



Nerve Conduction Studies in Asymptomatic Alcoholics

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ABSTRACT:

Introduction: *The association of Peripheral Neuropathy and Chronic Alcoholism in man has long been known. Neuropathy usually occurs after many years of drinking. The present study is undertaken with the aim of studying Electrophysiological patterns in Chronic Alcoholics who have not had any of the symptoms*

Materials and Methods: *The study group consisted of attendants of patients attending Neurology OP at Andhra Medical College who were asymptomatic and were having a habit of regular alcohol intake. The instrument used was VIKING SELECT EMG machine provided by VIASYS HEALTH CARE Ltd. The Nerves selected for the Study are Ulnar nerve, Radial nerve, Sural nerve, Common peroneal nerve, Tibial nerve.*

Results: *The findings in our study were decreased amplitude of compound muscle action potential recorded in common peroneal nerves, decreased amplitude of sensory nerve action potential recorded in radial nerves (49%).*

Conclusion: *In our study the radial sensory recordings and peroneal motor recordings are more common abnormal findings. Inference is these nerves are more vulnerable for compression during inebriated state due to their superficial position.*

Key words: *Nerve conduction, Asymptomatic Alcoholics*

Introduction

Though peripheral neuropathy is associated with chronic alcoholism, the exact relationship is not established.

Earlier writers stated that neuropathy resulted from alcohol itself^{1,2}. Shattuck³ proposed a nutritional aetiology. Strauss⁴ demonstrated that patients with alcoholic neuropathy improved with well balanced diet and vitamins in spite of daily alcohol intake.

It is now generally thought that the neuropathy of alcoholics is a deficiency disease, mainly due to lack of thiamine^{5,6,7}.

Chronic alcoholic patients who develop neuropathy have histories and clinical evidence of poor dietary habits. They often suffer deficiencies of substances such as folic acid⁸.

Neuropathy usually occurs after many years of drinking and initially the signs and symptoms appear in the distal portions of the lower limbs. Involvement is usually bilateral and symmetrical. Both motor and sensory fibres are affected. In later course, the upper extremities are involved as well.

It is now possible to accurately measure peripheral nerve conduction velocities in the fastest motor⁹ and sensory¹⁰ fibres in man because of advanced technology.

Alcoholic Beverages

An alcoholic beverage is a drink that contains ethanol. They are divided into beers(4-6% alcohol by volume), wines(9-6% alcohol) and spirits(20%).

Brandy, whisky and rum has alcohol content of 40-50% by volume.

Alcohol is a psychoactive drug that has a depressant effect¹¹

Alcoholic Beverages can be addictive and the state of addiction is called alcoholism¹¹.

Lethal dose of ethanol is 400mg/dl.

Blood alcohol concentration of 80-100mg/dl is considered as lethal drunkenness¹²⁻¹⁵.

Alcohol supplies the calories to the body. It is devoid of minerals, proteins and vitamins. It is

absorbed from the mucous membrane of mouth, esophagus, stomach and large bowels and from the proximal site of small intestine which is the major site of absorption¹²⁻¹⁵. It interferes with the absorption of vitamins in the small intestine and decreases their storage in liver producing deficiency of folic acid, pyridoxine, thiamine and nicotinic acid.

2% to 10% of ethanol is directly excreted by lungs skin urine but greater part is metabolized to acetaldehyde in liver by the enzyme alcohol dehydrogenase in cytosol and mitochondria. The acetaldehyde formed is rapidly destroyed by aldehyde dehydrogenase in cytosol and mitochondria¹²⁻¹⁵.

To the pathogenesis of alcoholic neuropathy includes: 1. direct toxic effects of alcohol on the cellular population of the central nervous system and other tissues, especially of parenchymatous organs (in particular of the liver), 2. indirect metabolic and exotoxic changes mediated by malabsorption, maldigestion and secondary caloric and energy deprivation, 3. effects of genetic factors².

Alcohol enhances GABA^A receptors (gamma aminobutyric acid A) and inhibits NMDA receptors (N- methyl –D-aspartate)¹²⁻¹⁵.

Alcohol produces after repeated exposure: ¹²⁻¹⁵ metabolic tolerance (30% increase in hepatic clearance), Cellular or pharmacodynamic tolerance, and behavioral tolerance. Cellular changes developed may not resolve for weeks after cessation of alcohol drinking as neurons require alcohol to function properly and the individual can be said physically dependent.

Terminology of nerve conduction study:

Principle of motor nerve conduction:

The motor/mixed nerve is stimulated at least at two points.

Pulse is adjusted to record a compound muscle action potential.

Surface recording electrodes are placed in the belly tendon montage (active electrode is kept

near to motor point; reference point is kept near to tendon).

Surface stimulation of healthy nerve requires square wave pulse of 0.1ms duration with an intensity of 5-40mA.

Filter settings for motor nerve conduction is 5-10 Hz and a sweep speed of 2-5m/sec

The measurement for motor nerve conduction include

1. Onset latency
2. Duration
3. Amplitude of CMAP

Onset latency: time in milliseconds from the stimulus artifact to the first negative deflection of CMAP. It is a measure of conduction of the fastest conducting motor fibres.

Amplitude correlates with the number of nerve fibres.

Duration correlates with the density of small fibre.

Conduction velocity =

Distance between two points of stimulation

Latency difference in milliseconds

$$= \frac{D}{PL-DL}$$

PL - Proximal latency DL ---- Distal latency

Minimal distance between two points of stimulation should be at least 10cm

Principles of sensory nerve conduction:

It can be measured antidromically or orthodromically.

For orthodromic conduction the distal portion of the nerve is stimulated and sensory nerve action potential recordings are taken from the proximal portion of the nerve.

In antidromic conduction, nerve is stimulated at a proximal point and action potential is recorded distally.

Recommended filter settings for sensory conduction is 10Hz to 2 kHz, sweep speed of 1-2ms/division and gain 1-5microvolts per division.

The measurement of sensory nerve conduction includes onset latency, amplitude, and duration of CMAP and nerve conduction velocity.

Latency of orthodromic conduction is measured from stimulus artifact to the initial positive or subsequent negative peak. It is shorter when compared to antidromic conduction¹⁶. The latency following orthodromic stimulation is shorter when compared to antidromic stimulation¹⁷.

The amplitude is higher in antidromic conduction when compared to orthodromic conduction¹⁸.

It is variable not only between two individuals but also between two sides of the same individual.

Amplitude of SNAP is reduced in antidromic conduction when the nerve is stimulated proximally.

Sensory conduction velocity is calculated by dividing the distance between the stimulating electrode and the recording electrode by the latency (in ms)

Late response waves (H Reflex & F wave)

Information about the conduction of impulses through the proximal segments of a nerve is provided by the study of the H Reflex and the F wave.

Sub maximal stimulation of mixed motor sensory nerves, insufficient to produce a direct motor response, none the less induces a muscle contraction (H wave) after a latency that is far longer than that of the direct motor response. This reflex is based on the activation of afferent fibres from muscle spindles and a long delay reflects the cumulative time required for the impulses to reach the spinal cord via the sensory fibres synapse with the anterior horn cells and to be transported along the motor fibres to the muscle. Stimuli of increasing frequency but low intensity cause a progressive depression and finally obliteration of H waves.

Supra maximal stimulation of mixed motor sensory nerve a second small muscle action

potential is recorded after a latency that is longer than for the direct motor response (latencies of 28-32ms in arms, 40-59ms in legs). This is F wave¹⁹. The name is attributed to their recognition in the small muscles of foot.

AIMS AND OBJECTIVES

A study of peripheral nerves is useful to know the damaging effects of alcohol on nervous tissue. With that objective the effect of alcohol abuse on nerve conduction is done in the study.

MATERIALS AND METHODS

The study group consisted of attendants of patients attending Neurology OP at Andhra Medical College who were asymptomatic and were having a habit of regular alcohol intake.

A total number of 40 male alcoholics were enrolled in the present study.

Out of them 12 subjects did not turn up and 8 were excluded from the study.

So finally the study group was reduced to 20 male alcoholics

The instrument used was VIKING SELECT EMG machine provided by VIASYS HEALTH CARE Ltd.

The nerves selected for the study are Ulnar nerve, Radial nerve, Sural nerve, Common peroneal nerve, Tibial nerve.

Criteria of inclusion are age between 25 to 50 yrs, consumption of 100 grams or above of alcohol at least 5 days a week and a minimum duration of alcohol intake of 2 yrs and above.

Criteria of exclusion

In the present study subjects with diabetes mellitus, alcohol withdrawal syndrome, leprosy and other neuropathies, HIV infection and Serological evidence of HbsAg positive, significant liver or kidney disease and malignancy were excluded from the study.

Screening test

In the screening tests Hemogram, Blood sugar levels, lipid profile, kidney function tests, liver function tests, Vitamin B12 levels, Serum protein A:G ratio, HIV Serology, CRP, RA factor, Anti DNA antibody, ECG, X ray Chest PA view, U/S Abdomen are done

After these test were done **NERVE CONDUCTION STUDIES** were performed at **NEUROLOGY DEPARTMENT, ANDHRA MEDICAL COLLEGE, VISAKHAPATNAM**

Reports presented in tabular form of 20 study subjects

Compound Motor Action Potential				Sensory Nerve Action Potential			
Amplitude		Latency		Amplitude		Latency	
Normal	Decreased	Normal	Increased	Normal	Decrease	Normal	Increased
13	7 (49% decrease in Amp)	19	1 (122% increase)	9	11	16	4

DISCUSSION

According to the previous studies most common electro diagnostic finding in patients with alcohol abuse is symmetrical axonal sensory motor neuropathy in the form of decreased amplitude, of sensory and compound motor action potential and

decreased velocity. This was attributed to malnutrition associated with alcohol intake by some and direct toxic effect by some workers.

In our study of twenty asymptomatic alcoholics, only 2 subjects had typical finding of absent or decreased amplitude of sural nerve potential

consisting with symmetrical axonal sensory motor neuropathy. Other findings in our study were Decreased amplitude of compound muscle action potential recorded in common peroneal nerves
Decreased amplitude of sensory nerve action potential recorded in radial nerves

The abnormal recordings in radial sensory recordings and peroneal motor are likely due to pressure occurring due to their superficial position in the spiral groove and at the neck of fibula respectively. These nerves are often vulnerable for compression during inebriated state.

The possibility of alcohol exerting a toxic effect on the roots remains a possibility.

Electroneurographic studies of alcoholic neuropathy show damaged sensory and motor nerve peripheral fibres caused by an axonal degeneration and consisting mainly of decreased sensory- and/or motor-evoked potential amplitudes with light involvement of conduction velocity (Kimura, 1989; Vuadens and Bogouslasky, 1998). Our electroneurographic findings indicate that the most frequent and earliest parameter to be affected is sensory-evoked potential amplitude, followed by sensory conduction velocity, motor-evoked potential amplitude and lastly motor conduction velocity. These results confirm the presence of emostly axonal neuropathy with more frequent and early involvement of sensory nervous fibres specially of the lower limbs.

As for aetiology and pathogenesis, the literature points out that alcoholic neuropathy is related to several factors: malnutrition, thiamine deficiency, direct toxicity of alcohol and, more recently, family history of alcoholism. According to some authors, alcoholic peripheral neuropathy is due mainly to malnutrition (Windebank, 1993), whereas according to other authors, neuropathy is related to the direct toxicity of alcohol on the peripheral nervous system (Estruch et al., 1993; Palliyath and Schwartz, 1993). In support of this latter hypothesis, an experimental investigation on animals showed that cytochrome P4502E1, an ethanol-inducible

isoenzyme of the P450-dependent pathway for ethanol oxidation in hepatocytes and neurons, may be involved in alcohol-related neurotoxicity (Wohrle et al., 1998). Moreover, a recent clinical cross-sectional study postulated that alcohol may have a dose-related toxic effect and could be considered an important risk factor for peripheral neuropathy (Monforte et al., 1995). With regard to family history of alcoholism and alcohol-related diseases, few studies have reported hereditary factors in chronic alcoholic patients in relation to hepatic (Hrubec and Omenn, 1981) or central nervous system diseases (Begleiter et al., 1983; Hill et al., 1986; Polich et al., 1994).

CONCLUSION

In our study the radial sensory recordings and peroneal motor recordings are more common abnormal findings.

Inference is these nerves are more vulnerable for compression during inebriated state due to their superficial position.

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