Original Article

Assessment of motor nerve conduction in healthy obese Indian population

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Abstract

Background and Aim: Motor nerve conduction studies help to understand the functional status of the peripheral and central nerves. Some of the studies in industrial workers have correlated the incidence of carpal tunnel syndrome with obesity and studied the function of the median nerve alone. Therefore, this study was aimed at assessing nerve conduction of major peripheral nerves in the obese individual without any comorbid condition or systemic complication.

Methods: Upon meeting the inclusion and exclusion criteria, 52 age-matched subjects were included in the study. They were divided into two groups (25 in control and 27 in study group; obese) based on their body mass index. Motor nerve conduction parameters (standardized distal motor latency in ms, amplitude of compound muscle action potential in mV and motor nerve conduction velocity in m/s) were recorded and the difference in these parameters between the groups was assessed using independent *t*-test.

Results: All the parameters depicted decreased motor conduction velocity in peripheral nerves (median, ulnar, tibial and common peroneal) in obese individuals compared with the control group. There was a significant prolongation of latency in all nerves and decrease in amplitude except in the tibial nerve. There was also a significant decrease in conduction velocity of tibial nerve in obese subjects compared to controls.

Conclusion: From the present study, we observe that in obesity, there are increase in motor nerve latencies, decrease in the amplitude of action potentials and conduction velocity, which indicate slow transmission in peripheral nerve fibers.

Key words: Body mass index, compound muscle action potential, motor nerve conduction, motor nerve conduction velocity, standardized distal motor latency

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INTRODUCTION

Motor nerve conduction study (NCS) is commonly advised in clinical practice as a part of electrodiagnostic procedures that help in establishing the types and degrees of abnormalities of the nerves. NCS establishes diagnosis very early and more accurately than other electrodiagnostic techniques because of its sensitivity to detect conduction slowing (or block), which is an early indicator of nerve entrapment or peripheral neuropathy.[1]

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In NCS of peripheral motor nerves, the latency, amplitude of action potential and nerve conduction velocity are the parameters recorded.

Motor nerve conductions are usually found to be affected in various clinical conditions like neuropathies (peripheral and central), metabolic and endocrine disorders such as diabetes, hypothyroidism. Obesity, a state of excess adiposity and a disorder of nutrition and energy homeostasis also affects motor nerve conduction by altering the metabolism and by creating a variety of co-morbid conditions such as insulin resistance, diabetes, hypertension, hyperlipidemia and hyperandrogenism, which can affect the motor neuron function.[2] Lipids are major components of a nerve cell. Therefore, both deficiencies of fat (malnutrition) and excess adiposity (obesity) are expected to interfere in neuronal structure and functions.[3,4] Body mass index (BMI) is a good indicator of body fat content and it can be easily

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calculated and also clinically applicable.[5] According to WHO classification, obesity is graded into three categories.[6]

Recently, it has been reported that obesity is associated with more incidence of carpal tunnel syndrome (CTS), which was suggested to be induced due to mechanical compression of the median nerve in carpal tunnel.[7-9] However, later it was found that the slowing of nerve conduction in CTS was due to metabolic complication of obesity rather than mechanical compression *per se*.[10] It was reported that median mononeuropathy at the wrist is 2.5 times more common in individuals classified as obese (BMI > 29) when compared to slender ones (BMI < 20).[11] It has been observed that obese people are more prone to CTS.[12] Wilkinson found that CTS was most likely to occur during the third trimester of pregnancy when weight gain is most rapid.[13] Thus, obesity acts as a risk factor for CTS both in adults as well as in children.[7,14,15] Lam and Thurston showed that obesity and CTS are statistically related.[16] Becker *et al*. suggested that the strong association between obesity and CTS could be due to accumulation of fat tissue inside the carpal tunnel or by an increase in hydrostatic pressure through this canal, exerting a compressive effect on the median nerve in carpal tunnel.[8] However, it was later investigated that slowing of median nerve conduction at the wrist is not due to influence of carpal tunnel pressure, rather due to endoneurial edema, which is proposed to be a metabolic complication of obesity.[10,17] Recently, it has also been observed that neural dysfunction occurs in obese Zucker rats.[18] There are also reports showing a decrease of stimulus-response slope with BMI.[19] However, till date no systematic study has been done in obese, asymptomatic individuals to assess their motor nerve conduction velocities in both upper and lower limb nerves. Therefore, in this study we have assessed motor nerve conduction velocities in obese individuals.

MATERIALS AND METHODS

After obtaining the approval of Institute Research Council and Ethics Committee, a total of 52 subjects were recruited from medicine out-patient department of Jawaharlal Institute of Postgraduate Medical Education and Research. The control group subjects were volunteers from hospital staff, medical students and relatives accompanying patients. The presence of neuromuscular or any musculoskeletal disorder was ruled out by a brief history and clinical examination by the medicine consultant.

Subjects with hypertension, diabetes mellitus, hypothyroidism, symptoms of numbness, tingling, pain, compression neuropathy, neuromuscular disorder, peripheral nerve injury, hereditary or genetic myopathy, neuropathy or radiculopathy, history of limb fracture, disorders of central nervous system were excluded from the study. The subjects were explained about the procedure, and informed consent was taken. Anthropometric measurements like age, sex, height (in meter), weight (in kg), head circumference (in cm), arm length (in cm) were recorded. BMI was calculated as weight in kg divided by height in meter squared.[20] They were divided into two groups: Study group (n = 27) and control group (n = 25). In subjects of the study group, fasting serum glucose and thyroid profile were assessed to rule out diabetes mellitus and hypothyroidism.

Totally 52 subjects of age group 17–50 years were included in the study out of which 30 were male and 22 were female. During history taking, none of the subjects were found to be using any artificial pacemaker. The laboratory temperature was maintained at 25–27°C.

Brief procedure

The motor nerve conduction recordings were performed using EP-EMG machine (NIHON KOHDEN-NEUROPACK Machine). The subjects were allowed to lie down on a couch and relax fully to ensure good recordings. The area of the skin was cleaned thoroughly with spirit to remove dirt, dead cells and grease. The cup or disc electrodes (Ag-AgCl) of 1 cm diameter filled with conducting jelly were fixed on the skin of recording area with transpore tape. These electrodes were connected to the oscilloscope through the preamplifier. After 10 min of rest and adaptation to the laboratory environment, electrodiagnostic tests were performed following the standard procedures.[21]

The recordings were performed with standard equipment settings of sensitivity 5 mV/division, sweep speed 5 ms/division, stimulus duration 0.2 ms, low frequency filter 10 Hz, high frequency filter 5 KHz by using supramaximal strength of stimuli. Motor NCS was performed for the peripheral nerves like median, ulnar in upper limb and tibial, common peroneal in lower limb. The recording electrode was placed on the motor point (point at which the nerve enters into the muscle), the reference electrode was placed distally, and the ground electrode was placed in between recording and stimulating electrodes. The cathode of the stimulating electrode was placed 8 cm proximal to the recording electrode. Each nerve was stimulated twice, once at a distal point and next at a proximal point following which the respective action potentials were recorded on the oscilloscope.[22] Recordings were taken only in the dominant side [Figure 1]. The nerve was stimulated to record the muscle action potential and the following parameters were measured: Standardized distal motor latency in ms, amplitude of compound muscle action potential in mV (CMAP) and motor nerve conduction velocity in m/s.

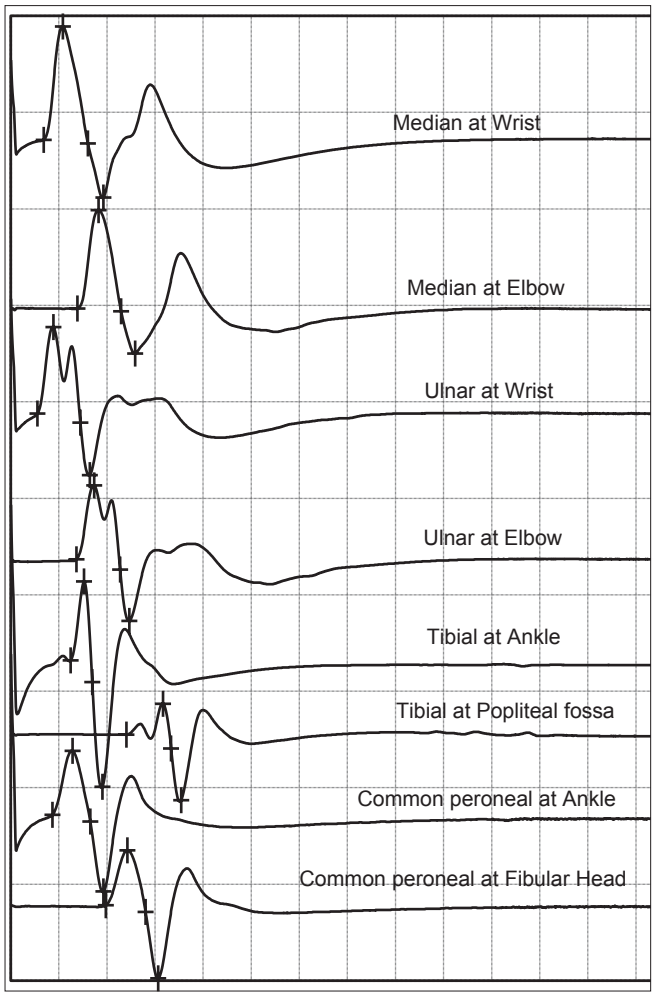

Figure 1: Motor nerve conduction wave forms recorded from median, ulnar, tibial and common peroneal nerve of right side

Standardized distal motor latency

This was calculated by measuring the period from the onset of stimulus artifact to the onset of the muscle action potential in ms. This is only calculated from distal recording obtained by stimulating the nerve at wrist.

Amplitude of compound muscle action potential

This was measured as peak to peak amplitude in mV.

Motor nerve conduction velocity

The motor conduction velocity (in m/s) was calculated by dividing the distance between the two points of stimulation by the difference of latencies (subtracting distal latency from proximal latency).

Statistical analysis of data

The statistical analysis was done by using Statistical Package for Social Sciences (version 16, SPSS Software Inc., Chicago, IL, USA). The demographic data (age and BMI) were presented as mean ± standard deviation and nerve conduction parameters were presented as mean ± standard error of the mean as the data was normally distributed. The level of significance was tested by using unpaired Student's *t*-test. $P < 0.05$ was considered statistically significant.

RESULTS

The demographic data obtained by descriptive statistics [Table 1] depicts the age and BMI of both the groups.

There was a significant prolongation of latency ($P = 0.000$) and decrease in amplitude ($P = 0.03$), but no significant change of conduction velocity of motor conduction of median nerve in obese subjects compared with their control [Table 2]. In ulnar nerve, there was a significant prolongation of latency ($P = 0.015$) and decrease of amplitude ($P = 0.042$), but the change in conduction velocity of motor conduction was not significant in obese subjects [Table 3]. There was a significant, prolongation of latency ($P = 0.000$), decrease of motor conduction velocity ($P = 0.000$) and increase in the amplitude of motor conduction of tibial nerve ($P = 0.008$) in obese subjects compared to control [Table 4]. Common peroneal nerve depicted a significant prolongation of latency ($P = 0.003$) and reduction of amplitude ($P = 0.02$), but there was no change in the conduction velocity of motor conduction in obese subjects [Table 5].

DISCUSSION

Motor nerve conduction is an important electrodiagnostic procedure for evaluation of structural and function integrity of motor neurons. The latency of CMAP indicates the speed of conduction in nerves, whereas the amplitude of CMAP refers to the density of nerve fibers and the muscle mass activated by stimulation of the motor nerve.[22] The conduction velocity of the nerve depends on the fiber diameter, degree of myelination and the internodal distance. As the axon increases in size, the myelin sheath becomes thicker, and the internodal distance becomes longer. The conduction, therefore, becomes faster. In the present study, motor conduction tests of both upper and lower limb revealed that there was significant increase in latency of CMAP of all nerves, and significant decrease in amplitude of CMAP of all nerves except in tibial nerve, where there was significant increase and decrease in velocity of nerve conduction only in tibial nerve in obese subjects compared to controls [Figure 2-4].

All the above findings suggest a decrease in motor conduction velocity of median, ulnar, tibial and common peroneal nerve in obese individuals [Figure 4], which is in contrast to the findings of McHugh *et al*. who reported that

BMI has no influence on nerve excitability.[23] However, our findings are similar to the report of Buschbacher except that they observed no difference in latency and amplitude of median and tibial nerves and in nerve conduction velocity of the median nerve [Figure 2-3].[24] Thus, the present study reveals dysfunctions of all components of the median nerve functions.

Normally, the motor conduction velocity depends on many physiological factors like thickness of the nerve fiber (degree of myelination), mechanical compression from surrounding peripheral tissues, ionic changes, temperature, age, height of the individual, and gender.[22] As conduction velocities recorded in the most of the nerves are peripheral nerves, the mechanical compression leading to conduction impairment in the present study could be one of the reasons. In case of CTS, it was proposed to be due to accumulation of fat tissue inside the carpal tunnel or by an increase in hydrostatic pressure as a result of fluid retention in the soft tissues of carpal tunnel, exerting a compressive effect on the median nerve.[8,11,12] Another possibility could be the increased translocation of blood volume from the legs after assumption of the recumbent position.[25] It was also suggested that slowing of median nerve conduction at the wrist is not due to influence of carpal tunnel pressure, rather due to endoneurial edema, which is proposed to be a metabolic complication of obesity.[10] However, the ionic changes and change in myelination that have not been assessed in our study might also contribute.

Most of the studies conducted before have been on patients of CTS, where obesity was one of the prominent associated features.[7,10,26] In CTS, there is prolongation of motor latencies.[27] Radecki found that prolongation of median latency and slowing of conduction was associated with increased BMI.[28] It was observed by Albers *et al*. that type II diabetic patients with median mononeuropathy had higher BMI than the remaining type II patients and there was a tendency for the frequency of median mononeuropathy to increase with increasing BMI.[26] This suggests that obesity *per se* affects median nerve conduction rather than increased serum glucose level.

The most significant finding of the present study is an increase in the amplitude of CMAP recorded from posterior tibial nerve. Posterior tibial nerve supplies abductor hallucis longus, which anatomically forms and maintains the medial longitudinal arch of foot.[29,30] This arch is the most weight bearing component of the body and muscle involved in strengthening this arch bears the brunt of the body weight. Therefore, in obesity abductor hallucis longus, which maintains the arch, might have undergone hypertrophy due to increased constant load on it, leading to the increase in amplitude of CMAP as normally height of CMAP depends primarily on the muscle mass. However, this must be investigated

Table 1: Demographic data of study group (*n*=27) and control group (*n*=25)

	Mean±SD	
	Control	Study
Age	23.40±7.211	22.26±8.263
BMI	20.98±1.340	33.54±1.561***

BMI: Body mass index, SD: Standard deviation

Table 2: Comparison of different parameters (latency, amplitude and conduction velocity) of motor conduction of median nerve in normal healthy subjects (control group; *n*=25) and obese subjects (study group; *n*=27)

	Latency (ms)	Amplitude (mV)	Conduction velocity (m/s)
Control	2.8±0.06	10.2±0.27	58.0±0.79
Obese	3.5±0.09***	8.9±0.50*	53.96±2.13

Values are mean±SEM. *P<0.05, ***P<0.001. SEM: Standard error of the mean

Table 3: Comparison of different parameters (latency, amplitude and conduction velocity) of motor conduction of ulnar nerve in normal healthy subjects (control group; *n*=25) and obese subjects (study group; *n*=27)

	Latency (ms)	Amplitude (mV)	Conduction velocity (m/s)
Control	2.59±0.103	9.73±0.32	62.53±1.35
Obese	2.94±0.094**	8.51±0.48*	61.84±1.04

Values are mean±SEM. *P<0.05, **P<0.01. SEM: Standard error of the mean

Table 4: Comparison of different parameters (latency, amplitude and conduction velocity) of motor conduction of tibial nerve in normal healthy subjects (control group; *n*=25) and obese subjects (study group; *n*=27)

	Latency (ms)	Amplitude (mV)	Conduction velocity (m/s)
Control	3.21±0.13	10.85±0.32	54.14±0.89
Obese	4.27±0.21***	13.36±0.82**	49.14±0.81***

Values are mean±SEM. **P<0.01, ***P<0.001. SEM: Standard error of the mean

Table 5: Comparison of different parameters (latency, amplitude and conduction velocity) of motor conduction of common peroneal nerve in normal healthy subjects (control group; *n*=25) and obese subjects (study group; *n*=27)

	Latency (ms)	Amplitude (mV)	Conduction velocity (m/s)
Control	3.64±0.08	6.2±0.41	55.14±0.95
Obese	4.14±0.14**	4.79±0.42*	52.8±1.04

Values are mean±SEM. *P<0.05, **P<0.01, Values as compared to control. SEM: Standard error of the mean

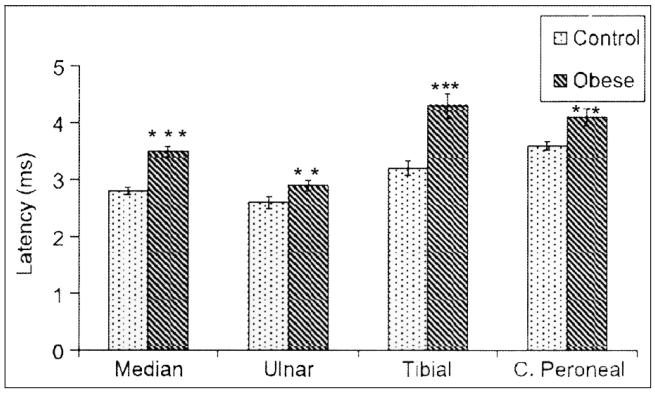

Figure 2: Comparison of standardized distal motor latencies as recorded in motor nerve conduction studies of various peripheral nerves. Values are mean ± standard error of the mean; ***P* < 0.01, ****P* < 0.001; values as compared to control

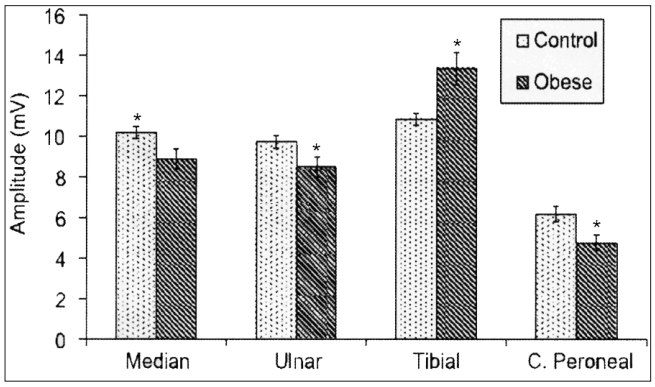

Figure 3: Comparison of compound muscle action potentials as recorded in motor nerve conduction studies of various peripheral nerves. Values are mean ± standard error of the mean; **P* < 0.05, ***P* < 0.01; values as compared to control

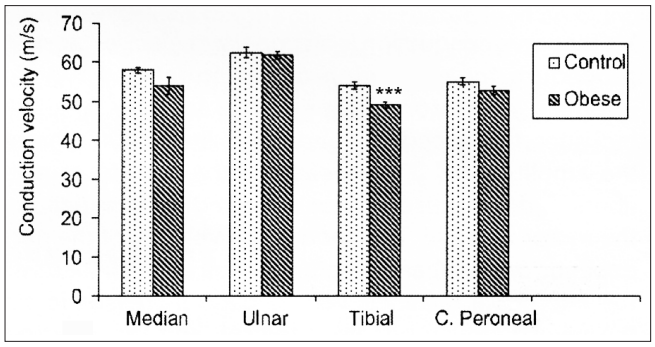

Figure 4: Comparison of motor conduction velocities as recorded in motor nerve conduction studies of various peripheral nerves. Values are mean ± standard error of the mean; ****P* < 0.001; values as compared to control

by muscle biopsy and detail molecular study of the muscle. As the study group subjects were asymptomatic without any comorbid condition, and the parameters in them were within the normal range, these findings do not point toward any disease process. However, a significant difference in parameters from control groups indicates decrease in functional status of neurons, which should not be ignored on a long-term basis. As obesity retards neuronal functions before clinical neurological manifestations, attempts should be made to detect these changes early through various electrodiagnostic procedures in obese individuals for their prevention. We suggest that steps should be taken to reduce obesity by various lifestyle modification measures like exercise and diet restriction to improve nerve function.

Limitations of the study

We have not assessed body composition and the contribution of body fat percentage to the nerve conduction deficits. H reflex and F-wave, that indicates conduction profile in the reflex arc in the spinal cord and the gender difference in the conduction velocity has not been assessed.

CONCLUSION

From the present study we conclude that in obesity, there is increase of motor nerve latencies and decrease in conduction velocity, indicating that transmission becomes slower in peripheral nerve fibers, and decrease in amplitude of action potentials except in tibial nerve CMAP, which suggests increased muscle mass of abductor hallucis longus due to increased load on plantar arch. Since the parameters recorded show a tendency toward delay or slowing in impulse transmission in nerve fibers in obese individuals in comparison to nonobese subjects, we suggest that obesity *per se* has some degree of deleterious influence on axonal functions.

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