

Passive Smoking and Alveolar Bone Density

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Abstract: The aim of this study was to study the influence of passive smoking on the alveolar bone density and to what extent bone loss and bone resorption occur. The study included 60 females aged from 30 to 45 years with mean age 37.7 years; the examinees were divided into two groups according to exposure to passive smoking. The first group (control) included thirty subjects not exposed to passive smoking. The second group (passive smokers group) included thirty subjects exposed to passive smoking at home or at work. Digital panoramic radiographs were taken for all the subjects. The radiodensitometric measurements in the three lower dental arch segments were performed for all the radiographs using the Digora soft ware system. Results showed that the clinical attachment loss (M_1) and the values of radiometric measurements measured from cemento-enamel junction to alveolar crest (alveolar bone loss, M_2) were higher in passive smoker group than control group while alveolar radiodensity measurements (D_1 , D_2 , D_3) were higher in control group than passive smokers group ($P < 0.01$). In conclusion, exposure to passive smoking is destructive and leads to decrease bone density and height of supporting structure of teeth.

Key words: Bone density - Panoramic radiograph - passive smoking

INTRODUCTION

Over the past 30 years, a growing body of scientific evidence has concluded that passive smoking can harm the health of non-smokers (Armstrong, 1987). Passive smoking (also known as second hand smoking, involuntary smoking, and exposure to environmental tobacco smoke) occurs when smoke from one person's burning tobacco product or the smoker's exhalation is inhaled by others. In 1987, Armstrong stated that the process of smoking produces three different types of tobacco smoke: Mainstream smoke (smoke directly inhaled by the smoker through a burning cigarette); Exhaled mainstream smoke (smoke breathed out by the smoker) and Side stream smoke (smoke which drifts from the burning end of a cigarette). Although, the passive smokers don't receive the same concentration of toxic chemicals as active smokers, the current scientific epidemiological studies demonstrated that passive smoking contains greater amount of ammonia, benzene, carbon monoxide, nicotine and some carcinogens from the same amount of burnt tobacco. They considered that, the passive smokers as the major preventable risk factor in the incidence and progression of many of health problems associated with direct smoking (US Department of Health and Human Services, 1986), (Bergstrom and Perber, 1994); (WHO Framework Convention on Tobacco Control; California Environmental Protection Agency, 2005). Chemicals from tobacco smoke can be absorbed into the bloodstream of pregnant women. Nicotine, carbon monoxide and other chemicals can cross the placenta affecting her unborn child (US Dept of Health and Human Services, 1986 and National Cancer Institute, 1999). Babies born to women exposed to passive smoking are more likely to have a slightly lower birth weight than they would otherwise (National Health and Medical Research Council, 1997; National Cancer Institute, 1999 and WHO, 1999). This would not necessarily adversely affect a healthy baby, but could further compromise a baby with other problems (National Cancer Institute, 1999 and WHO, 1999).

A correlation between smoking and the severity of periodontal pathology has been confirmed by many authors which may be explained by the action of nicotine present in tobacco products triggers the overproduction of cytokines in the body due to lowered oxygen levels. Moreover, when nicotine combines with oral bacteria, it results in formation of higher levels of cytokines leading to breakdown in the supporting tissues of the teeth (Axelsson *et al.*, 1998; Bergstorm *et al.*, 2000a; Khalaf *et al.*, 2005).

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Even in patients with good oral hygiene, smoking has a negative effect on bone metabolism. It was suggested that the combination of smoking and low systemic bone density negatively affects alveolar bone height and density (Hildebolt *et al.*, 2000; Payne *et al.*, 2000). One explanation for this finding is that, the Estrogen deficiency is associated with elevation with elevation of interleukin 1(IL-1), interleukin 6(IL-6), and tumor necrosis factor α (TNF α) which may affect both alveolar and systemic bone status (Pacifci, 1996; Morishita *et al.*, 1999). Tappia *et al.*, 1995 reported that the smokers have significantly higher plasma level of interleukin 1(IL-1), and tumor necrosis factor α (TNF α) than non smokers.

Radiographic analysis of the alveolar bone heights yield to that smoking is a risk factor for periodontal health and the progression of bone loss is significantly retarded in individuals who gave up smoking (Bergstrom and Eliasson, 1987; Bergstrom *et al.*, 1991; Bolin *et al.*, 1993; Bergstrom *et al.*, 2000a; Chen *et al.*, 2001).

Tobacco components have been shown to have direct effects on certain bone resorptive mediators. The combination of nicotine and bacterial lipopolysaccharide (LPS) increased Prostaglandin₂ (PGE₂) secretion by peripheral monocytes (Tappia *et al.*, 1995; Payne *et al.*, 1996; EL-Ghorab *et al.*, 1997; Bostrom *et al.*, 2000a). This endover aiming to study the influence of passive smoking on the alveolar bone density and to what extent bone loss and bone resorption occur.

MATERIAL AND METHODS

The study included 60 females aged from 30 to 45 years with mean age 37.7 years to avoid the menopausal age factor changes. The subjects were selected from out patient dental clinic of National Research Centre. All the subjects were chosen to be of the same socio-economic level and free from any systemic disease that may affect the bone density. The examinees were divided into two groups according to exposure to passive smoking. The first group (control) included thirty subjects not exposed to passive smoking. The second group included thirty subjects exposed to passive smoking at home or at work (passive smokers group), the duration of exposure not less than 5 years and not more than 15 years.

Methods:

Since, the alveolar bone loss or gain until 30% to 50% of bone mineral changes cannot be registered by conventional dental radiograph, the direct digital panoramic radiography was recommended for purpose of the standardization (radiometric and radiodensity measurements).

Panoramic radiographs were taken for all the subjects according to the standard procedure with magnification power 1.2.

The radiodensitometric measurements were performed for all the radiographs using the Digora soft ware system at different areas (mesial and distal sides for each individual tooth in the lower dental arch) to measure the alveolar bone loss and bone density in the supporting structures.

The mandibular arch was divided into three segments: frontal segment included the central incisors, lateral incisors and the canines i.e. the six anterior teeth; the middle segment included the first and second premolars in both sides. The posterior segment included 4 molars the first and second molars.

Radiometric Measurements:

The following points were traced on the scanned radiographs: alveolar crest point, cemento-enamel junction point, root apex point. For measuring the amount of alveolar bone loss at the crest level (M_1): vertical distances between the cemento-enamel junction point and the alveolar crest point mesially and distally for each individual tooth presents in the examined dental arch segment were measured. Means of these distances in each segment were calculated.

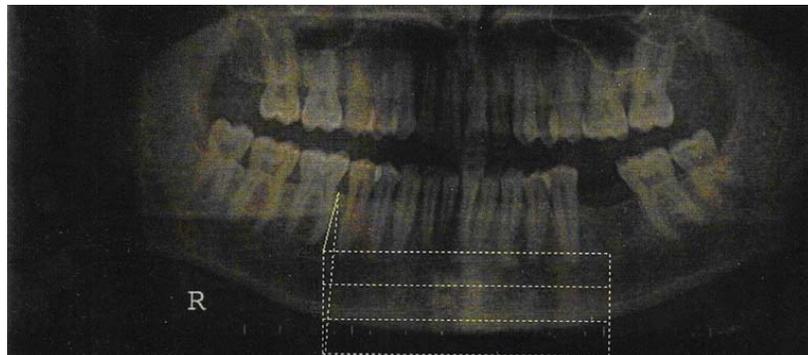
For measuring the alveolar bone height (M_2): the vertical distances from the alveolar crest point to the root apex point mesially and distally to each individual tooth presents in the examined dental arch segment were measured. The mean of these distances in each segment were calculated.

Radio Density Measurements:

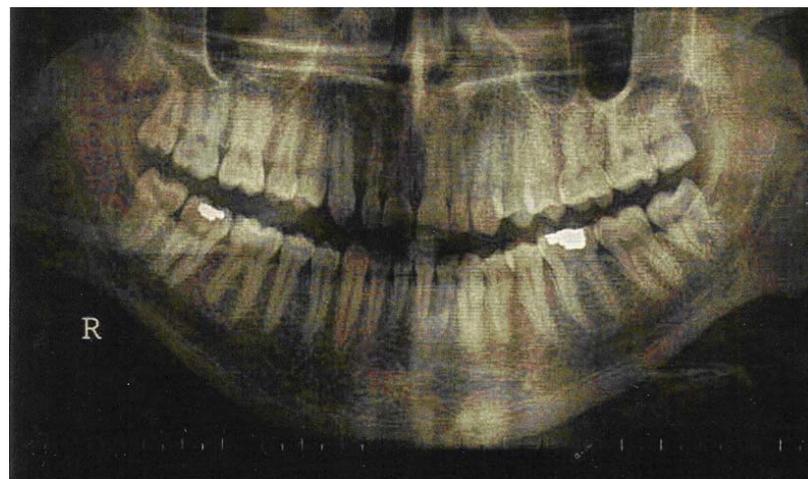
The measurements were performed for each tooth in mesial and distal sides at a line parallel to the fulcrum of each tooth at the following sites; alveolar crest point, mid point of the distance between the alveolar crest point and the root apex point. For each examined dental segment, the mean of alveolar crest level (D_1), mid point level (D_2), apical area levels (D_3) were calculated.



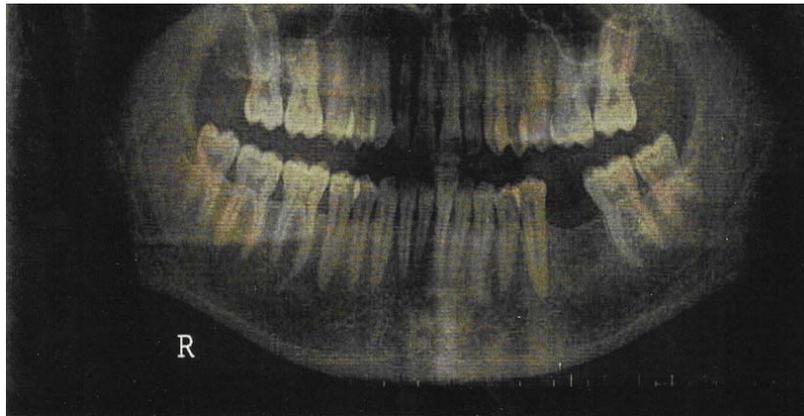
Radiometric Measurements made by Digora software



Radiometric Measurements made by Digora software



A direct digital panoramic radiograph of control patient



A direct digital panoramic radiograph of passive smoker

RESULTS AND DISCUSSION

Results:

Table (1) shows radiometric measurements of all teeth in the three different arch segments for the two examined groups. It is obvious that, there is statistically a significant differences between passive smoking group and control group in all measurements (M_1 , M_2), in the three dental arch segments with significant level $P < 0.01$ where control group significantly lesser in measurements concerned with amount of the bone loss at alveolar crest (M_1). On the other hand, the measurements concerned with residual alveolar bone height (M_2) it is clear that control group measurements are significantly higher. Regarding measurements concerned with the bone density as shown in table 2, the estimated means of bone density of control group are significantly higher in all sites (D_1 , D_2 , D_3) than the passive smoking group ($p < 0.01$).

Table 1: Means and Standard Deviations of Radiometric Measurements of Dental Arch Segment (Frontal, Middle, and Posterior) for Passive and Control Groups.

Arch Segments	Site	Passive Smoking Group.	Control Group
Frontal Segment	M 1	0.31*±0.00	0.24±0.01
	M 2	1.45±0.27	1.48*±0.26
Middle Segment	M 1	0.33*±0.09	1.53*±0.26
	M 2	1.41±0.27	0.26±0.08
Posterior Segment	M 1	0.37*±0.11	1.30*±0.26
	M 2	1.20±0.23	0.30±0.11

M 1: The mean vertical distances of both mesial and distal sides from cemento- enamel junction level to the alveolar crest level for each individual tooth in the examined segment (frontal, middle and posterior).

M 2: The mean vertical distances of both mesial and distal sides of the alveolar height from the alveolar crest level to root apex level for each individual tooth in the examined segment (frontal, middle and posterior).

* $P < 0.01$

Table 2: Means and Standard Deviations of Radio Density in the Different Dental Arch Segments (Frontal, Middle, and Posterior) for Passive Smoking and Control Groups.

Arch segments	Site	Passive smoking group X± SD	Control group X± SD
Frontal Segment	D ₁	63.30±12.13	68.84*±12.83
	D ₂	64.86±13.49	68.71*±12.85
	D ₃	67.78±11.68	73.46*±12.64
Middle Segment	D ₁	73.10±17.15	78.49*±20.10
	D ₂	76.24±16.09	82.87*±16.37
	D ₃	68.90±15.77	75.47*±16.26
Posterior Segment	D ₁	75.98±17.88	78.49*±21.94
	D ₂	82.30±18.89	89.85*±19.67
	D ₃	75.57±18.09	82.07*±18.37

D₁: The mean of bone density at the alveolar rest of both mesial and distal sides for each individual tooth in each examined segments.

D₂: The mean of bone density in the middle area alveolar height in mesial and distal sides for each individual tooth in the examined segments.

D₃: The mean of bone density in the periapical region for each individual tooth in each examined segments.

* $P < 0.001$

Discussion:

Passive smoking which is breathing in other's smoke is one of the key issues leading to smoking bans in workplaces and indoor public places, including restaurants. Research has generated scientific evidence that passive smoking causes the same problems as active smoking (Boyle *et al.*, 2003). It could be confirmed from the results of this study that exposure to passive smoking is a destructive process exactly as active cigarette smoking since both of them are produced by burning tobacco. This is in accordance with other studies that found that persons who had never used tobacco but exposed to passive smoking were more likely to have systemic diseases than those not exposed to passive smoking (Pirkle *et al.*, 1996; Arebes and Slade 2001). The volatile components of cigarette smoke have direct and indirect harmful toxic effects on bone metabolism through a variety of local effects acting directly on the periodontium and systemic effect e.g. cardio vascular diseases, pulmonary diseases and bone diseases [Johnson and Hill, 2004; Boyle *et al.*, 2003; Tomar and Asma, 2000]. Further more, the pathogenic effect differs by dose, duration of smoking and combination of the other risk factors that interfere the mechanism of bone metabolism (Seeman, 1996, Palmer *et al.*, 1999).

When evaluating hard tissue measurements such as vertical amount of bone loss at alveolar crest level (M_1) and the residual bone height (M_2), it is obvious from the results that the amount of alveolar bone loss was significantly higher in the three segments of dental arch in passive smoker group than the control one. However, the opposite was observed in measurements concerned the amount of residual ridge (M_2). As regard bone mineral density, the passive smoker group has a lower bone mineral density than the control group in the three different sites (D_1 , D_2 , D_3) of each examined dental arch segment. These results are in agreement with previous studies (Bergstrom and Ellison, 1987; Payne *et al.*, 1996), however, the explanation for these findings is that, the smoking whether passive or active may alter the mechanism of bone metabolism (El-Ghorab *et al.*, 1997; Bostrom *et al.*, 2000a). Since, the local effects of smoking including vasoconstriction (caused by nicotine) and reduction of the oxygen tension which creates a favorable environment for colonization by aerobic bacteria and dental flora. In addition to the heat from cigarette smoking which could have a local effect on the periodontium (Salvi *et al.*, 2000, Hanioka *et al.*, 2000). Systemic effects of the volatile component of cigarette include, impaired chemotaxis, phagocytosis of oral and peripheral neutrophil and reduced antibody production through decreasing the proliferating capacity of T cell which affect B cell function and antibody generation. Furthermore, nicotine, suppress the osteoblastic proliferation and stimulate the alkaline phosphates activity (Salvi *et al.*, 2000). Also, it has a deleterious effect on the fibroblast function, where the fibroblasts exposed to nicotine produce less fibronectin and collagen, as the collagenase production was increased. (Raulin *et al.*, 1988; Tanur *et al.*, 2000; Cattanlo *et al.*, 2000).

So, it appears reasonable to use the current finding to reiterate the known oral health hazards of tobacco conception as promulgated in the policies of the major public health organization which are target primarily towards elimination of active smoking as it is of course, the active smokers who generate the tobacco smoke inhaled by passive smokers.

Needless to say that smoker' arguments on their rights to light up could be over taken by the rights of non smoking majority to be protected from other peoples tobacco smoke. (D_1 , D_2 , D_3)

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