Effect of Habitual Cigarette Smoking on P Wave Duration and Dispersion

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ABSTRACT

In this study, P wave duration and dispersion (PWD) were measured in 30 heavy smoker, 30 light smoker and 30 nonsmoker subjects. There were no significant difference among heavy smokers, light smokers and nonsmokers with respect to maximum P wave duration and PWD (117±9ms, 116±8ms, 115±6ms ANOVA p=0.78, and 48±10ms, 45±11ms, 43±8ms, one-way ANOVA p=0.14, respectively). Minimum P wave duration was also similar in three groups. (69±10 ms, 70±13ms, 72±9ms, one-way ANOVA p=0.51). We also found no dose dependent-relation between the duration of smoking, the number of cigarettes smoked, and P wave duration. Habitual cigarette smoking alone does not alter P wave duration and PWD in otherwise healthy young subjects.

Key Words: Habitual cigarette smoking, sympathetic overactivity, P wave dispersion.

ÖZET

Kronik Sigara İçiciliğinin P Dalga Süresi ve Dispersiyonu Üzerine Etkisi

Bu çalışmada; 30 ağır sigara içicisi, 30 hafif sigara içicisi ve sigara içicisi olmayan 30 olguda P dalga süresi ve dispersiyonu (PWD) ölçülmüştür. Ağır sigara içicisi, hafif sigara içicisi ve sigara içicisi olmayan olgu gruplarında ölçülen maksimum p dalga süresi ve PWD sırasıyla (117±9ms, 116±8ms, 115±6ms ANOVA p=0.78 ve 48±10ms, 45±11ms, 43±8ms, one-way ANOVA p=0.14) olarak bulundu, anlamlı faklılık izlenmedi. Ayrıca olgu gruplarında ölçülen minimum P dalga süresi de benzer bulunmuştur (69±10ms, 70±13ms, 72±9ms, one-way ANOVA p=0.51). Bununla birlikte sigara içim süresi, içilen sigara sayısı ve P dalga süresi arasında doz bağımlı ilişki bulunmamıştır. Sağlıklı genç yaşlardaki olgularda tek başına kronik sigara içimi, P dalga süresi ve PWD üzerinde değişiklik oluşturmamaktadır.

Anahtar Kelimeler: Kronik sigara içimi, sempatik overaktivite, P dalga dispersiyonu.

INTRODUCTION

Smoking is a well established coronary risk factor, and it is known that the incidence of coronary artery disease and the rate of sudden death are higher among smokers than nonsmokers (1-3). Although the underlying mechanisms are still unclear, sympathetic nerve hyperactivity caused by smoking is believed to be one of the reasons for sudden death (4,5). On this direction, it has been shown that heart rate is lower after smoking cessation than during smoking, presumably because sympathetic nerve activity, which is elevated during smoking, decreases with smoking cessation (6,7). Previously, the influence of smoking on the heterogeneity of ventricular repolarization has been extensively studied although conflicting results have been reported (8-10). However, whether habitual smoking alters P wave duration and PWD has not been evaluated yet.

P wave dispersion (PWD) is a recent ECG marker that reflects discontinuous and inhomogeneous conduction of sinus impulses (11,12). P wave duration and PWD have been reported to be influenced by the autonomic tone, which induces changes in the velocity of impulse propagation (13). It has also been reported that increased sympathetic activity results in increased PWD (14). We have hypothesized that putative role of sympathetic overactivity as a consequence of habitual smoking may affect P wave duration and PWD. Therefore, the objective of this study was to investigate whether habitual smoking influences P wave duration and PWD and to determine whether or not there is a dose-dependent effect of active smoking on P wave duration and PWD.

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METHOD

Sixty healthy smokers with a history of active smoking, and age-gender matched 30 individuals, who never smoked, were included to the study. Active smokers were subclassified into two groups: 30 heavy smokers (with smoking habit of ? 1 pack/day, mean 6.6 ±1.7 years) and 30 light smokers (with smoking habit of ≤1 pack/week, mean 11.2±3.7 years). All study subjects were free from risk factors for coronary artery disease apart from smoking and no subject was receiving any medication at the moment of study. All subjects were asked to refrain from smoking, food intake and caffeine containing beverages at least 2 hours before attending the study. All participants were taken to the test room where they rested in supine position at least 15 minutes on a comfortable bed to stabilize heart rate. Then, the 12-lead ECG recordings at a paper speed of 50-mm/s and 2mV/cm standardization were obtained from all participants. Measurement of P wave duration was carried out manually using a caliper. The onset of P wave was defined as the point of the first visible upward departure of the trace from the bottom of the baseline for positive waves and as the point of first downward departure from the top of baseline for negative waves. The return to the baseline of the bottom of trace in positive waves and the top of the trace in negative waves were considered to be the end of the P wave. All P-waves were checked for noise and if it was not clear, the examination was repeated. At least three consecutive beats were measured in each lead. When the end of the P wave could not reliably be determined these leads were excluded from the study. Also, to improve accuracy all measurements were performed with magnifying lenses for defining the electrocardiogram deflection. The difference between the maximum and minimum P wave duration was calculated from the 12-lead ECG and was defined as the PWD. We have also investigated the association between the number of years of habitual smoking or the number of cigarettes smoked per day and P wave duration-PWD in smoker group. Analyses of ECG parameters were performed by two independent observers who were unaware of the clinical details. Intra and inter-observer coefficients of variation (standard deviation (SD) of differences between two observations divided by the mean value and expressed in percent) were found as 4.1 % and 4.3 % for maximum P wave duration and 4.3 % and 4.4 % for PWD, respectively.

Statistical analysis

All data were presented as mean value ± SD. Comparison of clinical and ECG variables among heavy smokers, light smokers and nonsmokers were performed with one-way ANOVA. Comparison of categorical data was performed by chi-square test. Relation between the number of years of habitual smoking and the number of cigarettes smoked per day, and P wave duration-PWD variables was assessed by Pearson's correlation coefficient. A p value <0.05 was considered as statistically significant.

RESULTS

Clinical characteristics of each group are shown in Table-1.

Table 1: General characteristics of the study subjects.

Variable	heavy smokers	light smokers	nonsmokers
Age (a)	39±8	38±8	NS
Sex (women)	10	14	NS
Recovery HR	76±12	71±9	NS
BP systolic (mmHg)	117±6	114±8	NS
BP diastolic (mmHg)	76±6	74±6	NS
MVA (cm^2)	1.42±0.32	-	-
Maximum mitral gradient (mmHg)	15.5±5.3	-	-
Mean mitral gradient (mmHg)	8.3±3.5	-	-
LA (cm)	4.16±0.5	2,93±0,5	<0.001
LVEF (%)	62±5	64±5	NS

There was no significant difference between each group in demographics of sex, age, blood pressure, and heart rate. On physical examination no clinically significant disorder were detected in any of the study subjects. All study subjects had sinus rhythm. The number of the leads in which P wave duration could be measured ranged from 8 to 12 leads. Maximum and minimum P wave duration were found to be similar in heavy smokers, light smokers and nonsmokers. (Figure1-2). PWD was also found to be identical in each group (Figure-3).

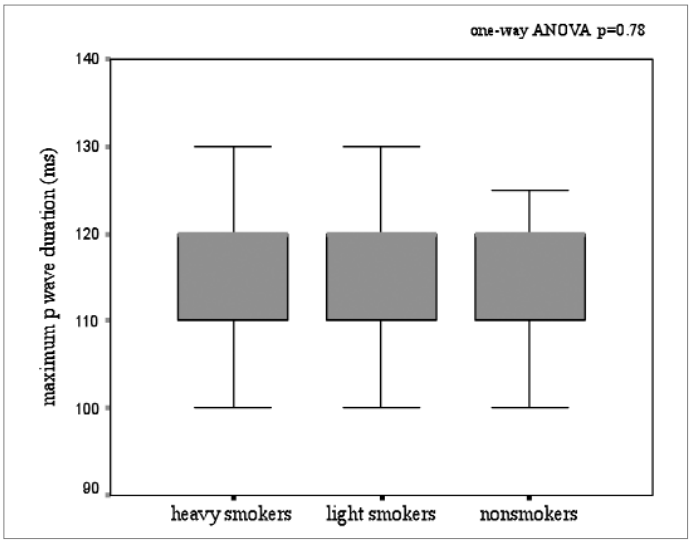

Figure 1: Comparison of maximum P wave duration in each group.

In addition, there was no significant correlation between the number of years of habitual smoking, the number of cigarettes smoked per day, and P wave duration or PWD in the smoker group.

DISCUSSION

The results of this study suggest that P wave duration and PWD are not altered either in heavy smokers or in light smokers compared to that of nonsmokers. In addition, there is no association between the number of ye-

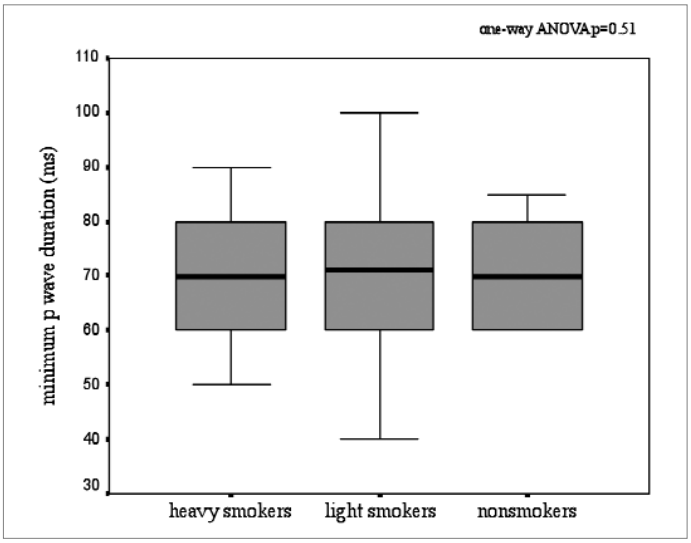

Figure 2: Comparison of minimum P wave duration in each group.

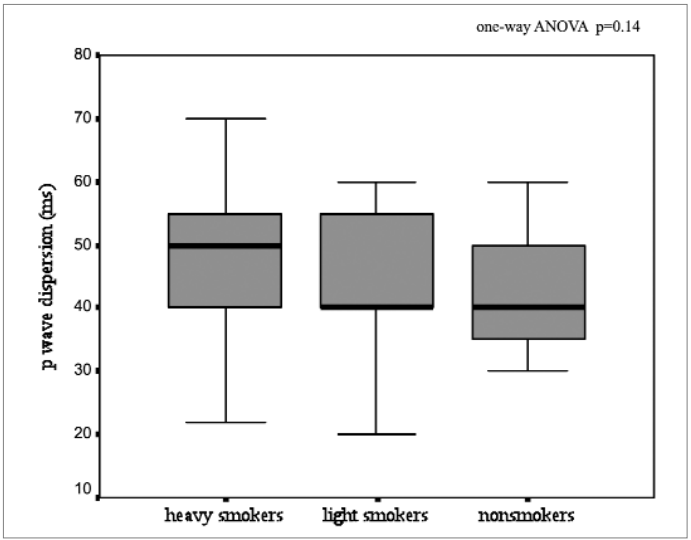

Figure 3: Comparison of PWD in each group.

ars of habitual smoking, the number of cigarettes smoked per day, and P wave duration or PWD.

P wave duration and PWD have been reported to be influenced by the autonomic tone, which induces changes in the velocity of impulse propagation (13). Prolonged P-wave duration and increased PWD have been reported to carry an increased risk for AF (1,11,15). Moreover, it has also been suggested that P wave duration and PWD are prolonged in patients with long-term AF compared to short-term AF (16). Therefore, PWD can be used to separate patients with a high risk of AF during sinus rhythm (13). Accordingly, Tukek et al.(15) suggested that increased sympathetic activity may cause significant increase in PWD. As they observed that P wave duration and dispersion were increased in patients with paroxysmal AF when compared with controls and that Valsalva maneuver normalized these changes, they supposed their findings could be related to beneficial effects of medications that decrease sympathetic tone in converting AF to sinus rhythm. More recently, beta-blocker therapy has been shown to reduce PWD in heart failure patients (17).

Cigarette smoking is a well recognized risk factor for cardiovascular disease and is known to promote the development of atherosclerosis, thrombosis, acute myocardial infarction and sudden death (1,3). Indeed, smoking has been shown to enhance adrenergic activity (4,18). Lucini et al. using spectral analysis of RR interval and systolic arterial pressure variability, have indicated that habitual cigarette smoking induces selective alterations in neuronal control of SA node (19). However, to our knowledge, effect of the habitual smoking on P wave duration and dispersion has not been studied, to date. Therefore, taking into account the increased sympathetic drive in habitual smokers we speculated that habitual smoking might affect P wave duration and PWD, and to our knowledge, our study is the first attempt to show effect of the habitual smoking on P wave duration and PWD.

From the clinical point of view, habitual cigarette smoking is a well established cardiovascular risk factor and is a public concern. The fact that P wave duration and PWD are not altered in habitual smokers does not ameliorate the other detrimental effects of cigarette. In addition, we included only a small number of young subjects, therefore, our results should be interpreted with caution. Moreover, all study subjects were free from the other cardiovascular risk factors.

CONCLUSION

Habitual cigarette smoking alone does not alter P wave duration and PWD in otherwise healthy young subjects. However, in case of accompanying coronary artery disease whether cigarette smoking to alter P wave duration and PWD remains to be determined.

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