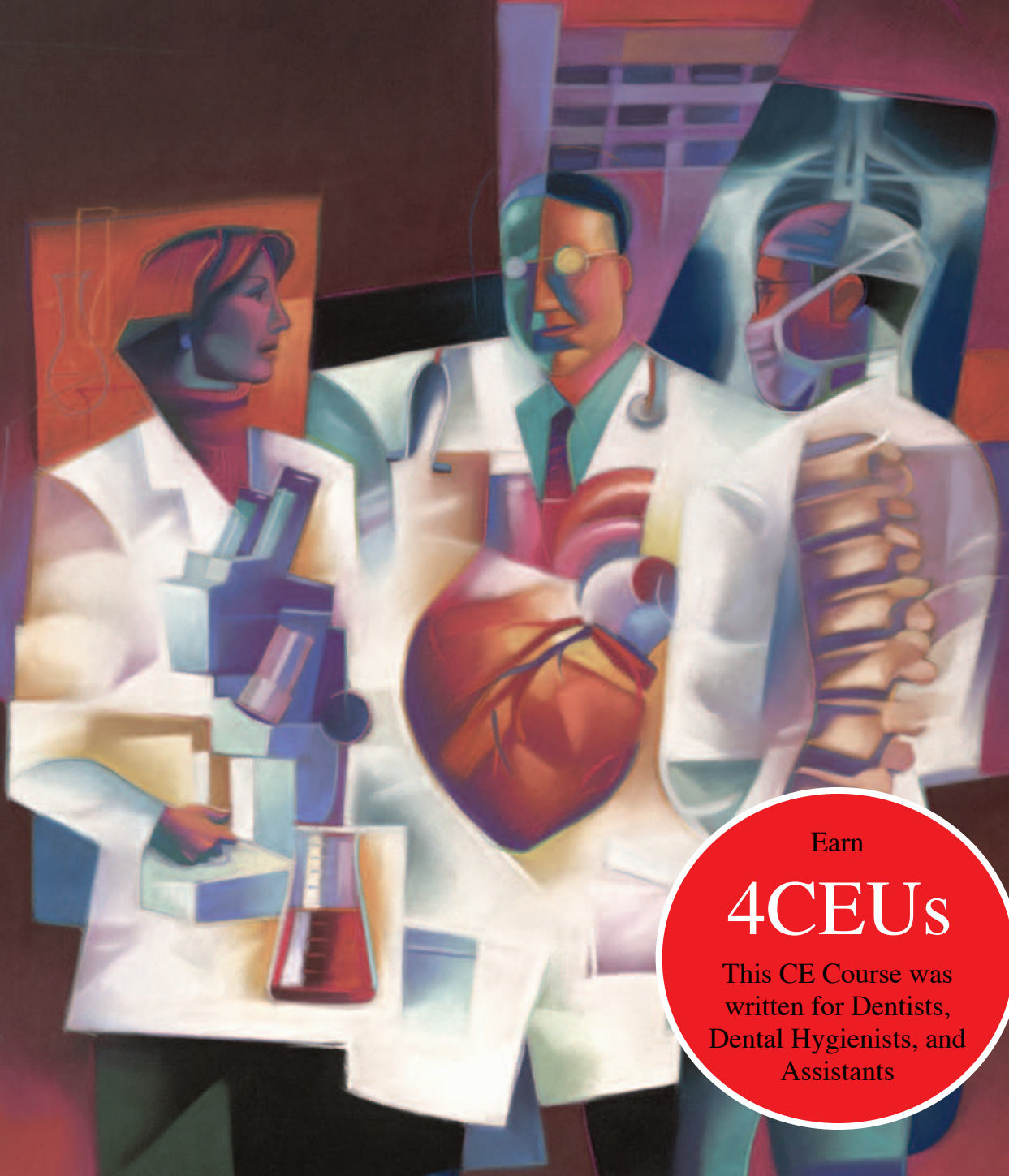


Periodontal Inflammation: The Oral-Body Health Connection

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Educational Objectives

The purpose of this course is to educate dentists and hygienists about the relationship between periodontal disease and systemic diseases such as cardiovascular disease, diabetes mellitus, respiratory disease, and osteoporosis. After completing this course the clinician will be able to

1. explain the mechanism by which periodontal disease causes production of inflammatory mediators that enter the systemic circulation and affect organ systems in the body;
2. explain the mechanism by which inflammatory mediators effect vessel plaque formation and lead to an increased risk of cardiovascular disease;
3. explain the mechanism by which periodontal disease mediators worsen the condition in diabetes mellitus;
4. describe the association of periodontal disease and the increased risk of development of pneumonia and COPD in the elderly;
5. describe the probable association of periodontal disease, tooth loss, and osteoporosis; and
6. clarify how appropriate treatment of periodontal disease can lead to improvement in outcomes in many of the above disease processes.

Introduction

Inflammation represents the body's protective response against injury and tissue destruction. This response consists of a spectrum of highly coordinated events that occur at the cellular and tissue level. Its purpose is to destroy, dilute, or sequester the injurious agent and the injured tissues in order to permit healing. Inflammation is a defensive mechanism intended to protect the host, but can also be potentially harmful. Clinical signs of inflammation are redness due to open blood vessels, heat due to warmth of blood, swelling due to edema, pain due to stimulation of pain receptors, and loss of function due to edema.¹

Periodontitis is the inflammation of the periodontium (gingiva and underlying connective tissue) resulting in features such as clinical attachment loss, alveolar bone loss, and periodontal pocketing. In periodontitis, one can see enlargement or recession of gingiva, bleeding upon probing, increased tooth mobility, drifting of teeth, and/or tooth exfoliation. Chronic periodontitis is a slowly progressing disease process that may occur continuously or in bursts of activity. Histologically, the gingival tissue from chronic periodontitis display junctional epithelium more apical to the cemento-enamel junction, loss of collagen fibers subjacent to the pocket epithelium, bone loss, numerous neutrophils (polymorphonuclear cells (PMNs), and dense inflamma-

tory cell infiltrate with plasma cells, lymphocytes, and macrophages.²

Tissue damage in chronic periodontitis is the result of major inflammatory and immunopathologic components activated by the host response. These include alteration of fibroblast function, activation of macrophage to release collagenase and other lytic enzymes, activation of lymphocytes, modulation of fibroblast growth, collagen synthesis, and stimulation of bone resorption. Prostaglandins and cytokines appear to be critically involved in tissue destruction caused by periodontitis.²

Concepts of the etiology of periodontitis imply a bacterial infection as the primary cause of the disease. Gingival inflammation is the result of plaque, or bacterial biofilm, which contains gram negative bacteria such as *P. gingivalis*, *B. forsythus*, *P. intermedia*, amongst many others.² These bacteria possess complex carbohydrates and proteins on their cell wall, called endotoxin or lipopolysaccharides (LPSs). When these molecules are detected by the host, a protective response ensues, resulting in inflammation, recruitment of white blood cells (WBCs), and release of cytokines and chemical mediators. The chemical mediators that cause the main systemic problems are IL-1, IL-6, and TNF-alpha. The function of IL-1 is to recruit osteoclasts (bone-resorbing cells), which remove bone matrix. The function of IL-6 is to increase fibrinogen, which helps in the clotting mechanism of injured blood vessels. The function of TNF-alpha is to primarily increase C-reactive proteins (CRPs) which recruit more macrophages to the site of injury. These mediators, although helpful in fighting insult to the body, can be harmful as well.

Periodontitis and Cardiovascular Disease

Atherosclerosis is the thickening and hardening of arteries which develops due to plaque buildup on arterial walls. This is a chronic inflammatory condition that affects injured arteries. There is growing evidence that infectious agents are causing this injury, thereby resulting in this chronic inflammation. This chronic inflammatory response increases the circulation of mediators such as C-reactive proteins and fibrinogen. There is a link between inflammation and atherosclerosis, and it suggests that chronic infections, such as those found in chronic periodontitis, may predispose someone to cardiovascular disease or exacerbate some with existing cardiovascular disease (CVD). The following is suggested regarding inflammatory mediators common in chronic periodontitis and CVD:³

- C-reactive proteins: found in chronic periodontitis and increased levels in the blood will cause damage to the smooth muscles of blood vessels and collection of macrophages.
- IL-1: found in association with increased risk of severe periodontitis and inside atherosclerotic plaques.

To see if arteries are inflamed as a result of atherosclerosis, a physician can test for CRPs by using a simple blood test at the same time the patient is being checked for cholesterol. The risk for CVD can be determined from the results.

CRP	Risk for CVD
Less than 1.0 mg/L	Low
1.0–2.9 mg/L	Intermediate
Greater than 3.0 mg/L	High

- **TNF-alpha:** also found in chronic periodontal infections. Systematically, it increases synthesis of triglycerides from the liver. Elevated triglyceride level is associated with lowered “good lipids,” known as HDL (high-density lipoproteins). This is associated with CHD (coronary heart disease).

- **Fibrinogen:** synthesized by the liver due to elevation of IL-6. IL-6 is also very high in chronic periodontitis. Fibrinogen is a clotting factor that is common in creating thrombi (clots) in the blood vessels resulting in myocardial infarction (MI or “heart attack”) and stroke.³

Similarities in the pathogenesis of atherosclerosis and periodontitis have suggested a common underlying biological mechanism for the two conditions. Most of the evidence supporting a relationship between chronic periodontitis and CVD is from studies performed in the late 1980s and have continued through 2000s, associating patients with a history of MI with worse oral health than control subjects. Those with acute MI had dental health worse than those who were healthy.⁸ The incidence of mortality and coronary heart disease increased as the severity of periodontal disease increased.⁹ Patients with a 20% increase of periodontal bone loss had a 40% increase in developing chronic heart disease (nonfatal MI, angina pectoris, and CHD deaths).⁴ *P. gingivalis* has the ability to induce the oxidation of “bad lipids,” known as LDL (low-density lipoproteins), which are pathogenic for plaque formation on arterial walls.^{5,6} CRP levels in patients with chronic periodontitis fall in the range of those who have cardiovascular disease.⁷ Gentle mastication can induce release of endotoxins from the mouth into the bloodstream, especially in patients with severe periodontitis. Also, periodontal pockets are a chronic source of passage of pro inflammatory bacterial components in the bloodstream.¹⁰ Periodontitis and gingivitis are independently associated with the risk of cerebral ischemia (stroke).¹¹

Healthy Heart Habits*

- Eat a heart-smart diet including a variety of fruits, vegetables, and grains.
- Exercise regularly.
- Limit soda, candy, alcohol, and sodium.
- Identify and reduce sources of stress.
- Know your blood pressure.
- Avoid smoking.

*American Heart Association

Monitor your patients’ blood pressure at initial exams and on a yearly basis, as they may have undiagnosed or uncontrolled hypertension. If medications are listed, ask if they adhere to dosing recommendations.

NORMAL	<120/<80
MILD HYPERTENSION	120–140/80–90
HYPERTENSION	>140/>90

Clinical manifestations with those who have cardiovascular disease are hypertension (See Box Above), fatigue after mild activity or when lying down, swelling of ankles, angina (chest pains), myocardial infarction (heart attack), or cerebral vascular accident (stroke). It is essential that a thorough medical history and blood pressure be taken during initial oral examinations. If a patient answers affirmatively to any of the above questions he/she may be taking medications, or in fact, be unaware of his/her condition. If the patient is not well controlled or if any question exists regarding his/her health status, a medical consultation is essential. A well-controlled hypertensive patient, for example, will see his/her physician every three to six months. However, as a general practitioner dentist, it is essential to check the patient’s blood pressure at initial examination and on a yearly basis.¹²

Chronic Periodontitis and Diabetes Mellitus

Diabetes mellitus is one of the most common diseases in the United States. It is a chronic metabolic disorder affecting carbohydrate, fat, and protein metabolism resulting in hyperglycemia. There are two types of diabetes mellitus: Type I is caused by defective secretion of insulin, while type II is a result of impaired insulin action due to tissue resistance. Appropriate measures can be taken to control blood glucose levels and prevent both acute and chronic complications.¹⁵

Patients with uncontrolled diabetes are prone to oral complications such as gingivitis, periodontal disease, fungal infections, and caries due to xerostomia. Uncontrolled diabetes also affects wound healing. However, those with controlled diabetes will heal similar to the nondiabetic patient. The most common oral symptom with diabetes is the increased prevalence and severity of periodontitis. Poorly controlled diabetes results in rapid progression of gingivitis and periodontitis. The degree of metabolic control and duration of diabetes are closely associated with the severity of periodontal disease.¹⁶

Periodontal disease has two components: bacteria and host response. There is a bidirectional relationship between periodontal disease and diabetes:^{17, 18, 19}

- The presence of diabetes increases the prevalence, incidence, and severity of periodontitis.^{17,18}
- Advanced glycation end products (AGE), which increase rapidly in poorly controlled diabetics, tend

to transform important inflammatory cells into more destructive cells, thereby resulting in more damage to many parts of the body as well as the periodontium.¹⁸

- Hyperglycemia results in impaired neutrophil chemotaxis. Neutrophils are the first cells in the host response against bacteria. Therefore, it can be concluded that the host response will be impaired during gingivitis and periodontitis.^{17, 18, 19}

- Diabetes leads to a two- to fourfold greater risk of severe bone loss. As glycemic control worsens, the effects of periodontal disease become greater.¹⁷

- Due to the high vascularity of acute periodontitis, the inflamed periodontium may serve as an endocrine-like source for TNF-alpha which antagonizes the effects of insulin. Therefore, glycemic control can be improved with periodontal treatment in these patients.¹⁸

- Treatment of chronic periodontitis helps glycemic levels in some patients.^{17, 18, 19}

The following has also been confirmed:²⁰

- The severity of periodontal disease increases with the duration of diabetes.

- Periodontitis is an infection that is twice as prevalent in diabetic individuals as in nondiabetics.

- Severe periodontal infections are much more common in type II diabetics and can worsen the metabolic control of diabetes.

- The chronic nature of periodontal infection represents a greater long-term risk for the diabetic than do acute infections.

- *P. gingivalis* is able to sustain systemic inflammation, which is another reason why diabetics need to have their chronic periodontitis treated.

- The increased inflammatory and cytokine response seen in diabetes is responsible for the dysregulation of lipid metabolism, insulin resistance, and long term microvascular complications. Chronic periodontitis could magnify the already elevated cytokine response and contributes to the overall burden of systemic inflammation.

- Mechanical periodontal therapy with adjunctive systemic doxycycline resulted in a 0.6% decrease in glycated hemoglobin.²⁰

Clinical manifestations of poorly controlled or uncontrolled diabetes are mostly polyphagia (increased hunger), polydipsia (increased thirst), polyurea (increased urination), pruritis (itching), weakness, increased susceptibility to infections, xerostomia leading to dental caries, burning mouth or tongue, altered taste sensation, candidiasis and other opportunistic infections, and severe gingivitis and periodontitis. It should be a common practice to ask patients if diabetes runs in the family. It is essential for the dental clinician to look for these signs and consider referring the patient for a work-up for diabetes.¹²

Look for clinical signs of uncontrolled or undiagnosed diabetes. Consider a physician referral when necessary.

The primary methods used for diagnosing diabetes mellitus are examining blood glucose levels for short- or long-term control via the glycated hemoglobin test, or HgbA1c test. This test is used because it monitors the patient's long-term control (a period of approximately 90 days due to the fact that red blood cells are replaced every 90 days). Consequently, the patient needs to be reassessed every three months for disease management. A normal value for the HgbA1c test is 6.0%–6.5%.¹⁴ If a patient's levels are greater than 7.9%, it is indicative of poor control and will require intervention with the patient's physician. Antibiotic prophylaxis will be necessary for any emergency dental treatment in these poorly controlled diabetic patients.¹²

Periodontal Inflammation and Respiratory Disease

The oral cavity has long been considered a potential reservoir for respiratory pathogens. Mechanisms of infection can be due to aspiration into the lung of the oral pathogens capable of causing pneumonia. In hospitalized patients and those in nursing homes, bacteria that colonize the teeth can potentially be aspirated into the lungs and can lead to pneumonia and decreased overall lung function measured by spirometry.¹³

Effective plaque control must be monitored in at-risk patients. Power toothbrushes and/or use of an antimicrobial toothpaste or rinse can be an effective option.

There are two routes of spreading oral microorganisms to the lower respiratory tract: hematogenous (via blood pathway) and aspiration. The hematogenous route is rare. However, aspiration is much more common. Aspiration of material from the upper airway occurs in 45% of healthy subjects and in 70% with impaired consciousness in one study. Periodontal disease and poor oral hygiene might result in a higher concentration of oral pathogens in the saliva, and the amount of these pathogens overwhelm local immune defenses. Dental plaque may also harbor colonies of pulmonary pathogens and promote their growth.¹³

The symptoms of chronic obstructive pulmonary disease (COPD) and pneumonia are difficulty breathing, constant coughing up of phlegm, flu-like symptoms, and fatigue. If a patient presents these symptoms, it is necessary to refer the patient to his/her physician for further workup.

Periodontitis and Osteoporosis

Osteoporosis is a debilitating metabolic bone disease that primarily affects elderly women. There is increasing evidence that osteoporosis and the underlying loss of bone mass is associated with periodontal disease and tooth loss. Current evidence, including several prospective studies, supports an association of osteoporosis and the onset and progression of periodontal disease in humans.²¹

Potential mechanisms by which host factors may influence onset and progression of periodontal disease directly or indirectly include underlying low bone density in the oral cavity, bone loss as an inflammatory response to infection, genetic susceptibility, and shared exposure to risk factors.²¹

Some studies have suggested that hormone replacement therapy is associated with retention of more teeth in old age. Nutritional factors also play a role in modifying periodontal disease, such as vitamin D and calcium.²² Calcium, a mineral, is used for building bones and teeth and in maintaining bone strength. Vitamin D plays a major role in calcium absorption. The relationship between calcium absorption and vitamin D is similar to that of a locked door and a key. Vitamin D is the key that unlocks the door and allows calcium to leave the intestine and enter the bloodstream. Vitamin D also works in the kidneys to help reabsorb calcium that otherwise would be excreted.²⁴

Five Steps to Bone Health*

1. Take recommended daily amounts of vitamin D and calcium.
2. Engage in weight-bearing exercise.
3. Avoid smoking and excessive alcohol.
4. Consult a physician about bone health.
5. Have a bone density test, and take medication when appropriate.

*National Osteoporosis Foundation

Systemic loss of bone density in osteoporosis, including that of the oral cavity, may provide a host system that is increasingly susceptible to infectious destruction of periodontal tissue. Understanding this association will aid health professionals to provide improved means to prevent, diagnose, and treat this disease.²¹

Osteoporosis tends to take place in postmenopausal women not receiving hormone treatment. Common signs of a person with osteoporosis are petite stature and common fractures to long bones. The diagnosis of osteoporosis can be via radiographs, symptomology, and blood workup. A common treatment for osteoporosis is hormone replacement with estrogen.¹² However, due to the onset of breast cancer, medications such as bisphosphonates (e.g. alendrolate— brand name Fosamax®) are being used. These medications prevent the action of osteoclasts. In addition, there is some evidence that some medications used to treat osteoporosis can be helpful in management of periodontal disease.^{21, 22}

Controlling Oral Inflammation

Inflammation is a protective process of the body to fight injury and infection. While its purpose is to help in healing and removal of infection, its actions can be harmful. Therefore, it is important that a patient maintain high standards in good oral hygiene. Dental professionals realize that this is not an easy task for most patients to accomplish on their own. Compliance to oral hygiene regimens can be time-

consuming, and many patients may lack the dexterity required to effectively remove plaque from all surfaces.

In order to fight oral inflammation, mechanical removal of pathogens via scaling and root planing in a diseased sulcus/pocket is important. Therefore, a prophylaxis/periodontal maintenance schedule based on the patient's needs (not insurance status) must be recommended. Locally applied antimicrobials (LAA) PerioChip®, Arestin®, Atridox®) can be beneficial in periodontal pockets 5 mm or more in depth. These products can release medication into the periodontal pockets for a length of time, reducing the bacterial/inflammatory challenge and creating an environment for better healing.

To increase the effectiveness of patient home care additional therapies are recommended. An option is the inclusion of an antimicrobial/anti-inflammatory toothpaste that contains triclosan with a copolymer (Colgate® Total®). Triclosan kills bacteria by interfering with the enzyme necessary for fatty-acid synthesis.²³ In addition, triclosan works directly on the inflammation process by directly inhibiting potent inflammatory mediators. Adding an antimicrobial in this fashion offers benefit without an additional step and therefore reduces compliance as an issue. Another means of adding an antimicrobial is in the form of a mouth rinse.

Chlorhexidine gluconate is a broad-spectrum antiseptic antimicrobial. It can be used as a rinse to prevent gingivitis by reducing and inhibiting the formation of plaque and as an LAA for the reduction in periodontal pockets (PerioChip®). Because it is positively charged, it binds to the oral mucosa and bacterial biofilm for up to 12 hours (and longer based on the delivery system). For this reason, chlorhexidine is a highly effective adjunct therapy. Another rinse option is stannous fluoride, which acts as an antibacterial and inhibits the formation of plaque.

If periodontal health cannot be obtained through regular periodontal maintenance and good oral hygiene, then the patient should be referred to a periodontist. A patient should also be referred if he/she has diabetes or cardiovascular disease along with periodontal disease for management of periodontal inflammation.

Conclusion

Inflammation, although a protective process, can be harmful. Periodontitis is the result of inflammation to the periodontium, and cytokines, or chemical mediators, are the result of inflammatory cells fighting against bacterial plaque. High amounts of these mediators can affect the body's systems, especially the arteries and can potentially cause more harm in a patient with compromised cardiovascular health. Dental professionals should include blood pressure screenings as part of their patient assessment practices and discuss heart-healthy strategies.

Dental professionals should assess risk for patients with diagnosed (or undiagnosed) diseases such as diabetes, respiratory diseases, and osteoporosis and refer patients to a physician or periodontist as necessary.

It is very important to evaluate and monitor oral hygiene in these patients. Consider the inclusion of antimicrobials as part of patient home care to enhance plaque control.

Fosamax® (MERCK & CO, INC., Whitehouse Station, NJ)
PerioChip® (OMNII Oral Pharmaceuticals, West Palm Beach, FL)
Arestin® (OraPharma, Warminster, PA)
Atridox® (CollaGenex Pharmaceuticals, Newtown, PA)
Colgate® Total® (Colgate Palmolive, New York, NY)

Author Profiles

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Dr. Richard Nejat received his Doctorate of Dental Surgery from New York University, where he graduated in the top of his class, receiving the prestigious OKU honors. Following graduation, Dr. Nejat began a three-year residency in periodontics and dental implants at Stony Brook University-State University of New York, where he earned his certificate in periodontics.

He is an active instructor in professional continuing education on topics including periodontal medicine, computer-guided and minimally invasive dental implant surgery, and periodontal plastic surgery.

Dr. Nejat is currently involved in numerous clinical research projects involving flapless dental implant surgery, minimally invasive periodontal plastic surgeries, and computer-guided dental implant surgery with immediate function.

Dr. Nejat is a Diplomate of the American Board of Periodontology. He maintains private practices in Manhattan, NY and Nutley, NJ.

Daniel Nejat, DMD

Dr. Daniel Nejat graduated magna cum laude from Drew University with a Bachelor of Arts. He continued his education by receiving a Doctorate in Dental Medicine at the University of Medicine and Dentistry of New Jersey, Dental School. Presently, Dr. Nejat is completing his postdoctoral periodontal residency at New York University College of Dentistry.

Morris Nejat, MD

Dr. Morris Nejat graduated cum laude from Drew University in New Jersey and obtained his medical degree from the University of Medicine and Dentistry of New Jersey, New Jersey Medical School. His internship and residency in pediatrics were completed at North Shore University Hospital, Cornell University Medical College. His allergy/immunology fellowship was performed at the R.A. Cooke Institute of Allergy at St. Luke's/Roosevelt Hospital Center, Columbia University College of Physicians and Surgeons. Dr. Nejat is Board Certified in pediatrics and has received appointments in pediatrics at St. Luke's/Roosevelt Hospital Center and Bellevue Hospital Center in New York City.

Dr. Nejat is frequently quoted in the media, including NBC news, Fox News, WB11 News, ABC news and numerous consumer publications. He also frequently lectures to physicians and consumers on the topics of allergy, asthma, eczema and food allergies.

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Course Questions

1. Inflammation represents the body's protective response against injury and tissue destruction.

- a. True
- b. False

2. Which of the following statements are true?

- a. Inflammation consists of coordinated events at both the cellular and tissue level.
- b. It can destroy an injurious agent (e.g. bacteria).
- c. It can be harmful to the host.
- d. All of the above.

3. Clinical signs of inflammation do NOT include

- a. redness and heat.
- b. edema and pain.
- c. headaches.
- d. swelling and loss of function.

4. Tissue damage in periodontal disease is the result of inflammatory components activated by the host response.

- a. True
- b. False

5. Inflammation occurs when the body detects endotoxins from host cells.

- a. True
- b. False

6. Cell mediators are messenger cells that recruit other cells into the area of infection.

- a. True
- b. False

7. The chemical mediator IL-1 recruits osteoclasts, which are bone-building cells.

- a. True
- b. False

8. The presence of elevated levels of chemical mediators can be harmful.

- a. True
- b. False

9. Atherosclerosis is the thickening and hardening of the

- a. PDL.
- b. blood-brain barrier.
- c. arteries.
- d. intestinal lining.

10. Atherosclerosis and periodontitis are chronic inflammatory diseases.

- a. True
- b. False

11. CRP is found in both chronic periodontitis and CVD. In increased levels in the blood, CRP will cause damage to smooth muscles of blood vessels. CRP levels in chronic periodontitis and CVD fall into the same range.

- a. True
- b. False

12. Research has shown that

- a. patients with a history of MI typically have good oral health.
- b. the incidence of mortality and CHD increased with the severity of periodontal disease.
- c. a 20% increase of periodontal bone loss has no effect on CHD.
- d. Both a and c.

13. Cellular mediator TNF-alpha is found in chronic periodontitis and is associated with lowering "good lipids" known as

- a. LDL.
- b. PDL.
- c. HDL.
- d. MOL.

14. Periodontal pockets are a chronic source of passage of pro inflammatory bacterial components into the bloodstream.

- a. True
- b. False

15. Diabetes is

- a. not very common.
- b. a chronic metabolic disorder.
- c. controllable to prevent complications.
- d. both b and c.

16. Patients with uncontrolled diabetes are prone to oral complications such as

- a. gingivitis and periodontal disease.
- b. fungal infections.
- c. caries due to xerostomia.
- d. all of the above.

17. There is a bidirectional relationship between periodontal disease and diabetes that indicates that the severity of

- a. diabetes will increase as periodontal disease gets worse.
- b. periodontal disease will increase as control of diabetes worsens.
- c. both a and b.
- d. none of the above.

18. The severity of periodontal disease increases with the duration of diabetes.

- a. True
- b. False

19. Clinical manifestations of poorly controlled or uncontrolled diabetics are

- a. burning mouth or tongue.
- b. polydipsia (increased thirst).
- c. altered taste sensation.
- d. all of the above.

20. Periodontitis is an infection that is not as prevalent in diabetic individuals as in nondiabetics.

- a. True
- b. False

21. Diabetes mellitus type I is caused by defective secretion of insulin, while type II is a result of impaired insulin action due to tissue resistance.

- a. True
- b. False

22. Antibiotic prophylaxis is necessary for any emergency dental treatment in poorly controlled diabetic patients.

- a. True
- b. False

23. The oral cavity is a potential reservoir for respiratory pathogens.

- a. True
- b. False

24. Bacteria that colonize the teeth cannot be aspirated into the lungs and lead to pneumonia and decreased overall lung function.

- a. True
- b. False

25. Oral microorganisms spread to the lower respiratory tract through

- a. a hematogenous route (via blood pathway).
- b. aspiration.
- c. osmosis.
- d. both a and b.

26. There is increasing evidence that osteoporosis and the underlying loss of bone mass are associated with periodontal disease and tooth loss.

- a. True
- b. False

27. There is some evidence that some medications used to treat osteoporosis can be helpful in management of periodontal disease.

- a. True
- b. False

28. Antimicrobials that are effective in the inhibition of plaque are

- a. triclosan.
- b. chlorhexidine.
- c. stannous fluoride.
- d. all of the above.

29. Triclosan is effective because it

- a. kills bacteria by interfering with the enzyme necessary for fatty-acid synthesis.
- b. directly inhibits potent inflammatory mediators.
- c. is an antiseptic, and bacteria will not develop resistance to it.
- d. all of the above.

30. The inclusion of antimicrobials into the oral hygiene process can help enhance plaque control.

- a. True
- b. False

Periodontal Inflammation: The Oral-Body Connection

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5 4 3 2 1 0
- Were the course objectives accomplished?
5 4 3 2 1 0
- Please rate the course content.
5 4 3 2 1 0
- Please rate the instructors' effectiveness.
5 4 3 2 1 0
- Was the overall administration of the course effective?
5 4 3 2 1 0
- How do you rate the authors' grasp of the topic?
5 4 3 2 1 0
- Do you feel that the references were adequate?
1 Yes 2 No
- Do you feel that the educational objectives were met?
1 Yes 2 No
- If any of the continuing education questions were unclear or ambiguous, please list them:

- Was there any subject matter you were unclear on? Please describe.

- Would you participate in a program similar to this one in the future on a different topic of interest? 1 Yes 2 No
- What additional continuing dental education topics would you like to see?

- | | |
|---------------------|---------------------|
| 1. (A) (B) (C) (D) | 16. (A) (B) (C) (D) |
| 2. (A) (B) (C) (D) | 17. (A) (B) (C) (D) |
| 3. (A) (B) (C) (D) | 18. (A) (B) (C) (D) |
| 4. (A) (B) (C) (D) | 19. (A) (B) (C) (D) |
| 5. (A) (B) (C) (D) | 20. (A) (B) (C) (D) |
| 6. (A) (B) (C) (D) | 21. (A) (B) (C) (D) |
| 7. (A) (B) (C) (D) | 22. (A) (B) (C) (D) |
| 8. (A) (B) (C) (D) | 23. (A) (B) (C) (D) |
| 9. (A) (B) (C) (D) | 24. (A) (B) (C) (D) |
| 10. (A) (B) (C) (D) | 25. (A) (B) (C) (D) |
| 11. (A) (B) (C) (D) | 26. (A) (B) (C) (D) |
| 12. (A) (B) (C) (D) | 27. (A) (B) (C) (D) |
| 13. (A) (B) (C) (D) | 28. (A) (B) (C) (D) |
| 14. (A) (B) (C) (D) | 29. (A) (B) (C) (D) |
| 15. (A) (B) (C) (D) | 30. (A) (B) (C) (D) |

Check this box to receive verification of your score.

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AUTHORS DISCLAIMER
 The authors of this course have no commercial ties with the sponsors or the providers of the unrestricted educational grant for this course.

EDUCATIONAL OBJECTIVES
 After reading this article, the clinician will be able to

- explain the mechanism by which periodontal disease causes production of inflammatory mediators which enter the systemic circulation and affect organ systems in the body;
- explain the mechanism by which inflammatory mediators effect vessel plaque formation and lead to an increased risk of cardiovascular disease;
- explain the mechanism by which periodontal disease mediators worsen the condition in diabetes mellitus;
- describe the association of periodontal disease and the increased risk of development of pneumonia and COPD in the elderly;
- describe the probable association of periodontal disease, tooth loss, and osteoporosis; and
- clarify how appropriate treatment of periodontal disease can lead to improvement in outcomes in many of the above disease processes.

INSTRUCTIONS

All questions should have only one answer. Grading of this examination is done manually. Participants will receive verification in the mail within two weeks after taking an examination.

SPONSOR/PROVIDER

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COURSE CREDITS/GOST

All participants scoring at least 70% (answering 21 or more questions correctly) on the examination will receive verification of 4 CEUs. The formal continuing education program of this sponsor is accepted by the AGD for Fellowship/Mastership credit. For current term of acceptance, please contact the ADTS. "DANB Approval" indicates that a continuing education course appears to meet certain specifications as described in the DANB Recertification Guidelines. DANB does not, however, endorse or recommend any particular continuing education course and is not responsible for the quality of any course content. Participants are urged to contact their state dental boards for continuing education requirements. The cost of this course is \$55.00.

EDUCATIONAL DISCLAIMER

The information presented here is for educational purposes only. It may not be possible to present all information required to utilize or apply this knowledge into practice. It is therefore recommended that additional knowledge be sought before attempting a new procedure or incorporating new technique or therapy. The opinions

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PARTICIPANT FEEDBACK

Questions can be e-mailed to jvillani@needce.com, faxed to 216-398-7922, or sent online via form available at www.needce.com.

RECORD KEEPING

The ADTS maintains records of your successful completion of any exam. Please contact our offices for a copy of your continuing education credits report. This report, which will list all credits earned to date, will be generated and mailed to you within five business days of receipt of your request.

CANCELLATION/REFUND POLICY

Any participant who is not 100% satisfied with this course can request a full refund by contacting the Academy of Dental Therapeutics and Stomatology in writing.

COURSE EVALUATION

We encourage participant feedback pertaining to all courses. Please be sure to complete the survey included within the answer sheet.