

Oral Body Inflammatory Connection



Marvin Slepian, MD



Neil R. Gottehrer, DDS

Cardiovascular disease (CVD) and periodontal disease are among the most prevalent conditions affecting adults today. Periodontal disease has been shown to, at some time, affect at as many as 75% of adults in the United States.¹ As for heart disease, the American Heart Association (AHA), in 2004, estimated that as many as 80.7 million adults (1 of every 3 adults) have cardiac disease.² Of the estimated 80.7 million American adults who have one or more types of CVD, 38.2 million are estimated to be less than 60 years of age. Cardiovascular disease includes the following categories: high blood pressure (HBP) (defined as systolic pressure 140 mm Hg and/or diastolic pressure 90 mm Hg, taking antihypertensive medication, or being told at least twice by a physician or other health professional that one has HBP)—73 million; coronary heart disease (CHD)—16 million; myocardial infarction (MI) (heart attack)—8.1 million; angina pectoris (chest pain)—9.1 million; heart failure—5.3 million; stroke—5.8 million; and congenital CV defects—650,000 to 1.3 million diagnoses.

The emergence of periodontal infection as a potential risk for CVD is leading to a convergence in oral and medical care that can only benefit its patients and the public's health.

THE ORAL-SYSTEMIC DISEASE CONNECTION

Recent clinical studies have suggested a strong connection between periodontal disease and CVD. In a 2005 paper, Desvarieux, et al³ studied the relationship between periodontal microbiota and carotid intima-media thickness (IMT) (The Oral Infections and Vascular Disease Epidemiology Study-INVEST). They stated that, “we report a positive independent relationship between carotid IMT and cumulative periodontal bacterial burden. Furthermore, we have shown

that the observed relationship with carotid IMT reflects both the burden and dominance of those pathogens etiologically related to periodontal disease in the subgingival microbial niche. These findings strengthen the hypothesis that oral infections may contribute to CVD morbidity and bolster the supposition that accelerated atherosclerotic development is a possible mechanism connecting chronic infections and CVD.”³

In a prior study, Arbes, et al⁴ concluded that there may be a greater risk for CHD-related events (such as MI) when periodontitis affects numerous teeth in the mouth, compared with subjects who exhibited a mild case of periodontitis affecting fewer teeth. In a 2007 paper published in the *New England Journal of Medicine*, Tonetti, et al⁵ stated that “endothelial dysfunction occurs early in the pathogenesis of arterial disease in response to a wide range of risk factors that have been shown to predict CV events in epidemiologic studies,” and that their study has “shown that the intensive treatment of periodontitis, a common potential source of low grade infection, results in an improvement in endothelial function.”

Studies conducted to determine if the treatment of chronic inflammation caused by other than periodontal disease (eg, chlamydia pneumonia) would reduce the clinical sequelae of CHD have not been able to show improvement in lessening the number of CHD events as the effective treatment of periodontitis has shown.⁵ The 5 dominant bacteria found to affect cardiac health are *Porphyromonas gingivalis*, *Tannerella forsythensis*, *Actinobacillus actinomycetemcomitans*, *Treponema denticola*, and *Micromonas micros*.

In the Atherosclerosis Risk in Communities study, completed in 2005, Beck, et al⁶ also appraised the connection of periodontal disease and CHD. They reviewed evidence that indicated that the chronic inflammatory burden of periodontal infection and the host response provide the basis for the observed association between periodontal disease and atherosclerosis and CHD. The strongest associations were then found with *P gingivalis*, *T forsythensis*, *T*

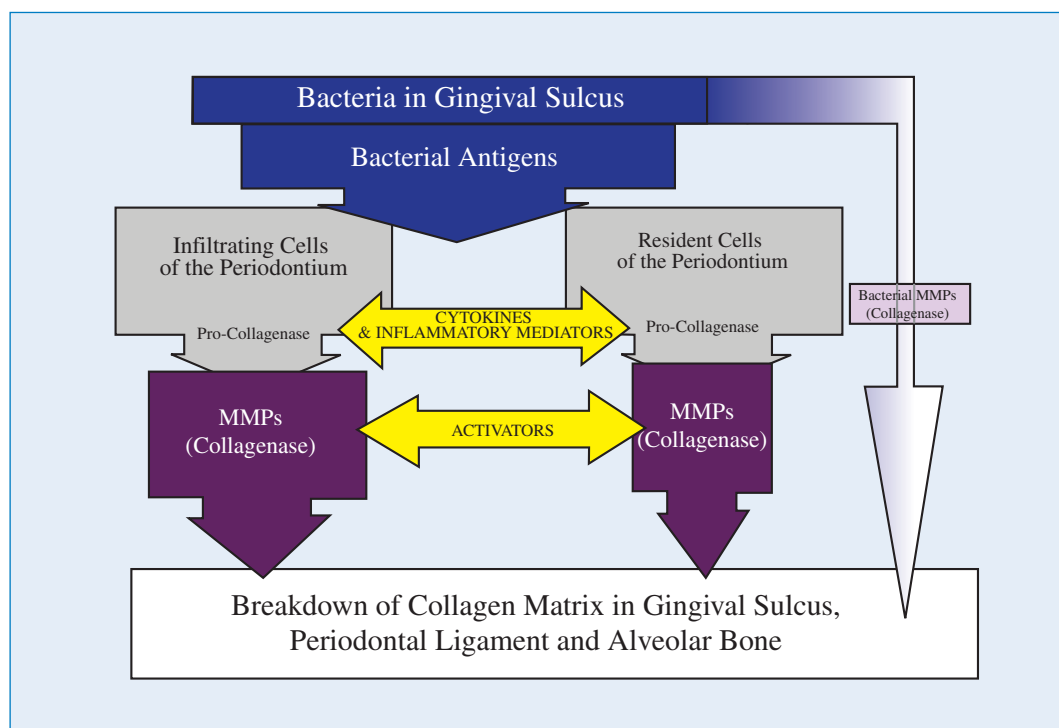


Figure 1. Host Response to bacterial antigens produces periodontal breakdown. (Gottehrer N. Managing risk factors in successful nonsurgical treatment of periodontal disease. *Dent Today*. Jan 2003; original Figure 1.)

continued on page 140

Oral Body Inflammatory...

continued from page 138

denticola, *Prevotella intermedia*, *M. micros*, *Prevotella nigrescans*, and *A. actinomycetemcomitans* (greater than 90%). Thus, they conservatively interpreted the results of this study to indicate that systemic exposure to oral organisms is related to the prevalence of detected CHD.⁶

In 2006, Demmer and Desvarieux⁷ reported that oral infection models have emerged as useful tools to study the hypothesis that infection is a CVD risk factor. They stated that periodontal infections are a leading culprit, after studying the reported associations between periodontal disease and CVD. They too, believe an association exists between the 2 diseases, but it is unknown whether it is causal or coincidental. They indicate that studies have enhanced the specificity of infectious exposure definitions, by measuring systemic antibodies to selected periodontal pathogens or by directly measuring and quantifying oral microbiota from subgingival dental plaque. They also indicate that results from these studies have shown positive associations between periodontal disease and CVD. They conclude that evidence supports an association among periodontal infections, atherosclerosis, and vascular disease.⁷

Periodontal treatment should be recommended on the basis of the value of its benefits for the overall health of patients, recognizing that patients are not healthy without

good oral health. The emergence of periodontal infection as a potential risk for CVD is leading to a convergence in oral and medical care that can only benefit patients and the public's health.

LOGICAL EXTENSION OF THE CONNECTION HYPOTHESIS

Because of the oral-systemic connection and association, clinical treatment studies have been performed to evaluate the effect of the aggressive clinical treatment of periodontal disease to the degree of heart disease. Iwamoto, et al⁸ studied chronic periodontitis patients, aged 43 to 75 years, and the risk of atherosclerosis. Treated nonsurgically, these patients (within one month of beginning treatment) recorded the lessening of elevated C-reactive protein (CRP) levels. They speculated that the periodontal infection may have contributed to the formation of the carotid plaque by means of prolonged subclinical inflammatory conditions.⁸

In 1997, Korman, et al⁹ (Figure 1) first summarized the various host-derived inflammatory mediators present in both the gingival crevicular fluid and periodontal tissues that may contribute to periodontal tissue destruction. These mediators include cytokines, which can mediate inflammation and activate osteoclasts to destroy bone, and matrix metalloproteinases (MMPs), which break down major structural tissue in the periodontal complex.⁹ Many of these same enzymes have

Periodontal disease severity correlated to the angiographic extent of coronary lesions, independent of systemic inflammatory status. ...these patients might benefit from an intensive periodontal therapy to prevent CAD progression.


been identified in patients with active CVD.

Blankenberg, et al¹⁰ studied plasma concentrations and genetic variations of MMPs, the same host-derived inflammatory mediators present in periodontal tissue destruction, and the prognosis of patients with CVD. Matrix metalloproteinases are involved in the breakdown of the extracellular matrix occurring during tissue repair and also in pathogenic conditions such as rheumatoid arthritis, periodontitis, and atherosclerosis. Collagenase MMP-8 (neutrophil collagenase or collagenase-2) is capable of initiating the degradation of the fibrillar collagens such as collagen type I, which is the major load-bearing molecule of the fibrous cap in atherosclerotic lesions. Several other MMPs, including MMP-2 and MMP-9, can further degrade the cleaved

collagen fragments. In their present population-based sample of middle-aged men followed for 10 years, they show that high serum MMP-8 concentration is an independent risk factor for acute MI, CHD, CVD, and death. The increased risk for CVD death was especially substantial in men with subclinical atherosclerosis at baseline: high serum MMP-8 concentration increased the risk for CVD death during the follow-up by 3-fold independently of other CVD risk factors.¹⁰

Amabile, et al¹¹, in a paper published in the *Journal of Internal Medicine*, agreed that periodontal disease has been recognized as a risk factor for systemic disease, but stated that its involvement in the pathogenesis of coronary artery disease (CAD) remains up for debate. They evaluated the potential relations between the severity of the periodontal disease inflammatory response and the angiographic lesion extent in patients with stable coronary artery disease. They evaluated 131 patients for the presence of CAD and divided them into 2 groups, one with lesions (85) and the other with absence of lesions (46). Mean periodontal pocket depth, high sensitivity CRP, serum amyloid A protein, and fibrinogen levels were measured. The results showed that the groups did not differ in the prevalence of traditional CV risk factors, but that the pocket depths were greater in patients with CAD, and that the systemic inflammation response was more pronounced with high levels of CRP, serum amyloid A, and fibrinogen. Multivariate analysis indicated a persistent high correlation between pocket depth and angiographic score, after adjustment for inflammatory markers.¹¹ They concluded that periodontal lesions predict presence of CAD stenosis in patients with CV risk factors.¹¹ Periodontal disease severity correlated to the angiographic extent of coronary lesions, independent of systemic inflammatory status. These results, they believe, suggest that these patients might benefit from an intensive periodontal therapy to prevent CAD progression.

Grau, et al¹² studied periodontal disease as a risk factor for ischemic stroke. The fact that chronic infectious disease may increase the risk of stroke prompted them to investigate whether periodontal disease, including periodontitis and gingivitis, is a risk factor for cerebral ischemia. They did a controlled study with 303 patients examined within 7 days after acute ischemic stroke or transient ischemic attack, 300 controls and 168 hospital controls with



Periodontal Record

Today's Date: _____

Patient Name: _____

Date of Birth: _____

Treatment Protocol Recommendations

Sharpen hand instruments prior to treatment | Use optical magnification
Use local, topical and or Nitrous Oxide anesthesia | Use an ultrasonic scaler

Stat-Ck™

Initial Periodontal Examination

Record a grade to every quadrant below

Grade	A	B	C	D	F	UR <input type="checkbox"/>	UL <input type="checkbox"/>
Pocket Depth	≤4mm at any site in quadrant	≤4mm at any site in quadrant	≤4mm at any site in quadrant	5-6mm at any site in quadrant	>6mm at any site in quadrant		
Bleeding on Probing (BOP)	No BOP at any site in quadrant	BOP at any site in quadrant	BOP at any site in quadrant	BOP at any site in quadrant	BOP at any site in quadrant		
Subgingival Debris	No Subgingival debris in any quadrant	No Subgingival debris in any quadrant	Subgingival debris in any quadrant	Subgingival debris in any quadrant	Subgingival debris in any quadrant		
Treatment Guidelines	Routine prophy	Scaling Prophy Host Modulation Full Mouth Ultrasonic	Scaling Root planing Prophy Host Modulation Full Mouth Ultrasonic	Scaling Root planing Prophy Host Modulation Full Mouth Ultrasonic Site specific Anti-infective	Scaling Root planing Prophy Host Modulation Full Mouth Ultrasonic Site specific Anti-infective	LR <input type="checkbox"/>	LL <input type="checkbox"/>

Place an asterisk (*) in the box to indicate quadrant(s) where site-specific anti-infectives (i.e. Arestin™) were placed.

Initial Periostat® Rx written on _____

Figure 2. STAT-CK documentation record for initial visit. (Gotthrehrer N. Antimicrobial host response therapy in periodontics: a modern way to manage disease. *Dent Today*. Sep 2006: original Figure 4.)

continued on page 141

Oral Body Inflammatory...

continued from page 140

nonvascular and noninflammatory neurological disease. A complete clinical and dental radiographic exam was performed for each patient. The individual mean clinical attachment loss measure at 4 sites per tooth served as the main indicator for periodontitis. The patients had higher clinical attachment loss than the controls. After adjustment for age, sex, number of teeth, vascular risk factors and diseases, socioeconomic and lifestyle factors, they found that the risk of cerebral ischemia increases with more severe periodontitis. The patients had a 4.3 times higher risk of cerebral ischemia than subjects with little or no periodontitis. Gingivitis and severe radiologic bone loss were also independently associated with the risk of cerebral ischemia, whereas caries was not. The study indicated that periodontal disease, a treatable condition, is an independent risk factor for cerebral ischemia in men and under age 60 subjects.¹²

These clinical studies provide strong evidence for physicians, faced with the challenge of keeping patients (who are at risk of CVD and adult onset diabetes) healthy, to request from the dentist the current periodontal status of their patients. Periodontitis has the potential to become, because of the connection, along with the present risks now identified by the AHA¹³, of tobacco smoke, high blood cholesterol, high blood pressure, physical inactivity, obesity and overweight, and diabetes mellitus, a recognized risk factor in cardiac disease.

NETWORK MANAGEMENT OF THE CONNECTION

Since periodontal disease results in inflammation, which can become systemic, *it is now critical for the dentist and physician to collaborate in helping patients reduce the inflammation*, which can become a significant risk factor for disease. Managing periodontal disease successfully may help in lowering risk factors for heart disease.

Golub and associates¹⁴ developed subantimicrobial dose doxycycline (SDD), taken at a 20 mg twice daily dosage, to treat periodontal disease by blocking tissue destructive enzymes and described its action in the *American Dentistry Association journal* in 1994. In 2000, Caton, et al¹⁵ described periodontal treatment with this SDD to improve the efficacy of scaling and root planing in adult patients with periodontitis. It is now considered the standard of care for nonsurgical periodontal

treatment in reducing the dental risk for cardiac disease, and has provided significant help in managing/controlling periodontal disease.¹⁶

With the knowledge that SDD improves the periodontal condition, Brown, et al¹⁷ studied the clinical and biochemical results of the metalloproteinase inhibition with SDD to prevent acute coronary syndromes (MIDAS) in a pilot trial. They described vulnerable plaque which demonstrated intense inflammation and macrophages secreted MMPs that degrade the fibrous cap, and ultimately leading to rupture. They hypothesized that SDD, 20 mg twice daily, would benefit patients with CAD by reducing inflammation and MMP activity and thus possibly prevent coronary plaque rupture events.¹⁷

They conducted a randomized, double blind, placebo-controlled pilot study for 6 months, using SDD or placebo treatments to reduce inflammation and prevent plaque rupture events.¹⁷ Fifty patients were studied, 26 taking SDD and 24 getting a placebo. Biochemical markers of inflammation were assessed in plasma at entry and at 6 months after therapy. In SDD-treated patients, high-sensitivity CRP protein was reduced by 46%. There was no significant reduction in placebo

patients. Interleukin-6 decreased from 22.1 to 14.7pg/ml. in SDD treated patients with no decrease of significance in placebo patients. On zymography, MMP-9 activity was reduced 50% by SDD therapy and was also unchanged in placebo treatment. They concluded that SDD appeared to exert potentially beneficial effects on inflammation, which could promote plaque stability.¹⁷

Based on Brown's study results¹⁷, physicians may be able to help manage a patient's periodontal condition by prescribing SDD, and actively work with the dentist to control the patient's periodontal risks. Dentists should take a stronger interest in patients' overall health and increasingly communicate with physicians. If dentists anticipate that they will be receiving more physician referrals, they must possess a basic knowledge of the science of inflammation and be prepared to help physicians understand the oral-systemic connection and how dentistry can help them with their patients. It is very helpful to have an organized approach to managing this information sharing and the interdisciplinary patient-related interactions that may arise from this.

An Initial Periodontal Examination should be completed for

every patient. It can be a limited exam, which can be easily done using a format that requires only screening and probing. The STAT-CK (Figure 2), developed by Dr. Neil Gottehrer and published in 2002, where all teeth are examined with the periodontal probe circumferentially, but not recorded at this initial visit, allows the patient's status to be graded A to F.¹⁸ Unlike traditional 6-point probing, this format is easily understood by the patient, allowing them a simple way of understanding their periodontal condition. This should be forwarded to the medical doctor (MD), with a copy given to the patient. The form gives the MD a report about the patient, as well as describing recommended treatment for the patient. With this information, the MD-DDS connection can be easily established and help to improve the patient's long-term health.

Figure 3 shows an image of one method used by Dr. Lee Ostler, author of *The Physician Marketing Handbook*, to report on a patient's oral health status and to ask for the physician's help in co-managing the patient. This Personalized Patient Report is part of a communications and referral-marketing system developed by Ostler to build beneficial relationships with medical doctors. It also helps put the emerging oral-systemic science into action in the dental office to benefit patients and enhance dental practices with new physician referrals.

Readers can go to the Web site mdreferrals.net and download a free sample copy of this form, as well as a science bibliography summarizing the current research. Additional information is also available on this informative website which will help dentists better serve their patients and build a strong connection with the physician to support future referrals in the MD-DDS network.

THE FUTURE OF THE MD-DDS CONNECTION

Major medical and dental insurance carriers are encouraging patients to take risk assessment tests for periodontal disease. CIGNA Dental¹⁹ has developed a new online tool for patients who may be at risk for gum disease. In a press release issued on August 28, 2008, they asked the question "Do you frequently have bad breath?" If so, they suggested that the consumer may need more than just a mint, as persistent bad breath is one of several signs that gum disease may be present. This question, along with many others, is part of CIGNA Dental's online Periodontal Disease Risk Assessment Tool©CIGNA 2008, to help people assess their own risk for gum (periodontal) disease.¹⁹

Fax Transmittal		
DATE:		
To: Dr.	Fax #	
From: G. Lee Ostler DDS, 1518 Judwin Ave, Richland, WA, 946-xxxx, 946-xxxx fax		
Re: Medical Co-Management for:		
Patient Name		Date of Birth
Re: Host Modulation therapy using Sub-antimicrobial Doxycycline Dose (SDD) Protocol for treatment of Periodontal Disease. *		
<p>This patient has been diagnosed with periodontal disease (persistent gingival bleeding and/or periodontal pockets). We are beginning a <u>dual therapy</u> program to treat this infection, which requires your involvement. The therapy includes:</p> <ol style="list-style-type: none"> 1- Non-surgical antimicrobial periodontal therapy combined with improved home care (the dental part), and 2- Host modulation therapy using a sub-antimicrobial doxycycline dose (SDD) protocol and medical monitoring (the medical part). <p>Our treatment goal is to reduce bleeding and inflammation around the teeth, reduce the microbial challenge in the mouth, and better manage the host response to this infection. In addition to the improved oral health, the extended objective is to reduce risk factors that are known to be associated with systemic disease conditions.</p> <p>While some physicians are comfortable with having us write these prescriptions and do this monitoring, we prefer to have the patient's physician manage the pharmaceutical and medical aspects of this approach, which in this case includes prescribing PerioStat (doxycycline hyclate 20 mg) 180 tabs, 1 tab BID for 90 days, one refill and doing the necessary blood work required to screen for related problems, and to monitor success from the systemic inflammation side of this treatment protocol. We believe this represents good medicine and allows the physician to be involved in what is truly a medical condition but which requires a dentist to diagnose and treat.</p> <p>Please indicate the course which you desire to take with the management of this patient, and fax this information back to me ASAP as our treatment program is beginning right away.</p>		
Physician Instructions & FaxBack Form		
Please fax to Dr. Ostler @ 509-946-xxxx		
(Please check # 1, 2 and 3)		
1	A	Have pt schedule medical appointment, for evaluation and Rx as requested.
	B	No appt necessary - Rx phoned in to: (Pref pharmacy).
2	A	Physician will order blood lab tests (to include CRP & HbA1c) and will send copy of labs to dentist.
	B	Blood work recently done (to include CRP & HbA1c) - will send copy to dentist.
3	A	OK for dentist to prescribe PerioStat - doxycycline, and order blood work with results copied to physician for medical management and follow up as necessary.
Doctor signature:		MD/DO
* Provide Dr. email to receive additional info including prescribing info, bibliography, and literature citations:		Dr. Email:
Confidential Information: If you are not the intended recipient of this fax, please destroy and contact sender.		

Figure 3. Sample of a MD fax for reporting patient diagnosis/treatment. Approval to reprint from Dr. Ostler.

CIGNA Dental insures approximately 10.9 million individuals including more than one-third of all Fortune 500 companies. Available in English and Spanish, this online quiz then gives a score that forecasts that person's risk for having gum disease. Results can be printed out and shared with their dentist at the next visit. It can also be shared with the physician who may now want to know the periodontal risk and order blood enzyme testing for physical evaluation.

"During my years as a practicing dentist, I saw first-hand how poor oral hygiene can affect an individual," said Dr. Michael Hahn, national dental director for CIGNA Dental, who was key in the creation of the Periodontal Disease Risk Assessment Tool. "CIGNA's development of this new tool will help people better understand their risk for gum disease. It's important for people to know that it is possible to have gum disease without having obvious warning signs, which is why regular dental check ups are so important."¹⁹ This test will stimulate early intervention and allow treatment of the disease before it causes major periodontal and CV problems.

CIGNA Dental's Periodontal Disease Risk Assessment Tool can be viewed by visiting www.cigna.com and typing "perio risk tool" in the search box. It serves as a companion to CIGNA Dental's online Cavity Risk Assessment Tool that was released in May 2007.¹⁹

Now that the consumers can do a risk assessment for periodontal disease, with an increased opportunity for them to consider treatment, a recent study supports the oral-systemic connection with statistics that periodontal treatment can have significant value relative to the cost of medical care. Albert, et al²⁰ conducted a study to evaluate the effect of periodontal treatment on overall medical costs. The study investigated the effect of periodontal treatment on Per Member Per Month costs within diabetes mellitus, CAD, and cerebrovascular disease. The study examined data from 144,225 enrollees with continuous coverage, who had one of these 3 chronic medical conditions for 2 years. It was concluded that periodontal care had an effect on the cost of medical care, with earlier treatment resulting in lower medical costs for these 3 medical conditions.²⁰

CLOSING COMMENTS

Knowing that a patient's medical risks may be reduced by periodontal treatment, and that this treatment can possibly reduce the burden of medical cost for the patient, it is now possible that more physicians

will participate in restoring a patient's periodontal health by suggesting a periodontal status exam from the dentist and possibly prescribing SDD, where indicated.

Chairside diagnostic tests should be developed to help the physician test for the specified enzymes and help make a periodontal diagnosis, which can make it simpler for the MD to refer the patient to the dentist for a periodontal assessment. Being made aware of the oral-systemic connection and having the diagnostic testing performed in the physician's office can be a very strong motivator for the patient to begin periodontal treatment. The future looks very bright, with physicians and dentists working together, to improve patient health and perhaps increase longevity of life. ♦

References

- Genco R, Offenbacher S, Beck J. Periodontal disease and cardiovascular disease: epidemiology and possible mechanisms. *J Am Dent Assoc.* 2002;133(suppl):14S-22S.
- Rosamond W, Flegal K, Furie K, et al. Heart disease and stroke statistics—2008 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation.* 2008;117:e25-146.
- Desvarieux M, Demmer RT, Rundek T, et al. Periodontal microbiota and carotid intima-media thickness: the Oral Infections and Vascular Disease Epidemiology Study (INVEST). *Circulation.* 2005;111:576-582.
- Arbes SJ Jr, Slade GD, Beck JD. Association between extent of periodontal attachment loss and self-reported history of heart attack: an analysis of NHANES III data. *J Dent Res.* 1999;78:1777-1782.
- Tonetti MS, D'Aiuto F, Nibali L, et al. Treatment of periodontitis and endothelial function. *N Engl J Med.* 2007;356:911-920.
- Beck JD, Eke P, Heiss G. Periodontal disease and coronary heart disease: a reappraisal of the exposure. *Circulation.* 2005;112:19-24.
- Demmer RT, Desvarieux M. Periodontal infections and cardiovascular disease: the heart of the matter [published correction appears in *J Am Dent Assoc.* Mar 2008;139:252]. *J Am Dent Assoc.* Oct 2006;137(suppl):14S-20S.
- Iwamoto Y, Nishimura F, Soga Y, et al. Antimicrobial periodontal treatment decreases serum C-reactive protein, tumor necrosis factor- α , but not adiponectin levels in patients with chronic periodontitis. *J Periodontol.* 2003;74:1231-1236.
- Kornman KS, Page RC, Tonetti MS. The host response to the microbial challenge in periodontitis: assembling the players. *Periodontol.* 2000;1997;14:33-53.
- Blankenberg S, Rupprecht HJ, Poirier O, et al; AtheroGene Investigators. Plasma concentrations and genetic variation of matrix metalloproteinase 9 and prognosis of patients with cardiovascular disease. *Circulation.* 2003;107:1579-1585.
- Amabile N, Susini G, Pettenati-Soubayroux I, et al. Severity of periodontal disease correlates inflammatory systemic status and independently predicts the presence and angiographic extent of stable coronary artery disease. *J Intern Med.* 2008;263:644-652.
- Grau AJ, Becher H, Ziegler CM, et al. Periodontal disease as a risk factor for ischemic stroke. *Stroke.* 2004;35:496-501.
- American Heart Association. Risk factors and coronary heart disease. AHA scientific position. <http://www.americanheart.org/presenter.jhtml?identifier=4726>. Accessed November 21, 2008.
- Golub LM, Wolff M, Roberts S, et al. Treating periodontal diseases by blocking tissue-destructive enzymes. *J Am Dent Assoc.* 1994;125:163-169.
- Caton JG, Ciancio SG, Blieden TM, et al. Treatment with subantimicrobial dose doxycycline improves the efficacy of scaling and root planing in patients with adult periodontitis. *J Periodontol.* 2000;71:521-532.
- Gottehrer NR, Martin JL. The standard of care for nonsurgical periodontal treatment for reducing the dental risk for cardiac disease. *Dent Today.* 2007;26:100-104.

- Brown DL, Desai KK, Vakili BA, et al. Clinical and biochemical results of the metalloproteinase inhibition with subantimicrobial doses of doxycycline to prevent acute coronary syndromes (MIDAS) pilot trial. *Arterioscler Thromb Vasc Biol.* 2004;24:733-738.
- Gottehrer NR, Shirdan TA. A new guide to non-surgical management of periodontal disease. *Dent Today.* 2002;21:54-59.
- Roman J. Are you at risk for gum disease? Cigna Dental's new online tool helps provide the answer. http://newsroom.cigna.com/article_display.cfm?article_id=942. Published August 28, 2008. Accessed November 21, 2008.
- Albert DA, Papapanou P, Conicella ML, et al. The effect of periodontal treatment on medical costs. Presented at the 35th Annual ADEA/ADR/CADR Meeting; March 8-11, 2006; Orlando, FL. Abstract 0058. http://iadr.confex.com/adr/2006Orld/techprogram/abstract_74102.htm. Accessed November 21, 2008.

Dr. Slepian is director of Interventional Cardiology and director of the Tissue Engineering Laboratory at the University of Arizona Sarver Heart Center. Dr. Slepian is professor of medicine (Cardiology) at the University of Arizona in Tucson. At Arizona he has also served as director of Interventional Cardiology at the Tucson VA Medical Center. In addition, Dr. Slepian is presently chairman and chief medical and scientific officer of SynCardia Systems Inc of Tucson, Arizona. Dr. Slepian received his AB degree in Biochemical Sciences from Princeton and his MD from the University of Cincinnati. He completed his internship and residency in Internal Medicine at NYU-Bellevue where he also served as chief resident. He trained in Cardiology at Johns Hopkins, completing a clinical and research fellowship. He subsequently received additional subspecialty training in Interventional Cardiology at The Cleveland Clinic, and also received postdoc-


torial training in Chemical Engineering and Polymer Chemistry at MIT and Washington University. He has also had a career in basic and translational research and technology transfer; his lab has developed many novel therapeutic solutions based on polymeric biomaterials that have found their way into clinical use today. He has been involved with bringing many new devices through the FDA regulatory process into clinical use, including most recently the total artificial heart. He is the author of more than 150 publications and 60 patents in the fields of vascular biology, polymeric biomaterials, local drug delivery, and artificial organs. He has received multiple awards for his academic and translational research activities and has been a frequent visiting professor and lecturer in both Medicine (Cardiology) and Biomedical Engineering, both nationally as well as internationally. He can be reached at (520) 626-8543 or slepian@email.arizona.edu.

Dr. Gottehrer has been in practice in suburban Philadelphia for more than 30 years, focusing his practice on cosmetics, implant dentistry, and periodontics. He is a graduate of the University of Maryland Dental School, received his postgraduate periodontal training at the University of Pennsylvania, and is a board-certified periodontologist. He teaches the senior elective course in Periodontics at the University of Maryland Dental School. He has published and lectured nationally and internationally, and is currently the president of the Institute of Advanced Oral and Physical Health in Havertown, Pa. He can be reached at (610) 449-9500 or dr.neilg@verizon.net.

Disclosure: Drs. Gottehrer and Slepian have no financial interest in any of the companies mentioned in this article.

OVERWHELMED?

Is all your dental literature driving you crazy?



Visit

DentistryToday.com

to have a 3,000-plus and growing archive
of clinical articles
at your fingertips.
It's free!
It's searchable!