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Original Research Article

Study of ECG Changes in Smokers Compared To Non Smokers

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Abstract

Smoking is one of the major cause of preventable morbidity and mortality in both developed and also in developing countries. Tobacco contains over 4000 chemical compounds and at least 400 toxins of which 60 are carcinogenic and its use is linked to over 25 diseases. The main cause of death related to smoking are atherosclerosis and cancer. Clinical observations in some areas shows that over 60% of heart diseases under 40 years of age are due to cigarette smoking. Regular smoking of any number of cigarettes is one of the important common risk factor for the development of myocardial infarction. The study is to see changes in ECG in smokers compared to nonsmokers. The study was conducted in young individuals of age between 18yrs to 25yrs .The individuals who does not smoke were selected as control and individuals who smoke are selected, ECG was recorded in control and smokers. Short term smoking of duration 02 to 05yrs produce significant cardiac electrical activity predisposing to cardiac risk. Study results showed that smokers are prone to cardiovascular risk. So in earlier stage 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.

Keywords: ECG, Smokers, Non smokers.

INTRODUCTION

Smoking is one of the major cause of preventable morbidity and mortality in both developed and also in developing countries. Smoking starts as a passion in youth and gradually becomes addiction. Smoking of dried tobacco leaves spread from America to the rest of the world after 16th century. Nicotine is the only alkaloid in the leaf. Earlier reports claimed amounts as great as 3.6mg per cigarette of which 90% of nicotine is to get absorbed. Tobacco contains over 4000 chemical compounds and at least 400 toxins of which 60 are carcinogenic and its use is linked to over 25 diseases. Tobacco contains dangerous psychoactive drugs and its effects are soothing and tranquilizing and also a stimulant action, physiological and psychological dependence occur. Smoking causes the most premature deaths in India. Consumptions of cigarettes increasing twice as fast in developed countries. In some parts of India over 70% of people will smoke. The main cause of death related to smoking atherosclerosis and cancer. Clinical observations in some areas shows that over 60% of heart diseases under 40 years of age are due to cigarette smoking. The major heart diseases are ischemic heart diseases, hypertension, thromboangitis obliterans. The cardiovascular responses to nicotine are due to stimulation of sympathetic ganglia and the adrenal medulla, together with the discharge of catecholamines from sympathetic nerve endings. Also contributing to the sympathomimetic response to nicotine is the activation of chemo receptors of the aortic and carotid bodies, which reflexly results in vaso constriction, tachycardia and elevated blood pressure [1]. Ventricular extra systoles occur. Cardiac output, work and oxygen consumption increase. Increased demand for blood flow that is not met because coronary vessels are narrowed by atherosclerosis may be a mechanism of tobacco – induced angina pectoris [2]. Cigarette smoking probably increases myocardial infarction through long term effects of atherosclerosis [3] and through readily reversible effects on hemostasis and the hemodynamic system [4]. The aim of the present study is to study ECG changes in smokers when compared to non smokers. Cigarette smoking has long been proved as a causative factor for coronary artery disease the aim is to evaluate ECG status of habitual smokers.

MATERIALS AND METHODS

The study was conducted after taking consent from thirty apparently healthy, male cigarette smokers of age group between 20-30 years and equal number of healthy age and sex matched controls. A smoker is defined as one who smokes one or more cigarettes daily for at least two years and a non smoker is defined as

one who had never smoked a cigarette at all. Duration of smoking was in the range of 2-5 years and cigarettes being smoked were in the range of 4-12 cigarettes per day. All subjects were non alcoholics, non – tobacco chewers, took no drugs and had a uniform pattern of diet and activity. They were not involved in any athletic or exercise programmed. They were sound physically and mentally .Their history and thorough clinical examination did not reveal any abnormality of any system. Smokers are instructed to refrain from smoking at last two hours prior to study of test. Procedure was explained to subjects and consent was taklen. Acute effect of smoking on electrocardiographic measurements was again determined in smokers, immediately after smoking are king size cigarette of gold flake filter brand of length 69mm. The instrument used to record electrocardiogram is the single channel electrocardiograph. The study was performed in physiology department. ECG;s were taken in healthy subjects and smokers. Smokers were advised to refrain from smoking at least for 2hrs before the recording was taken. After the ECG‟s were taken the smokers were asked to sit and they were given a cigarette (Goldflake filter king size) to smoke. As soon as they completed smoking the last puff of the cigarette, they were asked to lie down, lead connections were checked and ECG‟s were taken immediately to study the acute effects of smoking a cigarette. The ECG was evaluated for different parameters and results were drawn. Parameters considered were Heart rate, P wave, P-R interval, QRS complex, ST segment, Qt interval.

RESULTS

Heart rate

Heart rates are expressed in terms of beats/min. Heart rates are 77.2+ 6.386, 80.733 + 7.081, 86. 56 + 8.199 among controls, smokers before smoking and smokers immediately after smoking a cigaratte respectively (Table-1).

Heart rates were higher in smokers. Heart rate showed a statistically hightly significant value when controls were compared with smokers after smoking a cigratte and significant value when smokers were compared before and after smoking a cigarette.

Subjects	Heart rate (Mean + S.D.)
C - Controls	77.2 + 6.386
S1 - Smokers (Before smoking	80.733 + 7.081
S2 - Smokers (After smoking	86.567 + 8.199

Applying„ t statistic; C - S1: p > 0.05 (NS); C - S2: p > 0.05 (S) and also p < 0.001 (HS); S1 - S2: p < 0.05 (S)

P-wave

Duration

p wave duration (in seconds) of subjects are shown in Table-4 were 0.089 ± 0.010, 0.059 and 0.088 ± 0.018 in controls, smokers before smoking and smokers after smoking respectively. There is no increase or decrease in duration and there was no statistical significance (p > 0.05).

Amplitude

p wave amplitude were 1.03 ± 0.215, 1.05 ± 0.331 and 1.067 ± 0.341 respectively among controls, smokers before smoking and smokers after smoking shown in Table-4. There were no subjects with abnormal amplitudel. There is no statistical significance (p>0.05).

Table-2: P-Wave

Subjects	Duration (Sec) (Mean+ S.D)	Amplitube (in mm)
C-Controls	0.089+0.010	1.03±0.215
s1- Smokers (Before)	0.089+0.059	1.05±0.331
s2-Smokers (After)	0.088+0.018	1.067±0.341

Applying„ t‟statistic; C-s1: p>0.05 (NS); C-s2: p>0.05 (NS); s1-s2: p>0.05 (NS)

P-R interval

P-R interval measurements (in sec) in controls, smokers before smoking and smokers after smoking were 0.0146 + 0.019, 0.019 and 0.145 + 0.025 respectively (Table-3). One smoker had reduced P-R interval duration. But there was no statistical significance when the values of controls were compared with smokers before smoking and after smoking

Table-3: P-R Interval

Subjects	P-R Interval (in sec) (Mean ± S.D.)
C-Controls	0.146 ± 0.019
s1- Smokers (Before smoking)	0.145±0.019
s2-Smokers (After smoking)	0.145±0.025

Applying „t‟ statistic; c-s1 p>0.05 (NS); c-s2 p>0.05 (NS); s1_s2: p>0.05 (NS)

QRS complex

QRS complex measurements (in sec) were 0.082 + 0.009, 0.93 + 0.23 and 0.094 + 0.029 in controls, before smoking and after smoking a cigarette respectively (Table-4). Though two smokers had increase in QRS duration both before and after smoking there was no statistical significance ($p > 0.05$).

Table-4: QRS Complex

Subjects	QRS complex (in sec) (Mean ± S.D.)
C-Controls	0.146 ± 0.019
S1- Smokers (Before smoking)	0.093+ 0.009
S2-Smokers (After smoking)	0.094+ 0.029

Applying„ t‟statistic; C - S1 : $p > 0.05$ (NS); C - S2 : $p > 0.05$ (NS); S1 - S2 : $p > 0.05$ (NS)

ST segment

ST segment abnormality was not seen in controls. But three smokers had ST segment abnormality both before smoking and after smoking a cigaratte. By applying„ Z‟ statistic, it was statistically significant and smokers before smoking and after smoking. In smokers before and after smoking there was no difference and hence no statistical significance (Table-5).

Table-5: ST Segment

Subjects	Normal	Abnormal
C - Controls	30	0
S1 - Smokers (Before smoking	27	3
S2 - Smokers (After smoking)	27	3

Applying „Z‟ statistic; C - S1: $p > 0.05$ (NS); C - S2: $p > 0.05$ (NS); S1 - S2: $p > 0.05$ (NS)

T- wave

There were no T-wave changes in controls. But four smokers showed T -wave changes both before smoking and after smoking a cigarette. By applying „Z‟ statistic, it was statistically significant ($p>0.05$) when compared between controls and smokers before smoking and after smoking. In smokers before and after smoking there was no differene, hence there was no statistical significance (Table-6).

Table-6: Incidence of T-Wave Abnormality

Subjects	Normal	Abnormal
C-Controls	30	0
S1- Smokers (Before smoking)	26	4
S2-Smokers (After smoking	26	4

Applying „Z‟ statistic; C - S1: $p > 0.05$ (S); C - S2: $p > 0.05$ (S); S1 - S2: $p > 0.05$ (S)

QT_C interval

The QT_C interval values (in seconds) were 0.413 ± 0.012, 0.399 ± 0.028 and 0.420 ± 0.033 among controls smokers before smoking and smokers after smoking a cigarettestatistic. When„t‟wasappliedthere was statistical significance ($p < 0.05$) when values of controls were compared wit hsmokers before smoking and after smok-ing. But there was no statistical significance ($p > 0.05$) when values smokers before smoking and after smoking were compared (Table-7).

Table-7: QT_C Interval

Subjects	QT_ Interval
C- Controls	0.413 ± 0.012
S1- Smokers (Before smoking)	0.399 ± 0.028
S2-Smokers (After smoking)	0.402 ± 0.033

Applying „t‟ statistic; C - S1: $p < 0.05$ (S); C - S2: $p < 0.05$ (S); S1 - S2: $p < 0.05$ (NS)

DISCUSSION

Results from our study showed that heart rates were higher in smokers compared to non smokers and showed a statisticaly increase after smoking. Pickering and Sanderson [5] (1944-45) noted that smoking provokes anginal pain by raising blood pressure and increasing the heart rate. Boyle [6] and here collegues (1947) found in-crease in heart rate, blood pressure and cardiac output after intravenous injection of nicotine. P-wave duration and amplitude measurement doesnot show any statistically significant difference between the controls and smokers though there was slight decrease in duration and amplitude of p-wave in smokers. Cigarette smoking increases the velocity of conduction and shortens the effective refractive period at the AV node. Evan Fletcher *et al.*, [7] and Mujtaba FA [8]

found that in smokers there was little decrease in the duration of P-R interval after smoking a cigarette. But it was not statistically significant. The subjects in their study were habitual smokers between 20-45 years of age with more than 10 years of smoking habit. In our study there was no change in the duraiton P-R interval between controls and smokers. This may be because the subjects in our study were comparatively young and the duration of their smoking habit was only 2-5 years. QRS complex measurements in our study showed a slightly increase in smokers compared to controls. Mujtaba et al., found a slight decrease in QRS duration in smokers though it was not statistically significant. In a study by Cellina G. U *et al.*, [9] on „direct arterial pressure heart rate and ECG during cigarette smoking in unrestricted patient‟s conducted on nine patients, cigarette smoking caused angina pectoris in one individual and ECG showd ST segment depres-sion before the subjective apprectiation of pain. In our study, 3 subjects showed down sloping of ST segment. There was no ST segment variation seen in controls. Short term smoking of duration 2-5 years also produces significant ST segment changes. In our study four subjects showed T-wave inversions. The T-wave inversions was present both before smoking and after smoking. None of the controls had T-wave inversions. The T-wave inversions was present both before smoking and after smoking. None of the controls had T-wave inversion. These changes may be due to prolonged repolarisation leading to ischaemia [10]. Short term smoking of 2-5 years produces T-wave changes. QTC interval in the electrocardiogram reflect the time registered for depolarisation and repolarisation in the ventricular myocardium. Karjainen *et al.*, [11] in their study „QT interval on a cardiac risk factor in a middle aged population‟ reported that smoking was associated with shorter QTC than non smokers. I our study there was statistically significant decrease in QTC interval in smokers when compared to controls which indicates that smoking of 2-5 years of dura-tion produces significant decrease in QTC interval. But there was no significant change in QTC interval in smokers after smoking a cigarette.

CONCLUSION

Heart rates in smokers were higher than controls and it increased significantly immediately after smoking a cigarette. There is no significant change between controls and smokers in P-wave and P-R interval indicating that short term smoking of 2-5 years produces no change in atrial depolarization and AV nodal conduction time. Short term smoking of 2-5 years duration produces depolarization abnormalities which are represented as ST segment changes and T-wave changes. In our study ST segment depression was absorbed in three subjects and T-wave inversion was seen in four subjects. Short time smoking of 2-5 years duration produces a reduction in QTc interval. Short time smoking of 2-5 years duration produces a reduction in TP interval. Right bundle branch block was observed in some smokers in my study with short term smoking duration 2-5 years, but study in a larger population of cigarette smokers may confirm this. Short term smoking of duration 2-5 years produces significant change in cardiac electrical activity predisposing to cardiac risks. Our study results showed that smokers are prone to cardiovascular risk. So in earlier stage 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.

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