6. PERIODONTOLOGY

Mark S. Obernesser, D.D.S., M.M.Sc.

The Fundamentals of Gums and the Art of Gum Gardening 101:
" …'tis better to have longer teeth than teeth no longer.."
An anonymous periodontist

" You don't know your players without a program."

The genera of some of the bacteria and the names of disease states have changed. Here is a new "program."

FUNDAMENTALS OF THE PERIODONTIUM

1. What fibers are normally found in a healthy periodontium?

The fibers are described classically in histologic position as the dentogingival, dentoperiosteal, alveologingival, circular, and transseptal.

2. What is the major blood supply to the periodontal ligament? Adjacent gingival tissue?

The blood supply to the periodontal ligament derives from arteries and arterioles within the supporting bone (e.g., inferior alveolar artery) to the socket and periodontal ligament. Adjacent tissue is supplied by other superficial vessels.

3. What cell type is most frequently found in the periodontal ligament?

The predominant cell type is the fibroblast.

4. What immunologic cells are typically found in the healthy periodontium?

Immunologic cells typically found in the healthy periodontium include polymorphonuclear neutrophils (PMNs), mast cells, macrophages, and lymphocytes. The prevalence of these cell types shifts depending on the disease state.

5. What is the major macromolecular component of the cementum, alveolar bone, and periodontal ligament?

Collagen.

CLASSIFICATION AND ETIOLOGY OF PERIODONTAL DISEASES

6. What are the etiologic agents in periodontal disease?

Contrary to old wives' tales, periodontal disease is not caused by occlusal trauma, vitamin deficiencies, or hypercholesterolemia. The cause is bacterial plaque—specifically, gram-negative bacteria.

7. Does the presence of gram.negative bacteria predispose the patient to periodontal disease?

The bacteria are a critical element of the periodontal disease process; however, the host response to these bacteria is also a major component.

8. What is the chief component of plaque?

Bacteria. Approximately 90—95% of the wet weight of plaque is bacteria. The other 5—10% consists of a few host cells, an organic matrix, and inorganic ions.

9. How fast does plaque form?

As a rule of thumb, plaque accumulates in about 24 hours.

10. What are the basic types of plaque? How do they differ in composition?

The basic types of plaque are supragingival and subgingival. Supragingival plaque consists mostly of aerobes and facultative bacteria (mostly gram-positive), whereas subgingival plaque consists mostly of anaerobic bacteria (frequently gram-negative).

11. What type of plaque is associated with caries?

Naturally the supragingival plaque is associated with caries—predominantly the gram-positive cocci and rods (the acid producers).

12. What coating is responsible for the adherence of plaque to the enamel?

The salivary pellicle.

13. What are the basic types of subgingival plaque?

The three basic types of subgingival plaque are hard tissue, soft tissue, and loose plaque, all of which differ in composition. Hard tissue plaque adheres to the cementum, dentin, and enamel; soft tissue plaque adheres to the epithelial cells; and loose plaque floats in-between. Loose plaque has come under a great deal of investigation because of its possible role in attachment loss. The soft tissue plaque that adheres to the epithelial lining of the pocket has also sparked interest because of the potential involvement of the organisms in tissue invasion.

14. What is the major factor in determining the different bacteria in supragingival and subgingival plaque?

The major factor is oxygen. The redox potential of the gingival sulcus greatly influences the bacterial composition.

15. Do cariogenic bacteria promote colonization by periodontal pathogens?

On the contrary, the cariogenic bacteria tend to inhibit the gram-negative rods associated with periodontal disease.

16. What is the major mechanism by which cariogenic bacteria inhibit gram-negative periodontal pathogens?

Gram-positive cariogenic bacteria produce bacteriocins and other substances that inhibit gram-negative bacterial growth.

17. What is calculus? How is it basically formed?

Calculus is mineralized plaque. It is formed by bathing of the plaque in a supersaturated solution of Ca and P0 saliva.

18. Why is calculus frequently a dark color (e.g., black, brown, gray)?

After the plaque has been solidified to calculus and an inflammatory response has occurred, localized bleeding ensues. Red blood cells adhere to and permeate the calculus, hemolysis follows, and the hemoglobinliron colors the calculus.

19. What terms are used to describe healthy gingiva?

Healthy gingiva have scalloped, knifelike margins and a firm, stippled texture. In white people they are salmon-pink in color. African-Americans, Indians, Asians, and Africans frequently have pigmented gingiva. Salmon-pink naturally does not apply, but the other terms do.

20. What terms are used to describe inflamed gingiva?

The key word is inflammation, and the cardinal signs of inflammation are calor, rubor, tumor, and dolor. All may apply to inflamed gingiva. The margins are described as rolled, the gingiva as erythematous and edematous. The stippling is absent, and the gingiva are frequently described as boggy.

21. What is gingivitis? What bacterial groups are generally associated with gingivitis?

Gingivitis is inflammation of the gingiva. The bacterial groups associated with gingivitis are spirochetes, *Actinomyces* spp. (gram-positive filament), and *Eikenella* spp. (gram-negative rod).

22. What other terms are used in the clinical description of gingivitis?

Other terms describe severity (mild, moderale, severe), location (marginal or diffuse), and presence or absence of ulceration (desquamative), suppuration, and

hemorrhage. Other terms describing the architecture also may apply, such as blunting papilla and clefting.

23. What term is used to describe HIV gingivitis? How does it appear clinically?

Linear gingival erythema (LGE) is frequently used to describe HIV gingivitis. As the name implies, the gingival margin has a distinct red band, and the tissue may bleed easily.

24. Is gingivitis a forerunner of periodontitis?

No. Gingivitis is not necessarily a forerunner of periodontitis. Chronic gingivitis may exist for long periods without advancing to periodontitis.

25. Does periodontitis occur without gingivitis?

To the purist, the answer is yes. This situation may be particularly true in the case of localized juvenile periodontitis, in which negligible gingival inflammation may be accompanied by active periodontal disease. However, most patients with routine adult periodontitis also exhibit gingivitis.

26. What causes the transition from gingivitis to periodontitis?

The exact cause of the progression is most likely multifactorial, including a pathogenic combination of bacteria and an abnormal host response.

27. What are the histologic characteristics of the initial periodontal lesion?

Basically vasculitis of the vessels is accompanied by an increase of gingival exudate from the sulcus. PMNs migrate into the sulcus and junctional epithelium. The most coronal portion of the junctional epithelium is altered, and some perivascular collagen is lost.

28. What histologic changes are associated with the early periodontal lesion?

Many of the changes are a continuation of the initial lesion. PMNs continue to migrate into the epithelium, and other lymphocytes follow. The collagen network continues to break down, and the junctional epithelial cells proliferate.

29. What are the histologic features of the established periodontal lesion?

A key component of the established lesion is the predominance of plasma cells in the connective tissue with the production of antibodies, continued loss of connectivc tissue substance, and proliferation of junctional epithelium with or without apical migration.

30. What are the key histologic features of the advanced periodontal lesion?

Many of the features are similar to the established lesion. The advanced lesion extends to the periodontal ligament and alveolar bone with pocket formation and goes through periods of exacerbations and remission. There are more extensive cellular changes due to inflammation.

31. What are the clinical signs of acute necrotizing ulcerative gingivitis (ANUG)?

ANUG is an acute, recurring infection of the gingiva characterized by necrosis of the papilla (leading to blunting), spontaneous bleeding, pain, and fetor oris. It has been theorized that the disease is stress-related (e.g., taking the National Dental Board examinations, practical examina tions, being on death row at Alcatraz).

32. What bacteria are associated with ANUG?

The bacteria associated with ANUG are a fusospirochetal complex—fusiform bacteria and spirochetes.

33. What bacteria are associated with gingivitis of pregnancy? Why?

Bacteria associated with gingivitis of pregnancy are the black-pigmenting *Bacteroides* spp., which crave steroid hormones for their own metabolism. Therefore, pregnancy essentially selects for these I Patients who use birth control pills or receive steroid therapy (chronic autoimmune diseases) are also at risk.

34. What general terms are used to describe periodontitis?

Mild, moderate, and advanced or severe are commonly used. Other terms may include generalized or localized, refractory, rapidly progressive, adult chronic, or juvenile.

35. How is periodontitis classified?

The disease is classified according to its severity:

Type I	Gingivitis
Type II	Mild penodontitis
Type III	Moderate periodontitis
Type IV	Severe or advanced periodontitis

These categories are based on clinical criteria such as the amount of bone loss, pocket depth, and mobility.

36. What is the Periodontal Screening Program?

This program was established by the American Academy of Periodontology and the American Dental Association as a screening method for the general dentist to evaluate patients' periodontal health. Selected teeth are examined for the presence or absence of disease.

37. What term is used to describe HIV periodontitis?

HIV periodontitis has been updated to necrotizing ulcerative periodontitis. It involves severe pain, bleeding, rapid loss of bone and soft tissue, exposure of bone, sequestration, and tooth loss.

38. What bacteria are generally associated with active adult periodontitis?

The bacteria most frequently cultured from active adult periodontal lesions include Prophyoromonas gingivalis, *Actinobacillus actinomycetemcomitans, Campylobacter recta (Wolinella recta), Fusobacterium nucleatum, Pro vetella intermedia, Bacteroides forsythus, Eikenella corrodens, and Treponema denticola.*

39. What are the clinical features of localized juvenile periodontitis?

The periodontal destruction is localized to the first permanent molars and/or the permanent central incisors. Clinical signs of inflammation are less acute than would be expected from the severity of destruction. Other features include familial pattern, paucity of plaque, onset during the circumpubertal period, and preponderance of *A. actinomycetemcomitans* when the sites are cultured.

40. What bacteria are associated with rapidly advancing periodontitis?

P. gin givalis	*Bacteroides capillus*
P.intermedia	*E. corrodens*

41. What bacteria are associated with refractory periodontitis?

The major infectious agents are *B. forsythus, F. nucleatum, Streptococcus intermedius, E. corrodens, and P. gingivalis*. Although the diseases listed above have clinically distinct manifestations, many of the same players show up in cultural studies again and again. When the diagnosis of refractory or rapidly progressive periodontitis is made, the patient's medical and family history should be thoroughly investigated. There may be underlying systemic medical problems. Do not hesitate to use the clinical medical laboratory and to refer the patient for a complete medical examination.

42. What is the first cellular line of defense of the body against the periopathogens?

Other than the epithelial cell barrier, the first line of defense is the PMN.

43. Which periodontal diseases may involve bacterial invasion of the connective tissue?

- Localized juvenile periodontitis (LJP)
- Gingivitis
- ANUG

44. What bacteria may be associated with tissue invasion?

For UP the answer is again A. actinomycetemcomitans. For gingivitis and ANUG the culprits are spirochetes.

45. In what type of plaque are these organisms frequently cultured?

Because these organisms are associated with tissue invasion, they are most commonly isolated from soft tissue plaque and loose plaque in a periodontal pocket.

46. What is meant by a burn-out lesion in a patient with UP?

At one point the patient with UP had an infection with periodontal lesions in which the chief etiologic agent was A. actinomycetemcomitans. The body responds with an immunologic response and controls the infection, but the bony defect remains. The deep pocketing now becomes inhabited with bacterial flora more characteristic of adult periodontal lesions.

47. What bacteria are associated with HIV-related gingivitis and periodontitis?

Studies indicate that the bacteria complexes associated with HIV-related gingivitis (LGE) and periodontitis are similar and include *A.actinomycetemcomitans, P. intermedia, P. gin givalis, C. recta, and yeasts (Candida albicans)*. A major difference may be the number of *C. recta* that are isolated. Concentrations of *C. recta* tend to be higher in HIV-related periodontitis. Enteric bacteria also may be isolated.

48. Patients with deep periodontal pockets and heavy deposits of plaque and calculus may develop an acute periodontal abscess after scaling. Why?

After scaling and root planing of deep sites the coronal tissue heals (contracts and reattaches), but there may be infective material below. The process is analogous to tightening a pursestring.

49. What is a perioendo abscess?

A perioendo abscess is a combined lesion in which periodontal and end problems occur simultaneously. Symptoms may vary, but as a general rule the lesion demonstrates radiographic involvement of the periodontium and periapex with significant probing depths, percussion sensitivity, and pulpal sensitivity. Treatment may include scaling, root planing, periodontal surgery, and root canal therapy.

50. What treatment is frequently used for a periodontal abscess?

Initial treatment may consist of the establishment of drainage and the removal of the etiologic agents (incision and drainage, scaling, root planing, irrigation), followed first by a course of antibiotic therapy and then by surgical treatment. Variations exist. Be careful of the endoperio abscess.

51. When is it safe to treat a pregnant woman's nonacute periodontal problem?

In general, the second trimester is the window of treatment for most dental procedures. If antibiotics or other medications are indicated, consult with the obstetrician and *Physicians' Desk Reference.*

52. Which periodontal disease most nearly fulfills Koch's postulates?

Koch's postulates state that a pathogenic bacterium causes a disease, that the disease is transmissible through the bacteria, and that if you eliminate or control the bacteria, you eliminate the infection. LJP, caused by *A. actinomycetemcomitans*, most nearly fulfills Koch's postulates.

53. Why do most periodontal infections not fulfill Koch's postulates?

The answer lies in the preceding question. Most periodontal infections may be described as mixed anaerobic infections.

54. What is the paradox regarding an acute dental abscess?

The paradox basically pertains to bone loss associated with the lesion. An acute infection may involve rapid, extensive bone loss, but after the infection is eradicated, the lesion has great potential to heal completely.

55. What bacterial group is associated with root caries?

Root caries may be a problem for patients with gingival recession and xerostomia (whether induced by drugs, radiation, or some other agent). The bacteria associated with root caries are gram-positive rods and filaments, particularly *Actinomyces viscosus*.

CONCEPT OF DISEASE ACTIVITY

56. What is meant by active destructive disease?

Active destructive disease indicates a loss of periodontal attachment.

57. How is disease activity measured?

Classically disease activity (attachment loss) is measured by using a periodontal probe and a fixed reference point, such as the cementoenamel junction (CEJ). The change in the probing depth, excluding any changes in the gingival height due to inflammation, determines disease activity. Statistically, disease activity is frequently defined as an attachment loss of 1.5 mm or greater. A number of different types of probes are used to measure disease activity (e.g., Florida probe). Other methods may include subtraction radiography.

58. What is the classic definition of the presence of periodontal disease?

Radiographic evidence of bone loss.

59. How is the radiographic evidence of bone loss determined?

In the healthy periodontium, the bone approaches the height of the CEJ. In the case of periodontal disease, bone resorption has occurred, and the height is below the CEJ.

60. Which radiographs tend to be most accurate in the determination of bone loss?

The bitewings because of the parallelism. Vertical bitewings are useful to assess bone in severe cases.

61. What is bone sounding?

Sounding is used to provide the clinician with additional information about the amount of bone loss. The area in question is anesthetized, and a probe is forced through the epithelium until it strikes bone. Sounding may facilitate flap design.

62. How is periodontal disease activity described?

In the past. periodontal disease was thought to be a slow, Continuous process. Many of the older texts state that the disease progresses at a rate of 0.1 mm per year, but longitudinal studies have demonstrated otherwise. Current ideas revolve around the concept of random bursts of disease activity.

63. What is the nonspecific plaque hypothesis?

The hypothesis simply states that it is the quantity and not the quality of the plaque that causes periodontal disease. The specific plaque hypothesis states the converse.

64. Which hypothesis is more clinically accurate?

The specific plaque hypothesis. A prime example is LJP. Furthermore, a number of patients may exhibit heavy deposits of bacterial plaque and calculus with severe gingivitis, yet no bone loss.

65. What is meant by a shift in flora in comparing a healthy or diseased periodontal site?

The healthy periodontal site is characterized by a preponderance of gram-positive organisms and fewer gram-negative organisms. In the diseased state the opposite holds true.

66. What bacteria are associated with active destructive periodontal disease (adult periodontitis)?

The bacteria associated with destructive periodontal disease include *P. gingivalis, E. corrodens, F. nuc/eatum, C. recta, B. forsythus, and A. actinomycetemcomjtans*. The major player may be *P. gingivalis.*

67. What traditional clinical markers (other than a great change in attachment loss) may be significant in determining active periodontal disease?

One may think that the classic signs of inflammation (tumor, calor, rubor, and suppuration) are predictors of pending attachment loss. Data demonstrate the sensitivity and specificity only of calor (temperature) for predicting attachment loss. However, it is difficult to leave inflamed gingiva untreated.

68. What two inflammatory mediators may be indicators of disease activity?

Interleukin 1.-beta and tumor necrosis factor alpha may indicate disease activity.

PERIODONTAL DIAGNOSIS

69. What is periodontal pocketing?

Periodontal pocketing is the measurement from the crest of the gingiva to the depth of the pocket. Measurements range from < 1—3 mm in the healthy state (without inflammation).

70. What sites are routinely probed during a thorough periodontal examination?

Six sites are commonly checked: the mesio-, mid-, and distobuccal sites as the corresponding lingual/palatal sites. Most periodontists sweep the probe continuously through the sulcus to get a better feel for the pocket depths as a whole.

71. What is periodontal pseudopocketing?

Pseudopocketing is a condition in which pocketing occurs without attachment loss. A classic example is phenytoin (Dilantin) hyperplasia.

72. Which is more important: attachment loss or periodontal pocketing?

Attachment loss is much more significant because supportive structures are destroyed. Pocketing may increase or decrease, depending on the severity of gingival inflammation, without attachment loss. Frequently, extensive attachment loss and gingival recession, with poor prognosis for the tooth, may be accompanied by shallow periodontal pocketing.

73. What are the two most significant clinical parameters for the prognosis of a periodontally involved tooth?

The two most significant clinical parameters are mobility and attachment loss.

74. What is gingival hypertrophy?

Gingival hypertrophy indicates that the gingivae have increased in size and not number. Hypertrophy indicates inflammation, whereas hyperplasia may not.

75. What causes gingival recession?

The major causes are tooth brush or floss abrasion, parafunctional habits, periodontal disease, and orthodontics (if the bands are improperly placed).

76. Which area of the oral cavity has the least amount of attached gingiva?

The buccal mandibular premolar area commonly has the least amount of attached tissue.

77. What is a long junctional epithelium?

After a periodontal pocket has been scaled, root planed, and curetted, a soft tissue reattachment to the root surface may occur. This reattachment is called a long junctional epithelium. Pocket reduction is due to a gain in attachment, not to a decrease of inflammation. Fibrous reattachment is also possible.

78. What is the term for gingival cells that attach to the root cementum? How do they attach to the root?

The term is junctional epithelium; the cells attach by hemidesmisomes.

79. What is a mucogingival defect?

Mucogingival defects are defined by periodontal pocketing that goes beyond the mucogingival junction.

80. What are the major risk factors for periodontitis?

Major risk factors for periodontal disease include increased age, poor education, neglect of dental care, previous history of periodontal disease, tobacco use, and diabetes.

81. Is periodontal disease a risk factor for other disease?

Some epidemiologic evidence indicates that periodontal disease and other chronic infective diseases may be associated with coronary artery disease and stroke.

82. What is the crown-to-root ratio in a healthy dentition?

As a general rule, the crown-to-root ratio in a healthy dentition is 1:2 (for each tooth).

83. What root shapes generally have a more favorable prognosis?

As the preceding question suggests, the crown-to-root ratio is very important. Long, tapering roots are usually sturdier than short, conical roots.

84. What is the clinical significance of crown-to-root ratios?

Teeth with poor crown-to-root ratios tend to have a worsened prognosis, especially if mobility is significant.

85 What is a fenestration?

If you studied the classical languages, you will quickly surmise that fenestration refers to a window in the bone. Bony fenestrations are frequently treated surgically with grafts, with or without guided tissue regeneration.

86. What is a bony dehiscence?

A dehiscence is a V-shaped defect in the supporting bone—buccal or lingual plates. These defects are difficult to treat.

87. What is positive bony architecture?

In the healthy state the bone contours follow the gingival contours, a pattern that is usually described as scalloping. Negative bony architecture is another story.

88. What is negative bony architecture?

As described above, the bony architecture usually follows the gingival tissue. Negative bony architecture denotes intrabony defect(s). Many periodontists believe hat when osseous surgery is performed, it is necessary to recreate positive bony architecture, even at the expense of healthy supporting bone. Growing evidence suggests, however, that the recreation of positive bony architecture does not improve the periodontal prognosis.

89. What are the basic classifications of bony defects?

Bony defects are generally classified according to the number of bony walls that remain. For example, a one-wall defect has only one remaining wall of bone, two-wall defects have two remaining walls, and so on.

90. Which bony defect is most likely to repair or fill naturally after treatment?

Three-wall periodontal defects are most likely to repair naturally after therapy.

91. Why are three-wall defects most likely to repair after treatment?

Three-wall defects tend to be narrow, and three walls may contribute regenerative cells. Two- and one-wall defects lack that luxury.

92. Name the microbiologic methods of assessing bacterial plaque.

There are numerous ways to assess bacterial plaque. General categories include cultural, microscopic, enzymatic, and genetic methods.

93. How are furcations classified?

Furcations are classified according to probing. Class I furcations are found at the onset of probing: class II, approximately halfway into the furcation; and class III, throughout the furcation.

94. How is tooth mobility assessed?

Tooth mobility is important in the development of a prognosis and vital to treatment planning. Mobility is determined by gently tapping the tooth in a buccal/lingual direction with two instruments. Mobility is gauged by the motion back and forth in millimeters (range: 0 to 3+, also known as "flapping in the breeze").

95. What periodontal pathology do diabetes, Papillon-LeFevre, and Chediak-Higashi disease have in common?

With all of these diseases the normal cellular immunologic response is impaired. The white cells (PMN5) do not function properly. Therefore, patients are susceptible to periodontal infections. Watch for abscesses.

96. What is gingival crevicular fluid (GCF)?

GCF is an ultrafiltrate of serum. Therefore, it contains many of the components of serum, particularly complement and antibody. The flow rates of GCF have been used in attempt to predict disease activity. Furthermore, investigators have been interested in GCF for other markers of periodontal breakdown (e.g., beta-glucuronidase, interleukin, collagenase).

97. What enzymatic methods may be used to assess bacterial plaque? Disease activity?

Some of the enzymatic methods used to assess bacterial plaque associated with active disease include BANA (benzoyl-arginine-naphthylamide) hydrolysis, collagenase, and beta glucuronidase.

98. What genetically based techniques are used to assess bacterial plaque?

Most of these techniques are based on DNA/RNA homologies. DNA/RNA probes specific for a suspected periodontal pathogen are used to analyze plaque. Commercial probes are on the market. A chairside probe already in use in Europe awaits FDA approval for use in the United States.

99. Name the major immunologic techniqt for assessing bacterial plaque.

The major techniques are fluorescent antibody staining, enzyme-linked immunosorbent assay (ELISA), and Latex agglutination, all of which may have high-technology instrumentation applied to them. They are used most commonly as research tools.

100. What antibiotics are used frequently to treat a periodontal abscess?

After the establishment of drainage, whether it be via the sulcus or incision and drainage (I&D), penicillin or amoxicillin (500 mg every 6 hr) provides adequate antibiotic coverage.

101. What antibiotics may be well advised for the treatment of adult periodontitis?

For adult periodontitis, with high concentrations of *P. gingivalis*, doxycycline (50—100 mg 2 times/day) provides adequate coverage. *P. gingivalis* tends to be more sensitive to doxycycline than to tetracycline.

102. What is the appropriate response to refractory periodontitis?

This is the time to call out the cavalry. Broad-spectrum antibiotic coverage may be indicated, such as clindamycin (300 mg 3 times/day) or amoxicillin/clavulanic acid (500 mg every 6 hr) and metronidazole (250 mg 3 times/day). Other combinations exist.

103. How is LJP treated?

LJP has a preponderance of *A. actinomycetemcomitans* and is sufficiently treated with tetracycline (250 mg every 6 hr).

104. In a patient who is allergic to penicillin and erythromycin, what is the next antibiotic to be used for prophylaxis for a heart murmur?

Clindamycin, 600 mg 1 hour before treatment.

Note: The American Heart Association has recently revised the dosage of antibiotics re quired for prophylaxis. Refer to chapter 3 (Oral Medicine).

105. Why are third-generation cephalosporins frequently contraindicated for the treat ment of a periodontal abscess?

Frequently the spectrum of a third-generation cephalosporin becomes so specific that it does not provide adequate antimicrobial coverage. Penicillins should be the first choice; erythromycin or clindamycin may be is used in penicillin-allergic patients.

106. What complication may occur with broad-spectrum antibiotics?

A major problem is the development of pseudomembranous colitis, which is caused by the overgrowth and toxin production of *Clostridium difficle*

107. Why are tetracyclines used commonly in the treatment of periodontal disease?

Tetracycline is used primarily for antibiotic coverage, but it has advantages over other antibiotics because it concentrates at levels 2—4 times higher in the GCF than in the serum, binds to the root surface and can be released over a prolonged time, prevents bacterial reattachment to the root surface, promotes reattachment of fibers to the root surface, and inhibits collagenolytic activity.

108. What are some of the common guidelines or precautions that should be given to a patient in prescribing tetracyclines?

Use of any antibiotic involves the potential to upset the natural bacterial flora. Gastrointestinal distress, including nausea, vomiting, and diarrhea, is possible. Women must be advised of the potential of yeast infections. Other side effects include tinnitus, vertigo, and photosensitivity.

109. Are tetracyclines safe and effective for women who are taking birth control pills?

In general, a woman who is taking birth control pills should avoid the use of tetracyclines. Clinical studies have shown that tetracyclines may cause abnormal breakthrough bleeding during the menstrual cycle.

110. If a patient is not sure whether she is pregnant, should tetracyclines be used to treat an acute periodontal infection?

Tetracyclines exert their bacteriostatic effect by inhibiting protein synthesis at the ribosome. They also cross the placenta and inhibit fetal protein synthesis. Avoid tetracyclines in pregnant patients.

111. What directions should be given to the patient in prescribing oral tetracyclines?

Tetracyclines should be taken between meals (on an empty stomach) with a tall glass of water. Foods and antacids containing relatively high concentrations of calcium and iron should not be taken with tetracycline. Tetracycline acts as a chelator with these divalent cations, thereby interfering with its own intestinal absorption. Therapeutic dosages, therefore, are not achieved.

112. What are the major advantages and disadvantages of using doxycycline or minocycline in the treatment of periodontal disease?

The spectrum of doxycycline and minocycline may be slightly better, particularly in covering *P. gingivalis*. Other advantages include less photosensitivity, less chelating, and better patient compliance. Because both antibiotics are more fat-soluble, the dose is reduced to 50 or 100 mg 2 times/day. A big disadvantage is cost. Doxycycline and minocycline are much more expensive.

113. What is the major problem with the use of metronidazole?

When prescribing metronidazole, you should advise patients that they must refrain from alcohol or they may become violently ill from the combination

(Antabuse effect). Patients should always be advised not to mix any medicine with alcohol.

114. Why is metronidazole effective in treating a periodontal infection?
Metronidazole is most effective in areas of low redox potential, making it ideal for the treatment of anaerobic infections. It is also effective in treating Montezuma's revenge that is caused by a parasite.

115. What is localized drug delivery? How does it apply to periodontal therapy?

Localized drug delivery is being developed to deliver the drug directly to the site of intended use—the periodontal sulcus. The great advantage of such systems is that because they are local, systemic side effects are almost nil. The best studied system involves a tetracycline fiber, but other systems exist. This method is the wave of the future with antibiotics, antiinflammatory drugs, and growth factors.

116. How do localized delivery systems work?

One of the most popular localized drug delivery systems is for tetracycline. Basically the tetracycline is impregnated into an ethyl vinyl acetate strip. The fiber/strip is placed into the sulcus and secured into position. The fiber slowly releases the antibiotic into the sulcus, eradicating the bacteria. The fiber should be in place for 7—10 days, depending on the system used.

117. What preparation is required before placement of the fiber?

The teeth should be thoroughly scaled, root planed, and polished before fiber placement.

118. What pathway do nonsteroidal antiinflammatory drugs (NSAIDs) block?

NSAIDs block the cyclooxygenase metabolism of arachidonic acids.

119. Which mouth rinse appears to be most effective in the control of bacterial plaque?

Chlorhexidine gluconate is the most effective oral rinse for controlling bacterial plaque, particularly because it leaves the greatest residual concentration in the mouth after use.

120. What is sanguinaria? How is it used?

Sanguinaria, an extract from the blood root plant that exhibits antimicrobial properties, has been formulated into various dentifrices and mouthwashes. A major problem with sanguinaria is that it is easily washed from the oral cavity so that the antimicrobial effects are short-lived.

121. What is triclosan? How does it work?

Triclosan is a compound that has broad-spectrum antimicrobial properties. Therefore, it is effective against many of the gram-positive and gram-negative organisms involved with oral disease. Triclosan has recently been approved for use in dentifrices.

122. HIV-positive patients frequently manifest a condition called hairy leukoplakia in their oral cavity. What microbe is commonly associated with hairy leukoplakia? What is the treatment for this condition?

Candida albicans (yeast) is frequently associated with hairy leukoplakia and should be treated with antifungal medication, including nystatin or fluconazole. Chlorhexidine rinses should be included, because chlorhexidine is also effective against *C. albicans.*

123. What is the primary symptom of root sensitivity?

In general, the primary symptom is sensitivity to cold.

124. What is the cause of root sensitivity?

Root sensitivity is believed to be caused by the movement of fluid in the dentinal tubules, which stimulates the pain sensation (the hydrodynamic theory).

125. What factors may contribute significantly to dentinal sensitivity?

Tooth brush abrasion, periodontal and orthodontic treatment, gingival recession, acidic foods, and bruxism.

126. How is root sensitivity treated?

Treatment of root sensitivity usually involves seal-coating of the root. Substances routinely used are fluoride mouth rinses, fluoride toothpastes, desensitizing toothpaste, application of composite monomer, and iontophoresis.

127. How do root desensitizers work?

A number of methods are used, including protein precipitants (e.g., strontium chloride), dentinal tubule blockers (e.g., fluorides, oxalates), nerve desensitizers (potassium nitrate), and physical agents such as burnishing the root, composites, monomers, and resins.

128. What is iontophoresis? How is it used in periodontics?

Iontophoresis is analogous to electroplating. In periodontics it is used to treat dentinal sensitivity by electroplating fluoride to the root surface.

129. What new method is being tested to treat root sensitivity?

Investigators are testing the efficacy of lasers to seal the dentinal tubules.

OCCLUSAL TREATMENT

130. What is the role of occlusion in periodontal disease?

As a primary player, occlusion has little significance in the etiology of periodontal disease, but it may act as a contributing factor.

131. What are primary and secondary occlusal trauma?

Primary occlusal trauma refers to excessive force applied to a tooth or teeth with normal supporting structures. Secondary occlusal trauma refers to excessive force applied to a tooth or teeth with inadequate support (periodontal disease).

132. What is fremitus?

Fremitus is occlusal trauma associated with centric occlusion and may indicate a slight occlusal discrepancy. On examination the patient is asked to open slightly and tap gently. The examiner checks for minor tooth movement on tapping. This technique is used mostly for the maxilla.

133. When is a nightguard indicated?

A nightguard is indicated whenever the signs or symptoms of bruxism occur.

134. What are the clinical signs of bruxism?

Signs of bruxism may include faceting, temporomandibular joint (TMJ) symptoms, masticatory muscle soreness, fractured teeth or restorations, and widened periodontal ligament spaces (on radiographs). These signs may occur in various combinations.

135. What criteria should be followed in constructing a nightguard for the treatment of bruxism?

A nightguard should have four characteristics: (1) it should be made of hard acrylic; (2) it should snap gently over the occlusal surfaces of the maxillary teeth; (3) it should occlude evenly with the mandibular teeth; and (4) it should have even contacts in excursion and be comfortable so that the patient will wear it.

136. When should the splinting of teeth be considered?

Splinting of teeth is performed basically for patient comfort. Little evidence suggests that splinting improves the prognosis of periodontal mobile teeth. In fact, it may worsen the prognosis by limiting oral hygiene access.

137. What types of splints may be fabricated?

A wide range of splints may be provided from the simple to the elaborate. Examples in clude interproximal application of composite, composite with mesh reinforcement, Maryland bridge, and other fixed prostheses.

138. What do widened periodontal ligament spaces indicate?

Widened periodontal ligament spaces are indicative of occlusal traumatism (no underlying medical problems).

139. What situation may be considered to be controlled occlusal trauma?

Orthodontic tooth movement may be considered to be controlled occlusal trauma.

NONSURGICAL TREATMENT OF PERIODONTAL DISEASE

140. What is scaling? Root planing? Curettage?

Scaling is the removal of hard and soft deposits (plaque and calculus) from tooth surfaces. Root planing is the smoothing of the root surfaces with a scaler or cures. The objective of root planing is to remove additional deposits as well as affected cementum in an attempt to achieve soft-tissue attachment. Curettage is the removal of the lining of the periodontal pocket. This procedure is frequently performed with root planing to promote soft tissue attachment.

141. What is the treatment routinely used for ANUG?

Ti consists of debndement (scaling and root planing) with an antibiotic. Penicillin V/K, 260—500 mg 4 times/day for 7 days, should be sufficient. Pain relievers are prescribed if needed. Instructions for oral hygiene should be stressed.

142. What is the treatment for acute suppurating gingivitis?

The treatment is the same as that for ANUG. If the patient does not respond, you may consider changing the antibiotic. If the second antibiotic does not work, you may want to examine systemic factors; for example, diabetics are prone to this type of periodontal problem.

143. What is nonsurgical therapy for periodontal disease?

Nonsurgical treatment is centered on maintenance. Scaling and root planing are performed at greater frequency than in a normal recall schedule.

144. What is an appropriate interval for maintenance appointments for a patient treated nonsurgically?

Initially it is best to see the patient at 3—4-month intervals so that oral hygiene and disease progression may be assessed.

145. What is the Keyes technique?

The Keyes technique is a method of assessing bacterial plaque via microscopic means (wetmount slides) and correlating periodontal infection, particularly the numbers of spirochetes and motile rods. This technique was in vogue during the past 10—20 years, but in the author's opinion additional validation studies are required.

146. What are the advantages of periodontal surgery over nonsurgical treatment?

The most important reason for performing periodontal surgery is access. It gives you the opportunity to visualize the roots so that calculus may be removed more completely.

147. What are additional objectives of periodontal surgery?

Other objectives include pocket reduction and promotion of gingival reattachment.

148. Name the major complications that may be associated with periodontal surgery.

With any form of surgery, you run the risk of pain, fever, swelling, infection, and bleeding. In addition, other problems that may occur include gingival recession, root caries, and root sensitivity.

149. When is gingivectomy indicated?

Gingivectomies are indicated when there are copious amounts of attached tissue and no intrabony defects. The most common application is treatment of drug-induced hyperplasia.

150. What drugs may cause gingival hyperplasia?

Common causative drugs include phenytoin, nifedipine, and cyclosporine A. These med ications stimulate proliferation of gingival fibroblasts, causing an overgrowth of the gingiva. Other drugs that may cause gingival hyperplasia include calcium channel blockers (verapamil, felodipine, nisoldipine, diltiazem, amlodipine), antiepileptics (lamotrigine and mephenytoin), the immunosuppressive mycophenolate, the antidepressant sertraline, the antipsychotic pimozide, and interferon alpha beta.

151. How may pocket depth be indicated before performing a gingivectomy?

After the tissue has been anesthetized, a probe may be inserted to the depth of the pocket, and a second probe may be used to perforate the gingiva at that depth, creating a bleeding point (Black procedure). A series of bleeding points provides a guide for the amount to tissue to be excised. Connect the dots!

152. What instruments are commonly used to perform a gingivectomy?

Instruments may include the Buck and Kirkland knives, side-cutting rongeurs, electrosurgery apparatus, and laser.

153. What is a modified Widman flap?

A Widman flap is also known as open or flap curettage. Sulcular or submarginal incisions are made initially, and full-thickness flaps are elevated for debridement, scaling, and root planing. Flaps are then closed with sutures.

154. What is a full-thickness periodontal flap? A partial-thickness periodontal flap?

After the incision is made, a full-thickness flap involves elevation of the entire soft tissue, whereas a partial-thickness flap involves the splitting (dissection) of the gingival flap, leaving the periosteum adherent to the bone.

155. Why are inverse bevel incisions frequently used in flap surgery?

Inverse bevel incisions facilitate degranulation by thinning the flap. Furthermore, the thinning of the flap may promote reattachment of the gingiva to the root by placing connective tissue elements against the root when the flap is closed.

156. What is an apically positioned flap? When is it most frequently performed?

The definition is in the name. After the flap has been elevated and the necessary treatment has been performed, the gingiva is positioned at the crest of bone. This procedure is most frequently performed after osseous surgery (e.g., positive architecture, crown lengthening) and usually requires vertical releasing components.

157. What is osteoplasty? What is ostectomy?

Osteoplasty is the reshaping or recontouring of nonsupportive bone. An example is the recontouring and ramping of interproximal bone. Ostectomy is the removal of supporting bone. This procedure is usually performed to create positive architecture or to increase the clinical crown length.

158. What is cementoplasty? Where is it commonly applied?

Cementoplasty is the reshaping and smoothing of the root cementum. Teeth with developmental grooves in the roots, such as the premolars and maxillary lateral incisors, may develop localized periodontal defects as bacterial plaque and calculus run apically down the groove.

159. When is a crown-lengthening procedure indicated?

The procedure is indicated whenever clinical crown length is inadequate for the restoration. A general rule of thumb for a crown preparation is that you should have 4 mm between the margin of the preparation and the crest of bone to ensure adequate crown length. This measurement maintains a proper biologic width.

160. How are furcations routinely treated?

Formerly, as soon as a furcation became evident on the radiograph, the treatment was tincture of cold steel, better known as extraction. The treatment of furcations varies, depending on the type and the tooth. Treatment may range from simple management with scaling, root planing, and curettage to tissue-guided regeneration with bone-grafting material.

161. What is a distal wedge procedure? Where is it commonly found clinically?

As the name implies, in the distal wedge procedure a block of tissue is removed from the distal aspect of a tooth to reduce the pocket depth. Distal wedge procedures are frequently the sequel to the extraction of a third molar. After the third molar is extracted, the bone fill is poor, leaving a periodontal defect.

162. What is a palatal/lingual curtain procedure? Where is it frequently used? Why?

The palatal/lingual curtain procedure is a surgical procedure commonly carried out in treating the maxillary anterior teeth. Deep, interproximal buccal incisions are made to free the palatal tissue; the buccal flap is not elevated. After the palatal/lingual flap is elevated, debridement, scaling, and root planing are carried out from the palatal. The rationale behind this procedure is to maintain the buccal gingival architecture to minimize esthetic changes.

163. What is crestal anticipation?

This term is commonly used to describe flap design when surgery is performed, particularly when it is extremely difficult to position the gingival flap apically at the crest of bone. (In palatal and lingual gingiva, vertical releasing incisions are difficult or contraindicated.) Basically an inverse bevel gingivectomy to the crest is carried out.

164. When is a root amputation indicated?

Obviously the procedure applies only to multirooted teeth. In general, a root amputation may be performed when periodontal involvement of a single root is severe. Endodontic and prosthetic considerations also must be taken into account.

165. Which teeth are most frequently involved in root amputation procedures?

The requirement of multirooted teeth limits the number of candidates. A vast majority of root amputations involve the maxillary first and second molars.

166. Why are the maxillary first and second molars frequent candidates for root amputation?

Because of the convergence of the distobuccal root of the first molar and the mesiobuccal root of the second molar as the roots move apically, the first and second molars are commonly involved periodontal sites.

167. What are the major advantages of using a laser for periodontal procedures?

There are two major advantages of using a laser for periodontal surgery: (1) the incision is sterile, and (2) the laser cauterizes blood vessels during the procedure. It also has been reported that the postoperative period is less painful because of the desensitization of nerve endings.

168. Why may it be advantageous to use combination therapy (antibiotic and NSAID) in the treatment of periodontal disease?

Combination therapy attempts to kill two birds with one stone. It not only eliminates the etiologic agent but also attempts to control the resultant inflammation (hopefully to prevent bone loss).

169. What surgical procedure is performed as adjunctive therapy for orthodontic tooth rotation? How successful is it?

Routinely a fiberotomy is performed to prevent relapse of the tooth rotation. In general, a fiberotomy is not enough. The rotated tooth still requires some type of stabilization.

170. What medications may affect salivary flow? How may they affect periodontal health?

Many medicines may influence salivary flow. Prime suspects are tricyclic antidepressants and antihypertensives. Decreased salivary flow diminishes the natural cleansing of the oral cavity, thus increasing the incidence of periodontal disease and caries. Watch for both supra- and subgingival root caries.

GINGIVAL AUGMENTATION AND MUCOGINGIVAL SURGERY

171. When should a soft-tissue graft be considered as an appropriate treatment of gingival recession?

A soft tissue graft should be considered as soon as the mucogingival junction has been breached (i.e., probing extends beyond the mucogingival junction). Other factors also need consideration, such as location, frenum attachment, root sensitivity, root caries, and required restoration.

172. What is a free gingival graft? What other type of graft procedures may be used?

In a free gingival graft a section of attached gingiva is harvested from an area of the mouth. Routinely the hard palate is used, but any area with sufficient attached gingiva is appropriate. The graft is then sutured to the recipient site.

Other grafting procedures include the pedicle or lateral sliding flap, in which the graft is lifted from an area adjacent to the recipient site but not completely freed. This procedure maintains vascular supply to the graft.

173. How is bleeding controlled after the palate has been used as the donor site for a free gingival graft?

There are a number of ways to control bleeding at the donor site, including (1) pressure with a moistened gauze, (2) pressure with a tea bag, (3) vasoconstriction (epinephrine in the local anesthetic), (4) suturing (tie off the bleeders), (5) collagen with or without stent, (6) topical thrombin, and (7) chemical electrical cautery. If bleeding continues, it may not be a bad idea to assess prothrombin time (PT), partial thromboplastin time (PYF), and platelet count.

174. What is the primary reason for failure of a free gingival graft?

The chief reason that a free gingival graft fails is disruption of the vascular supply before engraftment. The second most common reason is infection.

175. What is meant by necrotic slough of a free gingival graft?

After a free gingival graft has been placed, the healing involves revascularization of the graft. The superficial layers of the graft are the last to be revascularized; therefore, the layer dies off, producing a necrotic slough. Pedicle grafts take their vascular supply with them; hence, no necrotic slough.

179. What type of flap is used at the recipient site of a free gingival graft? Why?

Partial-thickness flaps are used so that the periosteum remains attached to the bone. The reason is that the periosteum is the blood supply for the graft.

176. Why is the bone/periosteum scored during a grafting procedure?

The bone is frequently scored during a free gingival graft to prevent the ma of the graft. In other words, it helps to prevent the mucosa from covering over the graft. Additional methods to prevent this problem include suturing the base (apical) portion of the graft to the mucosa and tacking the mucosa to the periosteum.

177. Why is it difficult to place a free gingival graft in the buccal area of the mandibular premolars?

This procedure can be especially problematic when extensive recession has caused a mucogingival defect. The problem lies in the fact that you may encroach on the mental nerve/vascular bundle with the graft and cause problems with these structures.

178. When is a frenectomy indicated?

In general, frenectomy is indicated whenever a frenum is causing a problem. For example, a high attachment of a frenum may cause the crestal gingiva to pull away during phonation (ankyloglossia) and mastication, thus opening the pocket for food impaction. This situation frequently arises in the premolar areas.

179. What procedure may be performed in conjunction with a frenectomy to prevent recurrence?

A frenum also may cause a problem in the area between the maxillary central incisors, thus contributing to a diastema. The fibers of the frenum cross the height of the maxilla to the incisive papilla. The papilla may blanch when the frenum is pulled. A free gingival graft is pdrformed in conjunction with the frenectomy to prevent recurrence of fiber attachment to the papilla.

180. What other material has recently been developed for soft tissue grafting?

Freeze-dried allograft acellular human dermis.

181. What are the advantages of using allograft dermis?

The great advantage is that no donor graft is required. Thus the discomfort, bleeding, and infection associated with harvesting of tissue are avoided. In addition, the supply is potentially unlimited.

182. What are the disadvantages of using allograft dermis?

The two major disadvantages are acceptance and compliance. Some patients refuse to have any substance placed in their body from a cadaver source. In addition, after the allograft dermis has been placed, patients are advised not to brush their teeth with toothpaste but to rinse only for 7—10 days. The paste may inhibit engraftment.

183. What is a push-back procedure? Why was it used?

A push-back procedure was performed ona larger area of attached gingiva. As the name implies, an incision was made at the mucogingival junction, and the mucosa was pushed back, leaving exposed bone. Ouch! The area eventually granulated inward, followed by attached gingiva. Needless to say, the patient never spoke to you again because of the severe postoperative pain. As the question implies, this procedure is no longer in use.

REGENERATIVE PROCEDURES

184. What are the basic types of bone-grafting materials used in the treatment of periodon tal defects?

Grafts may be broken down into three fundamental categories: (1) autografts (intraoral and extraoral), (2) allografts, and (3) alloplasts. The

autografts may be harvested from the patients hip and rib (extraoral) or from a healing extraction socket, the chin, maxillary tuberosity, or retromolar areas (intraoral). Allografts consist of freeze-dried bone and freeze-dried decalcified bone from another source (usually cadaver bone). Alloplasts are synthetic materials; the most commonly used are tricalcium phosphate, calcium carbonate, and hydroxyapatite.

185. What is bone/blood coagulum? Where is it used?

Bone/blood coagtilum is another type of grafting material, normally obtained with a chisel or file during osseous surgery. The bone/blood shavings are collected and then packed into the defect in an attempt to promote new bone formation. Because the bone is predominantly cortical, the results are not predictable.

186. What is bone swagging?

Swagging is the bending and breaking of the bony walls into the periodontal defect. It, too, has poor predictability and is not used with great frequency.

187. When should an intraoral autograft from an extraction site be harvested?

As a general guideline, the intraoral autograft should be harvested 6—8 weeks after extraction. This gives the extraction site enough time to become organized with osteogenic components.

188. Which bone-grafting material has the greatest osteogenic potential with the fewest Sequelae in periodontal applications?

Osteogenic potential and sequelae are optimal with freeze-dried allografts (cadaver bone).

189. What sequelae may occur with autogenous bone grafts?

Possible sequelae include graft rejection, root resorption, and ankylosis.

190. What are connective tissue grafts? Where are they applied?

Connective tissue grafts are commonly used to augment a site that is now concave.

191. What sites are commonly used to harvest connective tissue for grafting?

Common sites include the hard palate, maxillary tuberosity, and retromolar area.

192. What growth factors may potentially be used to stimulate osseous regeneration?

The purpose of this question is to inform you of one of the new hot spots in periodontal research. Some day, after the etiologic agents have been removed and

the inflammation is under control, growth factors may be applied to regenerate the periodontium. Three growth factors that appear to have a great deal of potential are bone morphogenic protein, platelet-derived growth factor, and insulinlike growth factor. Surely others will emerge.

193. What is guided tissue regeneration (GTR)? Where is it most successful?

GTR involves the placement of a membrane (usually Gore-Tex) over a bony defect during periodontal surgery. A second surgical procedure is needed 6—8 weeks after initial surgery to retrieve the membrane. Defects amenable to this type of treatment are shallow furcations and narrow intrabony defects. GTR also may be applied to ridge augmentation procedures. A resorbable membrane is now commercially available. Therefore, no second surgery is needed to remove the membrane.

194. What is the purpose of the membrane?

The membrane prevents apical migration of the epithelium, which causes repocketing and prevents bone regeneration.

195. What surgical techniques may be used for ridge augmentation?

Common techniques use GTR membrane fixation or titanium mesh. In both cases, autogenous and/or allograft bone is placed and secured with these materials.

196. What are the indications for ridge augmentation?

Basically it is used whenever more bony mass is indicated. Examples inc future placement of an implant and filling a concavity after tooth extraction for esthetic reasons. More extensive augmentation is indicated when the bone becomes too atrophic for a prosthesis.

197. What are the two basic types of implant placement?

The two basic types of implant placement are submerged and nonsubmerged. Submerged implants require a second surgical procedure to uncover the fixture.

198. What is osseointegration?

Osseointegration is the same as ankylosis.

199. What bacteria are associated with periimplantitis?

Many of the same species associated with periimplantitis are also associated with adult periodontitis, including *A. actinomycetemcomitans*, *P. gingivalis*, and *P.intermedia*. Other species frequently detected by cultural methods are *Capnocytophaga* species, *C. recta*, and *E. corrodens*.

200. How are implants maintained?

Implants require maintenance, much like crowns and bridges and natural teeth. The same principle holds true: cleanliness is next to godliness. Implant systems may have different instruments associated with their maintenance. The instruments are usually plastic-tipped so that the surface of the implant is not scratched. Floss, superflpss, and braided floss are also handy.

BIBLIOGRAPHY

Classification of Periodontal Diseases and Etiologies

1. Haffajee AD, Socransky SS, Dzink JL, et al: Clinical, microbiological and immunological features of subjects with refractory periodontal diseases. J Clin Periodontol 15:390, 1988.
2. Kornman KS, Loesche WJ: Effects of estradiol and progesterone on Bacteroides melaningogenicus and Bacteroides gingivalis. Infect Immun 35:256—263, 1982.
3. Listgarten MA: The role of dental plaque in gingivitis and periodontitis. J Clin Periodoritol 15:485—487,1988.
4. Mandell ID, Gaffar A: Calculus revisited. J Clin Periodontal 13:249—257, 1986.
5. Moore WEC, Moore LH, Ranney RR, et al: The microflora of periodontal sites showing active progression. J Clin Periodontol 18:729—739, 1991.
6. Newman MN, Socransky SS: Predominant microbiota of penodontosis. J Periodontol Res 12:120—128, 1977.
7. Sooriyamoorthy M, Gower DB: Hormonal influences on gingival tissue: Relationship to periodontal disease. J Clin Periodontol 16:201—208, 1989.
8. Tanner ACR, Haffer C, Brathall GT, et a!: A study of the bacteria associated with advancing periodontitis in man. J Clin Periodontol 6:278, 1979.
9. Zambon JJ, Reynolds HS, Genco RJ: Studies of the subgingival microflora in patients with acquired immunodeficiency syndrome. J Clin Periodontol 61:699—704, 1990.

Concept of Disease Activity

10. Jandinski JJ, Stashenko P. Feder LS, et a!: Localization of interleukin I-beta in human periodontal tissue. J Penodontol 62:36—43, 1991.
11. Lindhe J, Haffajee AD, Socransky SS: The progression of periodontal disease in the absence of periodontal therapy. J Clin Periodontol 10:433—442, 1983.
12. Rossomando EF, Kennedy JE, Handjmichael J: Tumor necrosis factor alpha in gingival crevicular fluid as a possible indicator of periodontal disease in humans. Arch Oral Biol 35:431-434, 1990.
13. Socransky SS, Haffajee AD, Goodson JM, Lindhe J: New concepts of destructive periodontal disease. J Clin Periodontol 11:21—32, 1984.

Periodontal Diagnosis

14. Cochran DL: Bacteriological monitoring of periodontal disease: Cultural, enzymatic, immunological, and nucleic acid studies. Cun Opin Dent 1:37-44, 1991.
15. Goultschin J, Cohen HDS, Donchin M, et al: Association of smoking with periodontal treatment needs. J Periodontol 6 1:364-367, 1990.
16. Grbic iT, Lamster IB, Celenti RS, Fine JB: Risk indicators for future clinical attachment loss in adult penodontitis: Patient variables. J Periodontol 62:322—329, 1991.
17. Savitt ED, Keville MW, Peros WJ: DNA probes in the diagnosis of periodontal microorganisms. Arch Oral Biol 35(Suppl):153S—159S, 1990.
18. Schlossman M, Knowler WC, Pettitt DT, Genco Ri: Type 2 diabetes mellitus and periodontal disease. J Am Dent Assoc 121:532—536, 1990.

Adjunctive Periodontal Therapy

19. Bonesville P: Oral pharmacology of chlorhexidine. J Clin Periodontol 4:49—65, 1977.
20. Ciancio SA: Antibiotics in periodontal care. In Newman MG, Kornman KS (eds): Antibiotic/Antimicrobial Use in Dental Practice. Carol Stream, IL, Quintessence, 1990, pp 136—147.
21. Goodson JM: Drug delivery. In Perspectives on Oral Antimicrobial Therapeutics. Chicago, American Academy of Periodontology, 1987, pp 6 1—78.
22. Southard GL, Boulware RT, Walborn DR, et al: Sanguinarine: A new antiplaque agent. Compend Cont Educ Dent 5(Suppl):72—75, 1984.
23. Williams RC: Non-steroidal anti-inflammatory drugs in periodontal disease. In Lewis AJ, Furst DE (eds): Non-steroidal Anti-inflammatory Drugs. New York, Marcel Dekker, 1987, pp 143—155.

Nonsurgical Treatment of Periodontal Disease

24. Drisko CL, Killoy WJ: Scaling and root planing: Removal of calculus and subgingival organisms. Curr Opin Dent 1:74—80, 1991.
25. Hirshfeld L, Wasserman B: A long term survey of tooth loss in 600 treated periodontal patients. J Periodontol 49:225—237, 1978.
26. Pihistrom B, McHugh RB, Oliphant TH, Ortiz-Campos C: Comparison of surgical and non-surgical treatment of periodontal disease. J Clin Periodontol 10:524—541, 1983.

Surgical Treatment of Periodontal Disease

27. Becker BE, Becker W, Caffesse R, et al: Three modalities of periodontal therapy: 5-year final results. J Dent Res 69:2 19, 1990.
28. Kalkwarf KL: Surgical treatment of periodoptal diseases: Access flaps, bone resection techniques, root preparation, and flap closure. Cun Opin Dent 1:87—92, 1991.
29. Ramfjord SP, Morrison EC, Kerry GJ, et al: Four modalities of periodontal treatment compared over five years. J Clin Periodontol 14:445—452, 1987.

30. Ramfjord SP, Nissle RR, Shick RR, Cooper H: Subgingival curettage versus surgical elimination of periodontal pockets. J Periodontol 39:167—175, 1968.
31. Robertson PB: The residual calculus paradox. J Periodontol 61:65—66, 1990.
32. Tarnow DP, Fletcher P: Root resection vs. maintenance of furcated molars. NY State Dent J 55:34, 36,39, 1989.

Gingival Augmentation and Mucogingival Surgery

33. Allen EP: Use of mucogingival surgery to enhance esthetics. Dent Clin North Am 32:307—330, 1988.
34. Lang NP, Loe H: The relationship between the width of keratinized gingiva and gingival health. J Penodontol 43:623—627, 1972.
35. Miller PD: Regenerative and reconstructive periodontal plastic surgery: Mucogingival surgery. Dent Clin North Am 32:287—306, 1988.
36. Prato GPP, De Sanctis M: Soft tissue plastic surgery. Curr Opin Dent 1:98—103, 1991.

Regenerative Procedures

37. Becker BE, Becker W: Regenerative procedures: Grafting materials, guided tissue regeneration, and growth factors. Curr Opin Dent 1:93—97, 1991.
38. Branemark P1, Zarb GA, Albrektsson T: Tissue-integrated prostheses. In Osseointegration in Clinical Dentistry. Carol Stream, IL, Quintessence, 1985.
39. Lynch SE, Williams RC, Poison AM, et al: A combination of platelet-derived growth factors enhances periodontal regeneration. J Clin Periodontol 16:545—548, 1989.
40. Magnusson I, Batch C, Collins BR: New attachment formation following controlled tissue regeneration using biodegradable membranes. J Periodontol 59:1-6, 1988.