

**Beck JD, Offenbacher S. Systemic effects of periodontitis: epidemiology of periodontal disease and cardiovascular disease. J Periodontol 2005; 75:2089-2100. 55 (Refs)**

**Purpose:** To provide a more definitive answer as to whether oral disease is a risk factor or causal for cardiovascular disease (CVD) by clarifying which cardiovascular disease outcomes are affected by oral disease.

**Materials and Methods:** 1391 individuals (age 45-64 years old) randomly selected from Dental Atherosclerosis Risk in Communities Study (Dental ARIC). Clinical measures collected included PD, CAL on six sites for all teeth. Case definition of periodontal disease consists of following three degrees; 1) severe periodontitis: >2 interproximal sites (not on same tooth) with > 6mm CAL and 1 or more interproximal sites with PD >5mm; 2) initial periodontitis: > 2 interproximal sites with 4 or 5mm CAL (not on same tooth); and 3) healthy/gingivitis: the other individuals. Periodontal organisms selected were representatives from Socransky's clusters; *P.gingivalis*, *T. forsythensis*, and *T. denticola* from "red" and *P. intermedia*, *P. nigrescens*, *F. nucleatum*, *Peptostreptococcus micros*, and *Campylobacter rectus* from "orange", and *A.a.* Plaque organisms were measured using DNA checkerboards. The samples were assayed for IgG antibody levels. Distributions of the organisms and antibodies were divided into quartile and then dichotomized as high vs. low. Participants were defined as never smokers, former smokers, or current smokers, and subclassified light (< 20 pack-years) or heavy smokers (>20 pack-years). Age was dichotomized as <65 years or not. Race was either African American or Caucasian. Also diabetes, hypertension and obesity were reported. All collected data were analyzed with "principal component analysis"

**Findings:** The individuals with low organisms and low antibody levels (Q1) were most likely to have a healthy periodontal status. The quadrant characterized by low levels organisms and high antibodies (Q2) contained a higher % of individuals with initial (entry level) periodontitis. The quadrant with high organisms and high antibody levels (Q4) had the highest % of individuals with severe periodontitis. The quadrant characterized by high organism levels and low antibodies (Q3) contained individuals with periodontal characteristics similar to the severe group, but not as severe. The three periodontal conditions each distribute into one of the three types of bacterial-IgG response traits (i.e., Q1=health; Q2=early; and Q4=severe). However, microbial load and the antibody response contribute to the expression of disease, but it is not as highly correlated with these periodontal disease states as it is to other biofilm organism load/antibody response characteristics within each of the four quadrants. With respect to the demographic factors, Q1 is more likely to have individuals who are Caucasian, never smokers, not obese, not hypertensive, and not diabetic. All of these variables are more highly correlated with health than the AB/BUG (antibody/bacterial load) variables. Q2 also is more likely to have individuals who are former smokers, male, and aged >65 and have thick carotid arteries, CHD, or stroke. Significantly, both stroke and a diagnosis of CHD are closer to the Q2 centroid than any periodontal disease variables. Q4 is also characterized as being more likely to contain African-American individuals who are hypertensive, obese, and diabetic. Q3, which has individuals who have reasonably severe periodontal disease, is also

characterized as containing individuals who are female, aged <65, current smokers, but has relatively few individuals with cardiovascular disease. The results indicate four general patterns of biofilm load/antibody reactivity, but the strength of the associations and ability to predict disease are not quantified.

**Conclusions:** The cumulative evidence supports, but does not prove, a causal association between periodontal infection and atherosclerotic cardiovascular disease or its sequelae.

**Brook I, Lewis MA, Sandor GK et al. Clindamycin in dentistry: more than just effective prophylaxis for endocarditis? Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2005; 100:550-8.**

**Purpose:** to review literature on use of clindamycin in dental practice.

**Materials and Methods:** literature review.

**Findings:** Clindamycin has been in use for the last 3 decades. Historically, its limited use has been the concern of pseudomembranous colitis. Its routine use began as it was recommended for infective endocarditis prophylaxis.

Antimicrobial activities: Clindamycin inhibits protein synthesis at bacterial 50s ribosomal subunits. Clindamycin is a wide spectrum antimicrobial that has high level of in vitro activity against a variety of facultative and strictly anaerobes. This spectrum include gram positive, penicillin-resistant Staph. strains, B-hemolytic Strep. strict anaerobes, gram-negative species, B-lactamase producing strains. However, this drug has poor activity against G- facultative organisms including E. corrodens, Haemophilus spp. In addition, Clindamycin also reduces bacterial adherence to epithelial cells of mucosal surfaces and inhibits virulence factors.

Tissue concentration: Clindamycin reaches high concentration in saliva, GCF, and bone. Drug concentration in these tissue had been found to range from 40-50% to 300% of serum concentration. In one study, clindamycin concentration in bone and other was above minimal concentration at which 90% of isolates are inhibited (MIC<sup>90</sup>). High intracellular drug concentrations in tissue enhance opsonization, phagocytosis, and intracellular killing by macrophages and also yield a post-antibiotic effect.

Tolerability: In 3 decades of use, clindamycin had rare adverse/hypersensitivity reactions. Perceived pseudomembranous colitis association was not justified. Recent studies have shown that likelihood of pseudomembranous colitis is extremely low (6.7/100,000) in primary care setting and rarely in treatment of dental infections. Amoxicillin-clavulanate has higher relative risk of developing colitis vs. clindamycin.

Treatment of acute dental infections: In clinical trials involving periapical abscesses, the efficacy of clindamycin (600mg/day) had success rates of 97-100%. In addition, more rapid clinical improvement in pain, fever, and swelling was observed. The current recommendation for clindamycin ranges from 600-1200mg daily (150-300mg QID).

Periodontal disease: clindamycin was shown in 4 trials to have long term benefit for refractory periodontitis with 600mg/day x 7 days. One study had indicated that Clindamycin was superior to tetracycline at one year follow-up.

Peri-implantitis: no clinical trials to assess efficacy of Clindamycin of preventing peri-implantitis. Clindamycin may be an effective agent in peri-implantitis management.

**Conclusions:** according to the authors, a review of literature relating to maxillofacial infections has indicated that Clindamycin is a highly effective anti-microbial agent in dentistry.

**Brown RS, Rhodus NL. Epinephrine and local anesthesia revisited. OOOOE 2005;100:401-8. 74 (Refs)**

**Purpose:** To discuss the relative safety of epinephrine and local anesthetic formulations in patients with hypertension and other cardiovascular diseases.

**Materials and Methods:** Review article.

**Findings:**

- Rationale for combination of vasoconstrictor with local anesthetics: Vasoconstrictors in a local anesthetic are beneficial with regard to the duration, depth of anesthesia, blood loss, and reduction of systemic local anesthetic toxicity. Insufficient vasoconstriction results in inadequate pain control and increased levels of endogenous catecholamines like norepinephrine (NE) which in turn increase blood pressure and other cardiotoxic effects.
- Adrenergic system: Stimulation of Beta 1 receptors increases blood pressure, whereas stimulation of Beta 2 receptors decreases blood pressure. Epinephrine has both beta 1 and beta 2 activity, and hence does not dynamically increase blood pressure. Both epinephrine and NE are alpha-adrenergic agonists and cause vasoconstriction in the peripheral site but have limited systemic effects. Epinephrine has a very short plasma half-life (less than a minute) and is eliminated from the blood stream in approximately 10 minutes or less.
- Toxicity of vasoconstrictors: NE was removed as a vasoconstrictor from most formulations due as it caused rebound bradycardia. Epinephrine is a more potent alpha agonist (peripheral vasoconstrictor) and has a greater beta 2 activity compared to NE and levonordefrin.
- Changes in Mean arterial pressure (MAP): Epinephrine increases heart rate, stroke volume, systolic blood pressure, myocardial oxygen consumption, and cardiac automaticity but reduces diastolic blood pressure. Therefore MAP is relatively unchanged.
- Receptor dynamics: When receptors are exposed to large amounts of agonist they down-regulate and require greater amounts of ligand to produce the same biologic effect (eg: opioid addiction, pheochromocytoma) and hence small amounts of epinephrine have very little effect on these individuals.
- Drug-Drug interactions: Interactions between epinephrine and antidepressants like TCAs and MAOs have not been proven. There have been a few reports of interactions between epinephrine and propranolol, antihistamines, cocaine, general anesthetics.
- Vasoconstriction in patients with severe cardiac disease: The New York Heart Association recommends a maximum of 0.2 mg of epinephrine (<11 cartridges of 1:100,000 epinephrine) be used at 1 session in dental patients with heart disease always preceded by aspiration. Other studies show that 2-3 cartridges of 2% lidocaine with 1:100,000 epinephrine (36-54 µgms) appears to be well tolerated by most patients with hypertension and cardiovascular disease, and the benefits outweigh the potential disadvantages.

**Conclusions:** There is a relative absence of case reports noting adverse consequences

related to epinephrine in local anesthetics and clinical studies have repeatedly demonstrated their effectiveness and safety in dental patients.

**Cillo JE, Finn R. Correlation and comparison of body mass index on hemodynamics in hypertensive and normotensive patients undergoing intravenous sedation. J oral maxillofac surg 2006; 64:583-88. 32 (Refs)**

**Purpose:** To compare and correlate BMI on hemodynamics in hypertensive and normotensive patients undergoing IV sedation for outpatient dentoalveolar surgical procedures.

**Materials and Methods:** 263 consecutive male patients undergoing IV sedation for dental surgeries were included in this retrospective chart analysis. BMI was divided into 5 groups: underweight (<18.5), normal weight (18.5-24.9), overweight (25.0-29.9), obese (30.0-39.9), and extremely obese (>40). Hemodynamic measurements recorded at baseline and every 5 minutes during the procedure included systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), pulse pressure (PP), and pulse (P). The catheter for IV was placed and the anesthetic agents administered included methyprednisone, cafezolin, midazolam, fentanyl, and propofol. Data were subjected to statistical analysis.

**Findings:** There were no significant differences between the groups in terms of the amount of anesthetic received, time of procedure, or age of patients. Average hemodynamic values for normotensive patients were significantly lower than for hypertensive patients in all groups of BMI, except for pulse in the normal group, pulse in the obese group, and DBP in the extremely obese group. All intraoperative hemodynamic measurements defining percentage change from baseline were insignificant except for SBP, DBP, MAP in the underweight group, PP in the normal group, pulse in the overweight group, pulse and PP in the obese group, and pulse in the extremely obese group. Statistically significant positive correlations were found in both normotensive and hypertensive groups for BMI and baseline hemodynamic changes, except for pulse in hypertensive patients (insignificant moderate negative correlation).

**Conclusions:** Normotensive patients had lower hemodynamic values than hypertensive patients in all BMI groups. There was statistically significant moderate and positive correlation in BMI for changes from baseline for several hemodynamic measurements. The anesthetics and techniques used in this study for IV sedation for dentoalveolar surgeries provided hemodynamic stability for both normotensive and hypertensive groups regardless of BMI.

**Dye BA, Choudhary K, Shea S, Papapanou PN. Serum antibodies to periodontal pathogens and markers of systemic inflammation. J Clin Periodontol. 2005 Dec; 32(12):1189-99**

**Purpose:** To establish the relationship between serum IgG responses to two major periodontal pathogens, *Porphyromonas gingivalis*, *Actinobacillus actinomycetum* and two important inflammatory proteins (CRP & fibrinogen) that have been shown in epidemiological studies to be predictors of atherosclerosis, cardiovascular and ischaemic events.

**Materials and Methods:** Data on 2,973 participants aged 40 years and older from the third National Health and Nutrition Examination Survey, second phase (1991-1994) were used. Three logistic regression models adjusted for gender, race, educational attainment, diabetes, cigarette smoking, body mass index (BMI), and other inflammatory conditions were constructed, based on three different assumptions: (A) no access to dental/periodontal data; (B) knowledge of number of teeth present but not of clinical periodontal status; and (C) knowledge of both dental and clinical periodontal status

**Findings and Conclusions:** The regression analysis showed that an elevated IgG titre to Pg is independently associated with high serum CRP. High CRP (>0.4 mg/dl) was related to high antibody levels to *P. gingivalis* in models A [odds ratios (OR) 1.63, 95% confidence intervals (CI) 1.15-2.32], B (OR 1.69, 95% CI 1.18-2.41), and C (OR 1.58, 95% CI 1.12-2.23). In model C, high CRP was related to >30% extent of attachment loss of  $\geq 3$  mm (OR 1.58, 95% CI 1.19-2.08). In contrast, titres to Aa were not associated with CRP levels, and neither titer was associated with plasma fibrinogen. The association of antibody response to Pg and high CRP provides support to the notion that periodontal infections may contribute to systemic inflammation and thereby to atherosclerosis.

**Faria-Almeida R, Navarro A, Bascones A. Clinical metabolic changes after conventional treatment of type 2 diabetic patients with chronic periodontitis. J Periodontol 2006; 77:591-8. 46 (Refs)**

**Purpose:** To perform a clinical and metabolic comparison of the response to conventional periodontal treatment between type 2 diabetic patients and non-diabetic patients.

**Materials and Methods:**

Inclusions criteria	Exclusion criteria
Age of 35 to 70 years Presence of type 2 diabetes Generalized moderate chronic periodontitis (CAL 4-6mm, radiographic bone loss of 30-50%) Presence of $\geq$ 10 teeth/arch excluding third molars No previous periodontal treatment No modification in medication in the 2 mons before or during the study	Presence of systemic disease that could influence the course of the periodontal disease Intake of antibiotics or anti-inflammatories in the 4wk before the study Current smokers or former smokers Pregnancy or intention to become pregnant

The test group consisted of 10 type 2 diabetic patients. The control group included 10 nondiabetic patients. An initial examination included HbA1c and glucose level, periodontal examination (PII, BOP, PD, gingival recession, and CAL), oral hygiene instruction and supragingival prophylaxis. Four sessions of scaling and root planing were performed. Follow-up examinations were done at 3 and 6 months including all the elements of the initial examination. Statistical analysis was performed.

**Findings and Conclusions:** No statistically significant differences were found between the control and the test groups. The groups did not show significant difference in PII, BOP, gingival recession and CAL at any examination time. All variables showed improvement following scaling and root planing. Statistical significant differences in PDs were observed between the control and the test groups at all times. Results showed a positive metabolic response to the periodontal treatment with a lowering of HbA1c values at each follow-up. The test group showed a significant difference in this variable between baseline and 3 months and between baseline and 6 months. However, lowered values for blood glucose did not reach statistical significance.

**Ficarra G, Beninati F, Rubino I, et al. Osteoradionecrosis of the jaws in periodontal patients with a history of bisphosphonate treatment. J Clin Periodontol 2005; 32:1123-28. 30 (Refs)**

**Purpose:** To report nine consecutive cases of osteonecrosis of the maxilla and mandible in patients with disseminated hematological neoplasms and metastatic bone diseases who were receiving intra-venous bisphosphonate therapy.

**Materials and Methods:** Nine consecutive patients with severe periodontal disease and tooth loss who had been treated for disseminated hematological neoplasms or metastatic bone diseases and presented with jaw osteonecrosis were followed prospectively. Medical history, drugs received, anatomic locations and areas of osteonecrosis, relationship between previous extractions of periodontally involved teeth and presence or absence of oroantral fistulas were systematically documented. Radiographic and histopathological examinations were done on all patients. All patients were treated with conservative debridement of bone sequestra, local irrigation with providone-iodine and daily rinsing with 0.12% chlorhexidine mouthwash, oral nimesulide and prolonged antibiotic therapy with penicillin-type antibiotics. This treatment was repeated in case of recurrence of infection.

**Findings:** All patients showed lesions on the mandible and 2 of them also had lesions on the maxilla. In all patients tooth extraction preceded the onset of osteonecrosis. In 2 patients lesions occurred in multiple areas and also in distant bone sites that followed extractions. Symptoms included necrotic infected bone, pain, difficulty in eating, speaking, lower-lip paresthesia and in some cases trismus, halitosis and recurrent abscesses. In case of posterior maxillary involvement, chronic maxillary sinusitis and oroantral fistulas were also evident. The duration of bisphosphonate therapy at presentation of jaw osteonecrosis ranged from 10-70 months (median = 33months). All specimens revealed areas of necrotic bone and fibrinous exudates accompanied by a polymorphonuclear leucocyte infiltrate. Six patients had reached a pain free state by the end of the third month due to antibiotic treatment.

**Conclusions:** Conservative treatment with antibiotics and debridement of bone sequestra seems more appropriate in the treatment of osteonecrosis.

**Genco RJ et al. A proposed mode linking inflammation to obesity, diabetes, and periodontal infections. J Periodntol 2005; 76:2075-2084.**

**Purpose:** To examine the relationship between obesity and periodontal disease and to evaluate to what extent insulin resistance and associated systemic TNF-alpha levels and sTNF-alpha receptors may account this relationship.

**Materials and Methods:** Data from non diabetic patients who were part of the Third National Health and Nutrition Examination Survey (NHANES III) were used in this study. Data gathered included, fasting insulin levels, fasting glucose levels, serum cholesterol level, serum triglycerides levels, Glycated hemoglobin, C-reactive protein (CRP), and height and weight of the patient from which the Body Mass Index (BMI) was calculated. Periodontal assessments included measurements of gingival bleeding, calculus, probing depth, and attachment level carried out at two sites (buccal and mesiobuccal of two randomly selected quadrants, one upper and one lower. Individuals mean attachment levels (AL) of each subject was calculated and periodontitis was defined as  $AL \leq 1.5$ . A subset of the university at Buffalo myocardial infarction in periodontal disease case control study was used, which included 1221 adult controls, representing the highest and the lowest quartile of BMI, were assessed by extensive periodontal and medical examinations.

**Findings:** 12367 non-diabetic individuals (20-90 years of age) participated in the dental section of the (NHANESIII) study. Of these 53.1% were men and 46.9% women. 38.7% were whites, 27.7% were Black, 29.4% were Mexican Americans, and 4.2% were of other racial and ethnic groups. 43.1% of the patients were overweight ( $BMI \geq 27$ ). Over weight individuals were slightly older, less educated, and had a lower income compared to none overweight individuals. A greater portion of females, non Hispanic blacks, and Mexican Americans were over weight compared to those non-overweight. A greater level of attachment loss and more periodontal disease were seen in the overweight group. The severity of periodontal disease increase proportionally with increasing Insulin Resistance(IR). Most of the individuals in the highest quartile can be considered obese, as they exhibited a mean BMI of 31.8. Multivariate analyses revealed that BMI is positively and significantly related to severity of attachment loss. A weighted multiple regression analyses showed that this relationship is likely mediated by insulin resistance. Overweight individuals with IR in the highest quartile exhibited an odds ratio of 1.48 for severe attachment loss, whereas this association was not significant for subjects with high BMI and low IR. There was a highly statistically significant elevation of TNF- alpha RI and RII in those individuals with  $BMI > 30.8$  compared to those with  $BMI < 24.6$ . In the quartile with the lowest BMI a modest but statistically significant correlation of TNF alpha levels with periodontal disease was found.

**Conclusions:** Obesity was a significant predictor of periodontal disease and this relationship is suggested to be mediated by Insulin resistance independent of age, gender, race, ethnicity and smoking.

Graves DT, Liu R, Alikhani, Al-Mashat L, Trackman PC. Diabetes-enhanced Inflammation and Apoptosis—Impact on Periodontal Pathology. *J Dent Res* 2006 85: 15-21. 86 (Refs)

**Purpose:** To discuss how diabetes-enhanced inflammation and apoptosis may affect healing and matrix production in the oral environment after bacterial injury.

**Materials and Methods:** Review article.

**Findings:**

- Hyperglycemia observed in diabetes mellitus results from inadequate glucose transport from the vasculature into cells of the liver and muscle. Since both type 1 and type 2 diabetes are negatively affected by the death of beta cells in the pancreas, resulting in inadequate insulin production, the phrases 'insulin and non-insulin-dependent diabetes' are no longer used. Long-term manifestations of diabetes include retinopathy, neuropathy, nephropathy, angiopathy, atherosclerosis, periodontitis, and impaired wound-healing.
- Hyperglycemia-enhanced superoxide production has been linked to damage of vascular cells and may represent a common mechanism for diabetic complications.
- Diabetics have increased susceptibility to infection. Systemic levels of TNF- $\alpha$  and IL-6 are elevated in both type 1 and type 2 diabetes indicating systemic inflammation. Microbial infections such as *Chlamydia pneumoniae*, *Helicobacter pylori*, and periodontal pathogens through a greater response to endotoxemia or bacteremia are an important risk factor for cardiovascular diseases. Diabetes may enhance the inflammatory response to bacteria at both the site of infection and also systemically through a greater response to endotoxemia or bacteremia.
- Human gingival crevicular fluid from type 1 diabetics with periodontal disease has higher levels of both PGE2 and IL-1 $\beta$  and monocytes isolated from periodontal patients with type 1 diabetes produce significantly greater amounts of TNF- $\alpha$ , IL-1 $\beta$ , and PGE2 in response to lipopolysaccharide (LPS). Cytokine dysregulation associated with prolonged TNF expression may represent an important mechanism through which diabetes alters the host response to bacterial challenge.
- Diabetes also leads to greater net periodontal bone loss and contributes to increased risk of tooth loss by impairing the cycle of bone formation that occurs after bone resorption. Diabetes prolongs bacteria-induced apoptosis of bone-lining cells made up of periosteal cells and osteoblasts. Animal experiments showed that the amount of new alveolar bone formation was less than half that of normoglycemic controls and the level of apoptosis of bone-lining cells was much higher in diabetics and treatment with a caspase-inhibitor that blocks apoptosis significantly improved the formation of new bone in diabetic mice.
- Accumulation of advanced glycation end-products (AGEs) is greatly accelerated in chronic hyperglycemia. Treatment with sRAGE (receptor for AGE) decreased the levels of TNF- $\alpha$  and IL-6 in gingival tissue and suppressed alveolar bone loss and tipped the balance between matrix production and degradation toward formation by down-regulating matrix metalloproteinase activity in animal models. There are several mechanisms through which AGEs may affect cell behavior, such as

enhancing inflammation, stimulating apoptosis, or affecting production of extracellular matrix.

- Apoptosis (programmed cell death) occurs rapidly, within an hour of effector caspase activation. Since adequate healing requires sufficient number of cells to repair wounds, enhanced apoptosis of matrix-producing cells reduces tissue formation. Diabetes is associated with activation of polyol pathway, leading to the formation of AGEs and phospholipase C activation, higher levels of TNF- $\alpha$  expression, enhanced protein kinase C activation, and greater oxidative stress. Blocking apoptosis with a caspase inhibitor significantly improves the response to a bacterially induced injury in animals.

**Conclusions:** Healing response is altered in periodontal disease by enhanced production of ROS, TNF, and AGEs which impair wound healing by direct effects on osteoblastic or fibroblastic cells (reduced production of collagen), or indirect effects by promoting inflammation and apoptosis of matrix-producing cells.



**Holm SW, Cunningham LL, Bensadoun E, Madsen MJ. Hypertension: classification, pathophysiology, and management during outpatient sedation and local anesthesia. J Oral Maxillofac Surg 2006; 64:111-121.**

**Purpose:** The purpose of this paper is to review and summarize the new classification system based on the Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation and Treatment of hypertension and to review the guidelines, pathophysiology, clinical symptoms and diagnosis of hypertension.

**Materials and Methods:** Review of the Joint National Committee on the Prevention, detection, evaluation and treatment of hypertension.

**Findings and Conclusions:** For patients older than 50, SBP higher than 140 mm Hg is a much more important cardiovascular risk factor than DBP. The risk of cardiovascular disease doubles with each increment of 20/10 mm Hg above a baseline of 115/75 mm Hg. The lifetime risk of hypertension for patients who are normotensive at age 50 is 99 %. Patients with a SBP of 120 to 139 mm Hg or a DBP of 80 to 89 mm Hg should be considered pre-hypertensive and require changes in life style to prevent an occurrence of a cardiovascular accident. If blood pressure is more than 20/10 mm Hg above the goal blood pressure, thought should be given to utilizing two therapeutic agents, one of which could be a thiazide-type diuretic. The Joint National Committee has proposed three objectives in classifying hypertension and in evaluating patients for hypertension. They are: (1) assessing lifestyle and identifying other cardiovascular risk factors or diseases that may affect the prognosis and help guide treatment protocols; (2) reveal identifiable causes of high blood pressure and (3) assess the damage to target organs and cardiovascular disease. The classification of hypertension according to the Joint National Committee -7 is as follows: Prehypertensive = SBP 120-139 mm Hg, DBP - 80-89 mm Hg  
Stage I hypertension = SBP 140-159 mm Hg, DBP - 90-99 mm Hg  
Stage II hypertension = SBP >160 mm Hg, DBP > 100 mm Hg

Hypertension although mostly a chronic disease may also present in some patient as sporadic increases and decreases in blood pressure and it then termed labial hypertension. A small percentage of patients may manifest with a very rapid increase in blood pressure and such patients are said to have malignant hypertension. Essential or secondary hypertension may be caused by genetic or environmental factors. High salt intake, alcohol, obesity and reduced physical activity have been implicated as major contributing factors. Early signs of per-hypertension include fluctuations in BP and narrowing of the retinal arteries with/without hemorrhage. Symptoms of early hypertension include headache, vision changes, ringing in the ears or tingling of the hands and feet. Late signs may include ventricular hypertrophy, hematuria, proteinuria, heart failure, angina, renal failure or blindness. Renal failure is the leading cause of secondary hypertension. Secondary hypertension may be caused by disease of hormonal imbalances such as primary aldosteronism, Cushing's syndrome and pheochromocytoma. The JNC-7 guidelines for the management of hypertension include single or combination therapy with diuretics,  $\beta$ -blockers, or both for uncomplicated hypertension. The treatment of complicated hypertension may require combinations of medications such as ACE inhibitors, angiotensin II receptor blockers,  $\alpha$ -blockers,  $\alpha/\beta$  blockers,  $\beta$ -blockers, calcium antagonist,

and diuretics. The current recommended dose of local anesthetic for a patient with hypertension is two 1.8 ml cartridges with 1:100,000 epinephrine per appointment. Norepinephrine or levonordefrin should be avoided due to the unopposed activation of  $\alpha_1$  receptors in the hypertensive patient. There are no significant contraindications to the use of benzodiazepines for conscious sedation. Some researches have found that the use of barbiturates is associated with hypotension, cardiac arrhythmias, and bradycardias and therefore these drugs should be used with caution for patients with congestive heart failure. Hypertension does not contradict the use of opioids.

**Janket SJ, Wightman A, Baird AE, Van Dyke TE et al. Does periodontal treatment improve glycemic control in diabetic patients? A meta-analysis of intervention studies. J Dent Res 2005; 84(12):1154-59. 25 (Refs)**

**Purpose:** To review all published evidence systematically and to quantify the impact of periodontal treatment on HbA1c levels.

**Materials and Methods:** Using Medline, Evidence-based Medicine Reviews, and the Cochrane Central Register an internet search was performed for all published articles in English, post-1980 in humans. The authors identified 370 titles and from this pool selected 64 abstracts. All three investigators read these abstracts individually and selected 23 preliminary full text candidate articles. These were then narrowed down to 10 articles which provided the data for this meta-analysis. Statistical test were performed to evaluate publication bias, heterogeneity, and validity. These ten studies involved 456 type 1 and type 2 diabetic subjects.

**Findings and Conclusions:** Analysis revealed a statistically non-significant decrease in HbA1c levels by 0.38% in patients receiving periodontal non-surgical treatment. There appeared to be a correlation between diabetes type and the direction of outcomes. Type 2 diabetic HbA1c levels were more reduced, though not statistically significant, by periodontal treatment than type 1 patient groups.

Table 2. Quantitative Summary of Periodontal Treatment on HbA1C

	<b>Weighted Change in HbA1c</b>	<b>Average 95% Confidence Interval</b>
All Intervention Studies	-0.4%	-1.5, 0.7
Studies of type 2 DM only	-0.7%	-2.2, 0.9
Non-surgical debridement only	-0.4%	-2.1, 1.3
<i>Antimicrobial intervention in type 2 diabetic patients</i>	-0.7%	-2.3, 0.9

This meta-analysis found that periodontal treatment does not improve glycemic control in diabetic patients.

**Katancik J, Kritchesky R, Corby P, et al. Periodontitis and airway obstruction. J Periodontol 2005; 76:2161-7.**

**Purpose:** To evaluate the association between periodontal disease and airway obstruction

**Materials and Methods:** A total of 860 community (white and black, males and females) enrolled in the health, aging and body composition (Health ABC) study, were selected for this cross-sectional trial. The selection criteria included age 70-79 years, self-report of no difficulty walking one-quarter of mile or climbing 10 steps without resting, no difficulty performing basic daily living, no reported need of an walking device, no active treatment of cancer in the prior 3 years, and no plan to move out of the metropolitan or surrounding area. The periodontal evaluation was performed over years 2 and 3 of the Health ABC study and included four indices of periodontal health: Plaque index (PI), Gingival index (GI), probing depth (PD), loss of attachment (LOA). The evaluation of the pulmonary efficiency was conducted in year 1, and was performed according to the American Thoracic Society criteria, which included: Volume/forced vital capacity (FEV1/FVC) ratio and then the percentage of predicted FEV1 was used to categorized the severity of pulmonary obstruction. Airflow limitation was defined by the degree of reduction in FEV1. Severe limitation was defined as a predicted FEV1 below 50, moderate as predicted FEV1 between 50-70% and mild as a predicted FEV1 between 70-100%. In addition, cigarette status and pack/year was also recorded.

**Findings and Conclusions:** Means for all clinical indices for all subjects adjusted by age, race gender and examination site when compared with pulmonary severity showed that those with obstructive disease had significant worse LOA and GI but no differences in PI or PD.

Baseline Pulmonary Classification	N	Mean LOA	Mean GI	Mean PI	Mean PD
Normal	785	2.25 $\pm$ 0.05	0.93 $\pm$ 0.02	0.76 $\pm$ 0.02	2.01 $\pm$ 0.03
Mild obstructive disease	32	2.65 $\pm$ 0.24	0.96 $\pm$ 0.11	0.95 $\pm$ 0.09	2.10 $\pm$ 0.13
Moderate obstructive disease	28	2.70 $\pm$ 0.26	1.19 $\pm$ 0.11	0.93 $\pm$ 0.10	2.36 $\pm$ 0.14
Severe obstructive disease	15	2.90 $\pm$ 0.35	1.21 $\pm$ 0.16	0.75 $\pm$ 0.13	2.13 $\pm$ 0.19

Given the apparent graded association seen in former smokers and pulmonary obstruction, means levels of the periodontal indices adjusted for the former smokers by the level of obstructive disease, showed association of GI and LOA with the severity of obstructive disease. No association was found for PI or PD.

Adjusted (age, race, gender, site, and pack-years) Mean Periodontal Indices ( $\pm$ SE) for Former Smokers According to Level of Obstructive Disease					
Baseline Pulmonary Classification	N	Mean LOA	Mean GI	Mean PI	Mean PD
Normal	348	2.33 $\pm$ 0.77	0.93 $\pm$ 0.03	0.76 $\pm$ 0.03	2.00 $\pm$ 0.04
Mild obstructive disease	17	3.26 $\pm$ 0.36	1.10 $\pm$ 0.15	1.11 $\pm$ 0.13	2.70 $\pm$ 0.17
Moderate obstructive disease	12	3.01 $\pm$ 0.41	1.00 $\pm$ 0.18	0.96 $\pm$ 0.15	2.21 $\pm$ 0.20
Severe obstructive disease	9	3.53 $\pm$ 0.47	1.36 $\pm$ 0.20	0.77 $\pm$ 0.17	2.17 $\pm$ 0.23

For this cross-sectional study it could be concluded that an association existed between periodontal status and airway obstruction, predominantly in former smoker, but not direct cause and effect.

**Lopez NJ, Da Silva I, Ipinza J, Gutierrez J. Periodontal therapy reduces the rate of preterm low weight in women with pregnancy-associated gingivitis. J Periodontol 2005; 76:2144-53. 33 (Refs)**

**Purpose:** The purpose of the article was to address the following questions: 1) is pregnancy-associated gingivitis related to Preterm birth (PT)/and low birth weight (LBW)? And 2) does periodontal therapy reduce the PT/LBW rate in pregnant women with gingivitis?

**Materials and Methods:** The study is a randomized controlled trial that included 870 women with gingivitis who met inclusion criteria including  $\leq 22$  weeks gestation, gingival inflammation with  $\geq 25$  % of sites with bleeding on probing, at least 18 teeth, and no sites with clinical attachment level  $> 2$  mm. All women received a full-mouth periodontal examination. Gingival inflammation, probing depth (PD), clinical attachment levels, and the presence of BOP were measured. Oral hygiene status was assessed as the percentage of surfaces demonstrating plaque. The presence of plaque was positive when a continuous band of plaque was found in contact with the gingival tissue on the cervical portion of mesial, buccal, distal, and lingual tooth surfaces. Demographic factors were obtained and a medical, obstetric, and social history was taken. Information on known risk factors and obstetric factors were collected and included the following: in relation to pregnancy history, the number carried to full term, previous preterm deliveries, low weight births, previous pregnancies aborted, and live births. In relation to the current pregnancy, maternal age at time of study entry, onset of prenatal care, nutritional status, tobacco and alcohol use, use of illicit drugs, sexually transmitted diseases, asymptomatic bacteriuria, urinary infections, vaginosis, or any other maternal infectious disease, number of prenatal visits, intrauterine growth restriction, fetal death, gestational age, and birth weight were recorded. Prenatal care was given to all participants and included the following: blood pressure measurements, urine tests, blood tests, recording of maternal height and weight, and physical and pelvic examinations. At each prenatal visit, an evaluation of nutritional status was done to determine if weight-gain was adequate. Underweight women received supplementary nutrition. Women with symptomatic bacteriuria were treated with nitrofurantoin for 10 days. All women with vaginosis were treated with locally applied antibiotics according to the results of microbiological examinations of vaginal swabs. Randomization was done and the percentage of BOP sites was selected as the variable describing gingivitis. Patients were assigned to one of two categories: those with  $< 50\%$  and  $\geq 50\%$  BOP sites. The women were assigned in a 2:1 manner, with 2 entering the treatment group (and 1 the control. Women in the treatment group received OHI, supra and subgingival scaling, and crown polishing. At the beginning of treatment, each woman was provided with toothbrushes and 0.12% CHX and instructed to rinse once daily until delivery. Periodontal therapy was completed before 28 weeks of gestation, and maintenance therapy was provided every 2-3 weeks until delivery. Women in the control group were monitored two to three times during pregnancy, and repeated periodontal examinations were performed after 30 weeks of gestation to assess changes in periodontal status. Primary outcomes measured were preterm birth and low birth weight. Preterm birth was defined as a delivery before 37 complete weeks of gestation of an infant with birth weight below 2500 g which followed spontaneous labor and/or spontaneous rupture of the

membranes, regardless of route of delivery. An infant born at term with a birth weight less than 2500 g was diagnosed as having intrauterine growth restriction. The delivery date was calculated from the last menstrual period and from ultrasound examination. Women who had a preterm delivery or had a low birth weight infant (LBW) were grouped in the preterm/low birth weight group (PT/LBW) for the analyses of data to identify risk factors.

**Findings:** 834 women with live births completed the study, 553 in the treatment group and 281 in the control group. The mean age was 25.35 years, 15.5 % smoked, and 4.8% had a previous PT/LBW. Women in both groups had poor OH and extensive gingival inflammation, with a mean percentage of BOP sites > 50%. There were 31 PT/LBW infants (24 PT, 7 LBW). Women in the control group had a significantly higher incidence of PT than the treated group. Although the rate of LBW was higher in the control group, the difference was not statistically significant. The incidence of PT/LBW was more than three times higher in the control group. After multivariate adjustment for risk factors for PT/LBW, the only factor significantly associated with PT/LBW was gingivitis.

	<b>Treatment Group: N = 560</b>		<b>Control Group: N = 283</b>		<b>P value:</b>
	<b>N</b>	<b>%</b>	<b>N</b>	<b>%</b>	
<b>PT</b>	8	1.42	16	5.65	0.001
<b>LBW</b>	4	0.71	3	1.15	0.79
<b>PT/LBW</b>	12	2.14	19	6.71	0.002

**Conclusions:** The results of the present study show that eradication of periodontal infection significantly reduces the risk of PT/LBW.

**Madan G, Madan S, Madan G, Madan D. Minor oral surgery without stopping daily low-dose aspirin therapy: A study of 51 patients. J Oral Maxillofac Surg 2005; 63:1262-1265.**

**Purpose:** The purpose of this study was to report results and observations and propose that most minor oral surgical procedures can be performed safely without stopping low-dose aspirin therapy.

**Materials and Methods:** Fifty one patients, 32 of whom were males and 19 females ranging in age from 45 to 79 years participated in this study. All patients included in this study had been taking low dose aspirin therapy for a period ranging from 1 to 15 years. The indications for aspirin therapy ranged from angina to post-bypass surgery. All patients were assessed for values of bleeding time and platelet counts preoperatively. According to the study protocol, if the values were abnormal, patients would stop the aspirin for at least 7 days before the operation and be rescreened for the above mentioned tests. All surgery was performed on an outpatient basis under local anesthesia. The intraoperative blood loss was measured by weighing the swabs and by subtracting the volume of irrigation fluid from the total volume of fluid in the suction jar. Blood loss less than 30 ml was considered not excessive whereas more than 30 ml was considered excessive. Patients were checked 30 minutes after completion of the procedure. Follow up was completed after 24, 48, 72 hours and at 1 week and 2 week intervals.

**Findings and Conclusions:** Preoperative values for platelet count and bleeding time for all patients were within normal limits. The mean bleeding time was 2.86 minutes  $\pm$  0.54 and mean platelet count was  $259 \pm 47.57 \times 10^3/\text{mm}^3$ . Excessive intraoperative bleeding was encountered in only 1 case. This was controlled by soaking gauze in 1% feracrylum solution and applying pressure for 10 minutes.

The authors conclude that minor oral surgical procedures can be safely carried out without stopping long-term low dose aspirin therapy.

**Mattila KJ, et al. Dental infections and cardiovascular diseases: A review. J Periodontol 2005;76:2085-2088.**

**Purpose:** to review the data linking dental infections with CVD, with the emphasis on the most recent findings.

**Materials and Methods:** literature review

**Findings:** Cross sectional and observational studies: The association between periodontitis and CVD is complex and dependent on many factors including the age of the population studied.

Animal studies: repeated intravenous administration of *P.gingivalis* enhanced progression of atherosclerosis in the proximal aortas of apoE-deficient mice.

The results of animal studies must be interpreted with caution, since findings in experimental animals cannot be directly transferred to humans.

Effects of periodontitis on lipids, inflammation, and immune response: Increased attention has been paid to the effect of periodontitis on lipoproteins and inflammation, two major contributors to the pathogenesis of CVD. Recent data suggest that periodontitis may have more subtle but broad effects on the metabolism and properties of lipoproteins that may be reversed by periodontal treatment.

**Conclusions:** Periodontitis may not be confined only to a localized disease process. It has peripheral effects that include proatherogenic changes in lipoproteins and systemic inflammatory and immune responses. Interplay between altered lipoproteins and inflammation is recognized as a key factor in the pathogenesis of atherosclerosis. Periodontitis could bear a significant CVD risk, since it is a long-term disease process with a relatively high prevalence in Western populations and may not always respond to treatment. Observations that the risk is highest in individuals with periodontitis and elevated CRP concentrations and serum antibodies levels to periodontal pathogens may suggest that periodontitis increases CVD risk mostly in individuals who react to these infections by a systemic inflammatory and immune response. This may be due to genetic reasons and may also apply to other chronic low grade infections such as those caused by Herpes viruses and *C.pneumonia*

**Migliorati CA, Casiglia J, Epstein J, et al. Managing the care of patients with bisphosphonate-associated osteonecrosis An American Academy of Oral Medicine position paper. J Am Dent Assoc; 136:1658-68. 55 (Refs)**

**Purpose:** The purpose of this paper was to address the prevention of bisphosphonate-associated osteonecrosis (BON) and the management of care of patients with cancer and/or osteoporosis who are receiving bisphosphonates and who have BON.

**Materials and Methods:** Literature review with author's opinions.

**Findings:**

Background: The oral lesions associated with bisphosphonates are similar to those associated with radiation-induced osteonecrosis. The lesions appear as ragged oral mucosal ulcerations. The underlying bone is exposed and the lesions are extremely painful. The lesions of BON are persistent and do not respond to conventional treatment modalities such as debridement, hyperbaric oxygen therapy or antibiotic therapy.

A Review of Bisphosphonates: Bisphosphonates are synthetic analogues of inorganic pyrophosphate that have a high affinity for calcium. They are cleared from circulation, bind to bone mineral and concentrate in bone. They are potent inhibitors of osteoclastic activity. During bone remodeling, the bisphosphonates are taken up by osteoclasts and internalized in the cell cytoplasm. Bisphosphonates inhibit osteoclastic function and induce apoptotic cell death. Bisphosphonates inhibit osteoblast-mediated osteoclastic resorption. The medication also has antiangiogenic properties thus providing tumoricidal properties. As a result of bisphosphonate use bone turnover becomes suppressed and over time bone demonstrates little physiologic remodeling. Bone becomes brittle and unable to repair physiologic microfractures. Bisphosphonates are utilized to treat Paget's disease of bone, osteoporosis and hypercalcemia of malignancy. It is theorized that in a patient taking a bisphosphonate, the resulting microdamage is not repaired potentially setting the stage for oral osteonecrosis to occur. Bisphosphonate-associated osteonecrosis results from an interplay of bone metabolism, local trauma, increased demand for bone repair, infection and hypovascularity. Patients who use bisphosphonates intravenously are more susceptible to BON than those who take the medication orally. Other comorbid factors may also play a role including the presence of diabetes mellitus, overall tumor burden, extent of skeletal involvement, oral health status, presence of chronic or acute infection, history of head/neck radiation treatment, and the presence of myeloma or metastatic cancer at the BON site. Patients with multiple myeloma may also be taking other antiangiogenic agents such as thalidomide, glucocorticoids, and bortezomib thus setting the stage for a further increase in developing BON.

The Clinical Signs and Symptoms of Bisphosphonate-Associated Osteonecrosis: The most common clinical history associated with patients with oral BON is absent or delayed hard- and soft-tissue healing after dental extractions. Trauma induced by prosthodontic appliances has also been implicated in potentially causing BON. In the early stages of BON, no radiographic manifestations can be observed. Patients may be asymptomatic; however, severe pain may develop due to secondary infection of the site and necrosis of the bone. The osteonecrosis progresses to areas of bony exposure and dehiscence as well as paresthesia. In some patients, BON may develop spontaneously where by far the most common complaint is the sudden presence of roughness that leads to traumatized oral soft tissues surrounding necrotic bone. A diagnosis of BON may be made based on the medical and dental history of each patient as well as the observation of clinical signs and symptoms of the pathologic process of BON. Treatment strategies have included local irrigation with antibiotics, local surgical debridement, bone curettage, and

hyperbaric oxygen therapy. None of these therapeutic procedures was proven successful. The inability to manage BON lesions can compromise the nutritional, oncological and oral management of affected patients.

Treatment Management Recommendations: The treatment of patients using oral and intravenous bisphosphonate therapy is principally preventative in nature. Modifications of the dental treatment plan for a patient taking bisphosphonate medication are necessary to accomplish this goal. Dentists should follow existing guidelines for a dental consultation for the prevention of oral complications of cancer therapy (chemotherapy, radiation therapy, prehematopoietic stem cell transplantation). The elimination of all potential sites of infection is the primary objective of this consultation. The goal of therapy must be to obtain a state of good oral health so that during the active phase of bisphosphonate therapy, only 3 to 6 months of maintenance hygiene appointments will be necessary.

The following modalities should be observed during the consultation:

- a) A comprehensive extraoral and intraoral exam including the appropriate radiographic evaluation should be performed. Diagnosis of periodontal disease and caries, as well as evaluation of 3<sup>rd</sup> molars and the identification of metastatic cancer of the bone are necessary.
- b) For patients who are to begin bisphosphonate therapy, pocket elimination may be necessary to reduce plaque accumulation, minimize chronic periodontal inflammation and acute periodontal infections.
- c) Extraction of teeth should be completed prior to bisphosphonate therapy.
- d) Restorative dentistry should also be performed prior to bisphosphonate use, as well as prophylaxis and oral hygiene instruction.

For patients who have BON the following recommendations are provided:

- a) Routine restorative care may be provided under local anesthesia.
- b) Scaling and root planing as well as prophylaxis should be performed atraumatically.
- c) Avoid dental extractions unless the teeth have a mobility score of 3 or greater. Extraction should be performed atraumatically. Patient follow-up should be weekly for the 1<sup>st</sup> 4 weeks afterward and then monthly until the sockets are completely closed and healed. Antibiotic use may be indicated with the use of amoxicillin alone or in combination with clindamycin.
- d) Teeth with extensive caries may be endodontically treated and restored as overdenture abutments. This may be indicated for patients in whom a previous extraction had resulted in BON.
- e) The area of BON should be treated only with the objective of eliminating sharp edges of bone that may result in traumatized soft tissue. If the area surrounding the exposed bone exhibits tender erythema and suppuration and/or sinus tracts, the patient should be treated with antibiotics until the areas resolve. Chlorhexidine mouthwash may also be employed in order to reduce the bacterial load.
- f) Surgical debridement may be performed for patients with multiple myeloma who require hematopoietic stem cell transplantation.
- g) Soft vinyl appliances or obturators may be used to cover exposed necrotic bone to prevent further trauma to the soft tissues.
- h) Prosthetic devices may be relined with a soft liner in order to minimize soft-tissue trauma.
- i) Odontogenic infections should be treated aggressively with amoxicillin and/or clindamycin.

There are no prospective scientific studies to support specific recommendations regarding whether providing dental treatment for patients taking bisphosphonate medication places the patient at any risk of developing BON. There have only been a few cases of BON in patients taking alendronate and it was unknown if they had local comorbid factors. The risk for developing BON after implant placement, dental extractions and periodontal or other surgical procedures for patients taking oral bisphosphonates is currently unknown. There is also no scientific evidence to support discontinuation of bisphosphonate therapy to promote healing of necrotic osseous tissues. The half-life of intravenous bisphosphonates is

estimated to be of year's duration and cessation of bisphosphonate therapy may not be effective due to the fact that the medication may be incorporated into the bone.

**Conclusions:** According to this paper, prevention of BON is the primary approach to the management of this complication. Existing guidelines in treating patients who receive radiation or chemotherapy may be employed in treating patients who are at risk of BON. Communication between the physician and dentist is of paramount importance in treating patients who are on bisphosphonate therapy as well as patients who present with BON.

**Offenbacher S, Beck JD, A perspective on the potential cardioprotective benefits of periodontal therapy. JAHJ 2005; 149:950-54. 18 (Refs)**

**Purpose:** To summarize briefly what is known regarding the pathobiology of the oral infection-cardiovascular disease connection and why periodontal disease merits further study as a potentially new cardioprotective therapy.

**Materials and Methods:** Review article.

**Findings and Conclusions:**

Periodontal disease is a common biofilm infection that disseminates systemically: The chronic and cyclic nature of periodontal disease provides opportunity for repeated hematogenous dissemination of periodontal pathogens and direct exposure of the vasculature and liver to oral microbes and oral endotoxins. Periodontal pathogens are largely gram -ve and are tissue invasive and cause repeated bacteremias and endotoxemias. Periodontal pathogens are present in atherosclerotic plaques. Thus, repeated exposure of the vasculature to these pathogens provides a clear opportunity for endothelial inflammatory activation and functional impairment.

Periodontitis increases systemic inflammation as measured by serum (C-reactive protein) CRP and IL-6: Important infectious and inflammatory stressor that can increase the hepatic synthesis of CRP, which is a recognized predictor of acute MI and stroke. Raised CRP levels have been reported in people with extensive periodontal disease. Extensive periodontal disease and BMI are jointly associated with increased CRP levels in otherwise healthy middle-aged adults, suggesting the need for joint medical and periodontal evaluation. Studies have shown an association of periodontal disease with both increased CRP and impaired endothelial function as measured by brachial flow-mediated dilation. Studies have found increased serum IL-6 among periodontitis subjects with allele 2 for functional polymorphisms in IL-1A, TNF $\alpha$ , and IL-6 genes, suggesting that host genotype plays a role in increased deleterious effect on cardiovascular health.

Periodontal therapy decreases systemic inflammation and improves endothelial function: Scaling and root planing significantly reduced CRP levels and IL-6 levels. NSAIDs also reduced CRP levels.

**Wactawski-Wende J et al. The association between alveolar crestal height and osteoporosis in postmenopausal women. J Periodontol 2005; 76:2116-24.**

**Purpose:** to assess the cross-sectional association between alveolar crestal heights (ACH) and skeletal bone density in post menopausal women.

**Materials and Methods:** 1341 postmenopausal women (53-85 y.o) completed physical and oral exams, and bone density assessed and completed study questionnaires. Subjects were evaluated for bone density and ACH. ACH is defined as distance from CEJ to crestal height on oral radiographs. The average bone height loss in all teeth was calculated as mean ACH. (The larger ACH=the worse bone loss surrounding teeth). Bone density (BMD) was determined by dual energy x-ray absorptiometry, with severity determined by worst T score measured ( normal > -1.00; low -1.00 to -2.00; moderate -2.01 to -2.49; osteoporotic <-2.5). T score is the number of standard deviation BMD is above or below average peak bone mass for each skeletal site measured for race and gender.

**Findings:** The low, moderate, osteoporotic groups had odds of worse ACH increased by 39%, 59%, 230% respectively, compared to normal T-score subject group. After adjusting for weight, education, hormone use, Ca or Vitamin D supplementation, and smoking, above findings remained consistent. Further, the osteoporotic group increased 1.9fold upon age adjustment and became the worst ACH loss group. The result also showed the worse ACH loss with women older than 70 years of age was increased 2.5-4.6folds with decreasing T-score. After adjustment for all factors and age, odds ratio (CI 95%) for greater ACH loss were 2.66[1.12-6.29] for the low group, 2.31[0.89-6.01] for the moderate group, 3.57[1.42-8.97] for the osteoporotic group.

**Conclusions:** the study data supports a positive association between worsening DEXA T score and loss of alveolar crestal height of postmenopausal women. Age was a modifier in this association.