Annual Periodontal Review and Part 1 ABP Board Preparation Course
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Disclosure Statement
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- Lexi-Comp- Manual of Clinical Periodontics
- Oral B Guest Lecture Board, P&G Guest Lecture Board
- Maryland Part 1 Board Review Course
- LSU Part 2 Board Review Course
Multifactorial Infections
Current Concepts of Risk
RISK: The likelihood that a person will get a disease in a specified time period.
RISK FACTOR: The characteristics of individuals that place them at increased risk for getting a disease.
Risk factor is defined as “any characteristic, behavior, or an exposure with an association to a particular disease. The relationship is not necessarily causal in nature...”

Garcia, Nunn, Dietrich, 2009
RISK ASSESSMENT: The process of predicting an individual’s probability of getting a disease.
RISK INDICATORS: Probable or putative risk factors that have been identified in cross-sectional studies but not confirmed through longitudinal studies.
Need a susceptible host-risk factors contribute to, but do not directly cause, the initiation or progression of disease.
Risk Assessment - The new paradigm
Assessing Risk

- AAP Risk Assessment Tool- www.perio.org
- Previser- www.previser.com
Periodontal (Gum Disease) Risk and Disease Assessment

Risk of Gum Disease: 3

Risk predicts your future disease state. Your risk is determined by risk factors, which are distinct from the signs and symptoms of disease. Preventing disease requires treatment that reduces your risk factors. With routine dental care, tooth loss is 11 times more likely for an individual who has very high (5) risk compared to an individual who has low (2) risk. However, when risk is used to guide the selection of special treatment, tooth loss can be reduced 90% to 100%.

Your risk score of 3 is reflected against the chart to the left.

Disease State: 13

Localized mild and moderate periodontitis

Your disease state reflects the amount of damage caused by gum disease. As the disease state worsens, treatment increases in amount, complexity and cost. Tooth loss and the failure rate of repairs are greater for individuals with higher disease state scores. Treatment can repair the damage caused by disease, but tends not to help much in preventing new disease. Disease prevention requires treatment that reduces your risk factors. The best treatment incorporates both repair (where needed) and prevention.

Your Score: 13

What Changed? The information below shows the progression of your risk scores:

Characteristics of Health and Low Risk:
- No bleeding during exam or flossing
- X-rays show no bone loss
- No tartar below the gumline
- No fillings are below the gumline
- Not diabetic or under good control
- Bone fully fills fornix
- Minimal or no bacterial plaque
- No pockets deeper than 3 mm
- Not a current smoker
- X-rays show bone level is flat

Your Characteristics:
- No bleeding during exam
- Moderate bone loss
- No tartar below the gumline
- No fillings are below the gumline
- Diabetes is under fair control
- Bone fully fills fornix
- Minimal bacterial plaque
- Deepest pocket is 5-7 mm
- Former smoker
- X-rays show bone level is flat

Analysis:
- Best possible observation, but this could be incorrect and misleading if pockets are deeper than 5 mm
- Significant bone loss has occurred and additional bone loss could cause you to lose a tooth extracted
- Best possible observation, which could be incorrect and misleading for pockets deeper than 5 mm or that bleed
- Best possible condition
- Best possible condition
- Disease management and prevention is slightly compromised
- Pockets is beyond the reach of toothbrush and floss and possibly the dentist's tools
- Best possible situation
- Best possible condition
Characteristics of Health and Low Risk:

<table>
<thead>
<tr>
<th>Your Characteristics:</th>
<th>Analysis:</th>
</tr>
</thead>
<tbody>
<tr>
<td>No bleeding during exam or flossing</td>
<td>Best possible observation, but this could be incorrect and misleading if pockets are deeper than 5 mm.</td>
</tr>
<tr>
<td>X-rays show no bone loss</td>
<td>Moderate bone loss</td>
</tr>
<tr>
<td>No tartar below the gumline</td>
<td>Significant bone loss has occurred and additional bone loss could cause you to have a tooth extracted.</td>
</tr>
<tr>
<td>No fillings are below the gumline</td>
<td>No fillings are below the gumline</td>
</tr>
<tr>
<td>No diabetic or under control for blood pressure</td>
<td>Diabetes is under control</td>
</tr>
<tr>
<td>Bone fully fills function</td>
<td>Bone fully fills function</td>
</tr>
<tr>
<td>Minimal or no bacterial plaque</td>
<td>Minimal bacterial plaque</td>
</tr>
<tr>
<td>No pockets deeper than 5 mm</td>
<td>Deepest pocket is 5-7 mm</td>
</tr>
<tr>
<td>No current smoker</td>
<td>Former smoker</td>
</tr>
<tr>
<td>X-rays show some level of flat</td>
<td>X-rays show some level of flat</td>
</tr>
</tbody>
</table>

Active Intervention You May Need

- Generally most effective
- May be effective
- Less likely to be effective

**REDUCE POCKETS 5–7mm**

Pockets in the 5 to 7 mm depth range cannot be cleaned with a toothbrush and floss, and professional tooth cleaning tools don’t always reach the bottom of the pocket. Incomplete removal of plaque and calculus results in deeper pockets and tooth loss.

- SRP
- Chemo therapeutic (Special Circumstances)
- Gingival Flap Surgery
- Osteosurgical Surgery
- Extraction (Special Circumstances)
- Regenerative Therapy (Special Circumstances)
- Prophy or maintenance

**POCKETS < 5mm**

Pockets that are less than 5 mm deep can be thoroughly cleaned of bacteria and calculus most easily. However, if deep pockets were at one time less than 5 mm, so one should always watch for advancing disease. The optimal pocket depth is 3 mm or less.

- Prophy or maintenance
- SRP
- Chemo therapeutic (Special Circumstances)

Prevention and Maintenance

**VISIT THE DENTIST**

Symptoms are warning signs that frequently appear too late in the disease process for the simplest, most predictable, least costly treatment. Regular visits to detect disease in the early stages can prevent more complex and expensive treatment. Better still are regular visits for preventive care targeted to your risk factors.

- 3 x year
- Two times per year
- Four or more times per year
- less than 2 x year

**ORAL HYGIENE**

Oral Hygiene is of particular importance to you. You should learn improved methods of maintaining your teeth and gums.
Current View of Risk Factors for the Periodontal Diseases
The Pathogenesis Paradigm
Local Risk Factors

- Plaque
- Calculus
- Anatomic factors
- Occlusal factors
- Restorative factors
- Other factors
Plaque a.k.a. Biofilm
Biofilm (plaque) is necessary but not sufficient to produce periodontal inflammation.
Common Periodontal Pathogens

- Aggregatibacter actinomycetemcomitans
- Campylobacter rectus
- Eikenella corrodenens
- Fusobacterium nucleatum
- Peptostreptococcus micros
Common Periodontal Pathogens

- *Prevotella intermedia*
- *Porphyromonas gingivalis*
- *Streptococcus intermedius*
- *Tannerella forsythia (B. forsythus)*
- *Treponema sp.*
Biofilms
Calculus Composition

- Calcium
- Phosphorus
- Carbonate
- Na, Mg, K
- Hydroxyapatite \((\text{Ca}_5(\text{PO}_4)_3 \times \text{OH})\) is major crystal form in mature calculus. Whitlockite is the most common form in subgingival calculus.
- Also has octacalcium phosphate \((\text{Ca}_4\text{H}(\text{PO}_4)_3 \times 2\text{H}_2\text{O})\) whitlockite \((\text{B-Ca}_3(\text{PO}_4)_2)\), and brushite \((\text{CaH}(\text{PO}_4) \times 2\text{H}_2\text{O})\).
Calculus
Mechanisms of Attachment

- Secondary cuticle (organic pellicle that also calcifies).
- Mechanical locking into irregularities in cemental surface.
- Close adaptation of calculus undersurface depressions to unaltered cementum surfaces.
- Bacterial penetration of cementum (not universally accepted).
Calculus

- Mandel- JCP, 1986- Excellent review on calculus. “Subgingival calculus contributes significantly in the chronicity and progression of the disease, even if it can no longer be considered as responsible for initiation.”

Total Calculus
Removal: An Attainable Objective?
Kepic, O’Leary and Kafrawy- J Periodontol
Clinical Significance of Non-surgical Periodontal Therapy: An Evidence-Based Perspective

Furcation Anatomy


- 81% of maxillary and mandibular first molars have furcation entrances ≤ 1 mm.
- 58% of furcation entrances < 0.75 mm.
- Curette widths range from 0.7-1.0 mm.
Furcation Anatomy

Bower, 1979
Furcation Anatomy

Bower, 1979
Furcation Anatomy
Bower, 1979
Cervical Enamel Projections


- Cervical enamel projections were found in > 90% of mandibular isolated furcation involvements.
Cervical Enamel Projections
Restorative Factors

- Subgingival margins
- Overhangs
- Inadequate embrasures
Restorative Factors

- Open margins
- Marginal ridge relationships
- Root surface caries
Overhangs

- **Lang- JCP 1983-** debt-ridden dental students- 1 mm overhangs harbored black-pigmented *Bacteroides* even with good plaque control.

- **Jeffcoat and Howell- JP 1980-** the more severe the periodontal disease, the greater the role of the overhang appeared.

- **Brunsvold- JCP 1990-** at least 25% of restorations and 33% of patients had overhangs….and he was optimistic!!!
Overhangs

- **Pack- JCP 1990** - 56% of restorations had overhangs, 32% of pockets associated with overhangs bled.

- **Jansson- JCP 1994** - “The influence of a marginal overhang on pocket depth and attachment loss decreases with increasing pocket depth.”
Review Article: Detection of localized tooth-related factors that predispose to periodontal infections.

Local Tooth-related Factors

- Molars
  - Furcations
  - Root trunk length
  - Size of furcation entrance
  - Bifurcation ridges
  - Root concavities
  - Cervical enamel projections
  - Premolar concavities
Local Tooth-related Factors

- Overhanging restorations
- Subgingival margins and biologic width
- Restorative materials
- Prostheses- TKPs. Increased mobility, inflammation, and pocketing around abutments.
- Crowding
- Vertical root fractures
- Mucogingival deformities
- Adjacent hopeless teeth
Systemic Disease
Overview-
Risk Assessment in Clinical Practice
Risk Assessment in Clinical Practice

- Behavioral risk factors
  - Smoking
  - Compliance

- Systemic risk factors
  - Diabetes and glycemic control
  - HIV infection
  - Osteoporosis
  - Familial and genetic risk factors
  - Psychological factors
  - Aging
  - Microbiological risk factors
Diabetes and Related Disorders

- Diabetes
  - A metabolic disorder in which the body doesn’t produce or use insulin properly
  - Affects approximately 7% of people in the United States—almost 21 million
  - Sixth leading cause of death among Americans
Diabetes and Related Disorders

- Diabetes
  - Type I diabetes
    - Insulin is not created at all
    - Accounts for about 5% to 10% of diabetes cases
    - Requires daily insulin supplementation
  - Type II diabetes
    - Insulin is produced, but used ineffectively
    - Accounts for 90% to 95% of diabetes cases
    - Occurs most often in people who are overweight
    - May or may not require medication
Potential Pathogenic Mechanisms in Periodontally Related Destruction
Advanced Glycation Endproducts (AGEs)
“Expression of RAGE is enhanced in diabetics. AGE-EC RAGE interaction results in a shift to favor clot formation, increased monolayer permeability, and enhanced expression of VCAM-1 and IL-6. Also see increases in IL-1, IL-8, and TNF-a.” E. Lalla
6 Complications of Diabetes

- Cardiovascular disease
- Kidney disease
- Eye complications
- Neuropathy
- Foot and skin complications
- Periodontal disease - Loe - 1993
Periodontal Disease with Poor Diabetic Control

- Genco Group - Pima Indians which have the highest incidence of Type 2 diabetes, >50% of adults. Found that poorly-controlled diabetics had significantly more attachment loss and bone loss than well-controlled diabetics or non-diabetic controls. Smoking also contributed. Also found that treatment of perio disease may affect metabolic control.
Both Type 1 and Type 2 diabetes mellitus are associated with increased periodontal disease susceptibility. Conventional therapy appears to be effective in diabetics. It has not been demonstrated that chemotherapeutics are necessary for successful treatment in most diabetic patients. The effect of periodontal therapy may not be clinically significant.
Improvement in Glycemic Control with Periodontal Treatment


The best predictor for severe periodontal disease in subjects with T2D is smoking followed by HbA1c levels. T2D subjects should be informed about the increased risk for periodontal disease when suffering from T2D.
**Metabolic Properties of Fat**

- Increases levels of C-reactive protein (CRP), a key inflammatory marker.
- Produces cytokines such as TNF-α, IL-6, and others.
- Has resident macrophages that proliferate as fat increases.
Obesity and Inflammation

- TNF-α
- IL-6
- Plasminogen activator inhibitor-1
- Angiotensinogen
- Vascular endothelial growth factor
- C-reactive peptide
Obesity - now a major risk factor for periodontal disease
Health Complications of Obesity

- Hypertension
- Diabetes
- Abnormal blood fats
- Stroke
- Osteoarthritis
- Sleep apnea
- Cancer
- Gallstones
- Gout
Health Complications of Diabetes

- Cardiovascular disease
- Poor wound healing
- Altered inflammatory response
- Retinopathy
- Neuropathy
- Nephropathy
- Periodontal disease
An Update on HIV and Periodontal Disease

HIV Update

- HAART therapy- highly active antiretroviral therapies- reverse transcriptase inhibitors and protease inhibitors- viral loads may decrease to undetectable levels.
- With HAART, atypical HIV-associated oral lesions have decreased by 30%.
- Necrotizing disease suggests low CD4 counts.
- HIV patients have more AL than healthy patients.
- Periodontal treatment has not changed in 15 years.
Periodontitis Modified by Systemic Factors- HIV Infection

- HIV-infected individuals may exhibit the following:
  - Linear gingival erythema
  - Necrotizing ulcerative gingivitis/
    periodontitis
  - Severe localized periodontitis
  - Severe destructive necrotizing stomatitis
    affecting gingiva and bone

Influence of sex hormones on the periodontium

Primarily estrogens and progestins affect gingival tissues.

See selective overgrowth of *P. intermedia* by substituting estradiol and progesterone for menadione.

Pregnancy gingivitis in 30-100% of pregnant females and 10% develop pyogenic granulomas.

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**Hormonal Influences**

Hormone-Related Periodontal Disease

- Sooriyamoorthy and Gower. JCP 1989;16:201-208. A good review. High counts of *B. intermedius* (*P. intermedia*) have been observed in users of oral contraceptives and in the 2nd trimester. This is due to the competitive binding of progesterone and naphthaquinone, an essential nutrient for bacterial growth. See increased *P. intermedia* counts and pseudopockets, rather than attachment loss.
“There is a plausible pathophysiological basis...stress is associated with more severe periodontal disease.” Has not been definitively proven. Biologic plausibility is the support.
**Review Article**


- A MUST READ!
- Over 20 drugs cause enlargement.
- Prevalence info difficult to get- current thinking- phenytoin 50%, nifedipine 6-15%, CsA 25-30%.
- Enlargement of CT, collagen and ground substance. More fibroblasts controversial.
- Mechanism poorly understood.
- Fibroblasts react to IL-1B.
- Recurrence rate of overgrowth about 40%.
Medications

- Phenytoin
- Nifedipine, other calcium channel blockers
- Cyclosporin A
- Tacrolimus- (Prograf®)- 14% of subjects may have gingival overgrowth. (Sekiguchi, et al. 2007)
Periodontitis Modified by Systemic Factors

Medications - Anti-seizure drugs


Medications- Cyclosporin A

- Acts solely on cell-mediated immunity
- Resembles phenytoin overgrowth clinically and histopathologically, is seen in 30% of recipients, related to serum concentration of drug- Seymour, et al. JCP, 1987.
- Combination of nifedipine and cyclosporin shows greater overgrowth than cyclosporin alone- Ellis, et al. JCP, 1999.
Calcium Channel Blockers and Gingival Overgrowth

- **Dihydropyridines**
  - Nifedipine (Adalat®, Procardia®)- 10-15% often reversible with d/c of drug. Almost 80% of all CCB overgrowth reported cases.
  - Others (amlodipine- Norvasc®)- yes

- **Others- rare overgrowth**
  - Diltiazem (Cardizem®)
  - Verapamil (Calan®)
  - Bepridil (Vascor®)
Medications- Membrane-ion channel blockers

- Calcium-channel blockers influence fibroblasts to overproduce collagen matrix and ground substance when stimulated by gingival inflammation - Ellis, JP, 1999.
- No link with periodontitis
Heritability- the proportion of phenotypic variation within pairs attributed to genetic variation. A heritability estimate of 50% means that one half of the variance in the population is attributable to genetic variance. It does not mean that 50% of offspring of affected parents will be affected. Need large samples of reared together twins to estimate heritability accurately. Heritability estimates for MZ twins is about 50%.
Genetic Approaches

- Michalowicz- JP, 2000- Looked at MZ and DZ twins. Calculated that adult periodontitis had a 50% heritability (half of the variance in disease in the population is attributed to genetic variance)

Among other things, debunks the utility of IL-1 testing. Useful in only 30% of Europeans, 2.3% of Chinese, and low in African-American populations. Very good review.
The Immunology of Aggressive Periodontitis
Hypoinimmune
or
Hyperimmune???
Tobacco Use and Periodontal Disease
Smoking and Periodontal Disease

- Bergstrom, Preber Group- Several studies in JCP from 1987-2005. With other factors controlled for, found smokers had more bone loss than nonsmokers. One study was done in a group of dental hygienists with a high degree of oral hygiene. Also found that smoking affected therapy, causing less probe depth decrease. This was more evident after scaling the maxillary anterior segment. Rate of bone loss slows once a patient stops smoking. More calculus found in smokers- dose dependent.
Evidence for Smoking as an Etiologic Factor in Periodontitis

Criteria for Causation


- Strength of association - cross-sectional and case-controlled studies demonstrate a moderate to strong association between smoking and periodontitis
- Consistency - multiple studies of various designs have demonstrated this association
- Specificity - disease progression slows in patients who stop smoking compared to those who continue to smoke
Evidence for Smoking as an Etiologic Factor in Periodontitis
Criteria for Causation


- Temporality- Longitudinal studies show that smokers do not respond as well to periodontal therapy as non-smokers
- Biologic gradient- there is a dose-response effect in that heavy smokers have increased disease severity compared to light smokers
- Biologic plausibility- supported by tobacco’s negative effects on microbial and host response parameters
Evidence for Smoking as an Etiologic Factor in Periodontitis

Criteria for Causation


- Coherence- the effects of smoking on periodontitis are consistent with our knowledge of the natural history of periodontal disease

- Analogy- periodontal effects of smoking are analogous to other adverse smoking-related general health effects

- Experiment- evidence not currently available
Overlap leads to an increasing susceptibility to periodontitis.
Genco Group
5 Major Factors

- Age
- Smoking*
- Uncontrolled diabetes*
- *Bacteroides forsythus (Tannerella forsythia)*
- *Porphyromonas gingivalis*

**The double whammy!!!**
Periodontitis as a Risk Factor for Systemic Disease
Medical Conditions Related to Periodontal Disease

- Cardiovascular disease
- Pre-term low birth weight infants
- Rheumatoid arthritis
- Diabetes
- Pneumonia
Is it causality, contributory, or coincidence???
Criteria to Accept a Causal Relationship

- Biological plausibility
- Consistency of associations
- Strength of the association
- Temporal consistency
- Specificity of the association
Criteria to Accept a Causal Relationship

- Consistency of the findings
- Dose-response effect
- Support from experimental evidence
Calculating Odds Ratios
For example: If the incidence of oral cancer in a smoking population is 0.002 and in a nonsmoking population is 0.0004, the odds ratio is

\[
\frac{0.002}{0.0004} = 5
\]
Odds ratio $> 1$ indicates a positive association, $< 1$ a negative association, and $= 1$ no association.
When is an odds ratio meaningful?