A NOVEL TECHNIQUE USING MEASUREMENTS OF GALVANIC SKIN RESISTANCE AND HEART RATE VARIABILITY TO REVIEW THE AUTONOMIC CHANGES IN CHRONIC ALCOHOLICS

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**Background:** Alcohol has been widely consumed since prehistoric times by people around the world, as a component of the standard diet, for hygienic or medical reasons, for its relaxant and euphoric effects, for recreational purposes, for artistic inspiration and as aphrodisiacs. Alcohol is known to affect the sympathetic and parasympathetic activity, though the exact disturbances and degree of damage to the autonomic nervous system is not fully understood and possibility of reciprocal damage to these nerves have not been studied. **Methods:** Sympathetic nerve function was assessed by measurement of the galvanic skin resistance (GSR) Parasympathetic nerve function was assessed by R-R interval variation during deep breathing as measured by the heart rate variability (HRV) by the expiration/inspiration (E/I) ratio. 135 chronic alcoholics admitted to the Velankini rehabilitation ward were selected for this study. GSR was measured using GSR meter and HRV was assessed manually from calculation of the mean R-R interval and its standard deviation measured on short-term electrocardiograms. **Results:** Karl Pearson correlation was used to look for an association between GSR and HRV. A negative correlation of -0.919 was obtained. This signified that higher the sympathetic activity, lesser the vagal activity. **Conclusion:** Thus chronic consumption of alcohol has caused a proportional and reciprocal damage of the sympathetic and parasympathetic nerves in the human system.

**Keywords:** Chronic alcoholics, heart rate variability, galvanic skin response

INTRODUCTION

Autonomic neuro-cardial disturbances have been reported in alcoholics. The major reason for the interest in measuring HRV stems from its ability to predict survival after heart attack. Over half a dozen prospective studies have shown that reduced HRV predicts sudden death in patients with MI, independent of other prognostic indicators such as ejection fraction. Reduced HRV appears to be a marker of fatal ventricular arrhythmia. Moreover, a small number of studies have begun to suggest that reduced HRV may predict risk of survival even among individuals free of CHD.

To date, over 26 different types of arithmetic manipulations of R-R intervals have been used in the literature to represent HRV. The difference between the shortest R-R interval during inspiration and the longest during expiration (called the MAX-MIN, or peak-valley quantification of HRV) is one of them.

The autonomic responses of GSR are an excellent method for estimating the sympathetic activity and judging the disturbances taking place due to alcohol consumption. Increase in GSR indicates enhanced sympathetic activity.

Though individual measurements have been made of GSR and HRV, we chose to do estimate the two together in a novel method as they represent the sympathetic and parasympathetic activity respectively. Estimation of GSR and HRV together will help us to unravel the exact damage to the sympathetic and parasympathetic nerves which has occurred in the alcoholics.

MATERIAL AND METHODS

Chronic asymptomatic alcoholics admitted to the Velankini Rehabilitation ward, Father Muller’s Medical Hospital were selected as subjects. Ethical committee clearance was obtained from our Institute and Informed consent was received from all the subjects. 135 alcoholics were chosen for the study. All were males, 30-55 years in age, drinking Rum, Whisky, Gin, Brandy or Vodka for the past 10 years or more. Alcoholics with secondary somatic and mental diseases known to affect HRV and GSR measurements were excluded from the study; these included heart or lung disease, liver cirrhosis, hepatitis, pancreatitis, thyroid gland dysfunction, diabetes mellitus, malnutrition, multiple drug dependence (including chlormethiazole abuse), depression, anxiety disorders, and schizophrenia. The testing was done in the 1st week of admission.

In this study two parameters GSR for studying the sympathetic effects and HRV for studying the parasympathetic activity were tested. HRV was assessed manually from calculation of the mean R-R interval and its standard deviation measured on short-term electrocardiograms. The autonomic tests were performed in the Velankini Rehabilitation ward between 9.30am and 11 am to avoid circadian variation effects. The subjects were not allowed to take caffeine on the day of the
experiment. Patient was made to lie down in supine position for 20 minutes before the test began. A continuous ECG recording in Lead 2 was done for 10 minutes using BPL machine.

**Heart rate Response to deep breathing:**

The subjects were asked to relax in supine position. They were instructed to breathe at a rate of 6 breaths per minute. Throughout the period of deep breathing ECG was recorded with a marker to indicate the onset of inspiration and expiration. The maximum and minimum heart rate during breathing cycles was measured. The difference in maximum heart rate during inspiration and the minimum heart rate during expiration was calculated.

**GSR Measurement:**

The GSR was measured by the GSR Biofeedback Biotrainer, GBF-2000 (obtained from St. John’s medical College, Bangalore, India) with digital display in kilo-ohms, which measured the sympathetic activity. The recording was done in the sitting position. All GSR recordings were done immediately after the HRV measurements. For right-handed individuals, left hand values were taken. The left hand was placed comfortably resting from elbow to fingers on the table. The subject was seated in a revolving stool raised 20 inches from the ground. 2 points were marked exactly 1 inch away from each other in the hypothenar eminence using a scale, and jelly was applied liberally. The electrodes were placed on the marked points and secured with a transparent white cellophane tape. The subject was asked to relax completely, and was not allowed to watch the values. The instrument was switched on and 2 values were taken at an interval of 3 minutes, following, which the subject’s hand was wiped clean and the average was calculated.

**RESULTS**

The demographic profile was as follows: All alcoholics were males in the age group of 35-55 years (mean ± standard deviation: 45.9 ± 5.5 years); height: 167.08 ± 4.63 centimeters; weight: 49.18 ± 2.5 kilograms; Daily alcohol intake over 4 weeks before admission 1087 ± 329 g/day (range 1280-1560 g/day); duration of alcohol dependency 12.7 ± 8.1 years.

The mean galvanic skin resistance (GSR) was 10.43 ± 1.00 Kilo ohms; The mean heart rate variability (HRV) by the expiration/inspiration (E/I) ratio was 15.09 ± 1.81; The Karl Pearson correlation coefficient between GSR and HRV was – 0.919

**DISCUSSION**

The reason for reduced HRV could be as follows. Alcohol affects autonomic nervous system and heart has a predominant nerve supply by the Autonomic nervous system, sympathetic and parasympathetic. The difference between maximum and minimum heart rate in response to deep breathing showed a significant decrease in chronic alcoholics, suggesting that alcohol leads to autonomic imbalance especially a reduced parasympathetic activity and decreased vagal activity in alcoholics. This can cause alteration in heat rate variability.

<table>
<thead>
<tr>
<th>Chronic alcoholics</th>
<th>HRV</th>
<th>GSR</th>
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<tbody>
<tr>
<td>Mean</td>
<td>10.43</td>
<td>15.09</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>1.00</td>
<td>1.81</td>
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The reduced HRV which has been noted has occurred before any manifest heart conditions like cardiomyopathy or heart failure. Thus it can be a useful index to determine the reduced functioning of the heart even before there is manifested cardiac symptoms.

The possible reason for increased GSR is due to enhanced sympathetic activity. Previous reports state that alcohol increased sympathetic nerve activity by up to 239±22%. Acute increases in plasma alcohol increase heart rate and sympathetic nerve activity; blood pressure is not increased, probably because of vasodilator effects of alcohol.

Sympathetic and parasympathetic activity have been studied and correlated in other diseases like chorioretinopathy, diabetes mellitus, pre-prandial states and following heart transplantation. Nicolosi et al have reported no existence of a parallel involvement of peripheral somatic and autonomic cardiovascular nerve fibers in chronic alcoholism. But quantification of sympathetic and parasympathetic activity has not been undertaken in chronic alcoholics. The significant negative correlation thus demonstrates the depressed vagal activity and corresponding increased sympathetic activity in alcoholics.

The possible mechanism by which alcohol damages the nerves could be increased oxidative stress, damage to the mitochondria, interference with the activity of growth factors, effects on glial cells, impaired development and function of chemical messenger systems involved in neuronal communication, changes in the transport and uptake of the sugar glucose, effects on cell adhesion, and changes in the regulation of gene activity during development. 

**Significance:** Sympatho-vagal balance activity testing could help us to identify for finding ways to treat disturbances in sleep, sweating and other
impairments observed in day time in patients who consume alcohol

CONCLUSION

Reduction in parasympathetic activity and corresponding increase in sympathetic activity due to long standing alcohol consumption has been demonstrated by the reduced heart rate variability and enhanced galvanic skin resistance in chronic alcoholics.

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REFERENCES


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