

The Oral-Systemic Science: Emerging Evidence of a Strong Relationship

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The concept that there is a connection between periodontal disease and systemic health is not new. Dr. Willoughby Miller, in 1891, recognized and outlined the concept in a book called *The Dental Cosmos*, describing the mouth as a “focus of infection where microorganisms or their waste products obtain entrance to parts of the body adjacent to or remote from the mouth”. As oral-systemic science continues to develop, it will have a dramatic impact on the practice of dentistry.

Current estimates indicate that 75% of the North American population has some form of periodontal disease.¹ Mild to moderate forms of periodontal disease manifest with symptoms that are non-specific or even asymptomatic. Patients with periodontal disease often experience pain only when there are complications such as abscesses, alveolar bone breakdown or oral mucosal lesions. Without complications, patients experience no systemic signs of infection such as fever.² Any bleeding during scaling and root planing is attributed to instrumentation. Given these factors, there has been a certain amount of complacency in the treatment of periodontal disease as a serious infection by both, the patient and the clinician.

Studies show that untreated periodontal disease generates a destructive immune response that elevates systemic inflammation and that gingivitis may

be as damaging as periodontitis. Accumulated bacteria on the surfaces of the teeth in the form of a biofilm are responsible for the initiation and progression of periodontal disease. When biofilm is left undisturbed, a group of virulent anaerobic species emerge that activate the host immunoinflammatory processes. Although these bacteria initiate periodontitis, it appears that host modifying risk factors contribute to the severity and extent of the disease.²

Emerging evidence supports the relationship between periodontal disease and conditions such as cardiovascular disease, adverse pregnancy outcomes, diabetes, pneumonia, rheumatoid arthritis, osteoporosis and Alzheimer’s disease. (Figure 1)

PERIODONTAL DISEASE AND CARDIOVASCULAR DISEASE

Editors of the American Journal of Cardiology and the Journal of Periodontology simultaneously published a paper in 2009 making extensive clinical recommendations to both the dental and medical profession based on a number of studies including two separate systematic reviews and meta-analysis published in journals directed to the medical profession (Figure 2). The analysis of data concluded that periodontal disease is a risk factor for cardiovascular disease (CVD) independent of traditional risk factors. Perhaps the most significant recommendation is that patients with moder-

ate to severe periodontal disease should be informed that there may be an increased risk for atherosclerotic CVD associated with periodontitis.²

Another equally important recommendation was that for those patients with atherosclerotic CVD and no previous diagnosis of periodontitis, periodontal evaluation should include a comprehensive examination of the periodontal tissues with measurements of pocket depth, bleeding on probing and radiographic assessment of bone loss. These patients should be treated with a focus on reducing and controlling bacterial accumulations and eliminating inflammation.²

Further, when periodontal disease is newly diagnosed in patients with atherosclerotic CVD, dentists and physicians should closely collaborate to optimize CVD risk reduction and periodontal care.²

Other research³ has identified that periodontal disease in young individuals may be more of a risk indicator or risk factor for CVD than periodontal disease in older individuals. Patients who have pockets 4mm or greater in 15% of the teeth are more prone to have a heart attack than those whose tissues are healthy. Further, anaerobic infection was almost 4 times higher in those individuals who were hospitalized for cardiovascular disease than healthy individuals with no history of CVD.³

A simple test called the Papillary Bleeding Scale (Figure 3) can be used to assess the degree of inflammation present. This test consists of the insertion of a Stim-u-dent or Soft Pick between the teeth and then removing it. The amount of bleeding is graded using the scale of 0-5. With this test, the patient can clearly see that there is bleeding and would not attribute it to the “probing” by the clinician.

PERIODONTAL DISEASE AND LOW BIRTH WEIGHT OR EARLY DELIVERY BIRTHS

In February, 2010, the National Institute of Health published an announcement saying that premature birth affects 13 million infants world wide each year and that 1 out of 3 preterm infants is born to a mother who has a silent infection of the amniotic fluid. The findings support preterm labor as an immune response to infection or injury and substantial evidence indicates that inflammatory hormones, specifically Interleuken 1 and 6 may play a significant role in the labor process.⁴ The Lopez⁵ and Jeffcoat⁶ studies demonstrated that for the group of women who had scaling before delivery there was a substantial reduction in the rate of early delivery as compared to those women who had their teeth scaled after delivery. Other studies have also linked low birth weight babies and early delivery to levels of prostaglandin, an inflammatory response chemical.

PERIODONTAL DISEASE AND DIABETES MELLITUS

Although the relationship between periodontal disease, inflammation and overall health has been suspected, new studies are providing more comprehensive evidence for this link. We recognize that diabetes predisposes oral tissues to greater periodontal destruction but several

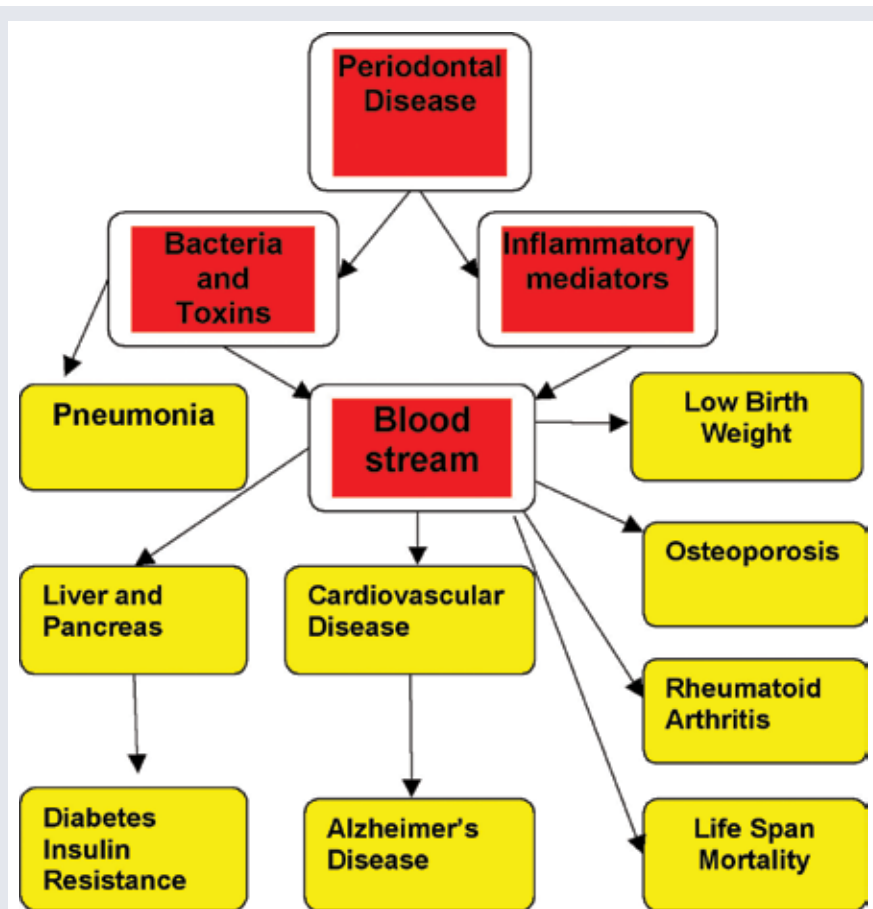


FIGURE 1—The Oral-Systemic Link.

studies have now identified that periodontal disease leads to poor glycemic control. Recent studies have presented evidence of a bidirectional adverse relationship between periodontal disease and diabetes mellitus, both type I and type II. Although diabetes is a metabolic disorder and periodontitis is an infectious disease, the relationship occurs through the ability of both conditions to induce an inflammatory response leading to the production of inflammatory mediators. These proinflammatory cytokines such as Interleukin-6 impair the glucose-stimulated release of insulin from the pancreas. Periodontal therapy, on the other hand, can stabilize glycemic control and reduce complications from unstable blood sugar levels.^{7,8}

In a U.S. population-based study of 12,367 non-diabetic indi-

viduals, it was found that there was an association of periodontal disease with body mass index (BMI). Approximately a 40% to 50% increase in the risk for periodontal disease was found in those with obesity. Studies show that fatty tissue produces pro-inflammatory mediators which lead to systemic inflammation. The result is a hyper-inflammatory state that can exaggerate the response to periodontal infection.^{7,8}

PERIODONTAL DISEASE AND PNEUMONIA

Health care associated (nosocomial or hospital acquired) pneumonia is a common cause of death in elderly patients. This pneumonia is caused by species that do not often colonize the oropharynx. The oral cavity has been suggested as an important reservoir for these respiratory pathogens and there is conclusive data that simple pre-

PATIENT	DENTIST	PHYSICIAN
1. Patient with moderate to severe periodontal disease	Inform patient that there may be an increased risk for CVD. If patient has two other risk factors refer for medical evaluation	If patient has other risk factors, (smoking, family history) consider medical evaluation.
2. Medical evaluation of patient with periodontitis		Include assessment of atherosclerotic CVD risk, blood pressure, blood lipid profile and (optional) plasma hsCRP
3. Risk factor treatment	Inform patient about smoking cessation and life style changes	Inform patients about smoking cessation, life style changes and provide pharmacologic therapy where appropriate
4. Patient with CVD and previous diagnosis of periodontal disease	Optimize periodontal care and CVD risk reduction.	Optimize CVD risk reduction and collaborate with dentist on periodontal care
5. Patient with CVD and no previous diagnosis of periodontal disease	a. Perform comprehensive periodontal evaluation, especially if some signs exist. b. If untreated or uncontrolled periodontal disease is present, reduce bacterial accumulations and eliminate inflammation.	Collaborate with dentist. Optimize CVD risk reduction and periodontal care
6. Patient with CVD and newly diagnosed periodontal disease	Collaborate with physician. Optimize CVD risk reduction and periodontal care	Collaborate with dentist. Optimize CVD risk reduction and periodontal care

FIGURE 2—Clinical Recommendations for Patients with Periodontitis.

ventive oral care such as helping the patient cleanse the mouth reduces mortality from pneumonia.⁹

PERIODONTAL DISEASE AND RHEUMATOID ARTHRITIS

Periodontal disease has also been associated with rheumatoid arthritis (RA), an autoimmune disease that inflames joints and causes destruction of cartilage, bone and ligaments. The two diseases share some basic characteristics: both diseased gingival tissues and joints affected by RA produce similar cytokines and growth factors. These chemicals promote the dissolution of bone, a problem shared by both diseases. This suggests the presence of a common underlying inflammatory mechanism. Levels of anti-CCP antibodies (anti-cyclic citrullinated peptide antibody) are considerably higher in RA patients with periodontal disease, suggesting that periodontitis may be a contributing factor in the pathogenesis of RA. Coincidentally, *P.gingivalis* produces an enzyme that induces citrullination of vari-

ous autoantigens. Patients with RA are likely to benefit from increased periodontal care.^{10,11}

PERIODONTAL DISEASE AND OSTEOPOROSIS

Bone loss is a condition shared between periodontal disease and osteoporosis. In the early stage, called osteopenia, there is a reduction in bone mass due to an imbalance between bone formation and bone resorption. Osteoporosis occurs as bone resorption becomes more prevalent and there is considerable demineralization. The common factor between osteoporosis and periodontal disease is the excessive osteoclastic activity and bone loss initiated through chronic inflammatory conditions. This shared chronic inflammatory response may predispose individuals with periodontitis to osteoporosis. Further, risk factors such as age, smoking and estrogen deficiency are the same for both, periodontal disease and osteoporosis.

Estrogen modulates cytokines that regulate bone metabolism

and the host inflammatory response. Lack of estrogen increases the number of osteoclasts causing an imbalance in bone metabolism and a reduction in bone density. Periodontitis also activates the inflammatory response and the osteoclasts. Many investigations have found significant correlation between periodontal disease and estrogen deficiency. These two risk factors, working together, can induce osteoporosis.⁷

PERIODONTAL DISEASE AND ALZHEIMER'S DISEASE

There is evidence that periodontal disease may be a risk factor for dementia through the bacterial and viral infections commonly found in periodontal disease. Oral infection can either directly or through systemic signals to the brain contribute to the development of Alzheimer's disease. Periodontal infections may result in elevating the systemic inflammatory response which in turn may contribute to existing brain and vascular pathologies that would impact brain function. Timely treatment of

periodontal infections that reduces oral pathogens would also reduce the risk of systemic infection and inflammation and would be most important to those individuals susceptible to infection and hyperinflammation.¹²

IS PERIODONTAL DISEASE A RISK MARKER FOR MORTALITY?

A study based in Sweden addressed the issue of periodontal disease as a risk marker for mortality. This study evaluated the relationship between periodontitis and premature death 16 years after the diagnosis of periodontitis. Results confirmed the hypothesis that periodontitis in young adults with any missing molars is a risk marker for premature death. The prematurely deceased women in the study were expected to live 36.1 years longer and the deceased men 31.6 years longer. The individuals who died were probably infected with periodontitis many years before the baseline registrations. Young individuals with periodontitis and missing molars seem to be at increased risk for premature death by life-threatening diseases, such as neoplasms, and diseases of the circulatory and digestive systems.¹³

Studies show conclusively that the mouth can be a source of chronic but silent infection and that dental cleanings may not eliminate the source of the infection. Research indicates that patients now rely on the dental team not just for their oral health but for their overall health.¹⁴ Given this responsibility, treatment of periodontal disease requires a paradigm shift. Inclusion of a microbiological assessment of oral biofilm from the tongue surface as well as the teeth will enable the clinician to screen for the presence and location of oral pathogens. Periodic sampling provides an avenue to monitor shifts in the biofilm and gives the dental team advance warning of infec-



P B SCALE

CATEGORIES	
Tissue is perfect	0
Tissue is not perfect	1
Spotting only	2
Bleeding Confined to fossa	3
Bleeding Spreads to other teeth	4
Spontaneous Bleeding	5

FIGURE 3—Papillary Bleeding Scale.

tion. The treatment of infection, systemic or oral, is complex and may require the short term use of antibiotics as well as other modalities. By subscribing to these changes, the practitioner is well underway to building the health care model of the future, where collaborative teams from dentistry and medicine, nutrition and pharmacy work together to assess and manage periodontitis and systemic diseases and reinforce health and wellness awareness to the public.²

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Anne Bosy is the creator of the Oravital System. Formerly a professor at George Brown College and Regency Dental Hygiene Academy, she is currently the Senior Vice President and founding partner of Oravital Inc. Oral Health welcomes this original article

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