INTRODUCTION

Alcoholism is one of the greatest epidemics of modern times, is a chronic progressive often fatal disease, which is more common in males of our population. Alcohol, specifically ethanol, is a potent CNS depressant with a range of effects on all systems particularly on Autonomic Nervous System. Cardiac automaticity is intrinsic to pacemaker tissue; heart rate and rhythm being largely under the control of autonomic systems.HRV refers to the beat to beat alteration in heart rate, i.e., the oscillation in the interval between consecutive heart beats as well as the oscillations between consecutive instantaneous heart rate. There has been no quantitative marker for Cardiovascular Autonomic Function. Both binge drinking and long-term heavy drinking can lead to strokes, even in people without coronary heart disease. Recent studies show that people who binge drink are about 56 percent more likely than people who never binge drink to suffer an ischemic stroke over 10 years. Binge drinkers also are about 39 percent more likely to suffer any type of stroke than people who never binge drink. In addition, alcohol exacerbates the problems that often lead to strokes, including hypertension, arrhythmias, and cardiomyopathy. Studies in the last 30 years have shown a great significance between ANS and CV morbidity, including sudden death and malignant arrhythmias [1]. Heart rate variability represents the hallmark of such markers. There is a significant decrease in the high frequency power of HRV analysis in Alcohol Dependant individuals compared with the normal subjects [2]. Based on previous studies, HRV analysis correlation was done in Alcohol dependant subjects with normal healthy subjects using Time domain parameters to rule out the possible variations accordingly.

MATERIALS AND METHODS

The Present study was conducted in the Department of Physiology in Stanley Medical College.
on 30 alcoholic depended patients and 30 controls after obtaining clearance from the ethical committee. Cases include Alcohol dependant individuals were diagnosed based on ICD-10 criteria. The age group of both alcoholic dependent subjects and controls were around 18-25 years.

Inclusion Criteria: 1) Age > 18 years, 2)Patients diagnosed to have Alcohol Dependence Syndrome based on ICD -10 Criteria, 3) Non smokers, not on any drugs.

Exclusion Criteria: 1) Age < 18 years, 2) H/0 Autonomic Dysfunction, 3) H/0 any other co-morbid medical illnesses like Diabetes, Hypertension, and Cardio vascular.

Selection of Controls: The Control group was obtained from the normal volunteers attending General Health Check Up under Master Health Check up Scheme in Stanley Medical College Hospital. Inclusion Criteria was healthy male individuals of 18 – 25 years. Free from CVS and any other chronic illness, non smokers, non alcoholic, not on any drugs. The subjects were made to sit in the lab for 10min to get adapted to the new environment after emptying the urinary bladder. The subjects have been clearly instructed not to have Coffee, Tea or cool drinks 1½ hours before test. Basic measures such as BMI, blood pressure, were measured. The course and purpose of study were then explained. Written consent was obtained.

Experimental Protocol
The tests were carried out in the neurophysiology of the Department of Physiology, Stanley Medical College, and Chennai between 10.00 am and 1.00 pm. The laboratory environment was quiet, the temperature between 25 - 28 degrees Celsius and the lighting subdued. Subjects were asked to empty their bladder before the tests. The tests did not involve intravascular instrumentation or administration of any drugs at any stage.

Equipment
ECG was acquired using RMS Polyrite D hardware 2.2 (India), an instantaneous heart rate at RR intervals were continuously plotted using RMS 2.2 software on a Microsoft Window-based PC.. Respiratory movements were recorded using respiratory belt which analysis inspiration and expiration. Blood pressure was measured using the automated non-invasive BP monitor (Planet 50) L & T India. This measures BP by the oscillometric method. A standard adult – size cuff measuring 23 cm by 12 cm was used for all subjects.

Methodology of Recording Parameters
The subjects were made to sit in the lab for 10min to get accustom to the new environment after emptying the urinary bladder. After through clinical examination to rule out any acute or chronic illness also for any autonomic dysfunction, then their height in meters and weight in Kg measured. The students were then explained about the procedure.

Procedure
Electrodes were fixed in the following position after cleaning the site with sprit to record the ECG. Exploring electrode placed in Left shoulder. Exploring electrode placed in Right shoulder. Reference electrode placed in Right leg. Respiratory belt was tied around the chest at the level of nipple to record respiratory movement. The electrodes and the respiratory belt were connected to RMS polarity D equipment. HR & HRV response to Lying: ECG was recorded for 10mins to determine the HRV at supine rest with the eyes closed with normal quite respiratory movement (12-16/min). HR & HRV response to standing: After recording in the supine position the subjects were asked to stand without support on a wooden plank within 3 seconds and his BP and HR were recorded at the end of 5 sec, 2 mines and 5 mines after assumption of standing position. HRV during Deep Breathing: After recording the standing position the subject was asked to lie down comfortably in the supine position. He was then instructed to breathe slowly and deeply at a rate of 6 breaths per minute in such a way that he takes 5 seconds for each inspiration followed 5 seconds for expiration. The entire procedure was monitored on the screen.

Time Domain Measures
1. From NN interval: SDNN--Standard Deviation of the NN interval.
2. From the differences between NN intervals: RMSSD--Square root of the mean squared differences of successive NN interval. NN 50--Number of interval differences of successive NN interval greater than 50 ms.

Statistical Analysis
Comparison between the cases and controls were done using Student independent t-test. P<0.05 was considered as statistical significant.

RESULTS

![Fig.1: Shows the Changes in the HRV during Supine Rest for Five](image-url)
Figure 1 shows that SDNN value is significantly reduced in cases as compared to controls (p<0.001). Both the RMSSD and NN50 also reduced in cases when compared to controls.

Figure 2 shows that SDNN is markedly reduced for cases during standing. The RMSSD and NN50 values also reduced. P=0.001 significant

Figure 3 shows that SDNN value is significantly reduced in cases as compared to controls (p<0.001). Both the RMSSD and NN50 also reduced in cases when compared to controls. (P < 0.001). Both in cases and in controls the Time Domain measures are showing high values during one minute deep breathing. There is a reduction of these high values in cases compared to controls.

DISCUSSION

The autonomic nervous system and the balance between PNS and SNS play an important role in overall cardiovascular homeostasis. Chronic effects of Alcohol are due to Thiamine deficiency. Chronic deficiency leads to degeneration of nerve cells, reactive gliosis and atrophy of cerebellum and peripheral nerves including Autonomic nerves. Thiamine deficiency affects as many as one fourth of chronic Alcoholics. On metabolism Ethanol is biotransferred into toxic acetaldehyde in liver and finally carbon dioxide and water by acetyl co A. Vacuolization also reported in Sympathetic neurons of young rats following exposure to large doses of ethanol [3, 4]. The excess number of observed deaths compared to the expected number of deaths we have observed was amongst the alcoholics had evidence of vigil neuropathy who had evidence of autonomic neuropathy. The survival of diabetics with autonomic neuropathy was less than that of our alcoholics with vigil neuropathy. In chronic alcoholics with vigil neuropathy may have improvement of vagal function tests following continued abstinence. The lower mortality rate amongst alcoholics with vigil neuropathy may therefore long periods of abstinence or complete abstinence in several of the subjects [5, 6]. Montfort R et al have shown the dose-related toxic effect of alcohol causing Autonomic and peripheral neuropathies in patients. R H Johnson et al in 1988 have studied extensively on Parasympathetic dysfunction affecting the vague nerve in heavy drinkers who shows depressed reflex heart rate responses. Hence in alcoholics there is a strong evidence of parasympathetic neuropathy. Our study supports the previous study that indicates that alcoholic autonomic neuropathy primarily affects the parasympathetic pathways rather than the sympathetic pathways [7]. Weise F et al. in 1985 stated that direct causative relationship of vagal neuropathy in the mortality of individual patients is generally unclear. Our result show that its presence cause reduced HRV by the reduction in SDNN. In Time Domain method, the reduction in RMSSD, more specific marker of vigil activity confirms the presence of vigil neuropathy. The value of NN50, another marker of vigil activity is also reduced showing a gross reduction in vagal activity. This is reduction in HRV [8]. Irisawa et al in 1993 have studied extensively on Parasympathetic dysfunction affecting the vagus nerve in heavy drinkers who shows depressed reflex heart rate responses. Our study adds that there is significant reduction in NN50 count in Alcoholic Dependant individuals which is lacking in previous studies. Autonomic function evaluations with HRV analysis is not without problems. The indices obtained are very complex and any valued interpretation needs a clear knowledge of mechanisms of cardiovascular regulation. Single measurements are even more difficult to interpret since there is wide variation in most of the HRV indices. HRV analysis requires well advanced data acquisition systems and expensive software and persons trained to analyze and interpret the result [9, 10]. Autonomic function evaluations with HRV analysis is not without problems. The indices obtained are very complex and any valued interpretation needs a clear knowledge of mechanisms of cardiovascular regulation. Single measurements are even more difficult to interpret since there is wide variation in most of the HRV indices. HRV analysis requires well advanced data acquisition systems and expensive software and persons trained to analyze and interpret the result [4, 7].
CONCLUSION
The results suggest that the previously reported universe associations between alcohol intake and heart rate variability, which have suggested that alcohol consumption is associated with reduced vagal activity. The reason for a positive association between alcohol intake and heart rate is unclear but possibilities include an increase in sympathetic activity secondary to vasodilatation or increased calcium entering into cardiac myocytes causes the positive effect on heart rate.

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REFERENCES