

# A Study of Sensory Nerve Conduction and Reaction Time Between the Chronic alcoholics and Non-Alcoholics

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## ABSTRACT

**Introduction:** Consuming Alcohol impacts the social, psychological, health, and economical spheres of our existence. The total deaths due to alcoholism and disability occurs early in life. There is a relationship between harmful use of alcohol leads to mental and behavioural disorders. There is a direct effect of alcohol in the nervous system and impaired performance on a wide range of psychomotor tasks. The relationship between Peripheral neuropathy and chronic alcoholism has to be determined. The present study to evaluate the sensory nerve conduction velocities in both upper limbs and lower limbs and compare the Visual reaction time and auditory reaction time in chronic alcoholics and non- alcoholic controls.

**Material and Methods:** The present study was carried out in the Department of Physiology after getting approval of the institutional ethical committee. Nerve conduction studies and reaction time were performed using RMS EEG-32 SUPER SPEC (P) LTD Chandigarh, Reaction time by YANTRA SHILPA Pune.

**Results:** There was increase in onset of latency time and decrease in amplitude and sensory nerve conduction velocity of sensory nerves in chronic alcoholics as compared to non-alcoholic controls. There was statistically very highly significant delay in visual reaction time and prolongation of auditory reaction time in chronic alcoholics as compared to non-alcoholic controls.

**Conclusion:** Nerve conduction studies and reaction time studies are useful in finding the effect of alcohol on auditory pathway, visual pathway and peripheral nervous system.

**Keywords:** Alcoholism, electrophysiological tests, Neuropathy, nerve conduction, reaction time.

## INTRODUCTION

Alcoholism is characterized by significant physiological, psychological and social dysfunctions associated with persistent and excessive use of alcohol. The World Health Organization estimates that there are 140 million people with alcoholism worldwide.<sup>1</sup> The harmful use of alcohol results in the death of 3.3 million people annually.<sup>2</sup> Long-term use of alcohol abuse can damage various organs causing cirrhosis of liver, pancreatitis, peptic ulcer, cardiovascular disease, central nervous system and peripheral nervous system can also occur. Wernicke's syndrome and Korsakoff's psychosis can occur together or separately due to the low thiamine levels in many alcohol-dependent people.<sup>3</sup> Peripheral neuropathy due to high alcohol levels in the body cause the nerve damage. Although neuropathy is frequent among chronic alcoholics, there are no uniform data about its prevalence, the association of peripheral neuropathy and chronic alcoholism in man has long been known, the exact relationship has not been completely determined. The study of the disorders of peripheral nerve function has been greatly facilitated by the development and application of refined electrophysiological techniques.<sup>4</sup>

The direct effect of alcohol on the central nervous system is essentially sedative and disorganizing as demonstrated by impaired performance on a wide range of psychomotor tasks such as simple and choice reaction time as rapidly as possible after the stimulus. Deterioration in cognitive performance is also evident in tasks involving memory for words and numbers, word fluency, mathematical reasoning and speed accuracy for numbers.

The present study was carried out to evaluate the long term effects of alcohol in the peripheral nervous system and reaction time by conducting neurophysiological tests.

Aim and objectives of the study were to study the sensory nerve conduction velocities in both upper limbs and lower limbs in chronic alcoholics and non- alcoholic controls and to study the Visual and Auditory reaction time of chronic alcoholics and non- alcoholic controls

## MATERIAL AND METHODS

The present one year case control study was carried out in Department of Physiology, Tirunelveli medical college after getting approval of the institutional ethical committee.

**Method of Collection of Data:** Complete central nervous system examination was performed. Nerve conduction studies were performed using RMS EEG-32 super spec (Recorders and Medicare System (P) Ltd Chandigarh, Reaction time by Yantra Shilpa Pune).

**Methodology:** Subjects of non-alcoholic controls and alcoholic without abstinence were given appointment to report in Department of Physiology in the morning hours at 10.00 am for measurement of anthropometric parameters and Neurophysiological tests.

All participants were given detailed information about research work. Informed and Written consent was obtained from every subject at the beginning of the study work.

**Selections of subjects:** 120 male subjects in the age group of 25-50 years were selected randomly from general population.

The subjects were divided into two groups:

**Study group:** It includes 60 chronic alcoholic men consuming alcohol more than 21 units/week (1 unit=10grams), 1 unit =30ml of alcohol for greater than 5 years of duration without abstinence

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**How to cite this article:** Pillai Karthik Piramanayagam, Thenmozhi. R. A study of sensory nerve conduction and reaction time between the chronic alcoholics and non-alcoholics. International Journal of Contemporary Medical Research 2016;3(7):2090-2093.

and not having clinical overt neuropathy.<sup>13</sup>

**Control group:** It includes 60 healthy men of same age, socio economic status, BMI and not consuming alcohol.

Detailed History of subject, type, quantity, frequency and duration of alcohol intake was recorded, Alcohol dependence screening was done by using alcohol dependence data Questionnaire (SADD) was used to detect alcohol dependence (Annexure B) Alcohol consumption in units were quantified.

General and systemic examination was carried out. ENT and Ophthalmic examinations was done. Onset latency (OL), Sensory nerve action potential (SNAP), Sensory nerve conduction velocity (SNCV), visual and auditory reaction time were also done. Sensory nerves- Ulnar and Sural were also examined.

**Exclusion criteria:** HIV, tuberculosis, diabetes, thyroid disorder, hypertension, smokers, visual and acoustic handicaps, head, ear, eye injury, stroke patients were excluded.

Patients on long term medication known to cause neuropathy, such as anti- epileptics, anti-psychotics, antidepressant drugs, Radiotherapy and chemotherapy were also excluded.

**Parameters selected and data collection:**

- 1) Height in cms.
- 2) Weight in Kgs.
- 3) Body mass index – wt in Kg/ ht in m<sup>2</sup>
- 4) Sensory Nerve conduction velocity (NCV): Distal latencies in (ms), Amplitude in (uv) and Nerve conduction velocity in (m/s) of sensory nerves both limbs Ulnar and Sural nerves were measured.
- 5) Reaction time: Both visual and auditory reaction time were

studied

- Auditory reaction time using low frequency and high frequency sounds.
- Visual reaction time using red and green light.
- Both measured by Response analyser: Yantra shilpa system, Pune.

**STATISTICAL ANALYSIS**

In our study simple t test and paired t test were done for comparison. The mean, standard deviation, standard error at 95% confidential interval were analysed with the help of SPSS version 21.

**RESULTS**

The baseline characteristics like age, height, weight and BMI was statistically not significant in chronic alcoholics as compared to non- alcoholic controls.

Table-1 shows, there was increase in onset latency and statistically very highly significant decrease in amplitude and sensory nerve conduction velocity of sensory ulnar nerves in chronic alcoholics as compared to controls.

Table-2 shows, there was increase in onset latency, very highly statistically significant decrease in amplitude and Sensory nerve conduction velocity of sural sensory nerves in chronic alcoholics as compared to controls.

Table-3 shows, there was statistically very highly significant prolongation of visual reaction time in chronic alcoholics as compared to controls.

Table-4 shows, there was statistically very highly significant prolongation of auditory reaction time of low frequency sound and high frequency sound in chronic alcoholics as compared to

Parameters	Ulnar sensory nerve	Non alcoholics (n=60) Mean±SD	Chronic alcoholics (n=60) Mean±SD	P value
OL(ms)	Rt	3.27±0.110	3.303±0.047	P<0.001
	Lt	3.183±0.118	3.256±0.042	P<0.001
SNAP(uv)	Rt	6.092±0.332	5.56±0.384	P<0.001
	Lt	6.086±0.332	5.63±0.374	P<0.001
SNCV(m/s)	Rt	56.568±1.301	54.580±0.746	P<0.001
	Lt	56.543±1.312	54.60±0.721	P<0.001

p<0.05 –significant, p<0.001 – highly significant

**Table-1:** Comparison of onset Latency, Sensory nerve action potential, Nerve conduction velocity of Ulnar sensory nerve in non- alcoholic controls and chronic alcoholics

Parameters	Sural Sensory nerve	Non alcoholics (n=60) Mean±SD	Chronic alcoholics (n=60) Mean±SD	P value
OL (ms)	Rt	4.16±0.112	5.21±0.114	P<0.001
	Lt	4.25±0.144	5.241±0.176	P<0.001
SNAP (uv)	Rt	11.07±0.520	7.98±0.237	P<0.001
	Lt	10.9±0.325	7.968±0.317	P<0.001
SNCV (m/s)	Rt	50.92±0.699	45.73±0.976	P<0.001
	Lt	51.23±0.805	45.721±0.780	P<0.001

p<0.05 –significant, p<0.001 – highly significant

**Table-2:** Comparison of onset Latency, sensory nerve action potential, Nerve conduction velocity of sural sensory nerve in non- alcoholic controls and chronic alcoholics

Parameters	Non alcoholics (n=60)	Chronic alcoholics (n=60)	P value
	Mean ±SD	Mean ±SD	
Red light(sec)	0.191±0.001	0.212±0.002	P<0.001
Green light( sec)	0.193±0.001	0.204±0.002	P<0.001

p<0.05 –significant, p<0.001 – highly significant

**Table-3:** Comparison of Visual reaction time in non-alcoholic controls and chronic alcoholics

Parameters	Non alcoholics (n=60) Mean ±SD	Chronic alcoholics (n=60) Mean ±SD	P value
Low frequency sound (sec)	0.163±0.001	0.1671±0.0008	P<0.001
High frequency sound(sec)	0.161±0.001	0.166±0.0001	P<0.001
p<0.05 –significant, p<0.001 – highly significant			
<b>Table-4:</b> Comparison of results of Auditory reaction time in non-alcoholic controls and chronic alcoholics			

controls.

The data obtained was subjected to statistical analysis and paired student “t” test was performed to compare the mean value of each parameter in alcoholics and non- alcoholics.

There was no statistical difference (p>0.05) in the mean age, height, weight and BMI in non -alcoholic controls and alcoholics group.

Sensory nerves of both ulnar and sural were statistically very highly significant increase (p<0.001) in onset latency of chronic alcoholics as compared to controls, there was also statistically very highly significant decrease (p<0.001) in sensory nerve action potential in chronic alcoholics as compared to non alcoholic controls.

There was statistically very highly significant (p<0.001) delay visual reaction time for red light and green light in chronic alcoholics as compared to non alcoholic controls. There was statistically very highly significant (p<0.001) prolongation of auditory reaction time of low frequency sound and high frequency sound in chronic alcoholics as compared to non alcoholic controls.

## DISCUSSION

Long -time effect of alcohol intake can cause decrease in neuronal conduction. It has been shown that electrophysiological deficits are greater in alcoholics with long term exposure. The histo-pathological changes in alcoholic neuropathy, segment demyelination was the most probable lesion accounting for reduction in sensory conduction of impulses. Axonal degeneration was the main pathological cause for decrease in motor nerve conduction velocity. Evidence of segmental demyelination secondary to axonal degeneration was also observed.

The chronic partial denervation due to wallerian like degeneration of fibres, ratio of myelin component to myelinated axon was increased in pathological nerve despite the reduction in myelin, indicating greater involvement of axonal component. However thiamine deficiency has been considered as main pathological factor for demyelination. The direct toxic actions of alcohol have also been claimed to play role in inducing nerve lesion. Appropriate nutritional therapy decreases the morbidity and mortality in alcoholics. Our study correlates with Maudsley and Mayer et al. revealed decrease in MNCV which was more pronounced in chronic alcoholics, with neuropathy as compared to those without neuropathy. A small decrease in sensory nerve conduction velocity as compared to controls.<sup>5</sup>

Richard and Mayer M.D et al. studied peripheral nerve conduction velocities in acute and chronic alcoholics and found decrease in Motor nerve conduction velocity and sensory nerve conduction velocity and latency was prolonged.<sup>6</sup> Walsh and McLeod et al. also studied peripheral Nerve conduction studies in alcoholics with neuropathy and found a slight reduction in maximal motor conduction velocity as compared to controls. The amplitude of sensory nerve action potentials was reduced

with only a slight increase in latency.<sup>14</sup>

T.G.H.C Fernandis et al. assessed peripheral nerve conduction studies in healthy subjects and two groups of chronic heavy drinkers consuming distilled alcohol and legal spirit and found both upper limb and lower limb motor and sensory nerve conduction velocity were significantly decreased in both group of alcoholics.<sup>7</sup>

Alcohol abuse leads to metabolic alterations in the nerve cells and degeneration of the axial flux. Each axon begins to generate from the most distal sections of the cell. In this way the longest axons are the first to be involved. As the disease progresses, the axonal flux becomes less and less efficient, and the degeneration begins to extend to portions of the axons closer to the cell body and is accompanied by destruction of the myeline sheaths. This is known as the ‘dying back’ phenomenon or retrograde degeneration. The pathogenesis of alcoholic polyneuropathy is still under debate. It could be due to nutritional deficiency, and especially deficiency of Thiamine, there is both clinical experimental evidence of a direct toxic effect of alcohol.

The mean values of reaction time for red light, green light was statistically very highly significantly (P<0.001) increased in chronic alcoholics as compared to non -alcoholic controls.

The mean values of low frequency sound and high frequency sound was statistically very highly significantly (P<0.001) in chronic alcoholics as compared to non- alcoholic controls.

We observed that both visual and auditory reaction time was longer in the alcoholic subjects compared to normal ones. It was consistent with the observations of Fein G and Chang M.<sup>6</sup> This could be due to involvement of both the sensory and motor pathways, which are sensed by the respective sensory receptors and carried through the optic nerve and auditory nerve. The processing of this information takes place in the brain and the motor pathways include the cranial nerves and the nerves which supply the muscles of the hand. If the person has good reflexes, these information processing is shorter and reaction time is shorter. In alcoholics the nerve damages occur which include both the autonomic and peripheral nerves. The neuropathies can thus reduce the speed of the impulse conduction and lead to the increase in the reaction time. The alcoholics cannot mobilize sufficient processing resources in terms of cognition and the poor motivation in alcoholics can cause insufficient effect to cognitive tasks.

Slow response to visual stimuli in chronic alcoholics has been reported by Talland GA and Vazhnova TN and Pogrebinskii SA.<sup>8,11</sup>

The delayed reaction time in chronic alcoholics as compared to control might be to following changes in neural system.

Alcoholics have retarded psychomotor speed, mild to generalised intellectual impairment and show premature aging process which is known to delay conduction.<sup>9</sup>

Chronic alcoholics also show impairment of perceptual – motor coordination, which is related to involvement of cerebellar hemispheres and diminished capacity for information

processing, which again may contribute to delayed response by alcoholics.<sup>8</sup> Prolonged use of alcohol leads to pattern of damage in central nervous system characterised by brain shrinkage including reduction in the white matter and cerebral grey matter coupled with extensive cerebellar degeneration. This causes delay in reaction time as cerebellar cortex is important for the timing and programming of accurate motor events.<sup>12,15</sup>

On prolonged exposure, alcohol affects number of brain regions. It leads to significant reduction in the volume of a number of sub-cortical grey matter and cortical Regions; involving basal ganglia, thalamus, mamillary bodies, hippocampus and superior frontal and parietal regions; important for higher cognitive function. This leads to cognitive impairment in chronic alcoholics.<sup>16</sup>

Impairment in recent and remote memory in chronic alcoholics leads to cognitive dysfunction resulting in sensory motor incoordination with delayed response in reaction time task.<sup>14</sup>

Chronic intake of high doses of ethanol causes peripheral neuropathy, which is related to deficiency of B vitamins, particularly thiamine. Individuals may complain of calf muscles tenderness, burning of soles of feet and paraesthesia in toes and fingers. Symptoms are more pronounced distally than proximally, which might be responsible for delayed response in alcoholics.<sup>17</sup>

Vazhnova TN et al 1984 has also been noted that the process of decision making is delayed in alcoholics indicating a lesion of anterior zones of brain, which leads to slowing of motor command. Also, the process of simultaneous activity of two hemispheres during processing of incoming visual information is disturbed (WHO offset 1977).<sup>17</sup> Alcoholics also suffer from dimness of vision or partial loss of sight without observable lesion in eye structures or optic nerves, called as alcoholic amblyopia which may be associated with malnutrition resulting in delayed visual time in chronic alcoholics.<sup>11</sup>

Visual – spatial organization is also impaired in chronic alcoholics which might be responsible for delayed response to visual stimuli (Chandler BC et al 1977).<sup>10</sup> This correlates with our study.

**Limitations of the Study** - The Quantitative analysis are required in further studies, which reveal the relationship of dose, duration and abstinence of alcohol on Brainstem evoked auditory response. Further longitudinal studies are recommended to see whether the neurological dysfunction in alcoholics is reversible with abstinence and with appropriate nutritional therapy.

## CONCLUSION

These electrophysiological tests can be used to detect subclinical central and peripheral neuropathy at an early stage of disease. So that suitable preventive and treatment methodologies can be implemented.

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**Source of Support:** Nil; **Conflict of Interest:** None

**Submitted:** 26-05-2016; **Published online:** 30-06-2016