Pak. J. Biochem. Mol. Biol. 2010; 43(1): 33-36

Compression of median nerve, an electrophysiological study

Husan Bano[1], Nelofar Sultana[2] and Shehnaz A. Sheikh[3]
Department of Physiology, Sindh Medical College[1], Karachi, Pakistan
Department of Physiology, Dow Medical College[2], Karachi, Pakistan
Department of Biochemistry, Liaquat National Medical College[3], Karachi, Pakistan

Abstract: To study the electrophysiological changes occurring in patients with compression of median nerve. The study was conducted in the Department of Physiotherapy, Jinnah Postgraduate Medical Centre, Karachi, during the period of April 2006-May 2007. Twenty five patients with informed consent having median nerve compression were examined during the course of study. The electrophysiological recordings of 14 uni and 11 bilateral limbs were obtained. "Surface electrodes" were used for determination of median motor nerve conduction velocity (M-MNCV) and "ring-electrodes" for median sensory nerve conduction velocity (M-SNCV). Median motor conduction time (M-MCT), amplitude of motor action potential (MAP), amplitude of sensory action potential (SAP) and median sensory latency (M-SL) were also recorded. Statistical analysis performed by applying" student's t-test." All p-values <0.01 were considered statistically significant. In patients with median nerve compression, M-MCT significantly increased whereas M-MNCV and amplitude of M-MAP were significantly low as compared to normal subjects (P<0.01). Median sensory digital nerve of finger III showed significantly increased M.-SL (P<0.01). Significantly decreased values of M-MSNCV and amplitude of SAP were also observed (P<0.01). The findings of decreased conduction velocity, reduced amplitude of action potential (sensory and motor), indicate the functional state of nerve and the degree of severity of entrapment of median nerve at the wrist. These observations confirmed that nerve conduction study is a sensitive test for early detection of abnormal nerve functions, which also directs the physician towards the appropriate disorder and line of treatment.

Keywords: Electrophysiology, nerve conduction, motor nerve, sensory nerve, entrapment neuropathy.
Received: February 14, 2010 **Accepted**: February 28, 2010
***Author for Correspondence:** h.bano@yahoo.com

INTRODUCTION

Median nerve compression is a common entrapment neuropathy in an upper extremity[1]. It causes numbness in the thumb, index, and part of the ring finger[2].

The median nerve is derived from the lateral and medial cords of the brachial plexus and receives fibers from C6, C7, C8 and T1 roots. It is a mixed nerve supplying muscles in the forearm, hand and skin over the palmer aspect of the thumb, index and middle finger and the lateral aspect of the ring finger[3] (Figure 1) . The entrapment or damage of this nerve may occur in relation to trauma or degenerative arthritis, presence of ganglion, pregnancy, acromegaly or myxedema. Pain and paresthesia are early symptoms and may be confined to the hand in the median nerve distribution4. Clinical examination reveals an impairment of cutaneous sensation in a median nerve distribution in the hand, and if motor involvement has occurred, there is weakness and wasting of the abducter pollicis brevis[4].

Nerve conduction tests are commonly used in the assessment of patients with numbness, tingling and pain in the hand of patients with median nerve compression and/ or carpal tunnel syndrome. A variety of median nerve (sensory and motor) tests have been introduced for the purpose of establishing the presence of median neuropathy in patients with Carpal Tunnel Syndrome[5]. Many investigators have reported that the median nerve entrapment neuropathy at the wrist can be accompanied by slowed motor conduction within the forearm [5, 6, 7].

It has also been reported that motor nerve conduction velocity (M-NCV) becomes slow in smaller nerve fibers and causes fiber loss; this could be due to segmental demyelination[8].

Studies in CTS patients on the palmer cutaneous median branch and ulnar sensory nerve conduction showed reduced conduction velocity. This indicates the extension of sensory symptoms outside the median nerve distribution in CTS patients.[9,10] Similar results have been observed in CTS patients with polyneuropathy[10].

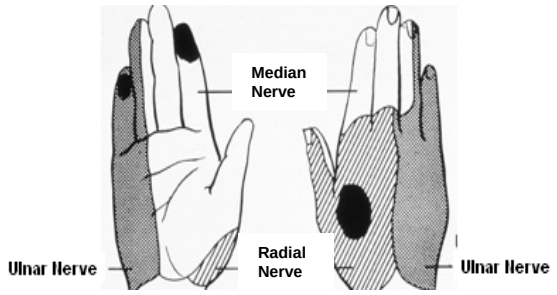

Figure 1: Median motor nerve.

The previous comparative studies on sensitivities of various parameters of "sensory nerve conduction" revealed that these tests did not significantly help in the diagnosis of carpal tunnel syndrome[11].

A study has observed that forearm mixed nerve conduction velocity is presumed to be indicative of conduction velocity of median nerve over the forearm[12]. In disease state, forearm mixed conduction velocity represents the conduction

velocity of palmar cutaneous branch[12,13]. Few studies have observed that proximal median NCV is usually affected by the distal lesion of Carpal Tunnel Syndrome[14-16].

The present study along with motor nerve conduction used other electrophysiological parameters such as median sensory nerve conduction velocity (M-SNCV), median sensory latency (M-SL) and amplitude of sensory action potential (SAP), in order to obtain the electrophysiological knowledge about the functioning of median nerve in normal healthy subjects and patients suffering from median nerve compression.

MATERIALS AND METHODS

Patients

During the study period of one year (April 2006-May2007), with informed consent twenty five patients of both the sexes suffering from median nerve compression with age range 16-70 years were examined and included in the study.

They were referred by neurologist to Physiotherapy department, Jinnah post graduate medical center (JPMC) for electromyography. The diagnosis of median nerve compression was made on the basis of patient's history. The present study included those patients of median nerve compression who met the following criteria[17].

Persistent sensory symptoms

Abnormal static 2-point discrimination (>6mm).
Diminished light touch sensation test.
Muscle wasting.
Positive provocative signs such as Phalen's and Tinel's sign[12].

Normal subjects

Twenty normal subjects without evidence of any neuro-muscular disorders were selected. They were M.Phil students of Basic Medical Science Institute (BMSI), JPMC and colleagues.

Apparatus and techniques

The action potentials in normal subjects and patients with compression of median nerve were recorded by electromyography.

Electrodes

"Surface electrodes" (stimulating and pick up) were used for the study of motor nerve conduction and "ring electrodes" for sensory nerve conduction. "Ground electrodes", to reduce the shock artifact. Before stimulating nerve, the equipment was carefully checked to insure proper setting of vertical position, focus, intensity and sweep speed. The calibration signals served as a source of known voltage to give specific vertical height. This calibration could be altered if necessary, to approximate the size of action potential obtained.

Median motor nerve

The conduction was obtained by placing the stimulating electrode along the nerve and by applying suprathreshold stimulus 200-250 volts for duration of 0.2 m sec (six times higher than motor threshold). This procedure was repeated at another point along the nerve as far as possible from first stimulating point. The response of muscle supplied by the nerve was picked up by surface electrode, displayed on cathode ray oscilloscope screen. The amplitude and duration of action potential were measured by the time and calibration signals. The distance between two points of stimulation (cathode to cathode) was then measured. The time taken for the impulse to travel between these two points was used to calculate the nerve conduction velocity.

$$NCV = \frac{\text{Distance (meter)}}{\text{Conduction Time (seconds)}}$$

Median sensory nerve

Conduction velocity was measured by placing ring electrode on the base of ring finger (Figure 3). The median sensory nerve was stimulated 13 cm proximal to the recording electrode.

RESULTS

The electromyographic study of 25 patients with clinically defined median nerve compression and 20 healthy subjects were obtained. Table 1 shows the variations of median motor conduction time (M-MCT), median motor nerve conduction velocity (M-MNCV) and amplitude of motor action potential (MAP).

Table 1: Comparative electrophysiological changes of median motor nerve in normal healthy subjects and patients with nerve compession.

Median nerve (Motor)	Normal subjects n=20 mean±SEM	Patients n=25 mean±SEM
M.MCT (m sec)	4.10* ±0.12	5.25* ±0.37
M-NCV (m/ sec)	58.33±0.74	46.52* ±1.25
MAP (mv)	6.55 ±0.45	2.26* ±0.41

*P<0.01

Comparison showed highly significant increased M-MCT (P<0.01), highly significant slowed M-MNCV (P<0.01) and significantly decreased MAP (P<0.01). Table 2 shows the comparative values of median sensory latency (M-SL), median sensory nerve conduction velocity (M-SNCV) and amplitude of sensory action potential (SAP) in digital nerve of finger III Significantly increased values were found in M-SL(P<0.01), significantly decreased values

were observed in M-SNCV and amplitude of SAP (P<0.01).

DISCUSSION

Entrapment neuropathy, nerve conduction velocity and amplitude of motor and sensory action potential is thought to be a sensitive indicator of severity of demyelination and ischaemia at the entrapment point[18]. Conduction velocity measurement in median nerve compression is of diagnostic significance, such as in carpal tunnel syndrome. Since conduction velocity measurement can identify sub-clinical lesions, it has particular value in initial diagnosis[19]. The present study agrees with the previous studies that nerve conduction velocity, distal latency and amplitude of motor action potential have useful diagnostic value[17, 18].

Table 2: Comparative electrophysiological changes of median sensory nerve in normal healthy subjects and patients with nerve compression.

Median sensory nerve (Finger III)	Normal subjects n-20 mean± SEM	Patients n=25 mean±SEM
M-SL (m sec)	1.97* ± 0.04	2.48* ± 0.17
M-SNCV (m/sec)	54.22 ±0.112	42.16* ± 2.44
SAP (mv)	27.90 ± 2.40	7.25* ± 2.42

*P<0.01

The distribution of nerve impairment in the present study was similar between males and females. Though females are more affected than males, perhaps because the carpal tunnel itself may be smaller in females than in males[2, 3]. In the present motor nerve conduction study, (Figure 2) the electrophysiological variables, M-MNCV and amplitude of MAP were found significantly reduced, where as M-CT showed higher values when compared to normal as reported earlier[5,6,10,17].

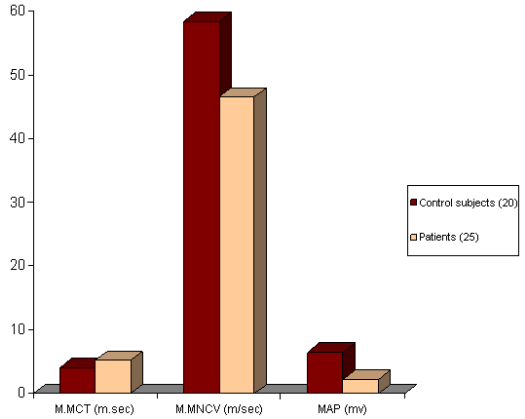

Figure 2: Comparative electrophysiological changes of median motor nerve in normal healthy subjects and patients with nerve compression.

A reduction in amplitude of MAP and increased conduction time may be due to either demyelination. [6,7,17] or could be due to the compression of median nerve which causes impairment of normal nerve functions.

In the present sensory median nerve conduction study, (Figure 3) reduced amplitude of SAP, slowed S-NCV and increased SL were found, which reflect the disease state of sensory nerve fibers in median nerve compression. It is suggested that the electrophysiological studies can identify signs of neurological impairment in the median nerve beyond the carpal tunnel. In the present study, there was a trend towards greater delay in MNCV and SNCV with increasing CT and SL where as decrease amplitudes of SAP and MAP reflect the functional impairment due to the severity of disease.

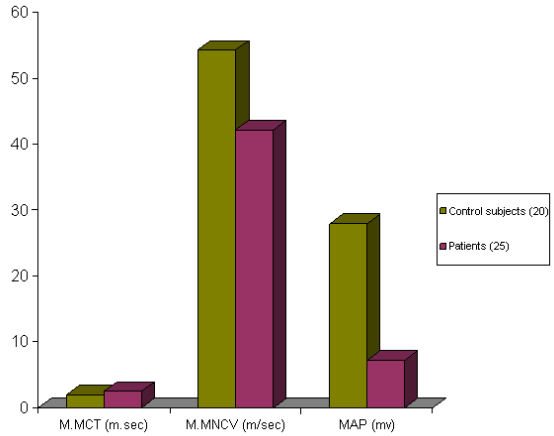

Figure 3: comparative electrophysiological changes of median sensory nerve in normal healthy subjects and patients with nerve compression

It is also suggested that if tests are conducted by skillful examiner using consistent methods the utility of SAP and MAP can be useful for assessing the clinical grading of median nerve compression or CTS.These parameters also have importance with respect to therapy selection. Despite the limitation of nerve conduction testing, it is the only tool currently available that can provide direct evidence of median neuropathy at the carpal tunnel to strongly support the diagnosis of CTS in symptomatic patients. Nerve conduction testing also provides an assessment of the severity of median neuropathy, which can be helpful in making decisions concerning type of treatment.

CONCLUSION

The diagnostic role of nerve conduction tests is gaining importance in today's clinical practice

physicians with varying degrees of experiences manage patients with median nerve compression and/or CTS.

Thus in treating patients with median nerve compression relying either on a) clinical examination of the hand or b) nerve conduction testing should be avoided and both of these examinations be done together.

REFERENCES

1. Isam A, Christina G, Ragnar J and Ewald O. Diagnostic properties of nerve conduction tests in population based carpal tunnel syndrome. *BMC Musculo Skeletal Disorder*, 2003; 4: 9
2. Lenman JA and Ritchie AE. Clinical electromyography 1st Ed, 1970; pp 90.
3. Aminoff MJ. Electromyography in clinical practice 1st Ed, 1978; pp 134.
4. Jablecki CK, Andary MT, Floeter MK, Miller RG, Quartly CA, Vennix MJ, Wilson JR. Second AAEM literature review of the usefulness of the nerve conduction studies and needle electromyography for the evaluation of patients with carpal tunnel syndrome. *Muscle Nerve,* 2002; 25:918-922.
5. Leif A, Jhon R, Hotson and Jan-Olof Kellerth. Correlation of median fore arm conduction velocity with carpal tunnel syndrome severity. *J. Clin. Physiol.,* 2006; 12: 11.
6. Pease WS, Lee HH and Johnson EW. Forearm median nerve conduction velocity in carpal tunnel syndrome. *Electromyogr. Clin. Neurophysiol.,* 1990; 30: 299-302.
7. Trojaborg W. Prolonged conduction block with axonal degeneration. An electrophysiological study. *J. Neurol. Neurosurg. Pschiat.,* 40: 50-57.
8. Bano H, Sultana N, Rukhsana N and Jafri TK. Study of electrophysiological parameters in healthy subjects and patients of motor mononeuropathy. *Med. Channel*, 2007; 13: 59-63.
9. Ratha Krishnan R, Therimadasamy AK, Chan YH and Wilder-Smith EP. The median palmar cutaneous nerve in normal subjects and carpel tunnel syndrome. *Clin. Neurophysiol.,* 2007; 118: 776-780.
10. Ayse TB, Feride G, Sumer G, Isik K and Mustufa G. The role of sensory nerve conduction study of the palmer cutaneous nerve in the diagnosis of carpal tunnel syndrome in patients with polyneuropathy. *Neurol. Ind.,* 2007; 55:17-21
11. Prakash KM, Fook-Chona S, Leoh TH, Dan YE, Nuriannah S, Tan YE and Lo YL. Sensitivities of sensory nerve conduction study parameters in carpal tunnel syndrome. *Clin. Neurophysiol.,* 2006; 23: 565-567.
12. Chang MH, Wei SJ, Chiang HL, Wang HM, Hsieh PF and Huang SY. Forearm mixed conduction velocityL: questionable role in evaluation of retrograde axonal atrophy in carpal tunnel syndrome. *Clin. Neurophysiol.,* 2003; 20: 196-200.
13. Chang MH, Wei SJ and Chen LW. The reason for forearm conduction slowing in carpal tunnel syndrome: an electrophysiological follow-up study after surgery. *Clin. Neurophysiol.,* 2003; 114: 1091-1095.
14. Tzeng SS, Wu ZA and Chu FL. Proximal slowing nerve conduction velocity in carpal tunnel syndrome. *Zhonghua Tixue. Zazhi. Taipal.,* 1990; 45: 86-90.
15. Pease WS, Lee HH and Jhonson EW. Forearm median nerve conduction velocity in carpal tunnel syndrome. *Electromyogr. Clin. Neurophysiol.,* 1990; 30: 299-302.
16. Chang MH, Liu LH, Wei SJ, Chiang HL and Hsieh PF. Does retrograde axonal atrophy really occur in carpal tunnel syndrome patients with normal forearm conduction velocity. *Clin. Neurophysiol.,* 2004; 115: 2783-2788.
17. Ogura T, Akiyo N, Kubo T and Kira Y. Relationship between nerve conduction study and clinical grading of carpal tunnel syndrome. *J. Orthoopaed. Surg.,* 2003.
18. Johnson EW and Olsen KJ. Clinical value of nerve conduction velocity determination. *J. Am. Med. Assoc.,* 1960; 172: 2030-2035.
19. Stetson DS, Silverstein BA, Keyserling WM, Wolfe WA and Albers JW. Hypothesis relating cumulative trauma to median nerve with subclinical nerve conduction deficits. *Am. J. Ind. Med..,* 1995; 27: 309-210.