Study Of Nerve Conduction Velocity In Type II Diabetes Mellitus

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Abstract: Background& Objectives: Diabetic neuropathy is the most common and troublesome complication of diabetes mellitus, leading to great morbidity and resulting in burden for diabetes care. The progression of neuropathy can be reduced by early detection and intervention. Nerve conduction studies are the most sensitive indices of the severity of neuropathy. These tests can be used to localize lesions and describe the type and severity of the pathophysiologic process, including alterations that are not recognized clinically. This study was undertaken to compare nerve conduction study results in diabetes mellitus patients with good glycemic control and poor glycemic control and to compare it with non-diabetic subjects. This study aims to signify the role of nerve conduction study in diabetes mellitus. This can help in identifying the asymptomatic stage of diabetic neuropathy so that suitable preventive measures can be taken. Methodology: Total 90 subjects were included in the study group. 30 were non diabetic subjects and 60 were known cases of Type II diabetes mellitus patients attending diabetic OPD at GMCH, Aurangabad of age 30-50 years with duration of 5-10 years. Glycated haemoglobin levels were estimated and on this basis the cases were divided into two groups; diabetic patients with good glycemic control and diabetic patients with poor glycemic control. Nerve Conduction parameters were measured by computerized micromed RMSEMG system. Results: There was an increase in mean latency and decrease in amplitude and velocity values in both the diabetic groups. Intergroup comparison showed that, the increase in latency and decrease in amplitude and velocity was more in diabetics with poor glycemic control as compared to other study groups and this difference was statistically highly significant. Conclusion: The study concluded that there is statistically significant changes in nerve conduction parameters in Type II diabetes. [Yadav N NJIRM 2015; 6(4): 36-43]

Key Words: Diabetes Mellitus, Glycated haemoglobin, Nerve Conduction Velocity.

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Introduction: Diabetes mellitus is a heterogeneous group of metabolic disorders characterized by hyperglycemia with disturbances of carbohydrate, fat and protein metabolism caused by either lack of insulin secretion or decreased sensitivity of the tissues to insulin.1, 2

Incidence of both Type I and Type II diabetes are rising. Currently, the number of cases of diabetes worldwide is estimated to be around 347 million. Of these >90% are Type II diabetics.[3]In India, number of diabetics is increasing fast and if this rise continues, according to global survey, India will surely become the capital of diabetes.[4]

It is associated with obesity, unsatisfactory diet, sedentary life style and increased urbanization. It is often discovered by chance. It is typically gradual in onset and affecting mainly middle aged and elderly people.[3]Without proper treatment, diabetics have a higher risk of developing chronic microvascular complications, including diabetic nephropathy, neuropathy, retinopathy and macrovascular complications, like coronary artery disease, peripheral arterial disease and stroke, which lead to significant morbidity and mortality.[5]

Among these, diabetic neuropathy is one of the most common and troublesome complication accounting for 28% of all complications in diabetes.[6]It usually results from microvascular injury involving small blood vessels that supply nerves (vasa nervorum) but macrovascular pathogenesis is also involved.[7]

50% of patients present with symptoms like sensation of numbness, tingling, sharpness or burning pain The symptoms tend to have a "glove and stocking" distribution.[8] Pain typically involves the lower extremities. As diabetic neuropathy progresses, the pain subsides and eventually disappears, but sensory deficit in lower extremities persists.[1]

It is a progressive process that has a long asymptomatic stage.[6]So, it is important to identify neuropathy in asymptomatic stage as diabetics are known to be at high risk of foot complications like foot ulceration, infections which may require amputation. As up to half of the patients may be

asymptomatic, so the diagnosis should be made with a careful clinical examination of the lower limbs and absence of symptoms should never be assumed to indicate an absence of signs.[9]Early identification and glycemic control are the key factors for preventing diabetic neuropathy. [6] The American Diabetes Association and the American Academy of Neurology recommend that, at least one parameter from each of the following five categories be measured to establish the presence of diabetic neuropathy: symptom profile, neurologic examination, nerve conduction studies, quantitative sensory testing (QST) and quantitative autonomic function testing (QAFT).[10]So, electrophysiological studies help us to detect abnormalities in diabetic patients that may not be clinically apparent.[11]

Therefore, this study was undertaken to assess the nerve conduction study results among known cases of Diabetes Mellitus with good glycemic control, diabetics with poor glycemic control and to compare it with non-diabetic subjects. This study aims to signify the role of nerve conduction study in diabetes mellitus. This can help in identifying the asymptomatic stage of diabetic neuropathy so that suitable preventive measures can be taken.

Material and Methods: This study was undertaken in the Department of Physiology in collaboration with Department of Biochemistry and Department of Medicine, Government medical college and hospital.

A total number of 90 subjects were included in the study group. Of them, patients of 30-50 years of age with a history of diabetes since 5-10 years attending the diabetic OPD were selected. Glycated haemoglobin levels were estimated and on this basis subjects were divided into diabetics with good glycemic control (Group 1) and diabetics with poor glycemic control (Group 2). The healthy controls of same age group were selected from relatives of patients and staff members.

Study Groups:

Group 1: 30 Type II diabetes mellitus patients with good glycemic control, of both sexes, of age group 30-50 years.

Group 2: 30 Type II diabetes mellitus patients with poor glycemic control, of both sexes, of age group 30- 50 years.

Group 3: 30 Non-diabetic healthy subjects, of both sexes, of age group 30- 50 years as a control group.

No one had any medical condition to be associated with polyneuropathy, unusual dietary habits, family H/O peripheral nerve disease and consumed alcohol or drugs with potential neurotoxic effects. Patients with Type I DM, Type II DM patients on insulin therapy were excluded.

A consent was obtained from each participant and procedure explained in their mother tongue. Ethical permission was taken from institutional ethical committee.

Computerized micromed RMSEMG System was used for electrophysiological analysis using surface electrodes. In present study, 3 parameters Nerve Conduction Velocity, amplitude, latency of sural nerve and median nerve (motor and sensory component) were recorded[12] (Table no. 1).

Table 1: Standard Nerve Conduction Values: [12]

Nerve		Latency (ms)	Amplitude	NCV(m/s)
Median nerve(sensory)		3.06 ± 0.41	8.91 ± 4.48(μV)	45.45 ± 9.40
Median nerve(motor)	Wrist	3.77 ± 0.40	8.10 ± 2.62(mV)	58.52 ± 3.76
	Elbow	7.62 ± 0.65	7.84±2.25(mV)	
Sural nerve		2.56 ±0.61	18.0 ± 10.5(μV)	50.9 ± 5.4

μV- Microvolt, mV- Millivolt, m/s- meter / second

A venous blood was collected for biochemical investigation HbA1c. Quantitative estimation was done by ion exchange resin using commercial kits from ERBA diagnostics[13] (Table no.2).

Table 2: Glycated haemoglobin levels in study groups[13]

Groups	HbA1c (%)
Group1-Diabetic; But; Good glycemic Control	5.5-6.8
Group 2-Poor glycemic Control	≥ 7.6
Group 3-Non- Diabetic subjects	4.2 - 6.2

HbA1c- Glycated Haemoglobin

Statistical analysis

The results were analyzed using Microsoft excel 2007. The data was analysed and compared by using unpaired student't' test. The values were expressed as mean ±SD. A p value of <0.05 was considered to be statistically significant. A p value of < 0.001 was considered to be statistically highly significant.

Results:

1. The mean values of glycated hemoglobin levels (%) in Group 1, Group 2 and Group 3 were 5.80 ± 0.25, 8.17 ± 0.54, 5.57 ± 0.45 respectively. The increase in glycated hemoglobin in Group 2 was statistically highly significant (p < 0.0001). (Table no. 3 and 4)

Table 3: Baseline characteristics and mean value of diabetic duration and glycemic status of the Study Groups

Parameters	Groups		
	Group 1 n = 30 Mean ± SD	Group 2 n = 30 Mean ± SD	Group 3 n = 30 Mean ± SD
Age (years)	47.73 ± 2.38	47.77 ± 1.78	46.33 ± 3.90
Height (cm)	170.03 ± 4.19	168.10 ± 4.50	169.0 ± 3.69
Weight (kg)	66.37 ± 5.59	66.03 ± 7.23	68.10 ± 6.63
BMI (kg/m2)	22.94 ± 1.59	23.33 ± 1.96	23.81 ± 1.80
Diabetic duration(years)	6.10±0.87	6.63±1.31	Not Applicable
HbA1c (%)	5.80±0.25	8.17±0.54	5.57±0.45

Table 4: Test of significance to show differences in glycemic status of the Study Groups by Unpaired't' test

Parameters	'p' value		
	G1VsG2	G2 VsG3	G1VsG3
HbA1c (%)	< 0.0001(HS)	< 0.0001(HS)	0.06 (NS)

Group 1- Diabetics with good glycemic control, Group 2-Diabetics with poor glycemic control, Group 3- Non diabetic controls, HbA1c- Glycated hemoglobin, NS- Not significant, HS- Highly Significant

2. The mean values of latency(ms), amplitude(mV) and velocity (m/s) of median (motor) nerve in Group 1 was 4.04 ± 0.60, 8.32 ± 2.46 and 54.13 ± 8.74 ,while in Group 2 was 4.58 ± 0.54, 6.29 ± 3.49 and 47.75 ± 9.08 and in Group 3 was 3.76 ± 0.43, 9.96 ± 2.58 and 59.69 ± 4.93 respectively.

The increase in latency and decrease in amplitude and velocity in Group 2 was statistically highly significant (p< 0.0001). (Table no. 5 and 6 and Graph no.1, 3, 4)

Table 5: Mean value of median (motor) nerve , median(sensory)nerve, sural nerve parameters of Study Groups

Nerves	**Parameters**	**Groups**		
		Group 1 n = 30 (Mean ± SD)	**Group 2 n = 30 (Mean ± SD)**	**Group 3 n = 30 (Mean ± SD)**
Median (Motor) nerve	Latency (ms)	4.04 ± 0.60	4.58 ± 0.54	3.76 ± 0.43
	Amplitude(mV)	8.32 ± 2.46	6.29 ± 3.49	9.96 ± 2.58
	Velocity (m/s)	54.13 ± 8.74	47.75 ± 9.08	59.69 ± 4.93
Median (sensory) nerve	Latency (ms)	3.38± 0.55	3.77±0.36	3.06±0.39
	Amplitude (µV)	11.69±3.64	8.34±3.72	15.18±4.16
	Velocity (m/s)	48.83±8.64	43.37±4.13	53.6±7.43
Sural nerve	Latency (ms)	3.34±0.44	3.64±0.39	3.07±0.23
	Amplitude (µV)	7.79±1.67	6.64±0.99	9.03±1.24
	Velocity (m/s)	45.58±3.62	42.83±3.65	49.05±4.18

Group 1- Diabetics with good glycemic control, Group 2-Diabetics with poor glycemic control, Group 3- Non diabetic controls, n - Number of subjects**,**ms - Millisecond , mV- Millivolt, µV= Microvolt , m/s-meter per second, SD - Standard Deviation

Table 6: Test of significance to show differences in median (motor) nerve, median (sensory) nerve, Sural nerve parameters of Study Groups by Unpaired 't' test.

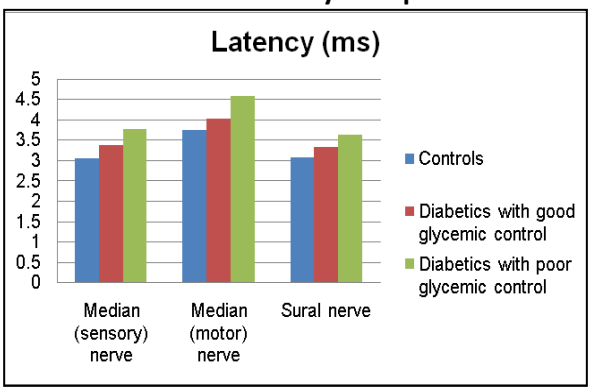
Nerves	Para-meters	'p' value		
		G1VsG2	G2 VsG3	G1VsG3
Median(motor) nerve	Latency (ms)	0.0006(HS)	<0.0001(HS)	0.04(S)
	Amplitude (µV)	0.01(S)	<0.0001(HS)	0.01(S)
	Velocity (m/s)	0.007(HS)	<0.0001(HS)	0.003(HS)
Median (sensory) nerve	Latency (ms)	0.001(HS)	<0.0001(HS)	0.01(S)
	Amplitude (µV)	0.0008(HS)	<0.0001(HS)	0.001(HS)
	Velocity (m/s)	0.002(HS)	<0.0001(HS)	0.02(S)
Sural nerve	Latency (ms)	0.006 (HS)	<0.0001 (HS)	0.005 (HS)
	Amplitude (µV)	0.002 (HS)	<0.0001 (HS)	0.001 (HS)
	Velocity (m/s)	0.004 (HS)	<0.0001 (HS)	0.001 (HS)

Group 1- Diabetics with good glycemic control, Group 2-Diabetics with poor glycemic control, Group 3- Non diabetic controls, ms - Millisecond, µV-Microvolt, m/s - meter per second, HS - Highly Significant.

3. The mean values of latency(ms), amplitude(µV) and velocity(m/s) of median(sensory) nerve in Group 1 was 3.38 ± 0.55, 11.69 ± 3.64 and 48.83 ± 8.64 while in Group 2 was 3.77 ± 0.36, 8.34 ± 3.72 and 43.37 ± 4.13 and in Group 3 was 3.06 ± 0.39, 15.18 ± 4.16 and 53.60 ± 7.43 respectively. The increase in latency and decrease in amplitude and velocity in Group 2 was statistically highly significant (p< 0.0001). (Table no. 5 and 6 and Graph no.1, 2, 4)

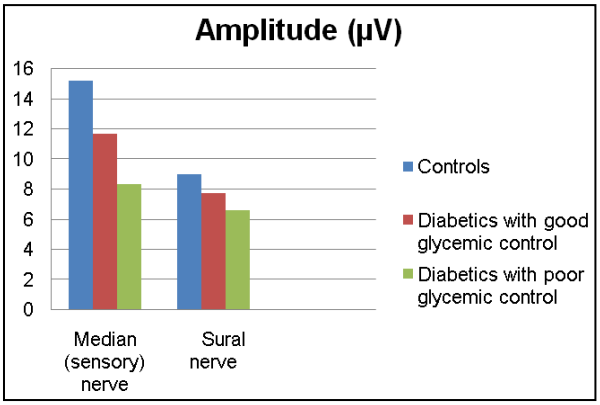
4. The mean values of latency(ms), amplitude(µV)and velocity(m/s) of sural nerve in Group 1 was 3.34 ± 0.44, 7.79 ± 1.67 and 45.58 ± 3.62, while in Group 2 was 3.64 ± 0.39, 6.64 ± 0.99 and 42.83 ± 3.65 and in Group 3 was 3.07 ± 0.23, 9.03 ± 1.24 and 49.05 ± 4.18 respectively. The increase in latency and decrease in amplitude and velocity in Group 2 was statistically highly significant (p < 0.0001). (Table no.5 and 6 and Graph no.1, 2, 4) The study showed both sensory and motor neuropathy in diabetes patients which was more pronounced in diabetics with poor glycemic control than good glycemic control.

Graph 1: Showing mean latency of median (sensory and motor component) nerve and sural nerve in Study Groups



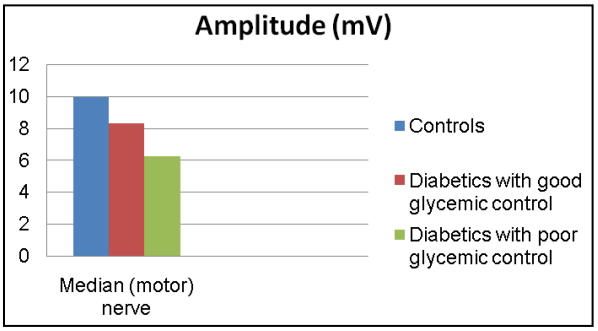
ms-millisecond

Graph 2: Showing mean amplitude of median (sensory) nerve and sural nerve in Study Groups



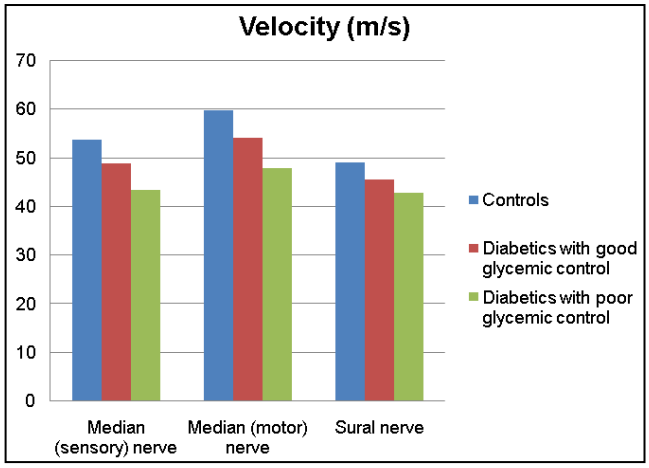
µV- Microvolt

Graph 3: Showing mean amplitude of median (motor) nerve in Study Groups



mV- Millivolt

Graph 4: Showing mean velocity of median (sensory and motor) nerve and sural nerve in Study Groups



m/s- meter per second

Discussion: The present study shows the effect of diabetes of duration 5-10 years on nerve conduction values in 60 diabetic patients and compared with 30 non- diabetic controls of both sexes in the age group 30-50years.

In the present study, nerve conduction parameters of both motor and sensory nerves were significantly changed. There was an increase in mean latency and decrease in amplitude and velocity values in both the diabetic groups. Intergroup comparison showed that, the increase in latency and decrease in amplitude and velocity was more in diabetics with poor glycemic control as compared to other study groups and this difference was statistically highly significant.

When diabetics with good glycemic control were compared with non-diabetic subjects, it was found that the diabetic group with good glycemic control still had increased latency and decreased amplitude and velocity when compared with non-diabetic subjects. This difference was found to be statistically significant.

This shows that in our study, even with good glycemic control, the nerve conduction parameters are affected.

The main responsible cause for changing conduction of impulse in nerve is degree of hyperglycemic hypoxia.[14]Dysfunction of ion conductance, especially voltage gated ion channels could contribute to abnormalities in the generation and conduction of action potential.[15] In hyperglycemic environment, oxidative stress leads to endothelial dysfunction and decreased capillary blood flow which in turn leads to endoneurial hypoxia causing death of nerve cells and so nerve conduction parameters are altered.[16]

Pathological changes in peripheral neuropathy due to diabetes mellitus are characterized by segmental demyelination, axonal degeneration, or a combination of the two.[9, 17]

Nerve conduction reflects several physiological components of peripheral nerve function including nerve size, degree of myelination, internodal distance, diameter of axons and nerve temperature.[10]

Nerve conduction velocity reflects integrity of the myelin sheath and indicates transmission time in the large myelinated nerve fibers.[10] Amplitude reflects the size and number of nerve fibers, and it's measurement is important for the evaluation of neuropathy.Both latency and conduction velocity depend on an intact, myelinated nerve as myelin and the saltatory conduction are essential for fast action potential propagation in normal subjects. In contrast, the amplitude of the waveform depends primarily on number of axons functioning within the nerve.[18,19]

Slowing of conduction velocity or prolongation of latency usually implies demyelinating injury, while loss of amplitude usually correlates with axonal loss or dysfunction.[8]

Abdulsalam A and Chopra et al [18]suggested that the early diabetic effects on the peripheral nerve are mainly demyelinating and confirmed the view of Bischoff that a metabolic Schwann cell lesion is the primary defect in diabetic neuropathy.

Bansal et al.[8]suggested that the slowing of nerve conduction velocity indicates the ongoing damage to the myelin sheaths and concluded that nerve conduction velocity is gradually diminished in diabetic neuropathy, with estimates of a loss of about 0.5 meter/second/year and they were also of opinion that the amplitude decreases with the

rising glycosylated haemoglobin levels, thus suggesting the onset of axonopathy.

Parveenkumar, Michael clark et al [19]showed that in the presence of hyperglycemia there is increase in osmolarity due to accumulation of sorbitol and fructose lead to schwann cell damage.In a study done by Clayton et al, .[20]it was shown that, the chemical conversion of glucose results in a depletion of nicotinamide adenine dinucleotide phosphate stores; which are important for detoxification of reactive oxygen species, and for the synthesis of the vasodilator nitric oxide. Nitric oxide plays an important role in controlling Na^+/K^+ ATPase activity, a diminution of which has been implicated in the pathogenesis of peripheral neuropathy.

Clayton W, Elasy TA [20]showed that the accumulation of sugar products results in a decrease in the synthesis of nerve cell myoinositol, which inhibits tissue Na^+/K^+ ATPase activity, required for normal neuron conduction. Decrease in activity of Na^+/K^+ ATPase pump results in Na^+ retention, edema, myelin swelling, axoglial disjunction and nerve degeneration; further contributing to the neuropathy.

Tavakoli M, Mojaddidi M, Fadavi H.[7,14] showed that blood vessels depend on normal nerve function, and nerves depend on adequate blood flow. The first pathological change in the microvasculature is vasoconstriction; this impairs supply of nutrients and oxygen. Malik R.A[21]explained that endothelial cell hyperplasia and platelet plugging are responsible for occlusive phenomenon in the pathogenesis of neuropathy.

Tavakoli et al,[14]in their study suggested that even short term hyperglycemia may be sufficient to induce oxidative stress and nerve cell dysfunction/ death leading to neuropathy. Neuropathy in diabetic patients is manifested in the motor, autonomic and sensory components of the nervous system.

Parveenkumar, Michael clark.[19]explained that elevated intracellular levels of glucose cause a non-enzymatic covalent bonding with proteins like hemoglobin, collagen, low density lipoprotein and tubulin in peripheral nerves .This leads to accumulation of advanced glycated end-products causing injury and inflammation via stimulation of factors like complement, cytokines,etc.

Other potential mechanisms through which glucose could impair cell function include inappropriate activation of protein kinase C, activation of cytokines (angiotensin II, endothelin), growth factor stimulation (transformation growth factor –β, vascular endothelium growth factor) and depletion of basement membrane glycosaminoglycans. Each of these factors plays a variable and interrelated role in development and progression of microvascular complications in diabetes.[22]

Thus, all these mechanisms explain the reasons for alteration in nerve conduction parameters in diabetics with poor glycemic control.

However, even with good glycemic control, it was found that there is affection of nerve conduction parameters even though the risk is reduced due to good glycemic control.

In these diabetics, the risk of diabetic complications was strongly associated with previous hyperglycemia. Any reduction in HbA1c is likely to reduce the risk of complications, with the lowest risk being in those with HbA1c values less than 6%.[23] However, the adjustment for HbA1c does not completely exclude a potential glycemia-mediated effect because of other factors, such as, glucose variability and the timing of changes in glucose level could have also played a role.[24]

In Type II diabetic patients, decreased nerve conduction velocity is probably one of the earliest neuropathic abnormalities and is often present even at diagnosis. Thereafter slowing of nerve conduction velocity generally progresses at a steady rate of approximately 1 meter/second/year.[25]

In a study done by Baba M et al,.[26] it was shown that, once polyneuropathy establishes, it become irreversible and finally leads to disability. This irreversible sensory deficit may be due to an increase in somatosensory central conduction time

between spinal cord entry time and the arrival time to sensory cortex.

Thrainsdottir et al.[27]demonstrated that increase in basal membrane thickening and reduction in capillary luminal area was associated with sensory peripheral neuropathy in impaired glucose tolerance and diabetic patients.This may be one of the reasons for slower velocities in diabetic patients in our study.

The results observed in our study further establish the importance of electrodiagnostic assessment in patients with diabetes mellitus. Early identification of diabetic polyneuropathy in these patients may encourage further intensification of glycemic control and more aggressive treatment in early diabetes, both of which may have clinical benefit. With all these evidences, we can now suggest to include the nerve conduction studies in the primary care settings for patients with diabetes.

An important limitation of nerve conduction study is that it only assesses large myelinated nerve fibers and overlooks possible small nerve fiber dysfunction.[10]

To search for small diameter nerve fiber dysfunction, measurement of intraepidermal nerve fiber (IENF) density and quantitative sensory testing (QST) should be done along with nerve conduction study.[28]

Conclusion: The present study showed that both, sensory and motor nerve conduction, are useful modality for detecting diabetic neuropathy in subclinical diabetics.

Nerve conduction studies are one of the important methods for diagnosis and evaluation of diabetic sensorimotor polyneuropathy especially for the subclinical neuropathies. Routine nerve conduction studies should be done in diabetics at least on yearly basis.

Our study also indicates that asymptomatic patients do occur; hence periodic screening should be carried out in diabetics to reduce long term complications like diabetic foot ulceration, etc.
The estimation of both, nerve conduction velocity and glycated hemoglobin levels in diabetics, is helpful in identifying the risk category for diabetic neuropathy which is one of the main causes for severe morbidity among the diabetes mellitus patients.

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