

STUDY OF ECG CHANGES IN SMOKERSM. Syamala Devi¹, D. Taraka Lakshmi², Nazia Farah³**HOW TO CITE THIS ARTICLE:**

M. Syamala Devi, D. Taraka Lakshmi, Nazia Farah. "Study of ECG Changes in Smokers". Journal of Evolution of Medical and Dental Sciences 2014; Vol. 3, Issue 49, October 02; Page: 11744-11747,

DOI: 10.14260/jemds/2014/3542

ABSTRACT: This study is intended to evaluate the changes in Electrocardiogram (ECG) in apparently healthy adult male smokers. This cross-sectional study covers 40 smokers, who smoked on an average 10 cigarettes per day for at least 5 years, and 40 non-smokers to find out the possible risk factors for cardiovascular disorders. This study was conducted during April 2011 to April 2012 in the Department of Physiology of Andhra Medical College, on subjects whose age ranged from 20 to 60 years. The ECG results were evaluated for different parameters like heart rate, P-wave, P-R interval, QRS complex, QT interval, and T-wave. The results were analyzed using student's t-test. The probability (p value) was calculated. The analysis showed that QRS and QT interval were shortened and that the QTc interval was widened in the smokers, although the values did not show any statistical significance. From the statistical analysis of the results obtained in the present study and their comparison with those of published reports, it appears that smoking 10 cigarettes per day for 5 Years does not cause major change in ECG wave forms.

KEYWORDS: ECG, smoking, cardiovascular disorders.

INTRODUCTION: Tobacco usage is considered one of the biggest public health hazards the world has ever faced, killing nearly six million people a year.^[1] More than five million of these deaths are attributable to direct tobacco use while more than 600000 are attributable to non-smokers being exposed to passive smoke. Approximately one person dies every six seconds due to tobacco use in some form, accounting for one in 10 adult deaths. Up to half of current tobacco users will eventually die of a tobacco-related disease.^[2]

Tobacco use can lead to heart attack, chronic obstructive pulmonary disease, cancer, peripheral vascular disease, hypertension and so on.^[3] Recording of ECG is one of the simplest methods of assessing cardiovascular dysfunction.^[4] An ECG is simply a representation of the electrical activity of the heart muscle as it changes with time. Smoking is a well-established risk factor for ischemic stroke and myocardial infarction.^[5] Smoking has varied effects on the cardiovascular system. The quality, quantity, duration and frequency of smoking play an important role in determining how much smoking is harmful to the cardiovascular system.^[6]

Given the importance of adverse effects of smoking, the present study has been undertaken to see ECG changes in male smokers, who are otherwise healthy.

MATERIALS AND METHODS: Eighty healthy male volunteers who were in the age group of 20 to 60 years, who attended the Out-Patients Department of King George Hospital attached to Andhra Medical College, were chosen for the study after obtaining written informed consent from them. The Institutional Ethical Committee clearance was also obtained. The study was conducted on an outpatient basis.

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Out of those who voluntarily agreed, 80 subjects whose age ranged from 20~60 years were selected, of whom 40 were non-smokers and 40 were smokers who had a history of smoking for more than 5 years. Major inclusion criteria were: age ≥ 18 years, smoking ≥ 10 cigarettes/day.

The major reasons for exclusion were blood pressure (BP) $>160/100$ mmHg; heavy alcohol use; history of seizure or serious head injury; use of contraindicated medications.

All the subjects were from different socio-economic classes and apparently healthy. Height, weight and resting blood pressure were measured using Diamond Blood Pressure BP Apparatus Deluxe Conventional Mercury Arm Type. 12-lead ECG was recorded after a light breakfast without any caffeinated beverages.

The subjects were made to rest in supine position, in non-exercising state and abstained from smoking two hours prior to the test.

The 12-lead electrocardiogram was recorded by using a single channel ECG cardioint (heart view 1200 ECG recorder-manufactured by Brown Dove Healthcare Pvt Ltd). The standard 12-lead ECG requires that 10 ECG leads be attached to the patient's body. The ECG leads are placed as follows:

1. LL: left leg, distally
2. RL: right leg, distally
3. LA: left arm, distally
4. RA: right arm, distally
5. V1: fourth intercostal space, to the right of the sternum
6. V2: fourth intercostal space, to the left of the sternum
7. V3: midway between V2 and V4
8. V4: fifth left intercostal space, mid-clavicular line
9. V5: at the horizontal level of V4, anterior axillary line (or midway between V4 and V6 if the anterior axillary line is not well defined)
10. V6: at the horizontal level of V4 and V5, mid-axillary line.

ECG of each subject was studied for rate, rhythm, axis, intervals PR, QRS, QT, time corrected QT duration (QTc). The heart rate was calculated by dividing 1500 with number of small squares between two R Waves. The QTc (corrected QT interval) was calculated by using Bazet's formula (QT interval/square root of the RR interval).^[7]

The PR interval, the QT interval, QRS segments were evaluated in seconds and the ST segment in millivolts. The results which were obtained were statistically analyzed by using the Student's t-test. The probability (p value) was calculated.

RESULTS: The analysis showed that P-R, QRS and QT duration was shortened and that the QTc interval was widened in the smokers, although the values did not show much statistical significance. None of the smokers show noticeable change in the heart rate.

T wave inversion is seen in smokers and none of the non-smokers showed any T wave abnormality. Smokers showed ST elevation and septal Q wave. One subject in smokers group showed left axis deviation.

Variable	Smoker (n=40) (mean±sd)	Non-Smoker (n=40) (mean±sd)	p Value	Significance
Heart Rate/min	74.30 ± 13.20	74.08 ± 11.53	0.935	nil
P-R interval in seconds	0.14 ± 0.022	0.13 ± 0.017	0.738	nil
QRS in seconds	0.13 ± 0.022	0.13 ± 0.017	0.545	nil
QT interval in seconds	0.37 ± 0.027	0.36 ± 0.023	0.25	nil
QTc interval	0.36 ± 0.042	0.32 ± 0.013	0.1187	nil
R-R interval in seconds	0.79 ± 0.17	0.82 ± 0.012	0.506	nil
ST segment in millivolts	0.093 ± 0.007	0.077 ± 0.046	0.131	nil
Mean Electrical Axis in degrees	51 ± 23.7	42 ± 22.7	0.056	nil
(p < 0.05 significant; p < 0.01 very significant; p < 0.001 highly significant;				
Table 1				

DISCUSSION: Tobacco inflicts huge damage on the health of India's people and could be clocking up a death toll of 1.5 million a year by 2020, if more users are not persuaded to kick the habit, an international report said. Tobacco smoking is a well-recognized risk factor for sudden cardiac death as well as other manifestations of coronary artery disease.^[8]

Nicotine, which is the main component of tobacco, causes sudden coronary death.^[9] It also has propensity to provoke ventricular arrhythmias.^[10,11] A longer retention of nicotine occurs in the blood and in other specific tissues including the heart via a constant exposure. In the heart it can result in profound electrophysiological alterations. Nicotine is a sympathomimetic substance that promotes the release of catecholamines and other neuro-transmitters, acting centrally and peripherally. This release of catecholamine is responsible for its cardiovascular effects such as increase in resting heart rate.

The cardiac effects of nicotine are attributed to the release of catecholamine, which are released due to the binding of nicotine to the nicotinic cholinergic gate on the cation channels in receptors (nAChRs) throughout the body. A longer retention of nicotine occurs in the blood and in other specific tissues such as the oesophagus, fundus, antrum, spleen, caecum, pancreas, testes, heart and the muscle via a constant exposure".^[12] Nicotine facilitates a conduction block and a re-entry and it increases the vulnerability to a ventricular fibrillation.^[13] Nicotine is a potent inhibitor of the cardiac A type potassium channels, which contributes to the changes in the electrophysiology and it also induces arrhythmias.^[14]

It is well known that the acute effects of smoking produce an increase in systolic and diastolic blood pressure, tachycardia, cardiac output and vasoconstriction, increase in carotid artery occlusion, and sometimes instantaneous MI.^[15,16] The present study revealed that 20% smokers showed abnormalities in ECG. According to a similar study done by Chatterjee S. et al., 6.7% smokers showed abnormalities in ECG.^[17]

The present study also showed decreased P-R, QRS complex and QT intervals and increased QTc interval in smokers which were found to be not statistically significant. Similar results are seen in study done by Venkatesh G. et al,^[18] who did a study in 2010 to compare the ECG changes in smokers with normal human beings.

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They found P-wave, P-R, QRS and QT duration was shortened and that the QTc interval was widened in the smokers, although the values did not show much statistical significance in ECG. Similar findings are also observed in a study titled "ECG changes in healthy adult male smokers" done by Khan et al.^[19] The findings showed that smoking causes no appreciable change in ECG wave forms except P-R interval. The present study showed decrease in QRS complex duration in smokers.

CONCLUSION: From the statistical analysis of the results obtained in the present study and their comparison with those of the earlier published reports, it may be concluded that, smoking 10 cigarettes per day for about 5 years causes no significant changes in ECG. However, in order to establish this further, there is need for undertaking a wider study covering different segments on the basis of the number of cigarettes smoked per day, and coupled with other risk factors.

REFERENCES:

1. Global Adult Tobacco Survey: India 2009-2010. Ministry of Health & Family Welfare, Govt. of India. Available from: <http://mohfw.nic.in/WriteReadData/l892s/1455618937GATS%20India.pdf>
2. Global Status Report on Non-communicable Diseases. WHO; April 2011. Available from: http://www.who.int/nmh/publications/ncd_report2010/en/
3. Health effects of cigarette smoking. Center for Disease Control and prevention. Available from: http://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/effects_cig_smoking/
4. Minami J, Ishimitsu T, Matsuoka H. Effects of Smoking Cessation on Blood Pressure and Heart Rate Variability in Habitual Smokers. *Hypertension*. 1999; 33: 586-590.
5. Feigin L, Rinkel E, Lawes M. Risk factors for subarachnoid hemorrhage: An updated systematic review of epidemiological studies. *Stroke*. 2005; 36: 2773-2780.
6. Baden L, Weiss ST, Thomas HE Jr., Sparrow D. Smoking status and the electrocardiogram: a cross sectional & longitudinal study. *Arch Environ Health*. 1982; 37: 365-9.
7. Bazett HC. An Analysis of the Time-relations of Electrocardiograms. *Heart*. 1920; 7: 353-70.
8. Health effects of cigarette smoking, Center for Disease Control and Prevention. Available from: http://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/effects_cig_smoking/
9. Lakier JB. Smoking and cardiovascular disease. *Am J Med*. 1992; 93:8S- 12S.
10. Chevalier HJ, Hunnigh C, Bandilla B, Dontenwill Hanel JW. Effect of high doses of nicotine in pig, I: Changes of the electrocardiogram. *Basic Res Cardiol*. 1976; 71: 68-75.
11. Escobed LG, Zack MM. Of sudden and non-sudden coronary deaths in the United States. *Circulation*. 1996; 93: 2044-36.
12. Chowdhury P, Doi R, Chang LW, Rayford PL. Tissue distribution of [3H]- nicotine in rats. *Biomed Environ Sci*. 1993; 6:59-64.
13. Yashima M, Ohara T, Cao JM, Kim YH, Fishbein MC, Mandel WJ, et al. Nicotine increases ventricular vulnerability to fibrillation in hearts with healed myocardial infarction. *Am J Physiol Heart Circ Physiol*. 2000 Jun; 278 (6): H2124-33.
14. Huizhenwang, Hang Shi MD, Baofeng Yang. Nicotine is a potent Blocker of the cardiac A- type K+Channels. *Circulation*. 2000; 102: 1165-71.
15. Pandey MR. Tobacco smoking and hypertension. *J Indian Med Assoc* 1999; 97: 367-9.

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16. Dwivedi S, Jauhari R. Smoking precipitating instantaneous myocardial Infarction. *Prev Cardiol* 1997; 1: 21-6.
17. Chatterjee S, Kumar S, Dey SK, Chatterjee P. Chronic effects of smoking on the Electrocardiogram. 1989 Nov; 30 (6): 827-839.
18. Venkatesh G, Swamy RM. A Study of Electrocardiographic changes in smokers compared to normal human beings. *Bio- medcal Research*. 2010; 21 (4): 389-392.
19. Khan IS, Rahman MA and Amin R. Study of ECG Changes in Apparently Healthy Adult Male Smokers. *Dinajpur Med Col J* 2011. Jan; 4 (1): 7-14.

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Date of Submission: 01/09/2014.
Date of Peer Review: 02/09/2014.
Date of Acceptance: 25/09/2014.
Date of Publishing: 01/10/2014.