

## MOTOR NERVE CONDUCTION STUDY OF LOWER LIMB NERVES IN DIABETICS

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### ABSTRACT

**Background :** The physiological properties of nerve and muscle are usually modified due to pathophysiological changes resulting from many diseases like diabetes. Diabetic peripheral neuropathy (DPN), the common complication of diabetes, can be assessed electrophysiologically by nerve conduction studies(NCS).

**Aim:** A motor nerve conduction study of lower limb nerves in diabetics.

**Material and Methods:** The peroneal and tibial motor nerve conduction study was carried out on 40 male type 2 diabetic patients attending diabetic clinic in the department of medicine and 40 healthy male volunteers who served as control. Motor Distal latency (MDL), Amplitude(Amp) and Conduction Velocity(CV) were measured by using Computerized RMS EMG EP Mk II and surface electrodes.

**Results:** On comparing the parameters of NCS it was found that motor distal latency of both the nerves were higher in diabetics than controls with statistically significant difference. Results also show decreased amplitude and conduction velocities of nerves of both(right and left) sides in diabetics (statistically significant). All the parameters were found correlated with blood sugar levels in diabetics.

**Key words :** Type 2 diabetes mellitus, peripheral neuropathy, peroneal nerve, tibial nerve , motor distal latency, conduction velocity.

### INTRODUCTION

Diabetes mellitus is one of the most common chronic diseases in nearly all countries, and continues to increase in numbers and significance, as changing lifestyles lead to reduced physical activity, and increased obesity.<sup>[1]</sup> It is frequently asymptomatic. The associated complications

of diabetes like neuropathy may be the first clinical indication of disease.<sup>[2,3]</sup>

Diabetic peripheral neuropathy(DPN) is a common complication of diabetes mellitus. It affects up to 50% of patients and predisposes the patients to severe functional limitations. Its end stage complications such as foot ulceration and amputation are associated with substantial health care costs and reduced quality of life.<sup>[4]</sup> .The rate of lower limb amputation is 15 times higher in diabetic patients compared with non diabetic patients, and more than 50% of diabetic amputees need a subsequent amputation of the contra lateral limb within 4 years of the loss of the first leg.<sup>[5]</sup> However progression of neuropathy can be reduced by early detection and intervention.<sup>[6]</sup> Nerve conduction studies(NCS) are helpful in early detection of neuropathy. Use of nerve conduction in assessing therapy in preventing or ameliorating neuropathy is desirable because abnormalities of nerve conduction are objective, most sensitive, specific, repeatable and therefore useful in the evaluation of diabetic neuropathy.<sup>[7]</sup>

The present study was designed to identify the presence of subclinical DPN by means of NCS. The findings will be helpful for early diagnosis of diabetic peripheral neuropathy.

### MATERIAL AND METHODS

The present study was carried out in the Department of Physiology in collaboration with the Department of Medicine of Pad. Dr. D.Y.Patil Medical College Pimpri, Pune, Maharashtra. A total of 80 male subjects in the age range of 40-65 years were selected for study. Out of these, 40 male subjects were diagnosed Type 2 diabetic patients attending diabetic clinic in the department of medicine and rest 40 were healthy male volunteers who

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served as control. The study was a comparative study between type 2 diabetic patients and age, sex, height, weight and BMI matched healthy non diabetic subjects. All the patients with any other type of neuropathy, chronic musculoskeletal disorders, retinopathy, nephropathy, any other chronic disease, alcoholics and smokers were excluded from the study. Detailed socio-demographic data, family history and medical history were taken from all the subjects and their physical and clinical examinations were done on very first day of the visit to OPD. The details of study were explained and informed consent was taken from each of the subjects. On the day of experiment fasting blood sample was collected for blood sugar analysis followed by nerve conduction study.

Anthropometric measurements (height and weight) were taken by using scales on bare foot. Body mass index (BMI) was found out. Both fasting and post prandial blood glucose levels were estimated by glucose oxidase(GOD/POD) method.<sup>[8]</sup> Motor nerve conduction study of Deep Peroneal and Tibial nerves were performed on both sides of the body in an environment with room temperature ranging from 23° C to 25° C using computerized RMS EMG EP MK II machine and surface electrodes. With the help of stimulating electrodes supramaximal stimulation was given at two different sites (distal site and proximal site) to obtain compound muscle action potential (CMAP). For peroneal nerve, the distal stimulation (S1) was given at ankle between the extensor digitorum longus and extensor hallucis longus tendon. Proximal stimulation (S2) was done behind and proximal to the fibular head. Active electrode for recording was placed over the belly of the extensor digitorum brevis muscle and reference electrode over the muscle tendon 3cm distal to the active electrode. For tibial nerve, the distal stimulation (S1) was given behind and proximal to the medial malleolus. Proximal stimulation (S2) was done in popliteal fossa, along the flexor crease of the knee, slightly lateral to the midline of the popliteal fossa. Ground electrode was placed between stimulating electrode and recording electrode for both the nerves. Distance between S1 and S2 was

measured in mm by measuring tape for both the nerves.<sup>[9]</sup> Motor Distal latency (MDL), Amplitude(Amp) and Conduction Velocity(CV) were measured.

The study was approved by the Ethics Committee of our institution. Analysis of data was done using Microsoft Excel and EPI INFO 2007. Relationship between predictors and outcomes were examined by Pearson correlation coefficient.

## RESULTS

In the present study 40 male diabetic subjects with duration of disease of  $2.28 \pm 1.51$  years were compared with 40 non diabetic (control) subjects of same age group, BMI and sex. The baseline characteristics of subjects are summarized in table 1. On comparing the parameters of nerves of both the groups (summarized in table 2) it was found that motor distal latency of (right and left) peroneal and tibial nerves were significantly higher ( $p < 0.05$ ,  $p < 0.0001$  respectively) in diabetics. Results also show significantly ( $p < 0.0001$ ) decreased amplitude of CMAP and conduction velocities of peroneal and tibial nerves of both sides in diabetics. Further, MDL was found positively correlated with blood sugar and CV was found negatively correlated with blood sugar in diabetics as shown in table 3.

**Table 1 : Baseline Characteristics Of Study Population**

Characteristics	Diabetics Mean $\pm$ SD	Controls Mean $\pm$ SD
Participants(n)	40	40
Age(years)	55.77 $\pm$ 7.12	53.62 $\pm$ 5.05
Weight(Kg)	74.6 $\pm$ 14.58	71.00 $\pm$ 8.66
Height(m)	1.71 $\pm$ 0.04	1.69 $\pm$ 0.03
BMI(Kg/m <sup>2</sup> )	25.2 $\pm$ 3.60	24.63 $\pm$ 2.11
Fasting Blood Sugar (mg%)	142.60 $\pm$ 31.38	94.05 $\pm$ 10.83
Post Prandial Blood Sugar (mg%)	235.02 $\pm$ 74.88	125.17 $\pm$ 12.4

**Table 2: Parameters of nerve conduction study in diabetics and non diabetics**

Parameters	Subjects	Peroneal		Tibial	
		Left	Right	Left	Right
MDL (mSec)	Diabetics Mean $\pm$ SD	3.50 $\pm$ 0.52	3.44 $\pm$ 0.48	3.99 $\pm$ 0.72	3.78 $\pm$ 0.80
	Controls Mean $\pm$ SD	3.23 $\pm$ 0.66	3.19 $\pm$ 0.63	3.38 $\pm$ 0.61	3.36 $\pm$ 0.56
	t value (P value)	2.01(0.047)	2.01(0.047)	4.08(0.0001)	2.69(0.008)
AMP (mV)	Diabetics Mean $\pm$ SD	5.09 $\pm$ 2.49	4.93 $\pm$ 2.16	12.16 $\pm$ 5.85	13.52 $\pm$ 6.54
	Controls Mean $\pm$ SD	7.21 $\pm$ 2.06	6.31 $\pm$ 1.59	19.71 $\pm$ 5.05	19.70 $\pm$ 5.49
	t value (P value)	4.14(0.0001)	3.25(0.0017)	6.16(0.0000)	4.56(0.0000)
CV (m/Sec)	Diabetics Mean $\pm$ SD	46.03 $\pm$ 6.39	45.25 $\pm$ 5.76	41.42 $\pm$ 4.42	41.63 $\pm$ 5.02
	Controls Mean $\pm$ SD	50.05 $\pm$ 4.49	49.86 $\pm$ 3.35	47.33 $\pm$ 1.63	47.73 $\pm$ 2.68
	t value (P value)	3.25(0.0017)	4.37(0.0000)	7.93(0.0000)	6.76(0.0000)

Note :

P< 0.05 Statistically significant

P< 0.0001 Statistically highly significant

#### Abbreviations

MDL (Motor distal latency)

AMP (Amplitude)

CV (Conduction Velocity)

#### Units

mSec :millisecond

mV : millivolt

m/s : meter/ second

**Table 3 .Correlation of Motor distal latency and Conduction velocity of nerves with Blood Sugar in diabetics (Pearson Correlation)**

Nerves	Blood Sugar	Motor distal latency (r value)		Conduction Velocity (r value)	
		Right	Left	Right	Left
Peroneal nerve	Fasting Blood Sugar	+0.068	+0.272	-0.002	-0.064
	PP Blood Sugar	+0.499	+0.449	-0.335	-0.181
Tibial nerve	Fasting Blood Sugar	+0.120	+0.020	-0.013	-0.047
	PP Blood Sugar	+0.002	+0.091	-0.001	-0.011

## DISCUSSION

The present study reveals alteration in electrophysiological parameters of peroneal and tibial nerves in diabetics. There are many mechanisms by which hyperglycemia causes nerve damage. Hyperglycemia reduces myoinositol but increases glucose influx through polyol pathway and reduces Na<sup>+</sup> K<sup>+</sup> ATPase activity. Increased Na<sup>+</sup>, K<sup>+</sup>, sorbitol and fructose concentrations in the nerve absorb water through osmosis. Accumulated water causes the compression of the nerve, which leads to decreased

axonal transport. Pathological features of diabetic peripheral neuropathy include distal axonal loss with fall out of large (myelinated) and small fibers, focal demyelination and regeneration.<sup>[10,11]</sup> Hyperglycemia also leads to elevated intracellular glucose and cellular toxicity in the endothelial cells of the capillaries associated with peripheral nerves.<sup>[12]</sup> In addition intracellular glucose can be converted to so called Amadori product, and these in turn can form advanced glycosylated end products (AGEs), which cross-link matrix proteins. This damages the blood vessels.<sup>[13]</sup> This results in ischemia of the nerves of the patient which may be responsible for neuropathy. Endothelial injury, and release of inflammatory mediators such as circulating cell adhesive molecules due to poor glycemic control have been also implicated. These molecules are important long term predictors of DPN.<sup>[14]</sup>

Many previous studies have also found NCS alterations suggestive of neuropathy in diabetics. Kimura J et al also found increased latency and decreased conduction velocity of peroneal and tibial nerves in diabetics as compared to normal subjects.<sup>[15]</sup> W. Hoffman et al found conduction velocity of peroneal nerve was significantly slower in the diabetic subjects than in the control.<sup>[16]</sup> Baba M found fall in motor amplitude of peroneal nerve in diabetics with no symptoms of neuropathy.<sup>[17]</sup>

This study also revealed association of fasting blood sugar and post prandial blood sugar with NCS alteration. MDL was found positively correlated with blood sugar and CV were found negatively correlated with blood sugar in diabetics.

## CONCLUSION

This preliminary study has been conducted on a relatively small population. Our findings suggest usefulness of NCS in early detection of peripheral neuropathy. Each newly diagnosed diabetic subject should be screened for neuropathy by electrophysiological evaluation especially of the lower limb nerves. Nerve conduction studies should be included in the routine evaluation of diabetic patients which may improve the diagnostic yield.

Aggressive management of hyperglycemia is needed to prevent nerve damage.

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