



## Original Article

# Nerve Conduction Abnormalities in Newly Diagnosed Diabetes Mellitus Patients

Authors

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## Abstract

**Background:** *Peripheral neuropathy is one of the major microvascular complications of diabetes mellitus. Diabetic peripheral neuropathy is defined as presence of symptoms and / or signs of peripheral nerve dysfunction in diabetic patients after exclusion of other causes. Though incidence of diabetic peripheral neuropathy correlates with duration of diabetes mellitus and degree of hyperglycemia some patients already have neuropathy at time of diagnosis itself.*

**Objectives:** *Assessment of nerve conduction abnormalities in patients with newly diagnosed diabetes mellitus.*

**Materials and Methods:** *Patients with newly diagnosed diabetes mellitus within one month duration were prospectively recruited. History of symptoms like tingling sensation, hyperaesthesia, burning sensation, history of weakness, gait abnormality and foot ulcer were noted. Complete clinical examination was done. Nerve conduction studies was performed in upper limb and lower limb of non-dominant hand side using a neuro space S1 machine.*

**Results:** *Twenty patients [12 men and 8 women] were included in this study with duration of diabetes mellitus <1 month. The mean age of the patients was found to be 58.76 ± 13.6 years. Among these 15 patients [75%] had abnormal nerve conduction studies and 5 patients [25%] were normal. Distal motor latencies were significantly higher in diabetics and also F-wave latencies were prolonged in both upper limb and lower limbs. Sensory conduction velocities decreased in diabetics compared to controls.*

**Conclusion:** *Patients with newly diagnosed diabetes mellitus have higher incidence of peripheral neuropathy. So performing nerve conduction studies is highly essential for early detection of diabetic peripheral neuropathy and proper management.*

**Keywords:** *NCS (Nerve conduction studies), Newly diagnosed diabetes mellitus, Diabetic peripheral neuropathy, F-waves., HbA1C-Glycosated haemoglobin level.*

## Introduction

Type 2 Diabetes mellitus (T2DM) is a chronic metabolic disorder manifested by defective insulin secretion or action and associated with hyperglycaemia.<sup>1</sup> The T2DM is markedly increasing in both developing and developed countries. The T2DM encompasses a period during which the disease is undiagnosed and the elapsed time before diagnosis is estimated to be about 10 years.<sup>2</sup> Hyperglycaemia imposes structural and functional impairment to various organs and leads to macrovascular and microvascular complications and in majority of the cases disease will be in undiagnosed stage. Mounting reports shows that in maximum of newly diagnosed T2DM subjects, there exists a chronic diabetic complications.<sup>3-5</sup>

Diabetic Peripheral neuropathy (DPN) is one of the major microvascular diabetic complications and elicits significant morbidity and mortality among the diabetic patients. Wide range of studies shows the prevalence of DPN which ranges from 8-59%.<sup>6,7</sup> There are various clinical scores such as diabetic neuropathy symptom score, diabetic neuropathy examination, and neuropathy disability scores for the assessment of DPN. However, nerve conduction velocity test remains the gold standard for the evaluation of DPN with high diagnostic accuracy.<sup>8</sup> The usually done nerve conduction studies (NCS) encompasses the assessment of motor functions of the median, ulnar, perineal and tibial nerves, and sensory function of median, ulnar and sural nerves in terms of onset latency, amplitude, and conduction velocity. The another motor response is the F waves elicited as result of the activation of antidromic of motor neurons as a result of peripheral stimulation of motor axons.<sup>9</sup> Previous study shows that evaluation of upper limb sensory conduction is highly sensitive in detecting DPN in newly diagnosed T2DM patients.<sup>10</sup> In another study, the motor conduction velocities are significantly impaired in newly diagnosed diabetics as compared to the sensory nerve conduction.<sup>11</sup> In this backdrop, the present

prospective study was carried out to evaluate the nerve conduction deficits in newly diagnosed T2DM patients.

## Materials and Methods

This was a prospective study conducted during the period from 2019 to 2020 among the 20 newly diagnosed T2DM patients attending the General Medicine, outpatient department of Rajah Muthiah Medical College and Hospital. The 20 healthy non diabetic patients were taken as control. The diabetes was diagnosed based on the criteria entitled in American Diabetes Association and the patients were recruited within one month after diagnosis after obtaining informed consent.

### Inclusion Criteria

1. Newly diagnosed type 2 DM <1month
2. Age more than 25 years
3. Non -diabetic patients without known musculoskeletal disorders

### Exclusion Criteria

1. Known diabetic patients more than one month
2. Chronic alcoholics
3. Patients With acute or chronic musculoskeletal Disorder
4. Patients on drugs which may improve mask or aggravate the normal course of neuropathy (pyridoxine NSAIDs, Metronidazole, hydralazine isoniazid)

## Nerve Conduction Studies

All the patients were underwent complete neurological examination and basic investigations like fasting blood sugar, post-prandial blood sugar, random blood sugar, HbA1C done including BMI. Nerve conduction studies were performed using Nihon Kohden MEB-9400 system at optimal temperature. A standard nerve conduction study is performed using two surface electrodes consisting of an active electrode and reference electrode. The active electrode is placed over the muscle belly at the end plate region and the reference electrode is placed more distally over the tendon. Stimulation is initiated at 0mA and slowly increased (upto 50mA) until a

maximal response is obtained. The resultant biphasic waveform is the compound muscle action potential (CMAP). The CMAP represents the summated muscle fiber action potentials within the muscle being recorded. The main CMAP parameters that are used for analysis include the amplitude, latency and conduction velocity. CMAP amplitude (millivolts) reflects the number of motor nerve fibers responding to the stimulus. The latency (milliseconds) is the time from the moment of stimulation to the onset or “takeoff” of the negative phase of the CMAP. The latency from stimulation at the distal site, known as the distal or onset latency, is reported in routine nerve conduction studies. The proximal latency is used for calculating conduction velocity. The conduction velocity (meters per second) is the speed of the fastest conducting nerve fibers.

Conduction velocity is then calculated in m/s by the formula-distance (d)/difference in latent periods (t).

For a sensory NCS, both stimulation and recording occur along the nerve. Stimulation intensity is gradually increased until there has been supramaximal stimulation (usually only 30-40mA is required). The resultant waveform is the sensory nerve action potential (SNAP), a biphasic or triphasic waveform that represents summated nerve action potential. The two primary SNAP measurements are the amplitude and peak latency. The amplitude (microvolts) reflects the number of sensory nerve fiber action potentials. The peak latency (milliseconds) is the time from the moment of nerve stimulation to the negative peak of the response. A common protocol for routine

sensory NCSs in the upper extremity consists of median, ulnar and superficial radial sensory stimulation. In the lower extremity, the sural and superficial peroneal sensory nerve are stimulated.

### Statistical Analysis

Data were recorded in Microsoft Excel and analysed using SPSS v 20. The parametric variables between newly diagnosed patients and controls were analysed using Independent sample t –test. P-value <0.05 was considered as statistically significant.

### Results

In this study out of 20 newly diagnosed diabetic patients, 12 were males and 8 were females. The mean age of the patients was found to be  $58.76 \pm 13.65$  years. The time range between the diagnosis of diabetes and inclusion to the study was 1- 25 days with a median of 5 days.

There was a significant elevation of FBS and PPBS in DPN patients as compared to the controls (FBS:  $224.20 \pm 40.12$  vs  $156.65 \pm 38.56$ ;  $p=0.004$ ; PPBS:  $325.65 \pm 45.76$  vs.  $257.29 \pm 34.56$ ;  $p<0.001$ ). The mean HbA1C level was significantly higher in DPN patients as compared to the controls ( $11.56 \pm 2.76$  vs  $5.23 \pm 1.23$  %;  $p=0.006$ ).

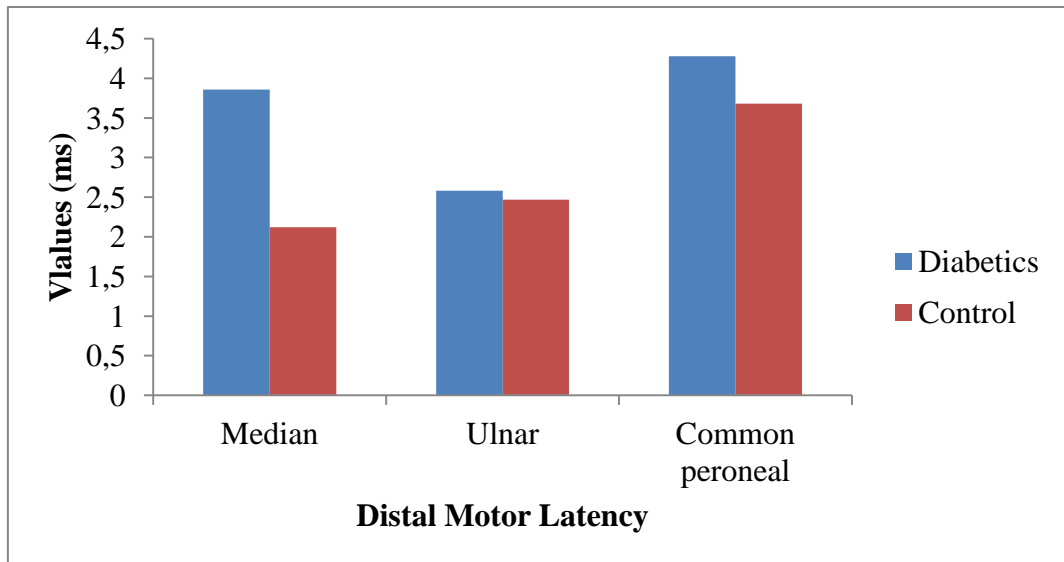
In newly diagnosed T2DM patients, the nerve conduction studies were normal in 5 patients (25%) and abnormal in 15 patients (75%).

In the present study, distal motor latencies of the median and the common peroneal nerves were significantly ( $P<0.05$ ) higher in diabetic patients as compared to the controls (Table 1).

**Table 1:** Distal motor latency among the diabetics and controls

Distal motor latency (ms)	Diabetics (n=20)	Control(n=20)	P –value
Median	$3.86 \pm 0.76$	$2.12 \pm 0.31$	0.001
Ulnar	$2.58 \pm 0.33$	$2.47 \pm 0.27$	0.32 <sup>NS</sup>
Common peroneal	$4.28 \pm 0.82$	$3.68 \pm 0.53$	0.001

P< 0.05- Significant; NS-Non Significant



**Fig 1:** Distal motor latency among the diabetics and controls

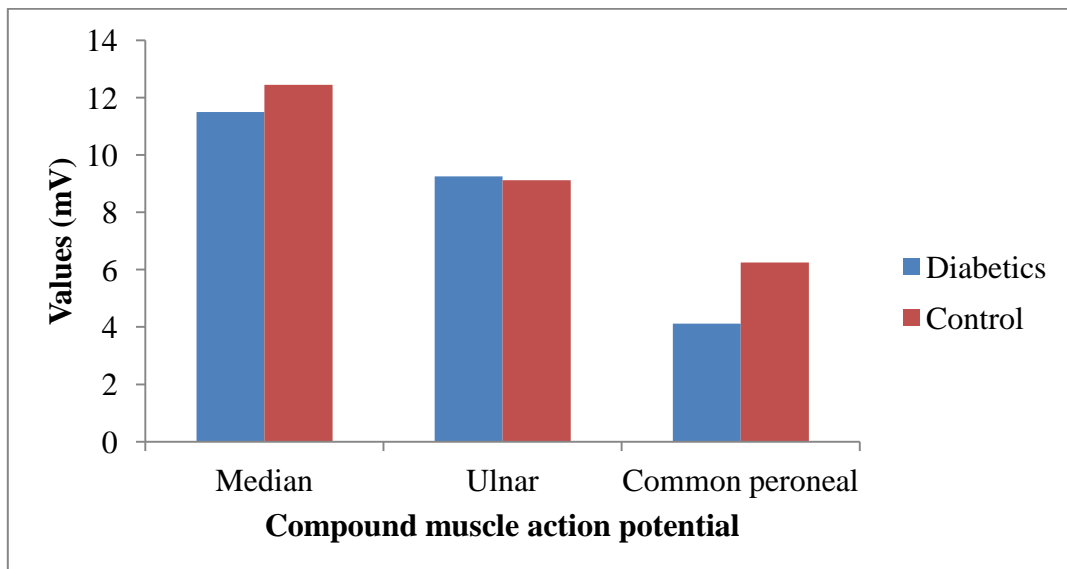
Further, Compound muscle action potential (CMAP) amplitude of the common peroneal nerve was significantly ( $p < 0.05$ ) reduced as compared to controls. However, there was no significant

alteration of CMAP amplitude in Median and Ulnar nerves among the diabetics and controls (Table 2).

**Table 2:** Compound muscle action potential among the diabetics and controls

Compound muscle action potential (mV)	Diabetics (n=20)	Control (n=20)	P –value
Median	11.50±3.21	12.45±3.76	0.65 <sup>NS</sup>
Ulnar	9.25±1.25	9.12±1.12	0.74 <sup>NS</sup>
Common peroneal	4.12±1.56	6.25±2.34	0.03

P < 0.05- Significant; NS-Non Significant



**Fig 2:** Compound muscle action potential among the Diabetics and controls

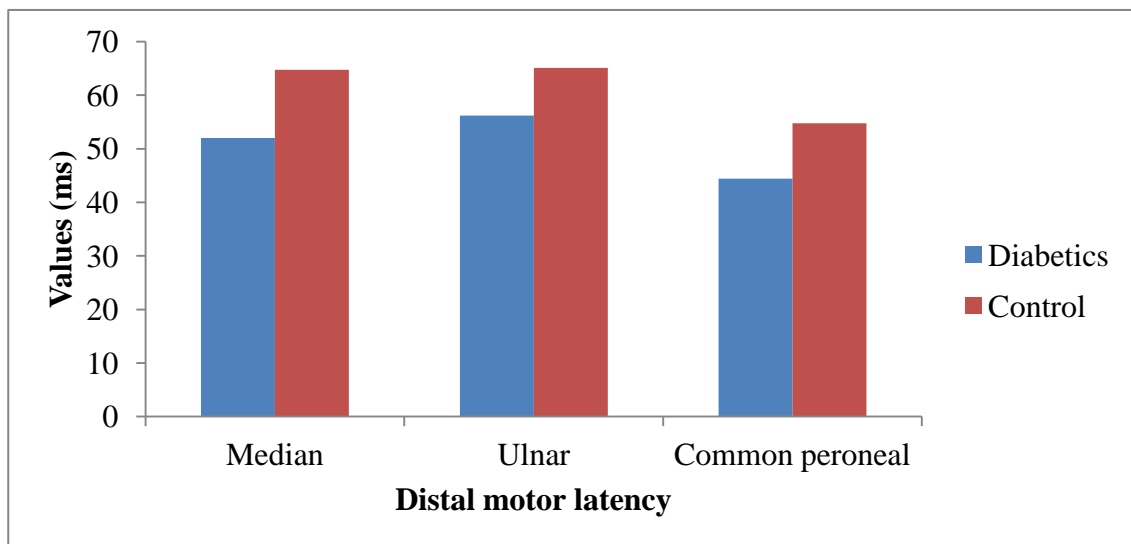
The median, ulnar, and common peroneal nerves motor conduction velocities were significantly

( $P < 0.05$ ) decreased in diabetics as compared to the controls (Table 3).

**Table 3:** Motor conduction velocities among the diabetics and controls

Motor conduction velocity (m/s)	Diabetics (n=20)	Control (n=20)	P –value
Median	52.02±5.45	64.75±5.25	0.000
Ulnar	56.21±4.12	65.12±8.55	0.000
Common peroneal	44.45±5.25	54.76±4.28	0.000

P< 0.05- Significant; NS-Non Significant



**Fig 3:** Motor conduction velocities among the diabetics and controls

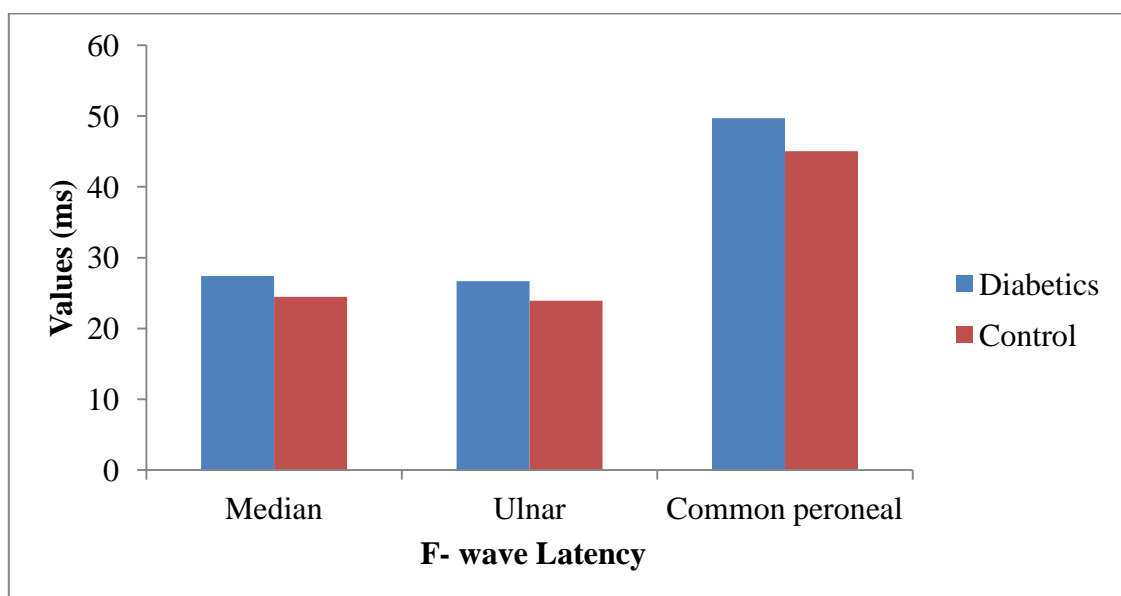
The F- wave latencies of median, ulnar, and common peroneal nerves were significantly

(p<0.05) prolonged in diabetics as compared to controls.

**Table 4:** F- wave Latency among the diabetics and controls

F wave latency (ms)	Diabetics (n=20)	Control (n=20)	P –value
Median	27.42±3.22	24.46±1.82	0.000
Ulnar	26.68±3.58	23.94±1.78	0.000
Common peroneal	49.68±6.86	45.03±4.84	0.000

P< 0.05- Significant; NS-Non Significant



**Fig 4:** F- wave Latency among the diabetics and controls

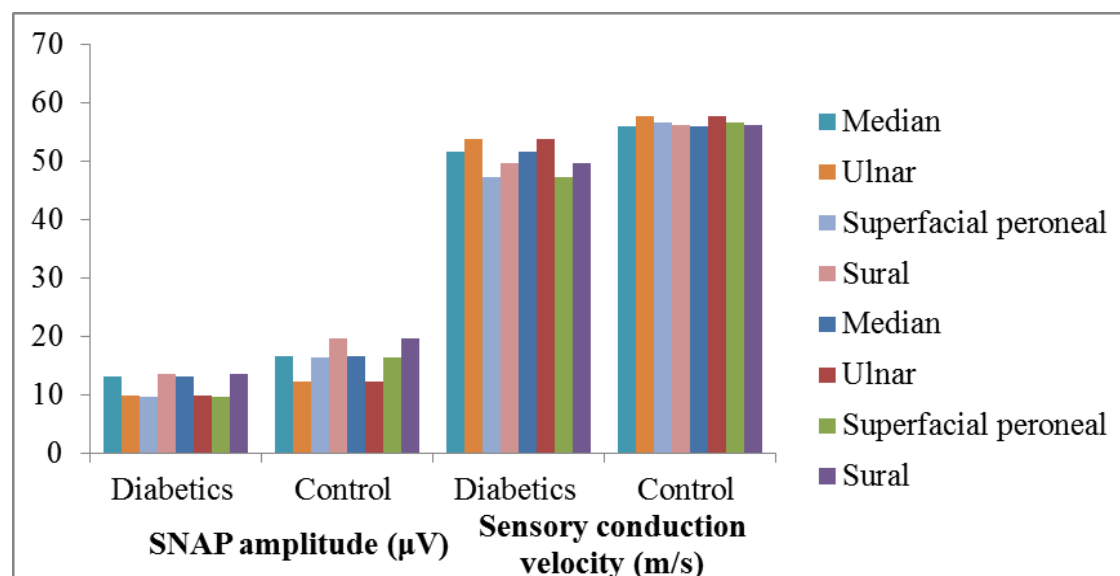
In our study, the sensory conduction velocities in median, superficial peroneal and sural nerves of diabetic patients were significantly ( $p < 0.05$ ) decreased as compared to the controls. Sensory

nerve action potential (SNAP) amplitudes of superficial peroneal and sural nerves were significantly ( $p < 0.05$ ) decreased in diabetics as compared to controls.

**Table 5:** SNAP amplitude and Sensory conduction velocity among the diabetics and controls

SNAP amplitude ( $\mu V$ )	Median	Ulnar	Superficial peroneal	Sural
Diabetics (n=20)	13.22 $\pm$ 7.82	9.84 $\pm$ 5.02	9.58 $\pm$ 5.38	13.48 $\pm$ 8.08
Control (n=20)	16.54 $\pm$ 5.76	12.26 $\pm$ 4.18	16.42 $\pm$ 9.52	19.65 $\pm$ 7.52
P –value	NS	NS	0.000	0.000
Sensory conduction velocity (m/s)	Median	Ulnar	Superficial peroneal	Sural
Diabetics (n=20)	51.48 $\pm$ 6.38	53.78 $\pm$ 6.38	47.28 $\pm$ 5.58	49.58 $\pm$ 6.42
Control (n=20)	55.88 $\pm$ 4.58	57.66 $\pm$ 6.48	56.64 $\pm$ 9.68	56.12 $\pm$ 8.18
P –value	0.000	NS	0.000	0.000

P < 0.05- Significant; NS-Non Significant



**Fig 5:** SNAP amplitude and Sensory conduction velocity among the diabetics and controls

**Discussion**

In newly diagnosed diabetic patients there has been a substantial evidence of symptomatic peripheral neuropathy as result of impairment in autonomic nerve function detected by nerve conduction studies.<sup>12</sup> The etiology of neuropathy during diabetes is as a result of enhanced vascular resistance and decreased blood. Further, reduced nerve myoinositol content activation of polyol pathway is also a noxious factor in the progression of DPN.<sup>13</sup>

In the present study, there has been a significant prolongation of distal motor latencies in diabetic subjects as that of the normal patients. Similar to our study, Rota et al. reported the NCS

impairment of distal median motor neuropathy in 42% of newly diagnosed diabetic patients.<sup>10</sup>

Previous report shows that decrease in motor conduction velocity and amplitude of SNAP in one of the early clinical manifestation of diabetic neuropathy in newly diagnosed subjects. These events may prelude to prolongation in the sensory latencies, decrease of sensory velocity, finally reduction of amplitudes of CMAP.<sup>9</sup> Similarly in our study, SNAP amplitude and sensory conduction velocity are significantly reduced in superficial peroneal and sural nerves. In addition we observed significant reduction of amplitudes of CMAP in common peroneal which is similar to the finding of de Souza et al.<sup>9</sup>

F-waves evaluates the excitability of complete length of motor units. Inclusion of F-waves latencies in the NCS improves sensitivity in the range between 3-36% in the detection of nerve conduction abnormalities in newly diagnosed diabetic patients.<sup>14</sup> Similarly in our study F-wave latencies was significantly prolonged in newly diagnosed T2DM patients as compared to the controls.

### Conclusions

Nerve conduction abnormalities is the most frequently observed clinical entity in newly diagnosed diabetic patients. Thus performing nerve conduction studies is highly essential in these patients for the early detection of DPN and proper management.

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**Conflict of interest:** Nil

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