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Original Research Article.

A Comparative Study of ECG Intervals in Young Male Smokers and Drinkers in Tertiary Care Hospital

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ABSTRACT

Background: Tobacco & Alcohol consumption has many worst health outcomes. Tobacco is consumed in many forms and one such form is cigarette smoking. Tobacco use leads to sudden coronary death, chronic obstructive pulmonary disease, cancer, peripheral vascular disease, hypertension and the list is endless. Alcohol is consumed in hard drink form. Alcohol is use leads to liver disease, brain damage, heart disease, pancreatitis, cancer risk and the list is endless.

Aim: To evaluate ECG intervals in apparently healthy young male smokers compared to non-smokers & Drinkers compared to non-drinkers using a 12-lead ECG record.

Objective: To evaluate and compare variations in ECG waves, segments and intervals in apparently 150 healthy young male smokers , non-smokers and 150 drinkers, non-drinkers.

Materials and Methods: Descriptive comparative study over total duration of 24 months. For convenience 300 young, apparently healthy smokers non-smokers, Drinkers and non-drinkers in the age group of 18 - 35 yrs. were taken.

Settings and Design: Department of General Medicine OPD of Vedantaa Institute of Medical Sciences, Palghar (Tertiary Care Hospital).

Results: PR interval was significantly shortened among smokers & drinkers. No statistically significant difference in QRS interval between the two groups (Smokers & non-Smokers) but statistically significant difference in QRS interval between the two groups (Drinkers & Non Drinkers). QTc interval was slightly higher among smokers than non-smokers also drinkers than non-drinkers. TP interval was slightly higher among non-smokers than smokers also non-drinkers than drinkers. There is statistically significant decrease seen in smokers & drinkers.

Conclusion: Smoking & Drinking induces significant alterations in cardiac electrophysiology like shortening of PR and TP interval, prolongation of the QTc interval in apparently adult male individuals which may predispose to cardiovascular morbidity and mortality in the long run.

Keywords: ECG, Healthy Young, Smokers, Non-Smokers, Drinkers & Non-Drinkers.

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INTRODUCTION

According to World Health Report (2002)[1], tobacco is the most preventable cause of overall as well as cardiovascular mortality worldwide. It was estimated that there were 100 million deaths worldwide in the 20th century. The total number of tobacco users in the world has been estimated at 1.2 billion, which is expected to rise to 1.6 billion during year 2020's. At present, tobacco use causes death of 3.5 to 4 million people globally and expected to increase by about 10 million during year 2020.[2] It has also been estimated that by 2030, there will be more than 8 million deaths every year.[3]

Environmental factors are important in the pathogenesis of cardiovascular disease. Smoking, diet and physical activity are major ecological factors affecting cardiac health. The effect of alcohol intoxication on the electrocardiogram (ECG) is usually difficult to determine due to absent knowledge of the preexisting condition of the heart.[4] According to the World Health Organization (WHO), alcohol use causes about 2.5 million deaths annually and is the leading risk factor worldwide for deaths among males between 15 and 59 years of age.[5] Acute alcohol intake in otherwise healthy subjects may predispose to cardiac

arrhythmias.[6] Chronic heavy alcohol consumption can result in systolic and diastolic dysfunction, left ventricular dilatation, conduction abnormalities, and decreased ejection fraction resulting in alcoholic cardiomyopathy.[7,8] While light to moderate alcohol consumption (2 drinks/day, 28 g for males and 1 drink, 14 g/day for women) has shown to have cardioprotective benefits, binge drinking has been associated with cardiac dysfunction, stroke, arrhythmias and sudden cardiac death.[5] Up to 15-20% of the patients with acute alcohol intoxication have atrial fibrillation, while others may have different supraventricular and ventricular arrhythmia.[9] On the electrocardiogram (ECG), nonspecific variations from normal may include alterations in the ST segment, P-wave changes, complete or incomplete left bundle branch block or atrioventricular conduction disturbances.[10] We performed a best evidence review of the literature, and in this report, we describe the ECG changes associated with acute alcohol intoxication in otherwise healthy individuals.[11]

Smoking has resulted in two-fold increase in the risk of Coronary Artery Diseases. The consumption of nicotine is the single biggest avoidable cause of death and disability.

The World Health Organisation (WHO) predicts that 70% of the deaths from smoking-related illnesses will occur in low- and middle-income countries by 2020. Smokers are found worldwide, while tobacco chewers are restricted to South East Asia. Uttar Pradesh is the third largest cultivator of tobacco leaf in India. Tobacco is produced mainly in Mainpuri, Moradabad, Farrukhabad and Etah districts in UP. There are many cigarette and gutka factories in the state.[12]

Black HR et al.[13], Jonas MA et al.[14], Willete et al.[15], MMWR, US[16], and Price JF et al.[17] have conducted epidemiologic studies, which strongly support the assertion that cigarette smoking (CS) in both men and women increases the incidence of myocardial infarction (MI) and fatal coronary artery disease.

Tobacco consumption has many worse health outcomes. Tobacco is consumed in many forms and one such form is cigarette smoking. Its use leads to sudden coronary death, chronic obstructive pulmonary disease, cancer, peripheral vascular disease, hypertension and the list is endless.[18]

Nicotine also causes cardiac death by provoking ventricular arrhythmias.[19,20] The cardiac effects of nicotine are attributed to the release of catecholamines[21], 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.[21] Nicotine facilitates a conduction block and a re-entry and it increases the vulnerability to a ventricular fibrillation.[22] 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.[23] It contains nicotine, which causes physical and psychological dependencies.

Hence, nicotine and other components of cigarette smoking produce profound changes in the heart which can be assessed by doing Electrocardiography (ECG), which is the easiest and the cheapest method for assessing cardiovascular abnormalities. Maintaining abstinence from smoking as early as possible can prevent further damage. This study was aimed at creating awareness on the smoking hazards.

AIM

To evaluate ECG intervals in apparently healthy young male smokers compared to non-smokers & Drinkers compared to Nondrinkers using a 12-lead ECG record

OBJECTIVES

To evaluate and compare variations in ECG waves, segments and intervals in apparently 150 healthy young male smokers, non-smokers and 150 drinkers, non-drinkers.

MATERIALS AND METHODS

Type of Study

Descriptive comparative study.

Sample Size

Total- 300.

Inclusion Criteria

1. Young males between the age of 18 - 35 years visiting tertiary care centre.
2. Randomized controlled trials, case control studies; in English language confirming changes in ECG with alcohol & smoking habits.
3. Studies including human participants of adult age and gender with a detectable variation from a normal ECG.

Exclusion Criteria

1. Males diagnosed with hypertension.
2. History of cardiac, respiratory, renal and endocrine disorders.
3. History of consumption of psychoactive substances.
4. Family history of hypertension and smoking.
5. History of cardiac diseases.
6. History of anxiety.
7. History of depressive disorders.
8. Passive smokers & drinkers.

Duration of Study

Total Duration: 24 months.

Study Setting

Medicine OPD of Vedantaa Institute of Medical Sciences, Palghar (Tertiary Care).

Cases: 150 young, apparently healthy smokers & Drinkers in age group of 18-35 yrs.

Controls: 150 young, apparently healthy Non-Smokers & Non-Drinkers in age group of 18-35 yrs.

Source of Data

Cases were apparently healthy male smokers & drinkers between ages 18-35 years, selected from among students and staff of the institute, and attendants of patients visiting outpatient departments at the hospital. Non-smoking & Non-drinking male controls of the same age group were selected from the same pool. The nature and purpose of the study was described to the subjects and informed written consent was obtained from those willing to participate in the study.

A pre-structured proforma was given to each subject to record personal details and pertinent medical history from both cases and controls. Details of smoking & drinking habit, that is duration and quantum of smoking & drinking, were obtained from cases. For each subject in the case group, number of pack years was calculated & number of bottles year was calculated. One pack year= 20 cigarettes smoked per day for a duration of one year & One bottle year = one bottle alcohol drinker per day for a duration of one year.

Physical examination included measurement of weight in kilograms, pulse rate was recorded by palpating radial artery and blood pressure recording with a mercury sphygmomanometer. Clinical examination of the cardiovascular and respiratory systems was done in detail.

The subjects were asked to visit the Outpatient Department of General Medicine, in the morning hours between 9AM to 1PM. Each subject was allowed to rest in supine posture for 20 minutes before recording was done. After a period of rest, pulse rate was recorded in beats per minute by palpating radial artery for one minute. Blood pressure was measured using mercury sphygmomanometer from the right upper arm with the subject in supine position with 3 readings at the interval of 5 mins were obtained and mean BP was taken into consideration.

Following the standard procedure, 12-lead electrocardiograms were recorded using Magic R 12-channel Electrocardiograph designed by Medline's team of biomedical engineers.

The ECG was evaluated for different intervals like PR interval (0.120 - 0.200 sec), QRS, QT interval, QTc interval (Male- < 0.42, Female- < 0.44 sec) and TP interval.

Statistical Analysis

The data was compiled in Microsoft Excel and analysed using SPSS (Statistical Package for Social Sciences) version 23. Level of significance was fixed at $p < 0.05$ with student's 't' test.

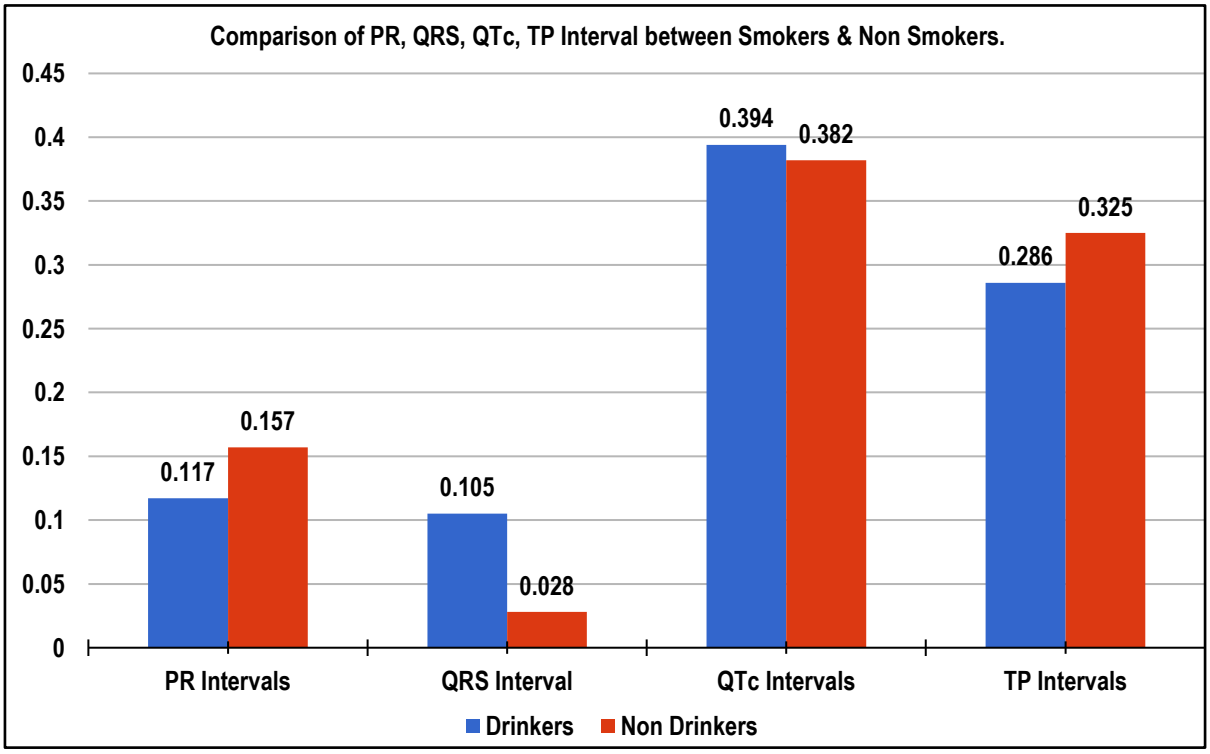
Table 1: Comparison of PR, QRS, QTc, TP Interval between Smokers & Non Smokers.

Measurement	Smokers (Mean +/- S.D.)	Non Smokers (Mean +/- S.D.)	P Value
PR Intervals	0.159 + / - 0.005	0.167 + / - 0.002	< 0.0001
QRS Interval	0.068 + / - 0.121	0.085 + / - 0.0023	0.226
QTc Intervals	0.48 + / - 0.025	0.42 + / - 0.021	< 0.0001
TP Intervals	0.32+ / - 0.021	0.37 + / - 0.014	< 0.0001

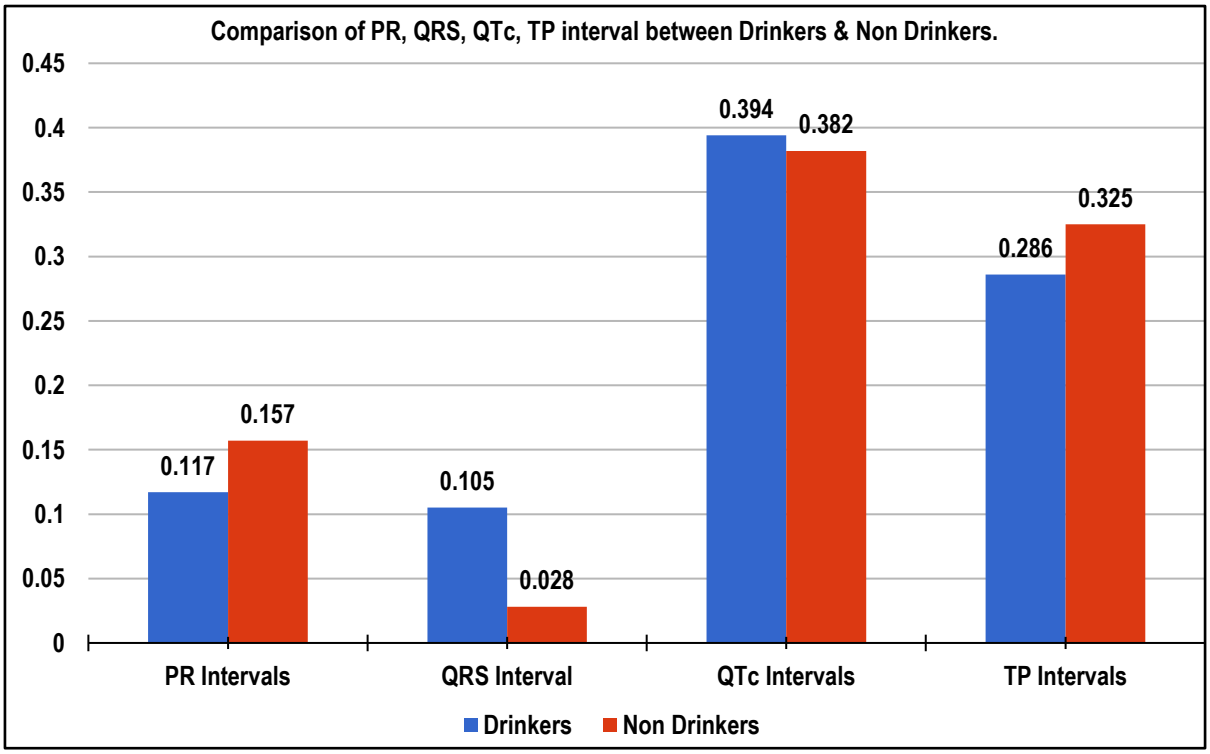
Table 2: Comparison of PR, QRS, QTc, TP interval between Drinkers & Non Drinkers

Measurement	Drinkers (Mean +/- S.D.)	Non Drinkers (Mean +/- S.D.)	P Value
PR Intervals	0.117 + / - 0.004	0.157 + / - 0.001	< 0.0001
QRS Interval	0.105 + / - 0.075	0.028 + / - 0.089	< 0.0001
QTc Intervals	0.394 + / - 0.031	0.382 + / - 0.017	0.0038
TP Intervals	0.286 + / - 0.013	0.325 + / - 0.009	< 0.0001

Graph 1: Comparison of PR, QRS, QTc, TP interval between Smokers & Non-Smokers.



Graph 2: Comparison of PR, QRS, QTc, TP interval between Drinkers & Non-Drinkers.



RESULTS

Table 1 shows that, PR interval was slightly higher among non-smokers (0.167 sec) than smokers (0.159 sec) but there was statistically significant among smokers & non-smokers (p<0.0001).

QRS interval was slightly higher among non-smokers (0.085 sec) than smokers (0.068 sec) but there was not statistically significant among smokers & non-smokers (p<0.226).

QTc interval was slightly higher among smokers (0.48 sec) than non-smokers (0.42 sec) but there was statistically significant among smokers & non-smokers (p<0.0001).

TP interval was slightly higher among non-smokers (0.37 sec) than smokers (0.32 sec) but there was statistically significant among smokers & non-smokers (p<0.0001).

Table 2 shows that, PR interval was slightly higher among non-drinkers (0.157 sec) than drinkers (0.117 sec) but there was statistically significant among drinkers & non-drinkers (p<0.0001).

QRS interval was slightly higher among drinkers (0.105 sec) than non-drinkers (0.028 sec) but there was statistically significant among drinkers & non-drinkers (p<0.0001).

QTc interval was slightly higher among drinkers (0.394 sec) than non-drinkers (0.382 sec) but there was statistically significant among drinkers & non-drinkers (p<0.0038).

TP interval was slightly higher among non-drinkers (0.325 sec) than drinkers (0.286 sec) but there was statistically significant among drinkers & non-drinkers (p<0.0001).

DISCUSSION

In this present study, PR interval was slightly higher among non-smokers as compared to smokers as well as non-drinkers as compared to drinkers. This resultant finding was in agreement with Prashantbabu et al and other studies,[24,25] but PR interval was more in smokers than non- smokers in study done by Salman S Siddiqui et al, Swati K et al, Amit Shrivastav et al and Venkatesh G et al.[26,27]

In our study QRS interval was slightly higher among non-smokers as compared to smokers. But there was not a statistically significant difference between Smokers and Non-Smokers. Other studies have demonstrated short QRS interval done by Salman S Siddiqui et al, Amit Shrivastav et al, Swati K et al but Venkatesh G et al and MR Renukadevi et al have observed that QRS interval was more in cases than contols.[26-28]

QRS interval was slightly higher among drinkers as compared to non-drinkers. But There was a statistically significant difference between drinkers and non-drinkers in our study.

In our study, QTc interval was slightly higher among smokers as compared to non-smokers. Also QTc interval was slightly higher among drinkers as compared to non-drinkers. Similar finding was reported by Prashanthbabu et al, Venkatesh G et al, Amit Shrivastav et al and Arvind Thangarasa et al.[28-30], but it was found that QTc was shorter in cases than controls by MR Renukadevi et al. The Ventricular repolarization is altered in young male cigarette smokers. The differences in the heterogeneity of ventricular repolarization between smokers and non- smokers are mainly due to heart rate differences between the 2 study groups.[31]

In our study TP interval was slightly higher among non-smokers as compared to smokers, also non-drinkers as compared to drinkers. In ECG strip if heart rate would increase in distance between T-wave (end of one cardiac cycle) and P-wave starting of new cardiac cycle would decrease and is the cause of shortening of TP interval, which was found similar in study done by Salman S Siddiqui et al, Amit Shrivastav et al and Venkatesh G et al.[30,31]

CONCLUSION

Smoking & Drinking induces significant alterations in cardiac electrophysiology like shortening of PR and TP interval, prolongation of the QTc interval in apparently adult male individuals which may predispose to cardiovascular morbidity and mortality in the long run.

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