Research Article

Electrocardiogram changes and heart rate variability during moderate exercise in chronic alcoholics

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Introduction

“Alcoholic” is a person, who has a compulsion to drink, exhibits prominent “drink-seeking behavior,” becomes tolerant to alcohol, and has obvious physical, psychological, and social problems related thereto.[1] Alcohol is a psychoactive drug, which has a depressant effect. High alcohol content in the blood reduces the attention and slows the reaction speed.[2] Alcoholism is called dual disease as it includes both mental and physical components. Significant alcohol intake produces changes in the brain’s structure and chemistry.[3]

Chronic and heavy alcohol consumption has deleterious effects on the cardiovascular system and may lead to progressive, chronic cardiac dysfunction such as heart muscle disorders (cardiomyopathy), heartbeat rhythm irregularities (arrhythmias), high blood pressure (hypertension), and stroke.[3] Most of the previous observations relate the abnormal electrocardiogram (ECG) to the organic myocardial diseases. However, ECG changes may occur in chronic alcoholic persons without any corresponding cardiomyopathy.[4]

Physical activity is defined as “any bodily movement produced by skeletal muscles, which results in energy expenditure.” Exercise capacity is one of the major determinants of cardiovascular health and its risk. It substantially affects the interpretation of a positive stress test. A few of previous studies have shown the presence of abnormal ECG at rest and during exercise in different pathological conditions but not essentially in apparently healthy chronic alcoholic persons.[5]
Thus, this study was conducted to establish the ECG changes and heart rate variability (HRV) in chronic alcoholic persons during moderate exercise and to compare the changes with nonalcoholic control individuals.

Materials and Methods

This study was conducted by monitoring and recording ECG in a “DYNATRAC” during moderate exercise on a treadmill test system in study subjects (chronic alcoholic persons) and controls (nonalcoholic persons), residing in and around Hubli city, Karnataka, India. The study was conducted in the Department of Physiology with the assistance of Medicine Department, Karnataka Institute of Medical Sciences (KIMS), Hubli, Karnataka, India. The study and its conduct were cleared by the ethical committee, KIMS, Hubli, Karnataka, India.

The study group consisted of 50 men, aged between 30 and 50 years with alcohol intake for more than 5 years of duration, without any known cardiac illness. Similarly, 50 nonalcoholic individuals of the same age, sex, and anthropometrically matched constituted the control group.

All the inclusion and exclusion criteria were considered while selecting the study subjects. The approximate number of alcoholic drinks consumed per month and duration of consumption was considered. Height and weight of each individual was recorded, and BMI was calculated. Vital parameters such as pulse rate and arterial blood pressure were recorded. A detailed clinical examination of respiratory, cardiovascular, abdomen, and central nervous system was conducted.

A stress test (exercise ECG test) was used to determine how well the heart and lungs function during physical activity. During the test, the subject was made to walk on a treadmill (STS DYNATRAC—USB-based treadmill test system—software version 3.8), while heart rate, blood pressure, and ECG were monitored. As the test progresses, the level of exertion gradually increased. The level of exercise was ascertained based on the pulse rate.

Statistical Analysis

Statistical analysis of the ECG changes and HRV in alcoholic persons and controls were done by Student’s t test. P-value < 0.05 was considered as “significant” in this study.

Results

Important clinical characteristics of ECG and HRV in study (alcoholic persons) and control (nonalcoholic persons) groups are presented in this section.

The mean (±SD) QT interval during exercise in control group was 0.41 ± 0.04 s and, in alcoholic persons, 0.46 ± 0.02 s. Thus, QT interval was significantly increased in alcoholic persons, compared with that of control group. Similarly, the mean (±SD) QTc interval during exercise in control group was 0.41 ± 0.05 s and, in alcoholic persons, 0.46 ± 0.04 s. Thus, QTc interval significantly increased in alcoholic persons, compared with that of control group [Table 1].

The mean (±SD) QRS frontal axis during exercise in control group was 63.10° ± 28.70° and, in alcoholic persons, 58.60° ± 31.00°. Thus, QRS frontal axis was significantly unchanged in alcoholic persons, compared with that of control group [Table 1].

The mean (±SD) HRV during exercise in control group was 4.1 ± 1.3 beats/min and, in alcoholic persons, 4.8 ± 1.0 beats/min. Thus, HRV was insignificant in alcoholic persons, compared with that of control group [Table 1].

Discussion

Excessive consumption of alcohol in the absence of underlying organic heart disease may produce electrocardiographic abnormalities. These abnormalities may imitate the changes produced by coronary artery diseases, although the prognostic significance of the abnormal ECG

Table 1. Comparison of ECG changes and HRV in alcoholic persons during moderate exercise with ECG and HRV of controls (nonalcoholic persons)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Alcoholic persons</th>
<th>t-value</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>P wave (s)</td>
<td>0.09 ± 0.01</td>
<td>0.08 ± 0.01</td>
<td>0.950</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>QRS complex (s)</td>
<td>0.08 ± 0.01</td>
<td>0.09 ± 0.03</td>
<td>0.918</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>T wave (s)</td>
<td>0.09 ± 0.02</td>
<td>0.1 ± 0.02</td>
<td>1.697</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>PR interval (s)</td>
<td>0.11 ± 0.01</td>
<td>0.13 ± 0.02</td>
<td>0.491</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>ST segment (s)</td>
<td>0.33 ± 0.01</td>
<td>0.38 ± 0.02</td>
<td>1.926</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>QT interval (s)</td>
<td>0.41 ± 0.04</td>
<td>0.46 ± 0.02</td>
<td>2.807</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>QTc interval (s)</td>
<td>0.41 ± 0.05</td>
<td>0.45 ± 0.04</td>
<td>4.362</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>TP interval (s)</td>
<td>0.05 ± 0.05</td>
<td>0.04 ± 0.04</td>
<td>0.081</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>QRS frontal axis (°)</td>
<td>63.10 ± 28.70</td>
<td>58.60 ± 31.00</td>
<td>0.753</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>125.56 ± 20.97</td>
<td>130.30 ± 22.70</td>
<td>1.084</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>HRV (beats/min)</td>
<td>4.1 ± 1.3</td>
<td>4.8 ± 1.0</td>
<td>1.593</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD (n = 50).
would be quite different.[6] Electrocardiographic findings in asymptomatic alcoholic persons are frequently abnormal, and these findings may be the only indication of heart disease in these individuals.[7]

In a previous study by Trejbal and Mitro, it was concluded that the ECG pattern very often shows nonspecific repolarization changes in ischemia, which is mostly asymptomatic in the form of silent myocardial ischemia and worsened in alcohol-intoxicated patients with ischemic heart disease. The resulting ECG may be influenced by many conditions, which often associates with inebriety, such as hypothermia and hypoglycemia to a large extent. There is convincing evidence that, not only the chronic alcoholism but also single episode of excessive alcohol consumption is associated with increased cardiovascular mortality.[8]

In this study, there were significant ECG changes observed in chronic alcoholic persons during moderate exercise. In these individuals, ST segment, QT interval, and QTc interval increased significantly compared with nonalcoholic persons.

This finding, ST segment prolongation, is contradictory to the general observation that, during exercise, there is a linear relationship between the changes in ST segment duration (shorten) and heart rate (increase). Previous study by Morvai et al.[9] showed that the T-wave irregularities and intraventricular conduction disturbances being the most common features in alcoholic persons.

QRS complex and ST interval represents the ventricular depolarization and repolarization, which involves the inter-ventricular septum and simultaneous left and right ventricular stimulations. Sympathetic stimulation increases conduction velocity, whereas ischemia tends to decrease the conduction velocity by slowing the rapid upstroke (phase 0) of the ventricular action potential. It has been postulated that the differences in QRS wave duration from rest to exercise might serve as a marker of ischemia. A subtle prolongation of QRS duration in alcoholic persons during exercise was demonstrated in a study by Ahnve et al.,[10] in which they concluded that a consistent increase in QRS duration and ST interval in alcoholic persons during exercise, although subtle, may be a marker of ischemia and consequently a potential diagnostic tool.

QT interval and QTc in the ECG include both ventricular depolarization and repolarization times and vary inversely with the heart rate. The correlation of an abnormally prolonged QTc with a disturbance of long axis lengthening, thus, represents an interrelation between electrical and mechanical events that are occurring synchronously. It is, therefore, possible for a causal relation to exist between the two, with abnormal mechanical activity modifying the time course of ventricular repolarization. Because diastolic disturbances are the earliest mechanical abnormalities to appear with ischemia, it is possible that the abnormal lengthening of QT and QTc might be a marker of ischemia.[11]

A study conducted by Kochegurov included the recording of ECG at rest and during the step test in 231 alcoholic patients and clinically normal male individuals. The most common ECG changes recorded at rest were shortened PQ interval, lengthened QT interval, rhythm and conductivity disorders, and flattened P waves. However, typical exercise-associated changes were elevated systolic parameters. As alcoholism progressed, there was an obvious tendency to increase incidence of conductivity disorders, flattened P waves, and elevated systolic parameters during exercise.[12]

However, in this study, it was observed that the important ECG changes in chronic alcoholic persons include increase in ST segment duration and lengthening of QT an QTc interval during moderate exercise. Furthermore, there is no QRS frontal axis deviation in alcoholic persons compared with the nonalcoholic persons. The probable reasons for only these findings in ECG may be the duration of alcohol consumption of alcoholic persons of this study.

HRV, the variation over a time period between consecutive heart beats, is predominantly dependent on the extrinsic regulation of the heart rate. It is a noninvasive electrocardiographic marker, reflecting the activity of the sympathetic and vagal components of the ANS on the sinus node of the heart. It expresses the total amount of variations of both instantaneous heart rate and RR intervals. In moderate exercise, the decrease in HRV can be attributed to a withdrawal in parasympathetic activity and an augmented sympathetic activity.[13]

In this study, there was a decrease in HRV in both the alcoholic and nonalcoholic persons during exercise compared with its levels at rest. However, this reduction in HRV was not significantly different in alcoholic persons compared with that of nonalcoholic persons. This association between alcohol intake and decreased HRV during exercise may be secondary to an increase in heart rate rather than a central or peripheral effect of alcohol on cardiac vagal activity. This study is different in a way that we have established the HRV and ECG changes in chronic alcoholic persons in a single study. However, a better result could be ascertained had we considered alcoholic subjects of history of more than 10 years of consumption in this study.

Conclusion

This study revealed that the alcohol consumption for more than 5 years of duration produces significant changes in ECG (prolongation of ST, QT, and QTc intervals but not the HRV) compared with nonalcoholic persons during moderate exercise. These changes may be considered as an early indicator of the effects of alcohol abuse on cardiovascular system. Precautions need to be taken in the alcohol abusers in the form of complete stoppage of alcohol consumption or moderation of alcohol consumption to prevent the onset of irreversible alcohol-induced cardiovascular diseases.

References


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