CE Course Handout

Evidence-Based Periodontal Therapy - Conquering the Unseen Enemy

Thursday, June 18, 2015
9:30am-12:30pm
“Evidence-Based Periodontal Strategies – Conquering the Unseen Enemy”

A Continuing Education Course for Dental Professionals
(half-day format)

Presented by
Dianne Glasscoe Watterson, RDH, BS, MBA

2825 Roderick Road, Frederick, MD  21704 (301) 874-5240 fax and office
Email – dglasscoe@northstate.net  ♦ Web – www.professionaldentalmgmt.com

© 2015. All rights reserved. No part of this book may be reproduced, stored in a retrieval system, or transcribed in any form without the prior written permission of Dianne Glasscoe Watterson.
Course Description

Periodontal diseases are multi-factorial, chronic infections that cause destruction of periodontal structures and influence many non-oral disease processes in the body. Treating periodontal diseases successfully involves understanding how and why the disease progresses and developing customized strategies that are aimed at disease control. This course examines the latest evidence-based information related to patient care protocol, including (but not limited to) pathogenicity, disease progression considerations, oral irrigation considerations, and home care modalities.

Course Objectives

Upon completion of this course, the attendee should be able to:

- Understand the role of the host and microbes in disease etiology.
- Implement evidence-based treatment strategies.
- Implement effective sequencing protocol for the periodontal patient.
- Understand the role of inflammation.
- Differentiate between antimicrobial irrigants.
- Discuss factors related to disease progression.
Pre-Test
Facts about Plaque and Periodontal Disease

True or False

(1) Supragingival plaque causes gingivitis but does NOT cause periodontitis T F
(2) Calculus deposits cause periodontitis. T F
(3) Once the dental papilla recedes, dental floss becomes increasingly ineffective. T F
(4) Toothbrushes do not reach more than 1-2mm and rarely 3mm into a pocket. T F
(5) Mouthwashes are effective in reducing gingivitis and periodontitis. T F
(6) Periodontal infection begins in shallow sulci. T F
(7) Periodontal disease occurs in episodes of activity and quiescence. T F
(8) Supragingival plaque control is the most important factor in stopping established periodontal disease. T F
(9) Periodontal pockets have been shown to re-populate to previously scaled levels within nine to eleven weeks following treatment. T F
(10) Plaque can be removed with means other than mechanical friction. T F
(11) Pre-procedural rinsing has been shown to reduce airborne pathogens by 94%. T F
(12) The top risk factor for periodontal disease is substandard oral hygiene. T F
(13) Tissue destruction associated with periodontal disease is caused solely by the action of pathogenic microbes. T F
(14) Probing depth cannot reveal a pathogenic infection or predict future attachment loss. T F
(15) Microbes known to be associated with periodontal disease include viruses, yeasts, and fungi. T F
(16) The microbes that initiate periodontal disease prefer calculus over cementum. T F
(17) The goal of periodontal therapy is to have no pockets over 3mm and no bleeding on probing. T F
(18) Bleeding on probing is an absolute sign of active periodontal disease. T F
(19) Hand scaling is superior to power scaling. T F
(20) Systemic antibiotics are not beneficial as adjuncts to treatment for chronic periodontal disease. T F
1. Infectious, chronic, inflammatory disease that results in destruction of the periodontal ligament and alveolar bone.

2. Calculus – an inert material from calcification of plaque. Full of lacunae.

3. Polymicrobial – more than 500 species with about 13 being the most pathogenic. Clonal types, especially with \( P_g \)

Pathogens most often associated with periodontal disease – *Socransky’s Red Complex*

- *Porphyromonas gingivalis*
- *Treponema denticola*
- *Tannerella forsythensis*

Also yeasts, viruses, fungi, especially herpesviruses (Slots, et al).

4. Shift in the balance of species from gram positive, facultative to gram negative, anaerobic species.

5. Bacterial fimbriae, the arms and legs of the bacteria, allow the microorganisms to adhere to other bacteria, epithelial cells, salivary proteins and other substances.

6. Fimbriae are also a major factor in bacterial virulence (the ability of a bacterium to cause disease), since these structures enable some bacteria to colonize human epithelial cells.

7. Bacteria that are able to stick to each other and to surfaces can form biofilms.

8. Viruses have appendages but not fimbriae. Spikes on the outer surface of a virus help it attach itself to a cell. Viruses are well-organized molecular parasites. Unlike a bacterium or a cell of an animal, a virus lacks the ability to replicate on its own.

9. Where microorganisms congregate: (1) tooth attached plaque, (2) unattached plaque, (3) epithelial associated plaque, (4) bacteria within connective tissue, and (5) bacteria on the bone surface.
10. Shift in microbes unfavorable to the host → signaling proteins called cytokines → send WBC to remove offending organism.

**Pathogenesis of Periodontal Disease**

- Biofilm formation
- Endotoxins
- Permeability of the sulcular wall (VSC)
- Immune response
- Krebs cycle
- Granulation tissue and swelling
- Sulcular bleeding

![Diagram of Pathogenesis of Periodontal Disease](image)

Fig. 1. The pathogenesis of periodontitis. LPS, lipopolysaccharide; PMNs, polymorphonuclear neutrophils. (Adapted from Page RC, Kornman KS. The pathogenesis of human periodontitis: an introduction. Periodontol 2000 1997;14:10; with permission.)

**Inflammation**

1. Inflammation is a process initiated by tissue irritation, injury, or infection.

2. The aim of the inflammatory cascade is to remove foreign organisms, remove and replace necrotic cells, and restore tissue health.

3. The non-specific aspect of inflammation often destroys important host tissues while attempting to restore health, such as the periodontal ligament in periodontitis and pancreatic islet cells in type I diabetes.

4. Bone loss in periodontitis is a function of increased osteoclastic activity and a decrease in osteoblastic activity, spurred by production of pro-inflammatory cytokines, such as TNF-α and IL-6.
5. Both the **microbes** and the **host inflammatory response** are responsible for tissue destruction.


7. The host changes associated with inflammation may be responsible for facilitating a more pathological flora.

8. Therapeutic modulation of inflammation is showing promise, i.e., resolvins and protectins (pro-resolving lipid-based agonists).

9. Aspirin facilitates the production of certain pro-resolving lipoxins. NSAIDS interfere with pro-inflammatory mediators, but also interfere with pro-resolving mediators, thus prolonging inflammation.

10. **Chronic inflammation** results from the body’s failure to turn off its inflammatory response.

11. Host immunity response is initiated by neutrophils, a type of white blood cell crucially important in eliminating infectious organisms by engulfing them through a process called phagocytosis – which literally means “cell eating.”

12. Neutrophils secrete proinflammatory chemicals that cause the acute phase of inflammation. Normally, at the end of this cycle, neutrophils would cease their secretions, and inflammation would subside. But in periodontal disease, the neutrophils continue churning out their proinflammatory chemicals that result in chronic destruction of bone and ligament.

13. Susceptibility to periodontal disease is genetically influenced.

14. Bleeding and inflammation foster increased numbers of periodontal pathogens, as pathogens are nourished by sulcular fluids.

15. **Infrabony defects.**
Biofilm Considerations

1. Biofilms are complex, well-organized micro-environments.

2. In 1968, Dr. Sig Socransky classified the predominant oral bacterial species. The Actinomyces species is closely related to members of the "yellow complex" (Streptococcus sanguis, Streptococcus mitis, Streptococcus gordonii and Streptococcus intermedius), "green complex" (Eikenella corrodens, Capnocytophaga gingivalis, Capnocytophaga sputigena), and "purple complex" (Veillonella parvula, Actinomyces odontolyticus), and appears to colonize the subgingival sulcus first. These complexes are shown to be associated with healthy periodontal sites. Noteworthy, new research has revealed the association with loss of attachment and presence of AA in patient with chronic periodontitis is not very strong. AA is definitely an important bacteria in the onset and progression of periodontal disease but recently also other bacteria have been associated with aggressive forms of periodontal disease, such as porphyromonas gingivalis (usually associate to chronic periodontitis) and also certain viruses.

3. The members of the "orange complex" (Eubacterium nodatum, Peptostreptococcus micros, and species from the genera Fusobacterium, Prevotella, and Campylobacter) are associated with gingivitis and presence of gingival bleeding. The species in this group were closely associated with one another and this complex appeared closely related to the red complex. Ali et al. (1994) found that P. intermedia was always detected in the presence of F. nucleatum in subgingival plaque samples from deep pockets in a group of adult periodontitis subjects. Similarly to the red complex, all species in the orange complex showed a significant association with increasing pocket depth. Further, treatment that included systemically administered metronidazole decreased levels of these species and improved periodontal status. The orange complex, in turn, is associated with the "red complex". The red complex is found to be associated with chronic periodontitis like deep pockets and recession (Newman 2011).

4. It has been shown in-vitro that biofilms grow under low shear conditions. When exposed to high shear, biofilm can be removed from the surface.

5. High velocity lavage will flush through the waterways in the biofilm removing the toxic waste that triggers gingival inflammation.

6. Aeration of biofilms has little, if any, effect on microbial survivability. The classification of microbes as aerobic or anaerobic refers to their optimal growth curves, not morbidity.

7. One explanation of why irrigation works is related to tonicity changes. Water is hypotonic (has lower osmotic pressure) compared to crevicular fluids. Water irrigation replaces a saltier fluid environment with a less salty one. While oral microbes are tolerant of O2 variations, they are less able to cope with tonicity changes.

8. For unexplained reasons, some microbes are able to survive in suboptimal environments.

9. Microbes within the biofilm matrix can communicate through quorum sensing.

10. Six hours after scaling, biofilms are fully mature with all major pathogens established in their most virulent form. (Biofilms, a new approach to the microbiology of dental plaque Jacob M. ten Cate, Review Article: Odontology, (2006) 94:1)
Oral Irrigation Efficacy

1. The efficacy of any oral irrigant depends on several factors:
   a) Delivery efficiency
   b) Concentration
   c) Duration
   d) Substantivity
   e) Dilution
   f) Compliance

2. **Povidone iodine**
   a) Betadine® or generic equivalent) is a 10% solution in water, yielding 1% (10,000 ppm)
   b) According to Greenstein et al, 10% povidone iodine should be diluted with 3 parts water and 1 part povidone iodine. This makes the solution 0.25% which is the minimum dilution to kill *P. gingivalis* in 5 minutes.
   c) Use of povidone iodine does not induce bacterial resistance.
   d) Allergic reactions, including itching, burning, and reddening and blistering in the area of application, so a patient's history of allergy to iodine or shellfish must be evaluated.
   e) Prolonged iodide intake can inhibit thyroid hormone synthesis and cause goiter, myxedema, or hyperthyroidism; therefore, povidone-iodine should not be used in patients with thyroid dysfunction, pregnant woman, infants, or in routine patient self-care.
   f) Povidone-iodine kills in vitro all major periodontopathic bacteria within 15 to 30 seconds and exhibits a wide virucidal spectrum, covering both enveloped (eg, herpesviruses) and non-enveloped viruses.
   g) Povidone-iodine used in periodontal treatment is applied subgingivally using, for example, a 3-mL endodontic syringe with a 23-gauge cannula that has a blunt end and side ports. The cannula is inserted into the base of the periodontal pocket to ensure maximum drug delivery. A single course of subgingival irrigation of the entire dentition takes about 1.5 minutes and is repeated at least 3 times for a total application time of 5-10 minutes.

3. **Sodium hypochlorite (NaOCl)** Among the most potent antiseptic and disinfectant agents against bacteria, fungi, and viruses.
   a) Sodium hypochlorite occurs naturally in human neutrophils and monocytes/macrophages, therefore, it does not evoke allergic reactions.
   b) It is not a mutagen, carcinogen or teratogen.
   c) It has a century-long safety record. Dilute sodium hypochlorite has no contraindications.
   d) Sodium hypochlorite rinsing exerts broad antimicrobial activity against experimental oral biofilms and reduces biofilm by 80-fold compared with water.
   e) Dilute sodium hypochlorite rinse (0.5%) has produced a 47% greater reduction in dental plaque mass compared with water rinsing.
   f) The lowest concentration of sodium hypochlorite solution that reliably inactivates bacteria in vitro is 0.01%.
   g) Galvan et al reported a highly significant (14.5-fold) difference in the number of teeth with no bleeding on probing after having participants rinse twice-weekly for 30 seconds with a .25% solution of sodium hypochlorite and water (3 parts water, 1 part NaOCl).
   h) More frequent rinsing may produce a brown-black extrinsic discoloration of the teeth.
   i) Diluted hypochlorite solutions gradually lose strength, so fresh solutions should be prepared for each use.
How much Clorox to a cup of water should we recommend for home use? The endpoint needed is 1,240 parts/million sodium hypochlorite (NaClO); the minimum inhibitory concentration (MIC).

### Chart Legend:

<table>
<thead>
<tr>
<th>Clorox Brand</th>
<th>NaClO Conc</th>
<th>1% = 10,000 parts/million</th>
<th>Divide PPM by the MIC endpoint needed</th>
<th>Ratio of Clorox to water</th>
<th>Tsp in a cup</th>
<th>Divide tsp/cup by needed PPM/MIC</th>
<th>Teaspoons per cup needed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regular*</td>
<td>5.25%</td>
<td>52,500</td>
<td>1,240 PPM</td>
<td>1:42</td>
<td>48</td>
<td>48/42=</td>
<td>1.14</td>
</tr>
<tr>
<td>Germicidal</td>
<td>8.25%</td>
<td>82,500</td>
<td>“</td>
<td>1:66</td>
<td>“</td>
<td>48/66=</td>
<td>0.73</td>
</tr>
<tr>
<td>Conc</td>
<td>8.25%</td>
<td>82,500</td>
<td>“</td>
<td>1:66</td>
<td>“</td>
<td>48/66=</td>
<td>0.73</td>
</tr>
</tbody>
</table>

*No longer in production


4. **Chlorhexidine.** Numerous studies and meta-reviews have confirmed its antiplaque and antigingivitis effects. The ability of chlorhexidine to adhere to the dental pellicle and oral mucosa prolongs its antiplaque effect.
   a) is absorbed into hydroxyapatite and is believed to inhibit bacterial colonization. After binding, the agent is slowly released in active form over 12 hours to 24 hours (substantivity).
   b) Chlorhexidine is inactivated by organic serum compounds in the gingival crevice fluid, and subgingival placement produces little change in microbial and clinical variables.
   c) As the antimicrobial action of the cationic chlorhexidine is neutralized by anionic compound surfactants in toothpastes, chlorhexidine should not be used in conjunction with toothbrushing.
   d) A major disadvantage of chlorhexidine is its propensity to dark stain tooth surfaces.
   e) It has been shown to increase calculus formation and alter taste over time. Benefit is dependent upon the frequency of use and ability to deliver into deep pockets.

5. **TheraSol** – Ethoxylated tertiary amine, capryl/capramidopropyl betaine – potent antimicrobial, non-staining, does not contribute to calculus formation, 6 hour substantivity, pleasant tasting, low alcohol.
6. **Hydrogen peroxide (H2O2)** There is good evidence for the safety of hydrogen peroxide when used at low concentrations on a daily basis over extended periods of time, in self-administered oral health care products such as dentifrices and mouth rinses. These low concentrations neither damage oral hard or soft tissues, nor do they pose a significant risk of adverse long-term effects. However, it does **not** kill anaerobes and is a weak antiseptic.

7. **Essential oils (Listerine)** – anti-plaque and semi-bacteriocidal activity (Charles, CA et al. Gen Dent. Jan-Feb; (61))
   - Listerine ZERO and Listerine Antiseptic Mouthwashes – 4 essential oils: eucalyptol, menthol, salicylate, thymol
   - Listerine Total Care and Total Care Zero – sodium fluoride 0.02%

8. **Salt water** – may only be bacteriostatic, but is soothing to sore tissues.

9. **Oil pulling.**
   a) It has its origins in Ayurvedic medicine dating back 3,000 years.
   b) For now, the ADA has no opinion on the matter and responded to interview requests by saying that it cannot comment on oil pulling because additional research is needed, and provided a link to its general Policy Statement on Unconventional Dentistry. "With the explosion of unrefereed information about oral health issues made possible by the Internet, the Association believes that the need for systematic evaluation of diagnostic and treatment efficacy and safety to assist practitioners in responding to patient inquiries is greater than ever," the statement reads.
   c) Coconut oil is preferred because 50% of the fat in coconut oil is comprised of the antimicrobial ingredient **lauric acid**. Lauric acid is very well known for its antimicrobial actions; it inhibits Strep mutans that are the primary bacteria that cause tooth decay.
   d) The premise is that microbes adhere to the oil and are removed with the oil is expelled. According to Jessica Emery, DMD; "As the oil hits your teeth and gums, microbes are picked up as though they are being drawn to a powerful magnet. Bacteria hiding under crevices in the gums and in pores and tubules within the teeth are sucked out of their hiding places and held firmly in the solution."

### Factors that Can Influence Disease Progression

1. **Home care**
   a. Is the patient apathetic?
   b. Is the patient financially strapped?
   c. Is the patient uneducated about oral hygiene?

2. **Susceptibility** - Good oral hygiene slows progression of disease. However OH plays less of a role for individuals NOT susceptible to the disease.
   a. Salivary pH
   b. Genetic predisposition
3. Systemic Disease, Conditions, and Drugs
   a. Diabetes
   b. Cardiovascular disease (AHA Scientific Statement, Apr. 2012 “Periodontal Disease and Atherosclerotic Vascular Disease: Does the Evidence Support an Independent Association?” “A link between oral health and cardiovascular disease has been proposed for more than a century…Observational studies to date support an association between PD and ASVD independent of known confounders. They do not, however, support a causative relationship. Although periodontal interventions result in a reduction in systemic inflammation and endothelial dysfunction in short-term studies, there is no evidence that they prevent ASVD or modify its outcomes.”
   c. Respiratory disease
   d. HIV/autoimmune disease
   e. Osteoporosis
   f. Pregnancy and low birthweight outcomes
   g. Prescription drug use

4. Fear
   a. of the unknown
   b. previous unpleasant dental experience
   c. injection
     • DENTSPLY™ survey of 700 patients - results 1 of 10 cancelled an appointment due to fear of injection.

5. Lifestyle factors
   a. Alcohol –
   b. Recreational drug use
   c. Tobacco
     • Smoking has toxic effect on bone dev. And reduces calcium absorption. According to NIH study published in 2000, 78.4% of perio is due to smoking.
     • All tobacco products, including e-cigarettes and hookahs, have nicotine. And it’s nicotine’s highly addictive properties that make these products harmful.

6. Local Oral Factors
   a. Overhanging restoration
   b. Poor margins
   c. Calculus
   d. Malpositioning
   e. Oral piercings

SOME PATIENTS HAVE NO IDENTIFIABLE RISK FACTORS
Review Questions

1. It has been established that all people are not equally ____________________ to periodontal disease, regardless of the level of home care.

2. Periodontal disease has been called a ____________________ disease.

3. The one medicament that has been shown to have continuing efficacy over five weeks is ________________ ____________________.

4. Inflammation is a process initiated by tissue ______________ , _______________, or ______________.

5. The top two risk factors for periodontitis are ________________ and ________________.

Limitations of BOP

1. Aspirin – Commonly taken for headaches and by many adults to protect against coronary disease. A single 325 mg tablet taken 7 days increases BOP by 12.4% Compared to placebo studies, aspirin takers are 5X more likely to exhibit BOP.
2. Hypertension Drugs – Estimated to be taken by 25% of adults. All increase BOP.
3. Menstruation – Bleeding varies with serum levels of estradiol, which peaks and drops during ovulation and premenstruation.
4. Oral contraceptives. Women who were on oral contraceptive pills had more extensive gingivitis and gingival bleeding than their matched controls not taking them. (JCDP, May, 2010)
5. Local Trauma – Recent food impaction and heroic hygiene before a visit to the dentist increase capillary fragility.
6. Probing Force – BOP varies directly with probing force and dramatically affects BOP. In clinical studies, probing forces vary from 5-125 g of force (20-30 g is considered ideal). For comparison, the weight of metal probe = 20 g.
7. Medications – Coumadin, Plavix, Pradaxa, Brilinta
8. Dietary supplements – the five G’S; Ginkgo, Ginseng, Garlic, Glucosamine, Ginger; Feverfew; Fish oils; St. John’s wort
9. Stress
10. Mouth breathing
11. Post-partum adjustment
12. Smoking cessation
13. Bruxism
14. Dietary anomalies
15. Systemic diseases
16. Vitamin deficiencies
Homecare Modalities and Customization

1. Different randomized studies have proven that interdental cleaning with devices other than floss are more efficient and thorough at plaque removal. (Jackson, 2006).

2. The interdental brush can be turned vertically to reach deeper pocket areas.

3. Vigorous horizontal interdental brushing can cause excessive wearing of root surfaces.

4. While flossing has been shown to reduce bleeding in gingivitis cases over tooth brushing alone, behavior modification of patients through education has not been successful in improving floss use. (Bagley, 1992).

5. A study by Hujoel, et al, (2006) stated: “No evidence on the effectiveness of floss in adults or under real-world clinical conditions could be identified. In particular, there was no evidence that flossing is effective in the presence of topical fluorides.”

6. From a Cochrane Review published in 2012: “There is some evidence from twelve studies that flossing in addition to toothbrushing reduces gingivitis compared to toothbrushing alone. There is weak, very unreliable evidence from 10 studies that flossing plus toothbrushing may be associated with a small reduction in plaque at 1 and 3 months. No studies reported the effectiveness of flossing plus toothbrushing for preventing dental caries.” (Sambunjak, et al)

7. Alternative forms of interdental plaque removal have been preferred by patients, suggesting improved compliance. (Spolsky, 1993).

8. Power brushes have been shown to more effectively remove plaque than manual brushes. (Dentino, et al, 2002)


10. In an evaluation of the efficacy of four floss types (woven, waxed, unwaxed, and shred resistant), Carr et al. allowed plaque to accumulate over 3 days, scored the plaque using O’Leary’s plaque index, and analyzed reductions in interproximal plaque after flossing. Plaque removal did not differ significantly among floss types.

11. Dentifrice is not necessary for plaque removal. (Paraskevas, 2007) However, dentifrice does provide benefits beyond plaque removal, i.e. reducing caries through fluorides, whitening, sensitivity reduction, fresh breath, calculus reduction, xerostomia relief. Colgate Total® reduces gingivitis through triclosan copolymer.

12. The use of baking soda as a dentifrice or toothpastes containing baking soda has been shown to be better at reducing plaque than non-soda toothpastes. (Putt, et al, 2008) In high concentrations, sodium bicarbonate is bacteriocidal against most periodontal pathogens. (Newbrun, E., 1997)

13. Non-floss options include interdental brushes, Soft-Picks by Sunstar Butler, Scrub Betweenes by Dentek, RotaPoints by DenMat.

14. An ideal interdental cleaning device should be userfriendly, remove plaque effectively, and have no deleterious soft-tissue or hard-tissue effects. However, not all interdental cleaning devices suit all patients, all types of dentitions and even not every interdental space. The
dental professional should, therefore, navigate the patient to the optimal devices tailored to their specific needs.

**Dianne’s Six Rules on Homecare**

1. Nobody will ever change anything about their homecare routine without first developing ________________________________.

2. If the patient cannot or will not floss, ___________________________ _____.

3. People do not brush or floss their teeth while ___________________________ _____ ___________________________.

4. Do not use ___________________________ ___________________________ such as, “You might want to think about flossing.”

5. Proper use of a good ___________________________ ___________________________ makes a good communicator become a GREAT communicator.

6. Please do not wait until the _________ of the appointment to teach home care instructions.
Patient Case Classification System

Healthy – Healthy tissue, light stain and/or calculus. Deposits primarily supragingival. Code 1110. (Code descriptor – “Removal of Plaque, calculus and stains from the tooth structures in the permanent and transitional dentition. It is intended to control local irritational factors.”)

Type I – Gingivitis. Gingivitis can vary from slight to severe. These patients will exhibit heavier debris than a healthy patient, but will not have bone loss. However, bleeding is the primary sign of bacterial activity. Shallow pockets have been shown to contain reservoirs of volatile sulfur compounds that are implicated in periodontal disease. Therefore, a two stage treatment protocol is necessary for thorough debridement.

Option 1
1st visit – full debridement (ultrasonics) to enable diagnosis Code 4355 (see flow chart)
2nd visit – prophylaxis and polish Code 1110

Option 2
1st visit – full debridement (ultrasonics) Code 1110
2nd visit – prophylaxis and polish Code 1110

Option 3
1st visit – right side debridement Code 4999
2nd visit – left side debridement Code 1110

**ALL 4999 (Unspecified periodontal procedure by report) must have a detailed narrative and intraoral photo if possible.

Type II – Early Periodontitis. Slight bone loss is detected with some pockets in the 4-6mm range. Some areas may need anesthesia to scale thoroughly. However, the disease has not progressed to the point of furcal involvement or mobility. Also, recession must be charted, not only because it shows previous disease, but because it is a more accurate representation of the patient’s periodontal status. Many insurance companies do not pay benefits for root planing unless there is at least 4 mm of attachment loss (not just probing depth). Attachment loss is the addition of the pocket measurement plus recession. The early periodontitis patient will have 3 or less teeth in the quadrant that are periodontally involved. They will require site specific periodontal treatment. The remainder of the dentition is appropriately treated with prophylaxis – D1110.

1st visit – prophylaxis for non-periodontally involved teeth Code 1110
2nd visit – UR/LR periodontal scaling Code 4342 (specify teeth)
3rd visit – UL/LL periodontal scaling Code 4342 (specify teeth)

Subsequent recare visits can be coded 4910, periodontal maintenance. Please note that for just a few isolated teeth with 4342, the patient may be maintained with prophylaxis in limited circumstances, in the clinical judgment of the dentist. Some insurance payors will not reimburse 4910 after a single or dual 4342 visit. It is highly variable among companies. Also, please note that some payors will allow 4342 on the same day as 1110 and some will not. Again, highly variable.

Type III – Moderate Periodontitis. Bone loss is in the 6-7mm range. There is moderately heavy calculus, both supra and subgingival. There can be furcation involvement and Class I mobility. Depending on the severity and number of teeth present, this type may need anesthesia throughout.
4 visits of quadrant scaling or 2 extended length visits  

Code 4341 X 4

Subsequent visits should be coded 4910, typically after 90 days of completion. Please note that many insurance carriers will pay 4910 for a limited time – 2-3 years – and then require code 1110. If the patient continues to exhibit signs of active disease, the patient will be appropriately coded 4910, which is periodontal maintenance. If the patient has no signs of active disease after a period of maintenance, the patient may be maintained with prophylaxis with the consent of the treating dentist.

**Type IV – Advanced Periodontitis.** Heavy bleeding, suppuration, and pockets in the 7mm or greater range. Mobility and very tenacious calculus (often ethnic) Will need anesthesia throughout the entire process. Heavy emphasis on ultrasonics and revisiting each previous quadrant scaled on subsequent visits.

4 visits of quadrant scaling with anesthesia  

Code 4341 X 4

All subsequent recare visits will be coded 4910, periodontal maintenance. The fee should reflect the degree of difficulty exhibited by the patient’s oral condition. Please note that many insurance carriers will pay 4910 for a limited time – 2-3 years – and then require code 1110. If the patient continues to exhibit signs of active disease, the patient will be appropriately coded 4910, which is periodontal maintenance. If the patient has no signs of active disease after a period of maintenance, the patient may be maintained with prophylaxis with the consent of the treating dentist.

---

*CLINICAL FLOW CHART*

(Used with permission, Charles Blair and Asso. *Coding with Confidence, 2012*)
Insurance Coding Recommendations

1. It is improper to report a periodic oral evaluation (D0120) or any other evaluation without the doctor actually examining the patient. A hygienist can screen but not diagnose unless specifically permitted by law.

2. It is improper to charge insurance patients for a periodic oral evaluation while the evaluation is ‘free’ for non-insurance patients. Treatment and fee protocols should be identical for both insured and non-insured patients.

3. CDT 2014 periodontal maintenance (D4910) does not include any type of periodontal oral evaluation. The code (D180), comprehensive periodontal evaluation can be used with the established periodontal patient in conjunction with (D4910).

4. The periodic oral evaluation (D0120) currently includes “periodontal screening, where indicated.” A periodontal screening, at the minimum should be performed and recorded at each D0120 visit. This does not mean periodontal probing and full charting is required at each periodic oral evaluation in order to report the periodic oral evaluation code.

5. Many offices erroneously report a periodic oral exam (D0120) instead of a comprehensive oral evaluation (D0150) in an effort to hold down the initial comprehensive oral evaluation fee for children. Doctors should establish two consistent fees (adult and child) for code D0150. To distinguish between the two, use D0150A (child) and D0150B (adult), or similar coding to distinguish the fee structure between the two. The computer software will strip the “A” or “B” for reporting purposes. With this approach, the reimbursement UCR will be higher for the child comprehensive evaluation (D0150) while the proper code is reported.

6. For an oral evaluation for a child less than three years of age, use code D0145.

7. It may be appropriate to also apply fluoride varnish for moderate to high caries risk patients at the under-three oral evaluation appointment. Use code D1206 in addition to D0145.

8. The comprehensive periodontal code (D0180) is not specialty-specific. The general practitioner can use it, as with any CDT code. However, the general practitioner would generally report the more comprehensive, extensive, and all-encompassing comprehensive oral evaluation (D0150) for the new patient, rather than D0180.

9. Some payors reimburse D0180 every 24 months, others every five years or “lifetime.” Reimbursement for D0180 is high variable. D0180 may be reimbursed only “once” per lifetime (like D0150) per doctor by some payors.

10. It is misleading to report extraoral panoramic film (D0330) and bitewing films (D0272/D0273/D0274) by the dental office simply ‘converting’ and reporting the two separate procedure codes as one code, intraoral complete series (D0210). A panoramic film is extraoral, not intraoral. NOTE: It is not illegal or improper for an insurance company to “remap” a submitted code to another code for payment purposes, according to the contract language. Payors routinely convert separately coded extraoral panoramic film and intraoral bitewing films to the lower, complete series (D0210).

11. Some insurance companies are now only allowing one prophy/year, depending on the patient’s risk factors. Pregnant patients can sometimes get benefits for four prophys/year.

12. It is improper to routinely use the D4355 code on new patients. (There is no such thing as an “exploratory prophy.”) The language for this code is very specific for those situations where there is so much bulky calculus that it is impossible to perform an examination. Many plans do not recognize this code. Some will pay only if submitted as D4999. Either way, it recommended that a narrative
be included. (Code descriptor for D4355 – “The gross removal of plaque and calculus that interfere with the ability of the dentist to perform a comprehensive oral evaluation. This preliminary procedure does not preclude the need for additional procedures.”)

13. Use code D4921 for subgingival irrigation. (New code for 2014 – not usually a covered benefit)

14. Some policies provide reimbursements for two – D4910 and D1110 – during a 12 month period. If the patient requires 3-month maintenance appointments, it is possible to bill the D4910 each time but **request an alternate benefit of D1110 every other time** since four distinct procedures are part of the plan. The narrative would read: “If D4910 is denied, please provide the alternative benefit of a prophylaxis. The ongoing periodontal maintenance visit included prophylaxis (D1110).” The D1110 is considered part of the D4910 by payers. The clinical record should reflect the fact that a “prophylaxis” was completed as part of the overall D4910 procedure.

Coding With Confidence: the “Go To” Guide for CDT 2014 by Charles Blair, DDS
www.drcharlesblair.com $109 + $10 shipping

Insurance Solutions - a bimonthly newsletter - a MUST HAVE for any office that takes dental insurance.
http://www.dental-ins-solutions.com

**Can a patient ever go back to 1110 ‘prophy’ after going through RPS?**

To quote from the CDT-13 concerning coding for a prophylaxis after periodontal therapy, “This is a matter of clinical judgment by the treating dentist. Follow-up patients who have received active periodontal therapy (surgical or non-surgical) are appropriately reported using the periodontal maintenance code D4910. However, if the treating dentist determines that a patient's oral condition can be treated with a routine prophylaxis, delivery of this service and reporting with code D1110 may be appropriate.”

**Can periodontal disease be cured?**

“Some therapists believe that periodontal diseases are chronic diseases and not curable, rather they are maintainable illnesses. However, this line of reasoning fails to recognize that most patients with periodontal diseases attain periodontal health after therapy at the vast majority of sites. The need for maintenance post-treatment is an important aspect of optimizing long-term success, but recognition of this fact does not justify characterizing periodontal diseases as incurable…The belief that chronic periodontitis is incurable in the presence of health after periodontal therapy seems contradictory. Maintenance may be an extension of active therapy, but the absence of disease after treatment fulfills the criteria for health and curability.” (Greenstein, JOP, August, 2002)
Step-By-Step Scaling and Debridement

- Seat patient and inquire about possible medical history changes
- Blood pressure screening
- Pre-procedural rinse
- Prepare for anesthetic and/or nitrous if needed and summon doctor
- Topical anesthetic
- Local anesthetic
- Disclosing solution. Demonstrate homecare procedures as needed
- Ultrasonic scaling, high power, large insert for gross debridement
- Ultrasonic scaling with fine insert at medium power
- Hand instruments
- Ultrasonic wash
- Hand instruments
- Ultrasonic wash
- Subgingival irrigation as necessary
- Apply fluoride varnish to newly exposed root surfaces in patients with severe attachment loss
- Instructions in home rinsing with chlorhexidine, salt water, fluoride, etc.

**Fluoride Varnish Ordering Information**

**PreviDent** Varnish (5% NaF) Colgate  www.colgateprofessional.com or your local Colgate rep

**VANISH** (5% NaF white varnish) OMNI Preventive Care/3M  1-800-445-3386

**Iris** (5% NaF) Benco Dental  1-800-462-3626

**VarnishAmerica Original** (5% NaF) **VarnishAmerica White** Medical Products Labs 800-523-0191

**Waterpik Ultra-Thin Varnish** 800-525-2774

(For more information on fluoride varnish evaluations, see September/October, 2008 issue of “Clinicians Report” by Gordon Christensen, 801-226-2121, www.cliniciansreport.org)
The Goals of Therapy

What causes periodontal disease?

Periodontal disease results from a combination of microbial effects and non-specific host responses acting at the only site in the body that is not protected by an intact epithelium covering.

What is the goal of periodontal therapy?

To stop the progression of the disease

How do we know when we have stopped the progress of the disease?

1. No continuing loss of periodontal attachment
2. No continuing loss of supporting bone

Inserts and Equipment

Burnett Power Tip by Parkell – 1-800-243-7446  www.parkell.com
GentleClean insert for implants - Parkell
THINsert by Dentsply®  www.dentsply.com
Manual Tune Ultrasonic - USI – 1-800-874-5332
American Eagle Instruments - www.am-eagle.com
Colorvue™ Probe by Hu-Friedy
Face Drapes by Practicon – www.practicon.com  1-800-959-9505
PropGard by Ultradent – www.ultradent.com  1-800-552-5512
Magnification by Designs for Vision - www.designsforvision.com  800-345-4009
Retipping service for instruments - Goldman Retipping Service – (708) 526-1166
Implant ultrasonic insert (Titanium Implant Scaler) – www.tonyriso.com  866-986-6974
Ultrathin inserts - www.tonyriso.com  866-986-6974
Closing Thoughts

- Periodontal disease is a function of microbes and a susceptible host.
- Re-infection happens.
- Some pathogens are tissue invasive, and lapses in home care lead to re-infection.
- High velocity water lavage will effectively remove plaque.
- Treating periodontal infections should involve strategies aimed at controlling microbes.
Bibliography


Gadsby, R. “The association of periodontal disease, diabetes and cardiovascular disease.” British Journal of Diabetes & Vascular Disease 2008; 8: 188. The online version of this article can be found at: http://dvd.sagepub.com/cgi/content/abstract/8/4/188


Haraszthy, Violet I., Sreenivasan, Prem K, & Zambon, Joseph J. “Community-Level Assessment of Dental Plaque Bacteria Susceptibility to Triclosan Over 19 Years.” BMC Oral Health. 2014;14(1)


Inflammation, Heart Disease and Stroke: The Role of C-Reactive Protein. April 2, 2008 http://www.americanheart.org/presenter.jhtml?identifier=4648


Weyant, Robert J. “No evidence that improved personal oral hygiene prevents or controls chronic periodontitis.” *Journal of Evidence Based Dental Practice*, Volume 5, Issue 2, June 2005, Pages 74-75.


Notes