

Poor Oral Health and Blood Pressure Control Among US Hypertensive Adults Results From the National Health and Nutrition Examination Survey 2009 to 2014

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Abstract—Periodontal disease is a chronic inflammatory disorder of the tissues surrounding the teeth, with evidence of systemic effects. Some studies showed the benefit of periodontal therapy on blood pressure (BP), but the impact of periodontitis on BP control is unknown. We retrospectively analyzed cross-sectional, nationally representative data from treated hypertensive adults aged ≥ 30 years with and without periodontitis. BP was examined as both continuous (mm Hg) and categorical (treatment goal achievement status according to guidelines: at goal and above goal) variable according to the presence or absence of periodontitis and its clinical parameters (probing depth, clinical attachment loss, and disease severity [mild, moderate, and severe]). Systolic BP means and odds ratios for uncontrolled BP according to the presence and severity of periodontitis were calculated using progressively adjusted models. Among treated hypertensive adults, mean systolic BP was about 2.3 to 3 mm Hg higher in the presence of periodontitis ($P < 0.0001$). Periodontitis was associated with unsuccessful antihypertensive treatment after multiple adjustments, with higher odds by disease severity. A good periodontal health is associated with better systolic BP profile during antihypertensive therapy by about 2.3 to 3 mm Hg and with lower odds of antihypertensive treatment failure. Dedicated studies are needed to test the impact of periodontal therapy on BP and the long-term effects on cardiovascular outcomes of this complementary approach to systemic health. (*Hypertension*. 2018;72:1365-1373. DOI: 10.1161/HYPERTENSIONAHA.118.11528.) • [Online Data Supplement](#)

Key Words: hypertension ■ inflammation ■ oral health ■ periodontal diseases ■ therapeutics

Periodontal disease is a chronic inflammatory disorder stemming from the tissues surrounding the teeth,¹ but with evidence of systemic effects on inflammatory markers.²⁻⁶ The established role of systemic inflammation as a major determinant of adverse cardiovascular outcomes⁷ has pushed research toward exploring the association of periodontal disease with a variety of cardiovascular conditions. Thus, many cardiovascular risk factors and related diseases, including endothelial dysfunction,⁸⁻¹⁰ hypertension,^{11,12} atherosclerosis,^{13,14} and major cardiovascular events,^{11,12,15-18} have been associated with periodontitis.

According to the National Health and Nutrition Examination Survey (NHANES) 2011 to 2014, hypertension is the third cardiovascular risk factor after physical inactivity and obesity in the United States¹⁹ and affects 30% to 45% of the general population worldwide,^{20,21} with great medical and human costs related to its treatment and complications.²²

Although some studies have reported on the benefit of periodontal treatment on blood pressure (BP) profile,^{23,24} data on the impact of periodontitis on BP control in treated hypertensive patients are lacking. Thus, the aim of the present study is

to examine the association between periodontitis and uncontrolled hypertension in treated hypertensive patients enrolled in the 2009 to 2014 NHANES campaign.

Methods

Data Source NHANES data can be accessed through the Centers for Disease Control and Prevention National Center for Health Statistics website at <https://www.cdc.gov/nchs/nhanes/index.htm>. The present study was deemed exempt from review by the Institutional Review Board at the University of L'Aquila.

Study Population

NHANES is a periodic survey conducted by the National Center for Health Statistics of the Centers for Disease Control and Prevention. It represents a stratified, multistage probability sample of the civilian noninstitutionalized population in the 50 US states and the District of Columbia.

The present study focuses on treated hypertensive adults ≥ 30 years of age, who answered the question: "Are you now taking prescribed medicine for high BP?" during 2009 to 2014 NHANES campaigns. These patients had been told at least once they had high BP. Participants < 30 years of age were excluded because periodontal evaluation was performed only above that age threshold. Oral

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health data collection protocols were approved by the Centers for Disease Control and Prevention, National Center for Health Statistics Research Ethics Review Board, Atlanta, and all survey participants provided written informed consent. All the examinations were conducted in a mobile examination center.

Periodontal Examination

Participants who had at least 1 natural tooth (excluding third molars) and did not meet any of the exclusion criteria (a history of heart transplant, artificial heart valve, congenital heart disease not including mitral valve prolapse, or bacterial endocarditis) were eligible for periodontal examination, consisting in full-mouth, 6-site-per-tooth assessment of periodontal pockets, recession, and loss of attachment. Disease severity was defined according to the gold standard full-mouth periodontitis surveillance protocol following suggested Centers for Disease Control and Prevention/American Academy of Periodontology case definitions.¹ In particular, periodontitis was classified as mild, in the presence of at least 2 interproximal sites with clinical attachment loss (CAL) ≥ 3 mm and at least 2 interproximal sites with probing depth (PD) ≥ 4 mm (not on the same tooth) or 1 site with PD ≥ 5 mm; moderate, defined as at least 2 interproximal sites with CAL ≥ 4 mm (not on the same tooth) or at least 2 interproximal sites with PD ≥ 5 mm (not on the same tooth); and severe, defined as having at least 2 interproximal sites with CAL ≥ 6 mm (not on the same tooth) and at least 1 interproximal site with PD ≥ 5 mm.¹

BP Measurement

Arterial BP was measured by trained and calibrated physicians using a mercury sphygmomanometer according to standardized BP measurement protocols,²⁵ and 3 consecutive BP readings were taken for each patient using the same arm. For the present analysis, an average of these readings, expressed as mean and SD, was calculated for each patient.

Additional Data

Consistent with previous studies^{26–28} and NHANES guidelines, additional data on general health status, medications use, laboratory findings, as well as socioeconomic and demographic background were selected for descriptive and inferential statistics, as appropriate. In particular, participants were stratified by categories of age (30–44, 45–64, and ≥ 65 years), sex (men/women), race (non-Hispanic whites, non-Hispanic blacks, Mexican-Americans, other Hispanics, and other, including multiracial), ethnicity (Hispanics, including Mexican-Americans and other Hispanics; non-Hispanics, including whites, blacks, and multiracial), glycemic status, body mass index, education (less than high school, high school, and more than high school), income (proportion to poverty level), and smoking status (current, former, or never smoker), as collected by NHANES. Ethnicity was examined in terms of being or not Hispanic, because of increased susceptibility of Hispanics to both high BP and periodontal disease.^{29,30} Glycemic status was defined by serum levels of HbA1c (glycohemoglobin A1c) as normoglycemia ($<5.7\%$), prediabetes (5.7%–6.4%), or diabetes mellitus ($>6.4\%$). According to body mass index, participants were classified as being underweight (<18.5), normal weight (18.5–25), overweight (25–30), or obese (≥ 30). Additional parameters of interest included serum levels of CRP (C-reactive protein), glucose, creatinine, and lipid panel (total and HDL [high-density lipoprotein] cholesterol and triglycerides).

Statistical Analysis

All statistical analyses were performed using R software (v3.4.2; R Foundation for Statistical Computing). Differences in demographic characteristics were evaluated with unpaired *t* tests for continuous variables and χ^2 tests for categorical variables. Bonferroni correction was applied as appropriate.³¹

Crude and progressively adjusted linear and logistic generalized additive models^{32,33} were used to evaluate the associations between periodontal disease and uncontrolled hypertension. BP was the dependent variable and was modeled in 2 different ways: (1) continuous (mmHg), in terms of mean systolic BP (SBP), given its stronger relationship with periodontitis than diastolic BP³⁴; (2) dichotomized as being below or above 130/80 mmHg. In accordance with the American College of Cardiology/American Heart Association 2017 Guideline for

the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults,³⁵ this threshold identifies the achievement of treatment goal in treated hypertensive patients. In untreated patients, the same BP values identify hypertension. Independent variables were selected among clinical and demographic characteristics of the examined sample (Table 1) and included age range, sex, ethnicity, body mass index ranges, smoking status, HbA1c ranges, total and HDL cholesterol, triglycerides, creatinine, education, poverty level, and CRP. Selection of smoothed variables was based on restricted maximum likelihood,^{36,37} resulting in the identification of total cholesterol as cubic spline. Following the initial crude model (model 1), 4 progressively adjusted models were generated (model 2: age range, sex, ethnicity, body mass index ranges, and smoking status; model 3: additional inclusion of HbA1c ranges, total cholesterol, HDL cholesterol, triglycerides, and creatinine; model 4: also adjusted for education and poverty level; model 5: full adjusted model incorporating CRP). SBP means were evaluated according to the presence or absence of periodontitis. In treated hypertensive adults, the same were then stratified by selected demographic characteristics (age range, sex, and race/ethnicity) and clinical parameters of periodontitis, namely PD, CAL, and disease severity (mild, moderate, and severe). Student *t* tests were used to evaluate differences in mean among groups.

Crude and adjusted odds ratios for uncontrolled BP during treatment according to the presence of periodontitis were obtained from logit generalized additive models.

Cubic splines with 3 knots located at the 25th, 50th, and 75th percentiles of the distribution³⁸ were generated to explore the relationship between SBP and PD/CAL.

Data were analyzed as recorded, without any imputation for missing data.

Statistical significance was set at $P < 0.05$. See Methods in the [online-only Data Supplement](#) for a detailed discussion of the statistical methods.

Results

A total of 11 753 participants of 19 528 underwent complete periodontal examination during the NHANES 2009 to 2014 campaigns. Among them, those who answered the question “Are you now taking prescribed medicine for high BP?” were 4095, of whom 3626 (88.5%) provided a positive answer, and 460 (11.5%) answered no. Demographic and clinical characteristics of treated hypertensive patients by periodontal disease severity are reported in Table 1. Briefly, 47.8% of patients were found to be free of periodontal disease. Among the remaining 52.2%, the majority had moderate disease (37.8%), followed by severe (11.5%) and mild (2.9%) disease. Compared with mild periodontitis, patients with moderate and severe disease tended to be older, men, Hispanic, smokers, normal weight, and to have lower income and education. Those without periodontitis were more often non-Hispanic white women, never/former smokers, and highly educated. Across NHANES campaigns, prevalence of periodontitis-free participants significantly increased over time. No differences in glycolipid profile, creatinine, and CRP were recorded. Mean SBP gradually increased across categories of disease severity (mild, moderate, and severe). Interestingly, participants with mild disease had lower SBP than those without periodontitis, but unmeasured factors might have accounted for this result.

Association of Periodontitis and Uncontrolled Hypertension

The unadjusted raw mean SBP was about 2.3 mmHg higher in treated hypertensive adults with periodontitis ($n=1834$; 133.43 ± 19.7 mmHg) than in those without the disease ($n=1694$; 131.17 ± 19.5 mmHg; $P < 0.001$). Such difference increased to about 3 mmHg after progressive adjustment ($P < 0.001$; Figure 1; Table 2).

Table 1. Demographic Characteristics of Treated Hypertensive US Adults Aged ≥30 y by Periodontal Disease Severity: National Health and Nutrition Examination Survey 2009 to 2014

Characteristics	Strata	Periodontal Disease				P Value
		None	Mild	Moderate	Severe	
n		1734	105	1370	417	<0.001*
Age, y; mean (SD)		62.60 (12.94)	57.32 (12.81)	64.17 (11.75)	62.64 (10.53)	<0.001
Sex (%)						<0.001
	Men	691 (39.9)	48 (45.7)	692 (50.5)	260 (62.4)	<0.001*
	Women	1043 (60.1)	57 (54.3)	678 (49.5)	157 (37.6)	<0.001*
Race/ethnicity (%)						<0.001
Hispanic	Mexican American	122 (7.0)	12 (11.4)	139 (10.1)	76 (18.2)	<0.001*
	Other Hispanic	133 (7.7)	7 (6.7)	122 (8.9)	31 (7.4)	1*
Non-Hispanic	Non-Hispanic white	891 (51.4)	49 (46.7)	554 (40.4)	114 (27.3)	<0.001*
	Non-Hispanic black	466 (26.9)	31 (29.5)	420 (30.7)	158 (37.9)	0.001*
	Multiracial	122 (7.0)	6 (5.7)	135 (9.9)	38 (9.1)	0.129*
SBP, mean (SD)		131.17 (19.54)	128.14 (19.19)	133.56 (19.14)	134.35 (21.32)	<0.001
DBP, mean (SD)		69.58 (13.88)	72.90 (12.37)	69.67 (14.32)	71.10 (14.73)	0.031
Cholesterol, mean (SD)		187.89 (44.34)	192.59 (41.56)	186.64 (42.22)	186.22 (42.33)	0.503
Triglycerides, mean (SD)		162.81 (106.95)	190.19 (172.88)	170.07 (127.16)	170.50 (134.74)	0.078
HDL, mean (SD)		52.03 (15.93)	50.88 (15.20)	51.19 (15.73)	50.89 (17.51)	0.404
CRP, mean (SD)		0.55 (0.84)	0.61 (0.73)	0.46 (0.71)	0.48 (0.62)	0.2
Smoking status (%)						0.001
	Smoker: current/former	798 (46.0)	41 (39.0)	700 (51.1)	235 (56.4)	<0.001*
	Never smoker	936 (54.0)	64 (61.0)	669 (48.8)	182 (43.6)	<0.001*
Current or former smoker (%)						0.001
	Everyday	216 (27.1)	8 (19.5)	188 (26.9)	88 (37.4)	0.017*
	Some days	33 (4.1)	4 (9.8)	24 (3.4)	16 (6.8)	0.149*
	Quitted	549 (68.8)	29 (70.7)	488 (69.7)	131 (55.7)	0.002*
Poverty level (%)						<0.001
	<100%	307 (19.4)	10 (10.5)	241 (19.4)	100 (27.0)	0.003*
	100%–200%	428 (27.0)	24 (25.3)	377 (30.3)	118 (31.8)	0.571*
	201%–300%	230 (14.5)	20 (21.1)	221 (17.8)	49 (13.2)	0.126*
	301%–400%	171 (10.8)	13 (13.7)	152 (12.2)	33 (8.9)	1*
	>400%	447 (28.2)	28 (29.5)	254 (20.4)	71 (19.1)	<0.001*
Education (%)						<0.001
	<High school	480 (27.7)	18 (17.1)	403 (29.4)	165 (39.6)	<0.001*
	High school	414 (23.9)	23 (21.9)	339 (24.7)	105 (25.2)	1*
	>High school	837 (48.3)	64 (61.0)	625 (45.6)	147 (35.3)	<0.001*
Age range, y (%)						<0.001
	30–44	184 (10.6)	18 (7.1)	91 (6.6)	19 (4.6)	<0.001*
	45–64	718 (41.4)	54 (51.4)	576 (42.0)	231 (55.4)	<0.001*
	≥65	832 (48.0)	33 (31.4)	703 (51.3)	167 (40.0)	<0.001*
BMI range (%)						0.012
	<18.5 (underweight)	16 (0.9)	0 (0.0)	10 (0.7)	2 (0.5)	1*
	18.5–24.9 (normal weight)	242 (14.2)	6 (5.8)	241 (17.8)	79 (19.1)	0.001*

(Continued)

Table 1. Continued

Characteristics	Strata	Periodontal Disease				P Value
		None	Mild	Moderate	Severe	
	25–29.9 (overweight)	571 (33.4)	35 (33.7)	428 (31.6)	132 (31.9)	1*
	≥30 (obese)	879 (51.5)	63 (60.6)	676 (49.9)	201 (48.6)	0.539*
HbA1c range (%)						<0.001
	<5.7%	642 (38.7)	31 (30.7)	414 (31.6)	125 (31.3)	0.001*
	5.7%–6.4%	662 (39.9)	46 (45.5)	562 (42.8)	160 (40.1)	0.897*
	≥6.5%	357 (21.5)	24 (23.8)	336 (25.6)	114 (28.6)	0.02*
Diabetes mellitus diagnosis (%)						0.185
	Yes	447 (25.8)	32 (30.5)	415 (30.3)	129 (30.9)	0.061*
	No	1208 (69.7)	67 (63.8)	886 (64.7)	271 (65.0)	0.051*
	Borderline	78 (4.5)	6 (5.7)	69 (5.0)	17 (4.1)	1*
Creatinine, mean (SD)						0.686
NHANES campaign (%)						<0.001
	2009–2010	533 (30.7)	81 (77.1)	479 (35.0)	154 (36.9)	<0.001*
	2011–2012	538 (31.0)	14 (13.3)	444 (32.4)	146 (35.0)	0.001*
	2013–2014	663 (38.2)	10 (9.5)	447 (32.6)	117 (28.1)	<0.001*

χ^2 test was used for comparing categorical data and *t* test for the continuous variables. BMI indicates body mass index; CRP, C-reactive protein; DBP, diastolic blood pressure; HbA1c, glycohemoglobin A1c; HDL, high-density lipoprotein; NHANES, National Health and Nutrition Examination Survey; and SBP, systolic blood pressure.

*Bonferroni corrections.

Periodontal disease was significantly associated with about 20% higher risk of unsuccessful antihypertensive treatment compared with the absence of the disease, except when CRP was included in the model (odds ratio, 1.19; 95% CI, 0.91–1.54; $P=0.205$; Table 3).

Quartiles strata were identified at 1.07, 1.53, 2.01, and 6.03 mm for PD and at 1.02, 1.56, 2.35, and 11.33 mm for CAL. Significant higher odds of uncontrolled BP were only observed among patients in the higher quartile of periodontal scores compared with those in the lower quartile (Table 4).

Subgroup Analysis by Age Group, Race/Ethnicity, Sex, and Disease Severity in Treated Hypertensive Patients

Stratified analysis by age groups confirmed a poorer SBP control across all age ranges in treated hypertensive patients with periodontitis (Δ SBP according to model 2: 30–44 years of age: 2.05 mm Hg, $P<0.0001$; 45–64 years of age: 2.30 mm Hg, $P<0.0001$; ≥65 years of age: 2.50 mm Hg, $P<0.0001$; Table S1 in the [online-only Data Supplement](#)).

According to race/ethnicity, non-Hispanic whites had the best, whereas non-Hispanic blacks had the worst, SBP profile compared with the other groups independent of periodontal status (Table S2) and age (data not shown). The analysis by sex confirmed the statistical difference in terms of mean achieved SBP in treated hypertensive men and women with or without periodontitis (Table S3). The analysis by periodontal disease severity showed that participants with moderate-to-severe disease had poorer BP control than those with mild disease (Table S4).

The curve of PD—a measure of acute illness—was J shaped below the age of 45 years, when the highest scores

were recorded; showed a gradual rise with SBP in the age range of 45 to 64 years; and was flat >65 years of age. Indeed, the same curve for CAL—a measure of chronic illness—was J shaped <45 years of age, when the lowest scores were recorded; showed a steep rise with SBP in the age range of 45 to 64 years; and had a progressive, gradual rise with SBP above the age of 65 years (Figure 2A and 2B).

Among treated Hispanics, the curve of PD showed an inverted U shape and was set well >1.5 mm of PD, whereas it was flat below the same value among non-Hispanics (Figure 2C). The same curve for CAL was less steep and showed an almost parallel behavior by ethnicity, with Hispanics showing about 0.2 mm greater CAL than non-Hispanics (Figure 2D). No difference in serum CRP by periodontal status was observed overall or across subgroups stratified by race and age ranges (Table S5).

Untreated Hypertensive Participants

Among untreated hypertensive patients ($n=460$), mean SBP was 2.8 to 7.6 mm Hg higher in the presence ($n=229$) than in the absence ($n=231$) of periodontitis, depending on adjustments (Table S6). Interestingly, there was no statistical difference in mean SBP between treated hypertensive adults with periodontitis and untreated participants without the disease ($P=0.669$). When the prevalence of having BP below or above the BP threshold of 130/80 mm Hg was examined, no difference according to periodontal disease was recorded in untreated patients ($P=0.552$), unlike what was observed among treated patients ($P=0.007$), where a greater proportion of participants with periodontitis was above threshold (Table S7).

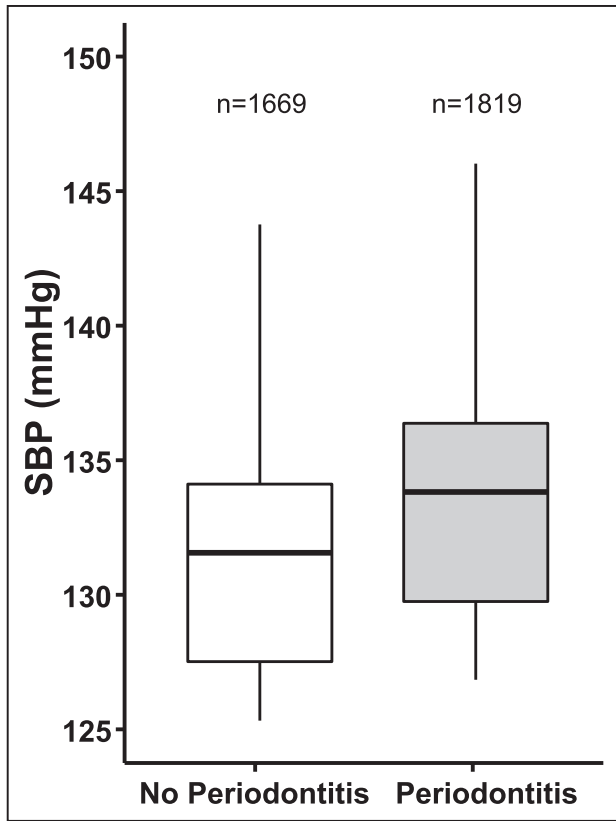


Figure 1. Box plots of systolic blood pressure (SBP) in treated hypertensive US adults aged ≥ 30 y with and without periodontitis: National Health and Nutrition Examination Survey 2009 to 2014. Data are adjusted by model 2.

Discussion

The results of our analyses on treated hypertensive participants in NHANES 2009 to 2014 campaigns who had complete periodontal evaluation show that periodontal disease is significantly associated with the worst SBP profile during antihypertensive therapy by about 2.3 to 3 mm Hg and with higher odds of unsuccessful antihypertensive treatment. This finding was independent of sex and persisted across age ranges and racial/ethnic subgroups, especially Hispanics. Interestingly,

treated adults with periodontitis achieved a mean SBP that was similar to that of untreated adults with good oral health. Taken together, these data suggest that antihypertensive therapy in the presence of periodontitis might not be as effective as in the absence of the disease, with an achieved SBP at best equal to, but not lower than, what was observed in the absence of periodontitis. Moreover, it seems that the severity of periodontal disease affects the odds of treatment failure. The results observed in the comparison group agree with our hypothesis. In fact, mean SBP among untreated hypertensives was about 2.8 to 7.6 mmHg higher in patients with periodontitis. Antihypertensive treatment reduces this spread, which, however, remains significant, suggesting that periodontitis might have detrimental effects on the efficacy of antihypertensive treatment.

Our findings are consistent with previous data reporting on the association of periodontitis with raised BP^{11,34,39} and add information of interest to the available literature on this topic. In particular, our results highlight the association of a good oral health not only with a better BP profile but also with successful antihypertensive treatment. A potential explanation for this is in the proinflammatory environment that characterizes the onset and progression of periodontal disease. As widely known, inflammation represents a cornerstone for the pathogenesis of cardiovascular diseases. It has been reported that the total surface area of inflammation in the presence of periodontitis can be estimated to equal the size of the palm of one’s hand.^{40,41} Such a large area of chronic inflammation reasonably dismisses large amounts of inflammatory mediators into the bloodstream, determining progressive vascular damage that affects cardiovascular health.^{42,43} The underlying mechanism is that inflammation can contribute to endothelial dysfunction, with consequent impaired vasodilation ultimately leading to alterations in the vascular structure: in agreement with this, meta-analytic data suggested a beneficial effect of periodontal treatment on endothelial function.⁴⁴ Low-grade bacteremia and endotoxemia, accumulation compounds formed under oxidative stress, as well as cross-reactivity or molecular mimicry between bacterial and self-antigens, have also been regarded as additional mechanisms potentially linking periodontal disease to systemic diseases.⁴⁵⁻⁴⁹ We did not observe significant differences in CRP by periodontal status;

Table 2. SBP Means (SD) of Treated Hypertensive Patients Stratified by the Presence/Absence of Periodontal Disease Are Presented for Each Model

Models	Periodontitis		No Periodontitis		Δ SBP	P Value
	n	SBP (SD)	n	SBP (SD)		
Model 1	1834	133.43 (19.7)	1694	131.17 (19.5)	2.26	<0.001
Model 2	1819	133.44 (4.6)	1669	131.08 (4.6)	2.36	<0.001
Model 3	1712	133.40 (5.4)	1558	130.89 (5.5)	2.51	<0.001
Model 4	1563	133.49 (5.6)	1430	131.09 (5.9)	2.40	<0.001
Model 5	592	133.18 (6.2)	434	130.12 (6.4)	3.06	<0.001

Mean SBP was used as the continuous dependent variable in GAMs. Model 1 is the crude model reporting raw SBP means according to the presence or absence of periodontitis. The crude model was progressively adjusted adding independent variables: age range, sex, ethnicity, BMI ranges, and smoking status (model 2); HbA1c ranges, total cholesterol, HDL cholesterol, triglycerides, and creatinine (model 3); education and poverty level (model 4); and CRP (model 5). Differences in mean SBP (Δ SBP) according to periodontal status and relative P values are also shown. BMI indicates body mass index; CRP, C-reactive protein; GAM, generalized additive model; HbA1c, glycohemoglobin A1c; HDL, high-density lipoprotein; and SBP, systolic blood pressure.

Table 3. Association Between Periodontitis and Unsuccessful Antihypertensive Treatment

Models	OR (95% CI)	n	P Value
Model 1	1.20 (1.05–1.37)	3512	0.008
Model 2	1.20 (1.04–1.37)	3472	0.011
Model 3	1.21 (1.05–1.39)	3256	0.010
Model 4	1.20 (1.03–1.39)	2981	0.021
Model 5	1.19 (0.91–1.54)	1021	0.205

The ORs and relative 95% CI were calculated using logistic GAMs. The dependent variable was dichotomized to identify treated hypertensive participants at goal or above goal as defined by 2017 ACC/AHA hypertension guidelines. ACC indicates American College of Cardiology; AHA, American Heart Association; GAM, generalized additive model; and OR, odds ratio.

however, chronic periodontitis has a relapsing-remitting behavior, with fluctuations in bacterial burden, inflammatory response, and tissue destruction,⁵⁰ which may explain our finding.

Our observation that PD scores—a measure of current periodontal inflammation—are more related to SBP in younger age, whereas CAL scores, which rather express historical periodontitis, have a steeper relationship with SBP at later age, further supports the concept of a relationship between hypertension and periodontitis mediated by inflammation. In fact, both clinical scores express a proflogistic environment, which is in its early, reversible phases in the case of PD and, conversely, at advanced, long-lasting stages in the case of CAL. According to the natural history of periodontitis,⁵¹ CAL is the result of persistent injury; therefore, it increases with age, whereas PD may reflect acute inflammatory events of the gingiva that may occur since the youth. Thus, it is possible that the specific pathophysiologic events behind each measure, all culminating in an inflammatory burst, may explain the observed relationship with increases in SBP at different ages.

The results by race/ethnicity highlight the greater burden of disease some racial/ethnic groups have to bear.³⁴ In fact, both periodontitis and hypertension seem to particularly

Table 4. ORs for Uncontrolled BP Among Treated Hypertensive Patients in the Higher Quartile (Q4) of Periodontal Scores (PD and CAL)

Periodontal Score	Quartiles	OR (95% CI)	Sample	P Value	Model
PD	Q4	1.26 (1.04–1.52)	3512	0.018	Model 1
	Q4	1.34 (1.10–1.64)	3472	0.004	Model 2
	Q4	1.35 (1.09–1.66)	3256	0.005	Model 3
	Q4	1.37 (1.10–1.70)	2981	0.005	Model 4
	Q4	1.45 (0.99–2.11)	1021	0.055	Model 5
CAL	Q4	1.26 (1.04–1.52)	3512	0.018	Model 1
	Q4	1.26 (1.04–1.54)	3472	0.021	Model 2
	Q4	1.30 (1.06–1.60)	3256	0.012	Model 3
	Q4	1.29 (1.04–1.60)	2981	0.021	Model 4
	Q4	1.26 (0.87–1.83)	1021	0.212	Model 5

The ORs of having uncontrolled BP were calculated using the first quartile (Q1) as reference. No statistical differences were observed for Q2 and Q3 compared with Q1. BP indicates blood pressure; CAL, clinical attachment loss; OR, odds ratio; and PD, probing depth.

affect blacks and Hispanics.^{27,52–55} The combination of the 2 may translate into a particularly unfavorable clinical setting. Given the long-term impact of hypertension on cardiovascular risk and the demonstrated benefit over BP of a good oral health,^{11,56} it seems reasonable that periodontal examination and treatment become part of the therapeutic algorithm in hypertensive patients. Our data suggest that all racial/ethnic subgroups, especially Hispanics, might benefit of such approach. Conversely, ignoring the additional burden of poor periodontal status on BP might translate into a higher cardiovascular risk in the long term. In support of this hypothesis is the observation of an association between history of periodontitis and incidence of cerebrovascular disease,⁵⁷ coronary heart disease,^{58,59} chronic kidney disease,^{60,61} and mortality.^{61,62}

The clinical implications of our findings are particularly interesting when considering that hypertensive individuals frequently need >1 drug to achieve adequate BP values. Besides costs, this might translate into less compliance to treatment.^{20,63,64} Thus, synergic strategies that contribute to BP control, including lifestyle measures and complementary therapies, might translate into potential benefits on the management of the hypertensive patient. As an example, lowering sodium intake by 100 mmol was associated with a 3-mmHg decrease in SBP,⁶⁵ which is similar to our findings, whereas lower sugar intake was observed to translate into a better BP profile by about 3.8 to 7.6 mmHg.^{66–68} Similarly, 2 recent meta-analyses indicated that regular aerobic and aquatic exercise may significantly decrease SBP by 4.7 and 8.4 mmHg, respectively.^{69,70} Thus, together with lifestyle measures, periodontal therapy may contribute to a certain degree to BP lowering, potentially limiting the need of additional drugs.

Another consideration must be done with reference to the long-standing interest of researchers and clinicians for pleiotropic effects of medications, particularly for those additional properties of drugs that seemed to mediate favorable effects on endothelium and inflammation.^{71–75} In fact, there are conflicting results on potential benefits exceeding the simple BP control with the use of the majority of BP drugs.^{76,77} In this perspective, periodontal therapy would represent, when appropriate, a nonpharmacological strategy for the reduction of low-grade systemic inflammation, with related cardiovascular benefits, thus contributing to traditional medical therapy to the achievement of a good global health.

This study has several strengths. To the best of our knowledge, it is the first study examining the magnitude of difference in achieved SBP by periodontal status in treated hypertensive adults. Although the cross-sectional nature of data does not allow any deduction of causality or temporal relationship between the analyzed variables, the finding is of interest for future studies testing the direction of such association. The multiracial composition of the survey allowed a stratified analysis based on ethnicity. Both qualitative and quantitative analyses of periodontitis, based on the most updated definition of the disease, were performed. In addition, data were tested with multiple adjustments. However, this study is not without limitations. First, bleeding on probing—a marker of periodontitis activity that was demonstrated to have the best association with raised BP³⁴—was not assessed in the selected cohorts. As a consequence, despite several mechanisms are thought to be implicated in periodontitis-related systemic effects, our

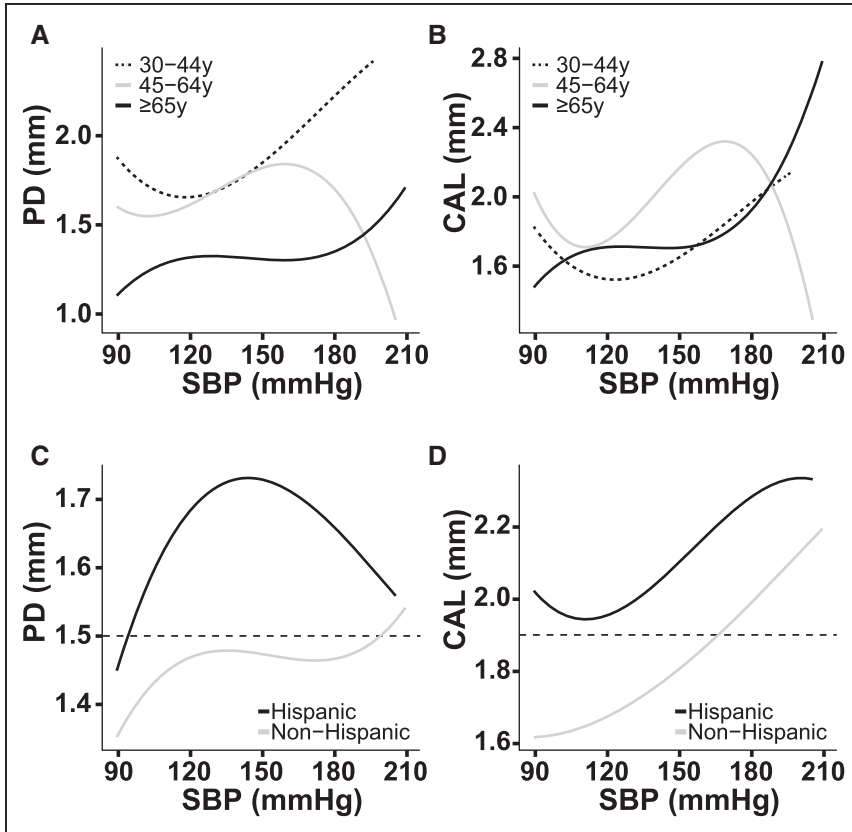


Figure 2. Cubic splines of the relationship between probing depth (PD)/clinical attachment loss (CAL) and systolic blood pressure (SBP) by age ranges (A and B) and ethnicity (C and D). Data are adjusted for model 2.

findings might not be generalized to active disease as assessed by bleeding on probing. Additional information of interest, that is hypertension drug classes, number and dose of medications, appropriateness of antihypertensive agents, adherence to therapy, use of antibiotics, or a history of periodontal treatment, was not available. Similarly, the impact of different degrees of oral health awareness or healthcare access, reflecting disparities in socioeconomic status, should be considered when interpreting the results. The survey excluded institutionalized patients; therefore, a certain degree of selection bias has to be considered. Finally, the analysis of costs/benefits related to periodontal therapy goes beyond the purpose of the present study and deserves specific evaluation in dedicated trials.

In conclusion, a good periodontal health is associated with a better SBP profile during antihypertensive therapy by about 2.3 to 3 mm Hg and with lower odds of treatment failure. Dedicated studies are needed to explore the impact of periodontal therapy on BP in treated hypertensive patients of different racial/ethnic descent and the long-term effects on cardiovascular outcomes of such a complementary approach to systemic health.

Perspectives

Observational data from NHANES 2009 to 2014 cohorts indicate that a good periodontal status is associated with a better SBP profile during antihypertensive therapy by a magnitude of about 2.3–3 mmHg. Low-grade systemic inflammation that is typical of periodontitis might explain this finding because it has been regarded as the mechanism underlying the association of periodontitis with several cardiovascular risk factors and diseases. However, because a causal relationship cannot be inferred from observational data, future studies should focus on the direction

of the reported association and the long-term impact of periodontal therapy on cardiovascular risk factors and outcomes in populations with different racial/ethnic background.

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Disclosures

None.

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Novelty and Significance

What Is New?

- Data on the impact of periodontitis on blood pressure control in treated hypertensive adults are lacking.
- A good periodontal health is associated with a better systolic blood pressure profile during antihypertensive therapy by a magnitude of about 2.3 to 3 mm Hg.

What Is Relevant?

- Low-grade systemic inflammation typical of periodontitis mediates its association with several cardiovascular risk factors and diseases.

- Dedicated studies are needed to test the impact of periodontal therapy as a complementary approach to systemic health.

Summary

Poor periodontal health is associated with the worst systolic blood pressure profile and unsuccessful antihypertensive treatment. Future studies should focus on the direction of this association and the long-term impact of periodontal therapy on cardiovascular outcomes.