

## **Electrocardiogram (ECG) as a diagnostic tool for the assessment of Cardiovascular status in alcoholics**

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### **Abstract**

**The Electrocardiogram (ECG or EKG) is a graphic recording of electric potentials generated by the heart. It is a simple and noninvasive, inexpensive test helps in assessing the cardiovascular status. Alcohol consumption causes ECG changes. Therefore this study is undertaken to detect the ECG changes in alcoholics and thereby assessing the cardiovascular status. The study protocol was explained to the participants and informed consent taken. 50 Alcoholic men of age group above 20years, confirming to DSM-IV Criteria were randomly selected from the general population. 50 age matched Controls were taken for the study. The groups were subdivided into three groups according to age (in years) as 20–39 (n=22), 40-49 (n=16) and > 50 (n=12). The ECG was recorded in resting state in lying down position. The ECG results were analyzed for Heart rate, P wave, PR interval, QRS duration, QT<sub>C</sub> interval, frontal axis, ST segment, T wave and TP interval. The results showed significantly increase in Heart rate and decrease in PR interval, QRS complex and TP intervals in alcoholic subjects. On comparison of subgroups, alcoholic subjects do not show statistically significant results. Our study results show cardiovascular risk in alcoholics. Therefore, earlier detection of ECG changes is useful in preventing the cardiovascular risk.**

**Key words:** Electrocardiogram, alcoholism, heart rate, age

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### **Introduction**

The Electrocardiogram (ECG or EKG) is a graphic recording of electric potentials generated by the heart. It is a simple and noninvasive, inexpensive, and highly versatile test helps in assessing the cardiovascular status. It is useful in detecting arrhythmias, conduction disturbances, myocardial ischemia and metabolic disturbances. The ECG waveforms are labeled alphabetically, beginning with the P wave, which represents atrial depolarization. The QRS complex represents ventricular depolarization, and the ST-T-U complex (ST segment, T wave, and U wave) represents ventricular repolarization [1].

Global alcohol consumption has increased in recent decades with most of all of this increase occurring in developing countries [1]. Alcohol consumption causes ECG changes which include cardiac conduction abnormalities, prolongation of the QT interval, prolongation of ventricular repolarization and sympathetic stimulation [2]. Sinus tachycardia or a Supraventricular arrhythmia, commonly atrial fibrillation and non-specific ST-T changes are also

observed in alcoholics [3]. Alcohol alters the endocrinal function like increase in adrenocorticotrophic hormone, oxytocin and electrolytes, which may indirectly causes myocardial damage [4]. In a cross-sectional study, habitual alcohol intake was positively associated with Blood Pressure and Heart Rate compared with nondrinkers [5]. Studies have showed that alcohol abuse in men younger at 40 with a first myocardial infarction [6]. Moderate dose of alcohol intake is associated with an increase in maximum and the minimum P-wave duration which have been reported to represent an increased risk for atrial fibrillation in patients with no underlying disease [7]. Cardiomyopathy associated with ECG changes like sinus tachycardia or a supraventricular arrhythmia, commonly atrial fibrillation with a rapid ventricular response and non-specific ST-T changes [3]. Alcohol, through its effect on electrolyte transport, could produce T-wave alterations [8].

Low voltage of the T waves is not unusual in old age, and is not necessarily abnormal. The aging myocardium is also liable to cause disorders of rhythm, ectopic Extrasystoles are a very common incidental finding at all ages,

they signify early myocardial degeneration or coronary sclerosis, so that some further assessment of their significance is important [9]. Therefore this study is undertaken

## Materials and Methods

This is a case control study undertaken in the department of Physiology JJMMC, Davangere. 50 Alcoholic men of age group above 20years, confirming to DSM-IV Criteria were randomly selected from the general population. 50 age matched Controls were taken for the study. The groups were subdivided into three groups according to age (in years) as 20–39 (n=22), 40-49 (n=16) and > 50 (n=12). The subject's detailed history was taken. A detailed physical and systemic examination was done. Height, weight, pulse rate, blood pressure, respiratory rate were recorded. Body Mass Index (BMI) was calculated based on height and weight.

Following detailed assessment of the subjects, they were screened for the presence of inclusion and exclusion criteria and dropped if any of exclusion criteria were present.

### Inclusion Criteria

Normal healthy individuals.

Men aged above 20 years consuming alcohol confirming to DSM-IV Criteria. [10].

### Exclusion Criteria

Subjects aged below 20 years.

Subjects associated with Hypertension, Diabetes mellitus and cardiovascular diseases like arrhythmias, MI etc.

Subjects on drugs or other substances which affect conducting system of the heart e.g digitalis, beta blockers, cigarettes etc.

Female are excluded because drinking was more prevalent in men than women cardiovascular diseases [11, 12].

The subjects were made to answer CAGE questionnaire [13] that has been proven helpful in making the diagnosis of alcoholism. Though simple, the questionnaire has been shown to be extremely sensitive and specific. A positive answer to two or more questions should create high index of suspicion for alcoholism.

### Recording of ECG

The subjects were made to rest for 5 minutes in supine position. All the electronic gadgets were taken away. A 12 lead electrocardiogram (Cardiant 108-T-MK-VI manufactured by BPL Electronics Ltd.) was recorded at 25mm/sec and labeled with subjects name and age. It was later analyzed for Heart rate, P wave, PR interval, QRS duration, QT<sub>C</sub> interval, frontal axis, ST segment, T wave and TP interval.

to detect the ECG changes in alcoholics and thereby assessing the cardiovascular status.

### Statistical Methods

Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean  $\pm$  SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance. Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups (Inter group analysis) on metric parameters, Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups. Analysis of variance has been used to find the significance of parameters across the age groups in alcoholics.

## Results

The results of this study are summarized in Tables 1–4. Mean age of controls and alcoholics were  $35.8 \pm 10.1$  years and  $40.8 \pm 10.9$  years respectively. Table 1 shows height, weight and Body Mass Index (BMI) measurements of controls and alcoholics subjects. BMI was calculated based on height and weight. Values are within the normal range and there was no statistical significance

**Table 1.** Comparison of Height, Weight, BMI between Controls and Alcoholics

Variables	Control	Alcoholic	P value
Height in cm	169.30 $\pm$ 8.50	168.92 $\pm$ 5.70	0.794
Weight in kg	58.30 $\pm$ 7.34	58.32 $\pm$ 9.06	1.000
BMI (kg/m <sup>2</sup> )	20.36 $\pm$ 2.21	20.48 $\pm$ 3.16	0.829

The electrocardiographic findings of controls and alcoholics subjects are listed in Table 2. Heart rate showed a statistically significant increase in alcoholic groups compared to controls. Values of P-wave duration and amplitude do not show any statistical significance. P-R interval and QRS complex measurements showed statistically significant reduction in alcoholic group. Both Isoelectric (IE) and Nonspecific ST segment (NSST) values are within normal range and does not have any statistical significance. QT<sub>C</sub> interval, frontal axis and T-wave values are within normal limits. Although two subjects showed axis deviation of -30 and it was not statistically significant

**Table 2.** Comparison of ECG report between Controls and Alcoholics

ECG report		Normal	Alcoholic	P value
Heart Rate	Beats/min	75.98±6.89	81.14±10.99	0.006**
	Duration	0.086±0.012	0.0802±0.021	0.105
P wave	Amplitude	1.02±0.20	0.98±0.30	0.438
P-R Interval	In seconds	0.143±0.02	0.131±0.02	0.002**
QRS complex	In seconds	0.076±0.01	0.055±0.01	<0.001**
ST Segment	IE	50(100.0%)	46(92.0%)	0.117
	NSST	0	4(8.0%)	
QTc interval	In seconds	0.404±0.06	0.403±0.08	0.893
T wave	Normal	50(100.0%)	48(96.0%)	0.117
	Abnormal	0	2(4.0%)	
QRS Frontal Axis	In degrees	51.0 ± 23.7	43.8 ± 22.9	0.662
TP Interval	In seconds	0.29±0.04	0.24±0.08	<0.001**

Mean ± SD (Standard Deviation)

Suggestive significance (P value: 0.05 < P < 0.10)

\*Moderately significant (P value: 0.01 < P ≤ 0.05)

\*\* Strongly significant (P value : P ≤ 0.01)

IE: Isoelectric

NSST: Non specific ST segment

T-P interval values shows statistically significant decrease in alcoholic group compared to controls.

In Table 3 Height, weight and Body Mass Index (BMI) readings of alcoholics of different age groups, 20–39, 40–49 and > 50 years were shown. On comparison of all the three subgroups these dependent variables are within normal range and do not show any statistical significance.

Table 4 shows ECG report of Alcoholics of 20–39, 40–49 and > 50 age groups. On comparison of these subgroups,

electrocardiographic readings of Heart rate, P wave, PR interval, QRS duration, QT<sub>C</sub> interval, frontal axis, ST segment, T wave and TP interval values are within normal limits and p value is >0.05 and thus the report does not have any statistical significance.

**Table 3.** Comparison of Height, Weight, BMI in Alcoholics for different age groups

Variables	Age in years			P value
	20-39 (n=22)	40-49 (n=16)	50 & above (n=12)	
Height in cm	167.32±5.96	169.06±5.36	171.67±4.97	0.102
Weight in kg	56.68±7.03	59.62±10.71	59.58±10.24	0.536
BMI (kg/m <sup>2</sup> )	20.34±2.99	20.97±3.53	20.20±3.13	0.832

**Table 4.** Comparison of ECG findings in Alcoholics for different age groups

ECG report		Age in years			P value
		20-39 (n=22)	40-49 (n=16)	50 & above (n=12)	
Heart rate	beats/min	80.63±11.38	83.75±11.11	78.58±9.78	0.453
	Duration	0.083±0.02	0.079±0.02	0.077±0.02	0.729
P wave	Amplitude	0.91±0.29	1.06±0.36	1.00±0.21	0.299
P-R Interval	In seconds	0.130±0.02	0.130±0.02	0.133±0.013	0.857
QRS complex	In seconds	0.057±0.015	0.053±0.010	0.053±0.009	0.435
	IE	20(90.9%)	16(100.0%)	10(83.3%)	NS
ST Seg	NSST	2(9.1%)	0	2(16.7%)	
QTc	In seconds	0.39±0.05	0.42±0.14	0.38±0.04	0.484
T wave	Normal	22(100.0%)	15(93.8%)	11(91.7%)	0.309
	Abnormal	0	1(6.2%)	1(8.3%)	
QRS Frontal Axis	In degrees	22	17	12	0.147
TP Interval	In seconds	0.26±0.08	0.23±0.09	0.24±0.05	0.488

Mean ± SD (Standard Deviation)

Suggestive significance (P value: 0.05 < P < 0.10)

\* Moderately significant (P value: 0.01 < P ≤ 0.05)

\*\* Strongly significant (P value: P ≤ 0.01)

IE: Isoelectric;

NSST: Non specific ST segment

## Discussion

Many workers did the work on effects of alcohol on electrocardiogram and showed positive results throughout the world over a long period of time. Excessive consumption of alcohol, in the absence of underlying organic heart disease, may produce electrocardiographic abnormalities. These may at times imitate the changes produced by coronary artery disease, but the prognostic significance of the abnormal electrocardiogram would be quite different [8].

Ryan and Howes [14] in their study showed alcohol consumption is associated with reduced vagal activity, may be mainly due to a positive association between alcohol intake and heart rate in the age group of 33–68yrs.

Tetsuya Ohira et al [5] showed habitual alcohol intake was positively associated with Heart Rate compared with nondrinkers. This increase in heart rate could be due to decrease in peripheral resistance and increase in cardiac output. Our study results are also similar showing significant increase in heart rate in alcoholics.

The PR interval reflects the time needed to activate the atria, to conduct the impulse to the AV node and His bundle and start the ventricular depolarisation. QRS complex is because of ventricular depolarization. Lorscheid A et al [15] showed that prolongation of the PR interval and QRS complex after acute ingestion of alcohol. In contrast our

study there is statistically significant reduction P-R interval and QRS complex measurements.

QT<sub>C</sub> interval in the electrocardiogram includes both ventricular depolarization and repolarization times and varies inversely with the heart rate [1]. Lorscheid A et al [15] studied acute effect of alcohol in healthy individuals and showed that QT<sub>C</sub> prolongation was seen in the subjects. In our study QT<sub>C</sub> interval does not show any statistical significance.

ST segment deviation from isoelectric line is a predictor of future coronary events in asymptomatic population. Klatsky [3] reported increasing intake of alcohol leads to cardiomyopathy and non specific ST-T changes in ECG. In our study three subjects showed non-specific ST changes in alcoholic group.

Robertson [9] in his study tells that as the age advances myocardium is also liable to cause disorders of rhythm, auricular fibrillation and low voltage of the T waves which may be due to early myocardial degeneration or coronary sclerosis. In our study on comparison of different age groups of alcoholic subjects does not show any statistically significant results indicating that electrocardiographic findings were similar in all 20–39, 40–49 and > 50 age groups.

In our study there is statistically significant decrease of TP interval in alcoholic group. TP interval change may be due to increased heart rate.

## Conclusion

Excessive consumption of alcohol, in the absence of underlying organic heart disease, may produce electrocardiographic abnormalities. In our study Heart rate was significantly increased indicating reduced vagal activity. There is reduction of P-R interval and QRS complex reflecting the reduced spreading of depolarization from the sinus node to the atria and ventricles. TP interval reduction may be due to increased heart rate. QT<sub>C</sub> interval does not show significant changes indicating no change in ventricular depolarization and repolarization. QRS frontal axis and ST segment does not show statistically significant results. In our study on comparison of different age subgroups of alcoholic subjects does not show any statistically significant results indicating that electrocardiographic findings were similar in all the age groups.

Our study results showed that alcoholics are prone for cardiovascular risk. Therefore, earlier detection of ECG changes is useful in preventing the cardiovascular risk.

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