Instant Update-

Getting Up To Speed in Periodontics for 2019

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Course Synopsis

Some things change and some things remain the same. The bedrocks of periodontal therapy are time-tested but new approaches to some of these therapies are providing better outcomes for patients. In addition, advances in the science of periodontics have led to both a better understanding of the disease processes and a new classification system for the periodontal diseases and conditions. In addition, as implant dentistry continues to solidify its position, complications are becoming more commonplace. This course will focus on four main areas:

- **The changes in science that have led to the new classification of the periodontal diseases and conditions.**
- **Current understanding of the perio-systemic connection.**
- **The "semi-surgical" approach to periodontal therapy.**
- **Peri-implant mucositis and peri-implantitis and what to do about it.**

At the end of this presentation, each participant will be able to:

- **Identify the differences between the 1999 and 2017 disease classification systems.**
- **Identify key factors and systemic diseases that have a strong association with the periodontal diseases.**
- **Develop a "semi-surgical" treatment plan for a patient with periodontitis.**
- **Understand the key factors that contribute to peri-implant disease and possible therapeutic approaches.**

Periodontitis is a disease of the non-mineralized and mineralized connective tissues- What causes and contributes to its breakdown?
Bacterial infections vs. Inflammation

Statistical vs. Clinical Significance

- **Clinical significance- Jacobson, et al. 1984- many definitions- usual criteria for a clinically significant result:**
  - **A change in outcome or a difference in outcome between groups that occurs that is of interest to someone;**
  - **The change or difference between groups must occur in am important outcome; and**
  - **The change or difference must be statistically significant.**
- **Minimal clinically important difference- may vary based on individual clinician.**

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 ASSESSMENT: The process of predicting an individual's probability of getting a disease.

Need a susceptible host-
risk factors contribute to, but do not directly cause, the initiation or progression of disease.

Previser

Persson GR, et al. Analyzing periodontal disease risk: a comparison of clinicians' assessment versus a computerized tool. J Am Dent Assoc 2003;134:575-582. The extent of variation among scores assigned by individual expert clinicians was greater than the authors had expected. Expert clinicians consistently assigned more subjects to PRC risk group 2 and fewer to risk group 5 than did the PRC. The authors observed very high heterogeneity in the risk scores expert clinicians assigned to patients in each of the PRC-assigned groups. Thus, expert clinicians varied greatly in evaluating risk and, relative to the PRC, they appeared to underestimate periodontitis risk, especially for high-risk patients. The authors' observations suggest that use of risk scores generated for individual patients by subjective expert clinician opinion about risk in periodontal clinical decision making could result in the misapplication of treatment for some patients and support the use of an objective tool such as the PRC. Use of the PRC over time may be expected to result in more uniform and accurate periodontal clinical decision making, improved oral health, reduction in the need for complex therapy and reduction in health care costs.

The parameters of care and responsibilities inherent in diagnosis and treatment of periodontal diseases do not differ for general practitioners and specialists."
Cobb, et al. 2003.

Three Phases of Therapy

- Initial therapy
- Advanced therapy
- Supportive therapy- maintenance

Examination> Diagnosis> Treatment Plan

The new- 2017- Periodontal Disease Classification, Staging, and Grading System
www.perio.org/2017wwdc

Main Goals

1. **Update the 1999 Classification**
2. **Create "Case definitions"**
3. **Provide diagnostic criteria to aid clinicians**

Key Changes

1. Chronic Periodontitis is replaced with periodontitis
2. Aggressive Periodontitis is replaced with periodontitis
3. Addition of Staging (severity) AND Grading (rate of progression)
4. The terms mild, moderate and severe have been removed and are replaced with a disease STAGE with respect to periodontitis.
5. Periodontal biotype is replaced with periodontal phenotype
    - ❖ Probe visible: thin (≤ 1mm)
    - ❖ Probe not visible: thick (> 1mm)
6. **Excessive occlusal force is replaced with traumatic occlusal force**
7. **Biologic width is replaced with supracrestal supporting tissues**
8. **Linear gingival erythema as a term is removed**
9. **Suggestion of "incipient periodontitis"**
10. **The term ulcerative has been removed from necrotizing periodontal diseases.**
11. **The Miller Classification for recessions has been replaced with recession types 1-3.**

Case definitions

Gingival health- Less than 10% bleeding sites with probing depths ≤ 3mm

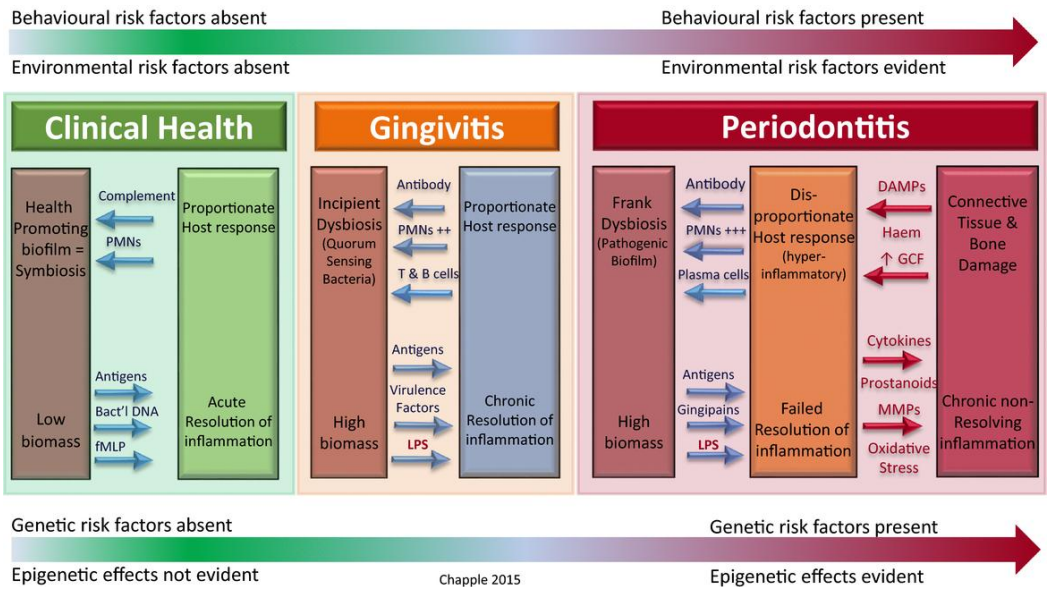
- Epidemiological definition

Characterized by successful treatment through control of local and systemic risk factors, resulting in minimal (< 10% of sites) BOP, no probing depths of 4 mm or greater that bleeding on probing, optimal improvement in other clinical parameters and lack of progressive periodontal destruction

- Clinical practice definition

Gingivitis- **≥10% bleeding sites with probing depths ≤ 3mm**

- **Epidemiological definition**
- **Localized is defined as 10% - 30% bleeding sites**
- **Generalized is defined as > 30% bleeding sites**
- **In clinical practice we should refer to the gingivitis look-up table to determine if we have a gingivitis case.**



Periodontitis- **Interdental CAL is detectable at ≥ 2 non-adjacent teeth, OR**

- Buccal or lingual CAL ≥ 3 mm with pocketing ≥ 3 mm is detectable at ≥ 2 teeth but the observed CAL cannot be ascribed to non-periodontitis-related causes such as :
    - 1. Gingival recession of traumatic origin
    - 2. Dental caries extending in the cervical area of the tooth
    - 3. Presence of CAL on the distal aspect of a second molar and associated with malposition or extraction of a third molar
    - 4. An endodontic lesion draining through the marginal periodontium
    - 5. The occurrence of a vertical root fracture

Three Steps to Staging and Grading a Patient

Step 1: Initial Case Overview to Assess Disease

Screen:
- Full mouth probing depths
- Full mouth radiographs
- Missing teeth

Mild to moderate periodontitis will typically be either Stage I or Stage II

Severe to very severe periodontitis will typically be either Stage III or Stage IV

Step 2: Establish Stage

For mild to moderate periodontitis (typically Stage I or Stage II):
- Confirm clinical attachment loss (CAL)
- Rule out non-periodontitis causes of CAL (e.g., cervical restorations or caries, root fractures, CAL due to traumatic causes)
- Determine maximum CAL or radiographic bone loss (RBL)
- Confirm RBL patterns

For moderate to severe periodontitis (typically Stage III or Stage IV):
- Determine maximum CAL or RBL
- Confirm RBL patterns
- Assess tooth loss due to periodontitis
- Evaluate case complexity factors (e.g., severe CAL frequency, surgical challenges)

Step 3: Establish Grade

- Calculate RBL (% of root length x 100) divided by age
- Assess risk factors (e.g., smoking, diabetes)
- Measure response to scaling and root planing and plaque control
- Assess expected rate of bone loss
- Conduct detailed risk assessment
- Account for medical and systemic inflammatory considerations

Stage descriptions drawn from Tonetti, Greenwell, Kornman. *J Periodontol* 2018;89 (Suppl 1): S159-S172.

Staging and Grading Periodontitis

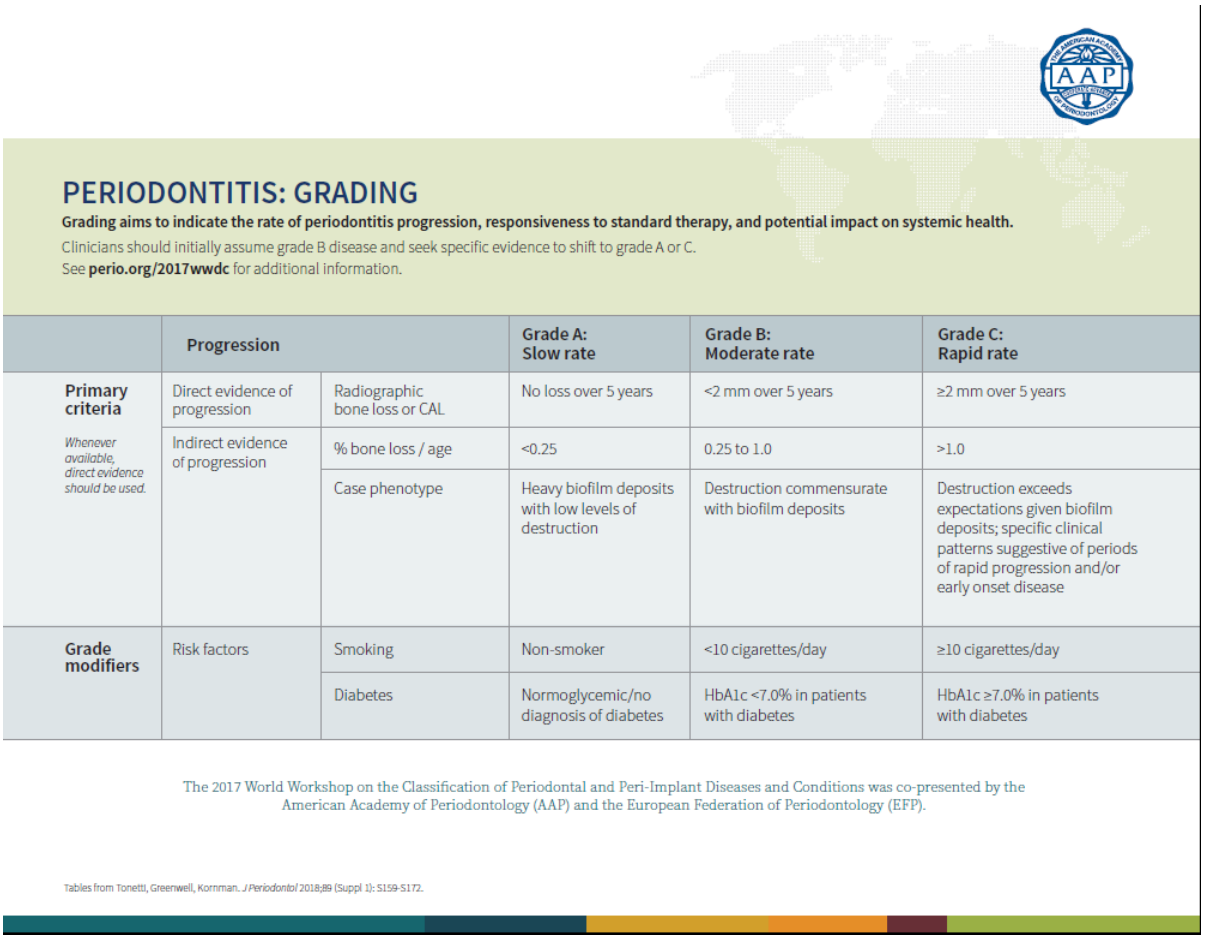
The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions resulted in a new classification of periodontitis characterized by a multidimensional staging and grading system. The charts below provide an overview. Please visit **perio.org/2017wwdc** for the complete suite of reviews, case definition papers, and consensus reports.

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	Direct evidence of progression	Radiographic bone loss or CAL	No loss over 5 years	<2 mm over 5 years	≥2 mm over 5 years
Whenever available, direct evidence should be used.	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).

Tables from Tonetti, Greenwell, Kornman. *J Periodontol* 2018;89 (Suppl 1): S159-S172.

Avenues to Success

- Plaque control
- Scaling and root planing
- Antimicrobials/antibiotics
- Occlusal therapy/ splinting
- Orthodontics
- Gingivectomy/ gingivoplasty
- Open flap debridement
- APF +/- osseous surgery
- Root resection
- Bone replacement grafts
- Guided tissue regeneration
- Guided bone regeneration
- Mucogingival surgery
- Periodontal plastic surgery
- Implants

- Supportive and adjunctive treatment

Local risk factors

- Plaque/ Biofilm
- Calculus
- Anatomic factors
- Occlusal factors
- Restorative factors
- Other factors

Biofilm is necessary but not sufficient to produce periodontal inflammation.

Common Periodontal Pathogens

- Aggregatibacter actinomycetemcomitans
- Campylobacter rectus
- Eubacterium nodatum
- Fusobacterium nucleatum
- Herpesvirus
- Peptostreptococcus micros
- Prevotella intermedia/nigrescens
- Porphyromonas gingivalis*
- Streptococcus intermedius
- Tannerella forsythia*
- Treponema denticola*
- Current list of suspects includes 20-30 bacteria
- * late colonizers- red complex
- Newer bacteria are being identified with advancements in microbiological techniques

Risk Assessment in Clinical Practice- Ronderos M, Ryder MI. Periodontol 2000, 2004;34:120-135.

- 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 periodontal disease

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

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.

Improvement in glycemic control with periodontal treatment

- Grossi, et al.- JP, 1997. Found significant reductions in glycosylated hemoglobin in all groups receiving antibiotics as part of periodontal therapy.
- Kiran M, et al. JCP, 2005. Initial therapy significantly decreased HbA1c levels in patients with periodontal disease.

- **Average improvement of A1c is in the 0.4% range. This is an improvement of the A1c value itself. Darre, et al. 2008; Simpson, et al. 2010, Cochrane; Teeuw, et al. 2010.**
- **Anecdotal reports state improvements up to 1%.**
- **Engebretson, et al. JAMA December, 2013 state that there is no improvement. Was study done to protocol or to clinical results?**

Metabolic Syndrome- at least three of the following 5 factors:

- Visceral (central) obesity
- Dyslipidemia
- Hyperglycemia
- Hypertension
- Low serum high density lipoprotein (HDL)
- Helps to identify persons at high risk for type 2 DM and CVD
- In the US, about 25% of the population has MS

Metabolic properties of fat

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

Etiology/potential risk factors/risk indicators for periodontal diseases

1. Biofilm as the primary risk factor/ etiology
2. Genetics- host response
3. Smoking
4. Viruses and other microorganisms
5. Inflammatory mediators- local and systemic
6. History of prior periodontal disease
7. Compromised host defense / systemic factors- obesity, diabetes as prominent factors
8. Race, ethnicity
9. Aging
10. Gender

11. Stress
12. Faulty dental care
13. Other local factor

Genco Group- 5 major risk factors for periodontal disease

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

***The double whammy!!!*

Hill's Criteria to Accept a Causal Relationshp

- Biologic plausibility
- Dose-response effect
- Temporal consistency
- Consistency of the findings
- Strength of the association- correlation value. Higher correlation, more likely a causal effect.

- **Specificity of the association- a single putative cause produces a specific effect. Absence of specificity does not negate a causal effect.**
- **Coherence- the association is compatible with current theory and knowledge.**

Odds Ratio

Alzheimer's and periodontal disease- P gingivalis. Gingipains. Small molecules from bacteria affect neurons

Keys to successful non-surgical therapy

- Time to do everything right the first time
- Examination
    - Medical
    - Hard and soft tissue
    - Periodontal
- Comprehensive diagnoses and treatment plan
- Understanding the basic biologic principles of pathogenesis and wound healing

- **Disruption of biofilm**
    - **Professionally**
    - **Daily**
- **Removal of biofilm retentive factors**
    - **Calculus**
    - **Overhangs, open contacts, other factors**
- **Sufficient time to heal and re-examination**

Bleeding on probing or lack thereof is still the major indicator of health or disease

Cause-related therapy

- Personal plaque control
    - Mechanical
    - Chemical
- Scaling and root planing
    - Powered instruments
    - Hand instrumentation
- Calculus detection
    - Tactile
    - Visual
    - Electronic
- Lasers in non-surgical therapy

Basic principles

- Treat early and aggressively.
    - What is conservative treatment?
    - IMHO- conservation of the *natural* teeth in health and comfortable function with good aesthetics for the life of the patient.
- Appropriate follow up and re-evaluation.
- Be realistic of what you can accomplish in your practice.

- **Be fair to the patient.**
    - **Do not use up their perio benefits if you know that you are going to refer anyway.**
- **Refer early- you have periodontists in your network.**
- **Watching that 5 mm probing go to 7 mm may be supervised neglect, but it is still neglect.**
- **Proper sequencing-**
    - **Do what the patient needs.**
    - **Remove calculus before doing interproximal restorations.**
    - **Can stagger restorative and periodontal treatment.**
    - **Cannot do all four quadrants of 4341 at the same time, but you can cluster scaling and extractions.**

Third molar extraction considerations

- Fully erupted?
- In occlusion?
- Teeth anterior to it compromised, especially in the mandible?
- Compromising periodontal health of the adjacent molar?
- Supraerupting?
- Causing an uncorrectable occlusal interference?
- Only remaining stable vertical stops?
- Distal abutment for RPD

Reasons to consider systemic antibiotics

- Patient has recurrent disease and previously has been treated surgically.
- Patient states that they are absolutely not interested in surgical treatment.
- Patient is not in a financial position to be treated surgically.
- Pushing the ‘‘day of reckoning’’ as far into the future as possible.
- Clinical manifestations of disease warrant adjunctive means as part of initial therapy.
    - **In general, amoxicillin and metronidazole are the agents of choice.**
    - **High dose, short period of time.**
    - **Must combine with mechanical disruption of biofilm.**
    - **Focus on therapeutic rather than prophylactic use.**

Recommended antibiotic regimens

- For combinations of anaerobic and facultative pathogens- metro + amoxicillin (Augmentin®) or metro + Cipro
- For *Aggregatibacter actinomycetemcomitans*- metro + amoxicillin (Augmentin®) or Cipro (adults only) or Cipro + metro.
- For anaerobic pathogens- metro + amoxicillin (Augmentin®), Augmentin® alone, metro, or clindamycin.
- For enteric rods, *Pseudomonads*- Cipro + metro

Serio recommendation- May want to consider amox 500 mg + metro 500 mg tid for 10 days.

Local antibiotics

- Actisite®- tetracycline fiber (no longer available)
- Atridox®- doxycycline gel- available from Denmat- www.denmat.com.
- PerioChip®- chlorhexidine chip- available from www.periochip.com.
- Arestin®- minocycline microspheres- available from OraPharma, www.arestin.com.

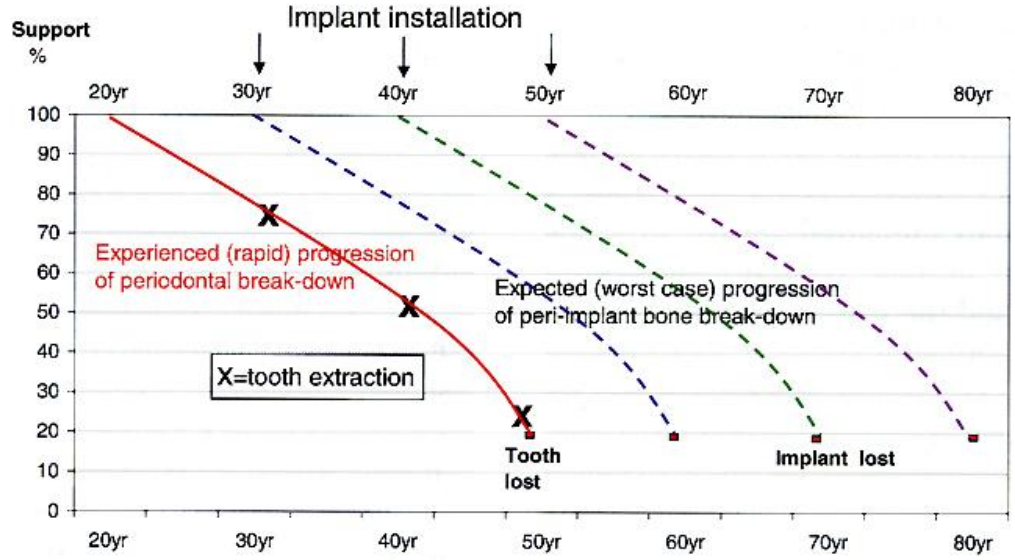
Semi-surgical therapy

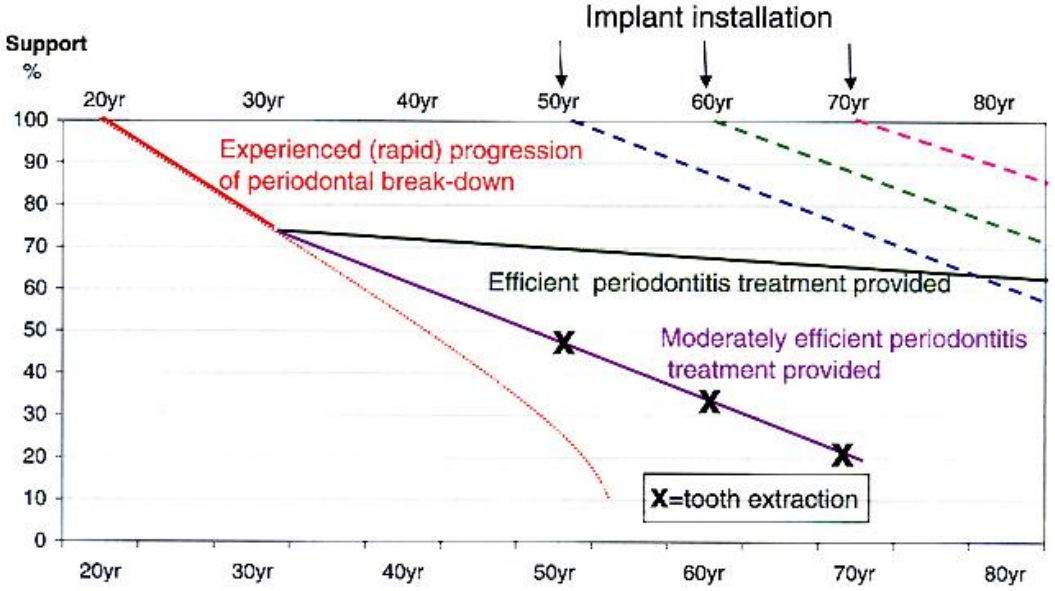
- The concept of ‘‘semi-surgical’’ therapy.
- Often done in patients who have never had scaling and root planing.
- Lots of local factors.
- Usually moderate to severe inflammation.
- Age related.
- Vigorous scaling and root planing with papillae removal.
- Subsequent use of systemic antibiotics.

Problems with Implants: Complications, Peri-implant mucositis, and Peri-implantitis

- ‘‘Atraumatic’’ extractions.
- Various approaches
    - The Zen of tooth removal- ‘‘See the tooth, feel the tooth, deliver the tooth.’’
    - Apical pressure and rotation only.

- Section multi-rooted teeth.
- Remove palatal bone to create space.
- Specialized instrumentation.





Definitions

- Peri-implant health- characterized by the absence of erythema, bleeding on probing, swelling, and suppuration. It is not possible to define a range of probing depths compatible with health. Peri-implant health can exist around implants with reduced bone support.
  - **Peri-implant mucositis: inflammation is confined to the soft tissues with no sign of supporting bone loss following initial bone remodeling during healing. Bleeding on gentle probing is the primary clinical characteristic. Erythema,**

swelling, and/or suppuration may be present. Biofilm is the primary etiological factor. Assumed to precede peri-implantitis.

- **Peri-implantitis: plaque-associated inflammation around the implant that includes both soft and hard tissue. The clinical signs of inflammation include bleeding on probing, and/or suppuration, increased probing depth, possible recession, and radiographic bone loss. May lead to severe bone destruction and implant loss.**
- **Hard- and soft-tissue deficiencies- refers to deficiencies at potential implant sites. Hard tissue deficiencies may be affected by periodontitis, endodontic infections, thin buccal plates, pneumatization of the maxillary sinus, agenesis of teeth, and prosthetic damage.**
- **Soft-tissue deficiencies relate to the amount of keratinized tissue and the location of the implant in relation to that tissue. While it is generally thought that a zone of KG is beneficial around implants, there is no good evidence supporting this position and implants have survived quite well surrounded by mucosa.**

Peri-implant mucositis

- Increased crevicular fluid earliest sign but not clinically practical.
- Bleeding on probing and/or suppuration
- Probe depths $\geq$ 4 mm.
- No radiographic bone loss after remodeling.
- Prevalence- In 80% of subjects and 48% of long term implants without bone loss (9-14 years). Other studies suggest up to 90% of implants affected.
- Reversible with non-surgical intervention- resolution may take longer than 3 weeks.
- Similar histology around teeth and implants.

Peri-implantitis

- Bleeding and/or suppuration.
- Significant bone loss in addition to remodeling.
- May see exposure of implant surface or threads.
- % radiographic bone loss may not be related to mobility.
- May progress more rapidly from peri-implant mucositis than periodontitis does from gingivitis.
- Bone destruction may pick up speed as lesion worsens.

- **Apical extension of inflammatory cell infiltrate more pronounced in peri-implantitis.**
- **More PMNs and macrophages in peri-implantitis lesions.**
- **Inflammatory cell infiltrate may not have epithelial covering at implant interface.**
- **Prevalence and incidence data is all over the map. Numbers range from about 7% of implants to 40% of implants over 7-10 year period. Differences based on varying definitions of implantitis. Same issue with defining periodontitis.**

Etiology and pathogenesis

- Many of the same bacteria but other forms may be influenced by the different environment, similar histological appearance except for encapsulation of inflammation.
- With newer technologies, other bacteria are being implicated.
- Biofilm formation- similar Gram- anaerobic bacteria.
- Same JE attachment to teeth and implants.
- Changes in connective tissues- less vascular, increased collagen:fibroblast ratio.
- Less calculus formation on implants.

- **Peri-implantitis- usual suspects (*P. gingivalis, T. forsythia, T. denticola*, others) along with possibly *S. aureus* and *Peptostreptococcus micra.***
- **Microbiota different around implants in patients with teeth and those who are edentulous. Identical or fraternal?**
- **Peri-implantitis may proceed more rapidly than periodontitis.**
- **No potential for self-limiting of the inflammation around implants. Do not see the connective tissue capsule around implant inflammation as is seen between inflammation and bone around teeth.**
- **No evidence that the host response differs around teeth or implants.**
- **Rough surfaces worse, although only a few studies suggest this.**

Risk factors

- Previous or existing periodontal disease.
- Poor OH, inability to clean, lack of compliance. Poor prosthetic design is a major factor.
- Smoking.
- Poor bone quality.
- Bone overheating during implant placement.
- Loss of bone due to flap reflection.
- **Genetic factors- no consensus on the role of IL-1 polymorphism. Garcia-Delaney suggests no role.**
- **Diabetes- better glycemic control, less chance of problems.**
- **Occlusal overload- the force has to go somewhere- overloaded bone, fractured implants, abutments, restorations, screws.**
- **Occlusal indicator wax.**
- **Premature loading.**
- **Excess cement remnants.**
- **Inadequate restoration-abutment seating.**
- **Improper contouring of restorations.**
- **Implant mal-positioning.**
- **Improperly located in bone.**
- **Implants too close together- minimally 3 mm apart surface to surface.**
- **Implant too close to adjacent tooth- minimally 2 mm apart surface to surface.**
- **Titanium particles in the soft tissue**

Diagnosis of ailing implants

- Group I- Success. No pain, mobility, or exudate and less than 2 mm radiographic bone loss.
- Group II- Satisfactory survival. No pain, mobility, or exudate and 2-4 mm bone loss.
- Group III- Compromised survival. No mobility, bone loss > 4 mm but less than half implant length, > 7 mm probings.
- Group IV- Failure. Pain, mobility, exudate, bone loss > half implant length.

Cluster failures of implants

- Usually occurs soon after implant placement.
- Genetic factors.
- Systemic factors- allergies?
- Heavy smoking.
- Heavy occlusion.
- Unusual biofilm.

Occlusion and implants

- Implants are, in effect, ankylosed teeth.
- Behave differently under occlusal load.
- Prefer long axis loading.
- How do you check when you have a mixed occlusion of natural teeth and implants?
- Must ensure that implants are not inadvertently overloaded.
- One suggestion- Kerr Occlusal Indicator Wax

Non-surgical approach to treat peri-implant mucositis

- Mechanical disruption of biofilm.
- Possible use of systemic antibiotics????
- Daily antimicrobial rinsing, syringe or WaterPik.
- Removing biofilm retentive features of the prosthesis.
- More frequent recare visits.
- Must be relentless about controlling inflammation.
- Compliance hovers at 50%. Better compliance- better success (Lagarvall and Jansson, J Periodontol, 2012)

Treatment of peri-implantitis

- Generally, nonsurgical therapy is not effective in treating peri-implantitis. May be able to improve soft tissue inflammation profile but there are no changes in bone or disease resolution.
- Surgical therapy is necessary but there are no generally agreed upon approaches to implant surface decontamination or surgical approaches.
- Several surgical approaches (case reports) will be presented.
- Many different materials have been used successfully for regeneration. Clinician's choice.

- **No one generally accepted recipe for implant decontamination although it seems that sterile saline works as well as anything else (Wilson and others) .**

- **No comparative clinical trials exist.**
- **Can use resective or regenerative approaches.**
- **Part of the approach is often to regenerate lost bone when feasible.**
- **How does laser therapy fit into peri-implantitis treatment?**