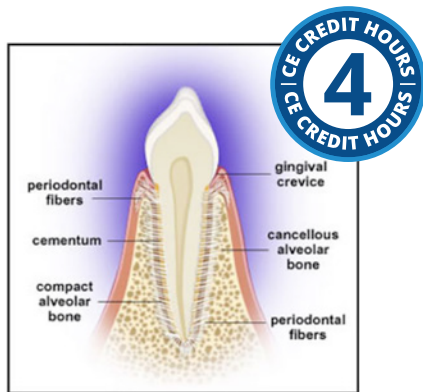


Periodontics: Oral Health and Wellness I. Understanding Periodontal Health, Recognizing Disease States and Choices in Treatment Strategies



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Disclaimer: Participants must always be aware of the hazards of using limited knowledge in integrating new techniques or procedures into their practice. Only sound evidence-based dentistry should be used in patient therapy.

Conflict of Interest Disclosure Statement

- The author reports no conflicts of interest associated with this course.

Introduction – Periodontics

Periodontics: Oral Health and Wellness I. Understanding Periodontal Health, Recognizing Disease States and Choices in Treatment Strategies provides information to assist clinicians in promoting the goals of oral health by first understanding periodontal health, recognizing disease states and providing choices in treatment strategies. This course reviews basic periodontal anatomy (to include connective tissue, bone, periodontal ligament and cementum) and physiology, periodontal disease classification, as well as the challenges, manifestations and implications of attachment loss. Evaluation of periodontal therapeutic strategies is best accomplished through a review of the scientific evidence on the topic. The objective of the therapeutic strategies reviewed is to improve the health and function of all periodontal attachment structures rather than only bone in isolation.

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Overview

Periodontal health depends on several factors, such as the periodontal microflora, oral hygiene, host response and other periodontal risk factors. Currently, we have yet to consistently and predictably restore periodontal attachment lost due to periodontal disease. The most frequently occurring periodontal disease is chronic periodontitis (formerly termed adult periodontitis). As the name implies, chronic periodontitis is often a slowly progressing disease which responds favorably to treatment and can be managed adequately; however, disease sequelae may not always be predictably restored.

Before one can recognize disease, one must understand periodontal health. The healthy periodontium is a strong yet flexible structure which attaches the teeth to the bones of the maxilla and mandible. In health, the periodontal attachment includes cementum, periodontal ligament, connective tissue and alveolar bone. The attachment bone plays a crucial role in maintaining the health and survival of the dentition.

This course provides information to assist clinicians in promoting the goals of oral health by first understanding periodontal health, recognizing disease states and providing choices in treatment strategies. The course reviews basic periodontal anatomy (to include connective tissue, bone, periodontal ligament and cementum) and physiology, periodontal disease classification, as well as the challenges, manifestations and implications of attachment loss. Evaluation of periodontal therapeutic strategies is best accomplished through a review of the scientific evidence on the topic. The objective of the therapeutic strategies reviewed is to improve the health and function of all periodontal attachment structures rather than only bone in isolation.

Learning Objectives

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

- Understand components of the healthy periodontium.
- Define periodontal health goals.
- Understand an overview of periodontal physiology and bone remodeling.
- Identify manifestations of diseased periodontium.
- Understand periodontal disease classification.
- Describe patterns of attachment loss and their prognostic and treatment implications.
- Evaluate choices of treatment strategies and how they meet periodontal health goals.

Introduction

This course provides information to assist clinicians in understanding the goals of periodontal health, which are to arrest disease progression, to improve clinical attachment levels and to give the patient a comfortable, esthetic and functional dentition that can

be maintained by both the patient and the therapist. It is essential to understand the role of the periodontal structures and why we need to maintain their health and function. The recognition and control of periodontal disease may help prevent future tooth loss and possibly may impact systemic health as well.

It is important for the clinician to focus on oral health goals and to make therapeutic choices that are supported by the best available scientific evidence. Evidence-based practice decision-making relies on a process of identifying and evaluating relevant information on diagnostics, treatments and prognoses.¹ Reliance on clinical experience and intuition, or poorly designed studies alone can be misleading. Inductive reasoning and uncontrolled observations often lead to false conclusions due to factors such as bias, the placebo effect (an improvement in health not attributable to treatment) and the nocebo effect (an ill effect resulting from the suggestion or belief that something is harmful). The findings of randomized, controlled clinical trials provide the gold standard for the highest level of evidence. In order for the study to be valid it must be reproducible, answer an appropriate question and measure the pre-determined variable among other criteria. Success criteria for clinical significance should be defined prior to the start of the study. Consecutive controlled case studies may be considered to demonstrate clinical success, but not clinical predictability. Case reports can show the potential for a technique to have a desired outcome. Variables that may confound study outcomes include bias, chance events not addressed by statistics, including systemic influences, diet, materials and patient expectations and their attributes. For comprehensive courses on understanding and evaluating a research report, please review the following:

- [Using Research for Clinical Decision-Making: Evaluating a Research Report](#)
- [Using Research for Clinical Decision-Making: The Elements of a Research Report](#)

This course will start by defining periodontal oral health and wellness goals for the periodontal patient. It will also review the concepts of periodontal health, physiology

and disease manifestations. Finally the course will evaluate the evidence supporting each therapeutic option and how it meets these goals.

Periodontal Health Goals

Define Periodontal Health Goals

The first learning objective of this course is to define periodontal health goals. Health has been defined as freedom from physical disease or pain. The American Academy of Periodontology (AAP) [Guidelines for Periodontal Therapy](#) position paper states the goals of periodontal therapy “are to preserve the natural dentition, periodontium, and peri-implant tissues; to maintain and improve periodontal and peri-implant health, comfort, esthetics, and function. Currently accepted clinical signs of a healthy periodontium include the absence of inflammatory signs of disease such as redness, swelling, suppuration, and bleeding on probing; maintenance of a functional periodontal attachment level; minimal or no recession in the absence of interproximal bone loss; and functional dental implants.”² Periodontal therapies will be evaluated as to whether or not, and to what degree they meet periodontal health goals.

Measuring Periodontal Health

The second learning objective of this course is to identify manifestations of diseased periodontium. The first step in identifying disease manifestations is to understand periodontal disease epidemiology and the degree to which its manifestations have been measured.

Periodontitis occurs frequently in the U.S. adult population. The exact prevalence of the disease in the U.S. and worldwide has been estimated to affect 20% of the adult population, but has not been definitively determined because studies lack a consistent definition of the disease and a consistent methodology. A large U.S. 1990 study by Brown et al of 15,132 subjects aged 18-64 years provides an insight into the epidemiology of periodontal diseases. It reported that 4-6 mm pockets were found in 13.4% of subjects, or 0.6 sites/subject and 1.3% of sites assessed. The same study reported pockets of 7 mm or greater were found far less frequently, in 0.6% of subjects, or 0.01 sites/subject and 0.03% of sites assessed. Additionally, the study reported that attachment loss greater or equal to 3 mm

was found in 44% of subjects, increasing with age and affecting an average of 3.4 sites/subject. Attachment loss greater or equal to 5 mm was found in 13% of subjects, increasing with age and affecting an average of 0.7 sites/subject.³ Because periodontal disease is relatively widespread, clinicians routinely see its manifestations.

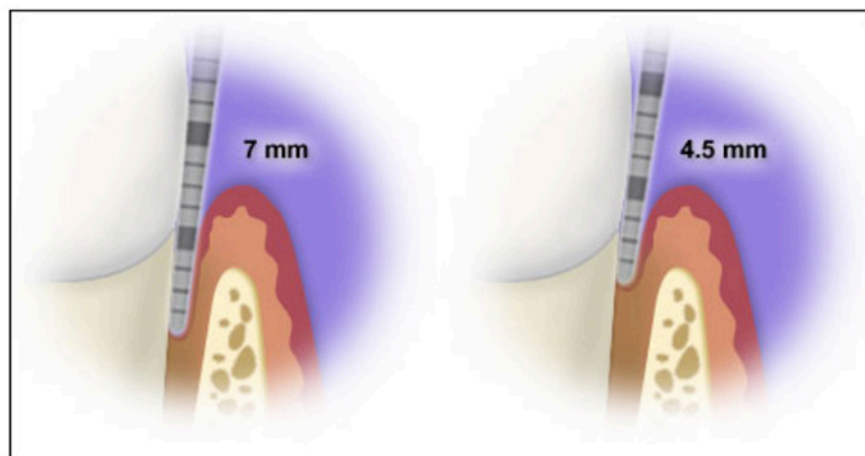
Disease severity ranges along a continuum of stage 1 to stage 4, and grading is determined to predict whether disease progression is slow, moderate, or rapid. Periodontitis often affects different areas of the mouth to different degrees. It is usually progressive, characterized by bursts of disease progression followed by periods where the disease is more quiescent. If left untreated, tooth loss may result due to the progressive nature of the disease. Clearly the goal is to attain and/or preserve oral tissue health with appropriate therapies. Success can be measured directly by preserving the dentition and periodontium, which includes the gingiva, periodontal ligament, root cementum and alveolar bone. Surrogate markers of success in measuring periodontal health include maintaining clinical attachment levels and reducing or eliminating pocket depths, inflammation, infection and bleeding on probing. Probing pocket depth (PPD) is a measurement of the distance between the gingival margin and the base of the probable crevice. Clinical attachment loss (CAL) is a measurement of the distance between the cemento-enamel junction (CEJ) and the base of the probable crevice or pocket. Furcation involvement may range from 1st degree, where

horizontal loss of support is less than 1/3 of the width of the tooth, to 3rd degree, where there is a horizontal loss of support from one side of the furcation through to the other side. Mobility can range from degree 1 (0.2-1 mm horizontal crown motion), to degree 2 (greater than 1 mm horizontal crown motion), to degree 3 (horizontal and vertical crown motion).

The Gingival Index, published by Löe in 1967, scores a 0 for no visible signs of inflammation, a 1 for slight change in color and texture, a 2 for noticeable inflammation and bleeding upon probing and a 3 for overt inflammation and spontaneous bleeding. The Plaque Index, published by Silness & Löe in 1964, scores plaque deposits on a 0-3 scale where 0 indicates that plaque is absent, 1 indicates plaque detected by gingival marginal probing, 2 indicates visible plaque and 3 indicates abundant plaque.

It is important to keep in mind that some of these measures are markers of past disease and do not reliably predict future disease progression. The presence or absence of inflammation have been clinically assessed by gingival redness, suppuration, bleeding on probing (BOP), measurements of gingival temperature and gingival crevicular fluid (GCF) and supragingival plaque. Further measures of success include maintaining and improving periodontal comfort, esthetics and function.

The patient's current, and historic periodontal health and plaque control status serves as a baseline when setting goals. The presence, absence, history, extent and severity of



Periodontal Probing

periodontal diseases indicate the need for various therapies. The Oral Risk Assessment (ORA) and Early Intervention System provide a methodology to organize patient "CARE":

- Collection of relevant medical and dental information, including full mouth probing of depths, full mouth radiographs, and missing teeth
- Assessment and assimilation of the collected information to establish staging and grading of periodontitis
- Recommendation of professional therapies and home care procedures and products
- Evaluation of treatment and healthcare outcomes

Periodontal health goals are set given the current health status of the patient, patient expectations, capabilities and values. Goals and therapies are defined for and with each patient prior to the development of a treatment plan. Patients are informed of the diagnosis of their condition, possible cause(s), therapeutic options and alternatives, susceptibility, risks and prognoses. Goals are set to achieve and maintain the nearest to an ideal state of periodontal health that the limitations and the degree of compliance of the patient will allow.

Systemic Health Implications

The patient's current systemic health also impacts the setting of periodontal health goals. Systemic conditions, infections, anomalies, trauma, etc. contribute to gingival and periodontal diseases. For example, diabetes, endocrine effects, medications, malnutrition, hematologic disorders, genetic disorders as well as other systemic considerations may affect oral health.

Finally, goals may be set for the possible benefit to systemic health that may follow improved periodontal health. For example, researchers are currently investigating the potential for an increased incidence of pre-term, low birth weight infants coinciding with increased incidence of periodontal diseases.⁴ Likewise the potential for a cause and effect link between periodontal disease and cardiovascular disease is under investigation. Additionally "periodontitis has recently been associated with the higher risk of ICU admission, needed assisted ventilation,

and death in COVID-19 patients, and with increased blood levels of biomarkers linked to worse disease outcomes."²⁶. Theoretically the systemic inflammatory response that can result in severe COVID-19 patient outcomes may be impacted by the systemic inflammation characteristic of periodontitis.

Anatomy in the Healthy Periodontal State

Anatomy in Health

The alveolar process is the portion of the mandible and the maxilla that supports the teeth. Tooth attachment is attained by the alveolar bone, the root cementum and the periodontal membrane. Compact bone lines the alveolus, or tooth socket, and is seen radiographically as the lamina dura. Cancellous bone, containing bone trabeculae, is found between the tooth sockets.

Alveolar bone is not a static structure; it constantly remodels through the action of osteoblasts and osteoclasts. Alveolar bone remodels in response to local factors such as directional forces, host response to microbial plaque and infection. It is also affected by systemic factors such as hormones, glucocorticoids and various disease states.



Bone Physiology Review⁸

The third learning objective of this course is to understand an overview of bone physiology and remodeling. Bone is often thought to be a static structure, but it actually is continuously remodeling through actions of osteoclasts (bone-resorbing cells) and osteoblasts (bone-building cells) and their products, under the influence of parathyroid hormone (PTH), vitamin D, estrogens, glucocorticoids, growth hormone, thyroid hormone and other factors such as cytokines, a unique family of growth factors. Remodeling is accomplished in the trabecular bone of the alveolar process, and elsewhere, by a team of cells that dissolves a pit-like area in bone and then fills it with new bone. This team of cells is called the Basic Multicellular Unit (BMU). Bone is remodeled through a sequence of steps that may take as much as 200 days, as follows:

Origination

Origination is the phase during which a BMU originates following an initiating “event” such as microdamage, mechanical stress, exposure to one or more of a group of biological factors, or even at random. The immune response begins in reaction to one of these events. Cytokines and other growth factors, such as parathyroid hormone (PTH), insulin-like growth factors (IGF), interleukin-1 (IL-1), interleukin-6 (IL-6) and tumor necrosis factor (TNF), are important in the Origination phase. IL-1 may be the most important factor in the immune response. Its function is to enhance the activation of T-cells in response to antigen. IL-6 is produced by fibroblasts and other cells. IL-6 enhances glucocorticoid syntheses. Overall it augments the response of immune cells to other cytokines. Tumor Necrosis Factor- α (TNF- α), like IL-1, is a major immune response-modifying cytokine produced mainly by activated macrophages. The presence of TNF induces osteoclast formation.

Factors such as estrogen can reduce occurrence of the Origination phase, thereby reducing the rate and occurrence of bone resorption. Biologically mediated strategies for improving bone growth in periodontitis patients work to modify the effects of factors that promote

bone resorption or to boost the effects of the factors that promote bone growth.

Osteoclast Recruitment

The lining cells that were activated during Origination secrete RANK-ligand, which may remain bound to the cell surface. Pre-osteoclasts are activated by RANK-ligand (RANKL) and then differentiate into mature osteoclasts which develop a ruffled border and resorb bone. Osteoprotegerin (OPG) can act to bind the RANK-ligand which reduces its effect. RANK-ligand is a potent bone-building agent. Osteoclasts are more effective at resorbing bone when RANK-ligand’s effect is reduced.

Resorption

Bone is resorbed by the mature osteoclasts for approximately two weeks at a given location until the osteoclasts undergo pre-programmed cell death. New osteoclasts are continuously activated as the BMU travels. Integrins and interleukins are immune factors that can act to increase osteoclast activity. Integrins are cell-surface receptors that bind ligands and reduce their bone-building effect.

Estrogen, calcitonin, interferon and TGF can reduce bone resorption during this phase. Calcitonin works in opposition to parathyroid hormone and can reduce its role in bone resorption. Similarly bisphosphonates, such as risedronate sodium inhibit osteoclast mediated bone resorption thereby reducing net bone loss. Additionally, researchers recently found and reported “that double stranded RNA molecules can activate the immune system response that leads to deterioration of bone”.

²⁷ This finding of an additional underlying mechanism of periodontitis progression may lead to new preventative measures.

Osteoblast Recruitment

Osteoblasts are derived from bone marrow stromal cells and are attracted by bone-derived growth factors, (including Cbfa1, BMP’s, IGF, PTH, and others) and perhaps the remains of the self-destructed osteoclasts. Cbfa1 activates the bone-specific protein, osteocalcin. Bone morphogenetic proteins

(BMPs) induce new bone formation by stimulating proliferation and migration of undifferentiated bone cell precursors.

Osteoid Formation

Osteoblasts start the process of bone-building by secreting layers of osteoid to slowly refill the cavity, as well as growth factors (including TGF-beta, BMP's and IGF) and proteins. The presence of glucocorticoids may retard osteoid formation.

Mineralization

The osteoid begins to mineralize utilizing calcium and phosphate when it is approximately 6 microns thick. Mineralization is controlled by osteoblast activity. The presence of pyrophosphate may reduce mineralization.

Mineral Maturation

Bone density increases over months after the cavity has been filled with bone because the mineral crystals become more closely packed.

Quiescence

During quiescence, some osteoblasts become lining cells which help regulate ongoing calcium release from the bones. Other osteoblasts become osteocytes which remain in bone, connected by long cell processes, which can sense functional stress on the bone.

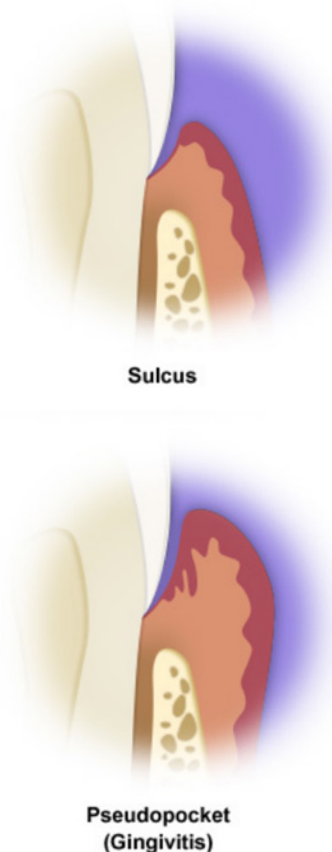
American Academy of Periodontology Disease Classification

The fourth learning objective is to understand periodontal disease classification. The AAP updated its classification system for periodontal diseases in 2018 to create a common terminology compatible with scientific knowledge of periodontal diseases.²⁵ Major changes included a new section classifying gingival diseases, replacement of the term "chronic periodontitis" with the term "periodontitis" and replacement of severity designations in specific locations with full mouth staging and grading levels.²⁵

Other less commonly occurring categories of periodontitis include disease associated with genetic disorders, necrotizing ulcerative

periodontal diseases, abscesses of the periodontium, periodontitis associated with endodontic lesions and developmental or acquired deformities and conditions.

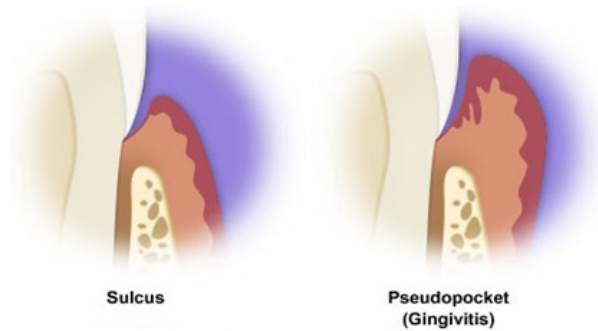
Periodontitis is commonly experienced by adults and it is known to occur in adolescents as well. Epidemiologic studies currently do not provide enough information to report exact prevalence due to the lack of consistency in study design, outcome measures and endpoints. In the 1996 Annals of Periodontology, Volume 1, Number 1, November, 1996, a review of the published data found that more severe forms of periodontal disease occur in approximately 10% of the population in developed countries, but that moderate attachment loss is more widespread. Variables of age, plaque and bleeding on probing are all found to be related to disease incidence and severity.⁶ Periodontitis is usually, but not always, a slowly progressing form of periodontitis



that often responds to treatment. The rate of progression is captured by grading the disease. For example, periodontitis staging considers severity via interdental clinical attachment loss (CAL) as slight at 1-2 mm, moderate at 3-4 mm, and severe at 5 or more mm of loss. Staging also considers disease complexity and extent or distribution. Periodontitis grading considers evidence of progression and risk factors that modify grading.²⁵

Studies show that patients may experience periods of relative quiescence and bursts of rapidly progressing disease activity.⁷ This course focuses on periodontitis; by far the most common form of the disease.

Anatomy in Diseased Periodontal States



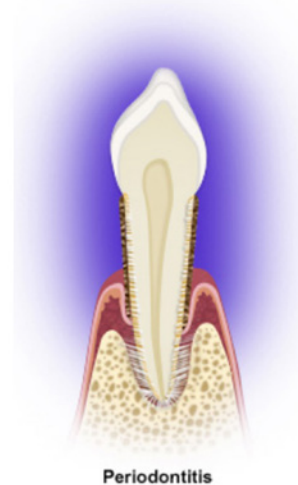
Recognizing Diseased Periodontal States

Gingivitis and periodontitis are classified as separate diseases. Gingivitis is an inflammation of the marginal gingiva that does not produce attachment loss nor loss of bone. Pockets that may occur with gingivitis are actually pseudopockets, and are due to gingival enlargement and do not involve apical migration of the gingival attachment nor bone loss.

Periodontitis occurs when the junctional epithelium and periodontal attachment move apically along the tooth root. Alveolar bone also resorbs towards the apex of the tooth during the disease process. It is believed that pathogenic bacterial plaque induces an inflammatory immune response, which may compromise periodontal structures. The plaque would be comprised of pathogenic organisms in numbers large enough to effect a response

in a susceptible host. Genetic and systemic factors may affect these events. Since bacterial plaque is generally considered the common denominator of periodontal disease, there has been a strong focus on antimicrobial strategies including scaling and root planing and routine oral hygiene measures.

Bone loss is routinely evaluated during the periodontal examination. Clinically, tooth mobility is measured as well as furcation involvement. Tooth mobility may vary from 1st degree, a horizontal movement of the tooth crown up to 1 mm, to 3rd degree, both horizontal and vertical movements of the crown. Radiographically, the alveolar bone is examined for height and horizontal or vertical bone loss and signs of furcation involvement. Radiographic bone loss findings may be

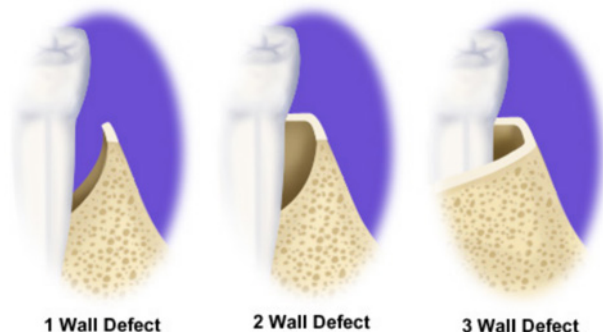


confirmed by direct visualization if open flap surgery is performed. If bone loss is horizontal, bone grafting is not indicated. If bone loss forms in an angular fashion forming infrabony defects, regenerative procedures may be helpful.

Analysis and Diagnosis of Attachment and Bone Loss Based on Morphology

The fifth learning objective of this course is to describe patterns of bone loss and their prognostic and treatment implications. Bone lost to chronic periodontitis can create bone defects with varying characteristics. For example, intrabony defects can have 1, 2 or 3

walls, they can be wide or narrow; and they can be shallow or deep. In general, deep and narrow defects with more bony walls have the greatest and most predictable chance for successful gain in attachment following periodontal surgery. Other factors strongly influencing the chances



for attachment gains include the patient's local, behavioral and systemic conditions and characteristics. Plaque control, smoking, systemic conditions clearly affect surgical outcomes. Other factors such as bone lost in furcation areas are complicating considerations.⁹

Bone-Preserving and Bone-Augmenting Therapeutic Options

Goals

The goals of achieving and maintaining periodontal health lead to the endpoint of obtaining a healthy and functional dentition for life. We track progress towards that endpoint by assessing the health of the periodontal attachment apparatus structures. Therapeutic options are evaluated by the degree to which those outcomes are met, as well as with the relative costs (measured in dollars, time, morbidity, comfort, esthetics, etc.) required to achieve them. All therapies which assist in limiting the deleterious effects of the host response to periodontal pathogens can be considered bone-preserving, as well as tooth-preserving therapeutic options. The most effective therapies may be those most under-rated in the minds of some dental professionals. Adequate plaque control by the patient and routine scaling and root planing and adjunctive therapies in the general practice and periodontal practice are essential to attain and maintain periodontal health for most

patients. Surgical therapies complement non-surgical therapies to provide a range of valuable options.

Gordon Douglass, installed as president of the AAP September 25, 2002, addresses treatment goals and strategies in his January 24, 2003 letter to the Medical/Health Editor of the New York Times, AAP treatment guidelines have always stressed that periodontal health should be achieved in the least invasive and most cost-effective manner. This is often accomplished through non-surgical periodontal treatment, including scaling and root planing (a careful cleaning of the root surfaces to remove plaque and tartar from deep periodontal pockets and to smooth the tooth root to remove bacterial toxins), followed by adjunctive therapy such as systemic and local delivery antimicrobials and host modulation, as needed and on a case-by-case basis. Most periodontists would agree that after scaling and root planing, many patients do not require any further active treatment, including surgical therapy. Most periodontists also agree that determining surgical needs before assessing a patient's response to non-surgical therapies is putting the cart before the horse. In fact, surgery is reserved for those situations when non-surgical therapies have failed to achieve the desirable clinical outcome of periodontal health, and to repair damage to gum tissue and bone as a result of periodontal disease."

Therapeutic Options

Overview

The sixth learning objective of this course is to evaluate whether bone preserving/augmenting strategies meet periodontal health goals. The dental professional's recommendations to the patient for plaque control procedures and products are essential to achieving the goal of a healthy periodontal attachment for life. Likewise, non-surgical approaches such as scaling and root planing (SRP), locally-delivered sustained-release antimicrobials and sub-antimicrobial systemic tetracyclines have been proven to improve periodontal health (measured by factors such as reduced pocket depth, increased attachment levels, increased bone, decreased need for surgical therapies)

in prospective, randomized, controlled clinical trials, the gold standard of evidence-based practice. Most surgical therapies have been clinically evaluated by the results of a series of case reports and the outcomes of research with animal models. In general, surgical therapy trials cannot be double-blinded and clinical outcomes may be less predictable due to variability in factors such as practitioners' skill, experience, bias, procedures, reproducibility of outcome measures, etc. Additionally, there may be a lack of consensus as to what constitutes acceptable periodontal health. Some may consider the retention of teeth and the absence of the signs of infection as acceptable endpoints. Others may consider optimum bone height as important. Despite these controversies and concerns, periodontal osseous and regenerative surgical therapies provide an option that is very effective for many appropriate patients. Surgical and non-surgical therapies can work together to meet patients' needs in managing a chronic, non-curable disease; although currently long term clinical data is mixed when comparing the outcomes of surgical therapies with those of non-surgical therapies.¹⁰ Most agree that the goal for all periodontal therapies is to reduce, eliminate and/or repair the damage to the periodontal structures resulting from the deleterious effects of the host response to pathogenic organisms.

Plaque Control Procedures and Products

The nature and severity of chronic periodontitis is a result of the interactions between microbial factors and a susceptible host. Pathogenic bacteria in the gingival sulcus or periodontal pocket are necessary for periodontitis to occur, but bacteria alone are only part of the picture. It is well known that the body's local and systemic response to bacteria and their toxins determines the extent and severity of the disease. Controlling pathogenic bacterial colonization minimizes the host response. That is best accomplished by routine brushing, flossing and otherwise disrupting plaque and biofilm. For more information on biofilm, please refer to the dentalcare.com course:¹²

- [Biofilm: A New View of Plaque](#)

Routine plaque control is the most reliable strategy in the prevention of bone and soft

tissue destruction and attachment loss that occurs with periodontal disease. One of the most important services the dental professional can provide is assistance with improved daily plaque control by the patient. The chances for plaque control improvement are increased when products with efficacy supported by the best available scientific evidence are recommended. Twice daily use of an effective toothbrush is critical to ensure efficient plaque removal. In a systematic review by the Cochrane Collaboration, power toothbrushes with rotation-oscillation motion were found to be better than manual toothbrushes at removing plaque and reducing gingival inflammation. No other powered designs were as consistently superior to manual toothbrushes.²² In a separate Cochrane review comparing power brush technologies, rotation-oscillation toothbrushes were found to reduce plaque and gingivitis more than those with side-to-side (sonic) action in the short term (4 to 12 weeks).²³ Another way to ensure adequate plaque control is use of chemotherapeutic agents to inhibit plaque regrowth. Dentifrices containing stabilized stannous fluoride or triclosan and oral rinses with bioavailable CPC or essential oils have been shown to reduce plaque and gingivitis. Published studies indicate dentifrice with stabilized stannous fluoride offer greater plaque control and gingival health advantages relative to other over-the-counter therapeutic dentifrices. Attaining routine plaque control by the patient is a very important factor in periodontal therapy. Some of the long-term success of surgical therapies can be attributed to increased access to sites that have been adversely affected by periodontal disease; giving the opportunity for better plaque control by the patient.

Scaling and Root Planing

While there is some lack of agreement as to what constitutes the optimal technique and procedural time and interval for each patient, scaling and root planing (SRP) is considered to be the gold standard for non-surgical periodontal therapies. Cobb reviewed numerous non-controlled clinical trials and case reports and found SRP to be very effective in improving clinical parameters, with pocket depth reductions averaging 2 mm, for chronic periodontitis patients.¹³ Properly controlled,

blinded, and powered clinical trials generally show a mean probing pocket depth (PPD) reduction of approximately 1 mm with SRP therapy alone.^{14,15,16} It is interesting that case reports and consecutive case studies summarized in the 1996 World Workshop generally show a mean PPD reduction of approximately 1.5 to 2 mm, which is nearly 1 mm greater, or almost 100% greater, than the PPD reduction found in randomized, controlled clinical trials. A breakdown of the groups shows that initially moderately deep sites of 4-6 mm show a mean pocket depth reduction of 1.29 mm; while the initially deep sites of ≥ 7 mm show a mean pocket depth reduction of 2.16 mm. Case study outcomes are generally considered to be less reliably predictive of expected patient outcomes due to the influence of non-controlled confounding variables and the influence of bias. There is still a need for more research to answer questions about the relative benefits of full-mouth disinfection vs. quadrant SRP, shorter vs. longer procedural time per tooth, etc.

Antiseptics

Antiseptics, delivered via rinsing and irrigation, have been shown to be effective in controlling gingivitis, but not periodontitis. The agents generally are not retained at the site long enough for their antimicrobial effect to provide a measurable benefit to pocket depth and/or attachment levels.

Locally-Delivered Sustained-Release Antimicrobials

Controlled clinical trials have consistently shown that locally-delivered sustained-release antimicrobials, along with scaling and root planing, have been shown to provide a clinically and statistically significant increase in the percentage of patients achieving predetermined periodontal benefits, than does scaling and root planing alone.¹⁵ Some trials have also shown that an agent alone can reduce probing depths as much as SRP alone.¹⁶ Generally, it is agreed that these agents provide better outcomes than SRP alone in sites where patient access for plaque control might otherwise be limited; i.e. wherever pocket depths are 2 mm or greater. Improved outcomes can be obtained with these products during both active and maintenance therapy.

Bacterial resistance to antibiotic therapies have not been reported, but resistance concerns can be avoided by the use of the locally-delivered sustained-release antimicrobial containing chlorhexidine, an antiseptic rather than an antibiotic which is not known to induce the emergence of resistant strains.

There remains a question as to whether these agents are appropriate for routine use or for only those instances where previously available therapies may fail. Generally, surgical needs are assessed after the patient receives optimal non-surgical therapies. All appropriate strategies should be considered in the effort to control a chronic disease like chronic periodontitis.

Examples of these products which have been available in the U.S. include tetracycline fibers, chlorhexidine chip, doxycycline polymer, and minocycline microspheres.

A 3 month phase II clinical trial found that 0.4% moxifloxacin gel, a fourth-generation fluoroquinolone, administered in a single subgingival administration adjunctive to scaling and root planing, resulted in additional pocket depth reduction compared with scaling and root planing plus a placebo gel.²⁰ Moxifloxacin is a broad spectrum antimicrobial active against aerobic and anaerobic bacteria. These results would need to be confirmed in a phase III clinical trial. Phase II trials generally assess efficacy and dose response prior to designing a larger phase III trial. Phase III studies are generally randomized controlled multicenter trials on larger groups of patients. The goals of a phase III trial usually compares efficacy as compared to the current gold standard treatment.

Another 9 month clinical trial indicated that microsphere formulation of doxycycline provided both a more sustained release and a high initial drug concentration as compared with doxycycline gel. All three groups (scaling and root planing alone, doxycycline microspheres and doxycycline gel) showed improvements in relative attachment levels at 9 months. The group receiving scaling and root planing plus doxycycline microspheres showed significantly greater gain in relative attachment

levels than the other groups.²¹ Again further research is needed comparing other agents and delivery systems.

Systemic Antibiotics

The advantages of systemic antibiotics include the ability for the drug to control pathogens in all periodontal tissues and other sites where they may have an impact. They may help to reduce future colonization. Another advantage may be a relatively low financial cost to the patient. However, systemic antibiotics may not reach the concentrations in the periodontal pocket needed to do the job, may be involved in drug side effects and interactions, may be subject to compliance issues and have been known to select and favor the growth of organisms that are not susceptible to the agent. Systemic antibiotics are not routinely used in chronic periodontitis due to increased concern about the induction of resistant organisms. They may be utilized in appropriately selected patients, such as refractory cases, along with other plaque control strategies.¹⁷

Sub-Antimicrobial Systemic Dosing of Doxycycline

Doxycycline is a tetracycline derivative. When it is dosed at 20 mg twice daily, the resulting plasma levels are below those required for an antimicrobial effect; therefore, the mechanism of action of this antibiotic is not antimicrobial, it reduces or inhibits the activity of collagenase, an enzyme produced by the body as a part of its host immune response to a bacterial challenge. For chronic periodontitis patients, when used in addition to SRP, sub-antimicrobial doxycycline is more effective than SRP alone for reducing probing pocket depth and for gaining clinical attachment. Tolerability was similar to placebo, but the usual tetracycline safety limitations apply. For example, systemic doxycycline is neither indicated for pregnant or nursing patients, nor for patients hypersensitive to tetracyclines. Additionally, compliance may not be optimal over the entire treatment period. This is an important new option to be considered for chronic periodontitis patients and the available evidence suggests it significantly increases the chance for better outcomes than SRP alone.^{18,19} Other host response modulating therapies have been studied including topical and systemically

administered non-steroidal anti-inflammatory drugs (NSAID's). Agents such as triclosan and bisphosphonates have been studied due to their potential to interfere with the periodontal disease process. Bisphosphonates may help inhibit bone loss.

Periodontal Surgery

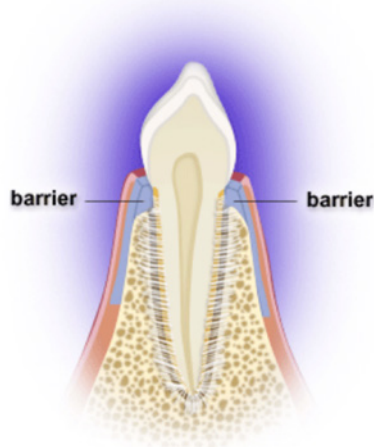
Osseous Surgery

Osseous surgery is generally considered to be resective surgery. The goal is to re-contour bone and tissues so that they are amenable to periodontal health and to allow for increased access for plaque control by the patient.

Regenerative Surgery

Regenerative procedures add the potential to restore lost tissues and attachment to the goals of osseous surgery. When the disease process is halted, or sufficiently slowed, the restored structures could be lasting. The procedures and materials include barriers to prevent the epithelium from growing into the bony defect and to provide an increased opportunity for bone growth within the defect. Other regenerative procedures include osseous grafting utilizing one of a large variety of natural and synthetic materials currently available. Biologically mediated strategies include matrix-derived proteins and/or growth factors.

Following periodontal surgery, epithelial, gingival connective tissue, bone and/or periodontal ligament cells may grow along the root surface. Barriers are often used to help guide periodontal ligament cells to grow along



the root surface to restore lost tooth support, while preventing the epithelial and gingival connective tissue cells from entering the space. Gains in probing attachment and bone fill may result. The process is called guided tissue regeneration, and one can utilize resorbable or non-resorbable barriers, calcium sulfate, etc. Non-resorbable barriers by their nature last longer, but require removal at a later surgery. Resorbable barriers are used more frequently, but some of them may resorb prior to the length of the time needed for optimal bone fill.

Many osseous grafting materials are currently available. Osseous grafts are generally most predictably successful in treating narrower and deeper 3-walled bony defects. Bony defects with fewer walls have less predictable bone fill results. Barriers can provide containment for grafts as well.

Bone grafting to stimulate bone deposition has been used in periodontal surgery since the 1970's. It involves a surgical procedure to place bone or bone substitute material into a bone defect with the objective of producing new bone and possibly the regeneration of periodontal ligament and cementum. Over time, most of the graft material will be replaced by new bone. In fact, over time all bone is eventually replaced by new bone as illustrated in the bone physiology review.

Autografts utilize the patient's bone, which can be obtained from intra-oral or extra-oral sites. They are the best materials for bone grafting, are very well accepted by the body and may produce the fastest rate of bone growth; however, there is the potential risk of additional discomfort and a secondary procedure. With autografts, the patient is assured of protection from disease transmission and/or immune reaction.

Allografts are obtained from another human source, typically highly processed bone powder from human cadavers. The age and health of the donor can affect the rate of bone regeneration. The risk of disease transmission and/or rejection is handled by processing and quality control. The allografts are freeze-dried at ultra-low temperatures and dried under high vacuum. They are available either demineralized or non-demineralized. Unlike

synthetic bone, which only provides scaffolding for osteoconduction, allografts include growth factors which are also osteoinductive. Allografts both induce bone growth and provide an environment that increases the body's regenerative process.

Xenografts are obtained from animal sources; usually cows and/or pigs. They include processed animal bone or growth proteins. Again, the risk of disease transmission and/or rejection is reduced by processing. A synthetic cell-binding peptide (P-15) combined with anorganic bovine-derived hydroxyapatite bone matrix (ABM) mimics autogenous bone and has been proven superior to grafts from some other sources. Like autogenous bone, ABM/P-15 is made of organic and inorganic components. The inorganic component in humans is calcium phosphate. ABM/P-15 contains identical calcium phosphate from a bovine source. The organic component of autogenous bone is collagen, a fibrous tissue which is important for cell binding and proliferation. The collagen component responsible for this function is P-15, which is synthetically replicated and irreversibly attached to the inorganic component. The processing at very high temperatures removes the risk of disease transmission or immune reaction. ABM/P-15 was evaluated in 31 patients and found to have promise as a bone replacement graft in 1, 2 and 3-wall bony defects along with open flap mechanical debridement, followed by 3-month recalls for three years.

Synthetic bone grafting materials carry no risk of disease transmission or immune system rejection. They help create an environment that facilitates the body's regenerative process. Examples of synthetic materials include natural and synthetic hydroxyapatites, ceramics, calcium carbonate (natural coral), silicon-containing glasses, and synthetic polymers.

Biologically mediated strategies include materials, such as enamel matrix proteins, that can be pre-mixed with vehicle solution. They are intended as an adjunct to periodontal surgery for topical application onto exposed root surfaces or bone to treat intrabony defects and furcations due to moderate or severe periodontitis. Enamel matrix protein

leaves only a resorbable protein matrix on the root surface which makes bone more likely to regenerate. These bone morphogenetic proteins help to initiate a cascade of events leading to the differentiation of progenitor cells into phenotypes involved in periodontal regeneration.

Growth factors and various extracellular matrix (ECM) proteins in bone regulate cell attachment. These proteins provide the osteogenic capacity observed with autografts and allografts. That is, these components provide the chemical cues to initiate and maintain those processes associated with bone formation. Synthetic bone graft materials, whether polymer, ceramic, etc., provide only an inherent osteoconductive capacity. They provide only a physical platform to support cell attachment and tissue development. By incorporating such biological growth factor components within synthetic bone scaffolds a closer imitation of human bone graft material is achieved. Thus a synthetic bone graft can be achieved with the inherent osteogenic properties of an allograft, without the immunological rejection or disease threat potential, and with the inherent osteogenic properties of autograft, without the

complications often associated with the graft donor site.

Conclusion

- Periodontitis results in the loss of periodontal attachment structures. If left untreated, tooth loss eventually may result.
- The currently available scientific evidence supports consistent plaque control at home and the routine use of SRP along with locally-delivered sustained-release antimicrobials and sub-antimicrobial systemic dosing of doxycycline in the professional treatment of chronic periodontitis.
- Osseous and regenerative surgery has been shown to provide desirable outcomes in consecutive case reports and animal models.
- Surgical and non-surgical therapies are complementary options available for consideration over time when managing the occurrence and progression of a chronic disease.
- More scientific evidence is needed to answer questions that will even better define therapies most likely to produce even better long-term outcomes.

Course Test Preview

To receive Continuing Education credit for this course, you must complete the online test. Please go to: <https://www.dentalcare.com/en-us/ce-courses/ce50/test>

- 1. Reliance on clinical experience, intuition, poorly designed studies alone can result in false conclusions and less than optimum care due to factors such as _____.**
 - A. bias
 - B. placebo effect
 - C. nocebo effect
 - D. All of the answers are true except E.
 - E. B and C
- 2. Consecutive controlled case studies provide a level of evidence of _____.**
 - A. predictable outcomes
 - B. no value
 - C. previous clinical success
 - D. scientific certainty
- 3. The American Academy of Periodontology Guidelines for Periodontal Therapy Position Paper states the goals of periodontal therapy include all of the following EXCEPT _____.**
 - A. restoring the periodontal attachment level to its original level
 - B. preserving the periodontium
 - C. absence of inflammatory signs
 - D. maintenance of a functional periodontal attachment level
- 4. Probing pocket depth (PPD) is a measurement of the distance between the _____.**
 - A. cemento-enamel junction (CEJ) and the base of the probable pocket
 - B. incisal edge and the base of the probable pocket
 - C. gingival margin and the base of the probable pocket
- 5. Furcation involvement _____.**
 - A. is a marker of past disease
 - B. does not reliably predict future disease progression
 - C. A and B
- 6. The Oral Risk Assessment (ORA) and Early Intervention System provide the following methodology to organize patient care:**
 - A. Collection of relevant medical and dental information.
 - B. Assessment and assimilation of the collected information.
 - C. Recommendation of professional therapies and home care procedures and products.
 - D. Evaluation of treatment and healthcare outcomes.
 - E. B, C, and D.
- 7. The term "chronic periodontitis" has been replaced by the term "periodontitis."**
 - A. True
 - B. False
- 8. Periodontitis incidence and severity is related to all the following EXCEPT _____.**
 - A. age
 - B. toothpaste choice
 - C. bleeding on probing
 - D. plaque levels

9. **Clinical attachment loss (CAL) of 1-2 mm is considered to be slight.**
A. True
B. False
10. **Alveolar bone does not remodel in response to _____.**
A. directional forces
B. host response
C. hormones
D. glucocorticoids
E. various disease states
F. mood.
11. **If bone loss forms an angular infrabony defect, _____.**
A. bone grafting may be helpful
B. not much can be done to help
12. **The following statements accurately describe bone except:**
A. Bone is a static structure.
B. Osteoclasts are cells that resorb bone.
C. A team of cells that accomplishes remodeling is called the Basic Multicellular Unit (BMU).
13. **Bone resorption cannot be reduced by which of the following:**
A. Estrogen
B. Interferon
C. Bisphosphonates
D. Topical rinses.
14. **Deep, narrow infrabony defects with more bony walls have the worst chance to gain attachment following periodontal surgery.**
A. True
B. False
15. **It is recommended to determine patients' surgical needs prior to assessing patients' response to non-surgical therapies.**
A. True
B. False
16. **Disrupting plaque biofilm is best accomplished through _____.**
A. periodontal surgery
B. locally-delivered antimicrobials
C. routine brushing and flossing
D. drinking lots of water
17. **The exact amount of time needed to optimally perform scaling and root planing has been scientifically determined.**
A. True
B. False
18. **Rinsing and irrigating with antimicrobials _____.**
A. may help control gingivitis
B. may help control periodontitis

- 19. Locally-delivered sustained-release antimicrobials all contain antibiotics.**
A. True
B. False
- 20. Systemic antibiotics feature all of the following EXCEPT _____.**
A. low cost to the patient
B. a routine benefit in chronic periodontitis
C. the ability to control pathogens within periodontal tissues
- 21. Subantimicrobial systemic dosing of doxycycline _____.**
A. as an adjunct to SRP, has been shown to reduce PPD better than SRP alone
B. has been shown to have tolerability similar to placebo
C. inhibits the activity of collagenase
D. is appropriate for use during pregnancy
E. cures periodontitis
F. A, B and C
- 22. The goal of osseous surgery is to _____.**
A. restore lost attachment
B. re-contour bone
C. allow increase access for routine plaque control
D. A and B
E. B and C
- 23. Barriers may be used in regenerative surgery to _____.**
A. prevent epithelial tissues from growing within the bony defect
B. modulate the host response
C. guide periodontal ligament cells to grow within the bony defect
D. A and C
E. B and C
- 24. Non-resorbable barriers _____.**
A. remain in place as long as needed
B. require removal at a later surgery
C. A and B
- 25. Bony defects with fewer wall generally have more predictable bone fill results.**
A. True
B. False
- 26. Autografts _____.**
A. utilize bone from a cadaver
B. can be obtained from intra-oral or extra-oral sites
- 27. Allografts _____.**
A. are freeze-dried
B. utilize the patient's own bone
C. may be demineralized
D. B and C
E. A and C

- 28. Xenografts are obtained from _____ sources.**
- A. artificial
 - B. animal
- 29. Synthetic bone does not _____.**
- A. need to be processed from calcium carbonate
 - B. facilitate the body's regenerative process
 - C. seem to be replaced by new bone over time
 - D. require immunosuppression
- 30. Biological growth factors are used with synthetic bone materials to simulate the osteogenic properties of an autograft.**
- A. True
 - B. False

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Additional Resources

- No Additional Resources Available.

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