INFLAMMATION:

The Relationship Between Oral Health and Systemic Disease

By JoAnn R. Gurenlian, RDH, PhD

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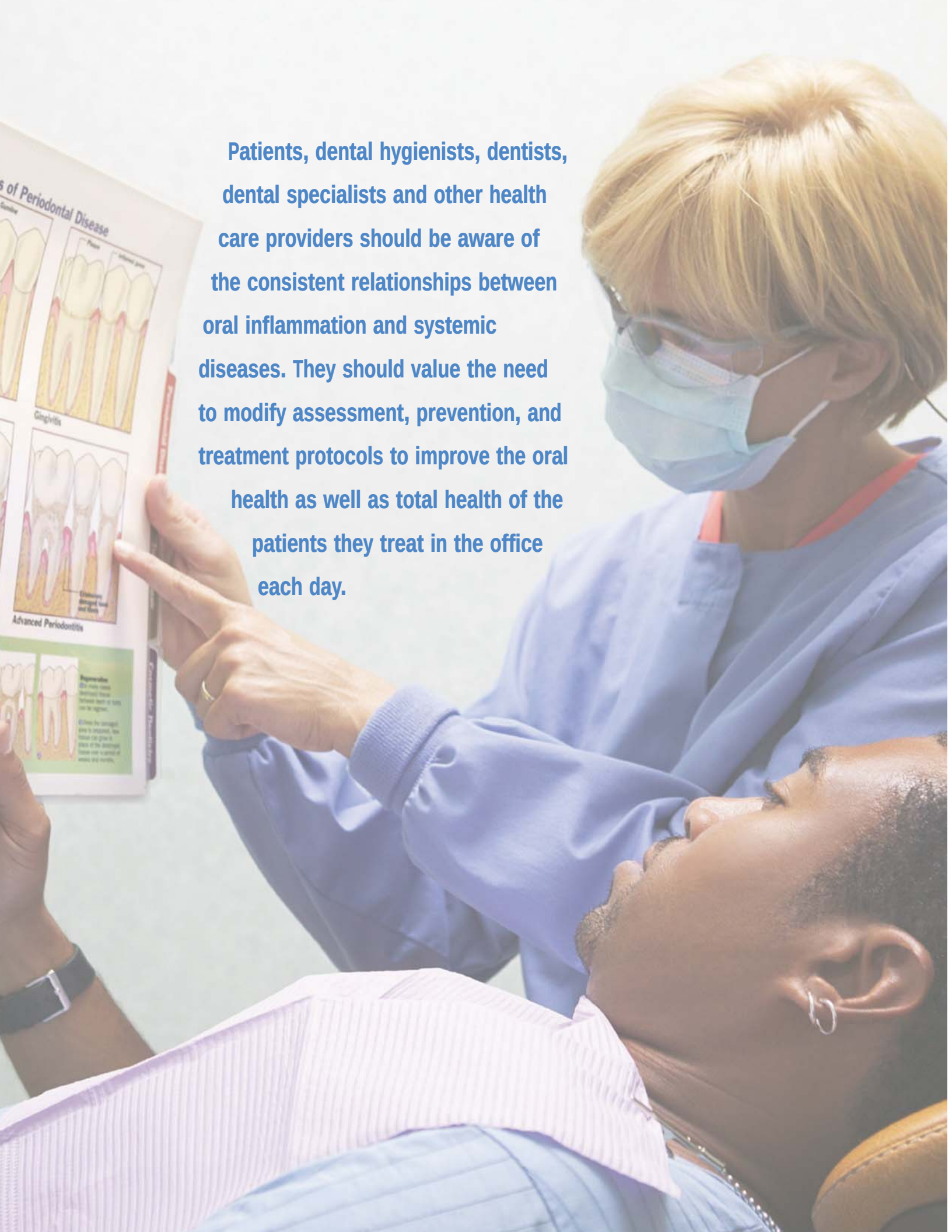

Patients, dental hygienists, dentists, dental specialists and other health care providers should be aware of the consistent relationships between oral inflammation and systemic diseases. They should value the need to modify assessment, prevention, and treatment protocols to improve the oral health as well as total health of the patients they treat in the office each day.

contents

JoAnn R. Gurenlian, RDH, PhD, is the owner of Gurenlian & Associates. She provides consulting and continuing education programs for health care providers. She has experience in general, periodontic, pediatric and orthodontic practices, and works part-time in a medical practice. She is an internationally recognized speaker on the topics of oral pathology, oral medicine, diabetes, and women's health. Dr. Gurenlian volunteers with local cancer, health and political organizations.

Clinical cover image courtesy of Dr. Randy Valentine, www.gumsbleeding.com.

Abstract

Since the mid 1990s, both the scientific community and the public have been inundated with articles addressing the association between systemic diseases and oral health. It seems that almost monthly there is an article in a fashion magazine reminding the public that tooth brushing and flossing can save their life. Some articles point to the notion that oral infection and bacteria may be linked to heart attack and stroke. Others dispel the association, indicating that there is not enough research to determine any relationship between the two. The questions that have been raised focusing on the relationship between periodontal diseases and systemic conditions now extend beyond cardiovascular disease and include diabetes, respiratory disease and adverse pregnancy outcomes. Research has demonstrated that the association between oral inflammation and systemic inflammation may be the key to understanding the deleterious effects on multiple organ systems. However, is the relationship so complex that it is like trying to crack the DaVinci Code, or can health care professionals and the public understand the role of inflammation in oral and systemic health?

Research has demonstrated that the association between oral inflammation and systemic inflammation may be the key to understanding the deleterious effects on multiple organ systems.

The purpose of this article is to review how the inflammatory process functions in the human body. The role of inflammation in oral and systemic health will be discussed. Translating this information into practical application for dental hygiene professionals will be addressed so that both inquiring patients and astute clinicians will capitalize on the opportunities for improving total health.

The Inflammatory Process

What is inflammation? Isn't this the process that is supposed to be good for our bodies? How can it now be something that causes harm to so many different aspects of the body? As we learn more about the biological mechanisms of inflammation, it becomes clear that this process is more complicated than was once thought.

Inflammation is the body's response to cellular injury. Despite the fact that the press has emphasized the harmful effects of inflammation, the fact remains that without this process, our bodies could not survive. Inflammation represents a protective response designed to rid the body of the initial cause of cell injury and the consequences of that injury. Cell injury may occur due to trauma, genetic defects, physical and chemical agents, tissue necrosis, foreign bodies, immune reactions and infections.

Inflammation is a local reactive change that involves the release of antibacterial agents from nearby cells that defend the host against infection. It also facilitates early tissue healing and repair. It contains—or "walls off"—the infectious or injurious agent and serves as a defense mechanism that the body can use to restore itself to a normal morphological form and function.

Table I: Physiologic Rationale for Cardinal Signs of Inflammation

Cardinal Signs of Inflammation	Physiologic Rationale
Rubor (redness)	Increased vascularity
Tumor (swelling)	Exudation of fluid
Calor (heat)	A combination of increased blood flow and the release of inflammatory mediators
Dolor (pain)	The stretching of pain receptors and nerves by the inflammatory exudates, and by the release of chemical mediators
Functio laesa (loss of function)	A combination of the above effects

McMahon RFT, Sloan P. Essentials of pathology for dentistry. Edinburgh: Churchill Livingstone; 2000, p. 26.

The inflammatory response consists of a vascular and a cellular reaction. These reactions are mediated by chemical factors derived from plasma proteins or cells. The classic signs of inflammation are redness, swelling, heat, pain and loss of function. The physiologic explanations for these signs appear in Table I. Other signs of inflammation include fever, leukocytosis or an increase in the number of circulating white blood cells, the presence of acute-phase proteins including C-reactive proteins (CRP), fibrinogen and serum amyloid A protein (SAA), and sepsis.

There are two types of inflammation: acute and chronic. Acute inflammation is characterized by a rapid onset and short duration. It manifests with exudation of fluid and plasma proteins, and emigration of leukocytes, most notably neutrophils. Chronic inflammation is of prolonged duration and manifests histologically by the presence of lymphocytes and macrophages and results in fibrosis and tissue necrosis. When inflammation continues for prolonged periods of time, it can be thought of as the healing process in overdrive, and deleterious changes can occur to localized tissues as well as the entire body.

In appreciating the inflammatory process, it is important to understand the role of chemical mediators. These are the substances that tend to direct the inflammatory response. These inflammatory mediators come from plasma proteins or cells including mast cells, platelets, neutrophils and monocytes/macrophages. They are triggered by bacterial products or host proteins. Chemical mediators bind to specific receptors on target cells and can increase vascular permeability and neutrophil chemotaxis, stimulate smooth muscle contraction, have direct enzymatic activity, induce pain or mediate oxidative damage. Most mediators are short-lived but cause harmful effects.[1] Examples of chemical mediators include vasoactive amines (histamine, serotonin), arachadonic acids (prostaglandins, leukotrienes) and cytokines (tumor necrosis factor and interleukin–1).

Inflammation and Oral Health

The inflammatory process significantly affects the periodontium. Plaque biofilm releases a variety of biologically active products as gram-positive and gram-negative bacteria colonize the tooth surface around the gingival margin and interproximal areas. These products include endotoxins, cytokines and protein toxins.[2] These molecules penetrate the gingival epithelium and initiate a host response that eventually results in gingivitis. Evidence of this can be seen clinically with changes in tissue color from pink to red, swelling, and bleeding upon probing.[3] Because gingivitis is typically not painful, it may remain untreated for years. Worse, it may be viewed by practitioners as something that requires less concern than periodontitis. Nevertheless, chronic gingivitis that persists for years may provide the basis for greater concern for systemic health than a periodontitis condition that is more readily treated.

As the biofilm continues to proliferate, soluble compounds penetrate the sulcular epithelium. This, in turn, signals the gingival epithelium to produce chemical mediators including interleukin–1 beta (IL-1,), prostaglandins, tumor necrosis factor alpha (TNF-α), and matrix metalloproteinases.[4] These products recruit neutrophils to the area and influence chemotaxis, and can cause increased permeability of gingival vessels that permits plasma proteins to emigrate from the blood vessels into the tissue. As the inflammatory process progresses, additional mediators are produced, and more cell types are recruited to the area including neutrophils, T-cells, and monocytes. Continued inflammation results in signaling of fibroblasts and production of proinflammatory cytokines in the tissues. Antibodies specific to oral bacteria circulate in the peripheral blood. The acute-phase response becomes activated and CRP, fibrinogen and complement are produced both by local cells and within the liver.[5,6] These proteins may further exacerbate

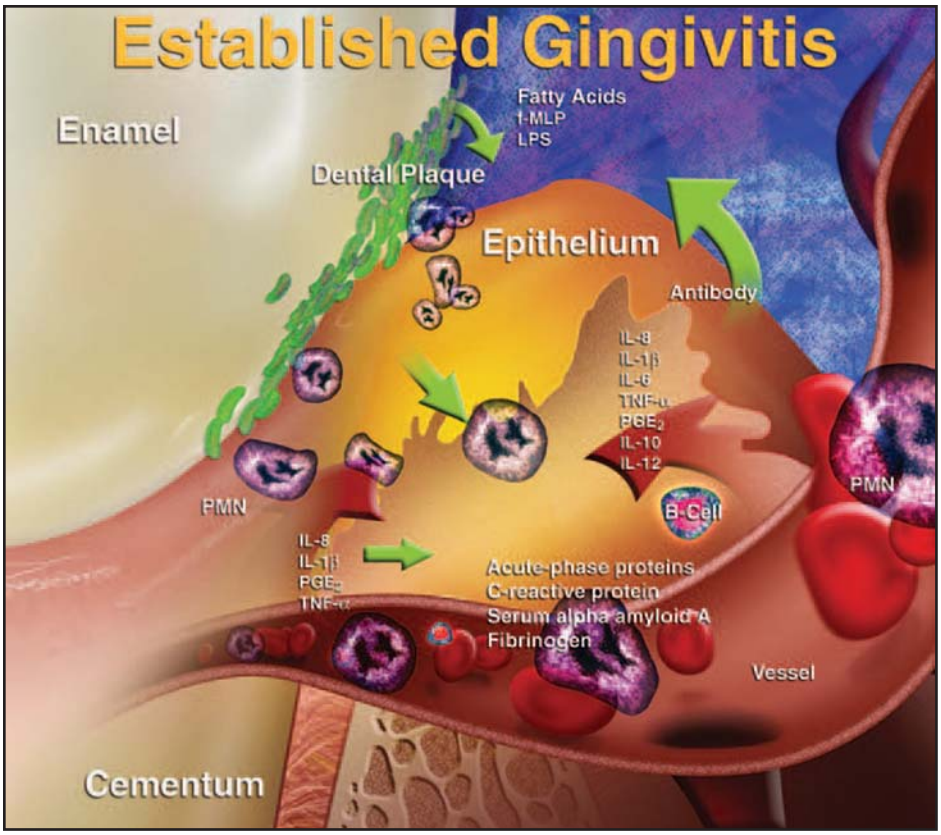

Figure 1: Mediators and cells present in established gingivitis

From: Scannapieco, FA: Periodontal inflammation: from gingivitis to systemic disease? Compend Cont Educ Dent. 2004; 25(7) (Suppl1): 16-24.

the local inflammatory response and may affect the initiation or progression of systemic disease (i.e., atherosclerosis).[7,8] This process of chronic gingivitis is represented in Figure 1.

It is important to note that even though an individual may have established or chronic gingivitis, the condition is still reversible. Thorough dental hygiene debridement and regular home oral hygiene care could return the gingival tissues to a state of health. In some individuals when the inflammatory process continues and expands, the collagen of the periodontal ligament breaks down and bone resorption occurs, thus resulting in periodontitis. Individuals with periodontitis have the same increased levels of proinflammatory mediators as those with chronic gingivitis, including CRP, fibrinogen, and IL-1, and 6. Fortunately, when periodontal treatment is performed and clinical inflammation decreases, the serum levels of these inflammatory mediators also decrease.[9]

The Oral-Systemic Relationship

Although periodontal diseases are well known as an oral problem, in the past decade, there has been a shift in perspective. Research has been focusing on the potential impact of periodontal diseases on systemic health. The relationship between periodontal inflammatory disease and systemic diseases such as cardiovascular disease, diabetes, respiratory disease and adverse pregnancy outcomes has been closely investigated. The basis for the biological mechanism of this relationship is beginning to emerge and further study may lead to an understanding of whether or not a true causal relationship exists.

Cardiovascular disease (CVD) is characterized by the build-up of inflammatory plaques that may cause thromboses and eventual myocardial infarction. Atherosclerosis is the term used for the thickening and hardening of the arteries that is produced by this plaque build-up. It represents a chronic inflammatory response that causes injury to the endothelium of elastic and muscular arterial tissue. One of the hallmarks of the early atherosclerotic lesion is the presence of neutrophils followed by monocytes and lymphocytes.[10] These leukocytes can affect the vascular endothelial lining and can cause oxidation of low-density lipoprotein (LDL) levels. Monocytes are induced to become macrophages, which take up modified lipoproteins and become lipid-laden "foam cells."[11] The local inflammation is sustained by secreting chemical mediators, and the atherosclerotic lesion begins to bulge within the luminal wall. As this lesion progresses, the extracellular matrix is degraded by proteolytic enzymes and becomes susceptible to rupture. Thromboses can occur, occluding blood flow to the heart, which may eventually lead to infarction.

Since atherosclerosis is considered to be inflammatory in nature, identifying inflammatory markers that correlate with disease state is beneficial. One of the most recognized and consistent markers of systemic inflammation and poor cardiovascular prognosis is the acute-phase protein CRP.[12,13] It is produced by the liver and released into the blood stream. It is positively correlated to IL-6, activates complement and accounts for LDL uptake by macrophages.[14-16]

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It has been proposed that bacteria or viruses may directly infect atherosclerotic lesions contributing to the inflammatory process. Further, distant infections may increase systemic inflammation through the release of toxins or the leakage of chemical mediators into the circulation.[17] It has been reported that studies of atheromatous lesions in the carotid arteries have found over 40% of atheromas contain antigens from periodontal pathogens including Porphyromonas gingivalis, Tannerella forsythensis, and Prevotella intermedia.[18] In addition, P. gingi-

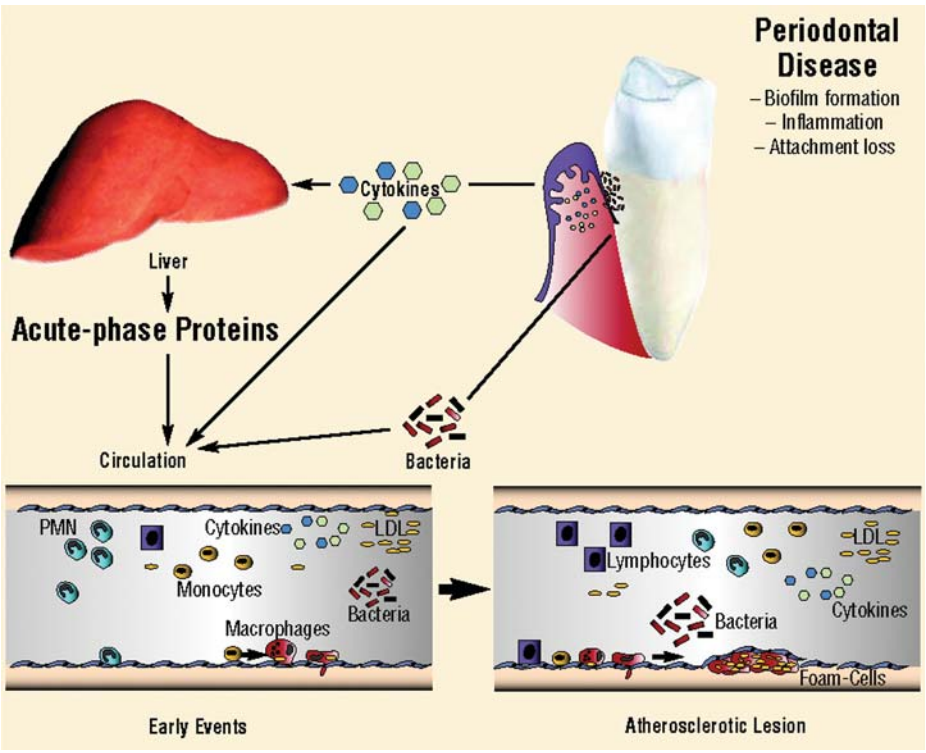

Figure 2: Connection between periodontal disease and atherosclerosis

From: Dave S, Batista EL Jr, Van Dyke TE: Cardiovascular disease and periodontal diseases: commonality and causation. Compend Cont Educ Dent. 2004; 25(7) (Suppl 1):26-37.

valis can induce platelet aggregation, a component of atheroma and thrombus formation.[19] This suggests a possible invasion of atheromas by oral pathogens as well as a possible contribution to their development. However, causality has yet to be established.

Animal model studies investigating the relationship between CVD and periodontal disease have demonstrated that clinically induced oral infection with P. gingivalis will increase atheroma size and elevate CRP levels.[20] More recently, a study of humans reported a positive independent association between carotid intima-media thickness (IMT) and bacterial burden including P. gingivalis, Actinobacillus actinomycetemcomitans, Treponema denticola, and Tannerella forsythensis.[21] Figure 2 represents the proposed connection between periodontal disease and atherosclerosis.

It is also thought that an autoimmune response may be involved in the development of atherosclerosis. Most humans have immune reactions against microbial heat-shock protein 60 (HSP60). Antibodies against bacterial versions of this protein may cross-react with human HSP60, causing an autoimmune response and stimulating athersosclerosis.[4]

Diabetes mellitus is another systemic condition with oral inflammatory connections. One of the major complications of diabetes is periodontitis.[22] While diabetes increases the probability of developing periodontal disease[22-24], periodontitis also increases the risk of poor glycemic control in people with diabetes when compared to those individuals with diabetes without periodontitis.[25] Fortunately, periodontal treatment can improve glycemic control by reducing the bacterial burden and the inflammatory response.[26-28]

There are several biological mechanisms proposed to explain the increased incidence and severity of periodontal disease in individuals with diabetes. Diabetes tends to increase susceptibility to infection—including oral infection—and the disease itself decreases the effectiveness of cells that kill bacteria.

Another explanation is that inflammation is enhanced in those with diabetes. Research has demonstrated elevated levels of inflammatory mediators in the gingival crevicular fluid of periodontal pockets of poorly controlled patients with diabetes as compared to those without diabetes or those with diabetes who are well controlled. These patients had significant periodontal destruction with an equivalent bacterial challenge.[24,29,30] In particular, the proinflammatory cytokine, TNF-α, plays a major role in this process. TNF-α has a significant role in insulin resistance, the primary cause of type 2 diabetes. It is produced in large quantities by fat cells. Periodontitis has also been associated with increased levels of TNF-α. Elevated levels of TNF-α may lead to greater bone loss by killing cells that repair damaged connective tissue or bone and may exacerbate insulin resistance and worsen glycemic control.[31-33]

It has also been hypothesized that diabetes interferes with the capacity to form new bone after periodontal diseases have caused bone resorption. Graves, et al., studied genetically diabetic mice with type 2 diabetes and nondiabetic littermates by injecting them with P. gingivalis. The death of osteoblasts was measured, and results indicated that there was a higher and more prolonged rate of osteoblast cell death in the diabetic group. It was concluded that the capacity to repair a bony defect by producing new bone would be severely limited when osteoblasts died prematurely. Yet further study is needed in this area to refine this concept.[34]

As with CVD and diabetes mellitus, there is a relationship between oral infection and respiratory disease. In particular, chronic obstructive pulmonary disease (COPD) and pneumonia have been associated with poor oral health.[35-38] It is likely that oral biofilm serves as a reservoir of infection for respiratory bacteria. Specifically, Pseudomonas aeruginosa, Staphylococcus aureus, and enteric bacteria that has been shown to colonize the teeth of patients admitted to hospitals or long-term care facilities. These bacteria may be released into saliva and then aspirated into the lower airway causing infection.[4] Another vehicle by which bacteria from the oral cavity can be introduced into the respiratory system is intubation.

Inflammatory mediators, such as cytokines produced by the periodontium, may be another mechanism by which respiratory disease are associated with oral health. These mediators present in inflamed gingival tissues enter the gingival crevicular fluid and then the saliva. Once aspirated, these mediators can have proinflammatory effects in the lower airway.

Further, studies have demonstrated that periodontal diseases have been shown to increase the risk of adverse pregnancy outcomes such as premature birth and low birth weight.[39-41] Uterine contractions are stimulated by oxytocin, which is produced by the hypothalamus, and by prostaglandins produced by the placenta. This process normally occurs in the third trimester and leads to birth. However, chronic infection can stimulate the inflammatory process, which leads to elevated amniotic levels of prostaglandins, TNF-α, and IL-1 and IL-6. These mediators then lead to premature rupture of membranes and preterm labor. Other work has suggested that periodontal pathogens may travel from the gingival sulcus to the placenta and stimulate preterm birth. Specifically, Han and colleagues found that periodontal bacteria, including Fusobacterium nucleatum, entered the bloodstream from the oral cavity and directly affected the birth process.[42] A summary of the biological mechanisms by which gingival inflammation effects systemic health appears in Figure 3.

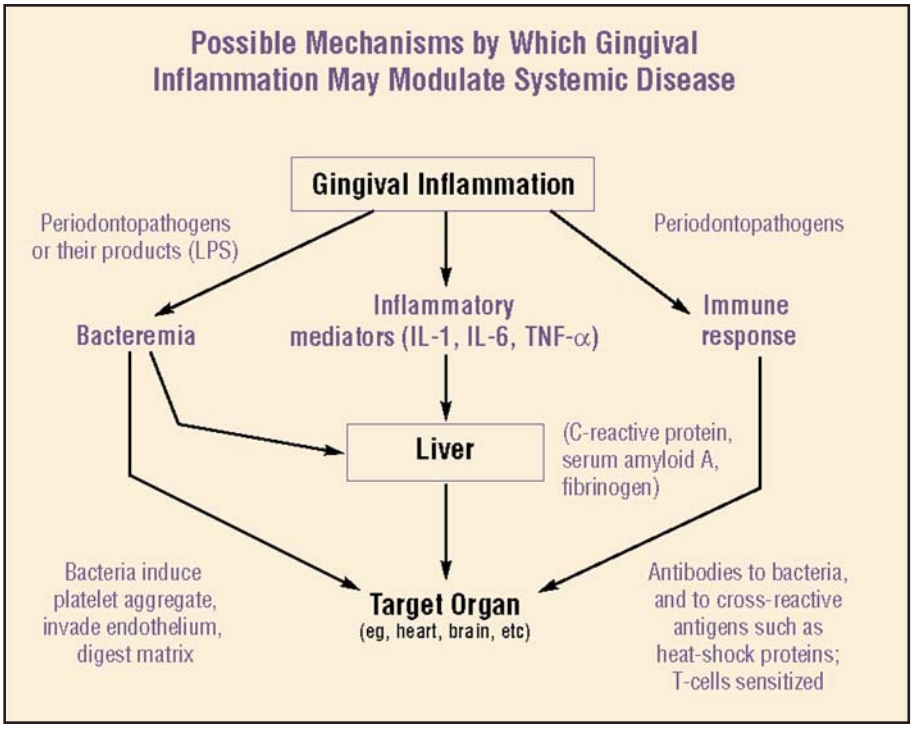

Figure 3: Model to explain oral inflammation-systemic disease association

From: Scannapieco, FA: Periodontal inflammation: from gingivitis to systemic disease? Compend Cont Educ Dent. 2004; 25(7) (Suppl1): 16-24.

Translating Science to Practice

As often occurs, research provides many answers and drives more questions. Despite the knowledge obtained through science, it is incumbent upon clinical practitioners to translate the evidence into practical use. What does the above information mean for clinical practice? Does it offer opportunities for changing dental hygiene interventions? How can we use this information to answer patients' questions when they inquire about CRP levels, or ask if their periodontal condition will give them heart disease? Are we better prepared to address their question when they say they read two conflicting articles about oral and systemic health in the same issue of Reader's Digest, Prevention magazine, or Every Woman and wonder which one is accurate?

Understanding the association between oral health and systemic health does provide opportunities for oral hygiene clinicians to reframe their protocols. The process begins by practicing oral medicine. First, comprehensive medical assessment is needed for each patient. A review of systems and vital signs evaluation should be part of that assessment process. During these assessments, identifying risk factors for specific systemic diseases is important, including age (over 40 years), hypertension, dyslipidemia, smoking, obesity/overweight, CVD, diabetes or symptoms of diabetes and women who are pregnant and have poor oral hygiene.

Comprehensive oral assessment is equally important. This may include thorough head and neck examination, radiographs (if clinically indicated), periodontal probing, bacterial monitoring of periodontal and carious pathogens, genetic testing for periodontal disease or other diagnostic tools that seem appropriate to each individual need.

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As these assessments are being performed, risk factors for oral and systemic diseases are being noted and explained to the patient. It is essential that the patient understand that the purpose of these assessments is to prevent problems from occurring or treat them as readily as possible. Patients are well acquainted with the idea that physicians prefer to treat a stroke or a heart attack before it occurs by identifying possible risk factors and trying to reduce them. They are used to attending a medical appointment and having certain assessments performed. They are even used to requesting certain tests or procedures based on their own education and experience. Thus, it is time

that we incorporate these approaches into dental and dental hygiene practice. Our patients need to become accustomed to the same comprehensive assessment process. Clinicians can then put together a picture for the patient that incorporates oral and systemic risk factor findings, and discuss how their chronic gingivitis or periodontitis condition may place them at risk for CVD, poor glycemic control or an adverse pregnancy outcome.

Once risks have been identified, those that can be modified are incorporated into the dental hygiene treatment plan and patient education process. Just as a physician will recommend a patient lose weight or prescribe an antihypertensive agent, the dental hygiene therapist may make recommendations for the patient to begin a smoking cessation program, use specific preventive oral care products, monitor their blood sugar regularly or complete a nutrition counseling program, in addition to having debridement of plaque biofilm and calculus. Certainly, patients will view the dental hygiene appointment as more than a "cleaning" if greater emphasis is placed on the patient's total health, risk factor assessment and risk factor modification.

In addition, once risk factors have been identified and appropriate treatment planned, it is important to be prepared to answer questions about medications and products that have anti-inflammatory and/or antibacterial properties. For example, in the past decade, several engineered therapeutic proteins and antibodies have been generated and are either currently in use or in the late stages of clinical trials. Patients may be familiar with:

Just as a physician will recommend a patient lose weight or prescribe an antihypertensive agent, the dental hygiene therapist may make recommendations for the patient to begin a smoking cessation program, use specific preventive oral care products, monitor their blood sugar regularly or complete a nutrition counseling program, in addition to having debridement of plaque biofilm and calculus.

- Etanercept (Embrel®), which binds TNF-α and prevents it from engaging its inflammatory functions
- Recombinant Protein C, which helps the body dissolve small clots triggered during inflammation
- Infliximab (Remicade), a monoclonal antibody that binds to TNF-α, and has been used to treat autoimmune inflammatory diseases such as rheumatoid arthritis and Crohn's disease

While these drugs are being used to treat systemic diseases, it is possible that they could be used to treat inflammation related to gingivitis and periodontal disease. Other engineered proteins under development may also be used to treat these oral infections.[43]

Another anti-inflammatory medication that has been shown to be effective for the treatment of periodontitis is low-dose doxycycline hyclate (Periostat). Periostat inhibits the collagenase activity by neutrophils, thus preventing the degradation of connective tissue and bone loss. Therefore, it is beneficial as part of host modulation therapy. It is administered twice daily at a dosage of 20 mg. Periostat is an antibiotic; however, the dose is too low to produce antibacterial effects. Studies have demonstrated that Periostat improves the effectiveness of routine scaling and root planing and that the progression of periodontitis is decreased.[43]

Optimal preventive education programs should include discussion of twice-daily brushing, flossing and use of a chemotherapeutic mouth rinse to reduce bacterial plaque and susceptibility to gingivitis.[44] Products recommended should be those that have been well-researched and demonstrated safety and efficacy. For example, Peridex® and Listerine® Antiseptic Mouthrinse are the only two chemotherapeutic mouth rinses that have been approved by the American Dental Association Council on Scientific Affairs. Their effectiveness has been well established. Similarly, a dentifrice containing triclosan/copolymer (Colgate Total® Toothpaste) has been shown to be effective in reducing plaque and gingivitis, controlling bacterial infection and preventing or slowing the progression of periodontal disease.[45] In addition, triclosan has been shown to possess potent anti-inflammatory properties. In vitro studies have demonstrated that triclosan has inhibited IL-1 stimulated prostaglandin production in human gingival fibroblast cells, inhibited the production of IL-1 by fibroblasts stimulated with TNF-α and has inhibited the production of collagenases by human bone cells and fibroblasts stimulated with IL-1 and TNF-α.[46,47] The antibacterial and anti-inflammatory properties of triclosan are reasons to recommend Colgate Total® toothpaste both for patients with periodontal diseases as well as for those whose systemic health has been compromised.

Conclusion

Research suggests that there is an interrelationship between oral infection, inflammation and systemic health. Patients, dental hygienists, dentists, dental specialists and other health care providers should be aware of the consistent relationships between oral inflammation and systemic diseases. They should value the need to modify assessment, prevention, and treatment protocols to improve the oral health as well as total health of the patients they treat in the office each day.

Take Advantage of the Opportunity to Earn CE Points

Based on the information presented in *Inflammation: The Relationship Between Oral Health and Systemic Disease*, you can earn 2 CE points by visiting www.adha.org and selecting "Continuing Ed" on the left-hand navigational bar. Then simply click on ADHA Continuing Education Courses and select this course.

A Case in Point

Mrs. White, a 45-year-old Hispanic female, presents to your practice for an initial dental hygiene appointment. She is new to the area, but reports that she faithfully had dental and dental hygiene care every six months. Mrs. White's medical history is significant for the following:

- Myopia for which she wears corrective lenses
- Borderline hypertension—no medications prescribed
- Prediabetes—diet and exercise recommendations made by nurse practitioner
- Overweight—diet and exercise recommendations made by nurse practitioner, advised to lose 20 pounds
- Smokes 1/2 pack of cigarettes daily for over 20 years

Oral history is significant for the following:

- Generalized gingivitis with moderate plaque present on lingual surfaces of mandibular premolar and molar teeth and supragingival calculus and plaque noted on the lingual surfaces of mandibular incisors
- No evidence of current or recurrent decay, previous history of decay with occlusal restorations present on all first molars, and a crown on tooth #31
- Presence of nicotine stomatitis

Mrs. White reports that she has been advised previously to quit smoking and has attempted to do so on three occasions without success. She states that she was told that she had gingivitis by her previous dentist and dental hygienist, but that it was not serious and that she should brush and floss more. Mrs. White admits that she does not floss regularly, but brushes twice daily with a manual toothbrush.

Given this information about Mrs. White, take a moment and imagine her sitting in the operatory. She is ready for her appointment. What is your next step? Do you need more information or are you ready to proceed with treatment? Have you mentally picked up your curet eagerly anticipating removing the debris from the mandibular region? If your answer is "Yes, let's get started," read this paper again. Mrs. White does not need the plaque and calculus removed yet nearly as much as she needs to know about her risk factors for oral and systemic health. Mrs. White needs you to take time out to review your findings from assessments and speak frankly with her about her health status. This is the perfect moment to discuss symptoms of diabetes that Mrs. White may not realize she has, to educate her about the links between smoking, hypertension, diabetes and CVD. Mrs. White is 45, overweight and Hispanic, placing her at greater risk for converting from prediabetes to diabetes. Nevertheless, with some effort, she can avoid that step through a concerted effort of diet and exercise. She may not realize that a modest weight loss will benefit her greatly in terms of improved general health. In addition, now is when you can begin discussing the link between her oral health and general health. The presence of chronic gingivitis coupled with prediabetes and borderline hypertension places Mrs.White at risk for further health issues. Also, she presents with nicotine stomatitis, another reason to incorporate smoking cessation as part of your education discussion and treatment plan. Mrs. White has known she has gingivitis, admits she does not floss regularly, but does brush daily. What recommendations would you make to help improve her oral home care regimen? Would you switch her to a powered toothbrush, have her use a mouth rinse, recommend Colage Total® toothpaste? How often would you want to see Mrs. White for follow-up?

In the course of reviewing this information, it is possible to see how the traditional dental hygiene appointment can be reframed. That 45-minute "cleaning" just does not fit the profile of needs for Mrs. White.

In the course of reviewing this information, it is possible to see how the traditional dental hygiene appointment can be reframed. That 45-minute "cleaning" just does not fit the profile of needs for Mrs. White. She deserves a schedule that allows for assessment and education, treatment and education, and re-evaluation and education. Is all this necessary for a simple case of gingivitis? Perhaps the real question we should be asking is, do we ever see simple cases of gingivitis? What have we been missing by not allowing adequate time to perform comprehensive assessment and risk factor analysis?

This case and the questions posed provide the dental hygiene reader an opportunity to reflect on the prospect of incorporating oral medicine into dental hygiene practice. Continually reviewing the literature related to oral and systemic health – and discerning relevant components – will enable dental hygienists to refine practice and continue to provide quality care to their patients.

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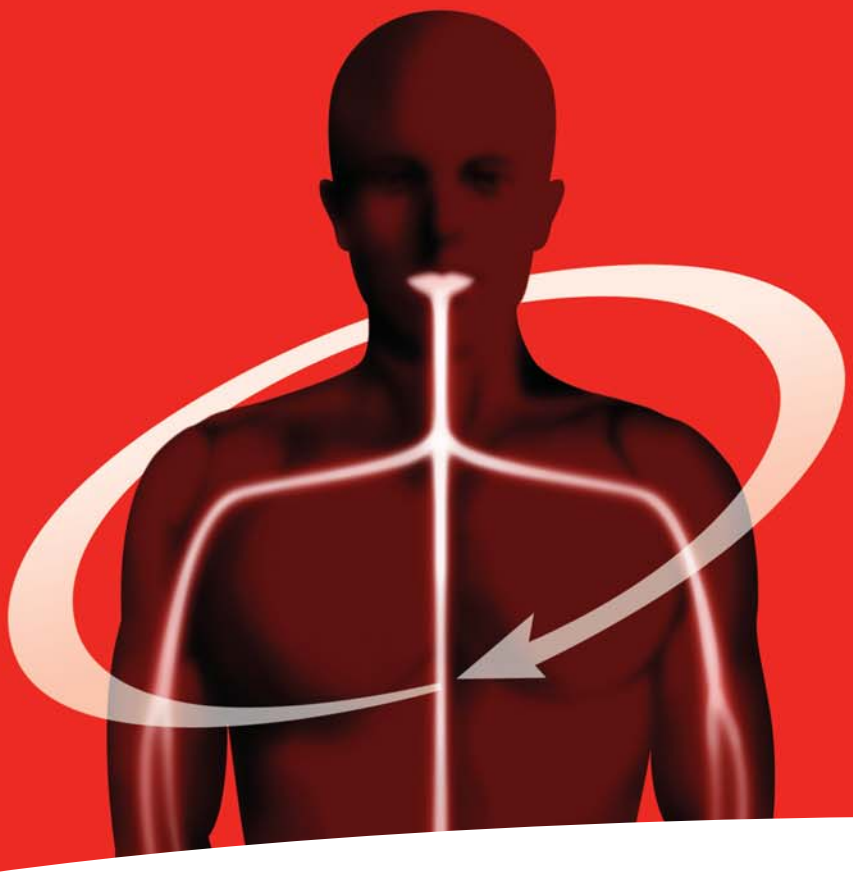

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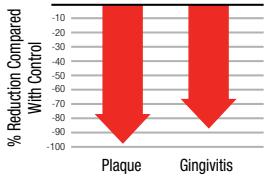

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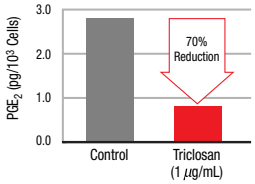

"Recent evidence suggests a strong relationship between periodontal inflammatory disease and systemic diseases such as cardiovascular disease. It is now generally accepted that inflammation plays an important role…"[7]
—Sheilesh, et al. *Compendium.* 2004.

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*vs ordinary fluoride toothpaste. †in vitro.

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