Phrenic nerve conduction in diabetic polyneuropathy

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We investigated phrenic nerve conduction in diabetic patients with neuropathic sympoms in order to assess the diagnostic utility of this nerve amongst other peripheral nerves in diabetic polyneuropathy. Forty diabetic patients and 30 normal individuals were studied. Their mean ages were 57.4±1.39 and 49.1 ±1.05 respectively. The patients were evaluated by using San Antonio Diabetic Conference's neurological examination and symptom scales. We recorded compound muscle action potentials of the diaphragm by placing surface electrodes over the 8th intercostal space bilaterally on the mid-axillary line. The reference electrodes were 2 cm behind active electrodes. We performed percutaneous stimulation by placing cathode over the posterior margin of the sternocleidomastoid muscle. Median motor and sensory, tibial and sural nerve conductions were also measured unilaterally. Mean phrenic nerve conduction time in patients was significantly increased compared with the control group. ($p<0.001$). Phrenic nerve latency abnormalities were seen in 85% of the patients. Side to side differences between phrenic nerve latencies were not significant. The differences between the percentages of abnormal findings of phrenic and other nerves tested were not statistically significant. In 3 patients phrenic neuropathy was the only abnormal finding. There were weak correlations between phrenic neuropathy and the duration of diabetes, mean symptom and neurological examination scores. We conclude that phrenic nerve latency measurement by using surface stimulation and recording technique is as useful as the measurements of peripheral nerves conduction velocities in the diagnosis of diabetic polyneuropathy. [Turk J Med Res 1995, 13(5): 184-186]

Key Words: Polyneuropathy, Diabetes mellitus, Phrenic nerve

One of the major complications of diabetes mellitus (DM) is peripheral neuropathy. This type of neuropathy is symmetrical, with a mild gradual onset and can manifest as sensory, motor symptoms or both. Neuropathy of a single cranial or peripheral nerve has also been observed. It may present an abrupt onset and involves only one nerve. The disease may affect any nerve, including the phrenic nerve. However, phrenic nerve conduction study is usually ignored in evaluating diabetic neuropathy. The aim of this study is to investigate phrenic nerve conduction in diabetic neuroptahy and compare the values with the peripheral nerves.

MATERIALS AND METDOHS

Patients: Forty patients were examined. They were referred to the EMG laboratory. Patients who had other causes for neuropathy were excluded. The patients had been hospitalized because of DM and had signs and symptoms suggesting neuropathy. Their mean age was 57.4±1.39, mean duration of DM was 9.77±1.02. The patients symptoms and neurological examination findings were scored according to- the neuropathy symptom score (NSS) and neurological disability scores (NDS) devised by Dyck et al (1). Peripheral nerves were examined unilaterally according to the protocol for electrodiagnostic tests recommended by the San Antonio Conference on Diabetic Neuropathy (2). Median nerve motor and sensory, posterior tibial and sural nerve conduction velocities (CV), F wave latencies, distal latencies and amplitudes of compound muscle and nerve action potentials were measured. Bipolar surface stimulating electrode (Dantec 13 K62) and surface recording electrodes (Dantec 13 L27) were used. For the electrical stimulation of the phrenic nerve each patient was placed in the supine position and a stimulus of 150-200 v. was administered at the posterior border of the upper margin of the thyroid cartilage at a rate of 1 Hz. The pulse duration was 0.2 msec. Bipolar surface electrodes

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were used for stimulation (Dantec 13 K62). The diaphragmatic muscle action potential was recorded bilaterally with silver cup electrodes placed 2 cm apart on the eight intercostal space with the anterior electrode on the anterior axillary line of the chest wall (3). A ground electrode was placed over the upper chest wall. Care was taken to see the contraction of the diaphragm and not to stimulate brachial plexus. At least three records were obtained.

Controls: Thirty patients with various diseases and had no symptoms, signs and neurological findings of neuropathy were examined. Their mean age was 49.1 ±1.05. Informed consent were obtained. Statistical analysis of the data was performed by Student's t test for the importance of difference between two percentages and Kendal rank correlation method.

RESULTS

Phrenic nerve conduction time in patients was significantly increased compared with the control group ($p<0.001$) (Figure 1). Phrenic nerve latency abnormalities were seen in 85% of the patients. Side to side differences between phrenic nerve latencies were not significant. This abnormality was bilateral in 30 patients and unilateral in 10 patients. The conduction velocities of the peripheral nerves are shown in Table 1. The differences between the percentages of abnormal findings of phrenic and other nerves tested were not statistically significant ($p>0.05$ (Figure 2). In 3 patients phrenic neuropathy was the only abnormal finding. There were no correlations between phrenic neuropathy and the duration of diabetes, mean symptom and neurological examination scores.

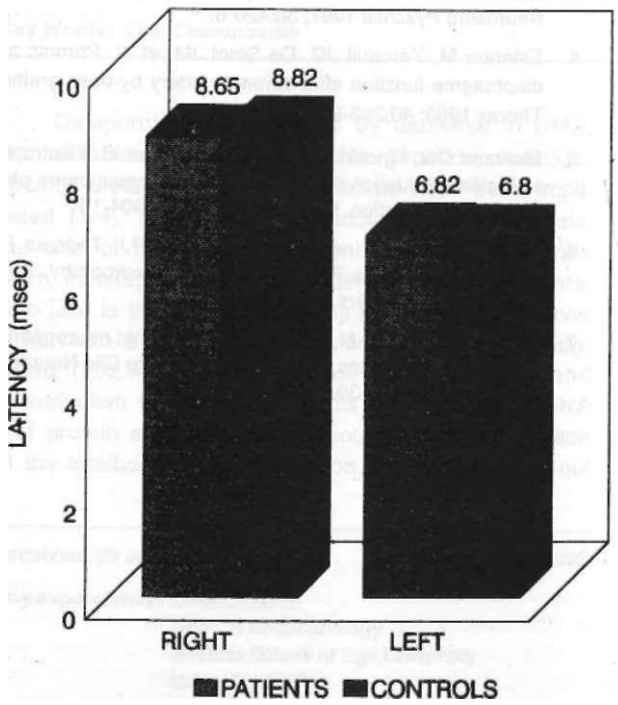

Figure 1. Phrenic nerve latency in diabetic patients and controls.

Table 1. The results of phrenic nerve latency and peripheral nerve CV's

Nerves	Controls (n=30)	Patients (n=40)
Phrenic/right latency Mean+SD (m/sec)	6.82+0.14	8.65+0.21*
Median motor CV Mean±SD (m/sec)	56.4011.12	47.17±0.84*
Posterior tibial CV Mean+SD (m/sec)	50.3±1.57	37.90+0.86
Median sensory CV Mean+SD (m/sec)	40.21+1.59	36.70+1.09*
Sural CV Mean±SD (m/sec)	46.96±1.46	35.57±1.43*

CV: Conduction velocity
*P<0.01

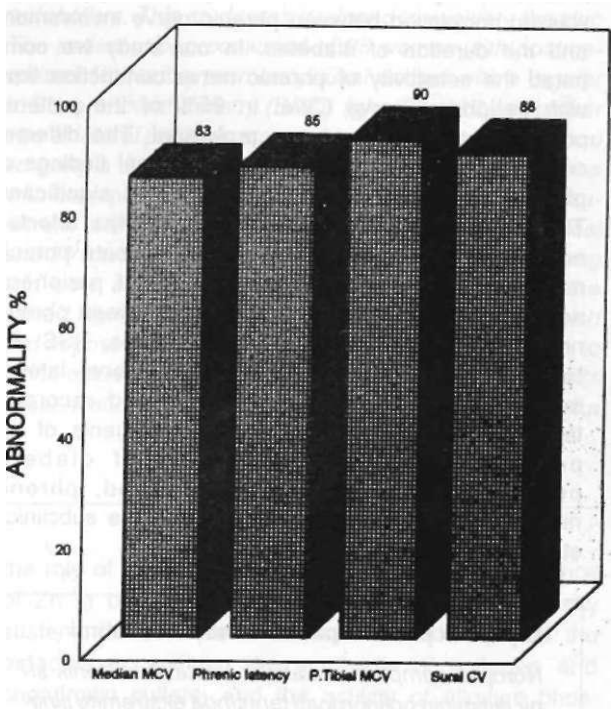

Figure 2. Abnormality percentages of phrenic nerve latency and peripheral nerve conduction velocities in diabetic patients.

Phrenic nerve conduction studies were used in patients after cardiac or thoracic surgery (4). Evidence of unilateral or bilateral diaphragmatic dysfunction were found in some of the patients. Phrenic nerve studies are also clinically helpful in the evaluation of the diseases of the peripheral nerves similar to the conventional conduction studies in the limbs. In patients with Guillaine - Barre syndrome, serial phrenic nerve studies may be helpful in detecting diaphragmatic in-

volvement before ventilatory insufficiency may manifest clinically (5). Other demyelinating polyneuropathies such as HSMN type **1,** are also associated with prolonged latencies of the diaphragmatic action potential (5). On the other hand, polyneuropathies associated primarily with axonal degenaration such as HSMN type 2 revealed either no or only minimal abnormalities in the latency and the amplitude of the diaphragmatic potential. In diabetic neuropathy, loss of both myelinated and unmyelinated fibers may occur with Schwann cell damage and axonal degeneration proceeding independtly (6). So, it is reasonable to measure phrenic nerve latency in diabetic patients. In this study we evaluated 40 diabetic patients with neuropathic symptoms using peripheral nerve CV, phrenic nerve latency, NSS and NDS. The only study in the literature reported 23.3% neuropathy in patients with randomly selected diabetic patients (7). There was no correlation between phrenic nerve involvement and the duration of diabetes. In our study we compared the sensitivity of phrenic nerve conduction time with peripheral nerve CV's. In 85% of the patients phrenic nerve latencies were prolonged. The differences between the percentages of abnormal findings of phrenic and peripheral nerves were not significant. This finding implies that phrenic nerve was affected equally with peripheral nerves. In 3 patients phrenic neuropathy was present despite normal peripheral nerve CV's. There was no correlation between phrenic nerve latency and the duration of diabetes, NSS and NDS. Thus, we conclude that phrenic nerve latency measurement by surface stimulation and recording technique is as useful as the measurements of the peripheral nerves in detection of diabetic polyneuropathy. Through this method, phrenic neuropathy may be discovered even at the subclinical stage.

Diyabetik polinöropatide frenik sinir iletimi

Nöropati semptomlu diabetik hastalarda frenik sinir iletiminin polinöropati tanısında ekstremite sinirleri ileti hızı ölçümleri ile birlikte kullanılabilirliğini araştırdık. Kırk diabetli hasta ve 30 sağlıklı kişi incelendi. Yaşlan sırasıyla 57.4±1.39 ve 49.1t1.05 idi. Hastalar San Antonio Diabet Konferansında kabul edilen nörolojik muayene ve semptom skalalarına göre değerlendirildiler. Kaydedici yüzeye! elektrodlar orta-aksiller çizgi üzerinde 8. interkostal aralığa konuldu. Sternokleidomastoid kasın arka kenarından uyarı verilerek diafragmatik aksiyon potansiyelleri kaydedildi. Ayrıca median sinir motor ve hissi, tibial motor ve sural sinir ileti hızları ölçüldü. Frenik sinir ortalama ileti zamanı kontrol grubuna kıyasla uzamış bulundu (Student's t testi, p<0.001). Hastaların %85'inde frenik sinir latansı uzamıştı. Frenik sinir ve ekstremite sinirlerinin anormallik yüzdeleri arasında anlamlı fark yoktu. Üç hastada sadece frenik sinir latansı uzamış bulundu. Frenik nöropati ile diabetin süresi, ortalama semptom ve muayene skorları arasında ilişki zayıftı. Sonuç olarak frenik sinir ileti zamanı ölçümlerinin diabetli hastalarda, polinöropatinin tanısında ekstremite sinirleri ileti hızı ölçümleri kadar duyarlı olduğunu, bazı hastalarda tek anormal bulgu olabileceğini ortaya koyduk. [Turk J Med Res 1995 (5): 184-186]

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