Periodontal Disease

The sixth complication of diabetes mellitus

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Periodontal disease is a chronic inflammatory condition characterized by destruction of the periodontal tissues and resulting in loss of connective tissue attachment, loss of alveolar bone, and the formation of pathological pockets around the diseased teeth. Some level of periodontal disease has been found in most populations studied and is responsible for a substantial portion of the tooth loss in adulthood.

The association between periodontal disease and diabetes has been explored in several studies over the years (1–5), and it is generally accepted that periodontal disease is more prevalent and more severe in persons with diabetes than in nondiabetic persons. Indeed, the periodontal signs and symptoms are now recognized as the "sixth complication" of diabetes.

Most of our information on periodontal disease in diabetes is derived from studies of persons with IDDM and only limited data are available on the relationship of periodontal disease with NIDDM. In the Pima Indians of the Gila River Indian Community in Arizona, type I diabetes is virtually nonexistent, whereas this population may have the world's highest prevalence of type II diabetes (6).

This review is limited to published and unpublished data from an ongoing longitudinal study of the periodontal condition in type II diabetes of the Pima Indians of the Gila River Community, as part of the Periodontal Research Center at the State University of New York at Buffalo under the leadership of Dr. Robert J. Genco (6–10) and our own studies of type I diabetes and periodontal disease in Danish men (2).

RESEARCH DESIGN AND METHODS — The Pima study population consisted of 2180 subjects 15 yr old or older whose heritage was at least 50% Pima, Papago, or a mixture of these two tribes, and who had undergone one or more biennial clinical examinations, including a comprehensive oral and dental examination, in the years between 1983 and 1988. For subjects seen more than once, the initial examination findings were used to determine the prevalence of periodontal disease. Incidence was computed for a subset of 746 subjects (295 men, 451 women) who were initially free of periodontal disease. Of this subset, 50 individuals had type II diabetes at the outset, whereas the remaining 696 did not have diabetes.

Periodontal disease status was determined without knowledge of the subject's medical status. Panoramic films were used to measure interproximal crestal alveolar bone loss, which was scored as a percentage of bone loss from the cementoenamel junctions to the apex at the deepest point on the mesial or distal surface of each tooth present, excluding third molars. Periodontal disease was determined on the basis of the percentage of bone loss and on clinical findings of tooth loss which, in this population, is overwhelmingly attributable to periodontal disease.

To be classified as nondiseased, subjects had to have a minimum of 24 teeth present, of which <6 had 25–50% bone loss and the rest <25% bone loss. Thus the nondiseased group included those with mild periodontal disease. Diseased subjects had <24 teeth present, or had at least six teeth with ≥25% bone loss, or had any teeth with ≥50% bone loss.

Diabetes status was determined using the World Health Organization criteria for epidemiological studies, namely the 2-h, postload plasma glucose concentration ≥200 mg/dl. The date of diagnosis of diabetes was determined from biennial examinations and from review of clinical records.

In the type I diabetes study, the material consisted of 102 Danish men 20–40 yr of age. Fifty-one individuals were selected at random among insulin-dependent diabetic outpatients in the Diabetic Clinic of the Second Department of Medicine at Aarhus Kommune Hospital (Aarhus, Denmark). The 51 nondiabetic subjects in the control group were also selected at random among patients calling at the hospital for minor ailments. All patients underwent a standardized medical examination, including urine tests for glucose and protein and ophthalmoscopy. The changes of the vascular bed of the retina ranged from mi-

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IDDM OR TYPE I DIABETES, INSULIN-DEPENDENT DIABETES MELLITUS; NIDDM OR TYPE II DIABETES, NON-INSULIN-DEPENDENT DIABETES MELLITUS; IGT, IMPAIRED GLUCOSE TOLERANCE; NGT, NORMAL GLUCOSE TOLERANCE.

Table 1—*Prevalence of periodontal disease in Pima Indians aged ≥15 yr, according to age, sex, and presence of diabetes*

		Nondiabetic subjects			Diabetic subjects		
	Age-group (yr)	Subjects (n)	Cases (n)	Prevalence (%)	Subjects (n)	Cases (n)	Prevalence (%)
Men	15–31	495	39	7.9	62	28	45.2
	35–54	109	58	53.2	133	118	88.7
	≥55	40	39	97.5	66	66	100
Women	15–34	643	48	7.5	94	45	47.9
	35–54	163	102	62.6	191	174	91.1
	≥55	37	35	94.6	147	144	98
Total		1,487	321	21.6	693	575	83

croaneurysms and hemangioma through fibrous proliferations, soft and hard exudates to capillary proliferations. Each control patient had a 3-h oral glucose tolerance test in order to exclude an undetected diabetes. All participants had a standardized periodontal examination using modern scoring and recording procedures.

RESULTS

Type II diabetes and periodontal disease among Pima Indians

Approximately 8% of the nondiabetic subjects <35 yr of age had advanced periodontal disease as defined in this study. In contrast, the prevalence of this level of severity of disease in persons with type II diabetes in the same age-group was 45–48% (Table 1). There were no major differences between men and women in either group.

As can be seen from Figs. 1 and 2, both loss of attachment and bone loss increase with age. However, diabetic subjects scored significantly higher for both of these indexes at all age levels, as compared with those with impaired glucose tolerance and those with normal glucose tolerance level.

Periodontal disease affected >95% of individuals ≥55 yr of age, regardless of their diabetes status. Therefore, measures of incidence were restricted to subjects 15–55 yr of age. Of the 746 individuals who were free of periodontal disease at the start of the study in 1983 and who were followed for up to 51/2 yr, 32 developed periodontal disease; 23 were among the 696 nondiabetic group and 9 belonged to a group of 50 persons with diabetes (Table 2).

The incidence rate of periodontal disease was similar in men and women. Age was strongly predictive of periodontal disease—this is consistent with other epidemiological studies, but diabetes status was even more predictive. After controlling for age and sex, the rate of periodontal disease in subjects with diabetes was 2.9 times—nearly threefold—what it was in individuals without diabetes. Thus, notwithstanding the fact that periodontal disease was common in this population, this study reaffirmed that diabetes confers an added risk. In addition, these studies provide longitudinal data that confirm previous cross-sectional observations of the association between diabetes and periodontal disease in this population.

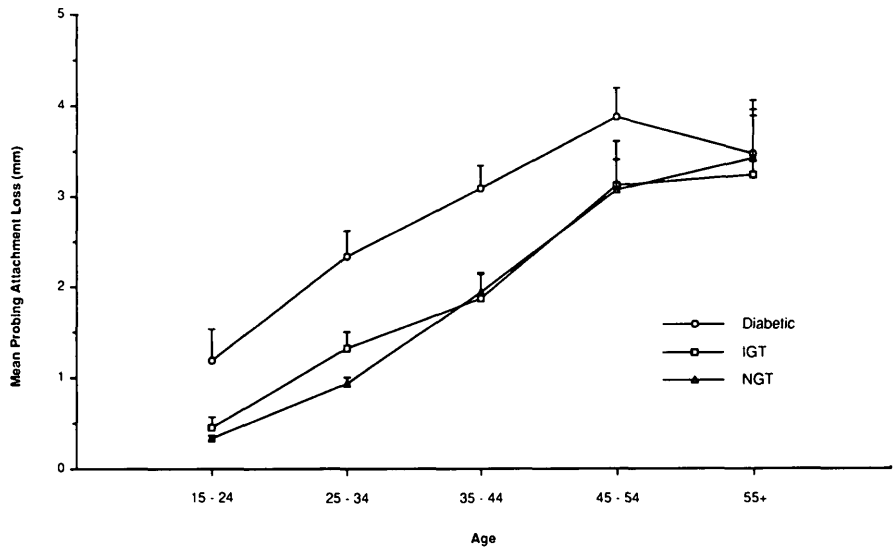

Figure 1—*Mean probing attachment loss with respect to glucose tolerance for all 1342 subjects. Diabetic subjects are those with a 2-h postload glucose level ≥200 mg/dl, IGT are those with a 2-h glucose level ≥140 mg/dl, but <200 mg/dl, and NGT refers to those with a 2-h glucose level of <140 mg/dl. From Emrich et al. (8).* © *by the Journal of Peridontology.*

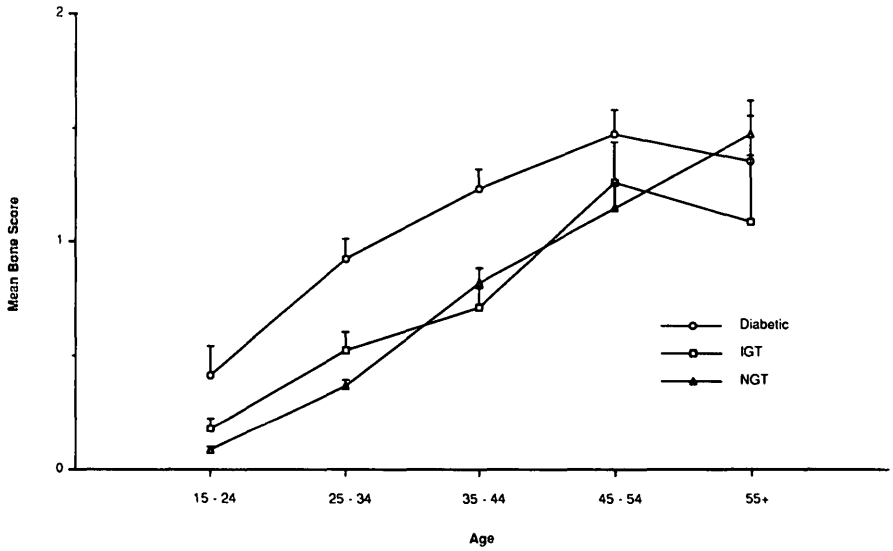

Figure 2—*Mean interproximal radiographic alveolar bone score with respect to glucose tolerance and age for all 1342 subjects. Diabetic subjects are those with a 2-h postload glucose level ≥200 mg/dl, IGT are those with a 2-h glucose level ≥140 mg/dl, but <200 mg/dl, and NGT refers to those with a 2-h glucose level of <140 mg/dl. From Emrich et al. (8). © by the Journal of Peridontology.*

It is also evident from Schlossman and associates findings (7) that, in this population, advanced types of the periodontal lesion occurred relatively early in life, increased with age, and were responsible for most of the tooth mortality and toothlessness at 40 yr and beyond (Figs. 3–5).

Based on preliminary analyses and after adjustment for age and sex, diabetic individuals had an average of 12 missing teeth compared with 8 for nondiabetic individuals. Patients with diabetes were 15 times more likely to be totally edentulous compared with nondiabetic individuals. The frequency of edentulousness increased with duration of diabetes, ranging from 7% edentulous for diabetes of 5-yr duration, to 14% for 10-yr duration to 75% for diabetes of 20-yr duration.

Also, diabetic persons with retinopathy were almost 5 times as likely to have periodontal disease than diabetic persons without retinopathy. No relation was observed between periodontal conditions and kidney complications or urine albumin concentrations. Finally, after controlling for age, sex, and duration of diabetes, the study showed that the odds of having periodontal disease for diabetic persons in poor control (>400 mg/dl) were more than twice that of a subject with a fasting glucose of <200 mg/dl.

Type I diabetes and periodontal disease

Although IDDM is not found among the Pimas, studies of other populations have indicated the IDDM is also associated with periodontal disease. A study (11) of prepubertal diabetic children showed greater frequency and severity of gingivitis than in normal controls, especially in those with poor metabolic control. An earlier prevalence study of 12- to 18-yr-olds in the U.S. indicated that the prevalence of periodontal disease of all forms in nondiabetic samples was ~3%. However, the prevalence appears to increase to between 11% and 16% among youngsters with type I diabetes in the same age-group (11).

Whereas population surveys and longitudinal studies make a strong case for an association and even a positive predictive value of diabetes for periodontal disease, they explain nothing about the mechanisms involved. Early studies conducted in Denmark 20 yr ago (2) compared 102 dentate men between 20 and 40 yr old with 51 age-matched diabetic individuals selected at random from an outpatient diabetes clinic. These studies demonstrated that diabetic indi-

Table 2—*Incidence of periodontal disease in Pima Indians aged 15–54 yr, according to age, sex, and presence of diabetes*

		Nondiabetic subjects			Diabetic subjects		
	Age-group (yr)	Person-yr at risk	Cases (n)	Incidence (new cases/1000 person-yr)	Person-yr at risk	Cases (n)	Incidence (new cases/1000 person-yr)
Men	15–34	463	4	8.6	23	2	87.0
	35–54	50	4	80.0	8	1	125.0
Women	15–34	724	8	11.0	59	2	33.9
	35–54	116	7	60.3	21	4	190.5
Total		1,353	23	17.0	111	9	81.1

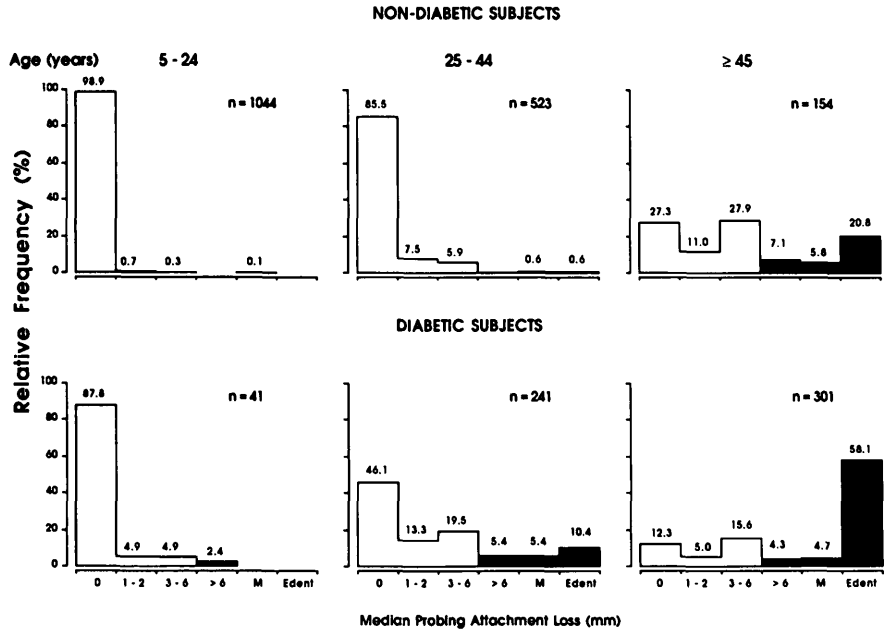

Figure 3—*Distribution of median probing attachment loss. M, missing ≥3 index teeth; Edent, fully edentulous. Shaded areas represent subjects with severe disease: >6 mm posterior axillary line, M, or Edent. From Schlossman et al. (7). © by the Journal of the American Dental Association.*

viduals >30 yr of age showed significant increase in periodontal tissue breakdown as compared with nondiabetic individuals (Fig. 6). Those persons with diabetes of >10-yr duration showed greater loss of attachment than those with more recent histories of diabetes (Fig. 7). Although increasing insulin dosage did not correlate with periodontal disease severity (Fig. 8), patients with retinopathy had significantly more loss of attachment than diabetic persons without retinal changes (Fig. 9).

The fact that the periodontal lesion is similar in type I diabetic persons and in nondiabetic persons and that a high correlation coefficient exists between gingival state and bacterial plaque in both diabetic and nondiabetic persons suggests that the initiation and maintenance of the inflammatory response are caused by the usual bacterial mechanisms. Given the advances in methodology for sampling and cultivating plaque microorganisms available today, it is of interest that a microbiological and immunological study of Pima Indian periodontitis patients with and without NIDDM diabetes (10) tends to confirm this suspicion. Although there was some evidence of a distinct serotype of *Porphyromonas gingivalis* found among the diabetic group compared with the nondiabetic group, the cultivable organisms were species that have often been implicated in the common adult form of chronic periodontitis.

CONCLUSIONS

- The prevalence of advanced periodontal disease was substantially higher among type II diabetic persons than in nondiabetic persons of the Pima Indian community.
- Loss of periodontal attachment and alveolar bone started early in the diabetic population.
- Incidence rate of advanced periodontal disease was the same in men and women; higher age predicted a greater incidence rate.
- The rate of periodontal disease in subjects with type II diabetes was almost

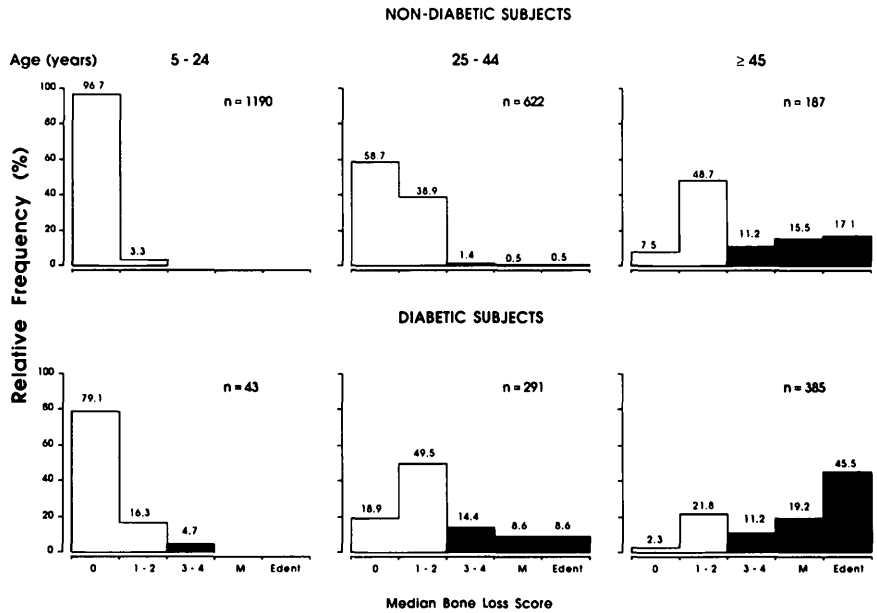

Figure 4—*Distribution of median alveolar bone loss. M, missing ≥14 teeth; Edent, fully edentulous. Shaded areas represent subjects with severe disease: >2 bone loss score, M, or Edent. From Schlossman et al. (7). © by the Journal of the American Dental Association.*

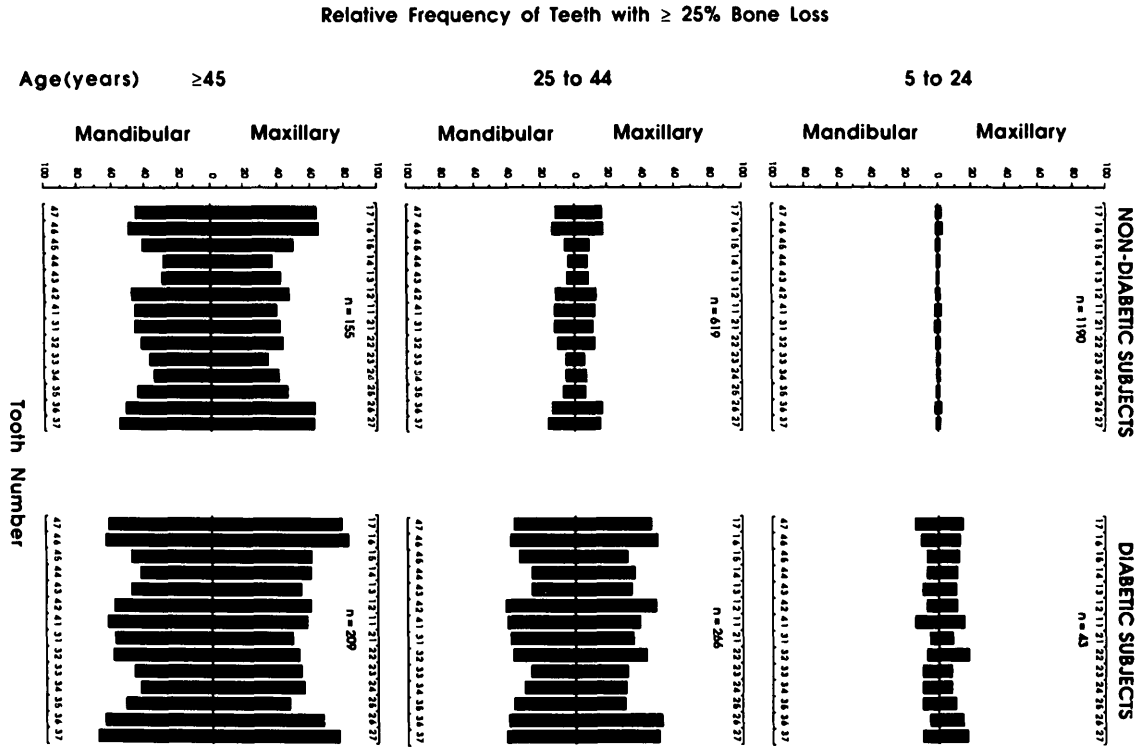

Figure 5—*Distribution of interproximal alveolar bone loss by tooth. Shaded areas represent the percentage of teeth affected by ≥25% bone loss determined by measurement from panoramic radiographs. From Schlossman et al. (7). © by the Journal of the American Dental Association.*

three times that in nondiabetic persons.
- Diabetic persons with retinopathy were almost five times more likely to have advanced periodontal disease than patients without retinopathy.
- Pima Indians with type II diabetes were 15 times more likely to be totally toothless than nondiabetic persons.
- When adjusted for age and sex, it was clear that edentulousness increased significantly with the duration of the diabetic condition.
- Patients 30–40 yr of age with type I diabetes had increased periodontal breakdown.
- Those suffering type I diabetes for >10 yr showed greater loss than those with a history of <10 yr.

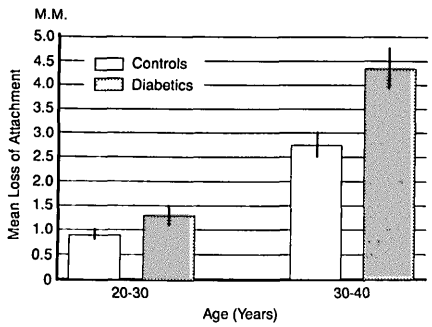

Figure 6—*Mean loss of periodontal attachment (in mm) according to the age of the diabetic and control patients. From Glavind et al. (2). © by the Journal of Periodontology.*

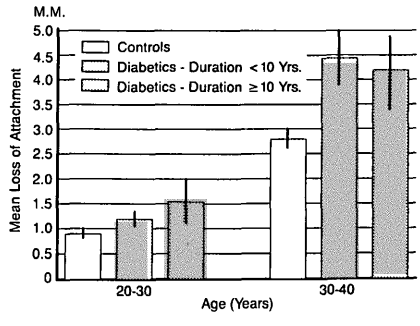

Figure 7—*Mean loss of periodontal attachment (in mm) according to age and diabetes duration. From Glavind et al. (2). © by the Journal of Peridontology.*

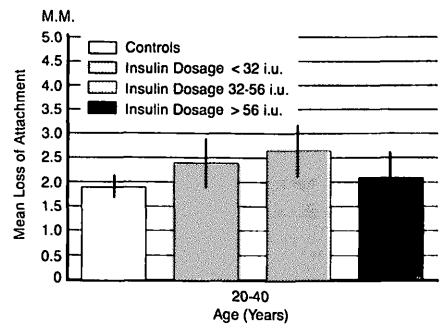

Figure 8—*Mean loss of periodontal attachment (in mm) according to insulin dosage. From Glavind et al. (2). © by the Journal of Peridontology.*

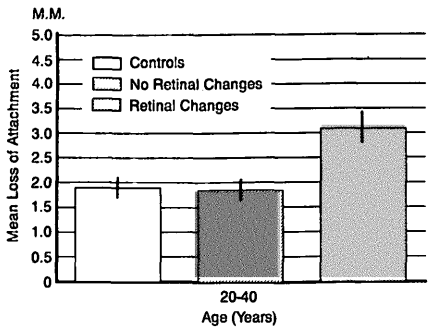

Figure 9—*Mean loss of periodontal attachment (in mm) according to vascular changes in the retina. From Glavind et al. (2).* © *by the Journal of Peridontology.*

- Insulin dosage did not seem to be related to the degree of periodontal destruction. However, type I diabetic persons with retinal changes had greater loss of periodontal attachment than others.
- Findings from studies of IDDM and NIDDM persons have demonstrated that both types of diabetes are predictors of periodontal disease, and that periodontal disease should be considered a complication of diabetes mellitus.

References

1. Belting CM, Hiniker JJ, Dummett CO: Influence of diabetes on severity of periodontal disease. *J Periodontol* 35:476–80, 1964
2. Glavind L, Lund B, Löe H: The relationship between periodontal state and diabetes duration, insulin dosage and retinal changes. *J Periodontol* 39:341–47, 1968
3. Campbell MJA: Epidemiology of periodontal disease in the diabetic and non-diabetic. *Australian Dent J* 17:274–78, 1972
4. Wolf J: Dental and periodontal conditions in diabetes mellitus: a clinical and radiographic study. *Proc Finn Dent Soc* 73 (Suppl. 6):1–56, 1977
5. Snajder N, Carraro JJ, Rugna S, Sereday M: Periodontal findings in diabetic and non-diabetic patients. *J Periodontal* 49: 445–48, 1979
6. Knowler WC, Bennett PH, Hamman RF, Miller M: Diabetes incidence and prevalence in Pima Indians: a 19-fold greater incidence than in Rochester, Minn. *Am J Epidemiol* 108:497–504, 1978
7. Schlossman M, Knowler WC, Pettitt DJ, Genco RJ: Type II diabetes and periodontal disease. *J Am Dent Assoc* 121:532–36, 1990
8. Emrich LJ, Schlossman M, Knowler WC, Genco RJ: Periodontal disease in non-insulin dependent (type II) diabetes mellitus. *J Periodontol* 62:123–30, 1991
9. Nelson RG, Schlossman M, Budding LM, Pettitt DJ, Saad MF, Genco RJ, Knowler, WC: Periodontal disease and glucose tolerance in Pima Indians. *Diabetes Care* 13:836–40, 1990
10. Zambon JJ, Reynolds H, Fisher JG, Schlossman M, Dunford R, Genco RJ: Microbiological and immunological studies of adult periodontitis in patients with non-insulin dependent diabetes mellitus. *J Periodontol* 59:23–31, 1988
11. Cianciola LJ, Park BH, Bruck E, Mosovich L, Genco RJ: Prevalence of periodontal disease in insulin-dependent diabetes mellitus (juvenile diabetes) (Abstract). *J Am Dent Assoc* 104:653–60, 1982