

Effect of Acute Cigarette Smoking on Atrial Impulse Propagation

Akut Sigara İçiminin Atriyal Uyarı İletimi Üzerine Etkisi

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ABSTRACT

Introduction: Acute cigarette smoking has been associated with sympathetic hyperactivity. We therefore sought to determine whether acute cigarette smoking alters atrial impulse propagation.

Materials and Methods: Twenty healthy smokers with a history of active smoking were included to the study. The 12-leads surface ECG recorded at a paper speed of 50 mm/s was obtained from all participants before and average 15 minutes after smoking one cigarette.

Results: The change in maximum and minimum P wave duration was measured manually and difference between two values was defined as P wave dispersion (PWD). Both maximum P wave duration and PWD remained unchanged while heart rate significantly increased after smoking one cigarette (104 ± 10 vs. 105 ± 7 ms, p > 0.05; 38 ± 10 vs. 42 ± 8 ms, p > 0.05, and 66 ± 5 vs. 72 ± 8 beat/min, p < 0.05, respectively).

Conclusion: We found that acute cigarette smoking alone did not alter atrial impulse propagation in chronic smokers.

Key Words: Cigarette smoking; smoking.

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ÖZET

Giriş: Akut sigara içiminin sempatik hiperaktiviteyle ilişkili olduğu bilinmektedir. Bu nedenle biz de akut sigara içiminin atriyal uyarı iletimini değiştirip değiştirmediğini araştırmak istedik.

Materyal ve Metod: Aktif sigara içimi öyküsü olan sağlıklı 20 sigara tiryakisi çalışmaya dahil edildi. Sigara içimi öncesinde ve 15 dakika sonrasında katılımcıların 12 leadli yüzey EKG'leri 50 mm/sn kağıt hızıyla çekildi.

Bulgular: Maksimum ve minimum p dalga sürelerindeki değişim manuel olarak ölçüldü ve bu iki değer arasındaki fark P Dalga Dispersiyonu (PWD) olarak tanımlandı. Bir sigara içiminin ardından kalp hızı anlamlı şekilde artarken, hem maksimum P dalga süresi hem de PWD değişmeden kaldı (sırasıyla 66 ± 5 vs. 72 ± 8 atım/dakika, p < 0.05; 104 ± 10 vs. 105 ± 7 ms, p > 0.05; 38 ± 10 vs. 42 ± 8 ms p > 0.05).

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Sonuç: Kronik sigara tiryakilerinde akut sigara içiminin tek başına atriyal uyarı iletimini değiştirmedeğini bulduk.

Anahtar Kelimeler: Sigara kullanımı; sigara.

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INTRODUCTION

Smoking is one of the established cardiovascular risk factor and it is known that smoking causes coronary atherosclerosis, inflammation, plaque rupture, unstable coronary syndrome and sudden death⁽¹⁻³⁾. Smoking-induced cardiovascular events have been partly attributed to sympathetic stimulation occurring as a consequence catecholamines release⁽⁴⁾. It has been shown that chronic smokers have higher pulse rate and blood pressure compared with nonsmokers, indicating sympathetic hyperactivity⁽⁴⁻⁶⁾. Similarly, acute smoking has been shown to cause a transient increase in pulse rate and blood pressure⁽⁷⁾. Acute smoking is also associated with a constriction of epicardial coronary arteries, an increase in coronary resistance, a reduction in the coronary flow reserve and is associated with coronary spasms in patients with coronary artery disease^(8,9). More recently, we have showed that acute smoking alters brachial artery endothelial function and carotid artery hemodynamics^(10,11). Although hemodynamic effect of smoking on cardiovascular system has been well defined, whether acute cigarette smoking alters atrial impulse propagation has not been studied yet.

P wave duration and dispersion (PWD), new electrocardiographic markers that reflect discontinuous and inhomogeneous conduction of sinus impulses, has been reported to be influenced by the autonomic tone, which induces changes in atrial size and the velocity of impulse propagation^(12,13). In addition, it has been shown that sympathetic hyperactivity may increase PWD^(14,15). Moreover, beta-blocker therapy has been shown to decrease P wave duration and PWD⁽¹⁶⁾. Due to possible role of high sympathetic tone on P wave duration and PWD, we have speculated that smoking may influence P wave duration and PWD. Therefore, in this study we attempt to investigate instantaneous effect of smoking one cigarette on P wave duration and PWD in smokers.

MATERIALS and METHODS

Twenty healthy long-term heavy smokers (with smoking habit of ≥ 1 pack/day) were included to the study. A

complete physical and echocardiographic examination was performed before the study. All study subjects were free from the other risk factors for coronary artery disease and no subject was receiving any medication at the moment of study. All participants gave their informed consent and institutional review board approved the study protocol. All subjects were asked to refrain from smoking, food intake and caffeine containing beverage at least 8 hours before attending the study. The study was performed between 09:00 am and 12:00 pm. The subjects were taken to the test room and rested in supine position at least 15 minutes on a comfortable bed to stabilize heart rate and 12-lead ECG was recorded for each subject at a rate of 50 mm/s and 2 mV/s in the supine position. After completion of baseline records, all participants were asked to smoke a cigarette (1.1 mg nicotine, 15 mg tar) and inhalation of smoke was encouraged. Then, second ECG records were obtained average 15 minutes after smoking.

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 of the top of the trace in negative waves, were considered to be the end of the P wave. The difference between the maximum (Pmax) and minimum P (Pmin) wave duration was calculated from the 12-lead ECG and was defined as the PWD. At least three consecutive beats were measured in each lead. All P- waves were checked for noise and if it was not clear, the examination was repeated. When the end of the P wave could not reliably be determined these leads were excluded from the study and also, to improve accuracy all measurements were performed with magnifying lenses for defining the electrocardiogram deflection. Moreover, to minimize measurement errors analyses of ECG parameters (Pmin, Pmax and PWD) were performed in duplicate on two separate days and by two independent observers who were unaware of the order of electrocar-

diagrams. 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 4.2% for Pmax and 4.2% and 4.4% for PWD respectively.

Statistical Analysis

All data were presented as mean value \pm SD. Binomial variables were analyzed with chi-squared test. ECG parameters before and after cigarette smoking were tested by Wilcoxon signed-rank test. Relation between the number of years of habitual smoking and the number of cigarettes smoked per day, and ECG parameters were assessed by Pearson's correlation coefficient. A p value < 0.05 was considered as statistically significant.

RESULTS

Clinical characteristics are shown in Table 1. On physical examination no clinically significant disorder were detected in any of the study subjects. Echocardiographic examination revealed no significant cardiac disorder. 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. Both Pmax or Pmin and PWD remained unchanged while heart rate significantly increased after smoking one cigarette (Table 2). In addition, there was no significant correlation between the number of years of habitual smoking, the number of cigarettes smoked per day and Pmax and PWD in smokers.

DISCUSSION

Previous studies have extensively investigated the detrimental effect of cigarette smoking; however, whether smoking alters atrial impulse propagation has not been clearly elucidated. Therefore, we attempted to investigate effect of acute cigarette smoking on atrial impulse propa-

Table 1. General characteristics of study subjects

Variable	
Number (male/female)	14/6
Age (years)	27 \pm 6
Body mass index (kg/m ²)	23 \pm 5
Systolic blood pressure (mmHg)	117 \pm 5
Diastolic blood pressure (mmHg)	68 \pm 5
Heart rate (beats/minute)	67 \pm 7
Cigarettes/day	23 \pm 15
Pack-years	17 \pm 8

Table 2. Comparison of P interval duration and dispersion before and after smoking one cigarette

Variables	Before smoking	After smoking	p
Pmax (ms)	104 \pm 10	105 \pm 7	NS
Pmin (ms)	66 \pm 13	63 \pm 7	NS
PWD (ms)	38 \pm 10	42 \pm 8	NS
Heart rate (beat/minute)	66 \pm 5	72 \pm 8	0.03

NS: Statistically not significant.

gation by P wave duration and PWD method. Our findings suggested that single dose smoking did not alter P wave duration and PWD in smokers.

PWD is a new electrocardiographic marker that has been associated with the inhomogeneous and discontinuous propagation of sinus impulses and the correlation between the presence of intraatrial conduction abnormalities and the induction of paroxysmal atrial fibrillation (AF) has been well documented^(14,15,17). P wave duration and PWD have been reported to be influenced by the autonomic tone, which induces changes in the velocity of impulse propagation^(14,15). Prolonged P-wave duration and increased PWD have also been reported to carry an increased risk for AF^(13,18,19). Accordingly, Tukek et al. suggested that increased sympathetic activity may cause significant increase in PWD⁽¹⁵⁾. As they observed that P wave duration and PWD were increased in patients with paroxysmal AF when compared with controls and that Valsalva maneuver normalized these changes, and 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⁽¹⁶⁾. Therefore, giving the effect of sympathetic stimulation on P wave duration and PWD we have speculated that acute cigarette smoking by sympathetic stimulation may alter atrial impulse propagation. However, we failed to observe any difference with regard to P wave duration and PWD.

It has been well known that cigarette smokers are at increased risk for coronary artery disease and sudden death. However, the mechanisms through which cigarette smoking suggest its detrimental effects are still unclear. Sympathetic nerve hyperactivity caused by smoking is believed to be one of the reasons for sudden death^(4,6). Previous reports have shown that habitual smokers reveal a marked disturbance of the neural control of the

sinoatrial node. At rest, habitual cigarette smokers are characterized by sign of increased sympathetic tone, as well as reduced vagal modulation and blunted baroreflex gain⁽²⁰⁻²²⁾. Heavy smoking has also been associated with long-term reduction in vagal cardiac control and blunted postural responses in autonomic cardiac regulation in young people⁽²²⁾. Indeed, it has been shown that heart rate is lower after smoking cessation than during smoking, presumably because sympathetic hyperactivity, which is elevated during smoking, decreases with smoking cessation^(4,6,23-25).

On the other hand, several previous studies have focused on the cardiovascular effect of acute cigarette smoking. Stefanadis et al. have observed a prompt increase in heart rate and blood pressure during the first 5 minutes after smoking⁽²⁶⁾. It has also been shown that circulating and locally released catecholamines rise to maximal at the end of 10-minute smoking period and return to baseline levels 30 minutes after the start of smoking^(4,26). It has been shown that short-term smoking causes an approximate 7% decrease in coronary flow velocity and a 10% increase in coronary resistance in patients with coronary artery disease⁽²⁷⁾. Most of the acute effects of smoking on neurocardiovascular regulation have been mainly attributed to nicotine which is the main constituent of cigarette. Indeed, nicotine is implicated in a wide spectrum of cardiac rhythm disorders, including transient sinus arrest and/or bradycardia, sinus tachycardia, atrial fibrillation, sinoatrial block, AV block, and ventricular tachyarrhythmias^(1,28,29). The mechanisms of the effect of nicotine include stimulation and blocking of the autonomic ganglia, liberation of adrenomedullary epinephrine, stimulation of carotid body chemoreceptors and aortic baroreceptors and direct action in the central nervous system^(30,31). In this study we observed that single dose smoking did not alter P wave duration and PWD despite sympathetic stimulation.

We studied on an extremely young population and the fact that no subject had concomitant cardiovascular disease might have affected our results. In addition, manual measurement errors of P wave duration and PWD may limit the value of our findings although this method has been well accepted and used in previous studies^(12,15,18).

We found that acute cigarette smoking did not alter atrial impulse propagation in long-term smokers. However, the application of our findings to older healthy subjects or patients with coronary artery disease should be studied in a larger population in the future.

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