

The Acute Effect of Passive Smoking on QT Dispersion in 95 Healthy Men

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Abstract

Background: Passive cigarette smoking is a known cause of a variety of diseases. However, most of the studies done in this field have focused on its chronic effects on human health. Studies considering the acute effect of smoking on QT dispersion, on the other hand, with its known predictive role in the occurrence ventricular arrhythmia and sudden cardiac death are sparse.

Objectives: This study aims at determining possible relation between acute exposure to others' cigarette smoke and QT dispersion in normal subjects.

Materials and Methods: In this prospective study, 95 healthy male volunteers 16 to 62 years (meanSD: 29.8±9.5) and normal baseline ECG were selected. Within 5 minutes of inhaling the smoke of one filtered cigarette burned at a distance of one meter a second ECG was obtained. We measured QT intervals in each of the 12 leads and corrected them according to the heart rate (QT_c). Difference of maximum and minimum measured QT and QT_c intervals amongst 12 leads (QT dispersion (QT_d) and QT_c dispersion (QT_{cd}), respectively) were compared.

Results: Mean±SD QT_d were 50.8±17.8 ms before and 73.6±29.2 ms after passive smoking. Mean±SD QT_{cd} were also increased from 62.2±20.9 ms to 85.2±30.8ms (p<0.001).

Conclusion: Passive smoking may acutely affect myocardial vulnerability to arrhythmia by increasing QT dispersion. More strict rules against smoking in all public places are endorsed in order to decrease the incidence of this preventable cause of illness and death.

Iran J Med Sci 2002; 27(3):117-119.

Keywords • QT dispersion • ventricular arrhythmia • sudden cardiac death • passive smoking

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Introduction

Cigarette smoking is one of the best known preventable causes of many human diseases. Not only it affects the smokers, but is also shown to be cumulatively harmful to those who inhale the cigarette smoke of others. Passive smoking is capable of inducing cardiovascular, pulmonary, and even malignant

Table 1: Dispersion of QT and its corrected forms, before and after passive smoking, in 95 healthy men

QT _d 's Passive smoking	Mean±SD QT _d *	Mean±SD QT _{c1d} *	Mean±SD QT _{c2d} *
Before	50.8±17.8	62.2±20.9	56.6±21.0
After	73.6±29.2	85.2±30.8	79.9±33.4
95% CI	-27.86 to -17.63	-29.58 to -16.90	-28.84 to -17.58

*Significantly different (p<0.001)

diseases in spouses and children of active smokers.¹⁻⁵ Exposure to environmental tobacco smoke (ETS) in work places⁶ and transportation systems is well known.⁷

One animal study has shown lowering of myocardial arrhythmia threshold in dogs forced to inhale cigarette smoke⁸. The electrocardiographic marker of myocardial repolarization, the period when some cardiac arrhythmias develop, is QT interval, from the onset of QRS complex to the return of T wave to the TP line. QT intervals are not exactly equal in all 12 leads of even a normal electrocardiogram. Increasing this difference from a defined point (58 ms)⁹ which is called increased QT dispersion has been shown to be related to vulnerability of cardiac muscle cells to ventricular arrhythmias¹⁰. Our study was designed to find if there was any acute effect attributable to passive cigarette smoking on QT dispersion in normal subjects.

Patients and Materials

We selected 95 healthy volunteer men with ages 16 to 62 years (mean±SD: 29.8±9.5) and explained the procedure for them. None of these men had history of heart or any other organ diseases and or drug consumption. Twenty of the subjects were current smokers.

We obtained a baseline ECG from each person in supine position, with a Cardiotest EK-51 ECG machine at a paper speed of 25 mm/s. A smoker sitting one meter apart from the subjects, smoked one filtered cigarette and within five minutes of finishing the cigarette, we obtained the second ECG. The QT intervals in each person's pair of ECGs were measured from the onset of QRS complex to the return of T wave to the TP line. Leads with

blurred-ended or flat T waves were omitted. For each ECG to be included in the study, we obtained at least nine leads with measurable QT intervals, with at least four precordial leads. We corrected QT intervals according to subjects' heart rate, using Bazett's formula as QT_{c1}= QT/√RR, and Hodges formula as QT_{c2}= QT+1.75× (heart rate-60). Difference between maximum and minimum QT intervals in each of the 12 leads, was defined as QT dispersion (QT_d). We compared mean QT_d's and QT_{cd}'s before and after passive smoking with the Student's t-test for paired samples.

Results

The mean±SD QT_d's of the subjects were 50.8±17.8 ms before and 73.6±29.2 ms after smoking, and these parameters for QT_{c1d} (Bazett's) were 62.2±20.9 ms and 85.2±30.8 ms, and those of QT_{c2d} (Hodges formula) was 56.6±21.0 and 79.9±33.4 ms, before and after exposure, respectively (p<0.001). There was a highly significant rise of QT_d and QT_{cd} after passive inhalation of one cigarette's smoke (Table 1). The same changes were observed in all 95 persons (Table 2). Inclusion of age groups of subjects did not affect these results (Table2).

The results of study were not different between smokers and nonsmokers.

Discussion

Several studies have shown that passive smokers live shorter than non-smokers. Passive smoking causes 53000 deaths annually in the United States.¹ It is the third preventable cause of death ranking behind active smoking and alcoholism. Aside from increasing mortality from passive smok-

Table 2: Age distribution of changes of QT_d after passive smoking

Age (yrs)	Number	Mean±SD QT _d		p value	%95 CI
		Before	After		
<25	27	48.9±17.7	70.1±25	<0.001	-30.10 to -12.34
25-40	56	52.2±17.6	76.2±32.2	<0.001	-30.77 to -17.23
>40	12	58.3±18.5	73.7±29.1	<0.05	-31.75 to -9.25
Total	95	52.0±17.8	79.2±29.8	<0.001	-27.19 to -17.20

* Wilcoxon matched-pairs signed-ranks test

ing, it is a main preventable cause of morbidity in the general population. One meta-analysis showed the estimated risk of lung cancer for nonsmokers married to smokers as 24%.

There are about ten times as many deaths from environmental tobacco smoke (ETS) induced heart disease as lung cancer. Aggravation of angina pectoris⁴, accelerated atherosclerosis of coronary arteries in adults, and increased incidence of asthma, chronic otitis media⁵ and pneumonia in children are among the other adverse effects of passive smoking.

There are few studies regarding the acute effects of passive smoking. We previously studied the harmful acute effect of active smoking on myocardial repolarization.¹¹ In this study we showed that passively inhaling the smoke of one filtered cigarette in an unventilated room causes inhomogeneity of myocardial repolarization in normal persons, (at least in men), regardless of their age and smoking habit.

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