

# Chronic Alcoholism and its Effect on Nerve Conduction Velocity

Sangita.R.Phatale<sup>1\*</sup>, Anjali Shete<sup>2</sup>

<sup>1</sup>Professor and HOD. Dept. of Physiology, MGM'S Medical College Aurangabad, Maharashtra, INDIA.

<sup>2</sup>Associate Professor. Dept of Physiology, Govt. Medical College, Aurangabad, Maharashtra, INDIA.

\*Corresponding Address:

[drsangita\\_phatale@rediffmail.com](mailto:drsangita_phatale@rediffmail.com)

## Research Article

**Abstract: Background and objective:** Alcoholism is common and serious problem. Alcohol most commonly consumed in the form of ethanol. The effects of alcohol are mainly caused by altering the function of membrane proteins in many different cell types especially cells of GIT, urinary tract, cardiovascular and nervous system. Alcohol also causes alcoholic peripheral neuropathy.

**Material and Methods:** The study was conducted in the department of physiology MR Medical College Gulbarga. The study was conducted in chronic alcoholic's subject and there age matched control and in three groups of 30 subjects in each groups. Group I – Normal healthy control, Group II – chronic alcoholic without neuropathy, Group III – chronic alcoholic with neuropathy. Nerve conduction study was carried out by using RMS- EMG,EP machine manufactured by Recorders and Medicare System (P) Ltd. The data was statistically analyses by using student unpaired' test.

**Result:** Motor and sensory nerve conduction velocity was compared with chronic alcoholic and healthy aged matched controls. The both sensory and motor nerve conduction was significantly decreased in chronic alcoholic without neuropathy and with neuropathy. **Conclusion:** From our study we conclude that alcohol addiction has adverse effect on the NCV Thus helps in early diagnosis as well as prognosis of alcoholic neuropathy

**Keywords:** Chronic alcoholism, NCV, neuropathy.

## 1. Introduction

Alcoholism is common and serious problem. Alcohol most commonly consumed in the form of ethanol. It is consumed in different forms containing different concentration eg – Beer (4% - 12%), wine (10 % - 15 %), Rum (37.5 % - 75.5 %), Whisky (40 % - 55%).(1) Ethanol has wide spread effects on the organ system of the body. The effects of alcohol are mainly caused by altering the function of membrane proteins in many different cell types especially cells of GIT, urinary tract, cardiovascular and nervous system.(2) Chronic alcoholism is a pathological condition resulting from the habitual use of alcohol in excessive amounts. Scientist suggested that a minimum of 100ml of ethyl alcohol per day for 5 years will precipitate the adverse effects.(3) National family health survey (NFHS- 3) showed that about 32% of total population is chronic user of alcohol. The effect of alcohol on brain can occur by both direct and indirect means.(4) Alcohol causes sleep disturbances,

numbness and legs, wernicke's syndrome and korsakoffs syndrome which can occur due to the low thiamine (Vit-B) levels. Korsakoffs syndrome affects memory and prevents new learning from taking place. Alcohol also causes alcoholic peripheral neuropathy.(5,6) Slowing of motor nerve conduction and reduction in sensory nerve action potential amplitudes in alcoholic neuropathy are due to consequences of axon loss in chronic alcoholism.(7) Alcoholic neuropathy also renders patient susceptible to compression of peripheral nerves at common sites of compartment including median nerve at carpal tunnel, ulnar nerve at elbow and peroneal nerve at fibular head(8). Nerve conduction study is a simple and noninvasive test and measures how quickly electrical impulses move along a nerve. Motor nerve conduction (MNCV) study is performed by electrical stimulation of a peripheral nerve and recording from a muscle supplied by this nerve. Sensory nerve conduction (SNCV) can be measured orthoromically or antidromically.

## 2. Material and Methods

The study was conducted in the department of physiology MR Medical College Gulbarga. The study was conducted in chronic alcoholic's subject and there age matched control and in three groups of 30 subjects in each groups. Group I – Normal healthy control, Group II – chronic alcoholic without neuropathy, Group III – chronic alcoholic with neuropathy. The age group of subjects was between 40 – 60 years. A detailed case history was taken for both control and alcoholic subject. Inclusion criteria for chronic alcoholic was alcohol consumption of more than 100ml daily for more than 10 years and without any other diseases causing neuropathy as well as any other neurological problem.

### Methodology

Nerve conduction study was carried out by using RMS-EMG,EP machine manufactured by Recorders and Medicare System (P) Ltd. All tests were done in a quiet room at a temperature of 26<sup>0</sup> to 30<sup>0</sup>C.

Motor nerve conduction velocities in ulnar and median nerves in upper limb and in deep peroneal and posterior tibial nerves in lower limb were studied. The nerves were stimulated per-cutaneously along their course where they are relatively superficial. The skin resistance was reduced

$$\text{Motor NCV(meters/sec.)} = \frac{\text{Distance between to 2 point of stimuli (mm)}}{\text{Difference in latencies (msec)}}$$

Sensory nerve conduction velocities of ulnar and median nerves were studied, recording is made using ring electrodes.

$$\text{Sensory NCV(meters/sec.)} = \frac{\text{Distance between the stimulating and recording site (mm)}}{\text{Difference in latencies (msec)}}$$

The statistical analysis was done using student's unpaired T- test.

### 3. Observation and Results

**Table1:** Shows mean value and Standard deviation(S.D) of motor nerve conduction velocity(m/s)with right hand.

Nerve	Group	Mean $\pm$ S.D	Group Compared	'P' value and significance
Right Median	I	54.8 $\pm$ 4.4	IandII	<0.001 S
	II	48.6 $\pm$ 3.8	IandIII	<0.001 S
	III	45.1 $\pm$ 4.5	IIandIII	<0.001 S
Right Ulnar	I	53.9 $\pm$ 4.9	IandII	<0.001 S
	II	47.8 $\pm$ 4.5	IandIII	<0.001 S
	III	45.4 $\pm$ 3.5	IIandIII	<0.05 S
Right Tibial	I	51.8 $\pm$ 2.9	IandII	<0.001 S
	II	45.3 $\pm$ 5.4	IandIII	<0.001 S
	III	39.8 $\pm$ 4.6	IIandIII	<0.001 S
Right Peroneal	I	52.8 $\pm$ 3.2	IandII	<0.001 S
	II	45.4 $\pm$ 4.4	IandIII	<0.001 S
	III	40.5 $\pm$ 4.1	IIandIII	<0.001 S

**Table 2:** Shows mean value and std.devition(S.D) of sensory nerve conduction velocity(m/s)with right hand.

Nerve	Group	Mean $\pm$ S.D	Group Compared	'P' value and significance
Right Median	I	54.9 $\pm$ 1.4	IandII	<0.001 S
	II	46.6 $\pm$ 3.8	IandIII	<0.001 S
	III	42.4 $\pm$ 3.5	IIandIII	<0.001 S
Right Ulnar	I	53.8 $\pm$ 4	IandII	<0.001 S
	II	45.4 $\pm$ 4.5	IandIII	<0.001 S
	III	42.6 $\pm$ 4.4	IIandIII	<0.05 S

### 4. Discussion

A nerve conduction study is a test commonly used to evaluate the function of the motor and sensory nerves of the human body. It measures how quickly electrical impulses move along a nerve. We observed that motor nerve conduction velocity (MNCV) in median, ulnar, tibial and peroneal nerves was decreased significantly ( $p < 0.001$ ) in groups of alcoholic patients with and without neuropathy; as compared to their age matched controls. It is also seen that MNCV was significantly decreased in chronic alcoholics with neuropathy; when compared to chronic alcoholic without neuropathy. The sensory nerve conduction velocity in median and ulnar nerves was decreased in group of alcoholic patient with and without neuropathy and statistically significant ( $p < 0.001$ ) when compared with age matched controls. Mawdsley and Mayer 1965(7), C.Coers and Hildebrand 1965(9), Juul-Jensen and Mayer 1966 (10), Walsh and McLeod 1970(11), M.Lefebvre D'Amour, B.T. Shahani

by rubbing with spirit swab; the active electrode was placed over muscle belly and reference electrode over tendon. The intensity of stimulus was increased gradually until the muscle action potential is viewed. Compound muscle action potential is recorded.

1979(12), J.P Ballantyne and S. Hansen 1980(13), Matti Hillbom and Arne Wennberg 1984 (14), H.J.De.Silva 2007(15) observed decrease in motor nerve conduction velocity of median, ulnar, peroneal, and tibial nerves and sensory nerve conduction in median and ulnar nerves in patients who did not have any clinical evidence of peripheral neuropathy. These matched with our findings. However our study did not matched with observations of Eileen Blackstaock, Rushworth and Dennis 1972(16), E.B.Casey and Pamela 1970(17), who revealed that maximum motor nerve conduction velocity was not altered significantly and was within normal range. The decrease in nerve conduction velocity in alcoholics can be explained on the following basis:

1. Myelin is an electrically insulating material that forms a layer, around the axon of a neuron. The function of myelin sheath is to increase the speed of conduction. It also prevents the leaking of electrical current.

2. Alcohol causes increase in lipid permeability of membrane of astrocytes and oligodendrocytes. Ethanol has been known to modulate ion channels and receptors, such as GABA, NMDA, glycine receptors, K<sup>+</sup> channels and intracellular signaling systems in neurons. Recent evidence shows that alcohol also affects glial cells and alters neuroglial interactions. It affects K<sup>+</sup> channels on astrocytes and astroglial gap junction permeability. This causes increased leakage of action potential current down the axon.(4)
3. In Chronic alcoholism there is always some degree of nutritional deficiency. Alcohol increases the metabolic demand for thiamin because of its role in the metabolism of glucose. Thiamine is important in three reactions in the metabolism of glucose; the decarboxylation of pyruvic acid, d- ketoglutaric acid and transketolase. Lack of thiamine in the cells prevents neurons from maintaining necessary ATP levels as a result of impaired glycolysis. Many alcoholics suffer from malnutrition as well as impaired intestinal absorption of thiamine, and other important cofactors for oxidative metabolism of glucose. (18)
4. An energy deficiency in Schwann cells causes disappearance of myelin on peripheral nerves, resulting in damage of axons and loss of function. All this leads to increase in leakage of current and slows signal transmission.
5. Acetaldehyde formed during the ethanol metabolism as toxic effect on peripheral nerves if it is not metabolized quickly.(19)
6. Damage to the nervous system takes place before symptoms appear in individuals with chronic alcoholism, beginning with segmental thinning and loss of myelin on peripheral ends of the longest nerves.(20)

## 5. Summary and Conclusion

- The present study was conducted to study nerve conduction velocities in chronic alcoholics and healthy controls.
- There was significant decrease in motor and sensory nerve conduction velocities in chronic alcoholics with and without neuropathy as compared to healthy controls.
- NCV helps to detect alcoholic neuropathy, before signs and symptoms appear.

## References

1. Morse R.M, Flavin D.K. "The definition of alcoholism. The Joint Committee of the National Council on Alcoholism and Drug Dependence and the American Society of Addiction Medicine to study the definition and criteria for the Diagnosis of Alcoholism" JAMA: the journal of American Medical Association ( August 1992) 268 (8): 1012 – 4.
2. Ramon Estruch, Jose M. Nicolas, Alcohol, alcohol abuse and alcoholism; Vol.28.No5.pp 543- 550 (1993)
3. Fiona Barter and Andrew R. Tanner: Neuropathy in an alcoholic population; Postgraduate Medical Journal (1987)63, 1033-1036.
4. Dr. Bill Boggan: Effects of Ethyl Alcohol on organ Function, Alcohol Chemistry and you; Alcohol and Alcoholism, vol.36 no 5, 2001.
5. Shy M.E. Peripheral neuropathies. In: Goldman L, Ausiello D, eds. Cecil Medicine.23<sup>rd</sup> ed. Philadelphia, Pa: Saunders Elsevier; 2007: chapter 446.
6. Christopher C.H.Book; B vitamin deficiency and neuropsychiatric syndromes in alcohol misuse; Alcoholand Alcoholism Vol 33,No4,pp.317-336,1998.
7. Mawdsley C and Mayer R.F: Nerve Conduction in Alcoholic Polyneuropathy, Brain (1965) 88, vol. 2,335-356
8. Taraksd S Ramchndran: Alcohol related neuropathy, Medscae drugs disease and procedure ,June 2009.
9. Coers C and Hildebrand J (1965) : Latent neuropathy in diabetes and alcoholism. Electromyographic and histological study. Neurology (Minneap) 15, 19 – 38.
10. Juul – Jensen, Mayer R.F(1966) : Threshod stimulation for nerve conduction studies in man. Archives of Neurology,15,410 -419.
11. Walsh .J.C and McLeod J.G.(1970): Alcoholic neuropathy An electrophysiological and histological study. Journal of Neurological Science,10,457 -469.
12. M.Lefebvre D'Amour, B.T. Shahani: The importance of studying sural nerve conduction and late responses in the evaluation of alcoholic subjects; Neurology 1979; 29,1600-1604
13. J.P Ballantyne, S Hansen: Quantitative electrophysiological study of alcoholic neuropathy.Journal of Neurology, Neurosurgery and Psychiatry,1980 May,43 (5),427-432
14. Matti Hillbom and Arne Wennberg; Prognosis of alcoholic polyneuropathy; Journal of Neurology, Neurosurgery and Psychiatry, 1984:47,699-703.
15. T.G.H.C.Ferdinandis, and H. J .De Silva (2007) illicit alcohol consumption and neuropathy – A preliminary study in Sri Lanka; Alcohol and Alcoholism Vol.43, No.2,pp.171 -173
16. Eileen Blackstock, Geoffrey Rushworth and Dennis Gath; Electrophysiological studies in Alcoholism; Journal of Neurology, Neurosurgery and Psychiatry, 1972,35,326-334.
17. E.B.Casey and Pamel; Electrophysiological evidence of distal lesion in alcoholic neuropathy, Journal of Neurology, Neurosurgery and Psychiatry, 1972, 35,624 - 630
18. Hell D, Six P, Vitamin B1 deficiency in chronic alcoholics and its clinical correlation salked R Schweiz Med Wochenschr.1976 Oct 23; 106(43)
19. Thompson, Warren and R Gregory Lande, 'Alcoholism' Mediscape drugs, disease and procedures, Feb18 2011
20. Robert D.Hienze, Danielle A. Bowers Acute ethanol effects on sensory/motor function in baboons with a history of chronic ethnl ingestion; Drug and Alcohol Dependence, Vol.30, issue 1, April 1992, pp85 -100.