Diseases of periodontium.

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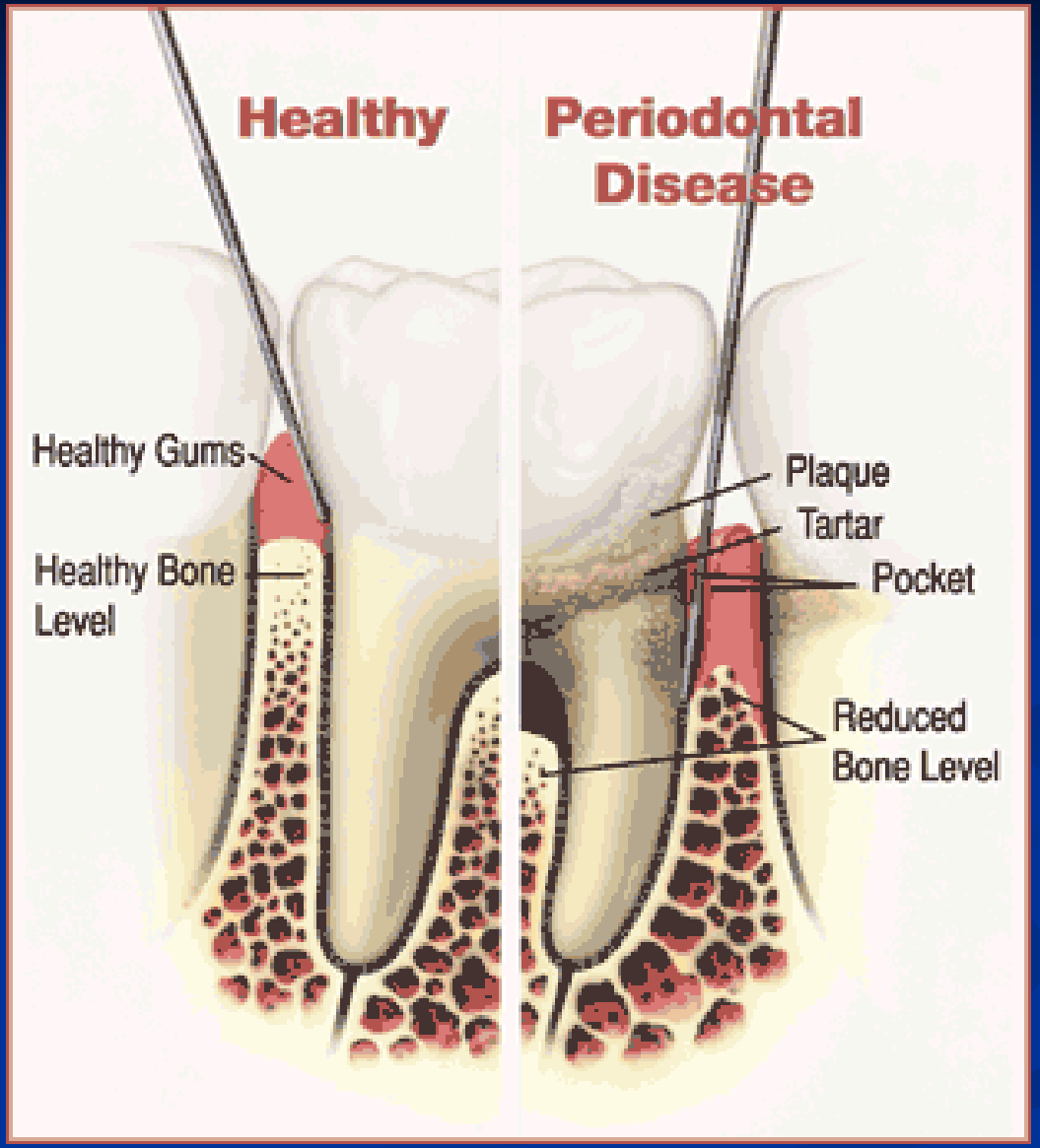
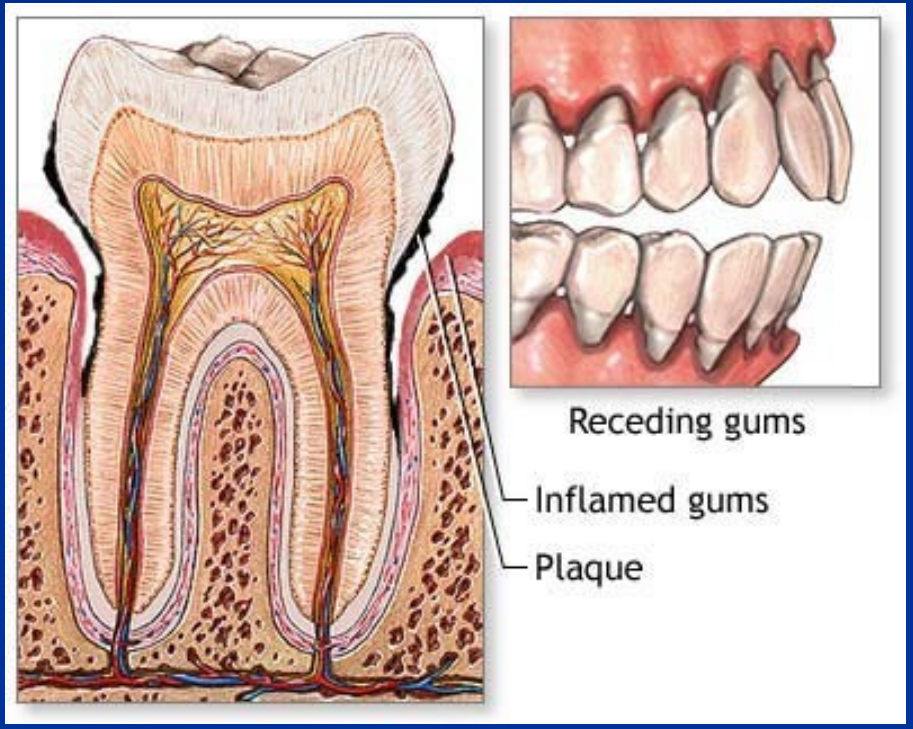
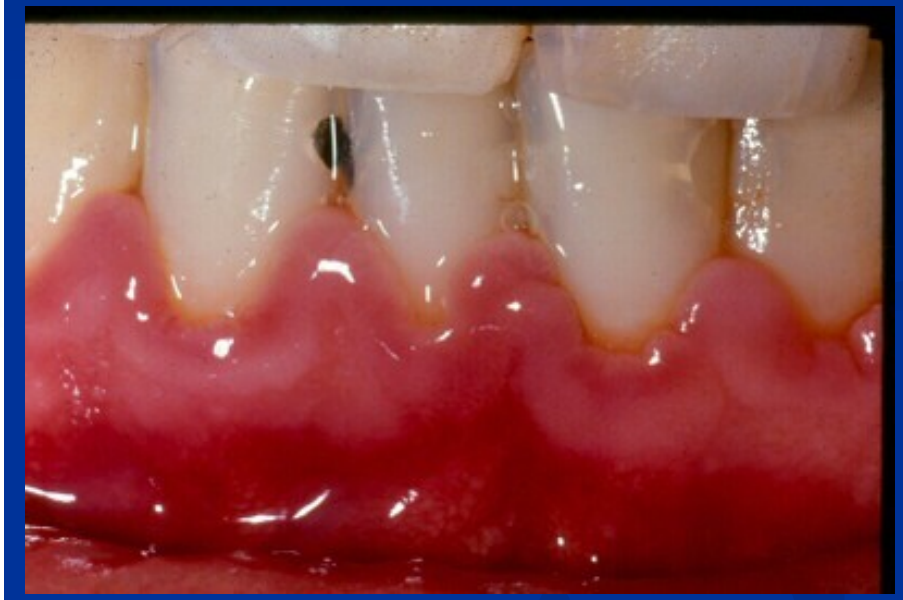

Healthy
Periodontal Disease
Healthy Gums
Healthy Bone Level
Plaque
Tartar
Pocket
Reduced Bone Level

- **Gingivitis:** inflammatory lesions confined to marginal gingiva

- **Periodontitis:** lesions associated with destruction of the connective tissue attachment of the tooth and loss of alveolar bone


Receding gums
Inflamed gums
Plaque



Epidemiology of periodontal disease

- Early periodontitis involves some of the teeth in the majority of adults
- The prevalence of pocketing/loss of attachment increases with age
- The proportion of teeth affected by periodontitis increases with age
- Advanced periodontal disease affects only a small percentage of the population

Classification of plaque-associated periodontal diseases

- **Gingivitis**
  - Associated with dental plaque only
  - Modified by systemic factors
  - Modified by medication
  - Modified by malnutrition
- **Chronic periodontitis**
  - Localised
  - Generalised
- **Aggressive periodontitis**
  - Localised
  - Generalised
- **Periodontitis in systemic diseases**
  - Immunocompromised patients
  - Genetic disorders

Based on International Workshop for a Classification of Periodontal Diseases and Conditions, 1999

PERIODONTITIS: STAGING

Staging intends to classify the severity and extent of a patient's disease based on the measurable amount of destroyed and/or damaged tissue as a result of periodontitis and to assess the specific factors that may attribute to the complexity of long-term case management.

Initial stage should be determined using clinical attachment loss (CAL). If CAL is not available, radiographic bone loss (RBL) should be used. Tooth loss due to periodontitis may modify stage definition. One or more complexity factors may shift the stage to a higher level. See **perio.org/2017wwdc** for additional information.

	Periodontitis	Stage I	Stage II	Stage III	Stage IV
Severity	**Interdental CAL** *(at site of greatest loss)*	1 – 2 mm	3 – 4 mm	≥5 mm	≥5 mm
	RBL	Coronal third (<15%)	Coronal third (15% - 33%)	Extending to middle third of root and beyond	Extending to middle third of root and beyond
	Tooth loss *(due to periodontitis)*	No tooth loss		≤4 teeth	≥5 teeth
Complexity	**Local**	• Max. probing depth ≤4 mm • Mostly horizontal bone loss	• Max. probing depth ≤5 mm • Mostly horizontal bone loss	In addition to Stage II complexity: • Probing depths ≥6 mm • Vertical bone loss ≥3 mm • Furcation involvement Class II or III • Moderate ridge defects	In addition to Stage III complexity: • Need for complex rehabilitation due to: – Masticatory dysfunction – Secondary occlusal trauma (tooth mobility degree ≥2) – Severe ridge defects – Bite collapse, drifting, flaring – < 20 remaining teeth (10 opposing pairs)
Extent and distribution	**Add to stage as descriptor**	For each stage, describe extent as: • Localized (<30% of teeth involved); • Generalized; or • Molar/incisor pattern			

PERIODONTITIS: GRADING

Grading aims to indicate the rate of periodontitis progression, responsiveness to standard therapy, and potential impact on systemic health.

Clinicians should initially assume grade B disease and seek specific evidence to shift to grade A or C.
See **perio.org/2017wwdc** for additional information.

	Progression		Grade A: Slow rate	Grade B: Moderate rate	Grade C: Rapid rate
Primary criteria *Whenever available, direct evidence should be used.*	Direct evidence of progression	Radiographic bone loss or CAL	No loss over 5 years	<2 mm over 5 years	≥2 mm over 5 years
	Indirect evidence of progression	% bone loss / age	<0.25	0.25 to 1.0	>1.0
		Case phenotype	Heavy biofilm deposits with low levels of destruction	Destruction commensurate with biofilm deposits	Destruction exceeds expectations given biofilm deposits; specific clinical patterns suggestive of periods of rapid progression and/or early onset disease
Grade modifiers	Risk factors	Smoking	Non-smoker	<10 cigarettes/day	≥10 cigarettes/day
		Diabetes	Normoglycemic/no diagnosis of diabetes	HbA1c <7.0% in patients with diabetes	HbA1c ≥7.0% in patients with diabetes

The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions was co-presented by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP).

Plaque microorganisms in health, gingivitis, and periodontitis

Main species	% aerobic/ anaerobic	% Gram+/ Gram -	Motile/ non-motile
Healthy gingiva *Streptococcus* *Actinomyces*	75/25	90/10	1:40
Chronic gingivitis *Actinomyces* *Streptococcus* *Porphyromonas* *Prevotella*	60/40	65/35	Number of motile rods and spirochaetes increases with disease
Chronic periodontitis *Actinobacillus* *Porphyromonas* *Bacteroides* *Prevotella* *Fusobacterium*	20/80	25/75	1:1 Abundant motile rods and spirochaetes

Summary: microbiology of periodontal disease

- Gram-positive cocci decrease as gingivitis progresses to periodontitis
- Gram-negative anaerobic bacilli increase as disease progresses
- Motile forms increase as disease progresses
- Periodontal disease involves interactions of mixtures of bacteria forming complexes in plaque
- Certain species (periodontal pathogens) are prevalent in destructive lesions

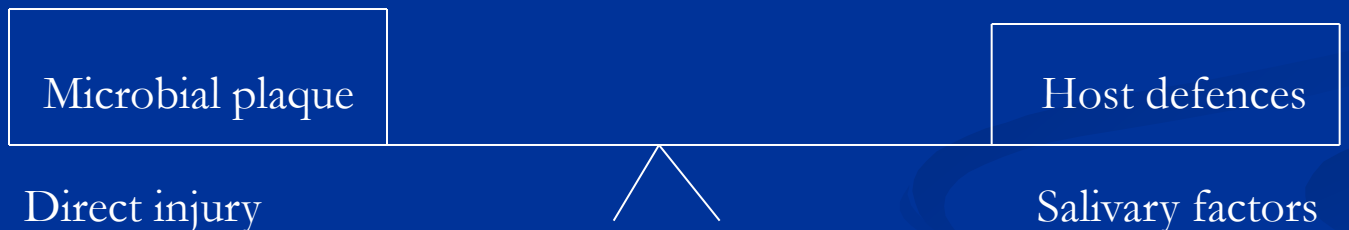
Other risk factors for periodontal diseases

- **Local factors**
- pre-existing anatomy of the teeth, gingiva, and alveolar bone
- alignment and occlusal relationships of teeth
- **Systemic factors**
- Diabetes mellitus
- Pregnancy and sex hormones
- Nutrition (avitaminosis C)
- Blood diseases
- Drugs
- AIDS
- Smoking

Drugs affecting periodontal tissues and the activity of periodontal disease

Anti-epileptics	Phenytoin	Gingival hyperplasia
Immunosuppressants	Azathioprine Corticosteroids Cyclosporin	Equivocal reduction of disease activity Gingival hyperplasia
Non-steroidal anti-inflammatory drugs	Indomethacin Ibuprofen	Equivocal reduction of disease activity
Calcium channel blockers	Nifedipine Verapamil	Gingival hyperplasia
Sex hormones	Oestrogen Progesterone	Exacerbation of pre-existing gingivitis

Host-parasite equilibrium at the plaque-gingival interface: chronic periodontal disease = disturbance of this balance = a dynamic process reflecting changes in the balance of the host-parasite relationship with time



Direct injury
Toxic products
Enzymes
Antigenic challange

Salivary factors
Crevicular fluid
Epithelial barrier
Migrating neutrophils
Immune response
Potential for tissue regeneration and repair

Initial gingivitis

- Microscopic area around base of gingival sulcus
- Acute inflammatory changes
  - Cellular exudate: enhanced migration of neutrophils
  - Fluid exudate: increased crevicular fluid flow
  - Number of chemical mediators of inflammation responsible

Early gingivits

- Lymphocytic infiltration
- Impairment of barrier function of junctional epithelium
- Gingival pocket formation; growth of subgingival plaque

Established gingivitis

- Expansion of area of inflammation and destruction of gingival connective tissue
- Predominance of plasma cells in inflammatory infiltrate
- Deepening of gingival pocket; thining/ulceration of pocket epithelium

Chronic periodontitis

- Apical extension of destructive inflammation
- Loss of connective tissue attachment and destruction of alveolar bone
- Apical migration of junctional epithelium and pocket formation
- Periods of quiscence/stability; random bursts of destructive activity

Degradation of the extracellular matrix (ECM) of gingiva, periodontal ligaments, and the destruction alveolar bone

- Matrix metallo-proteinases (MMPs) degrade ECM
- Tissue inhibitors of metalloproteinases (TIMPs) inhibit MMPs
- Activity of MMPs and TIMPs in balance in health
- Increased MMPs activity in disease; reflects fluctuations in cytokine activity (IL-1)
- Local mediators affecting bone resorption:
  - Cytokines (IL-1, IL-6, TNF)
  - Prostaglandins (PGE_2)
  - Growth factors (e.g. from osteblasts which regulate the osteoclast recruitment)

Pathogenesis of periodontal disease

- Disturbance of host-parasite balance
- Activation of host inflammatory and immune response
- Enhanced synthesis of inflammatory mediators/cytokines
- Periodontal connective tissue degradation/bone resorption
- New equilibrium in host-parasite realtionship as host response contains the challange for plaque bacteria

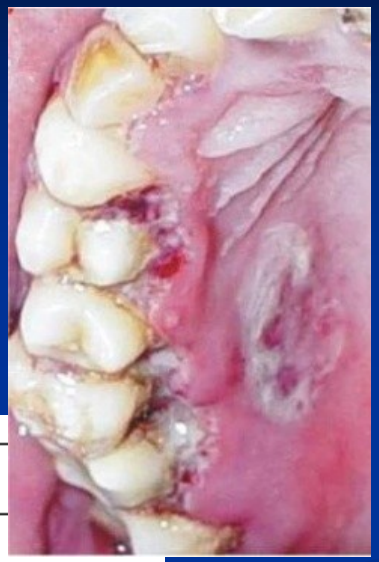
Clinical forms of periodontitis

- Chronic periodontitis
- Aggressive periodontitis
- Periodontitis in systemic disease

Aggressive periodontitis

- Usually juvenile
- F>M
- Rapid destruction of alveolar bone, vertical bone loss, deep intrabony pockets
- First molars and/or maxillary incisors
- Pathogenesis obscure; inflammatory and bacterial plaque??? (G-anaerobic rods *(Actinobacillus actinomycetemcomitans)*, genetic factors, abnormalities in cell-mediated immunity)

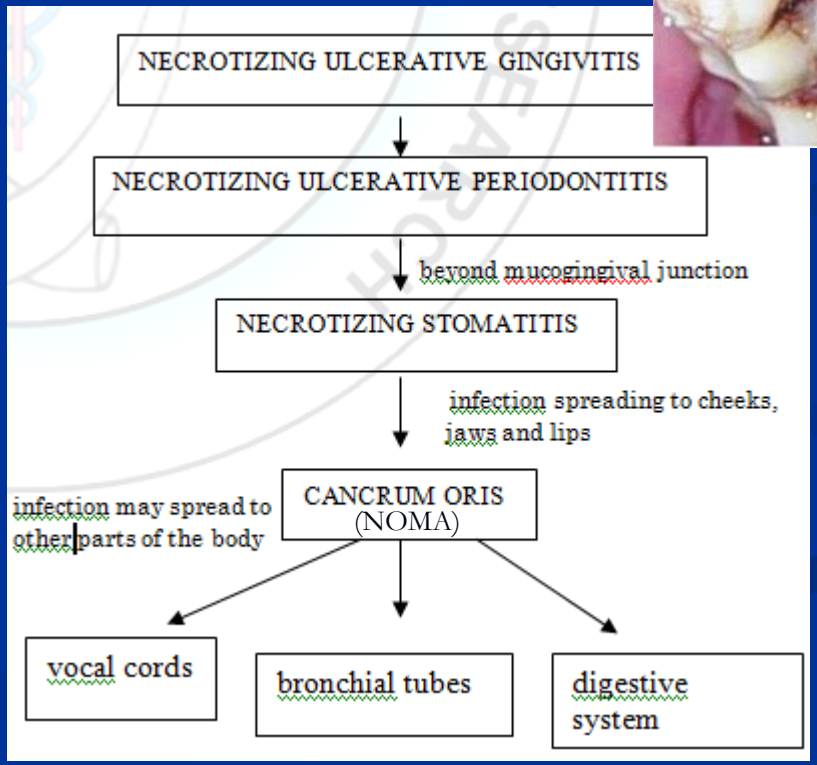
Necrotizing ulcerative gingivitis/parodontitis/stomatitis/noma



- Polymicrobial, endogennous infection: anaerobic bacteria, particularly Fusobacteria and spirochete species (Treponema, Prevotella, Porphyromonas, Selenomonas, Fusonacterium sp)

- Predisposing factors:
  - poor oral hygiene, preexisting gingivits
  - smoking
  - poor nutrition
  - psychological stress
  - weakened immune system, immunodeficiences, dysfunctions of neutrophils

- Malaise, fever, cervical lymphadenopathy



Periodontitis in systemic diseases

- **Diseases associated with major abnormalities of neutrophils**
  - Agranulocytosis
  - Cyclic neutropenia (AD, mutation in the gene for neutrophil elastase)
  - Chediak-Higashi syndrome (AR, mutation in lysosomal trafficing regulatore gene)
  - Job syndrome (hyper IgE syndrome, hereditary)
- **Diseases in which there may be associated neutrophils dysfunctions**
  - Papillon-Lefevre syndrome (palmar and plantar hyperkeratosis, severe periodontal destruction; AR, mutation in lysosomal enzyme cathepsin C gene)
  - Down syndrome
  - Juvenile-onset diabetes mellitus
- **Other systemic diseases**
  - Hypophosphatasia
  - Langerhans cell histiocytosis (histiocytosis X)
  - Ehlers-Danlos syndrome

Gingival enlargement

- **Fibrous overgrowths**
  - Gingival fibromatosis (hereditary, AD)
  - Chronic hyperplastic gingivitis
  - Drug associated hyperplasia (epanutin (anti-epilepticum), verapamil, nifedipin (cardiovascular diseases), cyclosporin (immunosuppressive drug))
- **Oedematous enlargement**
  - Oedematous gingivitis in puberty, pregnancy, oral contraceptives, scurvy (avitaminosis C)
- **Systemic disease**
  - Acute leukaemias
  - Wegener´s granulomatosis

Desquamative gingivitis

- **Gingival manifestation of several different diseases:**
- **Mucous membrane pemphigoid**
- **Lichen planus**
- **Local hypersensitivity reaction**
- **Orofacial granulomatosis** (in Crohn´s disease, sarcoidosis, other causes of granulomatous inflammation (infection, foreign bodies), idiopathic, Melkersson-Rosenthal syndrome (recurring facial paralysis, swelling of the face and lips, and the development of folds and furrows in the tongue), allergic reaction,…)

Thank you for your attention …