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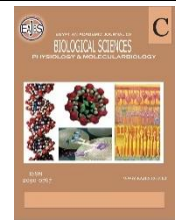
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Effect Of Cigarette Smoking on Nerve Conduction Velocity

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ABSTRACT

Background: Smoking causes changes in the myelin sheath leading to demyelination, causing blockage of nerve conduction and a decrease in conduction velocity. **Aim:** To assess the effect of cigarette smoking on nerve conduction velocity of median and ulnar nerve by nerve conduction study. **Methodology:** 50 smokers and 50 non-smokers were recruited and nerve conduction velocity of the median and ulnar nerve were studied in both groups. **Result:** The sensory nerve conduction velocity among smokers was significantly reduced when compared to non-smokers ($p < 0.05$). **Discussion:** The chemical oxidants that are released from cigarette smoke cause neural ischemia and also enhance oxidative stress. The sensory nerve is a thin nerve and it is more prone to damage by the chemicals released from cigarette smoke. **Conclusion:** The sensory nerve conduction velocity among smokers was reduced in our study.

INTRODUCTION

The chemical constituents in a cigarette include nicotine, tar, carbon monoxide, arsenic, ammonia, acetone, toluene, methylamine, pesticides, polonium-20, methanol etc (Abha Shrivastava *et al.*, 2017). Smoking causes an additional oxidative challenge by eliciting free radical activity and promoting oxidation and lipid peroxidation (Agrawal D *et al.*, 2007). Smoking and other tobacco use almost always begins at a younger age and a large percentage continue to smoke as adults, becoming lifelong smokers (Ahn HR *et al.*, 2011).

Lung cancer and ischemic heart disease such as atherosclerotic diseases of the myocardium and blood vessels are the two major fatal morbidities directly associated with smoking (Arnaud F *et al.*, 1990) (Arthur JR *et al.*, 1993). Chronic obstructive pulmonary disease (COPD) and other forms of cancer closely follow. Further, focusing chemicals in cigarette smoke have been implicated in causing changes in myelin sheaths of peripheral nerve (Asada A *et al.*, 2002). Myelin is a layer around the axon that is essential for the normal functioning of the nervous system. Smoking causes changes in the myelin sheath leading to demyelination. This can cause blockage of nerve conduction and decrease conduction velocity (Asada A *et al.*, 2002) (Baba S *et al.*, 2004).

Nerve conduction study is an essential tool in the evaluation of the peripheral nervous system (Baldassarri S *et al.*, 2016). The sensory nerve action potential (SNAP) provides information on the sensory nerve axon and its pathway from the distal receptors in the skin to the dorsal root ganglia, while the compound muscle action potential is an assessment of the motor nerve fibers from their origins in the anterior horn cell to their termination along muscle fibers (Banzet N *et al.*, 1999).

This test can accurately measure the degree of damage in large nerve fibres like the median nerve, revealing if symptoms are caused by degeneration of the myelin sheath. Conduction velocity is usually reduced in demyelinating neuropathies. The present study was conducted to assess if cigarette smoking has any impact on nerve conduction velocity.

MATERIALS AND METHODS

In this study, 100 male volunteers in the age group of 25 to 40 years, comprising 50 smokers and 50 nonsmokers were recruited from Makkalai Thedi Maruthuvam clinic. After explaining the procedure and purpose of the study, written informed consent was obtained. The case group included cigarette smokers, with no major illness like hypertension, diabetes mellitus, injury, trauma, or peripheral neuropathy in the past and present. Smokers were grouped on the basis of pack years

Pack years were calculated by multiplying the number of years smoked with the average number of packs per day (Scherer G *et al.*, 2006). Based on pack years of smoking, subjects were classified as:

Never smokers: 0.0 pack years.

Light smokers: 0.1 - 20.0 pack years.

Moderate smokers: 20.1 - 40 pack years.

Heavy smokers: > 40 pack years.

The control group included subjects who have never smoked and with no history of major illness like hypertension, diabetes mellitus, or peripheral neuropathy in the past and present. A detailed history was taken regarding their previous illness as well as any drug treatment that may affect the study. The study was approved by the Institutional ethical Committee.

Subjects were asked to come in the morning at 9.00 am after light breakfast. All the readings were recorded in a seating position at 25 degrees Celsius. The nerve conduction study was done using a 2-channel EMG equipment (Octopus). In the present study, motor and sensory nerve conduction of the median and ulnar nerves were measured. For motor nerve conduction velocity, the

median nerve was stimulated supramaximally at two points along its course respectively at the wrist and antecubital fossa (elbow). Recording and reference electrodes were placed over the abductor pollicis brevis along the thenar muscle border. The ground electrode was placed over the forearm. The stimulating site for median nerve motor study at the wrist is the middle of the wrist between tendons of flexor carpi radialis and palmaris longus and for motor study at the elbow, it is over brachial pulse. For sensory nerve conduction velocity, ring electrodes were placed at the proximal and distal interphalangeal joints of the index finger. These served as recording electrodes. Stimulating electrodes were placed at the wrist, cathode distal to the anode. The ground electrode was placed over the palm. With the help of stimulating electrodes, a sub-maximal stimulation was given over the middle of the wrist between the tendons of flexor carpi radialis and palmaris longus and antidromic conduction was recorded.

For motor nerve conduction velocity, the ulnar nerve was stimulated supramaximally at three points along its course respectively at the wrist, above the elbow and below the elbow. Recording and reference electrodes were placed over the abductor digiti minimi. The ground electrode was placed over the forearm. The stimulating site for ulnar nerve motor study at the wrist was the medial wrist, adjacent to flexor carpi ulnaris tendon, below the elbow is 3cm distal to the medial epicondyle and above the elbow was between biceps and triceps over medial humerus.

For sensory nerve conduction velocity, the recording electrodes were placed over the 5th metacarpal-phalangeal joint and over the distal interphalangeal joints. With the help of stimulating electrodes, a sub-maximal stimulation was given over the medial wrist, adjacent to the flexor carpi ulnaris tendon and antidromic conduction was recorded.

Statistical Analysis:

Data were tabulated by using mean and standard deviation for both motor and

sensory nerve conduction velocity. Paired t-test was used as a test of statistical significance between groups and a p-value less than 0.05 was considered to be statistically significant.

RESULTS AND DISCUSSION

The mean age of the cases was 37.73 ± 5.87202 . The mean pack years among

smokers were 3.613 ± 1.435 . The mean age of controls was 39.94 ± 6.110 (Table 1). shows the mean motor conduction velocity of both the median and ulnar nerves. Table 02 shows the sensory conduction velocity of both the median and ulnar nerves.

Table 1: Motor conduction velocity.

Nerve	Conduction velocity		p value
	Cases	Controls	
Right median nerve	53.364 \pm 5.852	57.018 \pm 5.820	*0.0023
Left median nerve	53.352 \pm 5.057	53.802 \pm 5.098	0.61
Right ulnar nerve	38.687 \pm 7.882	40.087 \pm 8.126	0.384
Left ulnar nerve	40.357 \pm 4.153	38.137 \pm 7.592	0.07

* - Significant

Table 2: Sensory conduction velocity

Nerve	Conduction velocity		p value
	Cases	Controls	
Right median nerve	50.312 \pm 4.918	56.133 \pm 7.238	*<0.0001
Left median nerve	50.901 \pm 6.149	58.544 \pm 6.422	*<0.0001
Right ulnar nerve	41.215 \pm 7.557	49.638 \pm 9.118	*<0.0001
Left ulnar nerve	36.973 \pm 5.434	39.801 \pm 6.682	*0.0223

* - Significant

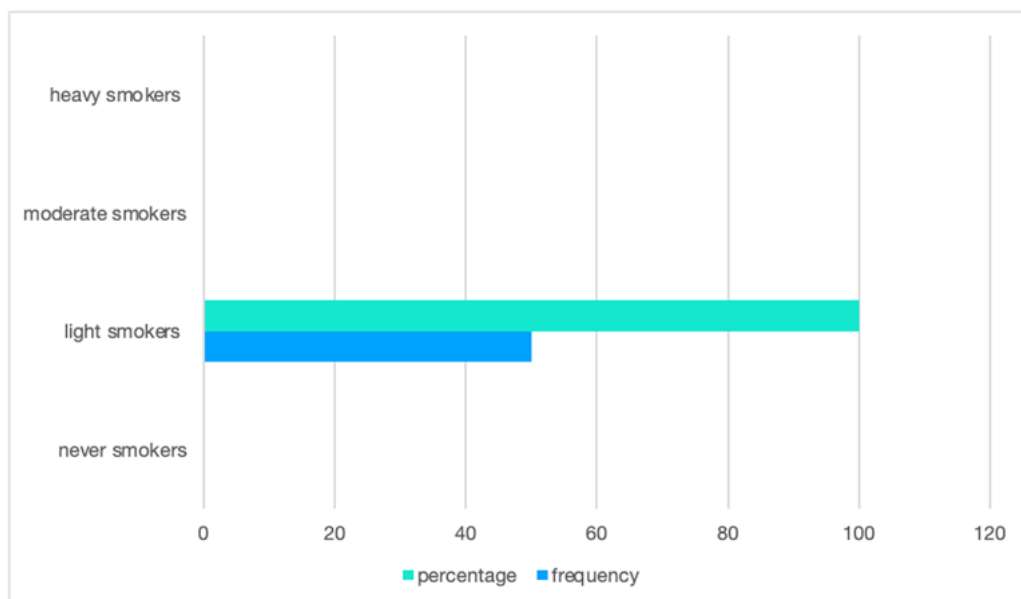


Fig. 1: Classification of smokers based on pack year.

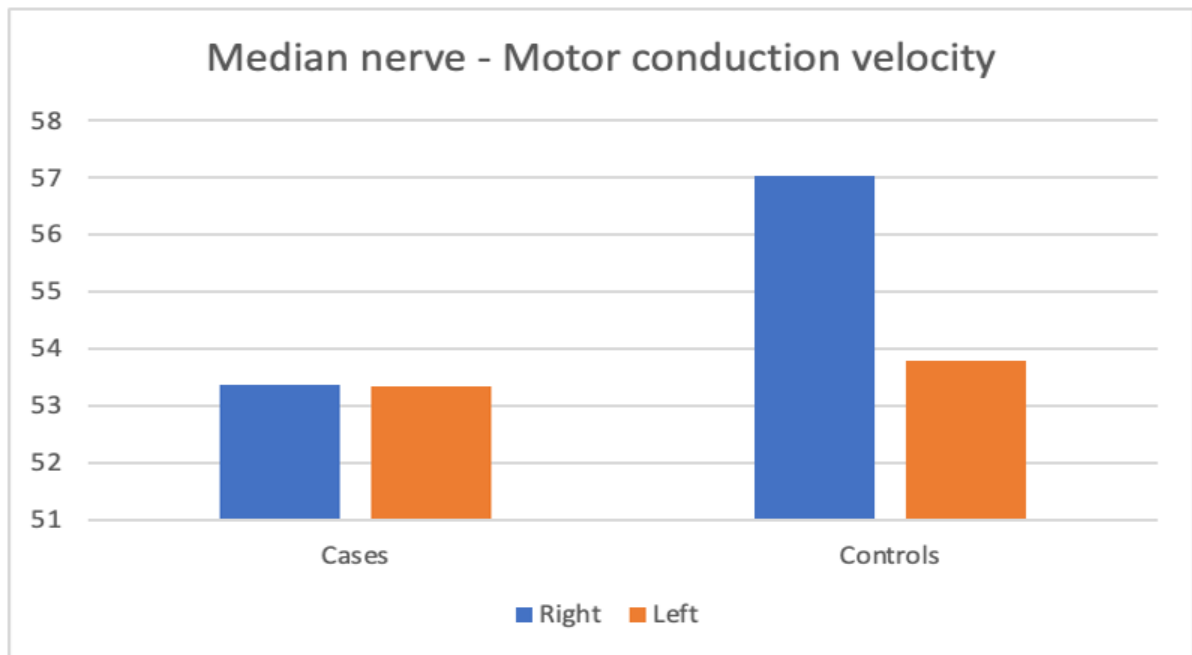


Fig. 2: The motor nerve conduction velocity of right and left median nerve among cases and controls.

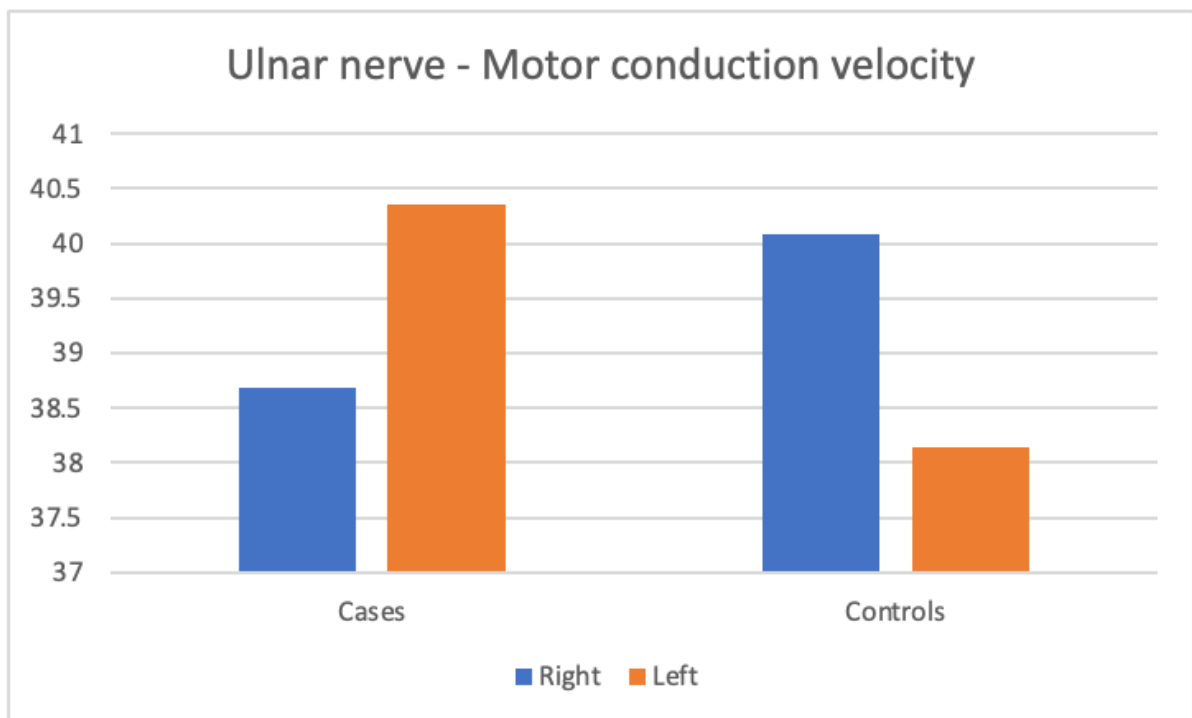


Fig. 3: The motor nerve conduction velocity of right and left ulnar nerve among cases and controls.

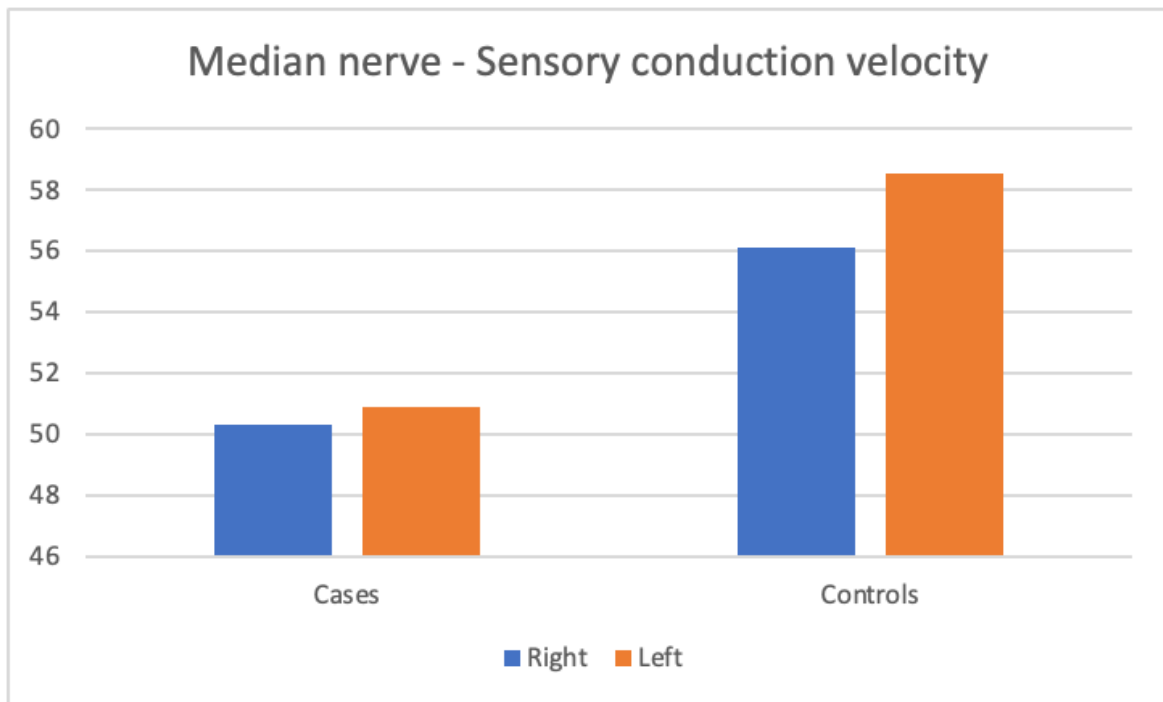


Fig. 4: The sensory nerve conduction velocity of right and left median nerve among cases and controls.

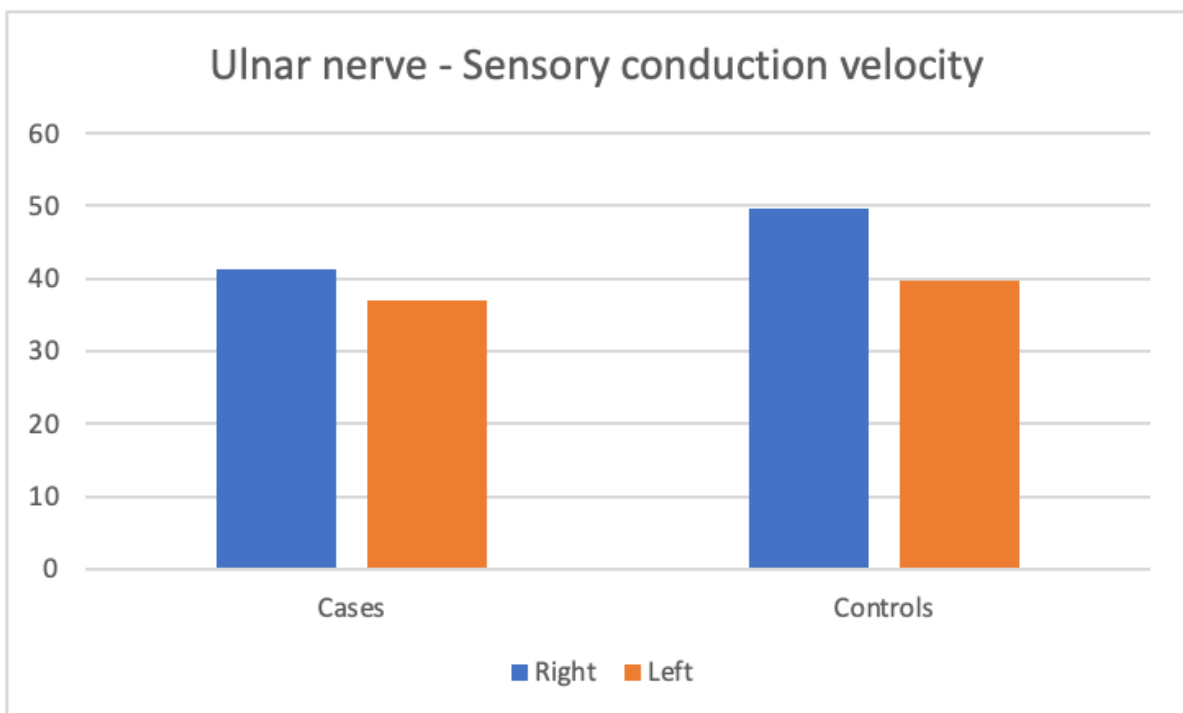


Fig. 5: The sensory nerve conduction velocity of right and left ulnar nerve among cases and controls.

In the present study, it was found that there were statistically significant changes in the conduction velocity of sensory nerves but not in motor nerves. Nerve conduction

studies provide a means of demonstrating the presence and extent of peripheral neuropathy (Calcerrada P *et al.*, 2009). Cigarette smoking causes a lot of impacts on the human body

(Carlson SG *et al.*, 2001). Cigarette smoking is a serious health problem. Cigarette smoke is a complex mixture of chemical compounds containing many free radicals and oxidants (Cazaubon S *et al.*, 2009). Exposure to highly reactive oxygen species generated by cigarette combustion can cause oxidative damage and trigger a strong inflammatory cascade that can lead to the onset and/or facilitate the progression of many CNS disorders (Chen JK *et al.*, 1998). Cigarette smoking can cause neural ischemia which may affect the peripheral ends of nerves and they slowly proceed towards centre (Damir Janigro *et al.*, 2010). Smoking induces subclinical changes in myelin sheath leading to demyelination of nerves and consequently decrease in conduction velocity. Chronic smoking can induce alteration in membrane permeability properties of tissue and can result in changes in signal transduction and electrolyte imbalance (De Jongh RT *et al.*, 2003). Neural tissue is highly susceptible to oxidative damage caused by free radicals since it consists of a pool of unsaturated lipids which are liable to peroxidation and oxidative modification (DeMaster EG *et al.*, 2001) (James W Albers *et al.*) (Misra UK, 2012) (Low PA *et al.*, 1997). The present study showed that there was a statistically significant decrease in the sensory nerve conduction velocity of ulnar and median nerves as compared to motor nerve conduction velocity. The results of our study were in accordance with those observed by Agarwal *et al.*, Motital *et al.* and Abha Shrivastava. Smoking enhances oxidative stress not only through the production of reactive oxygen radicals but also through the weakening of the antioxidant defense system (Low PA *et al.*, 1997). In our study all cases were in the range of light smokers and they all showed a significant reduction in the sensory nerve conduction velocity of the median and ulnar nerve. In our study, there were no significant changes observed in motor nerve conduction velocity of the ulnar and median nerve even though there was a reduction in conduction velocity. This decrease in sensory nerve conduction may be due to the fact that

sensory nerves are thinner than motor nerves and have shorter internodal distances.

Conclusion:

The present study showed that smoking was associated with a reduction in sensory nerve conduction of the median and ulnar nerves. The oxidative stress may be responsible for nerve conduction deficits among cigarette smokers. Further studies can be done to estimate the plasma antioxidant level among smokers and assess all peripheral nerves to find out the association.

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