

A Study of Electrocardiographic changes in smokers compared to normal human beings.

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Abstract

The present study was conducted to assess the electrophysiological and hemodynamic alteration in the heart and circulation. Recording of ECG is one of the easiest methods of assessing cardiovascular dysfunction. Hence this study is taken up to detect the electrocardiographic changes due to smoking. The study included male subjects above 18 years of age selected from the general population of Davanagere. They were divided into two groups, controls and smokers. Each group contained 50 subjects. Smokers were selected as per World Health Organization (WHO) 10th revision of International Statistical Classification of Diseases and related health problems (ICD-10) criteria of harmful use. After taking consent and a detailed history from subjects, electrocardiogram was recorded during resting supine position. The ECG results are evaluated for different parameters like heart rate, P-wave, P-R interval, QRS complex, QT_C interval, axis deviation, ST segment and T-wave. Two groups were compared statistically by student 't' test. p value of 0.05 or less ($p < 0.05$) was considered for statistical significance. There is statistically significant increase in the heart rate and decrease in QRS complex and T-P interval in smokers compared to controls. P-wave, P-R interval, QT_C interval, QRS frontal axis, ST segment and T-wave does not show statistically significant results. Our study results showed that smokers are prone for cardiovascular risk. So in earlier stages, abstinence from smoking can revert back the cardiovascular changes to nearly normal or in more severe cases to recovery with little residual damage to the heart.

Key words: Electrocardiogram, smokers, heart rate, axis, ventricular activation time, QRS complex, T-P interval

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Introduction

Cardiovascular diseases were on the constant rise in both developing and developed countries. Smoking produces electrophysiological and hemodynamic alteration in the heart and circulation. Cigarette smoking is one of the most harmful and addictive habits which is widespread all over the world [1]. There are many forms of tobacco which affects cardiovascular, respiratory and other body systems. It is perhaps the most dangerous of all psychoactive substances [2]. Many young people perceive smoking as an attribute to maturity [3]. The World Health Organization's (WHO) report on the Global Tobacco Epidemic in 2008 highlighted that approximately 5.4 million deaths every year are related to tobacco use. Unless urgent attention is taken, more than 80% of tobacco-related deaths will occur in low and middle-income countries by 2030 and could kill one billion during this century [4]. Smoking is a well-established risk factor for ischemic stroke, and

myocardial infarction (MI) [5]. Smoking cessation substantially reduces the risk of stroke and coronary heart disease, and quitting smoking is an important step toward preventing cardiovascular disease [6]. In a community based study showed, smoking is the risk factor for the development of major abnormal Q/QS wave patterns, T wave abnormalities and development of ST segment depression [7]. Western Electric Study provide evidence that minor isolated nonspecific ECG findings like ST-T abnormalities in middle-aged men which indicate increased mortality risk and warrant vigorous preventive management against the occurrence of clinical CHD. Major modifiable-preventable risk factors like cigarette smoking and others may give rise to this ECG finding [8].

Studies have shown that smoking habit induces changes in the normal ECG pattern, but the results are not consistent. Hence we intended to study the same in the Indian population where the smoking pattern, frequency and

smoking substances varied. We also intended to correlate the findings to that of the cardiovascular risk pattern.

Materials and Methods

The study was carried out in the Physiology Department, J.J.M. Medical College, Davangere from July 2003 to June 2005. Subjects were male individuals above 18 years age group selected from the general population. Following an explanation about the nature and purpose of the study, those subjects who were willing to participate were included after obtaining informed consent. They were grouped into two controls and smokers. Each group consisted of 50 subjects. A detailed assessment was done and various parameters were recorded which includes personal details like name, age, occupation, address, smoking history, personal history, family history and past history. Name was taken for identification purpose, but confidentiality was maintained.

Smokers were selected as per World Health Organization (WHO) 10th revision of International Statistical Classification of Diseases and related health problems (ICD-10) criteria of harmful us [9].

The subjects who were selected, a detailed physical and systemic examination was done like pulse rate, blood pressure, respiratory rate, cardiovascular system, respiratory system and central nervous system. Female are excluded because smoking was more prevalent in men than women [10] and prevalence of smoking was higher in males than in females [11].

Subjects with systemic illness like diabetes mellitus, hypertension, and bronchial asthma were excluded. After taking consent and a detailed history from subjects, A 12 lead electrocardiogram was recorded using single channel electrocardiograph Cardiant 108-T-MK-VI manufactured by BPL Electronics Ltd. during the resting state. The ECG results were evaluated for different parameters like heart rate, P-

wave, P-R interval, QRS complex, QT_C interval, axis deviation, ST segment, T wave and T-P interval.

Statistical Analysis

Continuous variables were presented as Mean \pm SD and Range values. Multiple group comparison were made by one way ANOVA followed by student 't' test for pair wise comparison. Categorical data was analyzed by Chi-square test. A p value of 0.05 or less ($p < 0.05$) was considered for statistical significance.

Results

Electrocardiographic recordings of controls and smokers were studied. Each group has 50 subjects. Subjects selected for the study were males of above 18 years of age. Detailed analysis of electrocardiograms was done. Mean age \pm SD (in years) were 35.8 ± 10.1 and 39.3 ± 10.0 in controls and smokers respectively. Heart rates were 76.0 ± 6.9 and 80.8 ± 10.2 among controls and smokers respectively. Heart rate showed a statistically significant increase in smokers groups compared to controls.

P-wave duration and amplitude, P-R interval, QT_C interval values, T-wave, and Axis measurements were within normal range. In smokers four subjects showed decrease in P-R interval and 2 smokers showed axis deviation of -30 but have no statistical significance.

In smokers group two subjects showed elevated ST segment and one subject with depressed ST segment, but no statistical significance.

QRS complex measurements (in seconds) were 0.08 ± 0.01 , 0.06 ± 0.01 , in controls and smokers respectively There is statistically significant decrease in smokers compared to control group.

T-P interval values (in seconds) were 0.29 ± 0.04 , 0.25 ± 0.07 , among controls and smokers respectively There is statistically significant decrease seen in smokers compared with controls.

Table 1 Electrocardiographic readings of Smokers and Controls

Variable	Particulars	Controls	Smokers	P value	
Heart Rate (beats/min)	Mean \pm SD	76.0 ± 6.9	80.8 ± 10.2	$p < 0.05$	
	Range	67 – 100	60 – 110		
P – Wave (in sec)	Mean \pm SD	0.09 ± 0.01	0.08 ± 0.02	NS	
	Range	0.04 – 0.10	0.06 – 0.12		
	Amplitude	Mean \pm SD	1.02 ± 0.20	0.99 ± 0.37	NS
		Range	0.5 – 1.5	0.5 – 2.0	

P – R Interval (in sec)	<i>Mean ± SD</i>	0.14 ± 0.02	0.16 ± 0.02	NS
	Range	0.08 – 0.20	0.14 – 0.16	
QRS Complex (in sec)	<i>Mean ± SD</i>	0.08 ± 0.01	0.06 ± 0.01	p < 0.001
	Range	0.04 – 0.12	0.04 ± 0.08	
QT _C –interval (in sec)	<i>Mean ± SD</i>	0.40 ± 0.06	0.41 ± 0.04	NS
	Range	0.27 – 0.50	0.33 – 0.48	
QRS Frontal axis (in degrees)	<i>Mean ± SD</i>	51.0 ± 23.7	42.0 ± 22.7	NS
	Range	+30 to +60	–30 to +90	
ST Segment	IE	50 (100%)	47 (94%)	
	NSST	-	3 (6%)	NS
	Normal	50 (100%)	48 (96%)	NS
T-wave (in sec)	Abnormal	-	2 (4%)	
	<i>Mean ± SD</i>	0.29 ± 0.04	0.25 ± 0.07	p < 0.01
T – P Interval (in sec)	Range	0.12 – 0.36	0.10 – 0.36	

Multiple group comparisons: One way ANOVA,

Pair wise comparisons

$p < 0.05$, $p < 0.01$

$p < 0.001$

$p > 0.05$

Student 't' test

:Significant

:Highly significant

Not significant

Discussion

Smoking has varied effects on the cardiovascular system. The quality, quantity, duration and frequency of smoking play an important role in determining whether it is beneficial or harmful to the cardiovascular system. Benowitz, Jacob and Rosenberg et al [12] showed that infusion of nicotine in 14 healthy young men increases the heart rate within 5-10 minutes. Majtaba [13] has shown that smoking increased the heart rate from 72 ± 3 to 96 ± 3 . In our study subjects showed increase in heart rate. The increase in heart rate could be due to stimulation of sympathetic ganglia and discharge of catecholamines from adrenal medulla.

Cigarette smoking increases the velocity of conduction and shortens the effective refractory period at the AV node [14]. Mujataba [14] found that in smokers there was a little decrease in the duration of P-R interval. But it was not statistically significant. In our study P-wave and P-R interval were within normal range and was not statistically significant.

QRS complex measurements in our study showed decrease in smokers. Mujtaba [13] found a slight decrease in QRS duration in smokers, though it was not significant.

QT_C interval in the electrocardiogram reflects the time registered for depolarization and repolarization in the ventricular myocardium. Karjalainen, Reunanen and Viitasalo et al [15] reported that smoking was associated with shorter QT_C than non smokers. QT_C interval in our study does not show any statistical significance.

In our study 2 smokers showed axis deviation of – 30 i.e. borderline left axis deviations. The axis deviation in the smokers may be due to right bundle branch block (RBBB), which is associated with other ECG changes also. RBBB may be produced because of ischemia.

ST segment deviation from isoelectric line is a predictor of future coronary events in asymptomatic population. Rywik, Gittings and O'Connor et al [16] reported that smoking causes down sloping of ST segment in the subjects. In our study 3 subjects showed non-specific ST changes in smokers group of no significance.

T-P interval in our study showed statistically significant decrease in smokers compared to controls. The ECG changes were T-P phenomenon i.e. close approximation of T and P wave.

Summary and conclusion

The present study was conducted to assess the electrophysiological and hemodynamic alteration in the heart and circulation. Recording of ECG is one of the easiest methods of assessing cardiovascular dysfunction. The ECG results were evaluated for different parameters like heart rate, P-wave, P-R interval, QRS complex, QT_C interval, axis deviation, ST segment and T-wave. There is no significant change seen in P-wave and P-R interval among both groups indicating that no change in atrial depolarization and AV nodal conduction. QRS complex is decreased significantly in smokers and alcoholics compared to controls. QT_C interval does not show statistically significant changes in all three groups indicating no change in depolarization and repolarization of ventricles.

T-P interval reduction is seen in smokers, which may alter the heart rate.

Our study results showed that smokers were prone for cardiovascular risk. So in earlier stages, abstinence from smoking and alcohol can revert back the cardiovascular changes to nearly normal or in more severe cases to recovery with little residual damage to the heart.

Limitations

Firstly in our study women were not included. Secondly, this is a cross sectional study, therefore there is a need for follow up studies

References

1. Wallece GM, Maxcy-Rosenean-Last RB. Public health and preventive medicine. 14th ed. USA: Appleton and Lange Publications; 1998: 817-845.
2. Lawrence DR, Bennett PN. Tobacco. Clinical Pharmacology. 6th ed. Edinburgh: ELBS Churchill Livingstone; 1987: 397-410.
3. Thun MJ, Day-Lally CA, Calle EE, Flanders WD, and Health CW Jr. Excess mortality among cigarette smokers-change in 20-year interval. Am J Public Health 1995; 85 (9): 1223-1230.
4. World Health Organization. Geneva: World Health Organization; 2008. WHO report on the global tobacco epidemic.
5. Feigin VL, Rinkel GJE, Lawes CMM, Algra A, Bennett DA, Gijn JV et al Risk factors for subarachnoid hemorrhage: An updated systematic review of epidemiological studies. Stroke. 2005; 36: 2773–2780.
6. Wannamethee SG, Shaper AG, Whincup PH, Walker M. Smoking cessation and the risk of stroke in middle-aged men. JAMA. 1995; 274: 155–160.
7. Moller CS, Byberg L, Sundstrom J, and Lind L. T wave abnormalities; high body mass index, current smoking and high lipoprotein (a) levels predict the development of major abnormal Q/QS patterns 20 years later. A population-based study BMC Cardiovasc Disord. 2006; 6: 10-22.
8. Daviglius ML, Liao Y, Greenland P, Dyer AR, Liu K, Xie X et al. Association of Nonspecific Minor ST-T Abnormalities with Cardiovascular Mortality JAMA. 1999; 281: 530-536.
9. Diagnostic criteria for research. ICD-10 Classification of mental and behavioral disorders WHO Geneva 1993: 75.
10. Ashraf A, Quayyum MA, Ng N, Minh HV, Razzaque A, Ahmed SM et al. Self-reported use of tobacco products in nine rural INDEPTH Health and Demographic Surveillance Systems in Asia. Glob Health Action. 2009; 2: 10-21.
11. Gupta R, Misra A, Vikram NK, Kondal D, Gupta SS, Agrawal A et al Younger age of escalation of cardiovascular risk factors in Asian Indian subjects BMC Cardiovasc Disord. 2009; 9: 28-44
12. Benowitz NL, Jacob P, Jones RT, Rosenberg J. Inter individual variability in the metabolism and cardiovascular effects of nicotine in man. J Pharm Exp Ther 1982; 221: 368-372.
13. Mujtaba FA. Effect of smoking on Electrocardiogram and blood pressure. IJPP 1977; 21 (4):393-395
14. Cellina, Honour AJ and Littler WA. Direct arterial pressure, heart rate ECG during cigarette smoking in unrestricted patients. Am H J 1975; 89: 18-25.
15. Karajlainen J, Reunanen A, Ristola P, Viitasalo M. QT_c - interval as cardiac risk factor in middle aged. Heart 1997; 77: 543-548.
16. Rywik TM, Zink RC, Gittings NS, Khan AA, Wright JG, O'Connor FC et al. Independent prognostic significance of ischemic ST segment response limited to recovery from tread mill exercise in asymptomatic subjects. Circulation June 1998; 97: 2117-2122.

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