Original Article

PennWell

GRAND ROUNDS
in Oral-Systemic Medicine™

Dental Economics RDH

MAINTAINING ORAL HEALTH IN THE AGING POPULATION: THE IMPORTANCE OF THE PERIODONTAL-SYSTEMIC CONNECTION IN THE ELDERLY

Anthony M. Iacopino, DMD, PhD†

Abstract
The rapid growth of the elderly population is having a dramatic impact on the practice of dentistry. In the future, dental professionals will need to collaborate with healthcare management teams as the community practice environment moves to a transdisciplinary model of care. That model will likely be based in part on what is known as the periodontal-systemic connection. Such a connection between systemic disease and periodontal health predicts that maintaining oral health through midlife should reduce healthcare expenditures for an individual's remaining lifespan. Ample evidence supports such a periodontal-systemic relationship, which is described in detail in this article, and justifies patient management approaches that include comprehensive transdisciplinary care. This approach is particularly relevant to elderly patients susceptible to systemic diseases or development of co-morbid conditions if periodontal health is neglected. Future comprehensive care models must include dental, medical, and other health professionals aware of the periodontal-systemic connection to manage periodontitis and systemic disease. Dental professionals should stress the importance of regular periodontal maintenance to patients and other health professionals. Preventive periodontal management will pay huge dividends as the population ages with regard to reducing potential systemic complications, especially those associated with chronic inflammation.

Citation: Iacopino A. Maintaining oral health in the aging population: the importance of the periodontal-systemic connection in the elderly. *Grand Rounds Oral-Sys Med.* 2006;3:25-37. (Digital version *Grand Rounds Oral-Sys Med.* 2006;3:25-37e.)

*(A complimentary copy of this article may be downloaded at **www.thesystemiclink.com**.)*

Key Words: Aging, periodontal disease, chronic inflammatory diseases, nutrition, masticatory function

Introduction

The number of elderly Americans surpassed 35 million in 2000, and by 2030 twenty percent of Americans will be 65 or older.[1] Rapid growth of this population will dramatically impact dentistry, since in this era, increased numbers of older adults retain their teeth.[2,3] Additionally, ethnic/cultural minorities account for a larger percentage of the elderly population, and individuals aged 85 and older are the fastest growing segment of the elderly population.[1] Thus, the diversity and number of frail/functionally-dependent elderly persons requiring care in nontraditional or institutionalized settings is increasing. The landmark 2000 report on oral health by the US Surgeon General documented profound disparities in the oral health status of older Americans.[4] Over 25% of people aged 65-74 have severe periodontal disease, and at any given time 5% of Americans aged 65 and older (currently approximately 1.7 million people) live in a long-term care facility where dental care is inadequate. In 2003, Oral Health America released its national grading report emphasizing that the oral health of older Americans is in a "state of decay".[5] Every state received failing or near failing grades in all categories of dental services for older adults, especially preventive and periodontal care.[6,7]

Adequate numbers of properly trained dental professionals are needed to ensure quality oral health care for older adults. These professionals must play a greater collaborative role with healthcare management teams as the community practice environment moves to a transdisciplinary care model.[2] For the dental profession to participate in this dynamic practice environment, practitioners must be prepared to address specific geriatric issues related to access,

† Associate Dean for Research and Graduate Studies; Professor, General Dental Sciences; Director, Wisconsin Geriatric Education Center, Marquette University.

risk assessment, prevention, cultural diversity, confounding medical conditions, and psychosocial influences, in addition to primary dental care.[8,9] Access is particularly important as low-income, minority, and institutionalized/homebound elderly are at high risk for oral disease but are the least likely to receive services.[10] A recent study by the Centers for Disease Control (CDC) reported that only 15% of these elderly had satisfactory oral health.[11]

The current state of geriatric dental expertise in the US is woefully inadequate.[8,12,13] Clinicians competent in geriatric issues are not being trained in numbers sufficient enough to meet projected workforce needs.[10] A recent report by the Institute of Medicine (IOM) criticized the lack of geriatric dental expertise, stating that "linkages between dentistry and medicine are insufficient to prepare practitioners for patients with complex medical problems".[14] Additionally, it noted that "the traditional isolation of dentistry from the rest of the healthcare team has been detrimental to the profession".[14] Transdisciplinary practice is a vital component of geriatric dental expertise because it exposes dental professionals to contributions of other health disciplines and fosters communication.[8,10] Other healthcare professionals must also develop appreciation for the abilities of dental personnel to manage oral health needs of the elderly. Such a transdisciplinary team management approach improves the prognosis of the elderly. In fact, studies demonstrate that elderly individuals whose comprehensive management includes dental care develop fewer co-morbid conditions and require less expenditure of healthcare dollars.[10]

The transdisciplinary concept has long been supported by the Health Resource Services Administration – Bureau of Health Professions (HRSA-BHPr), as noted in their white papers for health professions education.[15] Yet, inclusion of other healthcare disciplines in geriatric dental practice or oral health education for non-dental health professions is inadequate and lacks uniformity.[10,15] Dental professionals must be able to provide education and training for other healthcare practitioners and caregivers regarding 1) the relationship of oral health to general health, 2) the importance of daily oral care, 3) the means to provide basic oral care, and 4) the referral to a dental professional when necessary.[8,11,12]

Achieving an optimal level of transdisciplinary practice required for the elderly requires cooperation of like-minded healthcare professionals in community-based study clubs emphasizing transdisciplinary approaches. Involving dental professionals with these groups would provide comprehensive exposure to attitudes and skills required to provide dental care to older adults. As patients become disabled or experience cognitive dysfunction, dental teams will also need to address ethical issues relevant to dental diagnosis, treatment planning, and how care is provided.[8,11,12] In geriatric treatment planning, the focus should be on identifying an optimal level of care for the patient (ranging from none to very extensive). In other words, optimal care should not by definition be highest level technically possible. Rather, it should be to establish a level of care appropriate to maintain oral and general health. Other issues that should be considered within this framework are alternative treatment procedures or techniques, expanding the oral care team to include other health professionals or paraprofessionals, and the potential interactions of oral disease with systemic conditions in the elderly.

The field of geriatric practice will evolve as research is applied to patient care. Thus, dental professionals will need lifelong education in using health technology/informatics resources to update their skills.[16,17] Currently dental professionals need to be better acquainted with diagnosis/management of complex transdisciplinary cases and function more like oral physicians.[18] The shift toward prevention-oriented models must continue so that the traditional "repair" approach to dental care is transformed to one of early diagnosis and prevention. For the elderly population, professionals must familiarize themselves with evidence-based decision making and understand transdisciplinary management of complex cases in addition to acquiring expertise in restorative procedures and fabrication of dental appliances.[8,12,18] The most accessible and comprehensive transdisciplinary Web-based resource providing guidelines relevant to oral health in the elderly is currently available through the Wisconsin Geriatric Education Center and Marquette University School of Dentistry.[19]

Importance of the Periodontal-Systemic Connection in the Elderly

Recently the concept of a periodontal-systemic connection linking periodontal disease to systemic effects has emerged.[20] The implications of this connection, however, for the aging population have not been considered in depth.[21] In terms of public health impact, there is no doubt that poor oral health negatively impacts general health, particularly in the elderly.[22] Most chronic inflammatory diseases and conditions, including periodontal disease, are cumulative and thus manifested later in life. Decades of oral neglect contribute to additional health problems in the elderly population, which already consumes the majority of healthcare dollars nationwide, an outcome that places a significant burden on the healthcare system.[10,11] Given that periodontal disease has already been linked to development or exacerbation of systemic disease, maintaining optimal periodontal health in midlife may do more to reduce healthcare expenditures in one's remaining lifespan than any other public health measure. Thus,

optimal oral health cannot be reserved only for those who can afford basic care, but it must be a national priority to improve the overall health of all citizens.[22]

Physiological/Biochemical Linkage of Periodontal and Systemic Diseases

Many chronic inflammatory conditions share some common physiological and biochemical elements with periodontal disease (periodontitis).[23] Periodontitis is more than a localized oral infection. Recent data indicate that periodontitis may initiate changes in systemic physiology and biochemistry that alter immune function, serum cytokine/lipid levels, and tissue homeostasis.[23-25]

Periodontal Disease, Diabetes, and Insulin Resistance

The interrelationship of periodontitis and diabetes is the most obvious example of a systemic disease predisposing one to oral infection, and once that infection is established, it may in turn exacerbate the disease.[25-27] However, in this case, an oral infection might also predispose otherwise healthy patients to systemic disease.[23-25] Common cellular/molecular mechanisms underlie the reciprocity of diabetes and periodontitis and likely synergize when the conditions coexist. The proposed mechanistic link involves the broad axis of inflammation and includes activities of immune cells (polymorphonuclear leukocytes, monocytes, macrophages), serum lipid levels (low density lipoprotein cholesterol and triglycerides (LDL/TRG)), and tissue homeostasis.[23-30]

As a result of metabolic and enzymatic deficiencies, diabetic patients are prone to elevated serum LDL/TRG, even when blood glucose levels are controlled.[31-34] Elevated lipid levels alter immune cell function, producing an inflammatory immune cell phenotype (upregulation of pro-inflammatory cytokines from monocytes/polymorphonuclear leukocytes and downregulation of growth factors secreted from macrophages).[28-30,35-38] This occurrence predisposes an individual to chronic inflammation and progressive tissue breakdown and diminishes tissue repair capacity. Periodontal tissues are frequently affected because they are constantly wounded by substances emanating from bacterial biofilms. Thus much clinical and epidemiological evidence supports the idea that individuals with diabetes (both type I and type II) show higher incidence of severe or rapidly progressing forms of periodontitis than non-diabetics.[39-45]

Periodontitis-induced bacteremia/endotoxemia also elevates levels of serum pro-inflammatory cytokines such as interleukin-1 beta (IL-1ß) and tumor necrosis factor-alpha (TNF-α).[46-48] The activities of both cytokines alters lipid metabolism and leads to hyperlipidemia similar to that observed in diabetes. Additionally, these factors can produce an insulin resistance syndrome similar to that observed in pre-diabetes and may initiate destruction of pancreatic ß-cells, leading to diabetes. Elevated IL-1ß levels play a role in the development of type I diabetes through destruction of pancreatic ß-cells.[49-52] TNF-α has also been implicated as a causative factor in insulin resistance and type II diabetes, as elevated TNF-α levels alter intracellular signaling stimulated by insulin, reduce synthesis of the insulin-responsive glucose transporter, and mediate macrophage-dependent cytotoxicity in pancreatic islets.[53-57] Infection-induced insulin resistance syndromes, if long-standing or chronic, are precursors of active diabetes because of the pancreatic ß-cell destruction resulting from sustained elevation in IL-1ß/TNF-α.[51,53,57] In fact, a pro-inflammatory imbalance created by excess IL-1ß/TNF-α may be one of the most critical determinants of ß-cell loss in diabetes.[58] Thus, periodontitis may potentially exacerbate conditions associated with diabetes.

Recent clinical studies provide strong support for such a hypothesis, particularly with regard to insulin resistance.[59] The degree of insulin resistance has been shown to be directly related to the severity of periodontitis[60], and some investigators have demonstrated a relationship between pre-diabetes and periodontitis in non-diabetic patients.[61] Such reciprocity is further illustrated by recent studies demonstrating the effect of either periodontal disease or successful treatment of such disease on diabetes-induced hyperglycemia.[62-64] Periodontal treatment actually improved glycemic status, especially in type II diabetes, where controlled trials have shown improvement of glycemic control after periodontal therapy.[65] In another study, effective treatment of periodontal infection and reduction of periodontal inflammation were associated with lower levels of glycated hemoglobin.[66] Thus, chronic untreated periodontitis may induce diabetes.

Periodontal Disease and Atherosclerosis-induced Diseases

Investigators have hypothesized that periodontitis-induced elevations of IL-1ß and TNF-α and subsequent elevation of serum LDL/TRG levels trigger development of other systemic conditions that disproportionately affect the elderly, particularly cardiovascular disease (CVD), cerebrovascular disease (CBVD), rheumatoid arthritis (RA), and dementia.[23,24,67-71] In fact, recent studies suggest that in advanced periodontitis, serum levels of IL-1ß, TNF-α, and other inflammatory mediators/biomarkers are sufficiently elevated to be considered a significant systemic health risk, even in the absence of overt clinical symptoms of disease.[46,72,73] Even at low levels, these cytokines can initiate significant systemic responses, including inflammatory tissue and organ damage. Interestingly, chronic periodontitis is one of the strongest and most reliable elevators of serum pro-inflammatory cytokine/lipid levels, and treatment of peri-

odontitis has been shown to significantly reduce serum levels of these factors.[23-27,48,67,74,75]

The impact of periodontal disease on CVD and CBVD is closely tied to mechanisms underlying initiation and progression of atherosclerosis. This effect involves a combination of a systemic inflammatory state and dysregulation of lipid metabolism plus direct interaction of periodontal pathogens with vascular walls. Increasing evidence confirms that periodontitis facilitates atheroma formation.[67] The contribution of systemic inflammation and elevated serum LDL/TRG to atherosclerosis has been thoroughly described.[76] Periodontitis-induced elevation of serum pro-inflammatory cytokines, inflammatory biomarkers, and serum LDL/TRG has also been well documented.[23-27,67,72-74,77-81] Other studies demonstrate that periodontal pathogens interact with vascular endothelial cells (either directly or through inflammatory responses to antibodies directed against periodontal pathogens) and colonize developing atheromas, contributing to thickening of arterial walls and atheroma formation.[67,82-89] Thus, there is likely more than a casual relationship between periodontitis and pathogenic mechanisms underlying atherosclerosis. In the presence of systemic periodontal pathogens and antibody-mediated reactions to those pathogens, periodontitis-induced elevated serum LDL/TRG and pro-inflammatory cytokines may damage vascular endothelial cells, leading to recruitment of macrophages or foam cell formation and development of atheromatous plaques.

Periodontal Disease and Rheumatoid Arthritis

Periodontitis and RA share common pathogenic mechanisms and immunological/pathological outcomes. Patients with severe periodontitis or RA exhibit similar blood cytokine profiles distinct from disease-free individuals.[68] Recent evidence suggests a strong correlation between the extent and severity of periodontal disease and RA. Individuals with advanced RA will likely experience more significant periodontal problems than normal individuals and vice-versa.[69] Thus these conditions could be closely related through the systemic inflammatory state previously described, as accumulating evidence supports the notion that elevated pro-inflammatory cytokines and reduced tissue repair capacity are manifested in both conditions.[69] Additionally, some periodontal pathogens expressing the enzyme peptidylarginine deaminase (PAD) may generate antigens capable of stimulating production of rheumatoid factor-containing immune complexes and initiating inflammation in the synovium.[90] Of the pathologies previously described, evidence for this relationship is preliminary. Nonetheless, the immediate clinical lesson is that the periodontal status of patients with RA should be carefully monitored.

Periodontal Disease and Neurodegenerative Diseases

The inflammatory hypothesis of neurodegenerative diseases such as Alzheimer's and Parkinson's (the most common neurodegenerative disorders leading to dementia in the elderly) has moved from medical speculation to mainstream thinking.[70,71] Brain mononuclear phagocytes, particularly microglia (brain resident macrophages), protect the nervous system by acting as debris scavengers, killers of microbial pathogens, and regulators of immune responses. Microglia are activated by numerous environmental stimuli including pro-inflammatory cytokines and bacterial lipopolysaccharides, initiating a cascade of neuroinflammatory events.[70] Systemic inflammation is associated with signals transferred from blood to brain via perivascular macrophages and microglia.[91] Resultant neuroinflammatory responses include secretion of neurotoxic factors mediating neuronal cell injury and death. Over time a slow, smoldering inflammation in the brain may destroy sufficient neurons to cause clinical manifestations of Alzheimer's or Parkinson's dementia. Indeed, a recent population-based prospective cohort study showed that serum C-reactive protein and pro-inflammatory cytokine levels are increased prior to the clinical onset of dementia.[92] These interactions suggest that systemic infections, or indeed any challenge promoting a systemic inflammatory response, may contribute to the progression of chronic neurodegenerative disease and provide a potential link between early periodontitis-induced bacteremia/endotoxemia and subsequent neuronal injury.

Memory can be impacted by insulin activity. Insulin resistance is associated with age-related memory impairment and Alzheimer's disease.[93] Thus, the previously described periodontitis-induced insulin resistance may contribute to pathologic mechanisms underlying neurodegeneration, as might the described links between periodontitis and vascular disease. Increasing evidence indicates that several pathogenic mechanisms promoting atherosclerosis also function in neurodegenerative diseases.[94] Vascular disease and Alzheimer's disease share some biological mechanisms and risk factors, such as lipid metabolism dysregulation and systemic inflammation. Although the evidence for such a relationship is preliminary, atherosclerosis may be another important mechanistic link between periodontitis and neurodegeneration affecting the elderly.

Periodontal Disease and Respiratory Diseases

Periodontal disease may be connected more directly to respiratory disease. Although some studies of chronic obstructive pulmonary disease describe significant associations with periodontal disease and propose systemic mechanisms relating to inflammation and/or physiologic linkages, this data requires further support.[95-98] A stronger case has been made that bacterial components of den-

tal plaque are a major cause of respiratory infections in older adults, especially those in institutions.[99-102] Up to 48% of infections seen in nursing homes result from aspiration pneumonia, and the cost to treat patients developing pneumonia in these institutions has increased dramatically.[99] Aspiration pneumonia is a significant cause of morbidity, hospitalization, and mortality in the nursing home population.[100]

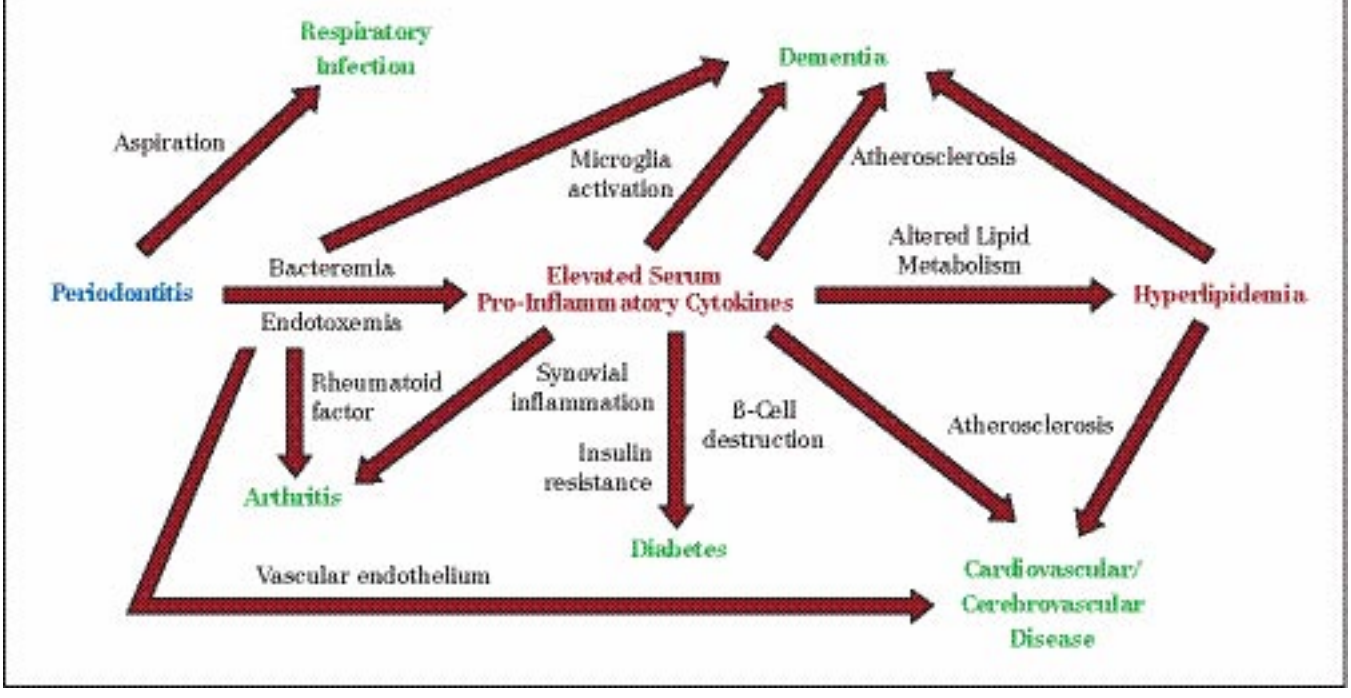
When host defense mechanisms are compromised because of disease, aging, poor nutrition, or other conditions associated with elderly patients in nursing homes, the aspiration of a large pathogenic inoculum from periodontally involved teeth overwhelms normal flora and significantly increases the risk of respiratory infection.[103] Bacteria constitute approximately 70% to 80% of solid plaque material and 1 mm^3 of plaque contains more than 106 bacteria of 300 different aerobic and anaerobic species.[99] Aspiration of plaque bacteria by older patients often leads to lower respiratory tract infections, such as aspiration pneumonia or pneumonitis, and recent evidence links anaerobic bacteria from periodontopathic biofilms with aspiration pneumonia in elderly persons.[103,104] Thus, poor periodontal health and accumulation of dental plaque is a major contributory factor in respiratory infections.[99,100,105] Furthermore, addressing periodontal health in nursing home patients is critical not only because older adults are now more likely to retain their teeth but because younger persons are being admitted to such facilities.[101,102]

Effect of Aging on the Periodontal-Systemic Connection

Age may also predispose individuals to the periodontal-systemic connection. It is well known that the incidence of periodontitis and the severity of untreated periodontal disease increase with age.[106-108] Secretion of pro-inflammatory cytokines from monocytes/macrophages and levels of soluble TNF-α receptor normally increase with age.[109,110] Thus, further increases in serum cytokines induced by periodontitis would not only be cumulative but could create a more destructive scenario through enhanced receptor interactions. Serum lipid levels also increase with age, particularly LDL.[111,112] Similar to the pro-inflammatory cytokines, periodontitis-induced LDL/TRG elevation could be more damaging in this context and further increase pathological lipid levels.

Aging is associated with increased insulin resistance.[113,114] Likewise, the incidence and severity of diabetes also increases with age.[115,116] These conditions may result from loss of an individual's capacity to respond to environmental challenges.[115] Some investigators attribute this association to an imbalance of important intracellular divalent cations such as calcium and magnesium that make cells vulnerable to ionic disturbances.[116] Many other systemic diseases/conditions associated with chronic inflammation also demonstrate increased incidence and severity with

Fig. 1: Linkage between periodontitis and systemic diseases/conditions predominantly affecting the elderly



advancing age.[117,118] The specific mechanisms by which advanced age promotes chronic inflammation are not yet understood. Nonetheless, age-related increases in pro-inflammatory cytokine/cytokine receptor expression and oxidative stress, along with decreased bioavailability of free radical scavenging systems, likely exacerbate the systemic inflammatory state in the elderly and promote development of chronic inflammatory diseases.[118,119]

Thus, with advancing age, factors mediating the periodontal-systemic connection are positioned to act synergistically, putting even healthy patients at risk to develop periodontitis. An overview of the link between periodontitis and systemic diseases/conditions predominantly affecting the elderly is shown in Figure 1 (previous page). Of perhaps greater significance relative to morbidity/mortality, elderly patients with chronic periodontitis also may be at risk for developing any systemic disease or condition associated with elevated pro-inflammatory cytokines/serum lipids. Future clinical studies are required to validate this premise.

Masticatory Function and Nutrition in Older Adults

Although recent discussions of the periodontal-systemic connection have focused on systemic diseases/conditions, there is significant evidence associating dietary imbalance with systemic illnesses. Oral health significantly influences dietary intake, particularly aspects of oral health related to masticatory function and edentulism.[122-129]

Mastication is the first step in digestion and is absolutely essential to optimize dietary intake. Masticatory function in older individuals is influenced by two variables: the number and health of natural teeth and the functional status of dental prostheses. Older people tend to have fewer natural teeth and there are higher rates of edentulism with increasing age. That pattern is, however, changing with a projected reduction in edentulism over the next 20 years.[2,3] Nevertheless, many older people rely on dentures for oral function, and even those who are dentate may require either partial dentures or a full denture in one jaw opposed by some natural teeth. Masticatory function in this group is often not much better than that seen in individuals who rely on full dentures.[124,127] Additionally, over 25% of those aged 65-74 have severe periodontal disease likely accompanied by varying levels of pain or dysfunction.[4] Compromised masticatory function causes variation in food choice to foods that an individual with impaired chewing can tolerate.[122-129] Thus, poor oral health, especially poor periodontal health and edentulism, may negatively impact systemic health by disturbing nutritional intake.

Tooth loss is correlated with changes in diet that may contribute to increased risk of developing chronic diseases.[12] Losing natural teeth and/or pain associated with oral infection impairs masticatory function.[124] This outcome is particularly relevant to people residing in institutional settings where mastication may not be monitored. Furthermore, the use of dental prostheses may not always restore full masticatory function, resulting in significant dietary changes through altered food choices or food preparation methods.[123] Indeed, denture wearers are often not aware of gradual adjustments made in food choice. As masticatory efficiency declines, people report increasing difficulty chewing foods and may choose not to eat foods difficult to chew, such as beef or steak, raw vegetables, or dry solid food like crusty bread. People handicapped by their dentition consequently suffer impaired intake of fruits, vegetables, and some key nutrients. Decreased intake of total calories, proteins, non-starch polysaccharides (dietary fiber) and vitamins is often accompanied by increased consumption of sugars and fats. These dietary links are supported by evidence by several large cross-sectional and longitudinal studies in Europe and the US linking oral health status to biochemical analyte levels of key micronutrients.[130] These studies (the National Diet and Nutrition Surveys in the UK and the National Health and Nutrition Examination Surveys in the US) cited significant reductions in some micronutrients (vitamin C, retinol, folate and ß carotene) in edentulous subjects compared with those with natural teeth. The outcomes were independent of effects of age, gender, regional variation within a country and socioeconomic group.

The negative impact of masticatory dysfunction is likely compounded by food preparation. Fresh foods can be over-prepared (by removing skin from fruits and vegetables) or over-cooked by or for a person with reduced chewing efficiency and nutrients are consequently lost. Those nutrients might prevent or antagonize disease and facilitate cellular defenses and combat aging (anti-oxidant vitamins C and E).[131,132] Reduction in dietary fiber (non-starch polysaccharides or dietary fiber) and in fruit and vegetable consumption is associated with increased risk of CVD and certain cancers (breast, prostate, cervical, pancreatic, gastric and colorectal), most likely because of the lipid-lowering capabilities of soluble fiber and the beneficial effects of antioxidants in fruits and vegetables.[131-139] Many dietary components that form an essential part of the cellular defenses against oxidative damage to DNA may be lost because of over-cooked meals. They include the trace elements selenium, zinc and manganese, vitamins A, C and E and other plant-derived micronutrients (ß carotene, luteine, lycopene and plant flavenoids).[131,132] The role of these factors in maintaining homeostasis is becoming increasingly apparent. Links have been demonstrated between deficiencies of these micronutrients, tissue breakdown, and cardio-vascular disease and stroke in the elderly.[135-138]

Recent studies confirm a direct relationship between compromised masticatory function and malnutrition in both community-dwelling and institutionalized older adults.[125-129] Elderly people with persistent untreated periodontal disease and/or dysfunctional prostheses had lower Healthy Eating Index (HEI) scores and significantly lower intake of vitamins A and B6.[125] Poor nutritional status demonstrated by the Mini Nutritional Assessment (MNA) and a questionnaire on eating problems was significantly associated with periodontal problems, edentulism, and dysfunctional prostheses.[126] These findings emphasize the importance of tools such as the MNA or HEI to assess nutrition along with dental evaluation for oral problems in a comprehensive care plan. Poorly fitting prostheses or chronic periodontitis as well as the potential for associated lesions and pain are of concern for individuals suffering from dementia or conditions that prevent them from articulating the cause for difficulty in chewing food.[140-143] Often, institutionalized elderly patients undergo rapid weight loss after being fitted for new dentures that are not monitored for comfort and functional efficiency.[141,142] Most often, poorly fitting dentures can be confirmed through the presence of lesions at the borders of the prostheses such as tissue overgrowth (epulis fissuratum) or ulcerations (denture ulcers).

Restoration of masticatory function by dental intervention alone will not necessarily lead to improved nutritional intake. Dental services should always be complemented by nutritional counsel, as has been confirmed in a recent clinical study where impaired chewing ability caused avoidance of hard and fibrous foods including fruits, vegetables and whole grains leading to a very low intake of non-starch polysaccharides and micronutrients.[123] In this case, provision of prostheses did not improve the diet. However, individualized dietary advice provided at the time of denture insertion resulted in increased consumption of fruits and vegetables and improved intake of non-starch polysaccharides. It is important to note that an individual's ability to respond to nutritional advice will be moderated by their oral health status. For example, an edentulous patient who has had a stroke resulting in paresis of the facial musculature will have considerable difficulty chewing foods because their ability to stabilize a complete denture during function will be impaired. The dental professional will need to work with the comprehensive care team to encourage a diverse and healthy dietary pattern. This could be accomplished in part while the patient receives instructions for use of complete dentures by challenging the patient to explore new foods and chewing methods. Dietary support and advice should always be given to patients being converted to edentulism for the first time, since using complete dentures as a masticatory tool is a challenge that will often be met by the blender unless positive support and advice is forthcoming from the dental team. It is widely recognized that a significant proportion of elderly people admitted to hospitals suffer from nutritional deficiencies and that adequate nutrient intake is an important determinant of recovery from illness.[144-147] Specific plans for nutritional support may be required for edentulous subjects during such recovery, and professional dental help may be required to help such patients cope with poorly fitting dentures.

Two case studies shown below, (Figures 2 and 3), provide strong support for the importance of maintaining oral and periodontal health in aging patients.

Case 1

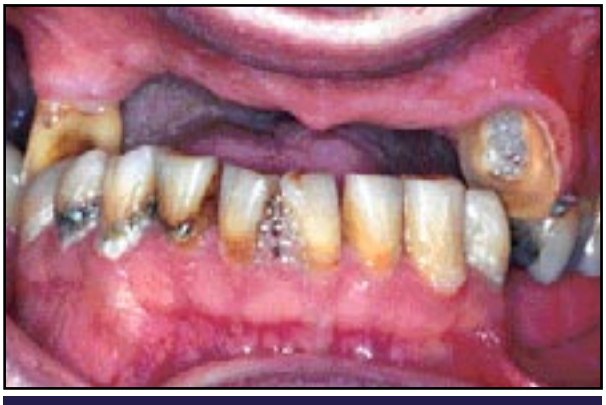


Fig. 2

Case 1. Clinical presentation of severe periodontal disease in an 80-year-old male who suffered a severe stroke two years ago.

Relevant Medical and Social History:

- 80-year-old male, suffered a severe stroke affecting the right side of his body resulting in dense right-sided hemiparesis of the arm and leg with facial paresis and impaired speech (he is able to swallow)
- History of hypertension with arrhythmia and adult onset (type II) diabetes mellitus
- Approximately 6 months after the stroke, hypertension and diabetes worsened and are now difficult to control
- Smokes 20 cigarettes per day and imbibes the equivalent of 20 alcoholic drinks each week
- Fiercely independent widower living alone with pets
- Children live 150 miles away and cannot provide consistent monitoring and care

Relevant Dental History:

- Has not seen a dentist for over 20 years

- Had no complaints prior to the stroke but grandchildren commented about his missing front teeth
- Reports being able to chew all varieties of food prior to his stroke, but his daughter reveals that his diet is limited in terms of variety since the stroke and that he often cooks his food very thoroughly to facilitate chewing

Clinical Findings/Problems:
- Functional contacts between only 2 pairs of natural teeth (he can no longer manipulate a food bolus with his tongue so that it can be chewed by these contacts)
- Poor oral hygiene as a consequence of the hemiparesis
- Mandibular cervical caries, severe wear and pulpal exposure of maxillary molars
- Collapsed vertical dimension of occlusion
- Has never worn a prosthesis
- Altered neuromuscular control and coordination as a consequence of the stroke

Treatment Considerations:
- Severely compromised masticatory function from dental neglect and stroke
- Had been able to function prior to the stroke because he could manipulate foods between his remaining tooth contacts
- Since the stroke, has no control over a food bolus because of loss of tongue function and has changed his diet and modified food preparation to facilitate chewing
- Recent exacerbation of existing systemic disease may be because of nutritional imbalances caused by changes in diet and food preparation
- Without masticatory rehabilitation, he can no longer function independently

Treatment Options:
- Provide basic periodontal care, restore mandibular carious lesions, perform endodontics and restorations for maxillary molars, modify diet to processed food combinations that are nutritionally balanced; the latter would require supervision by caregivers or personal care workers
- In addition to dental services listed, provide dentures to increase functional contacts and maximize masticatory efficiency (a significant challenge for this patient to learn to tolerate and use) along with nutritional counseling

Treatment Provided:
- Phase I periodontal therapy
- Restoration of mandibular cervical caries using a glass ionomer
- Single visit endodontics for maxillary molars
- Restoration of maxillary molars using composite resin
- Acrylic maxillary partial denture using zest anchors and increasing the vertical dimension
- Personalized oral hygiene plan for this patient including use of a daily fluoride mouth rinse and application of fluoride varnish to the overdenture abutment on a weekly basis
- Nutritional counseling in consultation with family physician and children for occasional monitoring and reinforcement

Follow-Up:
This treatment approach gave this patient a reasonably stable upper denture that improved his appearance and masticatory function. In most cases, use of maxillary zest anchors would not have been contemplated given his poor oral hygiene; however, it was more important to ensure adequate retention for the removable partial denture. The oral hygiene program was successful in maintaining the zest anchors. This patient adjusted to the denture and was compliant with nutritional advice. His systemic health significantly improved within six months of denture insertion. This patient survived another 10 years before dying from a second stroke.

Case 2

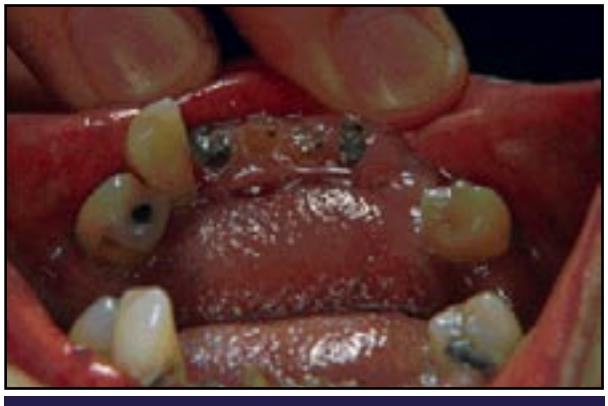


Fig. 3

Case 2. Clinical presentation of severe periodontal disease in an 82-year-old widow living in a nursing home for the past six years.

Relevant Medical and Social History:
- 82-year-old widow, living in a nursing home for the past six years
- Prior to admission, history of RA since age 60 with little change in condition
- Approximately six months after admission, she began to experience states of confusion and, after two years, a primary diagnosis of Alzheimer's-type dementia was recorded (currently being treated with amitryptiline)
- During the past three years, she has suffered from repeated occurrences of lobar pneumonia that did not

respond well to antimicrobial therapy and were slow to resolve
- During the past two years, there has been significant progression of the arthritis with marked deformity of the right wrist and fingers
- During the past year, has had significant weight loss of unknown origin

Relevant Dental History:
- Had seen her general dentist regularly for many years but had not had any dental examination or dental care since entering the nursing home (the facility did not have an oral healthcare policy and most residents were edentulous with the exception of this patient)
- Previous dental records indicated that she had 20 natural teeth remaining when she was last seen by her general dentist; half of her teeth had been restored but the periodontal condition was good and there was no evidence of active caries
- Had not brushed her teeth since the day of her admission to the nursing home and the facility staff were not aware she had her own teeth rather than dentures
- After six years in residence, she was referred for a dental consult by one of the visiting medical staff who was concerned that "she might have a problem with her teeth"

Clinical Findings/Problems:
- Facility had a free access kitchen area providing drinks and readily available snacks, such as bread and jelly
- This patient had a habit of getting cups of tea/coffee for other residents throughout the day and making a drink for herself each time using 2 teaspoons of sugar (estimated frequency of sugar intake was 16-18 sugar-laden drinks per day); the cariogenic effect of the sugar was compounded by the xerostomic effects of amitryptiline
- Rampant cervical caries involving all teeth in maxillary and mandibular arches (the mandibular central and lateral incisors were amputated below the crown)
- Poor oral hygiene and severe generalized periodontal disease

Treatment Considerations:
- Progression of dementia and arthritis along with frequent pneumonia appear temporally related to cessation of oral hygiene and development of rampant caries/periodontal disease
- Weight loss may be related to oral pain and discomfort and/or decreased masticatory function associated with decline of oral health and subsequent nutritional imbalance
- Treatment may need to be limited as a consequence of the extent of her oral disease, caries risk, and ability to tolerate dental procedures

Treatment Options:
- Removal of all remaining teeth and provision of immediate dentures
- Removal of grossly carious roots followed by transitional partial dentures planning for a phased movement to total edentulousness over the next 12-18 months
- Rigorous prevention program to overcome problems with xerostomia/caries and the addition of a partial denture (includes regular use of fluoride rinse, education of facility caregivers in appropriate oral hygiene techniques and the use of a mechanical brush)

Treatment Provided:
- Mandibular lateral and central incisor roots were extracted
- Phase I periodontal therapy
- Carious lesions were restored using chemo-mechanical caries removal with Aldara[i] followed by restoration using glass ionomer cement
- Nutritional counseling for facility caregivers
- Prevention program consisted of regular use of a fluoride rinse and education of facility caregivers in appropriate oral hygiene techniques including use of an automated toothbrush

Follow-Up:
This patient lived an additional 6 years. During that time, her remaining teeth were caries free and she did not develop any additional pneumonias. Her dementia and arthritis did not progress further. Her eating habits improved and she regained her normal weight.

Conclusions

A periodontal-systemic connection, although not fully understood on a molecular level, is relevant to the aging population and supported by ample literature. Evidence for such a connection is sufficient to base patient management approaches on paradigms including comprehensive transdisciplinary care. This is especially important for older patients susceptible to exacerbation of systemic diseases or to development of co-morbid conditions if periodontal health is not maintained. In the future, comprehensive care models must integrate dental and medical professionals with other health professionals to manage periodontitis and systemic disease. Dental professionals should appreciate the periodontal-systemic connection and stress the importance of regular periodontal maintenance to patients and other health professionals. Finally, insurers and policy makers must be convinced that investing in preventive periodontal management will pay huge dividends as the population ages with regard to reducing potential systemic complications, such as chronic inflammatory diseases/conditions.

[i] Carisolv®, 3M, St. Paul, MN

The relationship between periodontitis, systemic disease, and the systemic inflammatory state as defined by serum pro-inflammatory cytokine and lipid levels not only offers intriguing therapeutic possibilities but suggests that notions of adequate preventive patient management need revision. Lipid-lowering therapies or dietary interventions used to treat periodontitis in older patients may also counteract chronic inflammatory systemic diseases linked to periodontitis. Recent clinical studies have established a positive correlation between high LDL/TRG levels and periodontitis.[23-27,61,120,121] In fact, recent data demonstrate that high-fat diets and/or diets rich in omega-6 fatty acids produce a systemic inflammatory state associated with development of periodontitis.[148] Low fat diets, lipid-lowering drugs, and diets rich in omega-3 fatty acids appear to have the opposite effect and may be useful in preventive approaches or in patients with refractory periodontitis.[23-27,149,150]

There are significant interactions between oral health and nutrition. In the presence of dysfunctional dentition and/or oral disease, dietary alterations likely occur resulting in nutritional imbalance. Such deficiencies could exacerbate systemic disease and result in increased systemic illness. Appropriate care strategies to cope with this issue are not yet fully defined, but simple nutrient supplementation is unlikely to be effective without nutritional counseling from a comprehensive care team. Thus, elderly patients should be evaluated for masticatory function, and rapid weight loss without apparent systemic cause should trigger an oral assessment or a dental referral.

References

1. Administration on Aging, Washington, DC. Profile of older Americans. 2000. Available at www.aoa.gov/aoa/stats/profile/default.htm.
2. American Dental Association, Chicago, American Dental Association Health Policy Resources Center. Future of dentistry. 2001. Available at: http://www.ada.org/prof/resources/topics/futuredent/index.asp#future. Accessed Jun 8, 2006.
3. Murdock SH, Hoque MN. Current patterns and future trends in the population of the United States: implications for dentistry and the dental profession in the twenty-first century. *J Am Coll Dent*. 1998; 65:29-35.
4. Oral Health in America: A Report of the Surgeon General. U.S. Department of Health and Human Services, National Institute of Dental and Craniofacial Research, National Institutes of Health. Rockville, MD; 2000. Accessed at: http://www.nidcr.nih.gov/sgr/oralhealth.htm.
5. Oral Health America, Chicago, IL. A state of decay: The oral health of older Americans. 2003. Available at: http://www.oralhealthamerica.org/pdf/StateofDecayFinal.pdf. Accessed Jun 8, 2006.
6. Centers for Disease Control and Prevention. Surveillance for use of preventive health-care services by older adults, 1995-97. *MMWR Morb Mortal Wkly Rep*. 1999;48:51-88.
7. Bonito AJ. Executive summary: dental care considerations for vulnerable populations. *Spec Care Dentist*. 2002;22:5S-10S.
8. Niessen LC. Geriatric dentistry in the next millennium: opportunities for leadership in oral health. *Gerodontology*. 2000;17:3-7.
9. Ship JA, Chavez EM. Management of systemic diseases and chronic impairments in older adults: oral health considerations. *Gen Dent*. 2000;48:555-565.
10. Helgeson MJ, Smith BJ, Johnson M, et al. Frail elderly adults: dental care considerations of disadvantaged and special care populations. Conference Proceedings; HRSA-BHPr, Baltimore, MD, 2001.
11. Centers for Disease Control and Prevention. National Center for Health Statistics. Third national health and nutrition examination survey. 1997. Available at: www.cdc.gov/nchs/about/major/nhanes/nh3data.htm. Accessed Jun 8, 2006.
12. Ettinger RL, Watkins C, Cowen H. Reflections on changes in geriatric dentistry. *J Dent Educ*. 2000;64: 715-722.
13. Matear D. Why do we need education in geriatric dentistry? *J Can Dent Assoc*. 1998;64:736-738.
14. Field MJ, ed. Dental Education at the Crossroads: Challenges and Change. Institute of Medicine, Committee on the Future of Dental Education. Washington, DC: National Academy Press; 1995.
15. Klein SM, ed. A National Agenda for Geriatric Education: White Papers. Washington DC: HRSA-BHPr; 1998.
16. Valachovic RW, Haden NK. Trends in dental education 2000: the past, present, and future of the dental profession and the people it serves. Washington DC: American Dental Education Association; 2000. Available at: www.adea.org/publications/trends.htm.
17. Whipp JL, Ferguson DJ, Wells LM, et al. Rethinking knowledge and pedagogy in dental education. *J Dent Educ*. 2000;64:860-866.
18. Glassman P, Meyerowitz C. Postdoctural education in dentistry: preparing dental practitioners to meet the oral health needs of America in the 21st century. *J Dent Educ*. 1999;63:615-625.
19. Wisconsin Geriatric Education Center and Marquette University School of Dentistry, Milwaukee, WI. Geriatric Oral Health: The Missing Link to Comprehensive Care. Available at: www.wgec.org. Accessed Jun 8, 2006.
20. Scannapieco FA. Systemic effects of periodontal diseases. *Dent Clin North Am*. 2005;49:533-550.
21. Löesche WJ, Lopatin DE. Interaction between periodontal disease, medical diseases and immunity in the older individual. *Periodontol 2000*. 1998;16:80-105.
22. Petersen PE, Yamamoto T. Improving the oral health of older people: the approach of the WHO Global Oral Health Programme. *Community Dent Oral Epidemiol*. 2005;33:81-92.

23. Iacopino AM, Cutler CW. Pathophysiological relationships between periodontitis and systemic disease: recent concepts involving serum lipids. *J Periodontol*. 2000;71:1375-1384.
24. Cutler C, Iacopino AM. Periodontal disease: links with serum lipid/triglyceride levels? Review and new data. *J Int Acad Periodontol*. 2003;5:47-51.
25. Iacopino AM. Periodontitis and diabetes interrelationships: role of inflammation. *Ann Periodontol*. 2001;6:125-137.
26. Cutler CW, Machen RL, Jotwani R, et al. Heightened gingival inflammation and attachment loss in type 2 diabetics with hyperlipidemia. *J Periodontol*. 1999;70: 1313-1321.
27. Cutler CW, Shinedling EA, Nunn M, et al. Association between periodontitis and hyperlipidemia: cause or effect? *J Periodontol*. 1999;70:1429-1434.
28. Chu X, Newman J, Park B, et al. In vitro alteration of macrophage phenotype and function by serum lipids. *Cell Tissue Res*. 1999;296:331-337.
29. Doxey DL, Nares S, Park B, et al. Diabetes-induced impairment of macrophage cytokine release in a rat model: potential role of serum lipids. *Life Sci*. 1998;63:1127-1136.
30. Iacopino AM. Diabetic periodontitis: possible lipid-induced defect in tissue repair through alteration of macrophage phenotype and function. *Oral Dis*. 1995;1:214-229.
31. Howard BV. Lipoprotein metabolism in diabetes mellitus. *J Lipid Res*. 1987;28: 613-628.
32. Merrin PK, Elkeles RS. Treatment of diabetes: the effect on serum lipids and lipoproteins. *Postgrad Med J*. 1991;67: 931-937.
33. Kim DK, Escalante DA, Garber AJ. Prevention of atherosclerosis in diabetes: emphasis on treatment for the abnormal lipoprotein metabolism of diabetes. *Clin Ther*. 1993;15:766-778; discussion 765.
34. Garg A. Lipid-lowering therapy and macrovascular disease in diabetes. *Diabetes*. 1992;41:111-115.
35. Salvi GE, Collins JG, Yalda B, et al. Monocytic TNF alpha secretion patterns in IDDM patients with periodontal diseases. *J Clin Periodontol*. 1997;24:8-16.
36. Thomas CE, Jackson RL, Ohlweiler DF, et al. Multiple lipid oxidation products in low density lipoproteins induce interleukin-1 beta release from human blood mononuclear cells. *J Lipid Res*. 1994;35:417-427.
37. van der Poll T, Braxton CC, Coyle SM, et al. Effect of hypertriglyceridemia on endotoxin responsiveness in humans. *Infect Immun*. 1995;63: 3396-3400.
38. Jovinge S, Ares M, Kallin B, et al. Human monocytes/macrophages release TNF-alpha in response to Ox-LDL. *Arterioscler Thromb Vasc Biol*. 1996;16: 1573-1579.
39. Cianciola LJ, Park BH, Bruck E, et al. Prevalence of periodontal disease in insulin-dependent diabetes mellitus (juvenile diabetes). *J Am Dent Assoc*. 1982;104: 653-660.
40. Emrich L, Shlossman M, Genco R. Periodontal disease in non-insulin-dependent diabetes mellitus. *J Periodontol*. 1991;62: 123-131.
41. Katz PP, Wirthlin MR Jr, Szpunar SM, et al. Epidemiology and prevention of periodontal disease in individuals with diabetes. *Diabetes Care*. 1991;14:375-385.
42. Saftkan-Seppala B, Ainamo J. Periodontal conditions in insulin-dependent diabetes mellitus. *J Clin Periodontol*. 1992;19: 24-29.
43. Haber J, Wattles J, Crowley R, et al. Assessment of diabetes as a risk factor for periodontitis. *J Dent Res*. 1991;70: 414-415.
44. Grossi SG, Zambon JJ, Ho AW, et al. Assessment of risk for periodontal disease. Risk indicators for attachment loss. *J Periodontol*. 1994;65:260-267.
45. Loe H. Periodontal disease. The sixth complication of diabetes mellitus. *Diabetes Care*. 1993;16: 329-334.
46. Ebersole JL, Cappelli D. Acute-phase reactants in infectious and inflammatory diseases. *Periodontol 2000*. 2000;23:19-49.
47. Geerts SO, Nys M, De MP, et al. Systemic release of endotoxins induced by gentle mastication: association with periodontitis severity. *J Periodontol*. 2002;73:73-78.
48. D'Aiuto F, Parkar M, Andreou G, et al. Periodontitis and systemic inflammation: control of the local infection is associated with a reduction in serum inflammatory markers. *J Dent Res*. 2004;83:156-160.
49. Kristiansen OP, Pociot F, Johannesen J, et al. Linkage disequilibrium testing of four interleukin-1 gene cluster polymorphisms in Danish multiplex families with insulin-dependent diabetes mellitus. *Cytokine*. 2000;12:171-175.
50. Vassiliadis S, Dragiotis V, Protopapadakis E, et al. The destructive action of IL-1alpha and IL-1beta in IDDM is a multistage process: evidence and confirmation by apoptotic studies, induction of intermediates and electron microscopy. *Mediators Inflamm*. 1999;8:85-91.
51. Murzenok P, Goranov V. Do local immune-neuroendocrine disturbances initiate diabetes? *Can J Physiol Pharmacol*. 1999;77:147-155.
52. Sjoholm A. Aspects of the involvement of interleukin-1 and nitric oxide in the pathogenesis of insulin-dependent diabetes mellitus. *Cell Death Differ*. 1998;5:461-468.
53. Moller DE. Potential role of TNF-alpha in the pathogenesis of insulin resistance and type 2 diabetes. *Trends Endocrinol Metab*. 2000;11: 212-217.
54. Qi C, Pekala PH. Tumor necrosis factor-alpha-induced insulin resistance in adipocytes. *Proc Soc Exp Biol Med*. 2000;223:128-135
55. Hotamisligil GS. Mechanisms of TNF-alpha-induced insulin resistance. *Exp Clin Endocrinol Diabetes*. 1999;107: 119-125.
56. Peraldi P, Spiegelman B. TNF-alpha and insulin resistance: summary and future prospects. *Mole Cell Biochem*. 1998;182:169-175.

For additional references to this article, please consult the digital version of *Grand Rounds in Oral-Systemic Medicine*™ at www.thesystemiclink.com.