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Instrumentation for the Treatment of Periodontal Disease

Peer-Reviewed Publication
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Educational Objectives

The overall goal of this course is to provide the reader with information on nonsurgical periodontal therapy and instrumentation of periodontal sites. Upon completion of this course, the clinician will be able to do the following:

1. List and describe the prevalence of periodontitis in the population
2. Describe the periodontal disease process
3. List and describe the associations between oral and systemic health
4. List and describe therapeutic options for the treatment of periodontal disease
5. List and describe the mechanism by which ultrasonic instrumentation works
6. List and describe insert tip selection for periodontal sites and the rationale for their selection

Abstract

The initiation and progression of periodontal disease requires the presence of bacterial accumulations. Once periodontal disease exists, its progression depends on the host response. In order to treat periodontal disease, the biofilm must be disrupted and all hard and soft deposits removed from the tooth surfaces. In order to thoroughly remove deposits and debris without removing excessive tooth structure, instruments must be selected that are suitable for the intended site and technique. The selected debridement method should offer predictable results independent of operator skill level; be efficient to perform clinically, well tolerated by patients and cost effective; and have a low potential for adverse side effects.

Introduction

Periodontitis is common, with mild to moderate forms affecting 30% to 50% of adults and the severe generalized form affecting 5% to 15% of all adults in the United States.[1] More recent data suggests that the prevalence of periodontitis in the United States may actually be much greater than previously estimated.[2] Periodontitis seen in youth and early adulthood can probably be classified as aggressive periodontitis, and some degree of clinical attachment loss (CAL) in youth is well documented in population studies.[3]

Bacterial accumulations on the teeth are essential for the initiation and progression of periodontitis. This microbial infection is followed by a host-mediated destruction of connective and bone tissues caused by hyperactivated immune-inflammatory response.[4] The net result of the host response to initiating periodontal pathogenic bacteria is destruction of periodontal tissues and systemic interactions. Despite great understanding of the potential therapeutic benefit of host modulation, effective interruption of periodontal bacteria remains the cornerstone of effective periodontal disease intervention. Advances in debridement devices and techniques, as discussed below, can enhance a clinician's ability to successfully manage periodontal disease. Emerging information linking oral inflammation with serious, chronic diseases of aging underscores the importance of effective periodontal therapy. More effective approaches aimed at helping patients achieve and then maintain a preferred level of oral health can pay dividends to overall health.

Periodontal Disease Process

Periodontitis, viewed for years as primarily the outcome from infection, is now seen as resulting from a complex interplay between bacterial infection and the host response, often modified by behavioral factors. The host response is now seen as a key factor in the clinical expression of periodontitis, with only some 20% of periodontal diseases now attributed to bacterial variance. Additionally, genetic variance may be responsible for up to 50% of periodontal disease expression.[5] The clinical diagnosis of periodontitis historically has required evidence of loss of connective tissue surrounding the teeth and bone loss detected by radiography. For many years, clinical probing depth measurement was the primary factor used to determine which sites were in need of periodontal therapy. Current knowledge of the role that inflammation plays in the etiology of many systemic diseases suggests that incorporating other assessments into periodontal treatment decision pathways may be important. The destruction of periodontal tissues leads to deepening of the sulci adjacent to teeth, resulting in the formation of periodontal pockets. Despite the awareness that inflammatory mediators of oral origin can affect other body disease processes, periodontal therapy has been aimed almost exclusively at achieving and then maintaining pocket depths that the clinician considers accessible for the patient and for professional debridement efforts.

While there is little doubt that reduction in probing depth improves access to subgingival areas, focusing the management of periodontal disease solely on pocket depth may not be sufficient. Medical research underscores the important role that inflammation in the body plays in the development and progression of many of the serious, chronic diseases of aging. Emerging evidence continues to suggest that the mouth can be a significant source of inflammation when periodontal disease persists.[6] The entrance of bacteria, bacterial byproducts and inflammatory mediators released orally in response to the pathogenic periodontal bacteria can enter the bloodstream. Inflammation of periodontal tissues can have adverse effects beyond loss of periodontal attachment and bone.[7] Thus, in addition to management of probing depths, it seems prudent for oral inflammation to take on added diagnostic and therapeutic significance in the management of periodontal disease. The following therapeutic approach is based on assessment of patient, tooth and site risk factors. The intent is to more effectively target therapy to improve patients' oral and overall health.

Focusing the management of periodontal disease solely on pocket depth may not be sufficient.

Which Patients to Treat

Environmental and genetic factors, as well as acquired risk factors, accelerate destructive inflammatory processes in periodontitis.[8] The following non-oral risk factors associate strongly with increased risk for periodontitis and disease severity: tobacco use, diabetes mellitus, family history, mental stress and depression, obesity, and osteoporosis.[9] Realizing that risk factors for periodontal disease can make eradication of periodontal disease more difficult, more aggressive therapy is considered for patients who have known periodontal disease risk factors.

Table 1. Non-oral risk factors for periodontal disease

Tobacco use
Diabetes mellitus
Family history
Mental stress
Depression
Obesity
Osteoporosis

In similar fashion, adverse associations have been identified between periodontal disease and diabetes, cardiovascular disease, preterm low-birth-weight deliveries, respiratory diseases, certain cancers, kidney diseases and other systemic conditions.[10] It certainly seems advisable to treat more aggressively those patients who have other risk factors for the conditions that can be affected by periodontal inflammation. Allowing periodontal inflammation to persist in such patients will only add to their systemic disease risk. Rather than applying a basic therapeutic approach to all patients, determining if patients presenting for dental care have any of the factors indicating increased risk for periodontal disease severity and/or any of the other known risk factors for systemic diseases that can be affected when periodontal disease persists can be used to formulate a therapeutic approach proportionate to the level of risk.

Table 2. Adverse associations with periodontal disease

Diabetes mellitus
Cardiovascular disease
Respiratory disease
Certain cancers
Renal disease
Preterm low-birth-weight deliveries

Which Sites to Treat

Clinical and radiographic findings are commonly used to determine a patient's periodontal status. Often treatment resources are directed primarily to sites where probing depth has increased (where disease progression has already occurred). Diagnostic findings offering predictive value would allow the direction of treatment resources to sites at which breakdown was imminent. Bleeding on probing (BOP) is among the clinical signs used to predict disease progression.[11] Yet there is general agreement that an isolated incidence of BOP at a site is a poor predictor of disease activity at that site.[12] The predictive value of BOP increases substantially when BOP is persistent. Sites that continue to demonstrate BOP (at successive reevaluation visits) are more likely to break down.[13] In addition to signaling impending destructive activity, BOP is strongly correlated with gingival inflammation.[14] Gingival inflammation is typically expressed clinically as redness, edema and/or bleeding.

While preventing adverse changes in pocket depth has merit, the overwhelming evidence confirming the adverse relationship between oral inflammation and systemic disease suggests that elimination of inflammation should also be a goal of therapy. In addition to sites at which increases in probing depth are noted, those sites with persistent bleeding on probing or where other clinical signs of inflammation are found should be priority candidates for therapeutic attention.

Sites with increases in probing depth, persistent bleeding on probing or where other clinical signs of inflammation are found should be priority candidates for therapeutic attention.

Which Treatments

Bacterial biofilm accumulations on the teeth are essential to the initiation and progression of periodontitis.[15] Although periodontitis begins with a microbial infection, it is the host-mediated inflammatory response that causes clinically significant connective tissue and bone destruction.[16] Long-term clinical studies have clearly demonstrated that the regular and effective removal of bacterial biofilms on the teeth can prevent periodontitis.[17] Suppressing the host response has also been shown to play a critical adjunctive therapeutic role.[18] Dietary alterations intended to reduce the inflammatory response have also been shown to be of benefit in periodontal therapy.[19] Yet mechanical disruption of the biofilm remains the foundational approach for the resolution of inflammatory periodontal diseases. Biofilm disruption can be accomplished by mechanical means (hand instrumentation and/or ultrasonic instrumentation), systemic and local administration of targeted antibiotics, and laser-generated energy.[20] The chosen methodology is most often driven by the clinician's personal preference. However, the selected debridement method should offer predictable results inde-

pendent of operator skill level; be efficient to perform clinically, well tolerated by patients and cost effective; and have a low potential for adverse side effects.

Mechanical disruption of the biofilm remains the foundational approach for the resolution of inflammatory periodontal diseases.

Therapeutic Options

Periodontal therapy had long been focused on removal of visible plaque and clinically detectable calculus. The pathogens that initiate periodontal disease were thought to be deeply embedded in the cementum of subgingival root surfaces. For many years, the intention of mechanical debridement was the deliberate removal of the cementum, which was assumed to be pathogen laden. Increased dentinal hypersensitivity[21] and pulpitis[22,23] have been reported as undesirable side effects of excessive cementum removal. In the past two decades, studies have demonstrated that endotoxin is more superficially associated with the cementum. Thus, deliberate cementum removal is not necessary and may not even be prudent. While not conclusive, current research suggests that preserving cementum can improve the degree of periodontal regeneration.[24] In this regard, the consensus report from the 1996 World Workshop in Periodontics states that intentional cementum removal should not be included in current periodontal debridement techniques for the purpose of removing toxic substances from the root surface.[25] More recently, the American Academy of Periodontology added that "the goal of periodontal instrumentation is to effectively remove plaque and calculus, while causing the least amount of root surface damage."[26]

Current research suggests that preserving cementum can improve the degree of periodontal regeneration.

When used properly, similar clinical outcomes can be achieved with hand curettes and ultrasonic instrumentation.[26] However, the inherent operator variability due to the design and use of a bladed instrument makes achieving therapeutic root debridement less predictable with manual curette use. For a curette to actively remove biofilm without excessive removal of cementum, an adequate working edge must be created and maintained throughout the procedure. Additionally, that sharpened working edge has to be positioned against the root at the precise angle that permits the working portion of the instrument to engage the root in a way such that the movement of the instrument in a coronal direction will result in sheer force sufficient to remove endotoxin. Finally, the level of force applied during the working stroke needs to be sufficient to dislodge endotoxin from the surface without excessive cementum removal. With ultrasonic instrumentation, positioning the working portion of the instrument and applying sufficient force to selectively dislodge endotoxin is less operator-dependent. The cylindrical shape of most ultrasonic inserts is also more conducive to biofilm removal (better conforms to the surface) than the linear cutting edge of bladed instruments that were really designed for effective calculus removal.

Inherent operator variability makes achieving therapeutic root debridement more predictable with ultrasonic instrumentation than with manual curette use.

Ultrasonic Scaling

There are two categories of ultrasonic instrumentation: magnetostrictive and piezo. These categories differ in the way they are powered, which was thought to result in differing patterns of tip movement. Piezo devices, powered by a crystal, were believed to result in tip movement that is linear. Thus, only the sides of piezo-driven tips were thought to provide active debridement. The dimensional change in the metal stack of a magnetostrictive-driven insert was thought to be elliptical, with all sides of the tip capable of removing biofilm. The early studies suggesting this fundamental difference in tip motion were performed without load applied to the insert tips. Subsequent laser vibrotomy-based studies have demonstrated that as soon as the insert tip is loaded (placed against a tooth surface), both piezo- and magnetostrictive-driven instrument tips have elliptical patterns of movement.[27] Thus, anecdotal claims of increased root trauma with magnetostrictive devices compared to piezo devices due to the magnetostrictive tip "banging" into the root in multiple directions are unfounded. In reality, both piezo- and magnetostrictive-driven tips move in similar fashion once under any load. Indeed, the degree of damage to root surfaces via ultrasonic instrumentation is a factor of tip shape, lateral force, angulation and power setting regardless of the method of ultrasound generation. In other words, magnetostrictive, piezo and even hand curettes are all capable of inducing root damage. However, by using a preferred tip, at a preferred angulation, and using a preferred level of lateral force at a preferred power setting, the risk of root surface damage can be eliminated for all methods.[27] Tip angulation may be the primary determinant in causing root damage. Forces generated with a magnetostrictive-driven tip were lowest when the tip was parallel to the tooth surface and increased to its maximum point as the tip was moved ninety degrees to the tooth surface.[28] In contrast, forces from a piezo-driven tip increased and then peaked when the tip was moved to forty-five degrees to the tooth surface.[29] In other words, piezo is more technique-sensitive in terms of minimizing root damage.

Additionally, the degree of root substance removal via ultrasonic devices is significantly influenced by the tip designs, increasing for wider scaler tips as compared to narrow, probe-shaped inserts.[30] Thus, a suitable magnetostrictive-driven insert used at proper settings is less likely to result in root damage than a piezo-driven insert.

The degree of root substance removal via ultrasonic devices is significantly influenced by the tip designs, increasing for wider scaler tips as compared to narrow, probe-shaped inserts.

Cavitation

The removal of plaque and calculus from the tooth surface had originally been attributed mainly to the vibratory action of the probe tip. Walmsley theorized in 1984 that while the primary deposit removal action by ultrasonics is mechanical, cavitation activity causes fracture of the attached deposits through the resultant shock waves.[31] Despite conflicting laboratory findings and no reliable way to evaluate the clinical effects of ultrasonic cavitation, the force created via ultrasonic induced cavitation may be sufficient to disrupt the biofilm environment, thereby facilitating the mechanical removal of periodontopathic bacteria.[32]

Surface-Specific Tips and Device Settings

Clearly, contact between the active portion of an ultrasonic insert at a preferred power level, proper angulation to the tooth surface and minimal force is essential for adequate biofilm interruption. Tip selection should be based on the type of deposit and the anatomy of the surface to be debrided. The type of deposit encountered at a site should determine the amplitude (power level) needed for efficient removal. The force behind the tip movement is influenced by the diameter of the tip and the stroke range, with wider-diameter tips (standard) producing greater force for efficient removal of heavy or tenacious calculus and slim-diameter tips producing a lower level of force appropriate for efficient removal of light/soft deposits. The anatomy of the treatment site should then determine the shape of tip that will maximize contact of the active area with the root surface for thorough deposit removal.

Tip selection should be based on the type of deposit and the anatomy of the surface to be debrided.

A wide range of available magnetostrictive inserts permits a preferred two-stage approach to instrumentation. The objective of the first stage – scaling – is to reduce moderate-to-heavy calculus/stain deposits. To accomplish this most efficiently, a standard-diameter insert/tip and a higher level of power should be used. The objective of the second stage – debridement – is the definitive removal of all the light calculus and stain deposits that remain, as well as definitive removal of biofilm and endotoxin. Slim inserts are ideal for the second stage and can reach pocket areas deeper than 4 mm and furcation areas, provided an appropriately designed tip is used for the site's anatomy.

The use of slim insert tips also helps tactile identification of root morphology or remaining deposits, as the tip diameter is very similar to that of a periodontal probe. Data obtained from probing similarly can be obtained from the

Table 3. Magnetostrictive ultrasonic insert tip selection

DEPOSIT AND ROOT ANATOMY					INSERT SELECTION	INSERT SETTINGS		
Type of Deposit			Root Anatomy			Power Setting		
Light/ Biofilm	Moderate	Heavy	Straight	Curved	Type	Low	Low-Medium	Low-High
	X	X	X (supragingivally)	X (supragingivally)	Standard #3/Beavertail			X
	X	X	X	X (≤4mm)	Standard #10			X
	X	X	X	X (≤4mm)	Standard #100			X
	X	X	X	X (≤4mm)	Standard #1000/Triplebend			X
X	X		X	X (≤4mm)	Slimline #10		X	
X	X		X	X	Slimline Right or Left		X	
X	X		X	X (≤4mm)	Slimline #1000			X
X			X	X (≤4mm)	THINSert			X
X	X	X	X		SofTip (implant insert)	X		
	X	X	X (w/surgical procedure)	X (w/surgical procedure)	DiamondCoat			X

slim insert during use. Slim-insert use also allows biofilm removal without excessive cementum removal, thus reducing the likelihood of post-instrumentation dentinal hypersensitivity. It is still prudent to use an explorer specifically designed for calculus detection (ODU 11/12) following any scaling procedure. To complement root anatomy, right and left inserts are used to fully access the full root circumference in deep pockets, and by rotating the tip, full access to the roof of furcations can also be achieved.

Figure 1. Insert tip designs

Designs with curved tips, straight tips and optimized line angulations enable full pocket access ergonomically. New designs with line-angle adaptation have also improved the ability to access interproximal and subgingival areas, offering efficient removal of deposits with a slim-tip insert while maximizing patient comfort. A beveled edge design at the working end of the insert tip also helps the efficiency of deposit removal, as the ultrasonic energy is specifically targeted to each of the four corners rather than on the full circumference of a rounded working end.

Standard Diameter Inserts

The wider diameter and longer stroke range of standard inserts provide a range of force (amplitude) appropriate for the efficient reduction of moderate to heavy and/or tenacious calculus and stain deposits. The degree of force is further defined by the power setting of the scaling unit. The lowest power setting at which efficacy (removal/reduction of deposit) is achieved efficiently should be utilized.

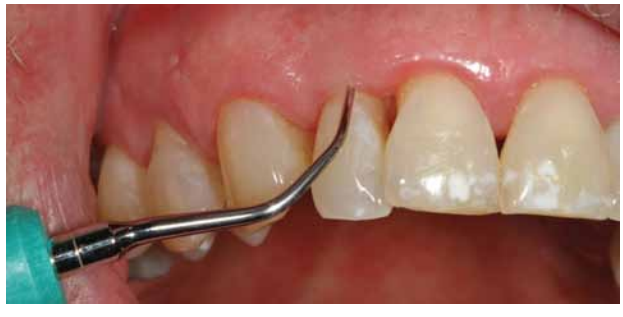
Tip designs available in standard diameter are straight with differing number of bends and include the #10, #100, #1000, and #3 (beavertail design). It is important to keep in mind that the objective of the scaling stage when heavier deposits are present is to reduce those deposits to a lesser degree. Hence, a straight insert provides a sufficient degree of contact to engage and reduce calcified deposits /stain, even in areas of more complex anatomy.

The #10 and #100 designs are similarly cylindrical in shape, with the #10 having one bend in the shank and the #100 having two bends in the shank. The length of either of these inserts is sufficient to enable contact of the active portion of the tip (terminal 4mm) to moderate-heavy subgingival calculus in deeper pockets.

The #1000, or triple bend, design features a third bend in the shank to facilitate adaptation around line angles and interproximally, as well as a beveled active area. This beveled edge design at the working end of the insert tip increases the efficiency of deposit removal as the ultrasonic energy is specifically targeted to each of the four corners rather than on the full circumference of a rounded working end. (Figures 2-4). With the length of the tip being reduced by the third bend in the shank, access to deep subgingival calculus with the #1000 is limited and better accessed by the #10 or #100.

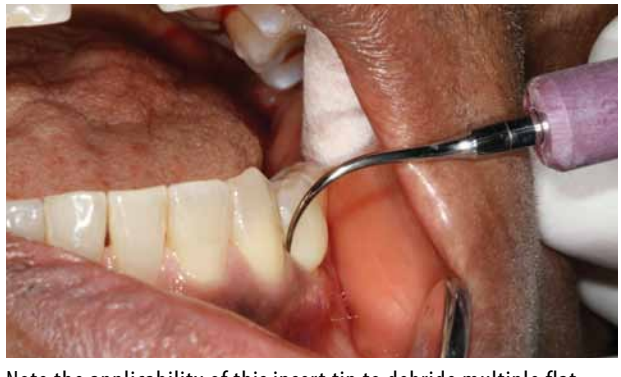
The #3 design, commonly recognized as the beavertail, is indicated for the breaking of heavy ridges of supragingival calculus and/or stain, most commonly on the lingual surfaces of the mandibular anterior teeth. Unlike the other inserts which utilize the terminal 4 mm of tip as the active area, the active area of the beavertail is the terminal edge of the tip, and is indicated for supragingival use only.

Figure 2. Triple bend insert tip



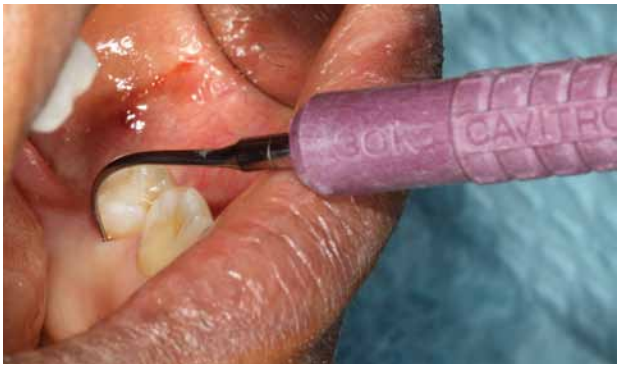
Note the applicability of this insert for line angles

Figure 3. THINSert tip lower left anterior quadrant



Note the applicability of this insert tip to debride multiple flat surface areas

Figure 4. THINSert tip lower left posterior area lingually



Slim Diameter Inserts

Slim diameter inserts are available with either straight or curved shanks.

Indicated for the definitive debridement of surfaces with flat or minimal contour, straight slim inserts include the popular #10 design as well as a newly available #1000 or triple bend design. As with the standard #10, the length of the slim #10 facilitates access to the depth of the pocket while the straight yet cylindrical shape conforms to relatively flat or minimally contoured surfaces, maximizing contact and therefore, disruption of biofilm.

The beveled tip and triple bends of the Slimline 1000 insert improve adaptation at line angles and access to interproximal and subgingival surfaces, offering the clinician an option for efficient debridement of minimally contoured surfaces while maximizing patient comfort

For definitive debridement of more complex root anatomy, as found in posterior roots, curved right and left inserts are needed to maximize access to and contact with contoured surfaces, and by rotating the curved tip, full access to the roof of furcations can also be achieved.

Ultra Slim Diameter Inserts

An ultra slim #10 insert with a 9° backbend (Cavitron THINSert) is available to facilitate thorough debridement of flat/minimally contoured surfaces where access with the slim #10 is limited due to tight contacts or relatively tight tissue. This insert is particularly useful for regular maintenance care (biofilm interruption) of patients with no significant hard deposits and minimal pocket depth (only relatively flat root surface involvement).

Patient Comfort

Patient comfort with the chosen method of debridement is essential. Tools to objectively measure patient pain perceptions in dentistry are lacking. Despite anecdotal claims to the contrary, there does not seem to be any significant difference in comfort levels of patients exposed to magnetostrictive or piezo devices.[33] Periodontal debridement, especially during the maintenance phase of therapy, typically requires relatively short working times at various sites throughout the mouth. For such procedures, traditional local anesthetic injections may not be the most suitable way to manage pain. Topical anesthetics that can allow for an adequate level of pain control without a needle injection may offer advantages. Indeed, patients are more likely to accept treatment with effective pain management, which increases comfort while reducing anxiety, yet a significant number of patients fear injections.[34] Thus, the appropriate use of needle-free anesthetics serves two purposes – pain management as well as reduced fear and anxiety compared to the use of local anesthetics. Topical anesthetics are convenient, but they offer short-duration anesthesia with variable pain control[35] and must thus be selected judiciously. A further option is the use of a locally applied noninjectable anesthetic containing 2.5 percent lidocaine and 2.5 percent prilocaine. This gel is syringed directly into the site of the pocket using a blunt cannula (without injecting it into the tissues) where site-specific anesthesia is then obtained. This anesthetic has been found to provide a level of anesthesia comparable to traditional injection anesthesia but without use of a needle, for twenty minutes' duration and without lingering anesthesia following completion of the procedure.[36,37,38]

Summary

Periodontitis is now seen as resulting from a complex interplay between bacterial infection and host response, often modified by behavioral factors, with the host response playing a key role. The overwhelming evidence confirming the adverse relationship between oral inflammation and systemic disease suggests that elimination of inflammation should be a goal of therapy in addition to preventing adverse changes in pocket depths and further clinical attachment loss. Mechanical disruption of the biofilm remains the foundational approach for the resolution of inflammatory periodontal diseases. Although similar clinical outcomes can be achieved with hand curettes and ultrasonic instrumentation, operator variability makes achieving therapeutic root debridement more predictable with ultrasonic instrumentation. Contact between the active portion of an ultrasonic insert at a preferred power level, proper angulation to the tooth surface and minimal force is essential for adequate biofilm interruption. Care should be taken to select tips that are appropriate based on the type of deposit and the anatomy of the surface to be debrided.

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Author Profile

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Dr. Timothy Donley is currently in the private practice of periodontics and implantology in Bowling Green, KY. After graduating from the University of Notre Dame, Georgetown University School of Dentistry and completing a general practice residency, he practiced general dentistry. He then returned to Indiana University where he received his Masters Degree in Periodontics. Dr. Donley is the former editor of the Journal of the Kentucky Dental Association and is an adjunct professor of Periodontics at Western Kentucky University. His course is part of the ADA Seminar Series. Dentistry Today recently listed him among the Leaders in Continuing Education. He lectures and publishes frequently on topics of interest to clinical dentists and hygienists.

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Questions

1. Mild to moderate forms of periodontitis affect _____ of adults.
   a. 10% to 30%
   b. 20% to 40%
   c. 30% to 50%
   d. none of the above

2. Severe generalized periodontitis affects _____ of adults.
   a. 3% to 12%
   b. 4% to 15%
   c. 5% to 15%
   d. 5% to 20%

3. The host-mediated destruction of connective and bone tissues in periodontitis is caused by a _____ response.
   a. hypoactivated immune-protective
   b. hyperactivated immune-protective
   c. hypoactivated immune-inflammatory
   d. hyperactivated immune-inflammatory

4. _____ remains the cornerstone of effective periodontal disease intervention.
   a. Effective intercession of periodontal bacteria
   b. Effective interruption of periodontal bacteria
   c. Removal of infected cementum
   d. none of the above

5. Periodontitis is now seen as _____.
   a. resulting from a complex interplay between bacterial infection and host response
   b. primarily the outcome from infection
   c. primarily the outcome from inflammation
   d. a and b

6. _____ of periodontal diseases now attributed to bacterial variance.
   a. 10 percent
   b. 20 percent
   c. 30 percent
   d. none of the above

7. The role of genetic variance in periodontal disease is now known to be _____.
   a. nonexistent
   b. insignificant
   c. significant
   d. none of the above

8. Focusing the management of periodontal disease solely on pocket depth _____.
   a. is sufficient
   b. is key
   c. may not be sufficient
   d. none of the above

9. _____ released orally in response to the pathogenic periodontal bacteria can enter the bloodstream.
   a. Bacteria
   b. Inflammatory mediators
   c. Bacterial byproducts
   d. all of the above

10. _____ factors accelerate destructive inflammatory processes in periodontitis.
    a. Acquired risk
    b. Environmental
    c. Genetic
    d. all of the above

11. _____ is strongly associated with increased risk for periodontitis and disease severity.
    a. Tobacco use
    b. Osteoporosis
    c. Obesity
    d. all of the above

12. _____ is not a non-oral risk factor for periodontal disease.
    a. Diabetes mellitus
    b. Depression
    c. Pulmonary embolism
    d. Mental stress

13. A therapeutic approach for periodontal disease should be developed _____ the level of risk.
    a. regardless of
    b. disproportionate to
    c. proportionate to
    d. a and c

14. There is general agreement that an isolated incidence of bleeding on probing at a site is _____ of disease activity at that site.
    a. an excellent predictor
    b. a good predictor
    c. a poor predictor
    d. a detractor

15. The predictive value of BOP increases substantially when BOP is _____.
    a. transient
    b. random
    c. persistent
    d. any of the above

16. Sites at which _____ are found should be priority candidates for therapeutic attention.
    a. increases in probing depth
    b. persistent bleeding on probing
    c. clinical signs of inflammation other than bleeding on probing
    d. all of the above

17. Suppressing the _____ has also been shown to play a critical adjunctive therapeutic role.
    a. thyroid gland
    b. host response
    c. antigen profile
    d. a and b

18. Dietary alterations intended to reduce the inflammatory response have also been shown to be _____ in periodontal therapy.
    a. of no benefit
    b. of benefit
    c. detrimental
    d. none of the above

19. Biofilm disruption can be accomplished by _____.
    a. mechanical means
    b. systemic and local administration of targeted antibiotics
    c. laser-generated energy
    d. all of the above

20. The selected debridement method should _____.
    a. be efficient to perform clinically
    b. be well tolerated by patients
    c. have a low potential for adverse side effects
    d. all of the above

21. Excessive cementum removal during instrumentation has been reported to result in _____.
    a. dentinal hypersensitivity
    b. enamel loss
    c. pulpitis
    d. a and c

22. According to the Academy of Periodontology, the goal of periodontal instrumentation is to _____.
    a. effectively remove plaque
    b. effectively remove calculus
    c. cause the least amount of root surface damage
    d. all of the above

23. Laser vibrotomy-based studies have demonstrated that as soon as the insert tip is loaded (placed against a tooth surface), _____.
    a. piezo driven instrument tips have elliptical patterns of movement
    b. magnetostrictive instrument tips have elliptical patterns of movement
    c. piezo driven instrument tips have vertical patterns of movement
    d. a and b

24. The risk of root surface can be eliminated during ultrasonic instrumentation by using a preferred _____.
    a. tip
    b. angulation
    c. lateral force at a preferred power setting
    d. all of the above

25. Tools to objectively measure patient pain perceptions in dentistry are _____.
    a. effective
    b. ineffective
    c. lacking
    d. none of the above

26. Slim probe-like insert tips remove _____ wider diameter insert tips.
    a. more root surface than
    b. the same amount of root surface as
    c. less root surface than
    d. none of the above

27. Walmsley theorized that cavitation activity causes _____ of the attached deposits.
    a. abrasion
    b. fracture
    c. erosion
    d. a and b

28. ____ of the biofilm remains the foundational approach for the resolution of inflammatory periodontal diseases.
    a. Mechanical disruption
    b. Chemical disruption
    c. Genetic disruption
    d. all of the above

29. The objective of the second stage of instrumentation is the definitive removal of all _____.
    a. heavy calculus, stain deposits, and biofilm
    b. endotoxins
    c. light calculus and stain deposits, biofilm and endotoxins
    d. a and b

30. It is prudent to use _____ specifically designed for calculus detection following any scaling procedure.
    a. a slim-tip insert
    b. an explorer
    c. a curette
    d. none of the above

Notes

ANSWER SHEET

Instrumentation for the Treatment of Periodontal Disease

Name: Title: Specialty:

Address: E-mail:

City: State: ZIP: Country:

Telephone: Home ( ) Office ( ) Lic. Renewal Date:

Requirements for successful completion of the course and to obtain dental continuing education credits: 1) Read the entire course. 2) Complete all information above. 3) Complete answer sheets in either pen or pencil. 4) Mark only one answer for each question. 5) A score of 70% on this test will earn you 2 CE credits. 6) Complete the Course Evaluation below. 7) Make check payable to PennWell Corp. **For Questions Call 216.398.7822**

Educational Objectives

1. List and describe the prevalence of periodontitis in the population
2. Describe the periodontal disease process
3. List and describe the associations between oral and systemic health
4. List and describe therapeutic options
5. List and describe the mechanism by which ultrasonic instrumentation works
6. List and describe tip selection for periodontal sites and the rationale for their selection

Course Evaluation

Please evaluate this course by responding to the following statements, using a scale of Excellent = 5 to Poor = 0.

1. Were the individual course objectives met? Objective #1: Yes No Objective #3: Yes No Objective #2: Yes No Objective #4: Yes No Objective #5: Yes No Objective #6: Yes No

2. To what extent were the course objectives accomplished overall? 5 4 3 2 1 0

3. Please rate your personal mastery of the course objectives. 5 4 3 2 1 0

4. How would you rate the objectives and educational methods? 5 4 3 2 1 0

5. How do you rate the author's grasp of the topic? 5 4 3 2 1 0

6. Please rate the instructor's effectiveness. 5 4 3 2 1 0

7. Was the overall administration of the course effective? 5 4 3 2 1 0

8. Do you feel that the references were adequate? Yes No

9. Would you participate in a similar program on a different topic? Yes No

10. If any of the continuing education questions were unclear or ambiguous, please list them.

_____

11. Was there any subject matter you found confusing? Please describe.

_____

_____

12. What additional continuing dental education topics would you like to see?

_____

_____

If not taking online, mail completed answer sheet to

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4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		21.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		22.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		23.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		24.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		25.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		26.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		27.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		28.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		29.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ		30.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

AGD Code 495

PLEASE PHOTOCOPY ANSWER SHEET FOR ADDITIONAL PARTICIPANTS.

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