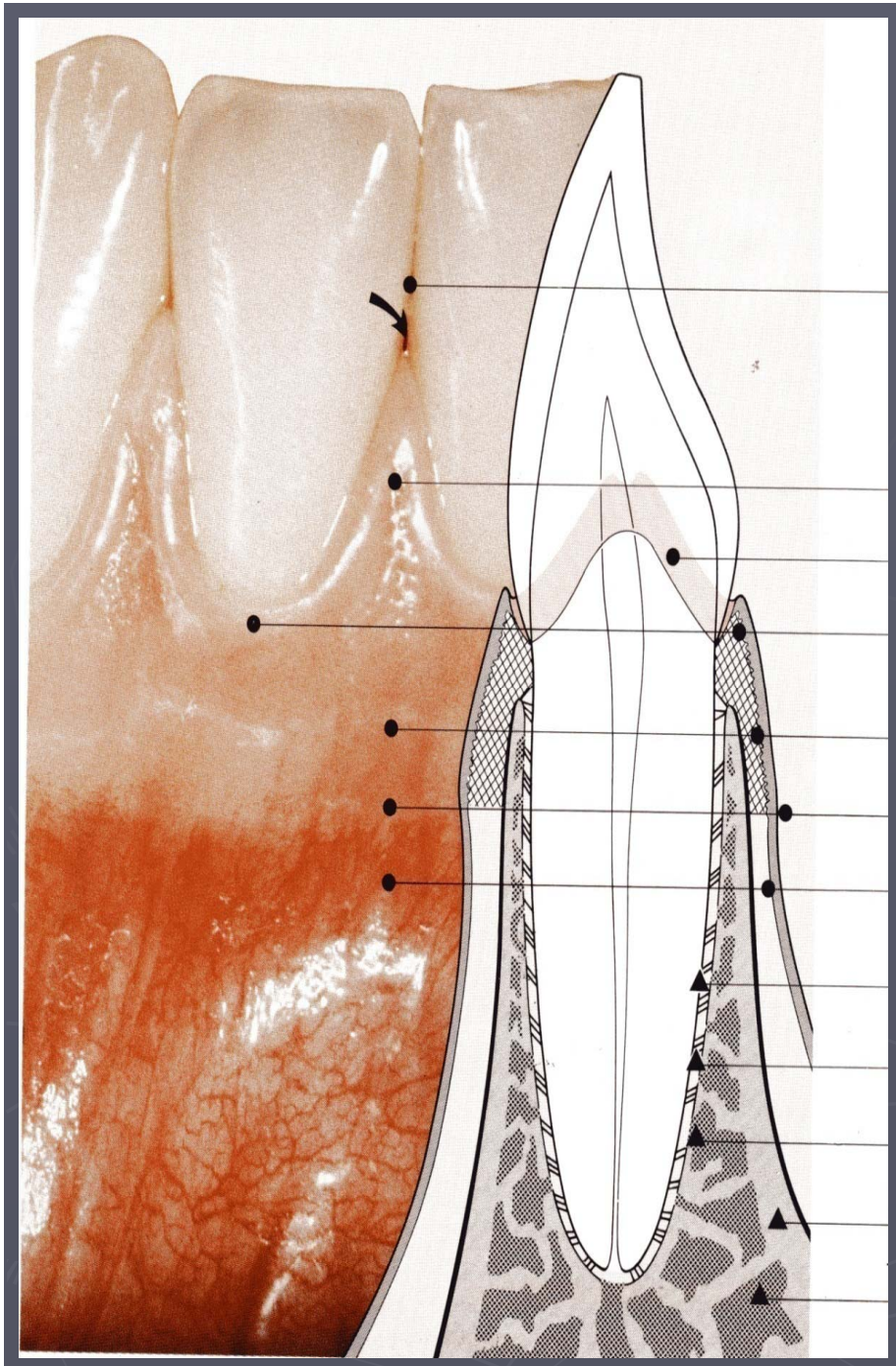


1 Contactpunt

2 Papil

3 Glazuur-cementgrens (tandhals)

4 Marginale gingiva of gingivarand

5 Aangehechte gingiva

6 Mucogingivale grens

7 Alveolaire mucosa

8 Wortelcement

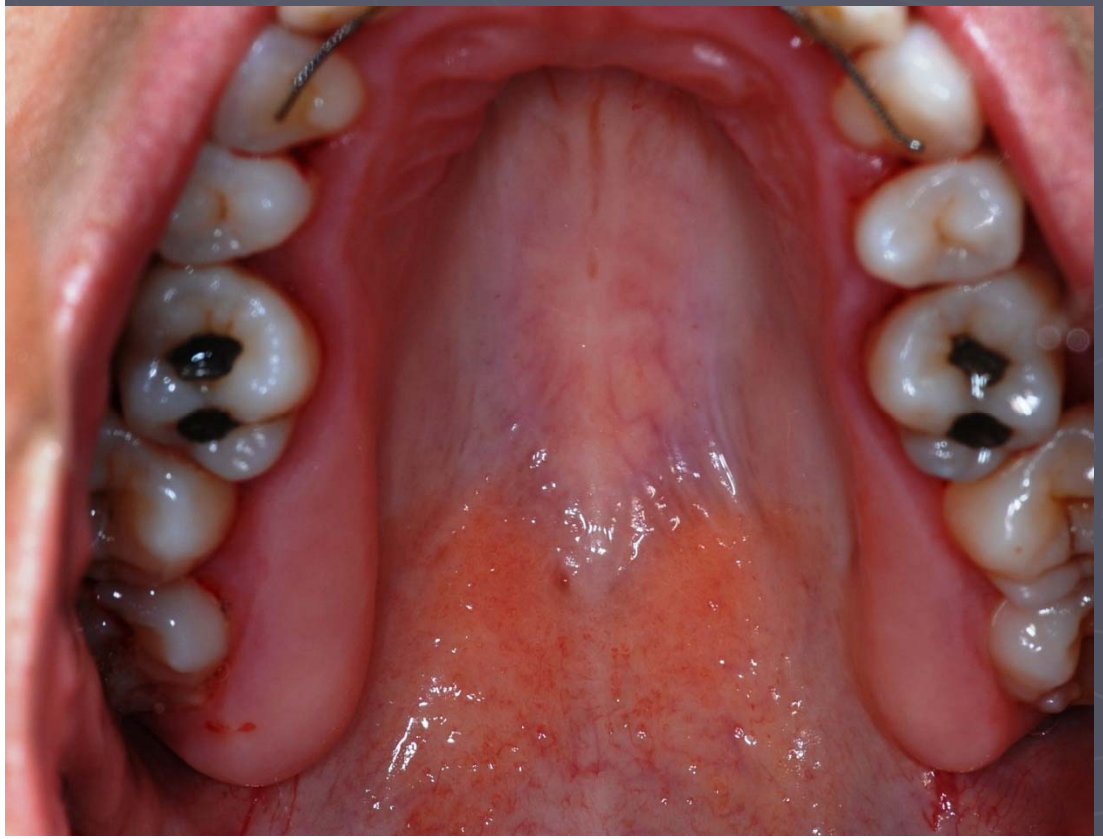
9 Parodontaal ligament

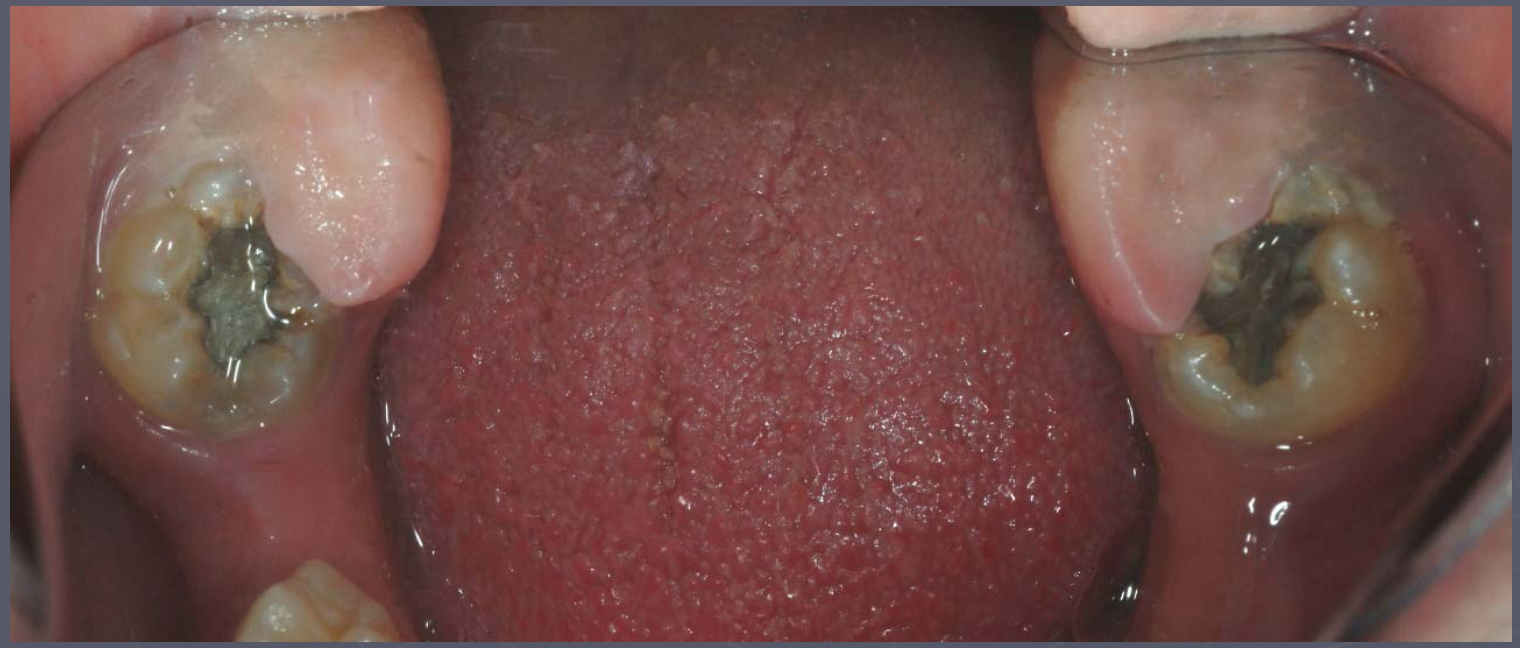
10 Alveolair bot

11 Corticaal bot

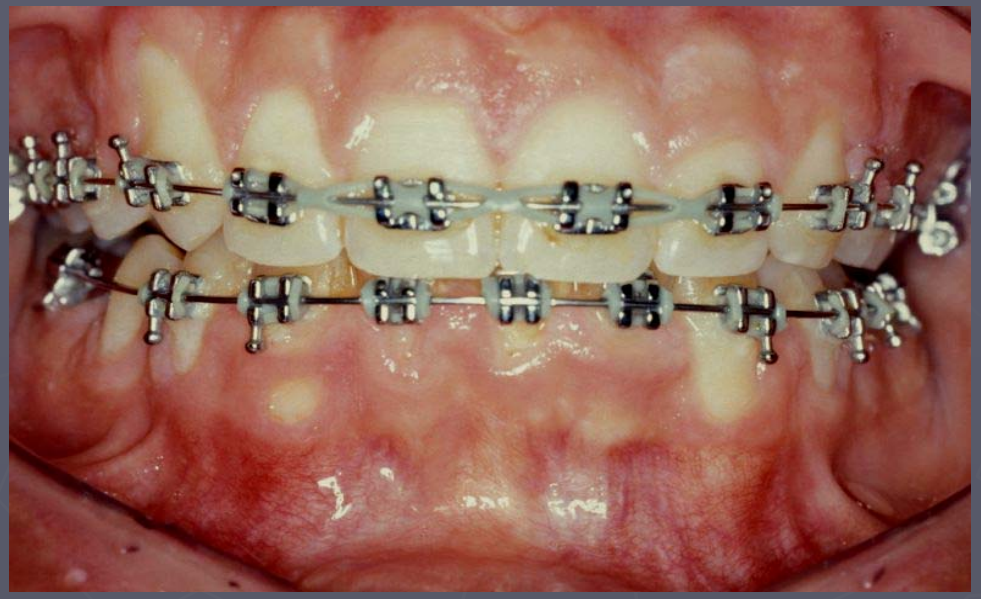
12 Basaal bot (medula)

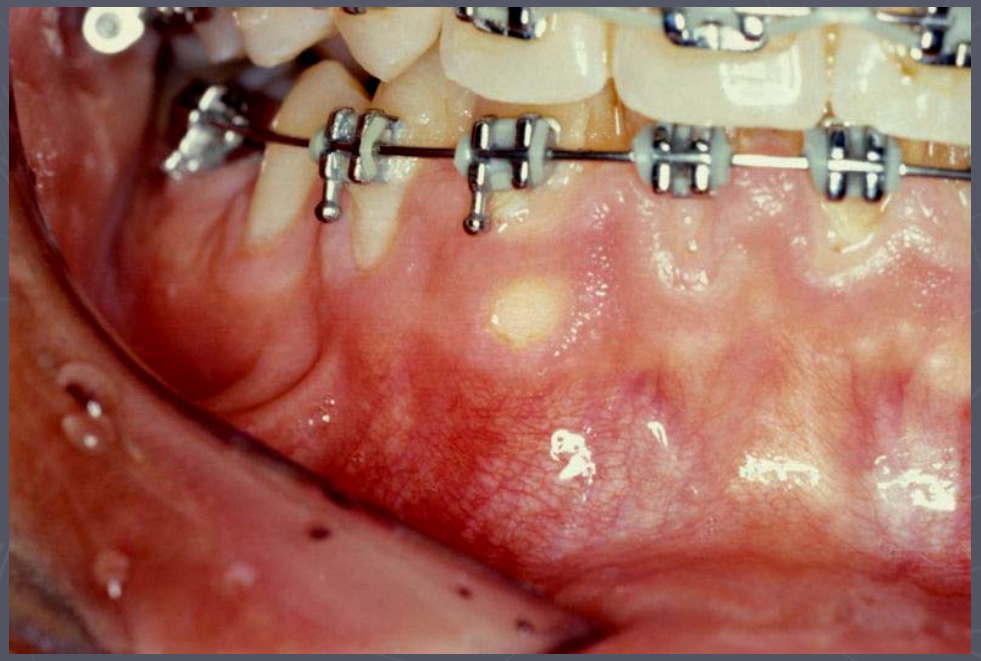
Hereditaire hyperplasie

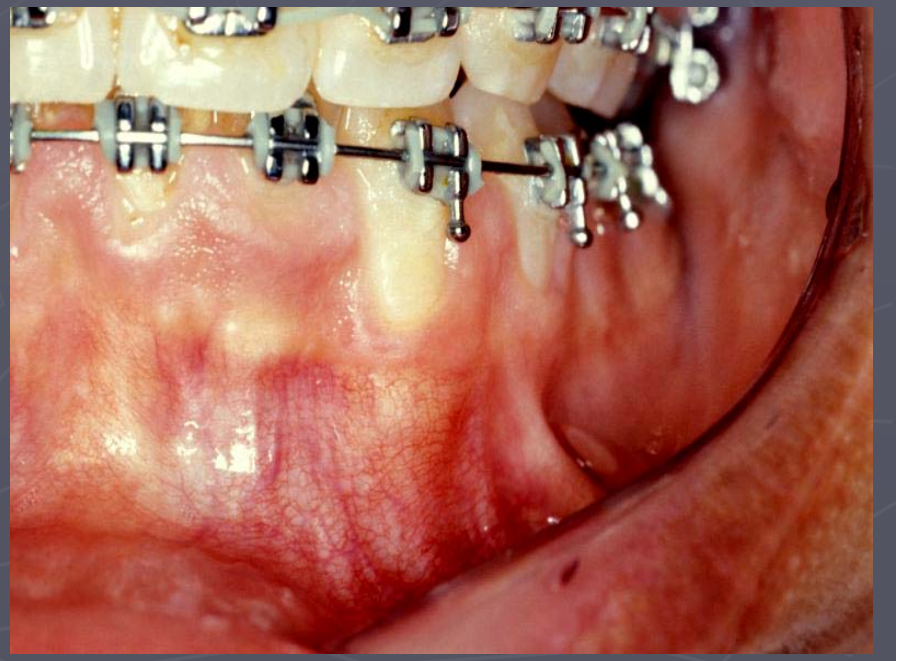




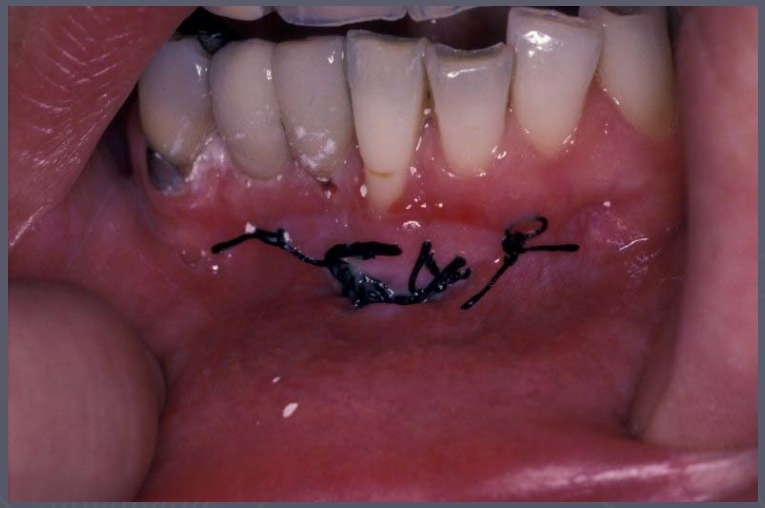
gingivarecessie







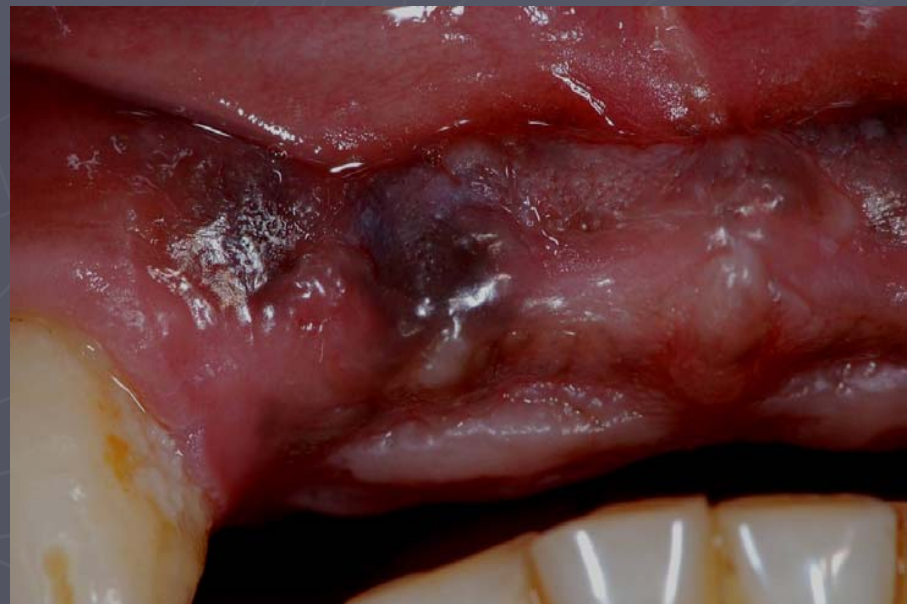
Vrij gingivatransplantaat



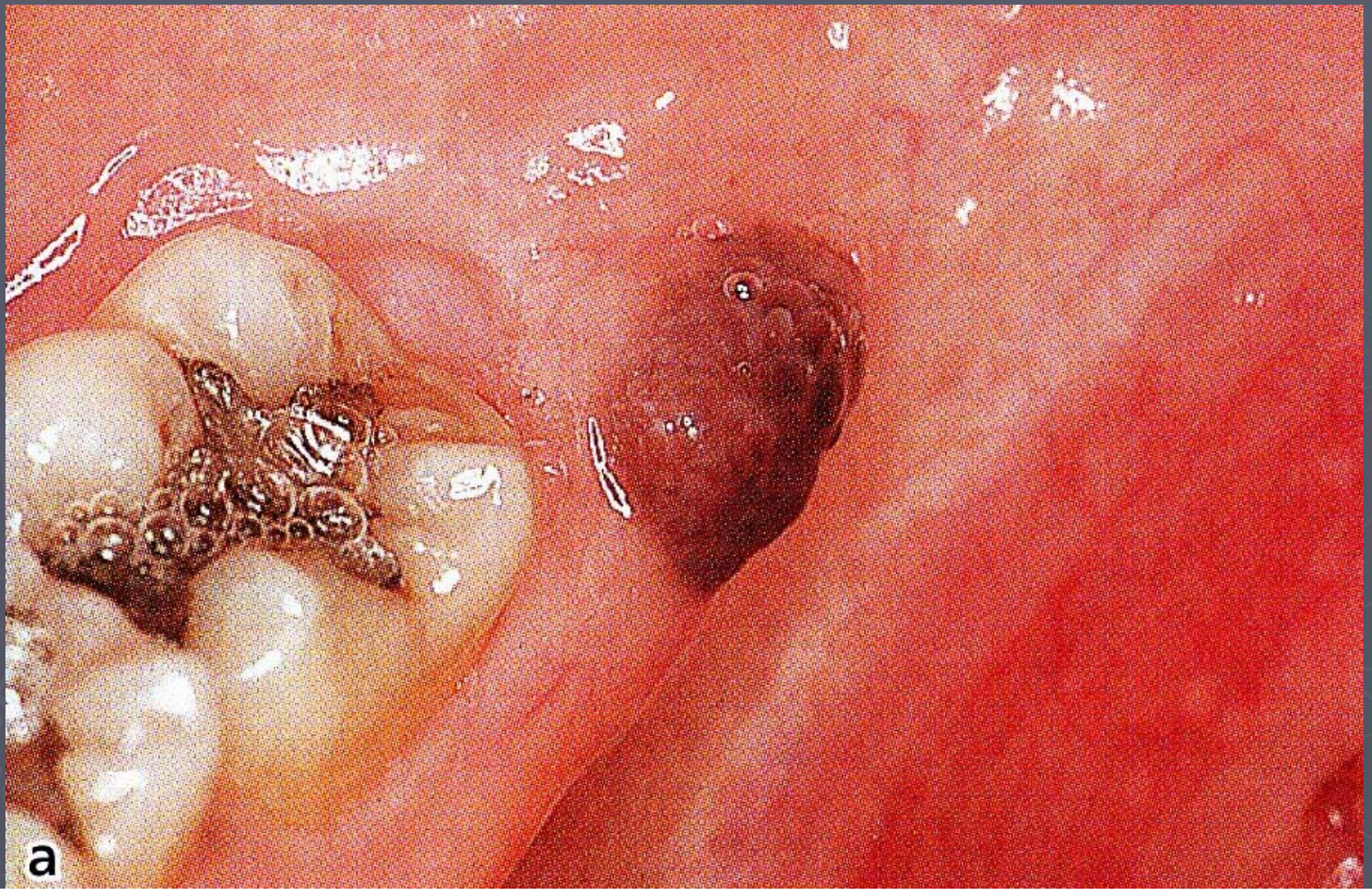
Hairy tongue

Lingua geographica

Raciale pigmentatie



nevus



pigmentations

Benign :

racial pigmentation

ephelides

smoking associated

intraoral nevi

Malignant :

melanoma

Endogenous etiology

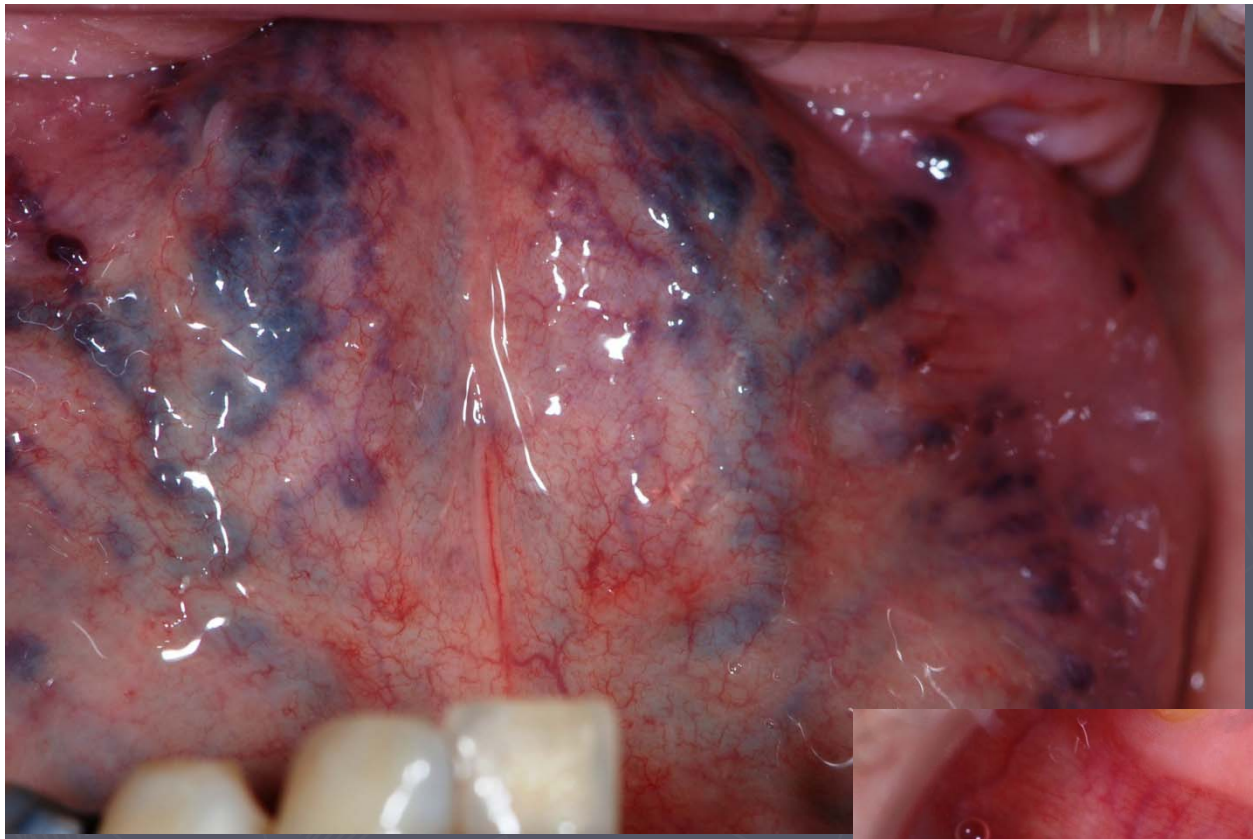
Addissons disease

Peutz-Jegers

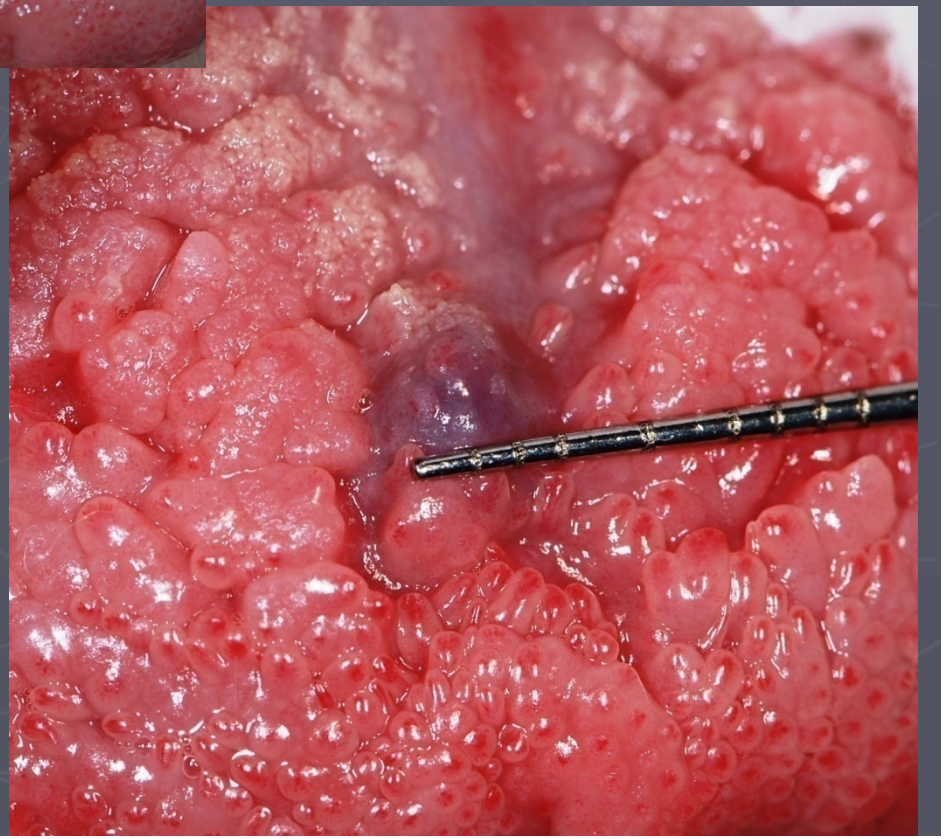
Exogenous etiology

amalgam tattoo

bismuth, lead



varices



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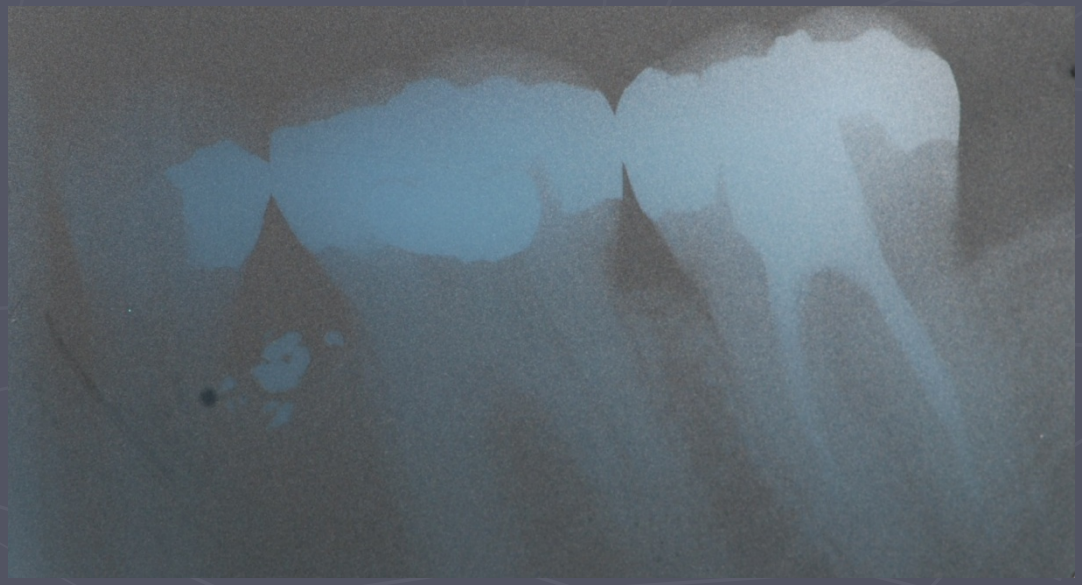
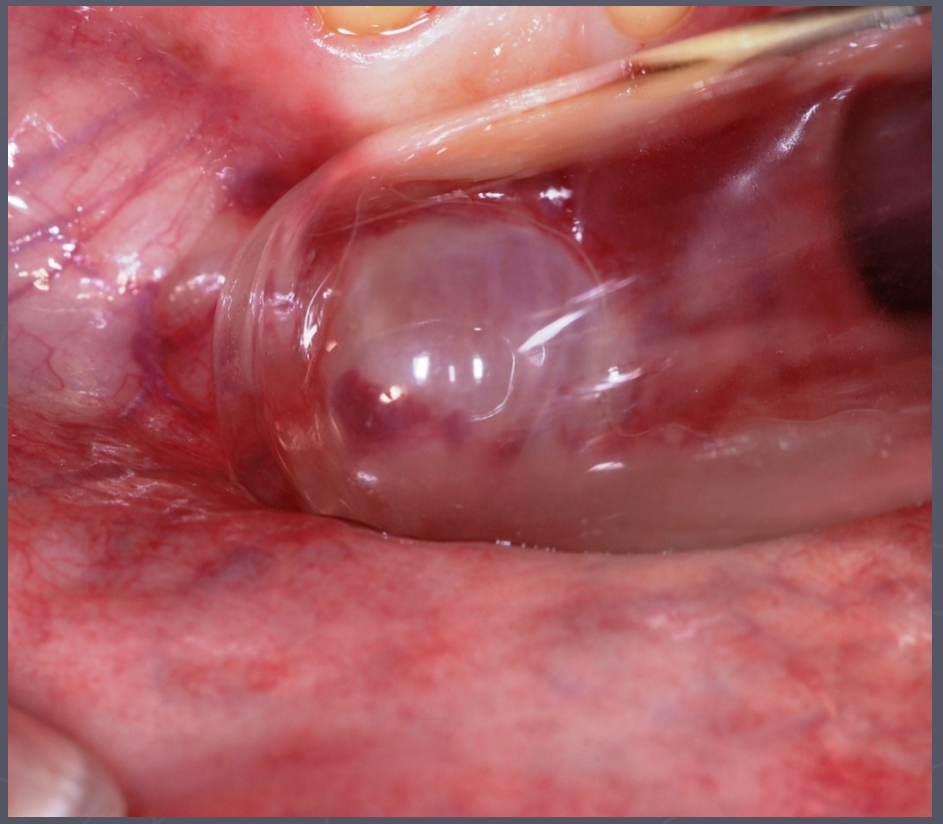




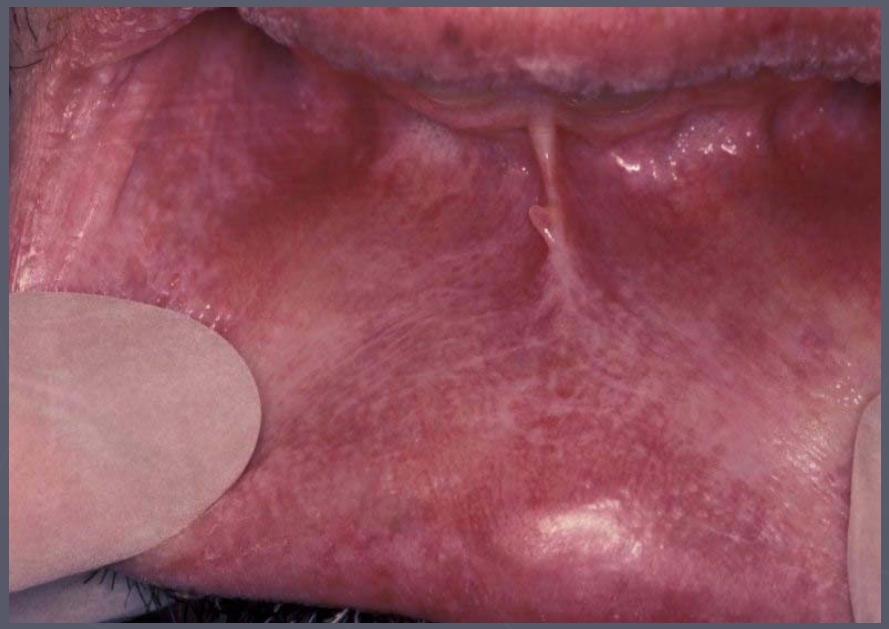
Fordyce spots

Linea alba

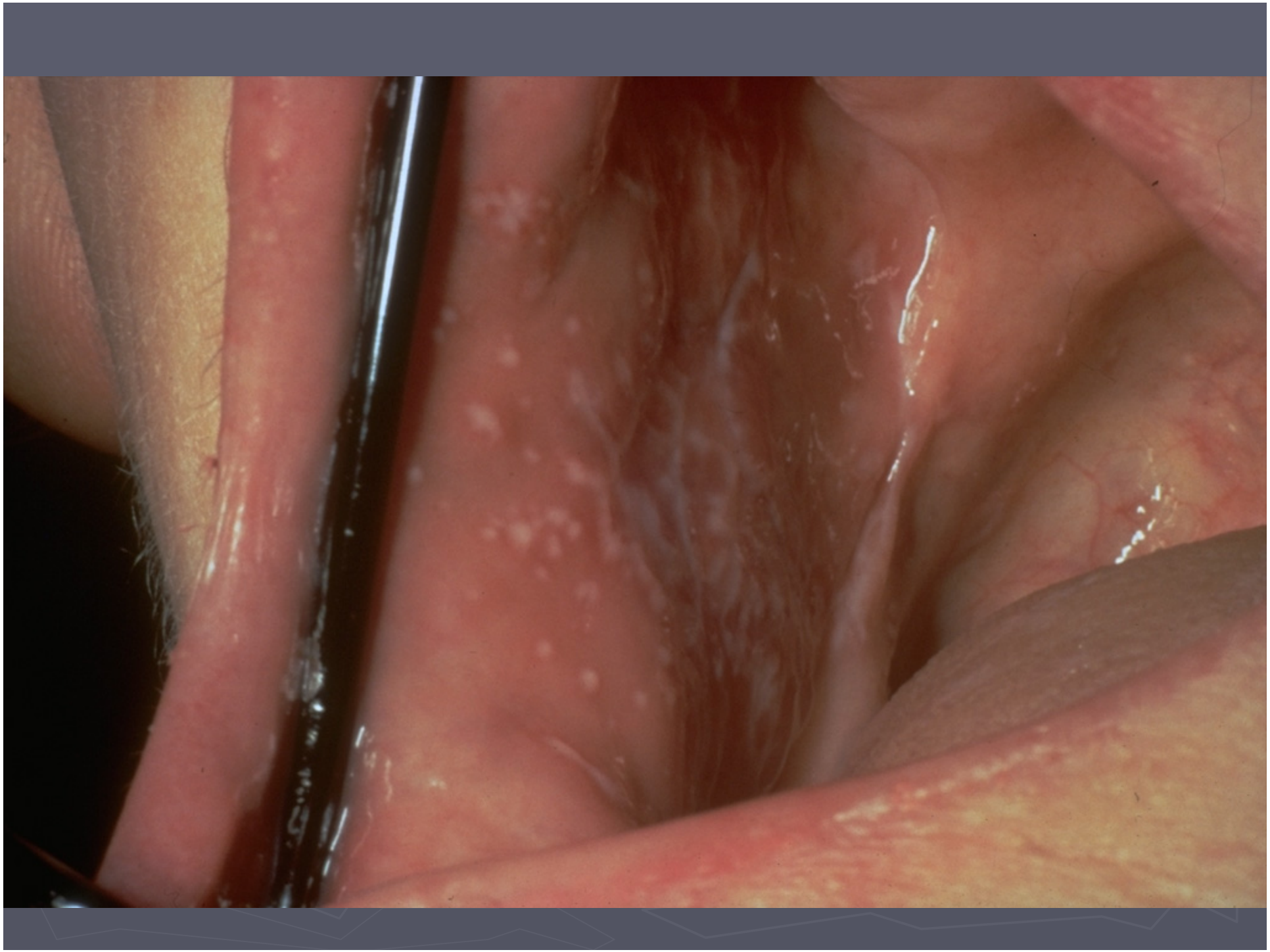
morsicatio

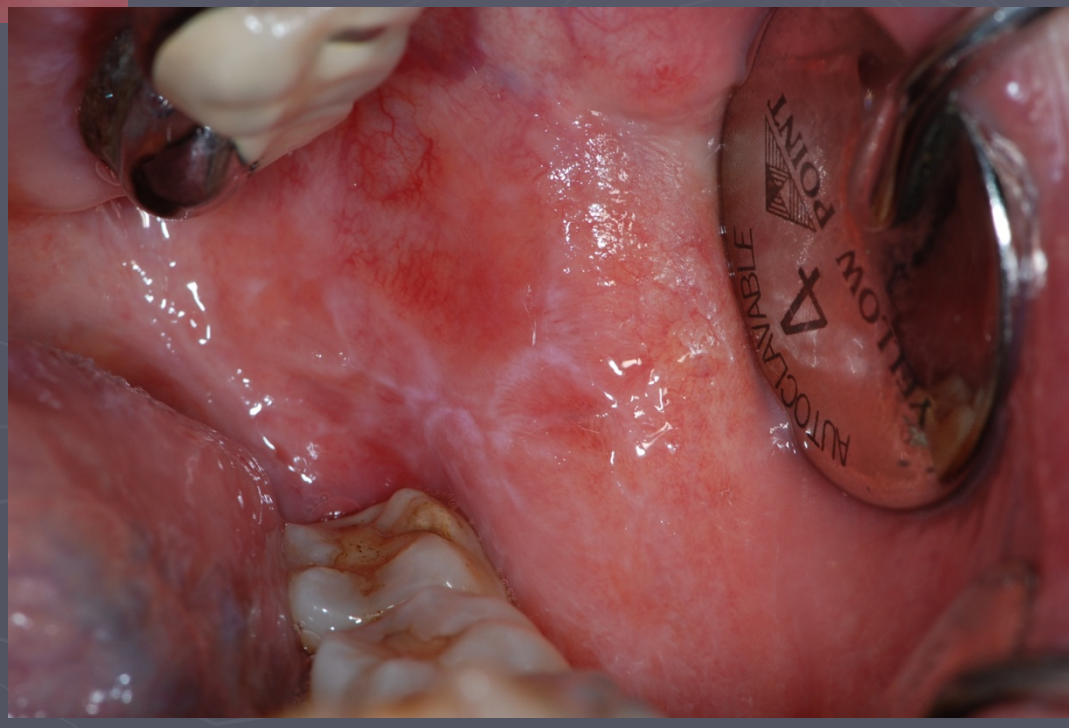
morsicatio


93 3 28



Lichen (ruber) planus

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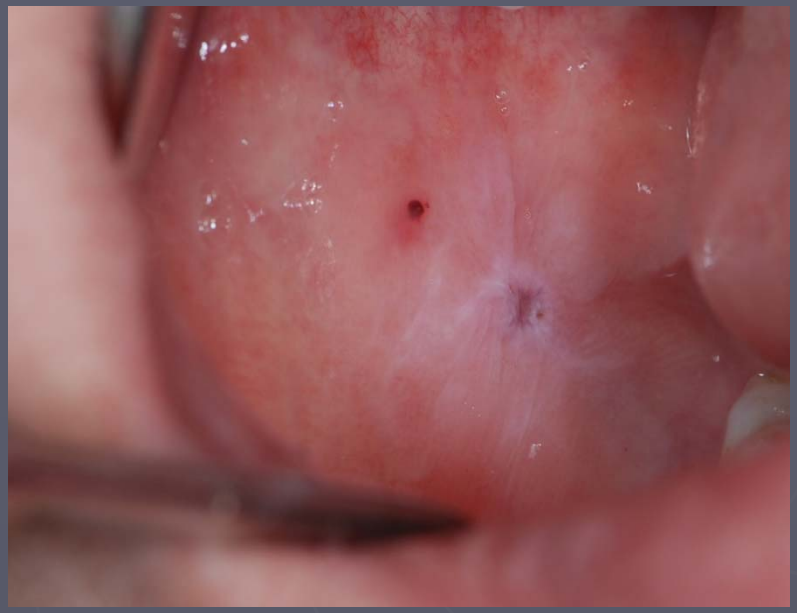
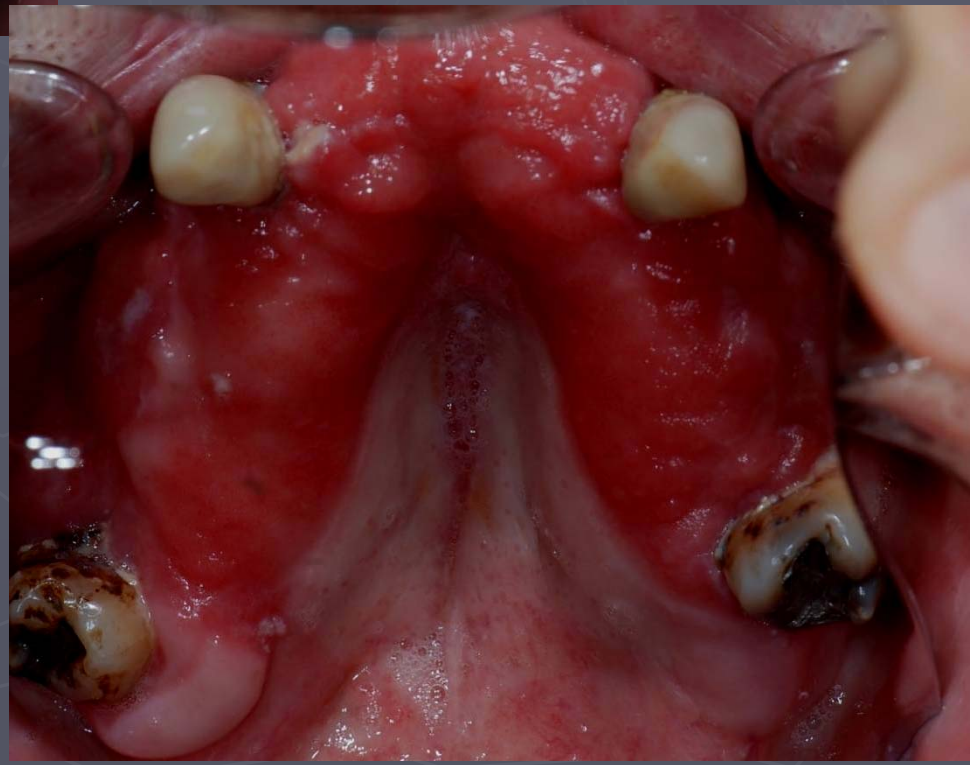
POINT

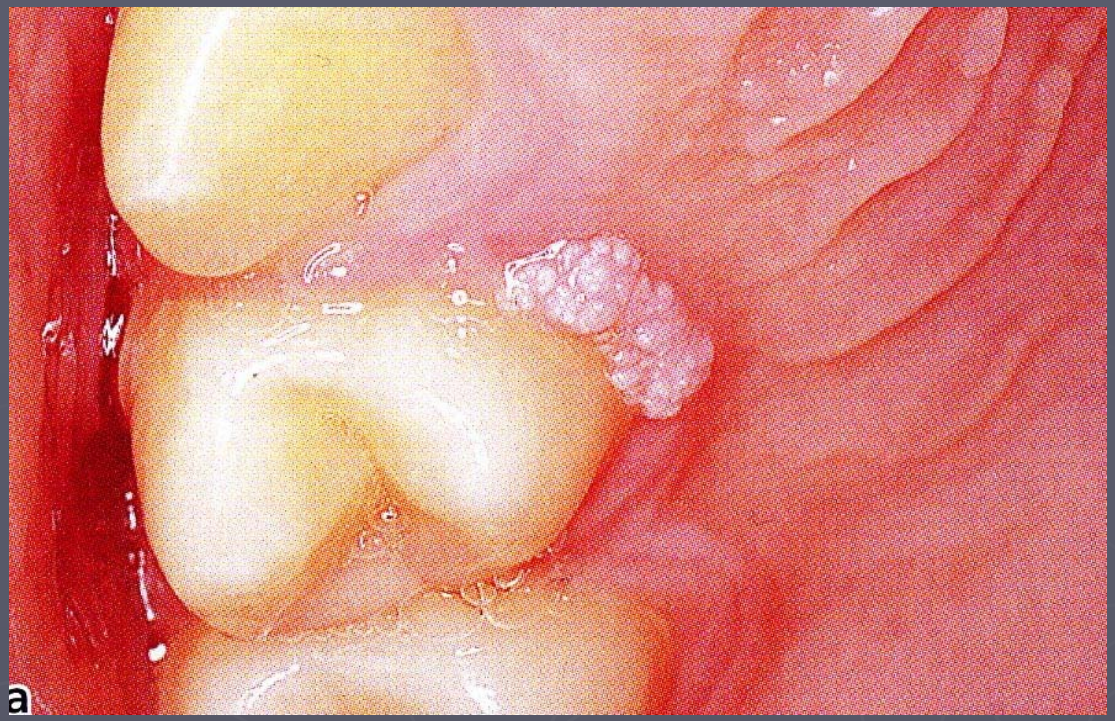
Lichen planus

- 0,1 – 4 % of population
- All ages, very seldom in children
- Mostly only intraoral lesions
- Different types : papular
  reticular
  plaque-like
  atrophic erosive
- Both gingiva and mucosa can be affected
- Precancerous 0,5 -2 %





Cheilitis angularis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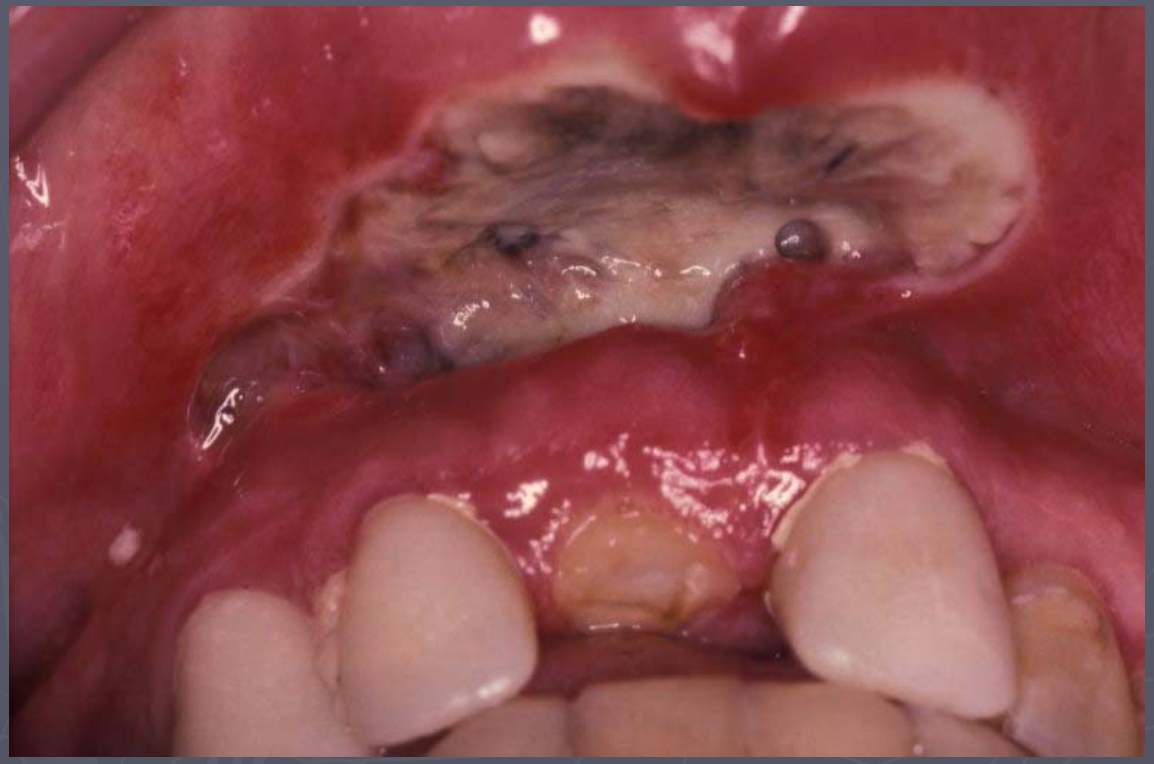
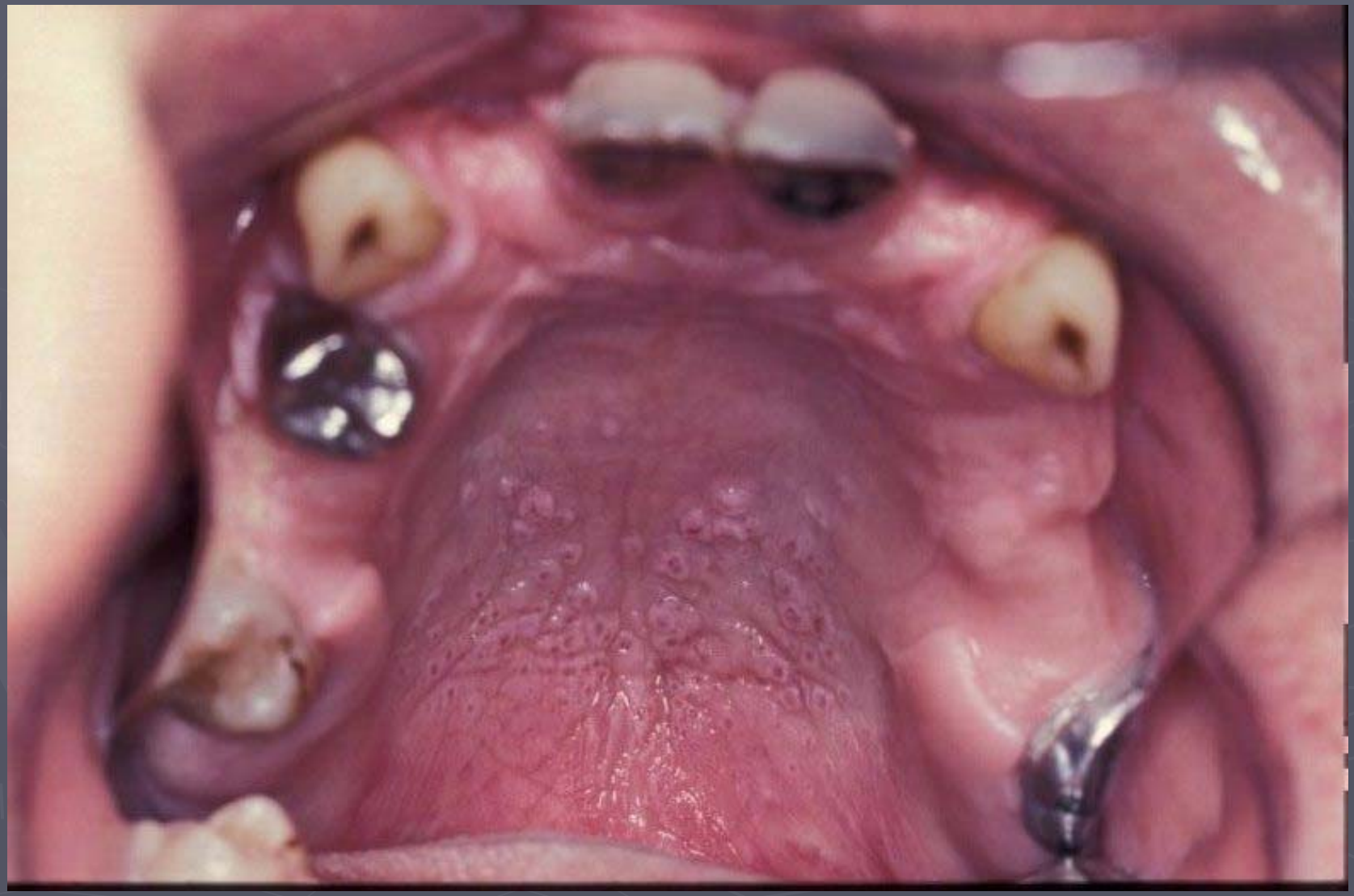
Primaire herpes

leucoplakie



Smokers palate

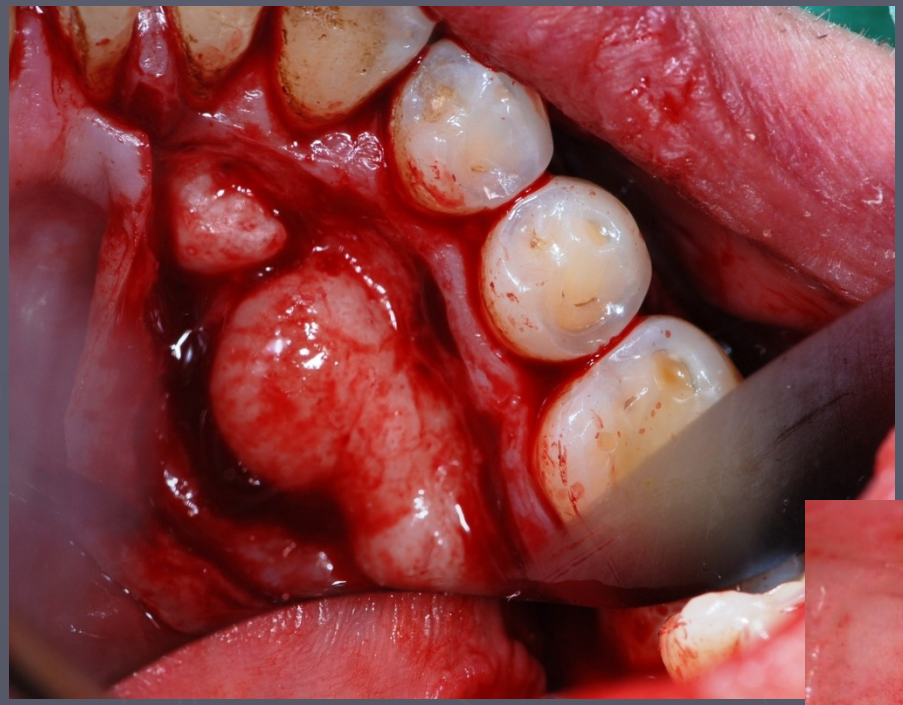


Mucosal lesions

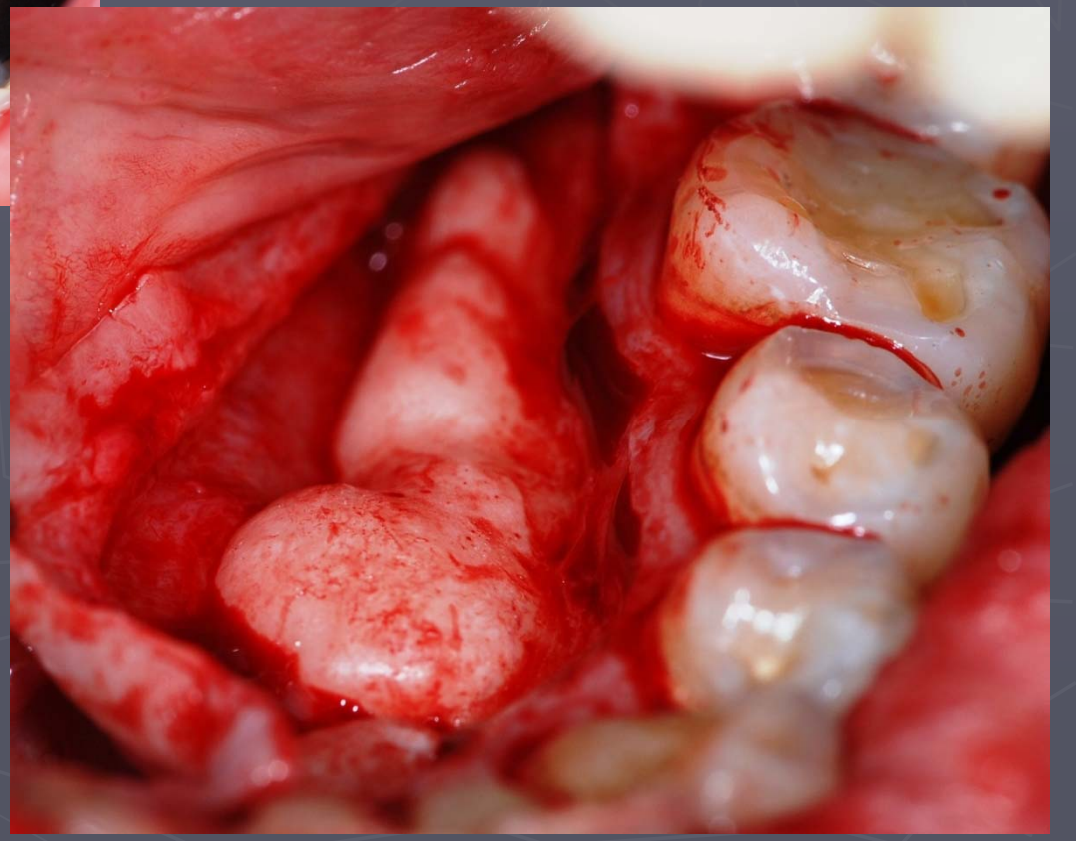
- Viral origine : herpes
- Fungal origin : candida
- Genetic origin : hereditary hyperplasia
- Systemic origin : lichen planus
  benign pemphigoid
  pemphigus vulgaris
  erythema multiforme
  lupus erythematosus
  allergic reactions

Mucosal lesions : origin

- Gastro-intestinal diseases : crohn's disease
- Hematological : leukemia
- Traumatic lesions : chemical injury
  physical injury
  thermal injury

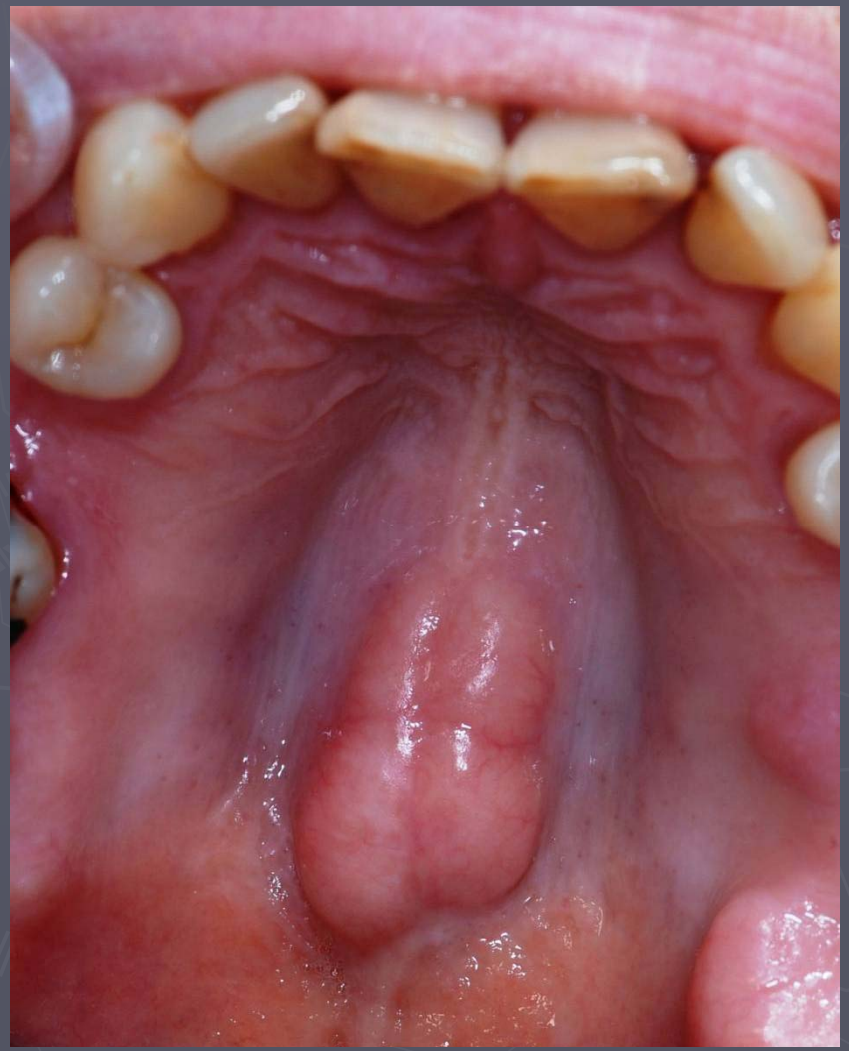


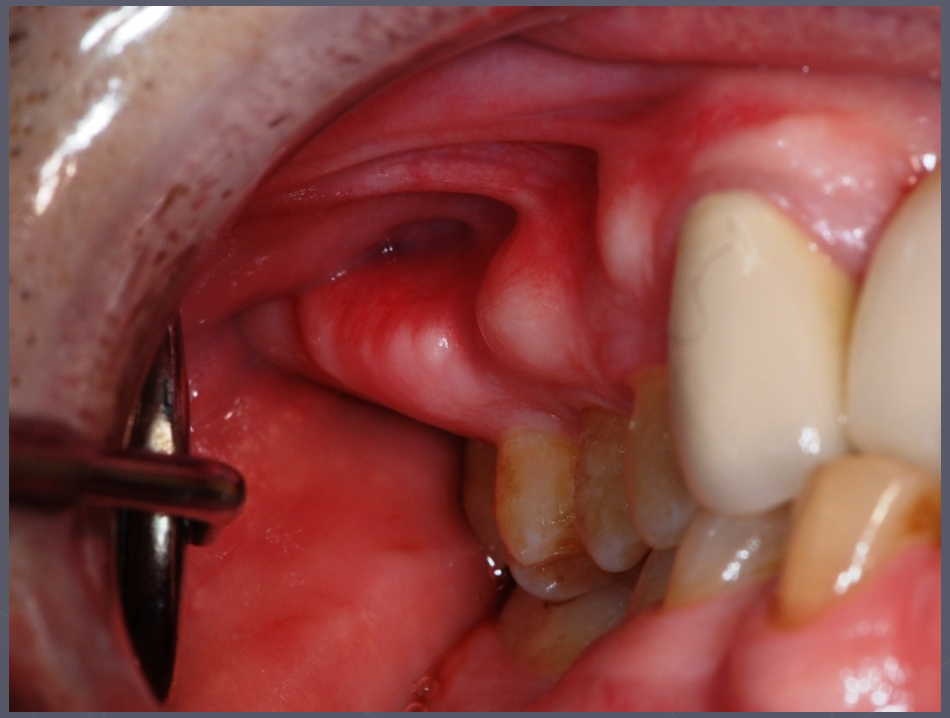
Torus lingualis



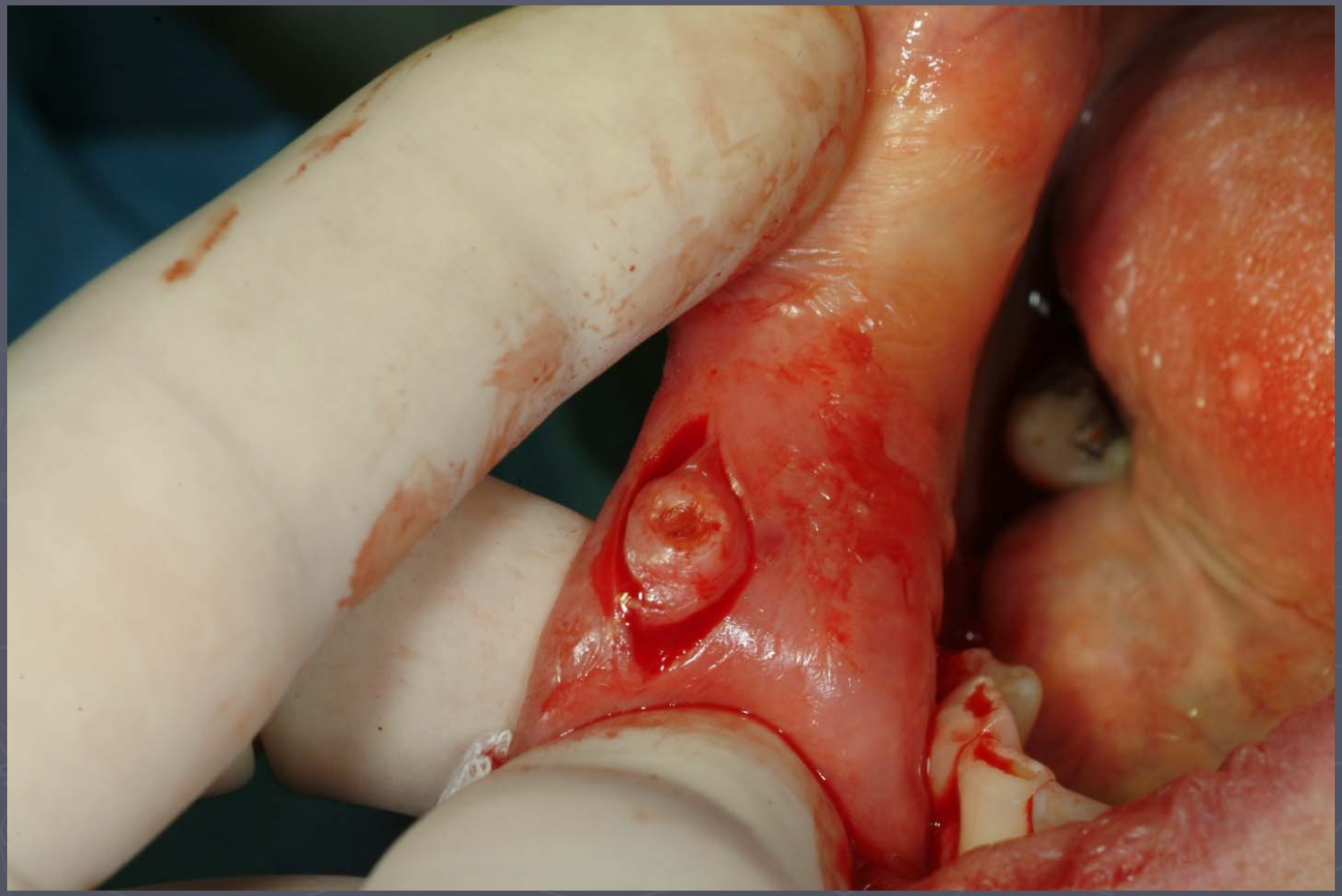
Torus palatinus

Torus palatinus

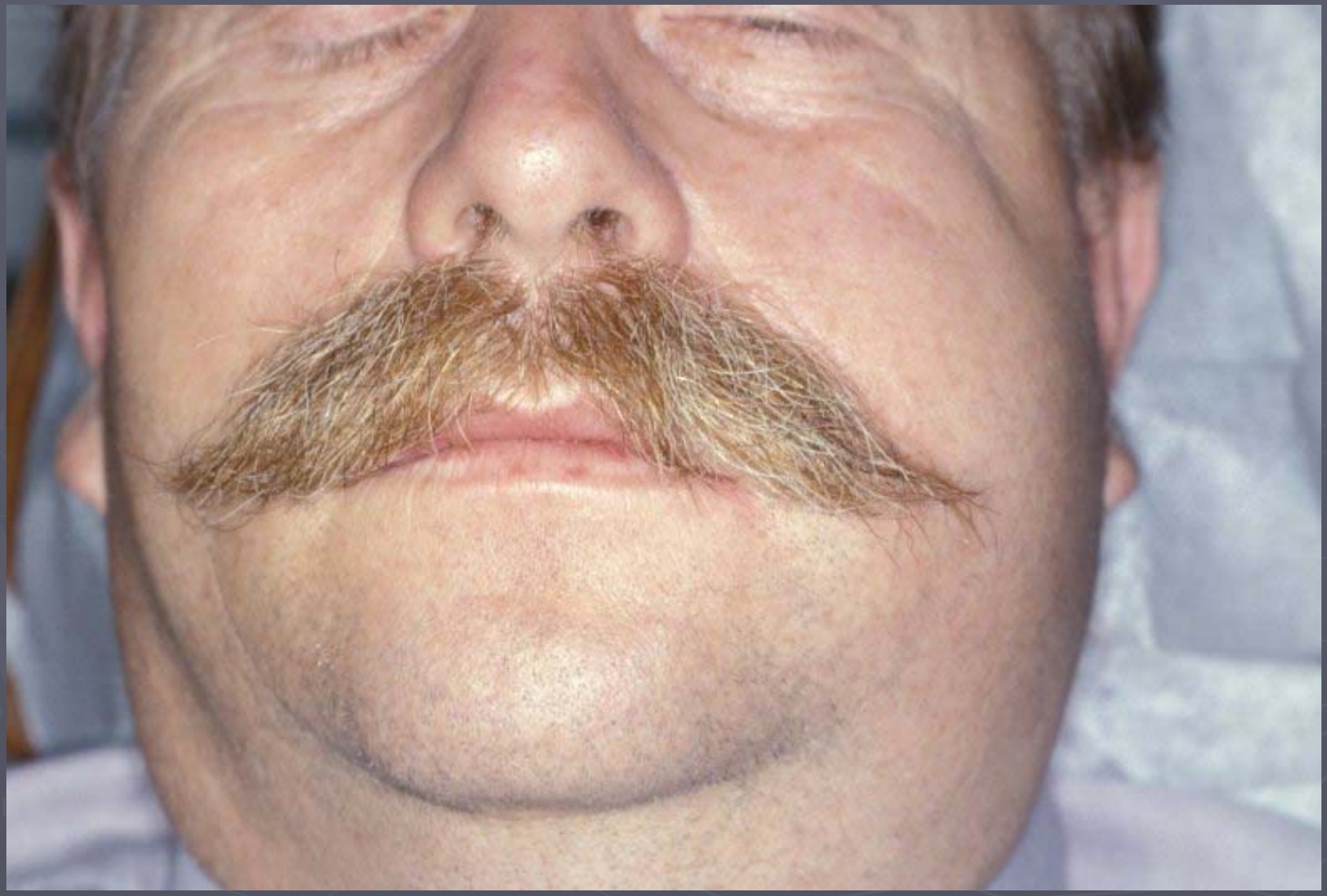


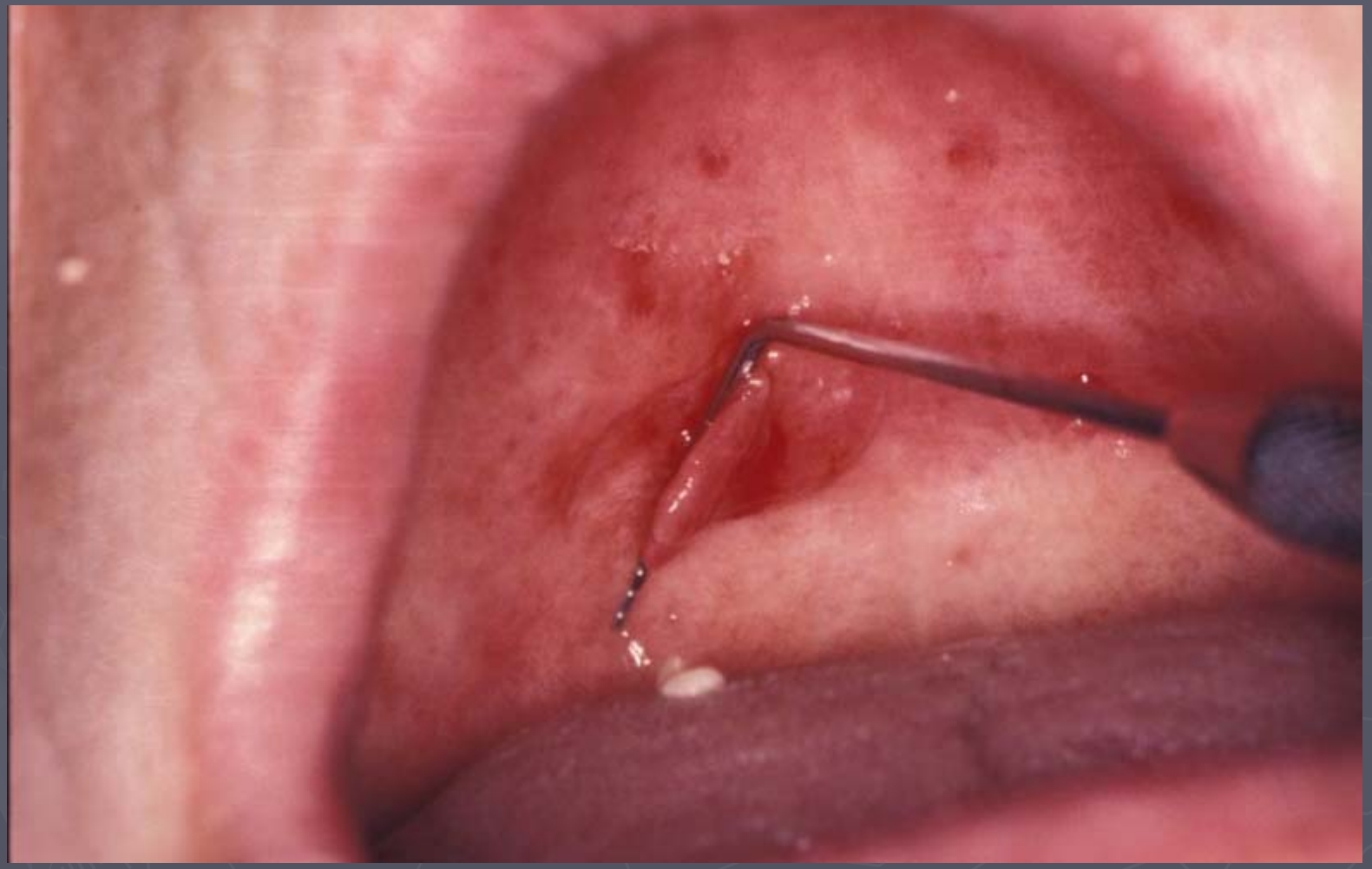


exostose

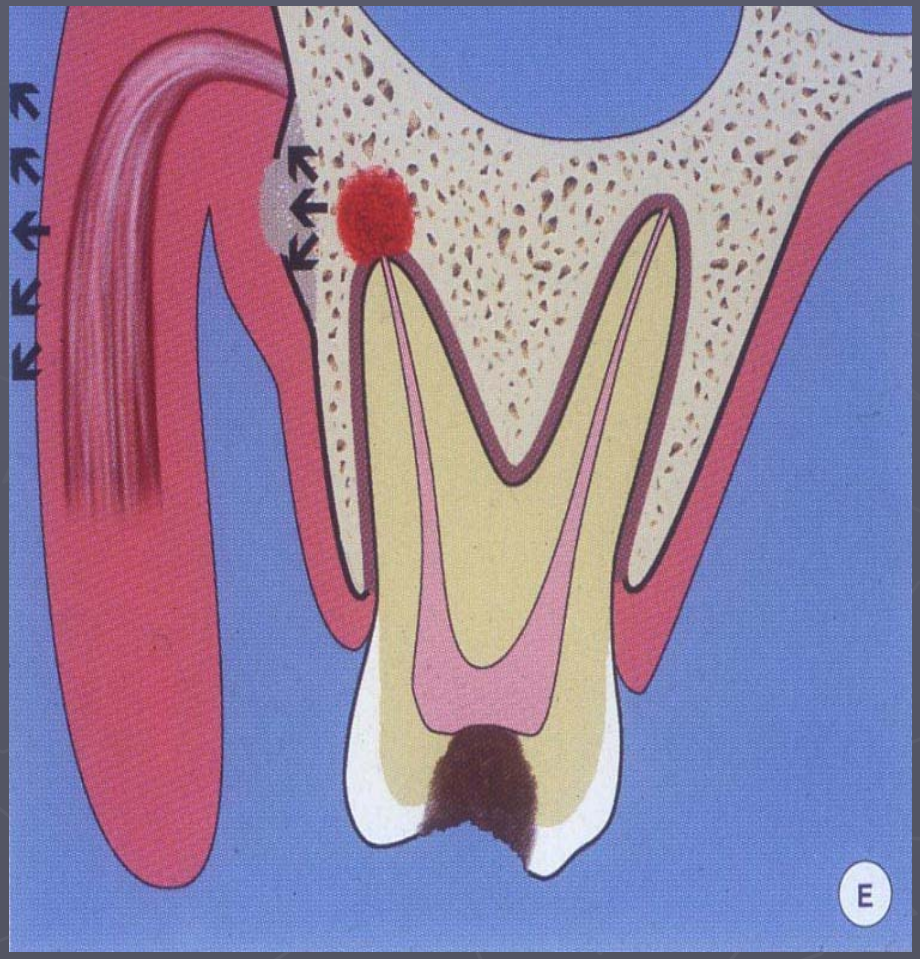


Parotiszwelling na spoelen CHX





abces



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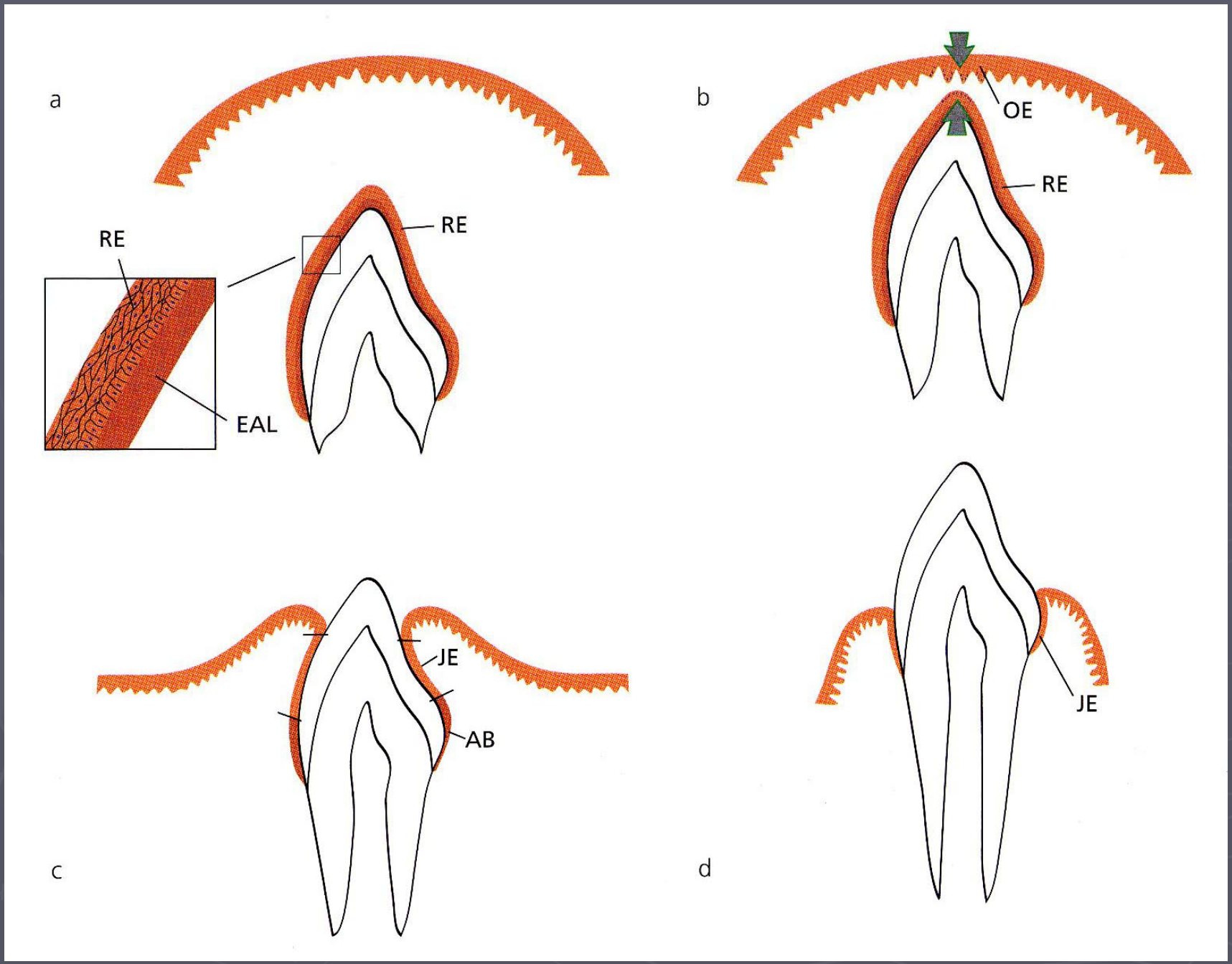
Periodontal infections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gingivitis


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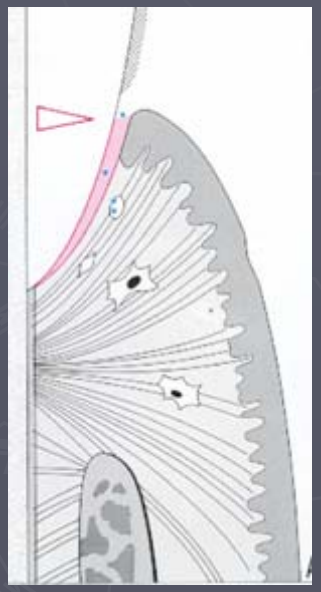
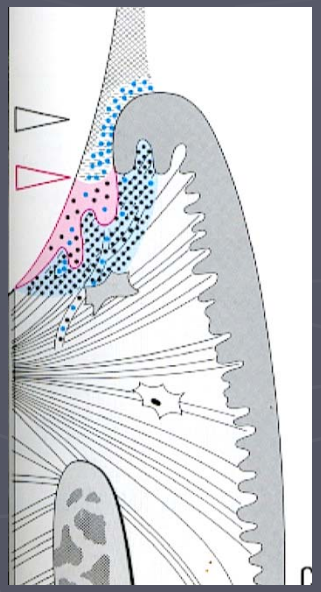
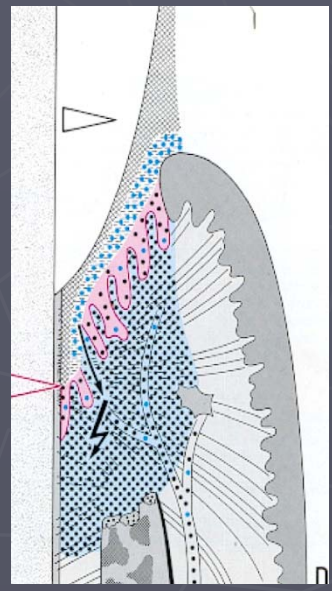
Etiologie

Pocketvorming

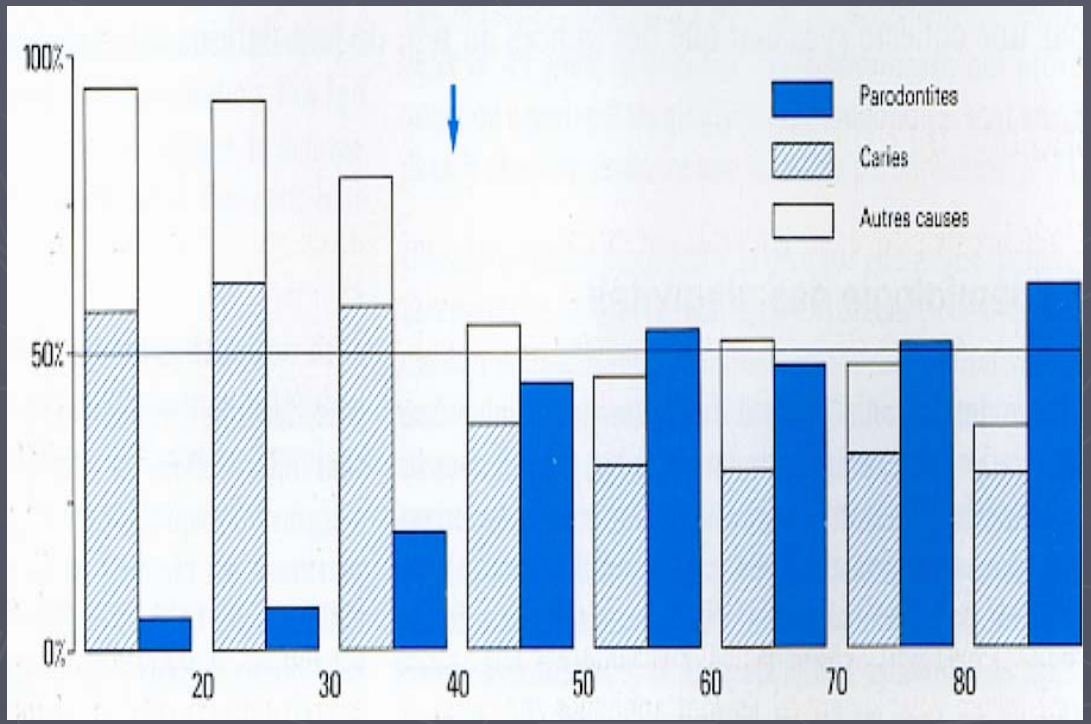
1. Onstekingsinfiltraat aan de marginale gingivarand
2. Afbraak van collageen weefsel
3. Aanhechtingsepitheel migreert apikaalwaarts
4. Botresorptie







Plaque - calculus



1977:

Na de leeftijd van 40 jaar is parodontitis de hoofdoorzaak van tandverlies

2005 :

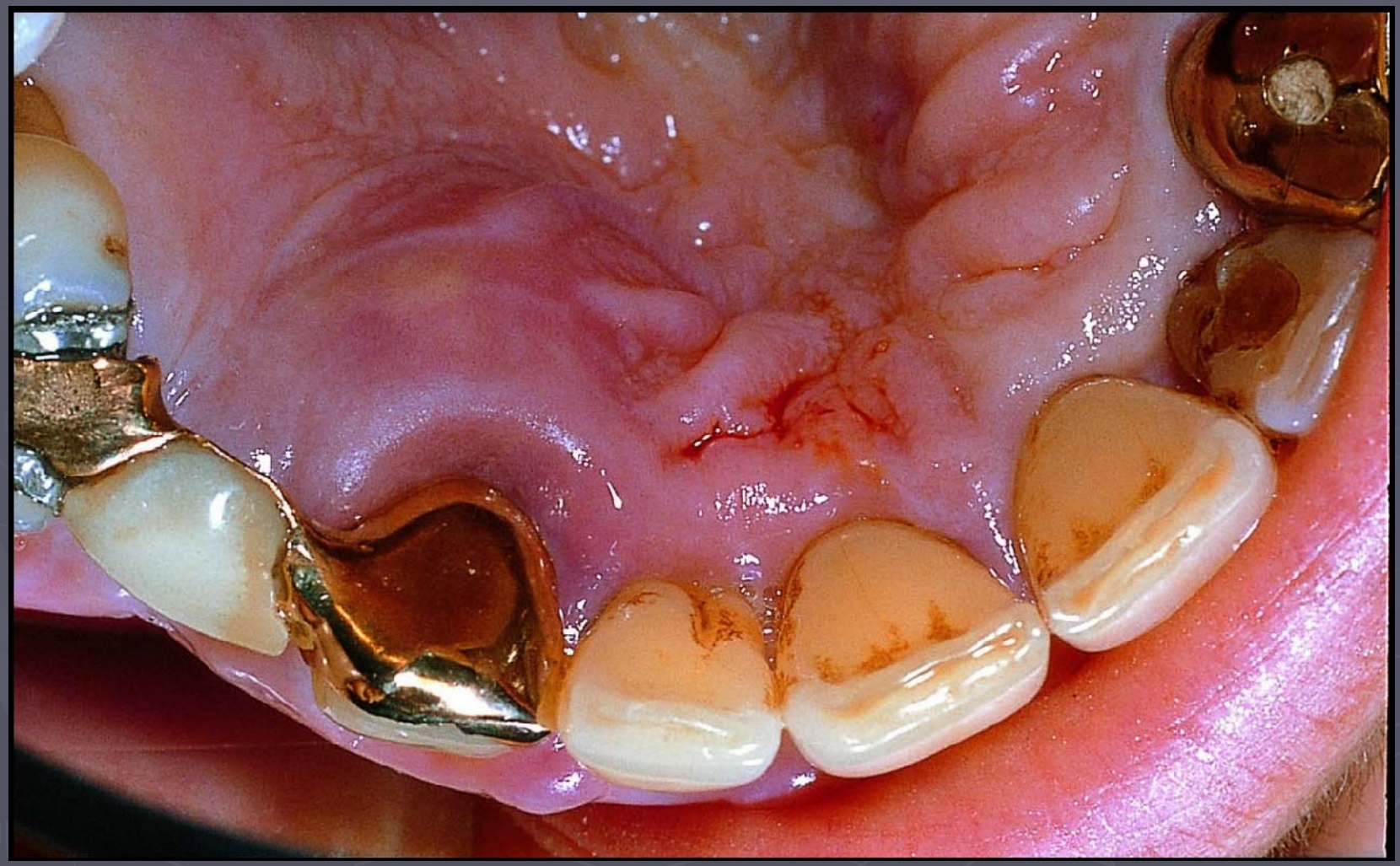
Parodontale aandoeningen vormen belangrijkste groep van orale / dentale pathologie

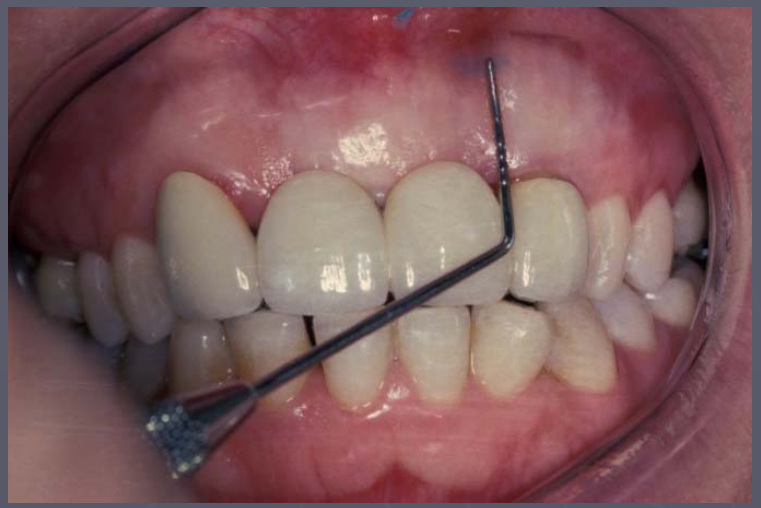
Curilovic, *1977.*

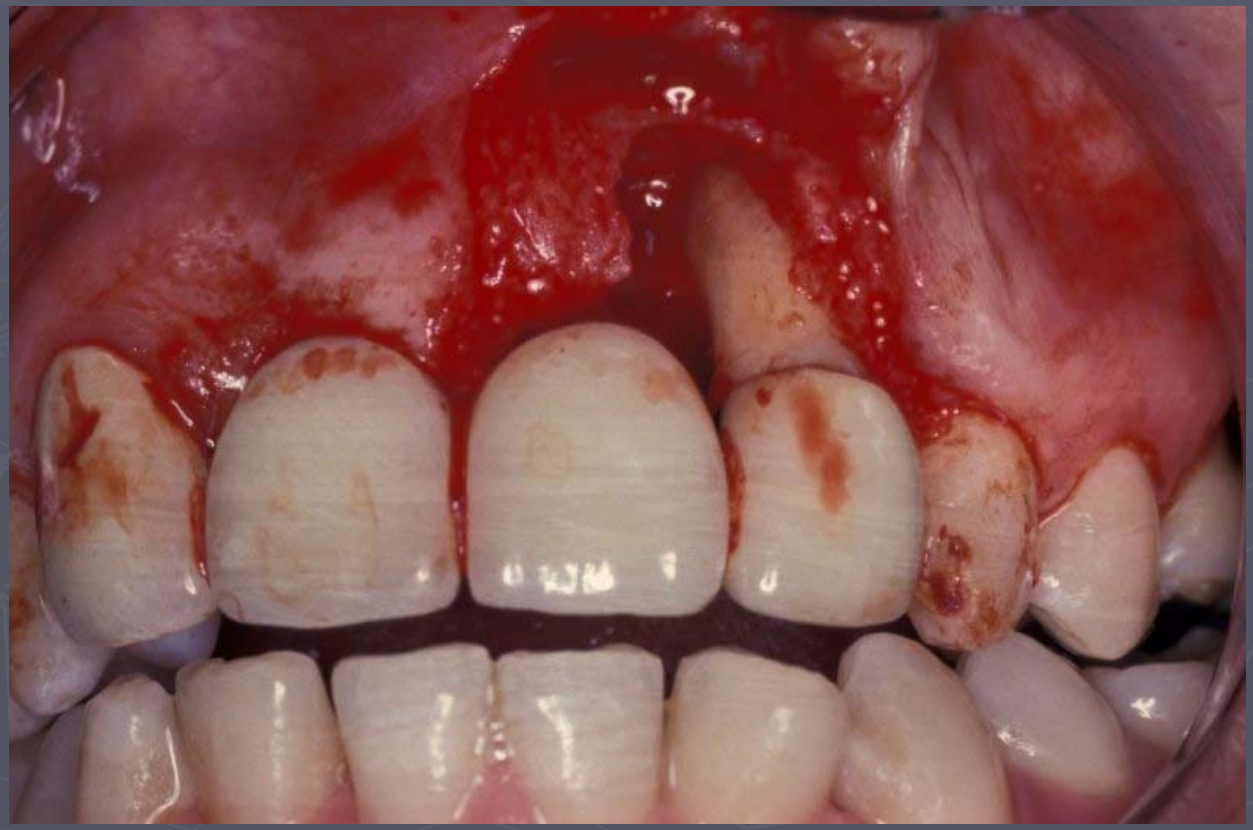
Plaque-induced periodontal diseases

- Periodontal abcess
- Chronic periodontitis
- Aggressive periodontitis
- Necrotizing periodontitis

Periodontal abcess

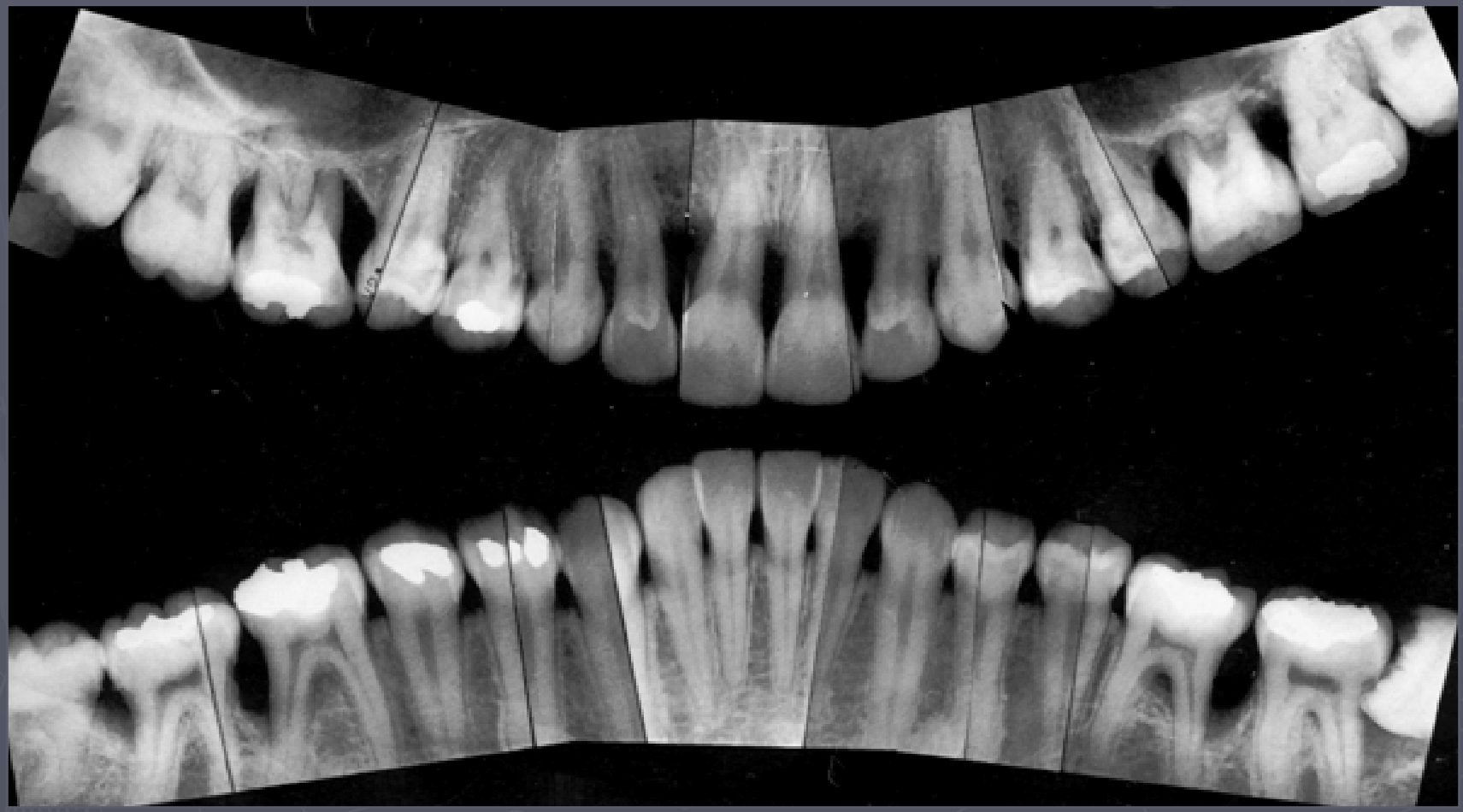






Parodontologie 2005
Belgische Vereniging voor Parodontologie

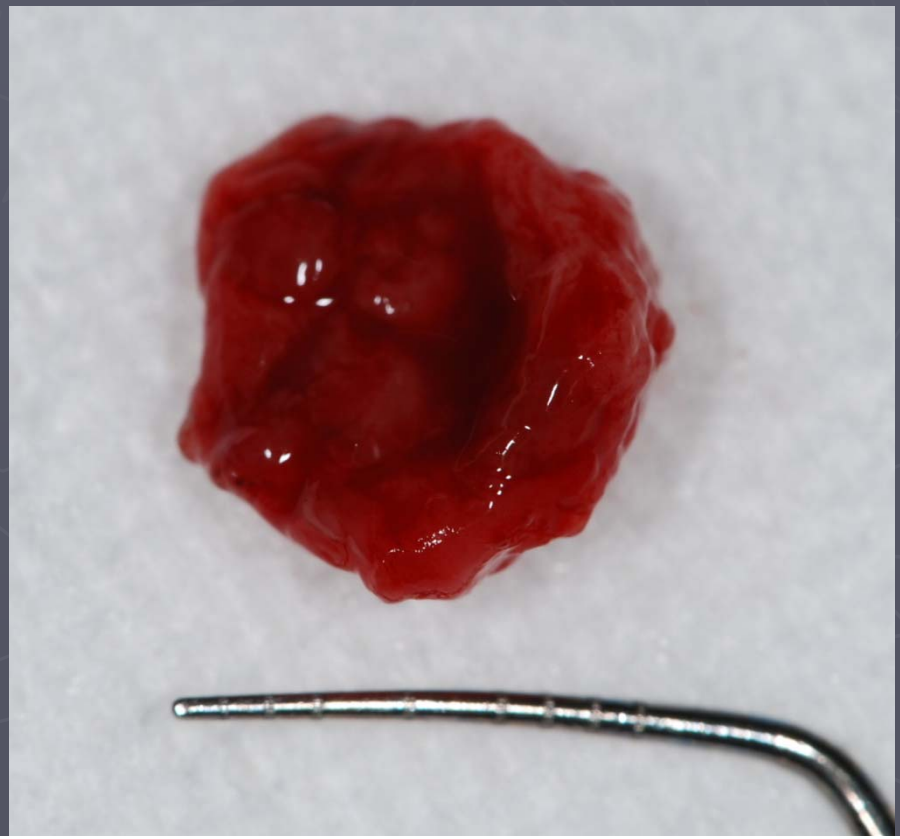
Parodontologie 2005
Belgische Vereniging voor Parodontologie



ulcererend letsel

- 30 à 50 cm² bij veralgemeende gevorderde parodontitis
- Gram – anaerobe infectie
- blootstelling aan permanente kauwkrachten

Dit ontstekingsletsel beslaat een oppervlakte van ongeveer 2,5 cm²



leukemia

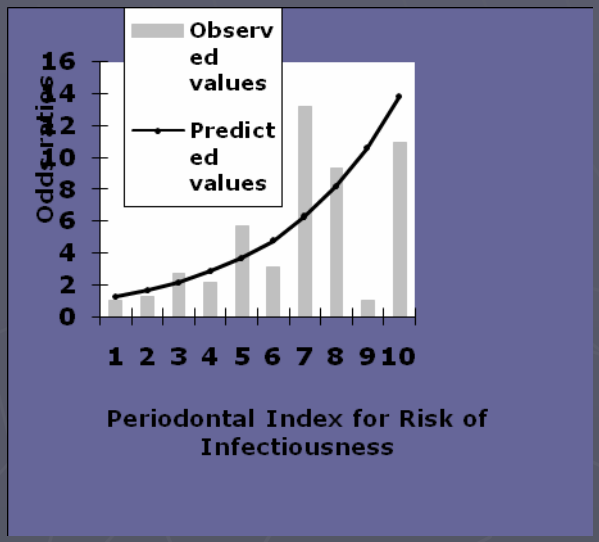
Bloedstaal (voorarm)a

- kauwen (chewing-gum) 50 x aan elke zijde
- 2de bloedstaal na 5 min.
- a

➔ Meting circulerende endotoxines

➔ Classificatie van patiënt naar gelang ernst parodonttis (PIRI: periodontal index for risk of infectiousness)

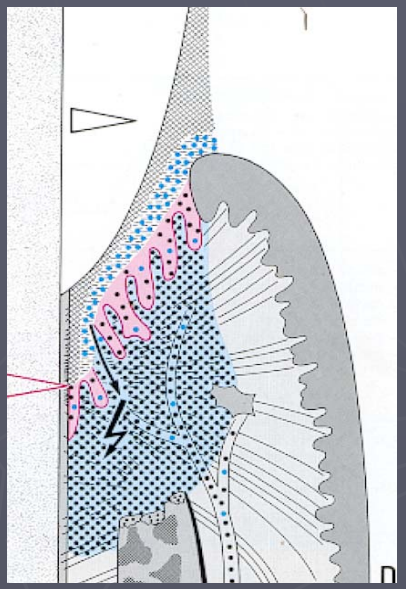
PIRI risk groups	Mastication		
	Before (n = 67)	After (n = 67)	Diff.(n = 67)
Low (O) (n = 25)	0.62 ± 1.57	1.79 ± 2.71	1.17 ± 1.83
Moderate (1-5) (n = 27)	1.33 ± 4.84	2.44 ± 4.84	1.11 ± 4.48
High (6-10) (n = 15)	0.55 ± 1.5	6.13 ± 9.34	5.58 ± 9.13
	p = 0.92	p = 0.037	p = 0.034

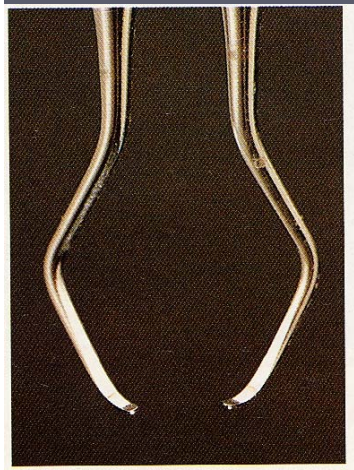


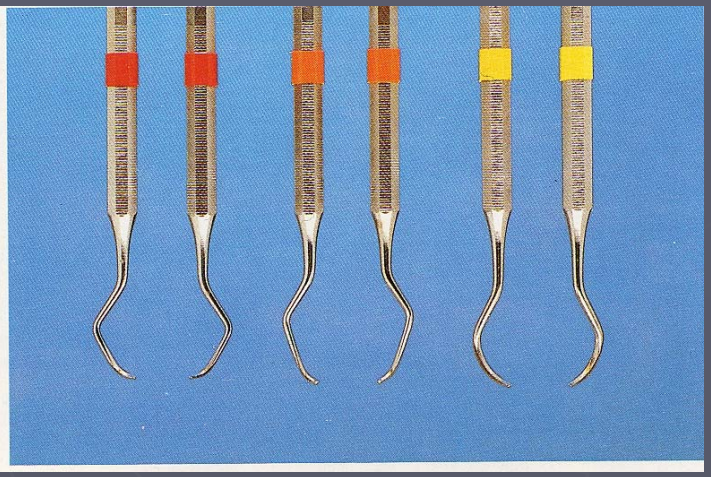
Presence of periodontal disease determines the risk of systemic spread of endotoxins

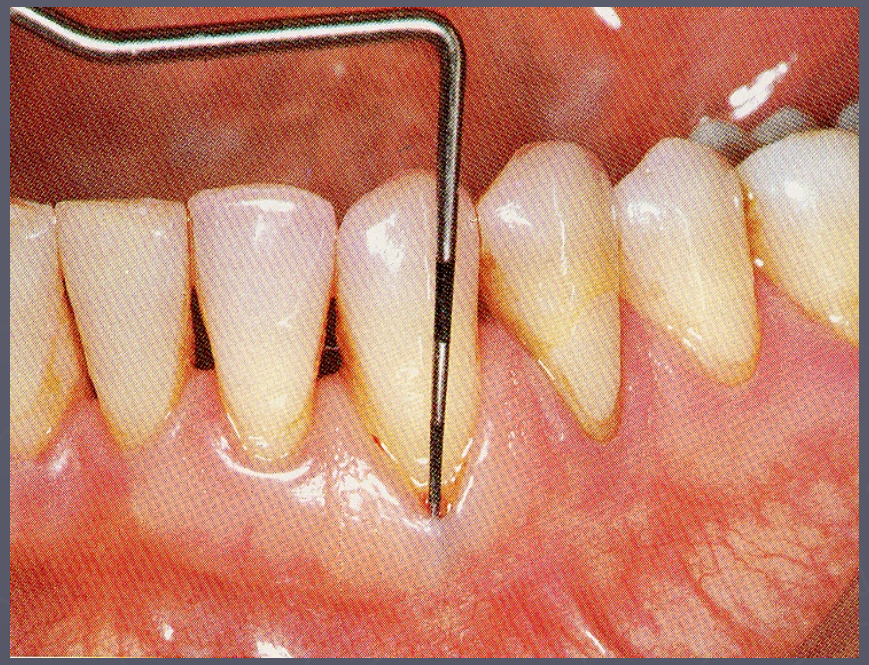
Systemic release of endotoxins induced by gentle mastication: association with periodontitis severity.
Geerts SO, Nys M, De MP, Charpentier J, Albert A, Legrand V, Rompen EH., J Periodontol. 2002 Jan;73(1):73-78.

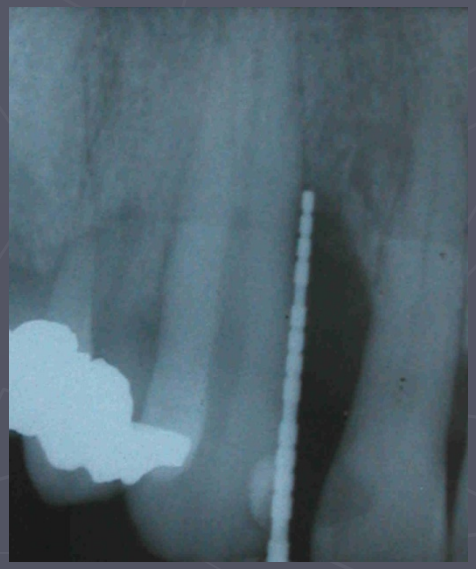
- De mondholte is een belangrijke leverancier van circulerende bacteriële componenten
- De hoeveelheid toxines in de bloedbaan is proportioneel gerelateerd met de uitgebreidheid van het ontstoken parodontium
- Het wordt steeds duidelijker dat een oorzakelijk verband bestaat tussen parodontale ontsteking en bepaalde systeemziekten

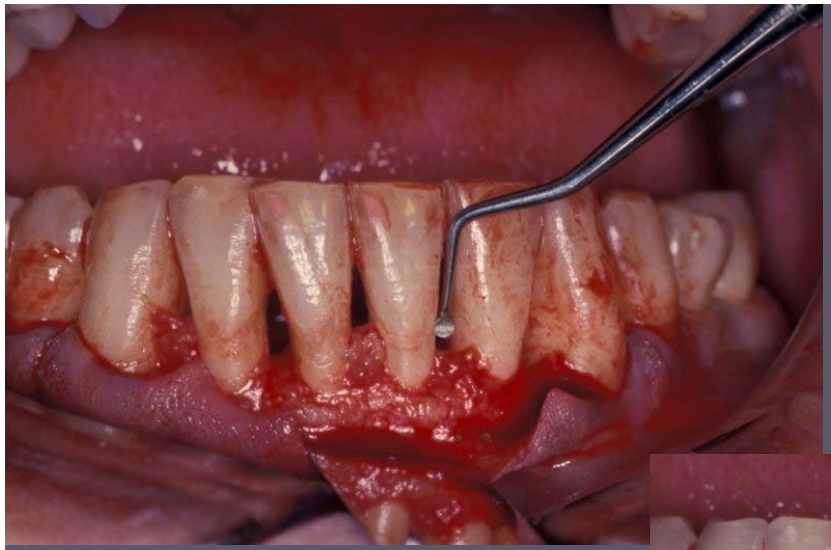


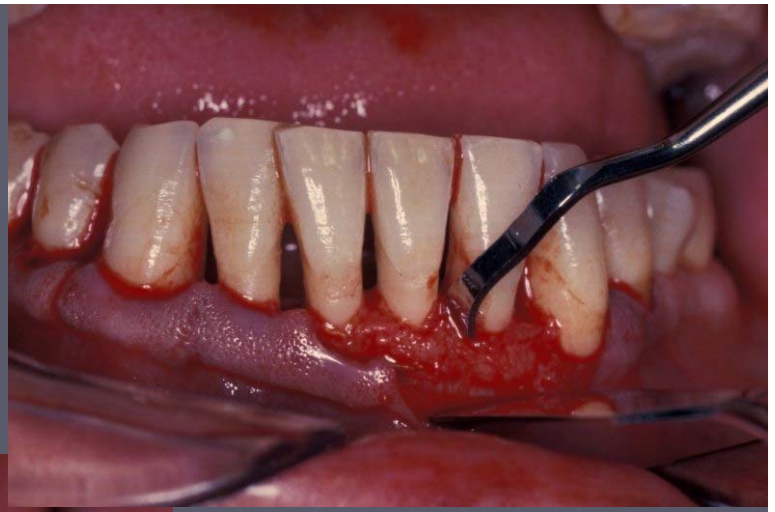


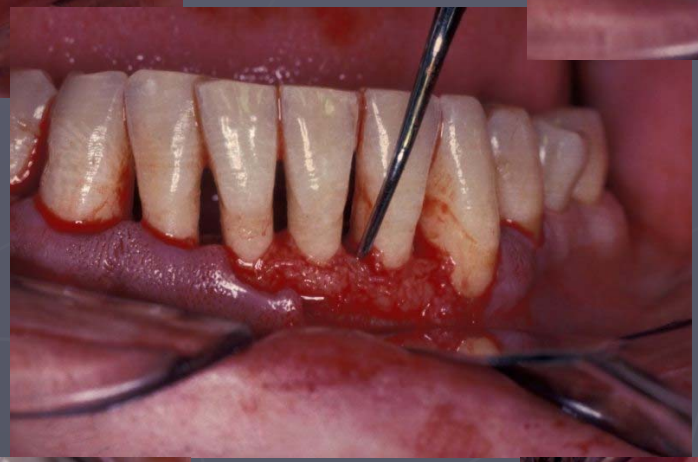












Oorzaken?

Allergie aan kunsthars(vulling)

Verkeerde poetstechniek

Medicatiegebruik

Roken

Diabetes

Specifieke bacteriële infectie

Denudatie bot met necrose oorzaken?

Overdreven plaqueopstapeling

Poetstrauma

Verbranding

Radiotherapie

Medicatiegebruik

Autoimmuunziekte

Specifieke bacteriele infectie

Periodontics and…

- Diabetes mellitus
- Cardiovascular diseases and atherosclerosis
- Preterm birth and low birth weight
- Rheumatoid arthritis
- Respiratory diseases

Diabetes Mellitus

Diabetes ⟺ Periodontitis

I. Diabetes ⇨ Periodontitis

II. Periodontitis ⇨ bad glycemic control (HbA1 level and plasma glucose)

Clinical periodontology and implant dentistry, Fourth edition, J. Lindhe
Perrino MA, 2007
Pihlstrom B et al., 2005

Diabetes Mellitus

I. Diabetes → Periodontitis

A. Effect on microbiota

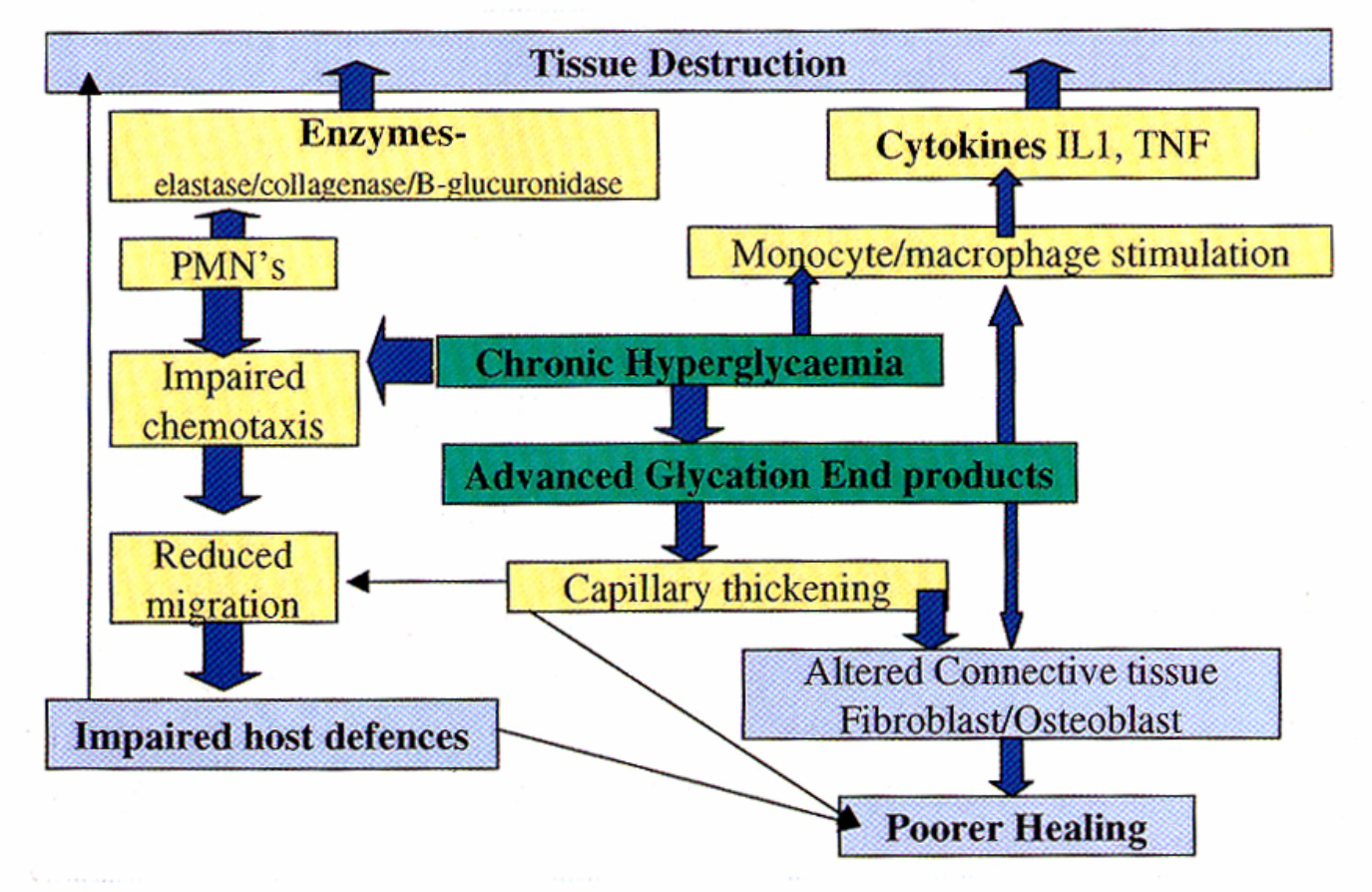
Hyperglycemia changes the bacterial load

↑ spirochetes
motile rods

↓ cocci

Diabetes Mellitus

B. Effects on host response



Diabetes Mellitus

C. Effects on healing and treatment response

Bad Healing

1. Decreased synthesis of collagen by fibroblasts
2. Increased degradation by collagenase
3. Defective remodeling and rapid degradation of newly synthesized, poorly cross-linked collagen

! Well-controlled diabetics react similar to healthy patients on periodontal treatment !

Diabetes Mellitus

II. Periodontitis:

Treatment ic control.

CONCLUSION:

A bidirectional approach is necessary to improve general health

CardioVascular Diseases

Periodontitis is a risk indicator of death due to CVD, especially in combination with smoking.

a) Clinical evidence:

Syrjanen, 1989:

‘ relation ‘

DeStefano, 1993:

2X higher risk

Jansson L, 2001

CardioVascular Diseases

Periodontitis is a risk indicator of death due to CVD, especially in combination with smoking.

Initiation/Progression of CVD by periodontal inflammation

b) Combination of periodontitis & CVD

1. LPS
2. Bactaeremia
3. CRP increased
4. von Willebrand factor AG
5. Cytokines perio=atheromatous
6. Bacteria=P.gingivalis,P.intermedia,P.forsythus,A.a.

Jansson L, 2001

CVD

!Dental infection is significantly correlated to death in CVD!

Periodontal treatment

↓serum inflammatory markers

↓C-reactive proteïn

↓

Lower risk for CVD or death

Johansson C, 2008

Preterm birth and low birth weight

There is a LINK between

periodontitis and preterm birth (PTB)
low birth weight (LBW)

Link?

Periodontitis → infection disease → PTB + LBW

Infection: PG E2
LPS
TNF-α
→ systemic circulation
→ infection uterus
→ contractions

Clinical periodontology and implant dentistry, Fourth edition, J. Lindhe
Pihlstrom B et al., 2005
Offenbacher et al., 1996

Preterm birth and low birth weight

Other factors to consider:

- maternal age (< 18 years)
- race
- use of alcoholics, drugs, tabacco
- maternal stress
- genetic background
- genitourinary tract infections
- socio-economic background

BUT even after adjusting for these factors,

an association between PTB & LBW and periodontitis remains

Pihlstrom B etal., 2005
Lopez R, 2005

Preterm birth and low birth weight

Periodontal treatment reduces risk on PTB and LBW.

When:

- Best before pregnancy.
- Otherwise in 2nd trimester of pregnancy.

<u>CONCLUSION:</u>

Women with childwish:

- check oral health
- referral to dentist or periodontologist

Respiratory Disease

Respiratory infections are associated with periodontal disease

Respiratory pathogens colonising the mouth can cause:

1. Bacterial pneumonia
2. COPD
3. Chronic bronchitis
4. Emphysema

Pihlstrom B et al., 2005

Respiratory Disease

Respiratory infections are associated with periodontal disease

Risc population: people in institutions
people in intensive care units

➡ Good oral health is important in this population.

Rinsing with chlorexidine can reduce the risk.

Pihlstrom B et al., 2005

Respiratory Disease

1° **LAB**:

bacteria from the pocket are the same as in sputum

2° **CLINICAL**:

patients with COPD show more periodontal attachment loss

HIV

I) HIV patient ⇒ immune compromised

=More prone to infections

¿Periodontitis?

SO: Carefull follow up of oral health of a HIV patient
Referral to a dentist (when necessary).

HIV

II) ANUG (acute necrotising ulcerative gingivitis/ periodontitis)

ANUP

Sometimes symptoms of presence of HIV / AIDS

SO: It's preferable to send these patients to a doctor for further examination.

Infection control of mouth is needed for general health reasons

HIV

HIV

Hairy leucoplacia

HIV

Kaposi Sarcoom

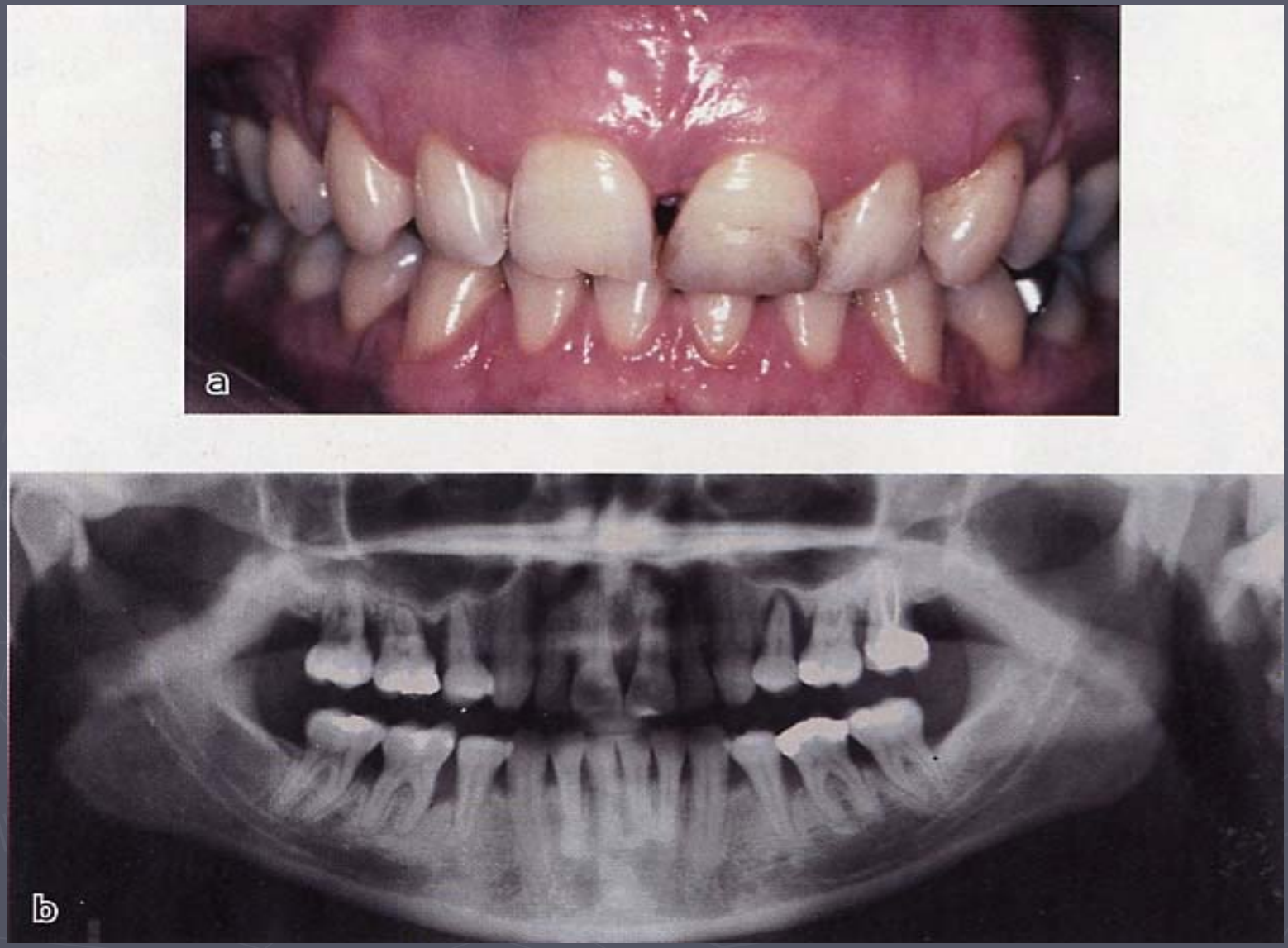
Smoking

Smokers have more periodontitis.

1. Deeper probing depths and larger number of deep pockets.
2. More attachment loss including more gingival recession.
3. More alveolar bone loss.
4. More tooth loss.
5. Less gingivitis and less bleeding on probing.
6. More teeth with furcation involvement.

Clinical periodontology and implant dentistry, Fourth edition, J. Lindhe

Smoking



Smoking

Why?

Different reasons.

I. Effects on plaque bacteria
II. Effects on the host response
III. Effects on healing and treatment response

Smoking

I. Effects on plaque bacteria

Smokers have higher levels of plaque

The plaque harbours more bacterial species which are associated with periodontitis.

Smoking

II. Effects on the host response.

Development of inflammation retarded:

less sites exhibiting redness
less bleeding on probing
⟵ Decreased vascularity
Nicotine VC

Defence : Higher levels of **PMN** in the blood
Lower level in the periodontal sulcus

- Neutrophil defects → more agressive periodontal breakdown
- PMN → elastase/collagenases → tissue destruction
- Smoking → reduced phagocytic capacity of PMN/neutrophils
  → affects B and T cell function
  → reduces most of the Ig except Ig E

Smoking

III. Effects on healing and treatment response.

- Surgery: impaired healing
- Non- surgical treatment : - less reduction probing depth
  - less gain clinical attachment

Stress

Emotional and psychosocial stress are risk indicators periodontal disease.

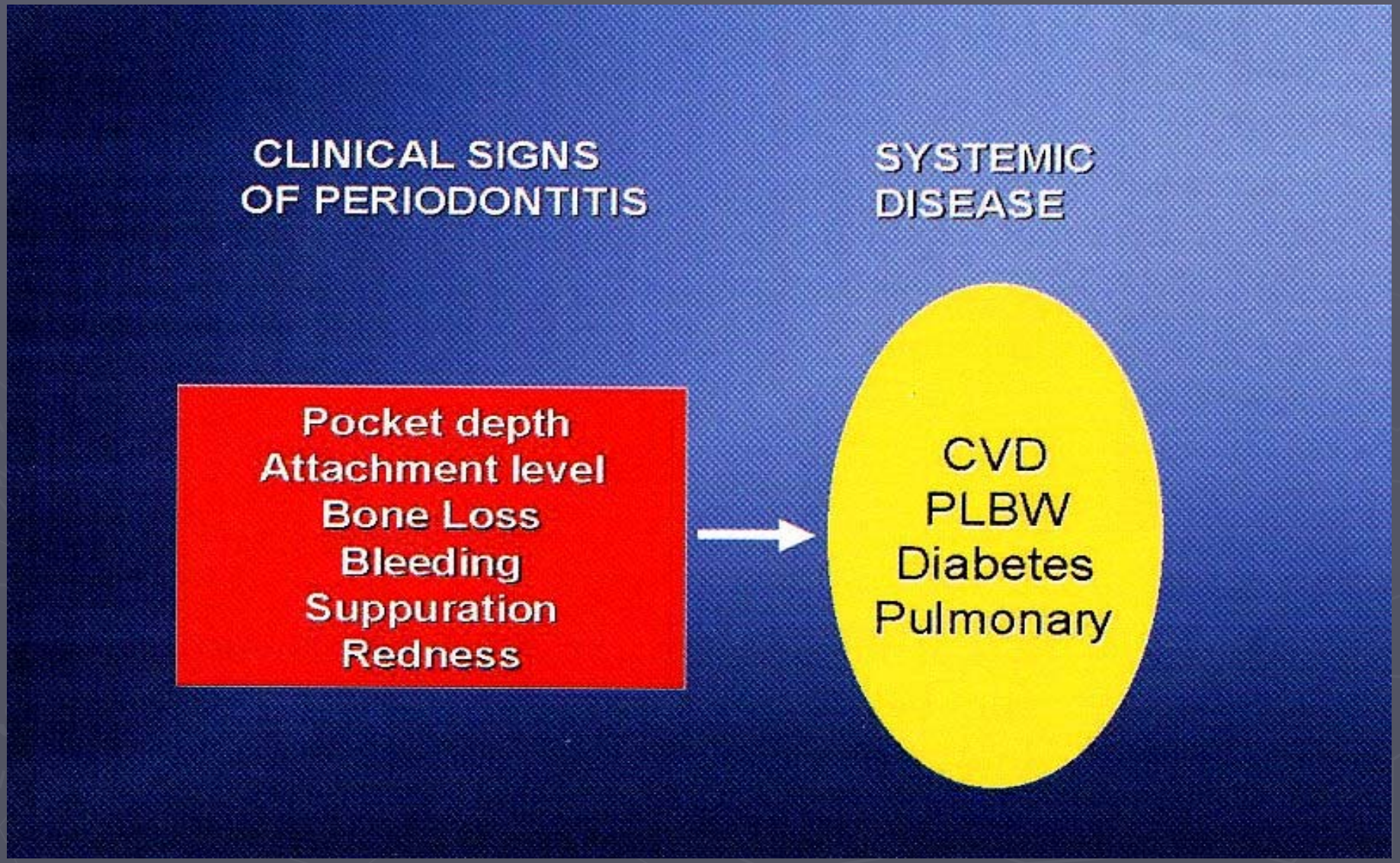
Anti - inflammatory effects

Immunosuppressive

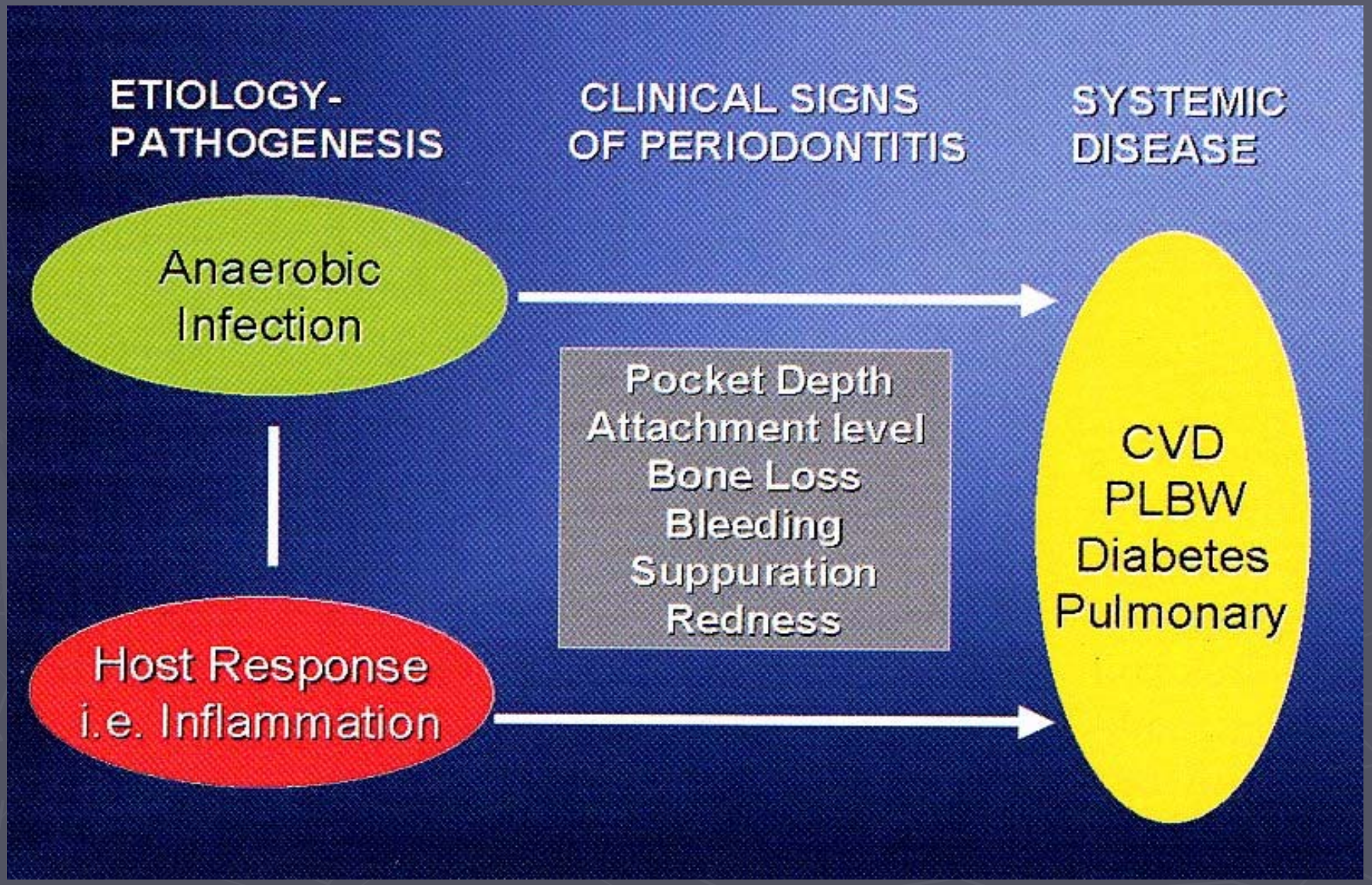
Clinical periodontology and implant dentistry, Fourth edition, J. Lindh

Pihlstrom B et al., 2005

Conclusion


CLINICAL SIGNS
OF PERIODONTITIS
SYSTEMIC
DISEASE
Pocket depth
Attachment level
Bone Loss
Bleeding
Suppuration
Redness
CVD
PLBW
Diabetes
Pulmonary

Conclusion


ETIOLOGY-
PATHOGENESIS
CLINICAL SIGNS
OF PERIODONTITIS
SYSTEMIC
DISEASE
Anaerobic
Infection
Host Response
i.e. Inflammation
Pocket Depth
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Bone Loss
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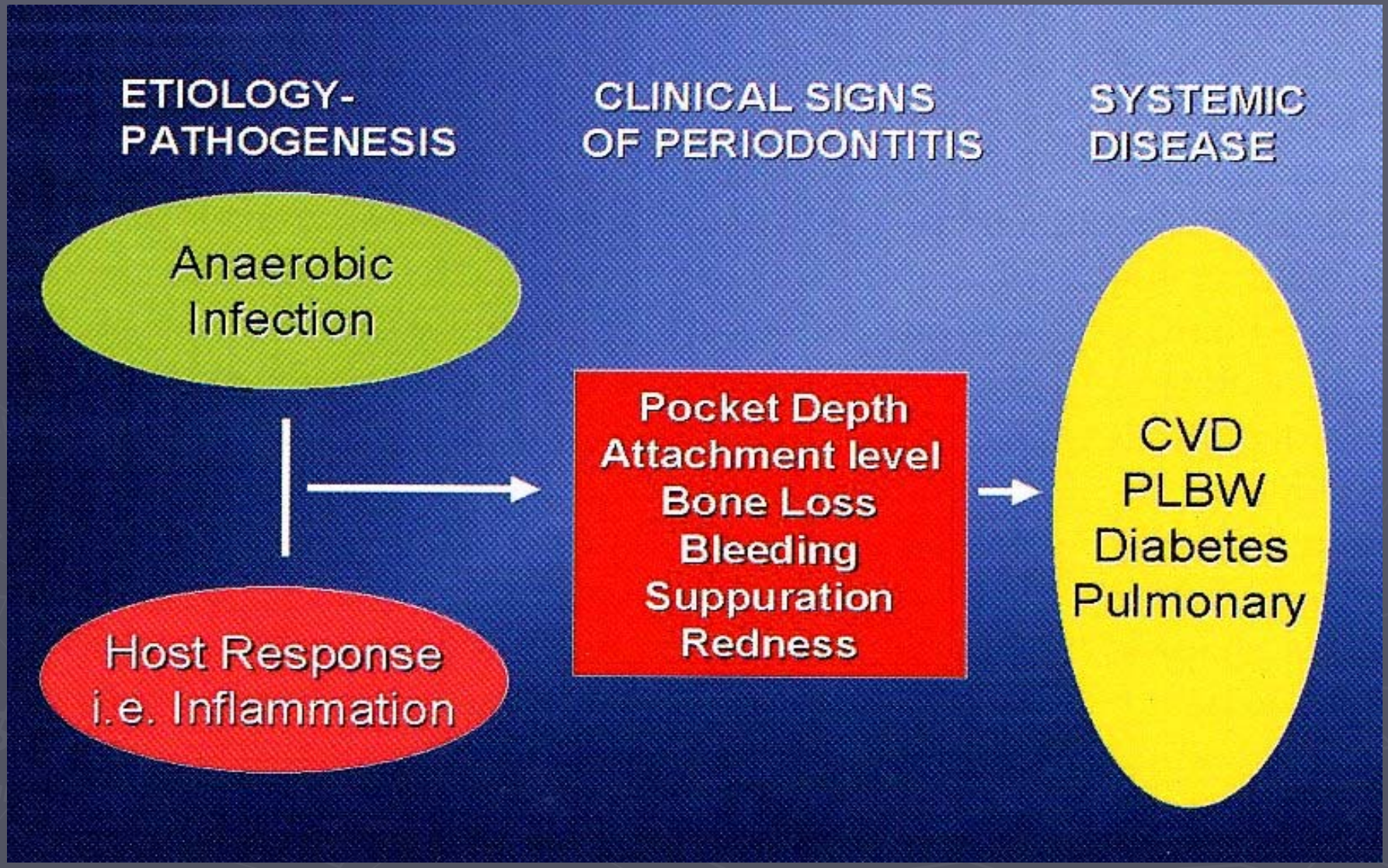
Conclusion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Pulmonary

Reference

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Medication

Quid periodontium?

- **Treatment of Periodontitis**
  - Is the use of *Antibiotics justified?*
- **Osteochemonecrosis**
  - ***B**isphosphonate **R**elated **O**steo**n**ecrosis of the **J**aw (BRONJ)*
- **Drug-Induced Gingival Overgrowth**
  - *Phenytoins*
  - *Cyclosporines*
  - *Calcium channel blockers*

Treatment of Periodontitis

Is the use of antibiotics justified?

Treatment of Periodontitis

- Prevention:
  - Oral hygiene instructions
  - Scaling (supragingival)
- Treatment
  - Non-Surgical therapy: Scaling and rootplaning
  - Surgical therapy: Flap
  - ***Antibiotics?***

Treatment of Periodontitis

Is the use of antibiotics justified?

- Antibiotics
  1. Additional benefits of systemic antibiotics
  2. Additional benefits of local antibiotics
  3. Additive microbiological effects
  4. Adverse effects of antibiotics
  5. Long-term effects
  6. Patient Selection
  7. Selection of Antibiotics
  8. Conclusions

Treatment of Periodontitis

Is the use of antibiotics justified?

1. Additional clinical effects: *systemic antibiotics*

DEEP POCKETS	PD reduction (mm)	CAL gain (mm)
SRP	1.80	1.24
SRP + sys. AB	2.84	1.54
DIFFERENCE	**1.04**	**0.30**

(Herrera et al. 2002)

Treatment of Periodontitis

Is the use of antibiotics justified?

2. Additional clinical effects: *Local antibiotics*

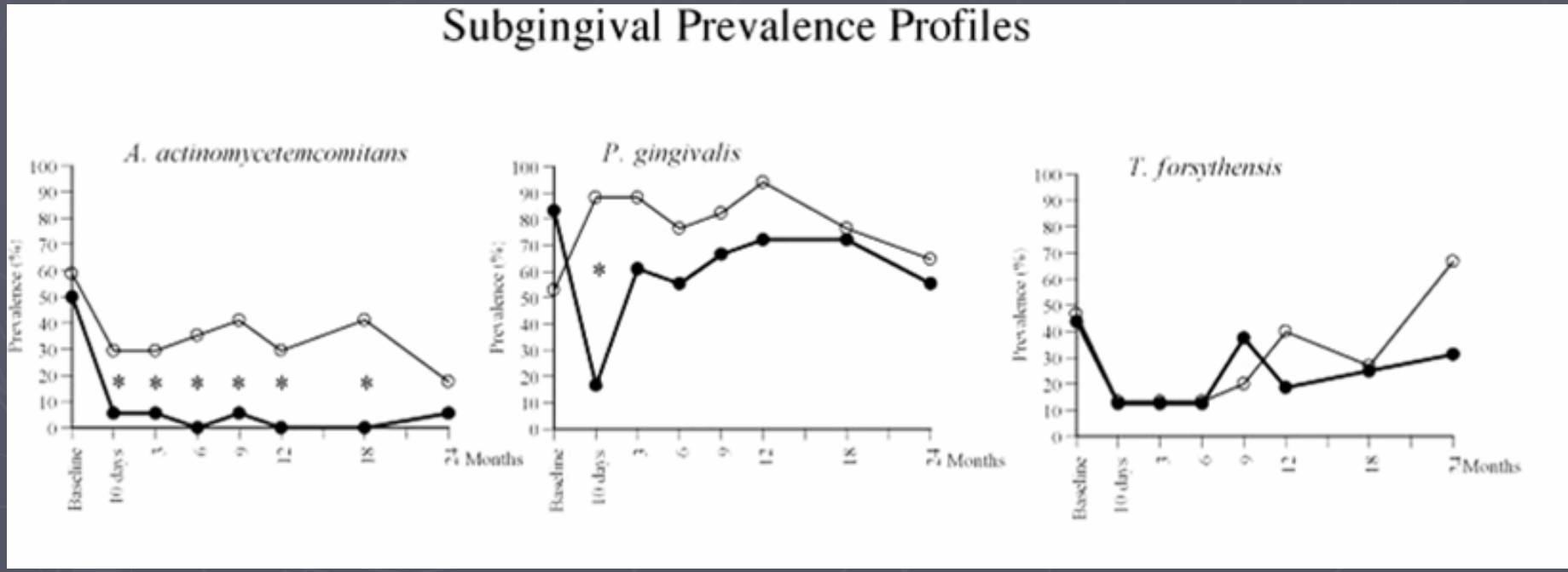
Local adjunctive therapy	PD reductions	CAL gain
	N positive studies / N studies	N positive studies / N studies
Tetracyclin	5/16	2/16
Minocyclin	4/8	3/8
Metronidazole	4/11	2/8
Other Antibiotics	1/1	1/1

(Bonito et al. 2005)

Treatment of Periodontitis

Is the use of antibiotics justified?

3. Additive microbiological effects



(Ehmke et al. 2005)

Treatment of Periodontitis

Is the use of antibiotics justified?

4. Adverse effects of antibiotics *(Slots et al. 2004)*
   - Bacterial resistance
   - Gastrointestinal disorders
   - Drug interactions
   - …
5. Long-term effects *(Serino et al. 2001)*
   - Prospective study in recurrent periodontitis patients over 5 years
   - Metronidazole + amoxicilline for 2 weeks
   - 75% showed relaps in 5 year follow-up period

Treatment of Periodontitis

Is the use of antibiotics justified?

6. Patient Selection
   - Non-responders conventional mechanical therapy:
     - Persistent pathogens
     - Impaired host resistance
   - Aggressive types of periodontitis
   - Severe infections: systemical manifestations
     - Abscess
     - Acute necrotizing gingivitis/periodontitis
   - Prophylaxis

Treatment of Periodontitis
Is the use of antibiotics justified?

7. Selection of Antibiotics (SPECTRUM)

- Monotherapy
  - Amoxycillin
  - Metronidazole
  - Clindamycin: *recurrent periodontitis*
    - *Cave: pseudomembranous colitis*
  - Doxycycline or Minocycline: *Aa*
  - Ciprofloxacin
  - Azithromycin
- Combitherapy
  - Metronidazole + Amoxycillin
  - Metronidazole + Ciprofloxacin
    - Allergie to B-lactam drugs

Treatment of Periodontitis
Is the use of antibiotics justified?

8. Conclusions
   - The use of antibiotics in the treatment of periodontitis is NOT justified
     - Additional clinical effects
     - Microbiological effects
     - Adverse effects
     - Questionable long-term effects
   - Patient selection
   - Selection of antibiotics: SPECTRUM

BRONJ

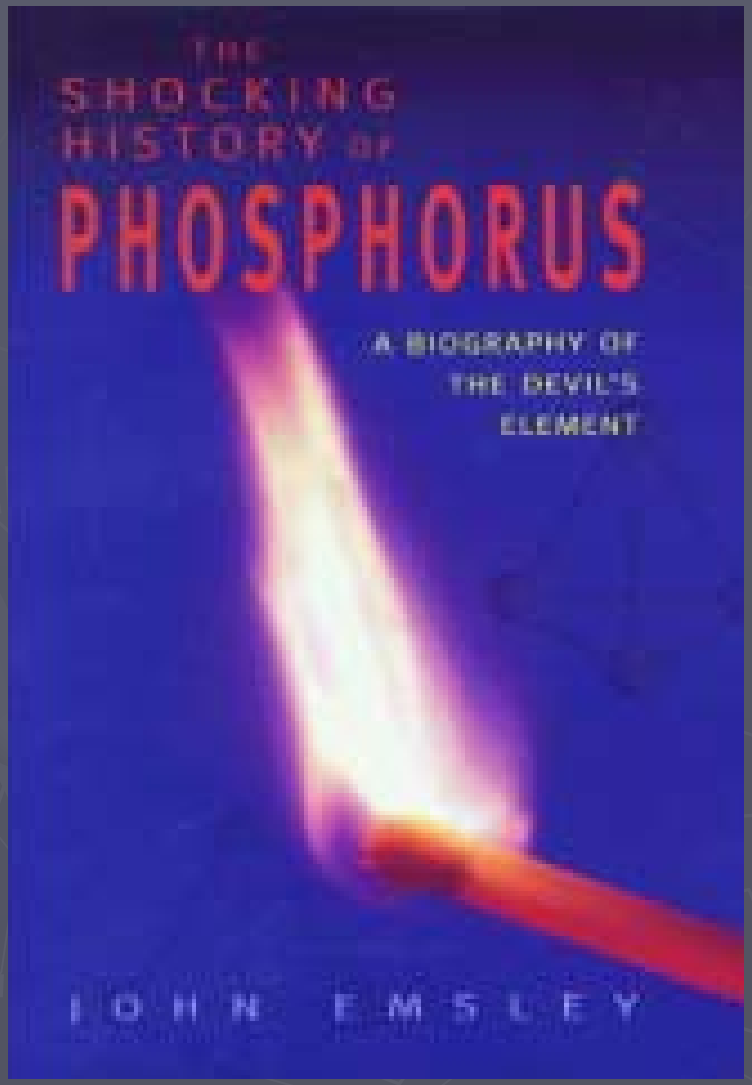
Bisphosphonates

Quid bone?

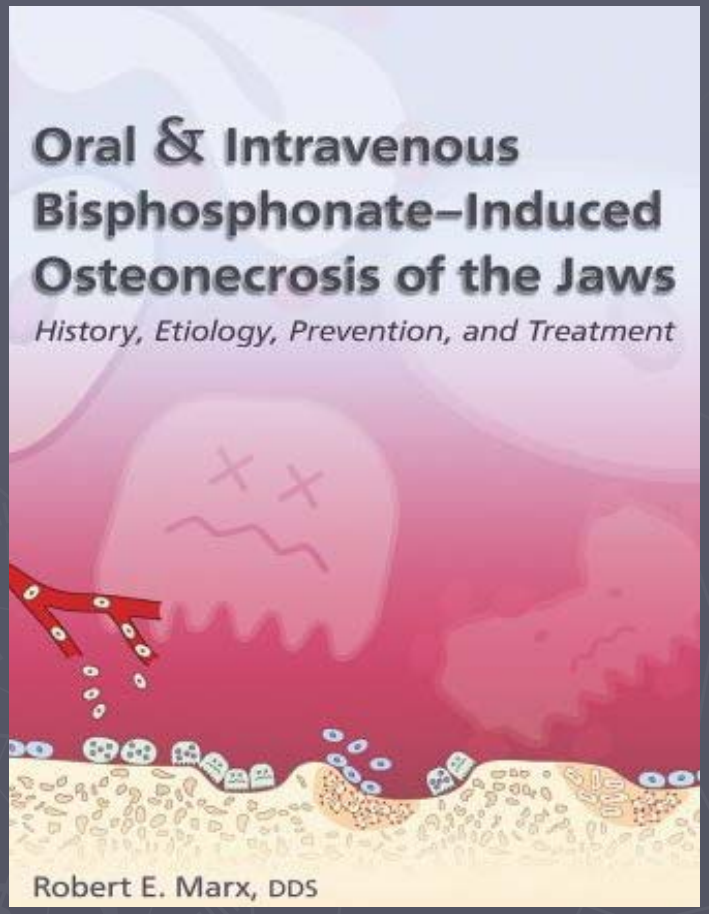
BRONJ

BRONJ

Return of the Phossy Jaw?



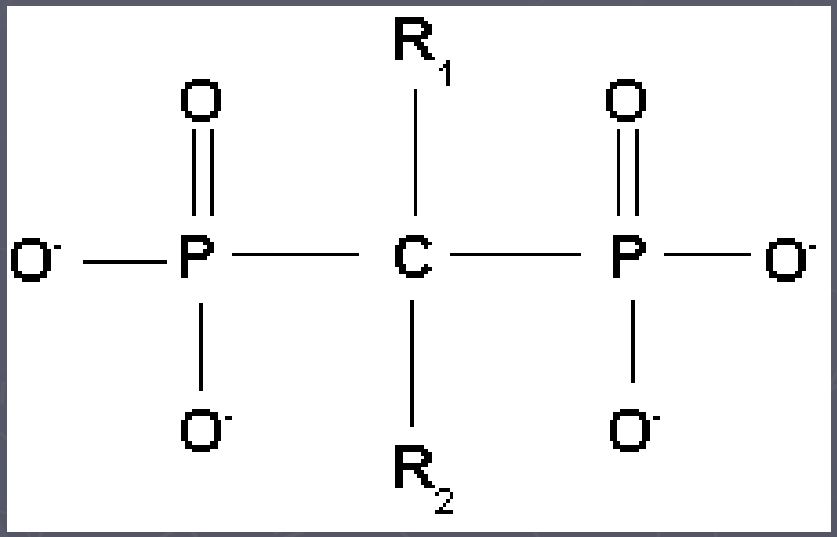
BRONJ

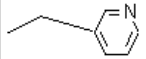
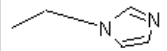


- Robert E. Marx 2003
- Novartis
- Recommendations treatment and prevention
- Incidence onco: 4-10%
- Incidence Osteoporose?

BRONJ
Bisphosphonates

- Chemical Structure



Agent	R_1 side chain	R_2 side chain
Etidronate	-OH	$-CH_3$
Clodronate	-Cl	-Cl
Tiludronate	-H	-S- -Cl
Pamidronate	-OH	$-CH_2-CH_2-NH_2$
Neridronate	-OH	$-(CH_2)_5-NH_2$
Olpadronate	-OH	$-(CH_2)_2N(CH_3)_2$
Alendronate	-OH	$-(CH_2)_3-NH_2$
Ibandronate	-OH	$-CH_2-CH_2N$ (CH_3, $(CH_2)_4-CH_3$)
Risedronate	-OH	
Zoledronate	-OH	

- Non-nitrogenous vs Nitrogenous
- PCP binding to hydroxyapatite
- R_1 OH binding to hydroxyapatite enhanced
- R_2 determines potency

BRONJ

Bisphosphonates

- Antiresorptive Relative Potency

Etidronate	1
Clodronate	10
Tiludronate	10
Pamidronate (ACTONEL®)	100
Alendronate (FOSAMAX®)	100-1000
Ibandronate	1000-10000
Risedronate (AREDIA®)	1000-10000
Zoledronic acid (ZOMETA®)	≥ 10000

BRONJ

Bisphosphonates

- Synthetic analogues of inorganic pyrophosphate
- Accumulate in bone
- May remain in body for years
- Potent inhibitors of osteoclastic activity
- Boneresorption→ Release→ phagocytized by osteoclasts→

APOPTOSIS

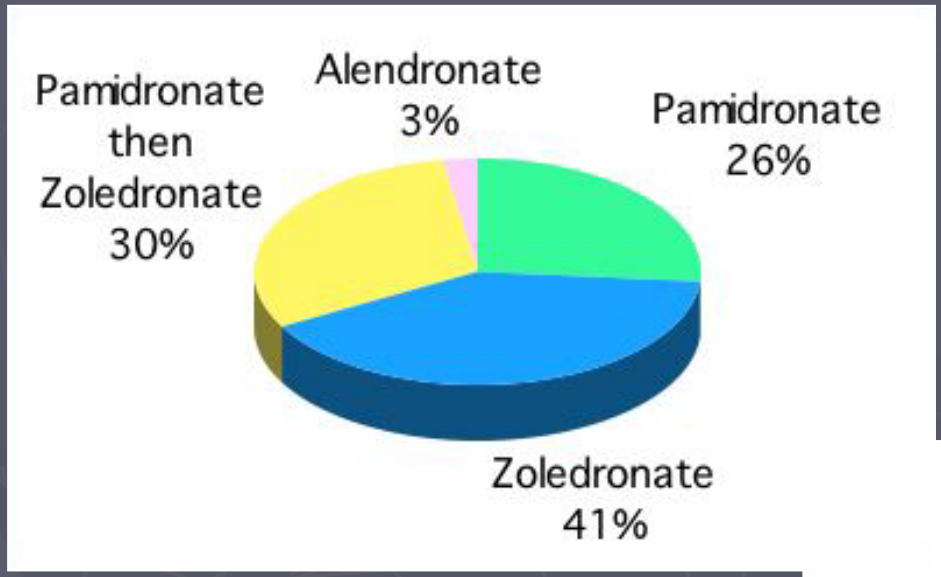
BRONJ

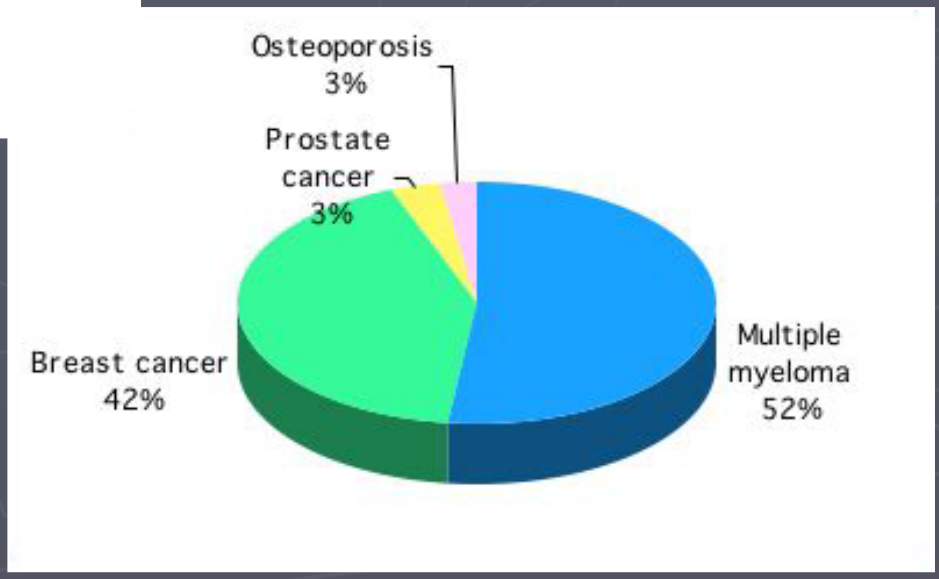
Bisphosphonates

- Indications:
  - Metastatis solid tumors in bone (breast-prostate) Multiple Myeloma
    - Intravenous application (pamidronate, zoledronate)
    - Treatment bone resorption defects
    - Stabilisation metastatic cancer deposits
  - Osteoporosis:
    - Oral application (eti-, rise-, tilu-, alendronate)
    - postmenopausal
    - preserve bone density
  - Paget's disease
  - Osteogenesis imperfecta

BRONJ

Bisphosphonates





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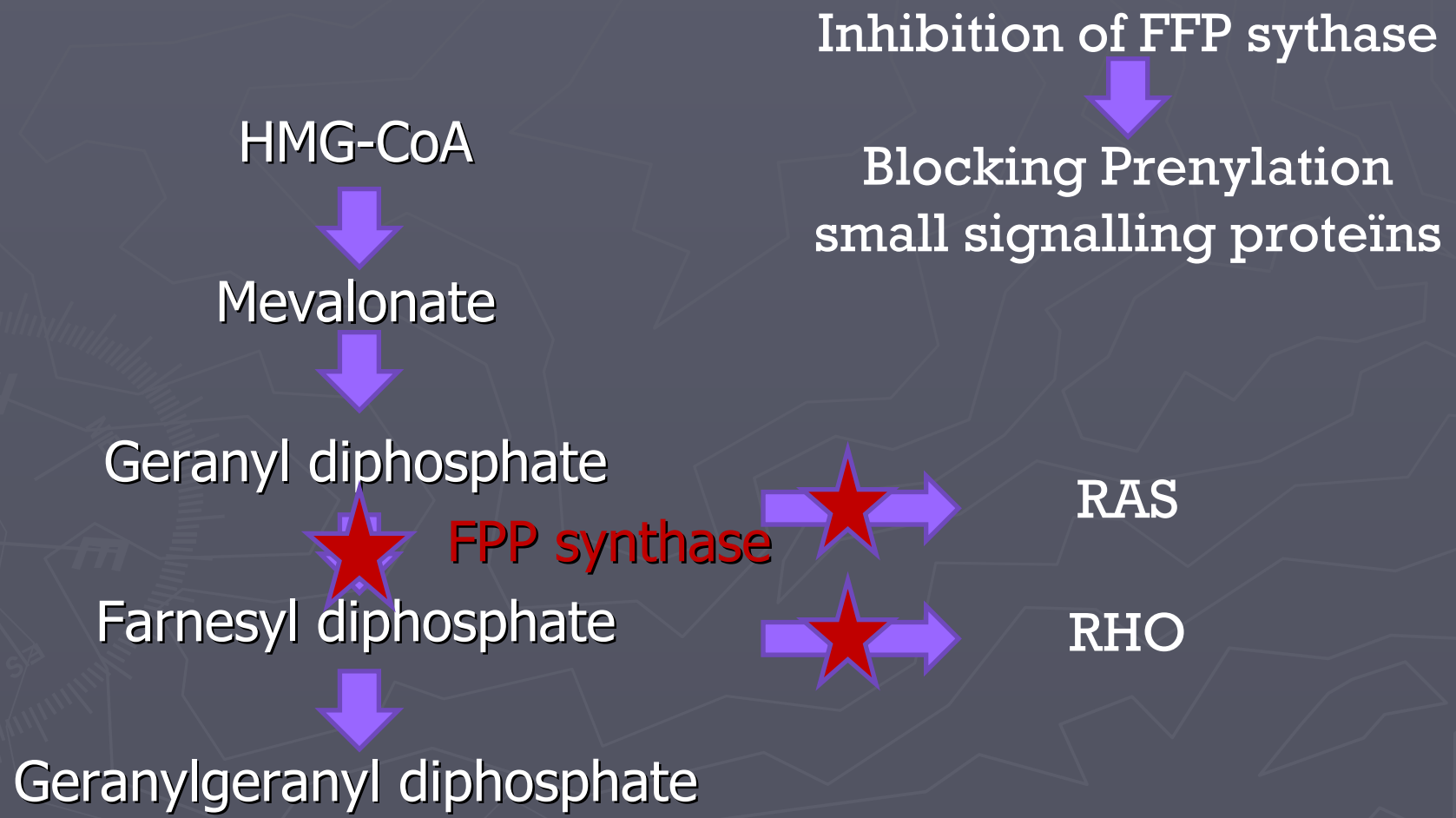
BRONJ

Bisphosphonates

- Mechanism of Action



BRONJ

Osteochemonecrosis of the jaw

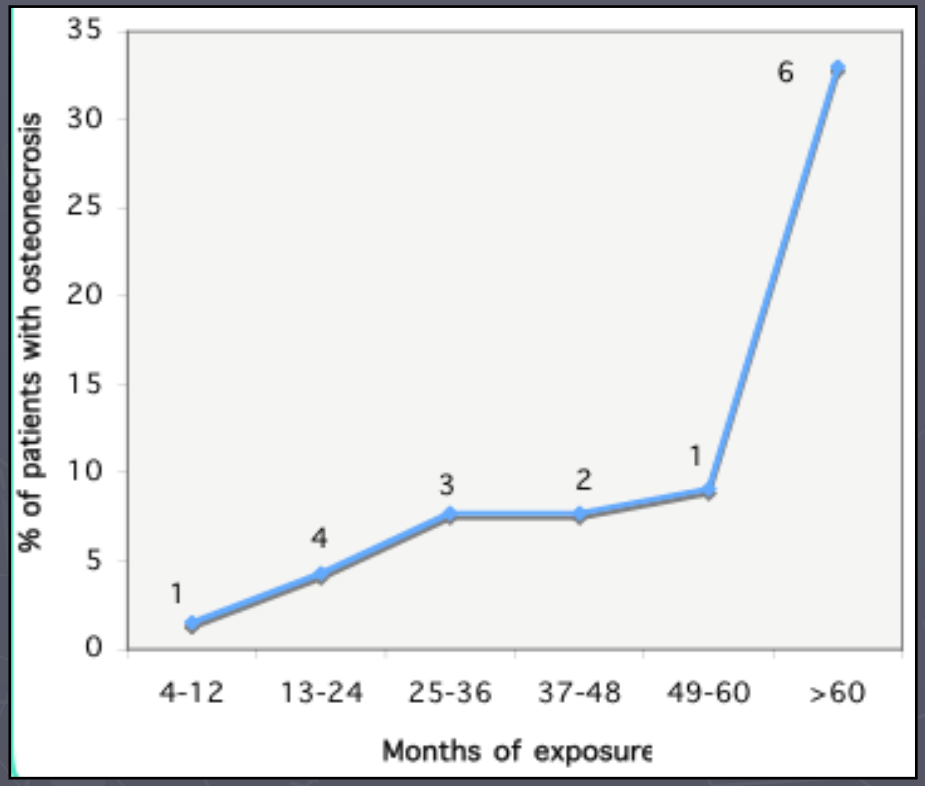
"OSTEOCHEMONECROSIS is a disfiguring jaw condition that includes serious infection and osteopetrosis, an abnormal build up of fragile bone"

This occurs in both maxilla and mandible, unlike osteoradionecrosis, which occurs in patients undergoing head and neck radiation, and is primarily in the mandible

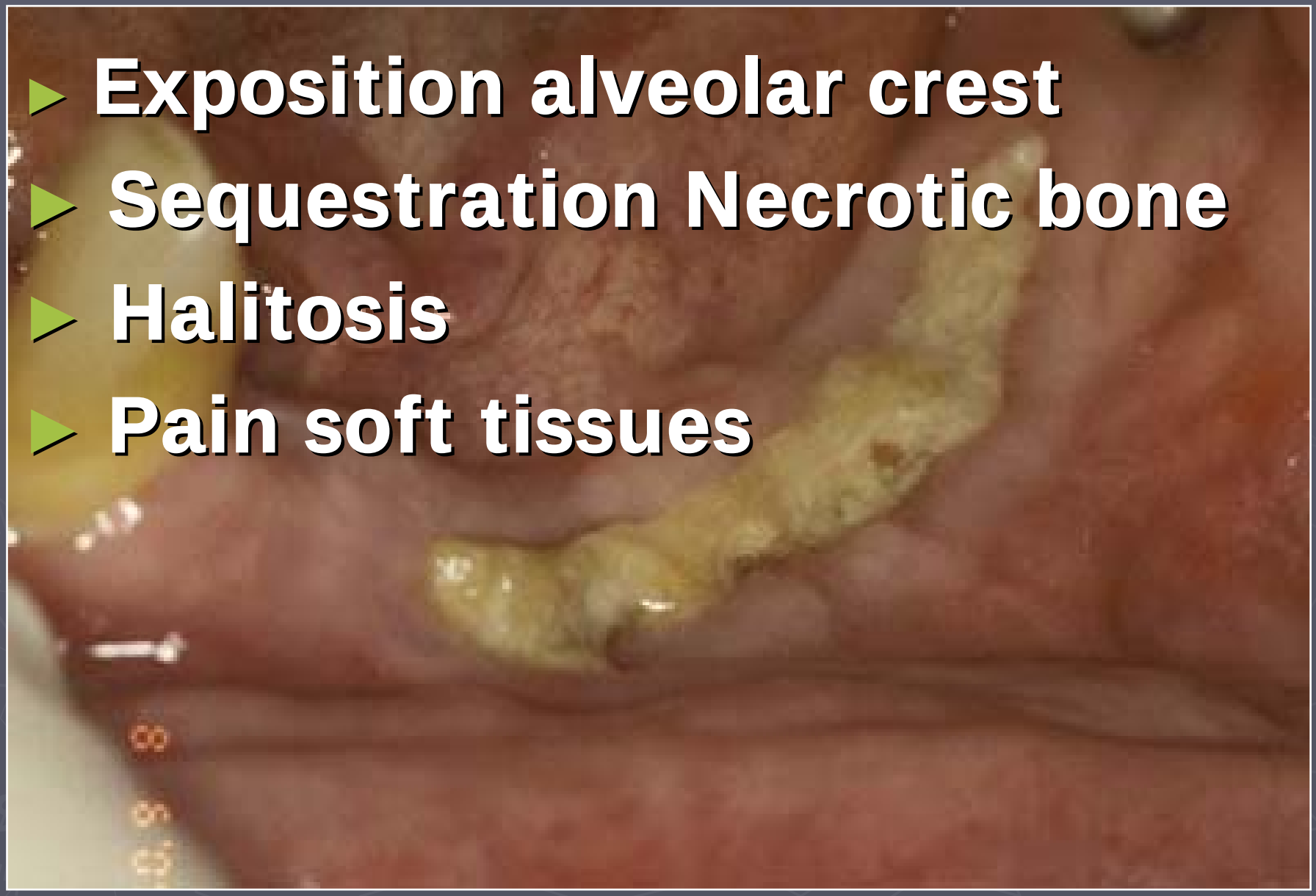
... The higher the potency and longer the duration of the bisphosphonate therapy, the more susceptible the patient is to osteochemonecrosis...

BRONJ

Osteochemonecrosis of the jaw

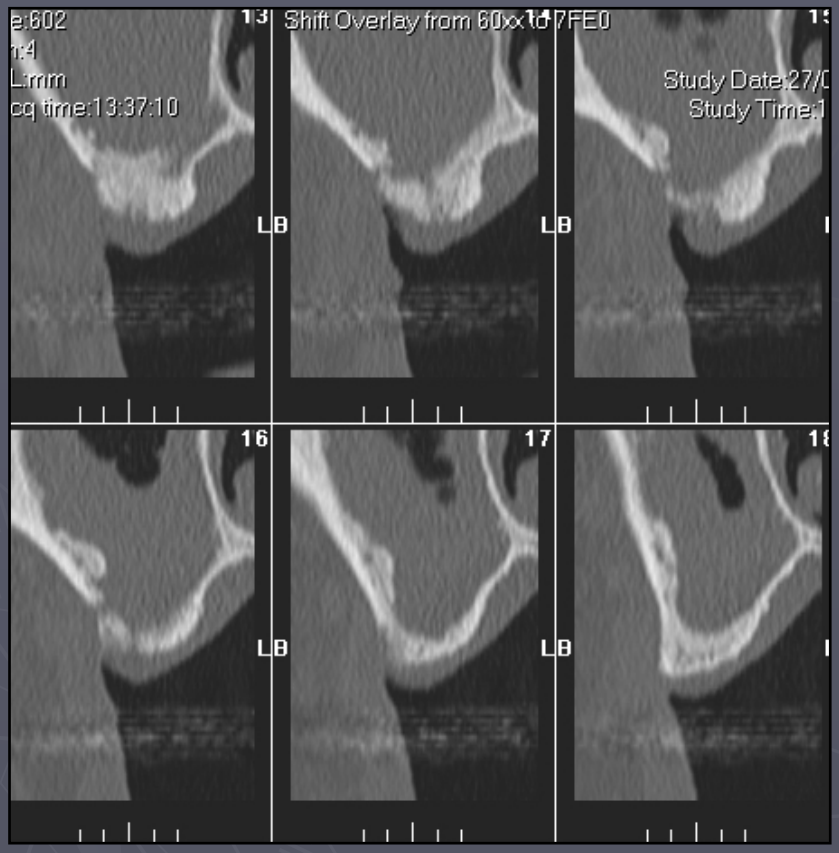


Osteochemonecrosis bij patiënten onder bisfosfonaten


Exposition alveolar crest
Sequestration Necrotic bone
Halitosis
Pain soft tissues

BRONJ

Osteonecrosis of the jaw



- **Extra-oral fistula**
- **Intra-oral fistula**

BRONJ

Patient Population at Risk

- Drug-related Risk Factores
  - Potency: IV vs Oral application
  - Duration of therapy
- Local Risk Factors
  - Dentoalveolar surgery
    - extraction,
    - periodontal surgery
    - dental implant placement
    - periapical surgery
  - Local anatomy
    - Mandible: lingual tori, mylohyoid ridge
    - Maxilla: palatal tori

AAOMS Position Paper on Bisphosphonate-Related Osteonecrosis of the Jaw
J Oral Maxillofac Surg 2007;65:369-376

BRONJ

Patient Population at Risk

- Concomitant Oral Disease
- Demographic and systemic factors
  - Age
  - Race: Caucasian
  - Cancer diagnosis
  - Osteopenia/Osteoporosis + Cancer diagnosis
- Possible Risk Factors
  - Corticosteroid therapy
  - Diabetes
  - Smoking
  - Alcohol Use
  - Poor oral Hygiene
  - Chemotherapeutic drugs

AAOMS Position Paper on Bisphosphonate-Related Osteonecrosis of the Jaw
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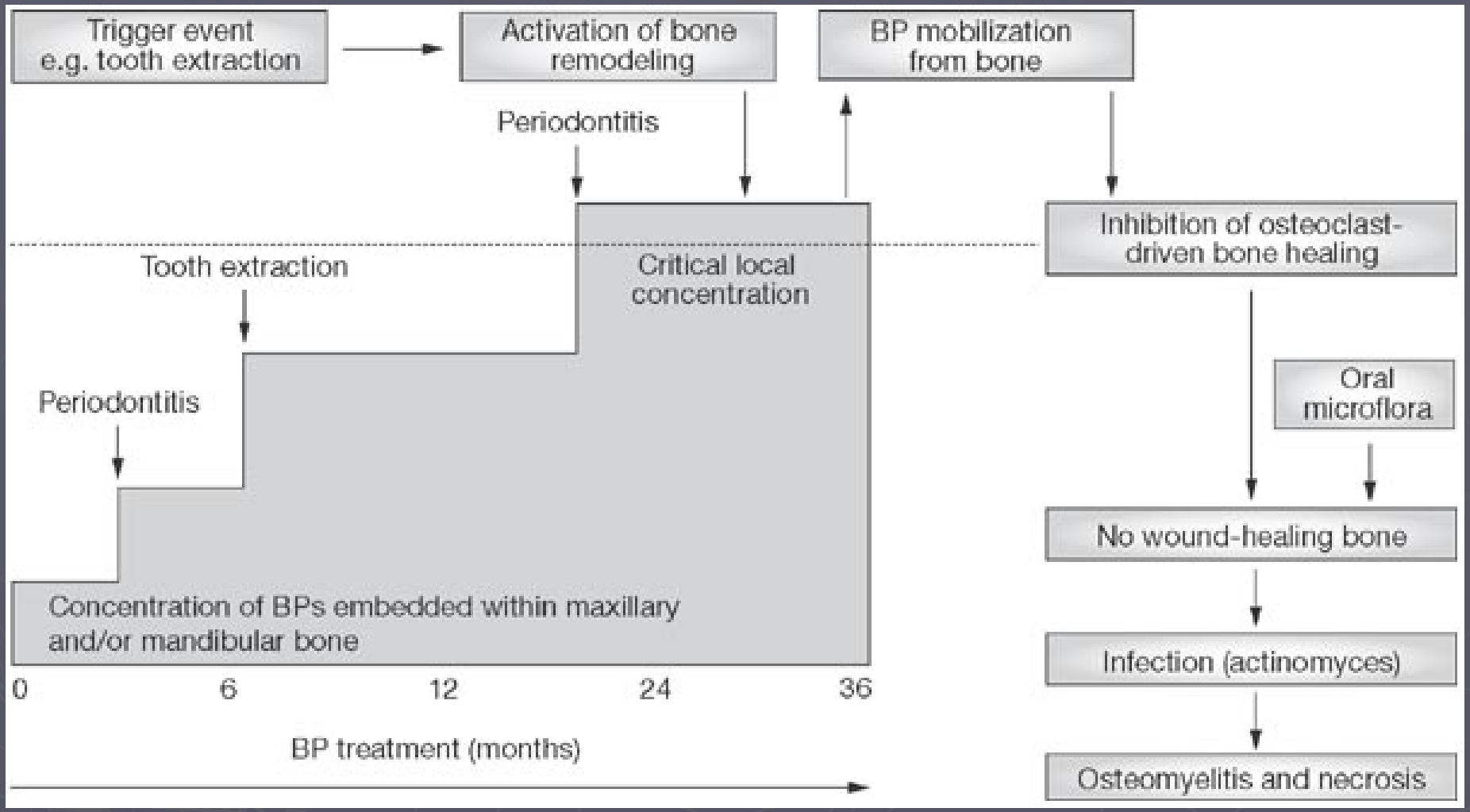


Francesco Bertoldo, Daniele Santini and Vincenzo Lo Cascio

Bisphosphonates and osteomyelitis of the jaw: a pathogenic puzzle

Nature Clinical Practice Oncology (2007)4:711-721

BRONJ
Prevention

- Before Bisphosphonate Therapy
  - Urgent oral examination (clinical, radiographic)
  - Eliminating infection
    - Tooth removal, periodontal surgery, root canal treatment, caries control, dental restorations, dentures
  - Removing
    - large tori
    - Impacted teeth with oral communication
  - Prophylactic antibiotic coverage
    - Invasive dental procedures
    - Penicillin
    - Quinolones/erythromycin + metronidazole
    - No clindamycin alone: ACTINOMYCES
      Eikenella Corrodens

Marx et al. 2005

BRONJ
Prevention

- During Bisphosphonate Therapy
  - Examination and surveillance schedule: *EXPOSED BONE* ?
  - Dental cleaning
  - Non-restorable teeth:
    - Root canal treatment + rootbanking
    - Avoid extraction!!!
  - Extraction: Antibiotic coverage
  - No elective surgery
  - Dentures: soft reline

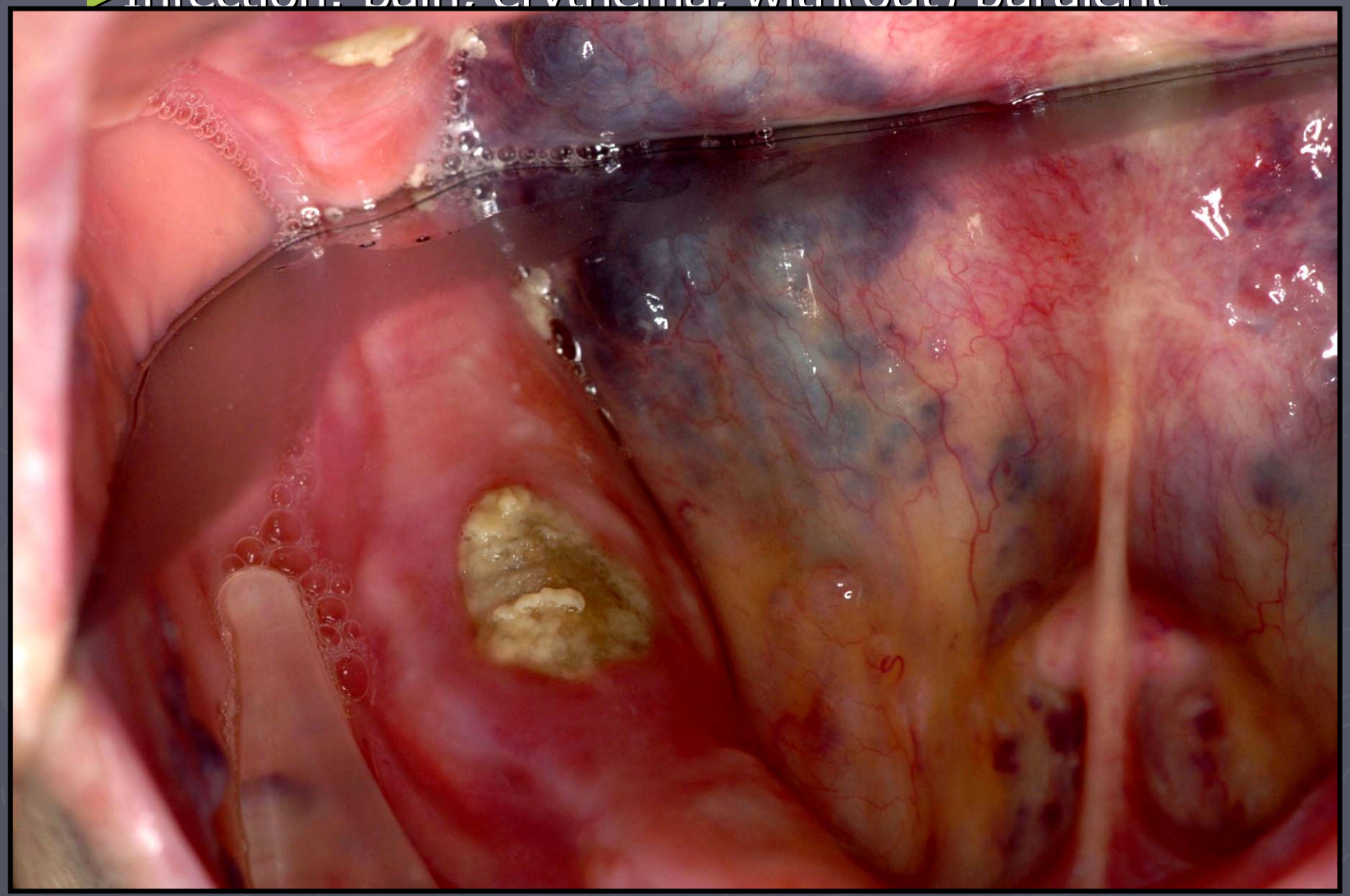
Marx et al. 2005

BRONJ
Treatment

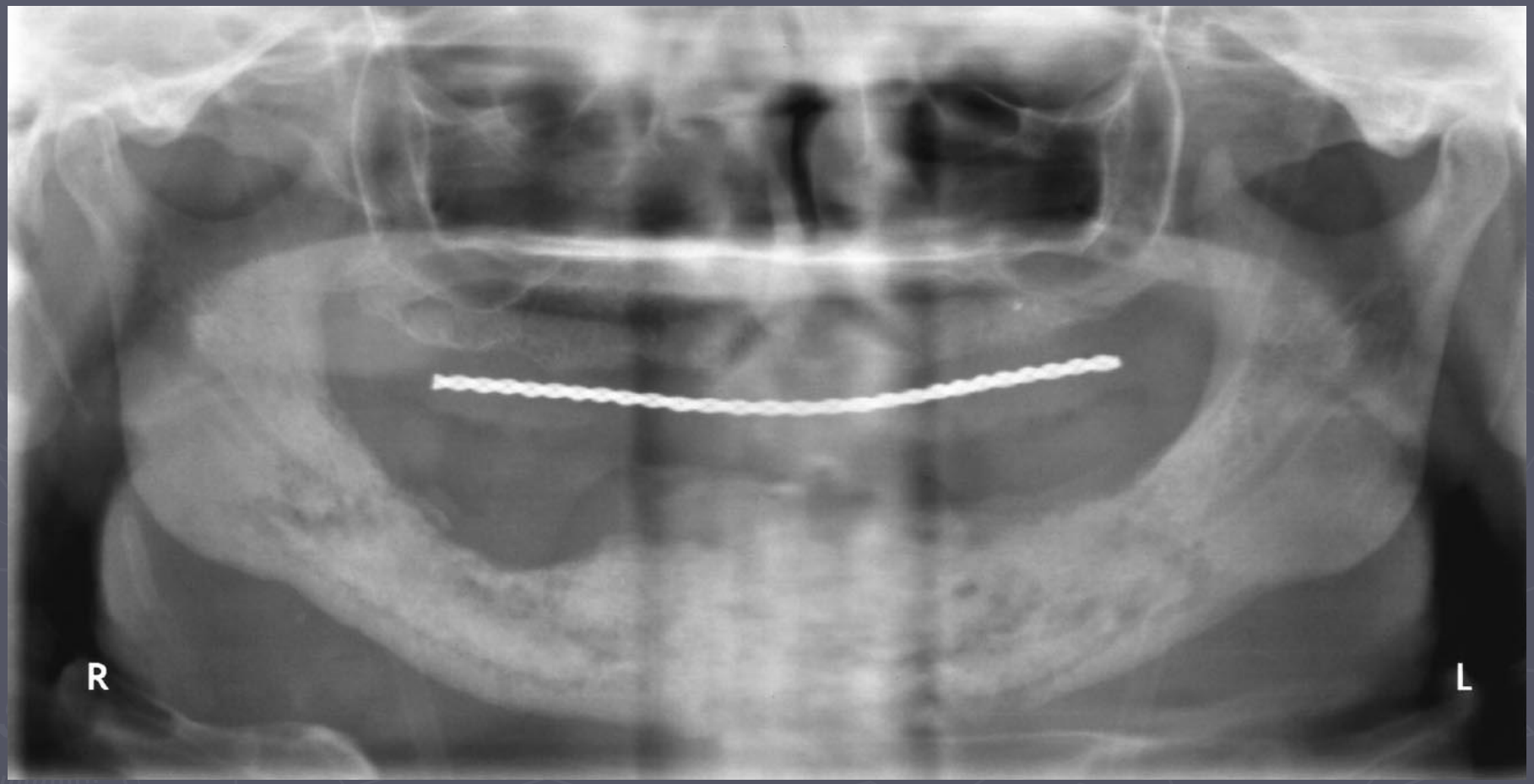
- BRONJ staging
  - Patients At risk :
    - No exposed or necrotic bone
  - Stage 1:
    - Exposed/necrotic bone
    - Asymptomatic, no evidence of infection

AAOMS Position Paper on Bisphosphonate-Related Osteonecrosis of the Jaw
J Oral Maxillofac Surg 2007;65:369-376

- Stage 2:
  - Exposed/necrotic bone
  - Infection: pain, erythema, with(out) purulent



- Stage 3:
  - Stage 2 + extra-oral fistula, pathological fracture or osteolysis inferior border



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Treatment

- Patient at risk:
  - NO TREATMENT → PREVENTION
- Stage 1:
  - Antibacterial mouth rinse
  - Clinical follow-up + patient education
- Stage 2 + 3:
  - Symptomatic treatment: Antibiotics
  - Pain control
  - Antibacterial mouth rinse
  - Superficial debridement/resection:
    - No soft tissue irritation
- *HBO2*

AAOMS Position Paper on Bisphosphonate-Related Osteonecrosis of the Jaw
J Oral Maxillofac Surg 2007;65:369-376

Hyperbare Zuurstof Therapy

- toename van de hoeveelheid beschikbare O_2 in de weefsels
- stimulatie van de angiogenese
- eliminatie van anaërobe kiemen

BRONJ

Conclusion

PREVENTION

Drug-Induced Gingival Overgrowth

Drug-Induced Gingival Overgrowth

1. Introduction
2. Mechanism of action
3. Risk factors
4. Management
    1. Non-surgical treatment
    2. Surgical treatment
5. Conclusions

Drug-Induced Gingival Overgrowth

Introduction

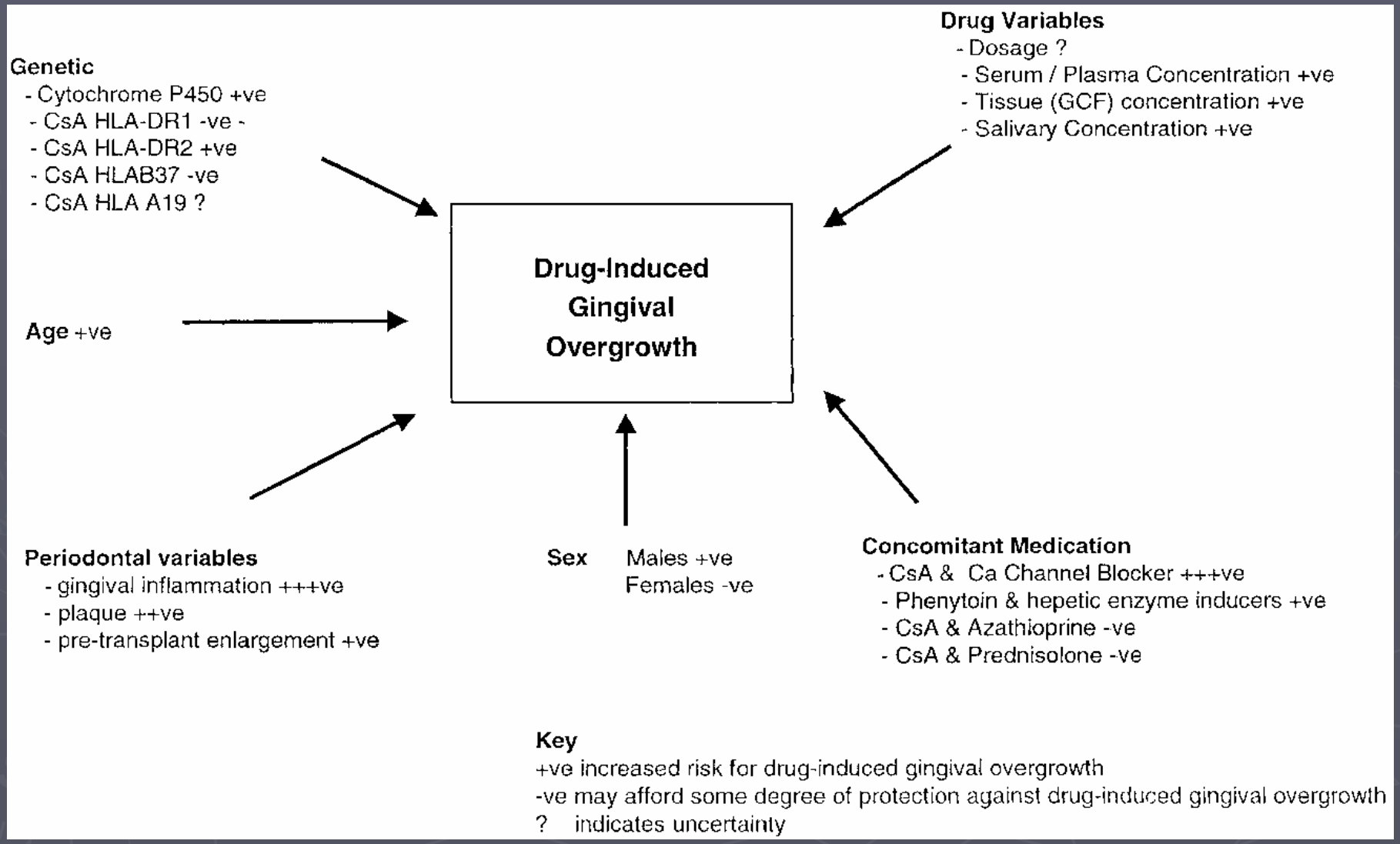
- Three types of Drugs
  - PHENYTOIN (anticonvulsant)
    - Prevalence: 50%
  - CYCLOSPORIN A (immunosuppressive agent)
    - Prevalence: 30%
  - various CALCIUM CHANNEL BLOCKERS
    - Prevalence: 10%

Calcium-antagonists

Drug-Induced Gingival Overgrowth

Risk Factors

- Age
- Gender
- Drug Variabels
  - Controversy
- Concomitant medication
  - Nifedipine + Cyclosporin
- Periodontal variables
  - Plaque + gingivitis
- Genetic factor


Genetic
- Cytochrome P450 +ve
- CsA HLA-DR1 -ve -
- CsA HLA-DR2 +ve
- CsA HLAB37 -ve
- CsA HLA A19 ?
Drug Variables
- Dosage ?
- Serum / Plasma Concentration +ve
- Tissue (GCF) concentration +ve
- Salivary Concentration +ve
Drug-Induced Gingival Overgrowth
Age +ve
Periodontal variables
- gingival inflammation +++ve
- plaque ++ve
- pre-transplant enlargement +ve
Sex Males +ve
Females -ve
Concomitant Medication
- CsA & Ca Channel Blocker +++ve
- Phenytoin & hepetic enzyme inducers +ve
- CsA & Azathioprine -ve
- CsA & Prednisolone -ve
Key
+ve increased risk for drug-induced gingival overgrowth
-ve may afford some degree of protection against drug-induced gingival overgrowth
? indicates uncertainty

Drug-Induced Gingival Overgrowth

Mechanism of Action

- Uncertain
- Previous terms
  - Gingival hypertrophy
  - Gingival hyperplasie

NO increase profileration fibroblasts
BUT accumulation ECM

- Metabolism of collagen
  - Synthesis vs Degradation
  - Degradation
    - Extracellular: secretion collagenase
    - Intracellular: phagocytosis by fibroblasts

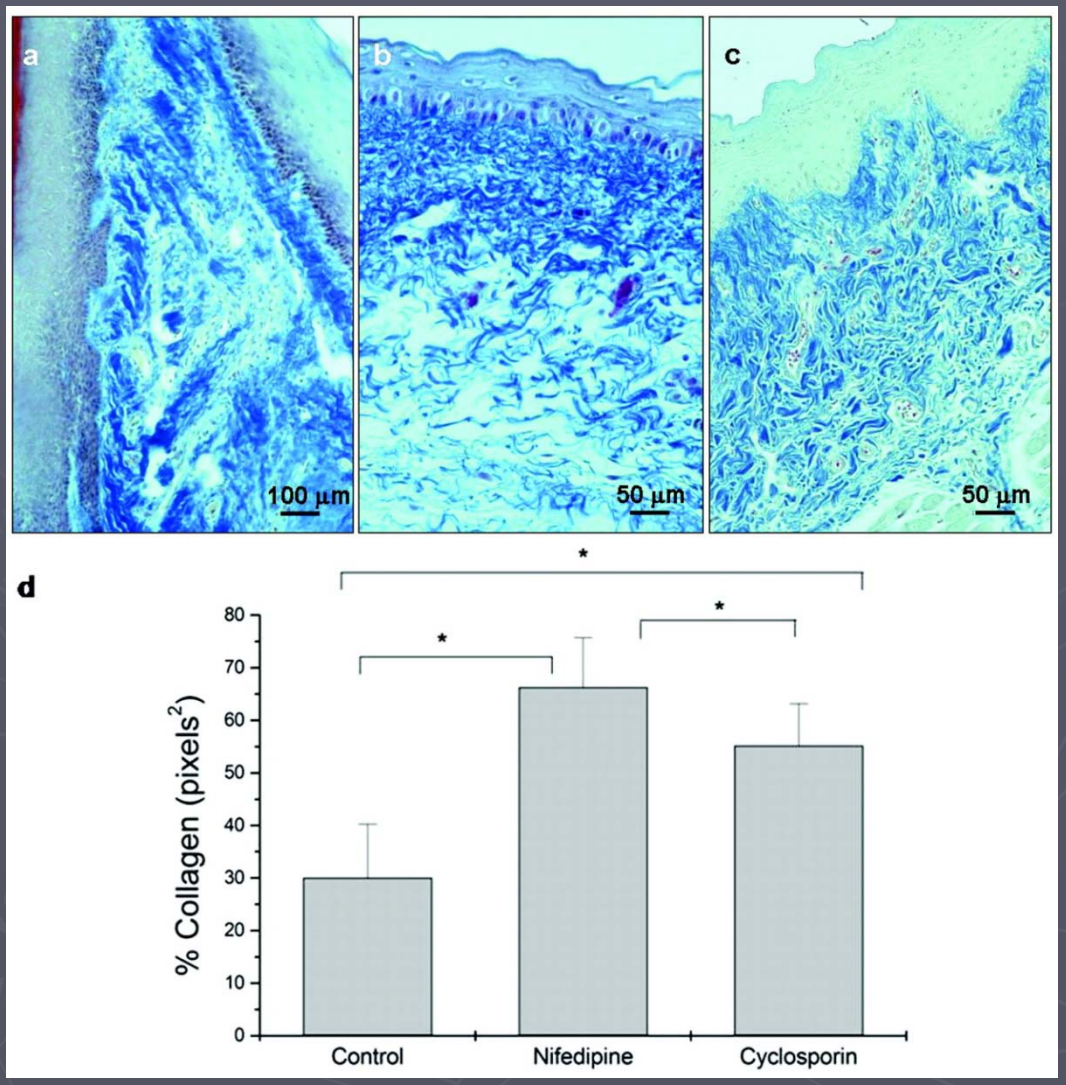
Drug-Induced Gingival Overgrowth

Mechanism of Action

- Role of α2-integrin
  - Integrin = transmembrane receptor
  - 17α - 8β subunits
  - α2 β1 integrin:
    - collagen type I binding to fibroblasts
    - initial binding step of phagocytosis
- expression α2 ★ collagen binding ➡ phagocytosis

Drug-Induced Gingival Overgrowth

Mechanism of Action



Different degree of fibrosis:

a)Control
b)Nifedipine
c)Cyclosporin

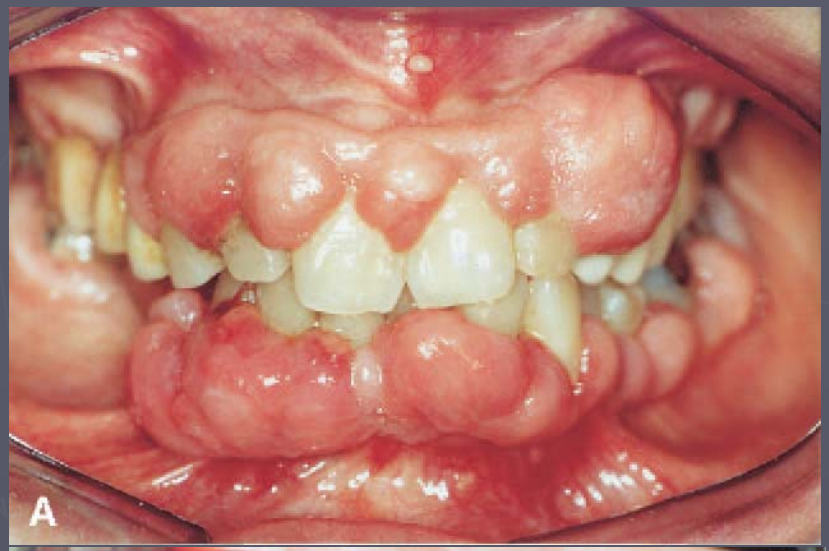
Bullon et al. Nifedipine and Cyclosporin affect Fibroblast Cacium and Gingiva .J Dent Res. 2007;86:357-362

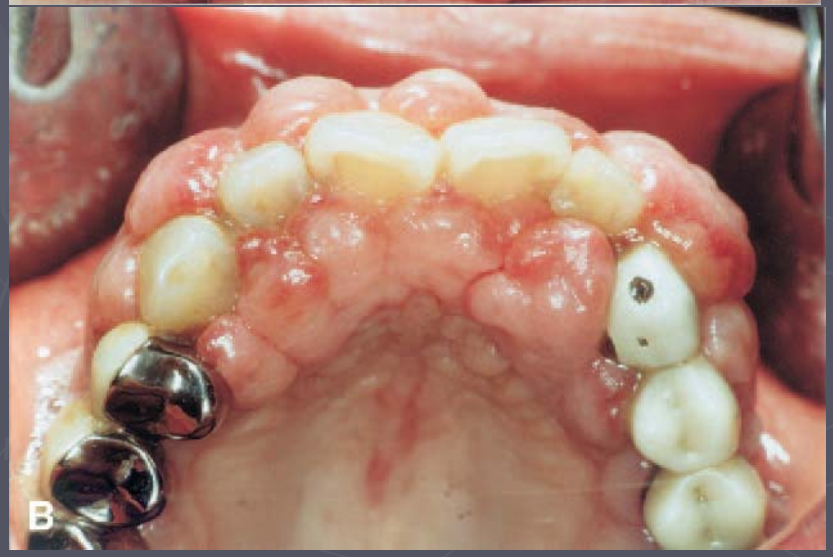
Drug-Induced Gingival Overgrowth
Management

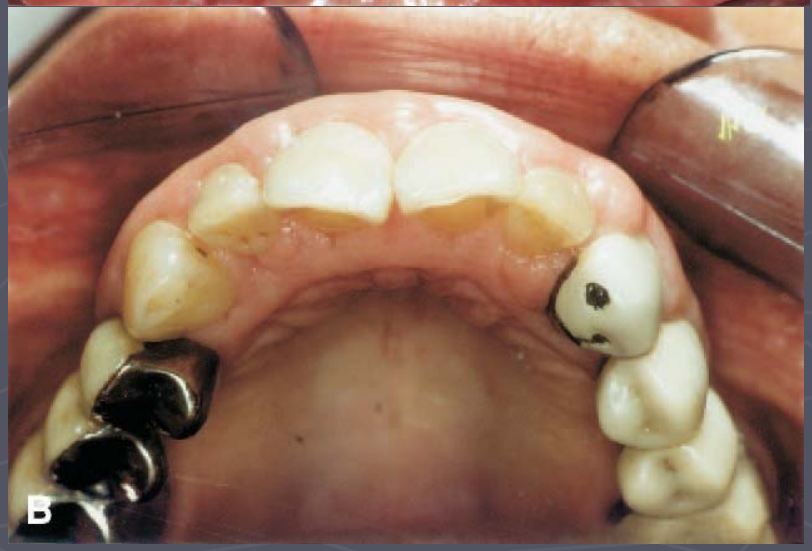
- Non-surgical approach
  - Reduce inflammation
  - Scaling / Rootplaning + oral hygiene instruction
- Antiseptic Mouthwashes
  - CHX: no long-term use
- Systemic Antibiotics
- Other Agents
  - Folic acid mouthwash
- Change in Medication

Drug-Induced Gingival Overgrowth

Management







Drug-Induced Gingival Overgrowth
Management

- Surgical Management
  - Scalpel gingivectomy:
    - long bevel incision
  - Electrosurgery:
    - cave thermal necrosis
  - Laser gingivectomy
  - Flap surgery:
    - overgrowth associated with bone/attachment loss

Drug-Induced Gingival Overgrowth

Management

LASER-gingivectomy

SCALPEL-gingivectomy

Drug-Induced Gingival Overgrowth
Conclusions

- Caused by Ca-antagonists
  - Phenytoin
  - Ca-channel blockers
  - Cyclosporin A
- Major Risk Factors
  - Gingival inflammation, plaque
  - Concommitant medication
- ECM accumulation
  - Decrease collagen degradation?
- Treatment:
  - Non-surgical vs Surgical approach

Mondhygiëne instructies

Hoe moet ik poetsen?

- Borstel 45° gedraaid
- Cirkelvormige bewegingen
- Alle vlakken:
  - Buiten
  - Binnen
  - Boven
- Tong en verhemelte niet vergeten!

Welke tandenborstel gebruik ik?

- Manuele tandenborstel

Welke tandenborstel gebruik ik?

- Electrische tandenborstel

Welke tanpasta gebruik ik?

- Een zachte fluoride houdende tandpasta.

Welkehulpmiddelen zijn er ?

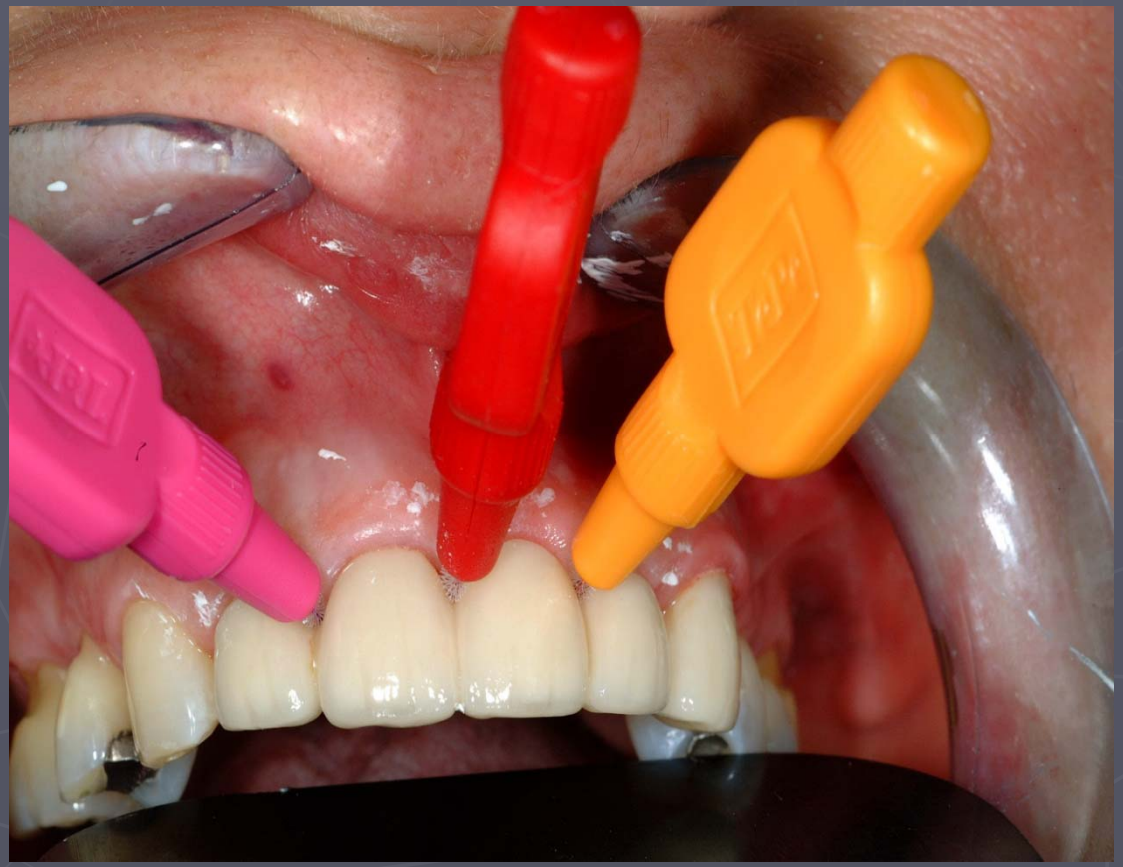
- Floss draad

Welke hulpmiddelen zijn er ?

- Tandenstokers ! Let op geen kaasprikkers

Welke hulpmiddelen zijn er ?

- Ragers



Welke hulpmiddelen zijn er ?

- Mondspoelmiddelen

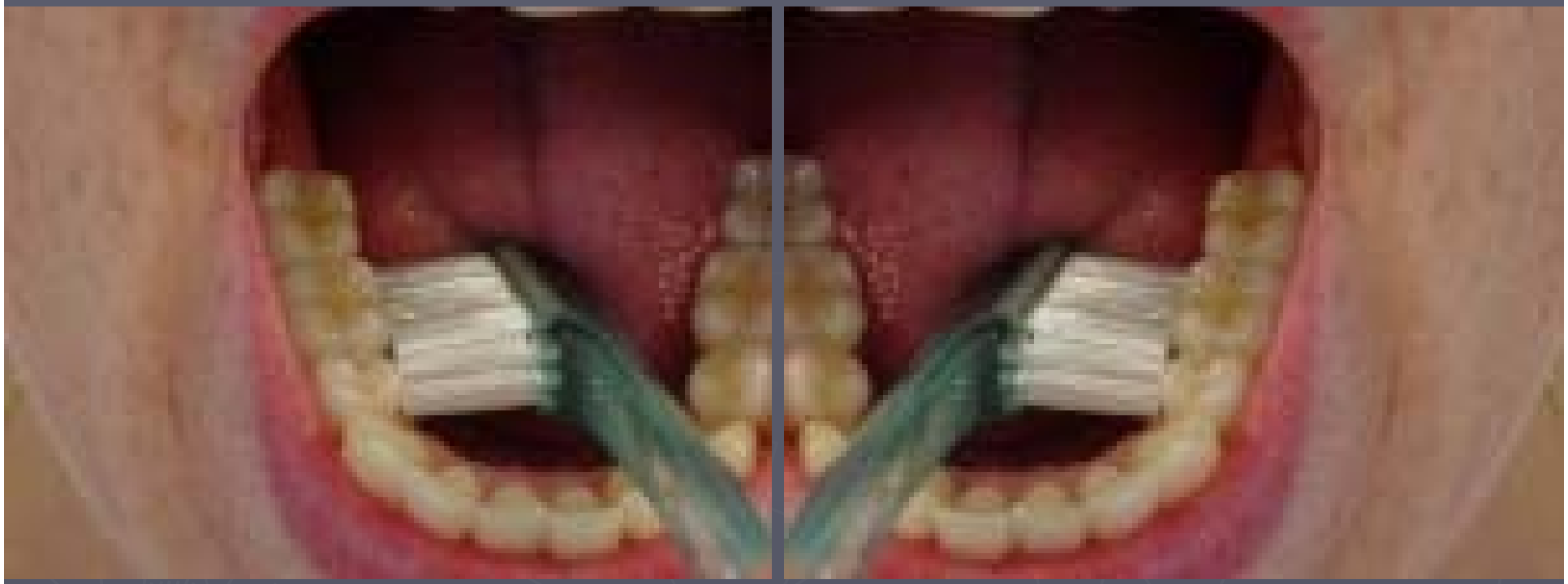
Bedankt voor jullie aandacht!

Poetsmethode

Onder-voorkant
Horizontaal

Boven-voorkant

Horizontaal



Onder-binnenkant kiezen

Horizontaal

Boven-binnenkant kiezen

Horizontaal

Onder-binnenkant voortanden

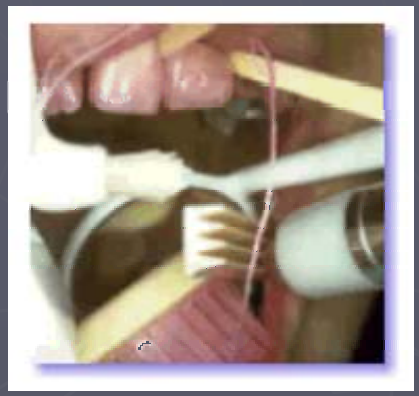
Diagonaal

Boven-binnenkant voortanden
Diagonaal


MAALTANDEN GOED SCHROBBEN

HYGIENE-MIDDELEN

- Het meest efficiente hulpmiddel is datgene wat de patient wil gebruiken
- Keep it simpel
  - 2-3 hulpmiddelen is maximum wat patient aan kan
- Laat patient achteraf voelen wat een schoon gebit is => polijsten



http://www.tandheelkunde.ugent.be
Afdeling parodontologie

HYGIENE-MIDDELEN

Efficient gebruik