Journal of Gerontology: MEDICAL SCIENCES
2008, Vol. 63A, No. 7, 739–744

Number of Teeth and Mortality Risk in the Baltimore Longitudinal Study of Aging

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Background. Findings from several studies suggested an association between oral health and several health outcomes including cardiovascular disease, aspiration pneumonia, malnutrition, poor quality of life, and mortality. Using data from the Baltimore Longitudinal Study of Aging (BLSA), we tested the hypothesis that number of teeth is indicative of mortality risk independent of other confounders.

Methods. Dentists conducted a standardized oral examination that included tooth count, tooth with coronal and cervical caries count, and gingival and periodontal index. Blood tests used in the analysis included fasting glucose, oral glucose tolerance test, serum low-density lipoprotein (LDL), high-density lipoprotein (HDL) cholesterol, triglycerides, and white blood cell counts. Physical activity, skin fold thickness, body mass index and chronic diseases were also evaluated.

Results. Of the 500 BLSA participants evaluated, 198 died an average of 130 (±75) months postdental evaluation, and 302 survivors were followed for a mean of 185 (±90) months. Based on multivariate Cox regression models, being edentulous or having than 20 teeth was independently associated with mortality.

Conclusion. The results of this study support the notion that number of teeth is a significant and independent risk indicator for early mortality. These findings suggest that the improvement of oral health may have a positive impact on general health and may delay mortality.

***Key Words*:** Aged—Mortality—Cohort studies—Tooth loss.

POOR oral health affects a large number of people around the world and contributes to increased morbidity and mortality (1). Findings from several studies suggest an association between oral health and several health outcomes including cardiovascular disease (2), aspiration pneumonia (3), malnutrition (4,5), and poor quality of life (6–8). Relationships between some aspects of oral health and mortality have also been investigated, but without adequate control for important confounding factors that may be related to oral health and are also independent risk factors for mortality (9–16).

Studies have shown that self-assessed masticatory disability (14), number of missing teeth (4,9,12), and urgent need of dental treatment (12) predict mortality and use of adequate dentures reduces the risk of mortality (11).

It is important to emphasize that oral diseases are linked to noncommunicable chronic diseases primarily through shared common risk factors such as age, lifestyle behaviors, dietary habits, smoking, and low socioeconomic status (16,17). Accordingly, there is some evidence that, after adjusting for these risk factors, the relationship between oral health and cardiovascular mortality is weakened (16).

Two mechanisms by which poor oral health may directly affect all cause and cardiovascular mortality also have been hypothesized. Poor oral health is a major cause of a pro-inflammatory state and through this mechanism may accelerate the atherosclerotic process or precipitate a plaque rupture. In addition, poor oral health may affect eating behavior and contribute to poor nutrition, which has also been identified as a risk factor for mortality (10).

Using data from the Baltimore Longitudinal Study of Aging (BLSA), we tested the hypothesis that number of missing teeth, a simple but reliable proxy measure of oral health, is a risk indicator for mortality independent of other confounders. The BLSA is an ideal study for testing this hypothesis because the oral examination was done concurrently with assessment of major cardiovascular conditions. In addition, participants were evaluated at regular intervals for many years with an average follow-up for mortality of 10 years.

Methods

The BLSA is America's longest-running scientific study of human aging (starting in 1958). Detailed information on dental data was collected between 1978 and 1999. Dental variables assessed included dental caries, periodontal status, dental prosthetic status, salivary gland function, oral health-related behaviors, and access to dental services. All participants had a dental care insurance plan.

There were 773 participants with dental files in the BLSA, of which 500 had information on number of teeth and were included in this study. Mean age was 57.46 (±17.37). Four hundred eight individuals (81.6%) were men. Participants were followed for mortality from time of study entry through 2004. Mortality ascertainment of inactive participants was done by telephone follow-up, correspondence from relatives, and annual searches of the National Death Index.

Dental assessment was performed by dentists, and included number of teeth, number of teeth with coronal and cervical caries, number of teeth with caries, and the DMFT index (sum of the number of decayed, missing and filled teeth). This index provides information on the dental status related to dental caries. DMFT index was assessed in accordance with World Health Organization (WHO) criteria (18). All teeth were examined, except the third molars. Periodontal assessment included the Gingival Index (GI) (19) and Periodontal Index (PDI) (20). Six teeth were examined, 16, 21, 24, 36, 41, and 44. Six sites were examined for each tooth, mesiobuccal, buccal, distobuccal, mesiolingual, lingual, and distolingual. For GI and PDI, six scores were assigned per tooth; an average score was calculated per tooth, and an average for the six examined teeth was calculated. The GI is based on the status of gingival tissues. The scores are ''zero'' for normal gingiva, ''one'' for mild inflammation with a slight change in color, slight edema, and no bleeding on probing; ''two'' for moderate inflammation with redness, edema, glazing and bleeding on probing; and ''three'' for severe inflammation with marked redness and edema, ulceration and tendency to spontaneous bleeding. The PDI was accessed in accordance with Ramfjord criteria (20). If the gingival margin did not extend apically to the cement–enamel junction at any of the measured areas, then the PDI score is the same the GI score (0, 1, 2, or 3 according to GI criteria). The score 4 is applicable when gingival margin extends apically to the cement-enamel junction but not >3.0 mm. The score 5 is applicable when gingival margin extends apically to the cement-enamel junction with a distance of 3.1–6.0 mm. The score 6 is applicable when gingival margin extends apically to the cement-enamel junction with a distance of >6.0 mm.

Frequency of tooth brushing was assessed by asking participants ''how often do you brush your teeth''; and frequency of visits to the dentist was assessed with the question ''How often do you visit a dentist?'' Number of teeth was categorized as follows: Edentulous (number of teeth $= 0$), 1–19 teeth, and ≥ 20 teeth (functional dentition) (21).

Sociodemographic data included age (categorized as ≤ 65 years or >65 years), years of formal education, and sex. Smoking was categorized as current smoker versus former or never smoker. Self-rated health status was assessed by asking participants whether they rated their health as ''robust,'' ''average,'' or ''poor.''

Blood tests used in the analysis included fasting glucose, serum glucose assessed 2 hours postglucose challenge (75 g of glucose in 300 mL of solution), LDL cholesterol, HDL cholesterol, triglycerides, and white blood cell counts. Laboratory assays were performed at the time of the participant's visit within the clinical laboratories at the National Institute on Aging.

Physical activity was assessed using an open-ended questionnaire covering the full gamut of possible activities (22). Participants were asked to report the amount time spent performing 97 activities, including high-intensity physical activities such as vigorous calisthenics, running, or lap swimming. The intensity of each activity was estimated in metabolic equivalents of oxygen uptake (METs) (23). One MET approximates resting energy expenditure in terms of oxygen uptake.

Abdominal skin fold thickness was measured on the right side of the abdomen with a large caliper. Lower waist circumference was measured level with the top of the iliac crest. Cut points used in the analysis were in accordance with the American Heart Association. Waist circumference was considered elevated when it exceeded 88 cm in women and 102 cm in men (24). Body mass index (BMI) was calculated as weight (in kilograms) divided by height (in meters) squared.

Myocardial infarction (MI) was defined as documented MI by medical and hospitalization history and/or typical Q waves on a resting electrocardiogram. Angina pectoris was identified by medical history and current use of anti-ischemic agents or positive ischemic treadmill stress testing and clinical symptoms (Rose criteria). Cancer was defined as non-skin cancer documented by medical history and hospitalization report. Clinical diabetes was defined as history of diabetes with current use of insulin or hypoglycemic agent or two consecutive positive oral glucose tolerance tests (200 mg/dL or more at 120 minutes after 75 g glucose beverage intake). History of stroke was confirmed by medical history and hospitalization records. Transitory ischemic attack was characterized by history of sudden numbness or weakness in the face, arm, or leg; confusion or difficulty in talking or understanding speech; trouble seeing in one or both eyes; and difficulty with walking, dizziness, or loss of balance and coordination that last for few minutes to <1 hour and absence of clinical and image evidence of stroke. Congestive heart failure (CHF) was characterized as documented CHF by medical history and hospitalization report. This study was approved by the Institutional Review Board and all participants provided written informed consents.

Statistical Analysis

The main characteristics of participants who died versus those who remained alive over the follow-up are reported as mean $\pm$ standard deviation or percentages. Differences between groups were tested using age- and sex-adjusted analysis of covariance (ANCOVA) models.

Cox proportional hazards analysis was used to evaluate the univariate and multivariate associations between characteristics at study entry and time to death, with emphasis on number of teeth. Time to death was determined from the time of the first visit in the BLSA study until the day of death or end of the follow-up, whichever came first.

Assumptions of the Cox model were checked by looking at the distribution of Cox–Snell residuals (censored participants), Schoenfeld residuals (proportionality assumption), Martingale residuals (functional form) of the association, and deviance residuals for identifying poorly predicted participants. Data were analyzed using the SPSS 12.0 statistical software (SPSS Inc, Chicago, IL).

Associations between number of teeth and mortality were adjusted for age, sex, education, smoking, self-rated health, serum fasting glucose, 2-hour postchallenge serum glucose,

Table 1. Sociodemographic and Medical Characteristics of BLSA Participants, by Survival Status

Sociodemographic and Medical Variables	Survivors ($N = 302$)	Decedents ($N = 198$)	p*
Age, y	48.38 ± 14.52	71.31 ± 11.10	.001†
Sex, % males	80.5%	83.3%	.48†
Education, y	15.81 ± 3.85	15.41 ± 4.14	.65
Current smoker	13.6%	12.1%	.03
Self-rated health			.79
Robust	42.5%	27.1%	
Average	54.7%	60.4%	
Poor	2.8%	12.5%	
Serum cholesterol, mg/dL	214 ± 37	216 ± 40	.14
Serum LDL, mg/dL	119 ± 32	119 ± 33	.28
Serum HDL, mg/dL	45 ± 11	46 ± 12	.55
Serum triglycerides, mg/dL	110 ± 68	107 ± 33	.96
Fasting glucose, mg/dL	97.89 ± 8.41	99.28 ± 10.11	.55
Two hours postglucose challenge, mg/dL	123.30 ± 37.76	138.53 ± 37.81	.84
White blood cell count, /mm^3	6566 ± 1410	6798 ± 1691	.01
High-intensity physical activity, in METS	149.62 ± 181.39	74.61 ± 134.28	.09
Total physical activity, in METS	2181 ± 394.6	1994 ± 396.5	.87
Skinfold thickness right abdomen, cm	1.63 ± 0.83	1.43 ± 0.77	.10
Lower abdomen circumference			
Normal	75.1%	73.8%	.77
Elevated	24.9%	26.2%	
Body mass index, kg/m^2			
Underweight	2.3%	5.6%	.89
Normal	48.8%	46.0%	
Overweight	39.2%	38.4%	
Obese	9.6%	10.1%	

Notes: *Analysis of covariance model adjusted for age and sex, except for age and sex. Data are referred as mean values ± standard deviation or percentages. †Unpaired t test or Chi-square.

BLSA = Baltimore Longitudinal Study of Aging; LDL = low-density lipoprotein; HDL = high-density lipoprotein; METS = metabolic equivalents.

LDL and HDL cholesterol, triglycerides, white blood cell counts, high-intensity physical activity, total physical activity, abdominal skinfold thickness, lower waist circumference, BMI, MI, cancer, clinical diabetes, angina, stroke, transitory ischemic attack, CHF, number of teeth with coronal and cervical caries, DMFT, average GI, average PDI, frequency of tooth brushing, and frequency of visits to the dentist.

All variables associated with mortality with a p value $<$ .25 were considered potential confounders and were included in the multivariate model predicting mortality. Variables not contributing to the model fit (i.e., $p > .25$) were removed from the final model using a backward selection algorithm.

Results

Of the 500 BLSA participants available for the analysis, 302 survived and 198 died over a mean 164 (±89) months of follow-up. Survivors were followed for a mean of 185 (±90) months. The range of follow up for survivors was 22–324 months. Participants who died were followed for a mean of 130 (±75) months after baseline evaluation. The range of follow-up for deceased was 2–316 months.

Sociodemographic and medical characteristics of study participants are shown in Table 1, according to survivorship and adjusted for age and sex. Dental characteristics of participants according to the survival status are given in Table 2. Only the average GI, an indicator of gingival status, differed between survivors and deceased participants. Table 3 shows the results from chronic diseases of BLSA participants according to the survival status.

In univariate Cox regression models, age, male sex, self-rated health of ‘‘average’’ or ‘‘poor’’ and 2-hour post-challenge serum glucose, MI, cancer, clinical diabetes, angina, transitory ischemic attack and CHF were signifi-

Table 2. Dental Characteristics of BLSA Participants

Dental Variables	Survivors ($N = 302$)	Deaths ($N = 198$)	p*
Number of teeth			
>20 (ref.)	90.4%	63.1%	.27
1–19	7.6%	27.8%	
0	2.0%	9.1%	
Teeth with coronal caries	0.84 ± 1.58	0.54 ± 1.13	.94
Teeth with cervical caries	0.30 ± 0.8	0.62 ± 1.24	.26
Teeth with caries	0.97 ± 1.44	0.93 ± 1.79	.72
DMFT	17.21 ± 6.36	20.97 ± 5.94	.52
Average GI	1.52 ± 0.65	1.73 ± 0.65	.01
Average PDI	4.04 ± 0.88	4.58 ± 0.86	.88
Brushes teeth ≥ 2/d	63.8%	61.6%	.43
Visits dentist ≥ 1/y	82.6%	80.1%	.39

Notes: Data are referred as mean values ± standard deviation or percentages.

*Analysis of covariance model adjusted for age and sex.

BLSA = Baltimore Longitudinal Study of Aging; DMFT = sum of the number of decayed, missing and filled teeth; GI = Gingival Index; PDI = Periodontal Index.

Table 3. Chronic Diseases of BLSA Participants, by Survival Status

Chronic Diseases	Survivors ($N = 302$)	Deaths ($N = 198$)	p*
Myocardial infarction			
Never	99.0%	89.8%	.01
Past	1.0%	10.2%	
Cancer[†]			
Never	99.0%	91.9%	.07
Past	1.0%	8.1%	
Clinical diabetes			
Never	98.7%	88.9%	.01
Past	1.3%	11.1%	
Angina			
Never	95.0%	75.3%	.03
Past	5.0%	24.7%	
Stroke			
Never	99.7%	99.0%	.53
Past	0.3%	1.0%	
Transitory ischemic attack			
Never	98.7%	91.4%	.20
Past	1.3%	8.6%	
Congestive heart failure			
Never	99.7%	99.0%	.48
Past	0.3%	1.0%	

Notes: Data are referred as percentages.

*Analysis of covariance model adjusted for age and sex.

[†]Except skin nonmelanoma.

BLSA = Baltimore Longitudinal Study of Aging.

Table 4. Hazard Ratios (and 95% Confidence Intervals) for Mortality in the BLSA Sample

Independent Variables		Adjusted Hazard Ratios (95% CI)	p
Age >65 y	≤65 y (Ref.)	1.00	.001
	>65 y	8.76 (5.88–13.03)	
White blood cell count/1000		1.15 (1.05–1.27)	.004
Number of teeth	≥20 teeth (Ref.)	1.00	.001
	1–19 teeth	2.17 (1.50–3.13)	.001
	0 teeth	1.76 (1.04–2.98)	.04
Myocardial infarction	Never (Ref.)	1.00	.001
	Past	2.45 (1.41–4.26)	
Clinical diabetes	Never (Ref.)	1.00	.002
	Past	2.23 (1.33–3.74)	

Note: The following variables were in the initial multivariate model: age, sex, self-rated health, glucose at 2 hours, high-intensity physical activity, total physical activity, abdominal skinfold thickness, smoking, white blood cell count, myocardial infarction, cancer, clinical diabetes, angina, transitory ischemic attack, number of teeth, frequency of brushing teeth, difficulty of chewing, number of teeth with cervical caries, number of teeth with coronal caries, DMFT (sum of the number of decayed, missing and filled teeth), average Periodontal Index, and average Gingival Index.

cantly associated with all-cause mortality. Of the dental variables, univariate Cox regression models showed that the number of teeth, teeth with cervical caries, DMFT and average PDI were associated with mortality risk.

In multivariate Cox regression models, age, white blood cell counts, and being edentulous or having <20 teeth, past MI, and clinical diabetes were independently associated with mortality, even after adjusting for potential confounders (Table 4).

Discussion

Using data from the BLSA, we found that missing teeth was a significant risk factor for mortality, even after controlling for potential confounders such as serum fasting glucose, total cholesterol, BMI, past clinical diabetes, and other oral health–related variables that included caries and periodontal disease. Findings from this study are in accordance with other studies that showed a strong association between oral health-related variables and mortality, particularly among elderly participants (9–17).

It has been hypothesized that the association between oral health and mortality, particularly that stemming from cardiovascular disease, may be due to the fact that both dental and cardiovascular diseases share common risk factors, including age, smoking, and low socioeconomic status (16). This hypothesis, however, is not supported by recent findings showing that associations between tooth loss and both all-cause and cardiovascular mortality remained statistically significant even after controlling for sociodemographic variables and behavioral risk factors such as smoking (25). In the current work, additional potential confounders such as serum cholesterol, fasting glucose, and 2-hour postchallenge serum glucose, serum triglycerides, and chronic diseases such as diabetes and cardiovascular diseases were accounted for and thus our findings support and expand those reported by Cabrera and colleagues (25).

Tooth loss results mainly from dental caries and periodontal diseases, the most common oral infections which result from, among other factors, poor oral health. Thus, poor oral health would lead to oral infections, particularly periodontal diseases, which are chronic inflammatory conditions able to produce a mild but persistent systemic inflammatory response (26) known to be associated with elevated levels of systemic inflammatory markers such as interleukin (IL)-1 and IL-6 (26–31).

Nutrition and its relationship to oral health status have been described as possible mediators for the excess mortality observed in people with tooth loss. Pain, infection, and missing teeth can impact mastication, food choice, and nutrition (32). Poor diet and food choices are associated with declining numbers of teeth and increasing age (33). People who have lost teeth can become quite handicapped by their dentition and, as a consequence, substantially reduce their intake of fruit, vegetables, and other key nutrients (34) shown to be associated with increased survival and lower cardiovascular mortality (35). Moreover, adults who have no natural teeth and wear complete replacement dentures tend to have a high fat, low fiber diet (36).

It may be hypothesized that the absence of teeth mediates the complex relationship between oral and general health. Perhaps inflammation cannot fully explain the association of oral health with mortality because oral health could affect people physically and psychologically. It has both biological and social determinants (37) that could act as confounding factors in analyses.

Whether tooth loss increases the chance for mortality by modifying the inflammatory profile due to periodontitis (38), nutrition intake due to masticatory dysfunction (39) or represents a proxy for general health status is open to debate. There is, however, an increasing number of well-designed studies providing evidence that periodontitis is associated with a steady increase in circulatory levels of pro-inflammatory cytokines (26–30) and that tooth loss is associated with worse nutritional status (40,41). However, the importance of the increase in circulating levels of cytokines mediated by periodontal inflammation and of the malnutrition mediated by a compromised dentition on mortality remains to be determined.

One important limitation of the study is that the sample consists largely of well-educated white men. In addition, only a subset of participants had received a dental examination. This limited sample size may have reduced the precision of the analysis. This limited sample size did not allow us to examine the importance of tooth loss on disease-specific mortality, especially on mortality due to cardiovascular disease, reducing the precision of the results. Another limitation is the use of general scores to represent periodontal disease. Use of general indexes presumes that periodontal disease is present in the entire mouth. However, it is well known that periodontitis can be localized to a single region in the mouth which may have evaded detection if not included in the standardized area assessed. This may have resulted in some misclassification error which would tend to underestimate the importance of periodontal disease for mortality (42).

The results of this study support earlier observations, confirming that the number of teeth is a significant risk indicator for mortality, even after controlling for several important confounders. In addition, our study expands the existing knowledge, because it represents one of the first studies that analyzed the relationship between number of teeth and mortality, taking into account important biological confounders in addition to age, smoking, and socioeconomic status. The findings of this study also have significant implications in terms of public health, because they add to previous evidence suggesting that improving oral health and preventing tooth decay may substantially improve the health status of the population and increase longevity.

Acknowledgments

This research was supported (in part) by the Intramural Research Program of the National Institutes of Health (NIH), National Institute on Aging, Clinical Research Branch, Longitudinal Studies Section. Support for this study was provided by the Brazilian Education Ministry (CAPES) and Brazilian National Council for Scientific and Technological Development (CNPq). D.M.P.P and J.B.H. received support from a CAPES grant (1746-05-7 and 0651-06-0, respectively), and F.N.H. received support from a CNPq grant (200256/2006-7).

At the time this manuscript was written, the authors were employed or affiliated with the National Institute on Aging. Data for these analyses were obtained from the Baltimore Longitudinal Study of Aging a study performed by National Institute on Aging.

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References

1. Watt RG. Strategies and approaches in oral disease prevention and health promotion. *Bull World Health Organ.* 2005;83:711–718.
2. Desvarieux M, Schwahn C, Volzke H, et al. Gender differences in the relationship between periodontal disease, tooth loss, and atherosclerosis. *Stroke.* 2004;35:2029–2035.
3. Terpenning MS, Taylor GW, Lopatin DE, Kerr CK, Dominguez BL, Loesche WJ. Aspiration pneumonia: dental and oral risk factors in an older veteran population. *J Am Geriatr Soc.* 2001;49:557–563.
4. Kayser-Jones J. Malnutrition, dehydration, and starvation in the midst of plenty: the political impact of qualitative inquiry. *Qual Health Res.* 2002;12:1391–1405.
5. Mojon P, Budtz-Jorgensen E, Rapin CH. Relationship between oral health and nutrition in very old people. *Age Ageing.* 1999;28:463–468.
6. Hugo FN, Hilgert JB, Silva DD, Sousa MLR, Pucca Jr GA. Correlates of partial tooth loss and edentulism in Brazilian elderly. *Community Dent Oral Epidemiol.* 2007;35:224–232.
7. Ingram SS, Seo PH, Sloane R, et al. The association between oral health and general health and quality of life in older male cancer patients. *J Am Geriatr Soc.* 2005;53:1504–1509.
8. McMillan AS, Leung KC, Pow EH, Wong MC, Li LS, Allen PF. Oral health-related quality of life of stroke survivors on discharge from hospital after rehabilitation. *J Oral Rehabil.* 2005;32:495–503.
9. Abnet CC, Qiao YL, Dawsey SM, Dong ZW, Taylor PR, Mark SD. Tooth loss is associated with increased risk of total death and death from upper gastrointestinal cancer, heart disease, and stroke in a Chinese population-based cohort. *Int J Epidemiol.* 2005;34:467–474.
10. Genkinger JM, Platz EA, Hoffman SC, Comstock GW, Helzlsouer KJ. Fruit, vegetable, and antioxidant intake and all-cause, cancer, and cardiovascular disease mortality in a community-dwelling population in Washington County, Maryland. *Am J Epidemiol.* 2004;160:1223–1233.
11. Hamalainen P, Meurman JH, Kauppinen M, Keskinen M. Oral infections as predictors of mortality. *Gerodontology.* 2005;22:151–157.
12. Hamalainen P, Meurman JH, Keskinen M, Heikkinen E. Relationship between dental health and 10-year mortality in a cohort of community-dwelling elderly people. *Eur J Oral Sci.* 2003;111:291–296.
13. Jansson L, Lavstedt S, Frithiof L. Relationship between oral health and mortality rate. *J Clin Periodontol.* 2002;29:1029–1034.
14. Jansson L, Lavstedt S, Frithiof L, Theobald H. Relationship between oral health and mortality in cardiovascular diseases. *J Clin Periodontol.* 2001;28:762–768.
15. Nakanishi N, Fukuda H, Takatorige T, Tatara K. Relationship between self-assessed masticatory disability and 9-year mortality in a cohort of community-residing elderly people. *J Am Geriatr Soc.* 2005;53:54–58.
16. Tuominen R, Reunanen A, Paunio M, Paunio I, Aromaa A. Oral health indicators poorly predict coronary heart disease deaths. *J Dent Res.* 2003;82:713–718.
17. Yoshida M, Morikawa H, Yoshikawa M, Tsuga K, Akagawa Y. Eight-year mortality associated with dental occlusion and denture use in community-dwelling elderly persons. *Gerodontology.* 2005;22:234–237.
18. World Health Organization. *Oral Health Surveys: Basic Methods.* 4th Ed. Geneva: World Health Organization; 1997.
19. Löe H, Silness J. Periodontal disease in pregnancy. *Acta Odontol Scand.* 1963;21:533–551.
20. Ramfjord SP. The periodontal disease index. *J Periodontol.* 1967;38:602–610.
21. Global goals for oral health in the year 2000. Federation Dentaire Internationale. *Int Dent J.* 1982;32:74–77.
22. Verbrugge LM, Gruber-Baldini AL, Fozard JL. Age differences and age changes in activities: Baltimore Longitudinal Study of Aging. *J Gerontol Soc Sci.* 1996;51B:S30–S41.
23. Talbot LA, Metter EJ, Fleg JL. Leisure-time physical activities and their relationship to cardiorespiratory fitness in healthy men and women 18–95 years old. *Med Sci Sports Exerc.* 2000;32:417–425.
24. Grundy SM. Metabolic syndrome scientific statement by the American Heart Association and the National Heart, Lung, and Blood Institute. *Arterioscler Thromb Vasc Biol.* 2005;25:2243–2244.
25. Cabrera C, Hakeberg M, Ahlqwist M, et al. Can the relation between tooth loss and chronic disease be explained by socio-economic status?

A 24-year follow-up from the population study of women in Gothenburg, Sweden. *Eur J Epidemiol.* 2005;20:229–236.
26. Figueredo CM, Gustafsson A, Asman B, Bergstrom K. Expression of intracellular elastase activity in peripheral neutrophils from patients with adult periodontitis. *J Clin Periodontol.* 2000;27:572–577.
27. D'Aiuto F, Nibali L, Parkar M, Suvan J, Tonetti MS. Short-term effects of intensive periodontal therapy on serum inflammatory markers and cholesterol. *J Dent Res.* 2005;84:269–273.
28. 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.
29. Fredriksson MI, Figueredo CM, Gustafsson A, Bergstrom KG, Asman BE. Effect of periodontitis and smoking on blood leukocytes and acute-phase proteins. *J Periodontol.* 1999;70:1355–1360.
30. Montebugnoli L, Servidio D, Miaton RA, Prati C, Tricoci P, Melloni C. Poor oral health is associated with coronary heart disease and elevated systemic inflammatory and haemostatic factors. *J Clin Periodontol.* 2004;31:25–29.
31. Ajwani S, Mattila KJ, Narhi TO, Tilvis RS, Ainamo A. Oral health status, C-reactive protein and mortality—a 10 year follow-up study. *Gerodontology.* 2003;20:32–40.
32. Petersen PE, Bourgeois D, Ogawa H, Estupinan-Day S, Ndiaye C. The global burden of oral diseases and risks to oral health. *Bull World Health Organ.* 2005;83:661–669.
33. Daly RM, Elsner RJ, Allen PF, Burke FM. Associations between self-reported dental status and diet. *J Oral Rehabil.* 2003;30:964–970.
34. Sheiham A, Steele J. Does the condition of the mouth and teeth affect the ability to eat certain foods, nutrient and dietary intake and nutritional status amongst older people? *Public Health Nutr.* 2001; 4:797–803.
35. Tucker KL, Hallfrisch J, Qiao N, Muller D, Andres R, Fleg JL. The combination of high fruit and vegetable and low saturated fat intakes is more protective against mortality in aging men than is either alone: the Baltimore Longitudinal Study of Aging. *J Nutr.* 2005;135: 556–561.
36. Allen PF. Association between diet, social resources and oral health related quality of life in edentulous patients. *J Oral Rehabil.* 2005; 32:623–628.
37. Sheiham A. Oral health, general health and quality of life. *Bull World Health Organ.* 2005;83:644.
38. Desvarieux M, Demmer RT, Rundek T, et al. Relationship between periodontal disease, tooth loss, and carotid artery plaque: the Oral Infections and Vascular Disease Epidemiology Study (INVEST). *Stroke.* 2003;34:2120–2125.
39. N'Gom PI, Woda A. Influence of impaired mastication on nutrition. *J Prosthet Dent.* 2002;87:667–673.
40. Marcenes W, Steele JG, Sheiham A, Walls AW. The relationship between dental status, food selection, nutrient intake, nutritional status, and body mass index in older people. *Cad Saude Publica.* 2003; 19:809–816.
41. Nowjack-Raymer RE, Sheiham A. Association of edentulism and diet and nutrition in US adults. *J Dent Res.* 2003;82:123–126.
42. Baelum V. The epidemiology of destructive periodontal disease. Causes, paradigms, problems, methods and empirical evidence. Aarhus: University of Aarhus; 1998.

Received July 13, 2007
Accepted October 19, 2007
Decision Editor: Darryl Wieland, PhD, MPH