# COMPARISON OF TERMINAL LATENCY INDEX IN PATIENTS WITH ENTRAPMENT AND GENERALIZED NEUROPATHY


M.M.U. Kabiraj, M.D.[1]*, V.P. Gupta, Ph.D.[2], A.A. Jamil, M.D.[2] and A.H.Shah, Ph.D., D.Sc.[2]

1Department of Neuroscience, Riyadh Armed Forces Hospital, P.O.Box N-641, Riyadh-11159, Saudi Arabia

2Riyadh Medical Complex, Central Laboratories, P.O.Box 9120, Riyadh-11413, Saudi Arabia.



## ABSTRACT

The terminal latency index (TLI) of 31 patients with clinical and electrdiagnostic evidence of Carpal Tunnel Syndrome (CTS) having retrograde changes reflected by reduced conduction velocity of the median nerve in the forearm of the affected hand (Group I) and 34 diabetic patients with frank peripheral neuropathy (Group II) was calculated and compared with those of the control group (n = 38). The TLI in group I was significantly abnormal ($P < 0.005$) but it was within normal limits for group II. Nerve conduction studies showed significant changes ($P < 0.005$) of median nerve conduction velocity (MNCV), its sensorimotor terminal latency (STL) and amplitude of the compound muscle action potential (CMAP) for both groups as compared to those of the control. The normal ulnar conduction in group I and highly slow ($P < 0.005$) ulnar conduction observed in group II were suggestive of the involvement of peripheral neuropathy. In patients with peripheral neuropathies (Group II) an equal slowing across the carpal tunnel was observed. It was, therefore, concluded that TLI is a sensitive test to detect CTS even with retrograde changes. However, it is not sensitive evidence in detecting peripheral neuropathy of demyelinating nature.



(Key Words: carpal tunnel syndrome; CTS; terminal latency index; TLI; neuropathy; nerve conduction; clinical studies)


## INTRODUCTION

Various sensorimotor parameters have been proposed for complete investigations of patients suffering from carpal tunnel syndrome (CTS) (Simpson 1956; Johnson et al., 1987; Kabiraj et al. 1998). The reduction of the median nerve motor conduction velocity (MNCV) in the forearm and an increased distal latency were found to be a prominent change (Thomas et al. 1967; Buchthal et al., 1974; Kimura and Ayyar 1985). Buchtal (1975) demonstrated that the slow conduction of the median nerve in the forearm is not proportional to the increase in distal latency. Recent studies (Uchida and Sugioka 1993) revealed that both reduced MNCV in the forearm and reduced amplitude of evoked mixed nerve action potentials (EMNAP) can indicate retrograde degeneration in CTS.

For the proper diagnosis and classification of peripheral polyneuropathy, different electrophysiological tests were considered essential (Kaeser and Lambert 1962; Lambert 1962; Albers and Kelly 1989; Cornblath 1990). It was found that routine motor nerve conduction studies correlate well with demyelinating lesions, however, patients with anoxal neuropathy might show disproportionate delay in distal motor latency (Longibuan et al. 1994). Shahani et al. (1979) successfully demonstrated a significant reduction in the ratio of proximal conduction time (PCT) for equal distances across the wrist in patients with carpal tunnel syndrome and those with peripheral neuropathy. A careful literature review revealed that extensive studies are lacking in patients of this category to understand the significance of TLI and to provide support to earlier findings. Furthermore, no report is available to assess the role of TLI in identifying the retrograde changes in CTS patients and patients with demyelinating neuropathy.

In continuation of our work (Kabiraj et al. 1998) in the present study, we wish to report our findings on the usefulness and sensitivity of TLI in patients with CTS associated with retrograde neuropathy and patients with frank peripheral neuropathy.

## MATERIALS AND METHODS

The data obtained from the hands of normal subjects (n = 38) i.e. male (n = 19) and female (n = 19), aged 20-79 years served as control in the present study. The control subjects were hospital staff and volunteers who were completely free from peripheral neuropathy and any systemic conditions such as diabetes mellitus, excessive alcohol consumption, or exposure to toxins.

The patients were divided into two groups: Group I consisted of 31 subjects suffering from CTS (10 males and 21 females, age 27-68 years). A total of 41 hands of these CTS patients with electroclinical evidence were studied. In each case the diagnosis required the fulfillment of at-least two of the three neuro-physiological criteria: median distal medial motor latency > mean +2SD of the control group; distal latency for sensory action potential recorded from the index finger ≥ mean + 2SD control; and the amplitude of the compound action potential recorded from abductor pollicis brevis (APB) < mean - 2SD of the control as described earlier. Tinnel sign and Phalen tests were also positive in these patients (Kabiraj et al. 1998).

Group II comprised of 68 hands of 34 patients (18 males, 16 females, aged 20-86 years). All patients in this group suffered from demyelinating peripheral polyneuropathy with obvious muscle wasting of the limb muscles. The confirmed diagnosis was based on the following criteria: symmetrical proximal and distal weakness in the upper and lower extremities, a reflexia or hyporeflexia, pins and needles sensations and/or numbness in the extremities, nerve conduction studies with features of demyelination, and EMG findings suggestive of neurogenic changes.

The electrophysiological studies were conducted using MEDLEC Mystro instrument. The nerve conduction studies were performed according to the standard technique described by Kimura (1982). The skin temperature of subjects was kept more than 32 C. For motor conduction studies, belly-tendon recording technique was used. Orthodromic sensory conduction studies were done for the peripheral nerves. EMG was performed using bipolar concentric needle electrodes and abductor pollicis brevis muscles were sampled. In each case recording of fibrillation potentials, positive sharp waves with longer duration polyphasic potentials on volitional effort was regarded as neurogenic EMG (denervation).

Terminal latency index (TLI) for the median nerve was calculated according to the formula of Shahani et al (1979):

TLI = Terminal distance (mm) / MNCV proximal (m/sec) x Terminal motor latency (msec)

The results between groups were compared by using one-way analysis of variance (ANOVA). A probability value equal to or less than 0.05 was considered as a significant change.

## RESULTS

The results of our present study are presented in Table 1 and 2. The mean terminal latency index (TLI) in Group 1 patients suffering from CTS was found to be significantly low ($P < 0.005$) as compared to the control group. There was no significant differences in the TLI of Group II patients as compared to the control. The terminal latencies observed in Group I and Group II patients were significantly prolonged ($P < 0.005$) as compared to those of the control.

The results of the median nerve conduction velocity (MNCV) are shown in Table 1. The forearm motor conduction velocities of the median nerve were found significantly low ($P < 0.005$) in both the groups.

Table-1: Median nerve conduction study.

| Parameters | Control (n = 38) (mean ± SD) | Group I (n = 31) (mean ± SD) | Group II (n = 34) (mean ± SD) |
|---|---|---|---|
| Motor conduction velocity (m/sec) Median nerve | 56.57 ± 4.50 | 43.92 ± 3.35* | 41.63 ± 6.16* |
| Motor Terminal Latency (msec) Median | 3.27 ± 0.36 | 6.03 ± 1.36* | 4.94 ± 1.35* |
| CMAP (mV) Median | 5.04 ± 2.17 | 2.85 ± 1.04* | 2.00 ± 1.77* |
| Sensory Terminal Latency (msec) Median | 2.61 ± 0.04 | 3.91 ± 0.46* | 3.44 ± 0.81* |
| Terminal Latency Index | 0.43 ± 0.05 | 0.26 ± 0.06* | 0.40 ± 0.10 |

| | | | |
|---|---|---|---|
| Median | | | |

*P < 0.005 (Students' *t*-test).

The mean terminal motor latencies (TML) were 3.27 ± 0.36 msec, 6.03 ± 1.36 msec, and 4.94 ± 1.35 msec for the control, Group I and Group II respectively. However, the sensory conduction of the median nerve in both groups (I and II) was significantly low ($P < 0.005$) as compared to the control.

The motor conduction studies of the ulnar nerves (Table 2) showed that the mean values of CV, TL, and CMAP amplitude of Group II patients were significantly lower ($P < 0.005$) as compared to Group I. The results of the ulnar nerve conduction study in Group I did not show any significant change as compared to the control. Hence, parameters of ulnar nerve for Group I are taken as a control for Group II (Table II).

Table-2: Ulnar nerve conduction study.

| Parameter | Group I (n = 31) (mean ± SD) | Group II (n = 34) (mean ± SD) |
|---|---|---|
| CV (m/sec) | 59.00 ± 6.31 | 39.47 ± 8.1* |
| Terminal Latency (msec) | 2.61 ± 0.45 | 4.10 ± 1.23* |
| CMAP (mV) | 5.32 ± 3.00 | 3.12 ± 1.26* |

*P < 0.005 (Students' *t*-test).

**DISCUSSION**

The observed decreased TLI in Group I patients is indicative of disproportionate conduction of the median nerve across the carpal tunnel. These findings were substantiated by the highly significant prolongation of the motor terminal latency (MTL) caused by mechanical compression of the median nerve in the carpal tunnel in these patients.

The significantly lower proximal motor conduction (PMC) of the median nerve observed in the patients of Group I may be attributed to the retrograde changes in the nerve due to long-standing entrapment neuropathy. Our findings are in agreement with the earlier reports where such changes were observed in severe CTS (Gutmann and Holubar 1951; Kiraly and Krnjevic 1959; Cragg and Thomas 1961). There are contradictory reports on the disproportionate conduction between the proximal and distal segments of the median nerve across the carpal tunnel (Thomas 1960; Buchthal et al. 1974). However, studies done by Stoehr et al. (1978) revealed that the reduction of MNCV proximal to the wrist in CTS depends on the severity of the nerve lesion.

The amplitude of compound action potential recorded from abductor pollicis brevis in the present study was found significantly low which indicated axonal loss in CTS patients. The clinical observations correlated well with these findings and in the patients, besides weakness in abduction of the thumb, denervation was confirmed by EMG investigations. Our findings are in agreement with earlier reports (Kimura and Ayyar 1985) where in severely-affected CTS patients increased polyphasic motor units and decreased recruitment of the motor units along with decreased amplitude of the CMAP were observed.

Based on the results of our study it is concluded that significantly low LTI in patients (Group I) is indicative of axonal neuropathy. Our results support the findings of Longiguan et al. (1994) who suggested that disproportional slowing of proximal conduction and prolongation of distal motor latency with low CMAP amplitude might be considered as criteria for axonal neuropathy.

It is worth-mentioning that our patients in Group II with peripheral polyneuropathy had normal TLI reflecting proportionate reduction of proximal and distal conduction across the carpal tunnel. This observation was further supported by the significant reduction of the MNCV in the forearm of the patients and prolonged distal motor latency (Table 1) which reflected demyelinating neuropathy. In earlier studies it was also found that patients with demyelinating disease had marked reduction of conduction velocities. However, conduction velocity was nearly normal in patients with motor neuron disease and in experimental degeneration and axonal degeneration induced by thallium (Lambert 1956; Kaeser and Lambert 1962).

Based on the results of our present study it is concluded that TLI plays important role in detecting entrapment neuropathy as in CTS even if there are electrodiagnostic clues of retrograde changes. However, in cases of predominant demyelinating peripheral neuropathies, the TLI has negligible role. Further studies are warranted to pin-point the role of TLI in patients suffering from pure axonal or conduction block.

## REFERENCES

Albers, J.W. 1989. "Acquired inflammatory demyelinating polyneurophathies: clinical and electro-diagnostic features". Muscle Nerve. 12: 435-451.

Buchthal, F., A. Rosenfalck and W. Trojaborg. 1974. "Electrophysiological findings in etrapment of the median nerve at wrist and elbow". J. Neuro. Neurosurg. Psychiatr. 37: 340-360.

Carroll, G.J. 1987. "Comparison of median and radial nerve sensory latencies in the electrophysiological diagnosis of carpal tunnel syndrome". Electroenceph. Clin. Neurophysiol. 68: 101-106.

Cornblath, D.R. 1990. "Electophysiology in Guillain-Barre syndrome". Ann. Neurol. 27 (suppl), s17-s20.

Gellman, H. D.R. Chandler and J. Petrasek. 1988. "Carpal tunnel syndrome in paraplegic patients". J. Bone & Joint Surg. 70A: 517-519.

Gragg, B.G. and P.K. Thomas. 1961. "Changes in conduction velocity and fibre size proximal to peripheral nerve lesions". J. Physiol. 157: 315-327.

Johnson, E.W., M. Sipski and T. Lammertse. 1987. "Median and radial sensory latencies digit I: normal values and usefulness in carpal tunnel syndrome". Arch. Phys. Med. Rehab. 68: 140-141.

Kabiraj, M.M.U., S. Al-Rajeh, A.R. Tahan, M. Abdusljabbar, M. Al-Bunyan, A.K. Daif and A. Awada. 1998. "Carpal tunnel syndrome: a clinico-electrophysiological study". Med. Sci. Res. 26: 631-633.

Kaeser, H.E. and E.H. Lambert. 1962. "Nerve function studies in experimental polyneuritis". Electroenceph. Clin. Neurophysiol. 22 (suppl): 29-35.

Lambert, E.H. 1962. "Diagnostic value of electrical stimulation of motor nerves". Electroenceph. Clin. Neurophysiol. 22 (suppl): 9-16.

Lesser, E.A., D.O.S. Venkatesh, D.C. Preston and E.L. Logiguan. 1975. "Stimulation distal to the lesion inpatients with carpal tunnel syndrome". Muscle-Nerve. 18: 503-507.

Longiguan, E.L., J. John, B. Kelly Jr. and L.S. Adelman. 1994. "Nerve conduction and biopsy correlation in over 100 consecutive patients with suspected polyneuropathy". Muscle-Nerve 17: 1010-1020.

Loong, S.C. and C.S. Seah. 1971. "Comparison of median and ulnar sensory nerve action potentials in the diagnosis of the carpal tunnel syndrome. J. Neurol. Neurosurg. Psychiatr. 34: 750-754.

Simpson, J.A. 1956. "Electrical signs in the diagnosis of carpal tunnel and related syndromes. J. Neurol. Neurosurg. Psychiatr. 19: 275-280.

Stoehr, M., F. Petruch, K. Scheglmann and K. Schilling. 1978. "Retrograde changes of nerve fibers with the carpal tunnel syndrome". J. Neurol. 218: 287-292.

Szabo, R.M., S.R.H. Gleberman and M.P. Dimick. 1984. "Sensibility testing in patients with carpal tunnel syndrome". J. Bone & Joint Surg. 66-A: 60-64.

Thomas, J.E., E.H. Lambert and K.A. Cseuz. 1967. "Electrodiagnostic aspects of the carpal tunnel syndrome". Arch. Neurol. 16: 635-641.

**About the Authors**:

Mohammad Kabiraj, M.D. is a Consultant/Neurophysiologist at the Armed Forces Hospital in Riyadh. Dr. Kabiraj earned his MBBS (1969) and his Masters degree (1974) with honors, from Dhaka University, Bangladesh in the field of Physiology. Dr. Kabiraj has conducted physiology studies as a research scholar in Sweden and as an Assistant Professor of Physiology at the King Saud University in Riyadh. His primary research interests lie in the areas of electrodiagnostic medicine and intra-operative monitoring for epilepsy surgery.

Vijay Prakesh Gupta, D.F.M., Ph.D. presently works for the Ministry of Health in Riyadh, Kingdom of Saudi Arabia as Legal Specialist and Clinical Toxicologist. Dr. Gupta also serves as an Adjunct Professor at Greenwich University. He earned his M.B.B.S. from Agra University in India, his D.F.M. in Forensic Medicine from Bangalore University, India and his Ph.D. in Forensic Medicine and Toxicology from Greenwich University. Dr. Gupta is a member of ten international medical, forensic and toxicological societies; has written more than 17 published papers on forensic science and toxicology and received awards for his work in both India and the Kingdom of Saudi Arabia.

Anis Ahmad Jamil, M.D., D.C.H. currently serves as a Senior Consultant/Pediatric Neurologist and Head of the Department of Pediatric Neurology and Clinical Neurophysiology at The Children's Hospital, Department of Pediatric Neurology, Riyadh Medical Complex. Dr. Jamil formerly served as the Director of the Arab and Saudi Board Residency Program (Pediatric) and as the Co-Chairman of the Department of Postgraduate Medical Education and Academic Affairs. Dr. Jamil brings over 25 years of clinical experience in pediatrics and neurology to his research and has authored or co-authored over 30 professional papers. Dr. Jamil completed his M.B.B.S. in 1973, his D.C.H. in 1976, and earned his Doctorate in Pediatrics (M.D.) in 1981. Additionally, he is currently completing a Ph.D. in Neurology at Greenwich University. His main research interests lie in the areas of epilepsy, epileptic syndromes, and movement and neurometabolic disorders.

Arif Hussain Shah, Ph.D. D.Sc., currently serves as the Head of the Central Instrumental, Drug Stability & Research Departments at the Central Laboratory for Drug & Food Analysis, Ministry of Health in Riyadh, Saudi Arabia, where he is also the Drug Analysis Specialist/Consultant. Professor Shah also currently holds teaching positions the Open International University and Greenwich University. He has also held teaching and research posts at King Saud University and Gomal University. Prof. Shah has authored or co-authored over 125 research articles in various international journals on topics of structural determination of new compounds, toxicity evaluation, and assay methods for drug products. He received his B.Sc. and M.Sc. from the University of Peshawar in Peshawar, Pakistan and his M.S. and Ph.D. from the Institute of Organic Chemistry & Biochemistry, in Bonn, Germany.

**Cite As**: Kabiraj, M.M.U, V.P, Gupta, A.A. Jamil, and A.H. Shah. 2000. Comparison of Terminal Latency Index in Patients with Entrapment and Generalized Neuropathy. *Greenwich Journal of Science and Technology*. 1(1):4-10.

Return to GJST Home Page