Clinical Neurophysiology 119 (2008) 2800–2803

Contents lists available at ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

The role of forearm mixed nerve conduction study in the evaluation of proximal conduction slowing in carpal tunnel syndrome

Ming-Hong Chang [a,b,*], Yi-Chung Lee [a,b,c], Peiyuan F. Hsieh [a,b]

[a] Section of Neurology, Taichung Veterans General Hospital, No. 160, Chung-Kang Road, Section 3, Taichung 40705, Taiwan
[b] Department of Neurology, National Yang-Ming University, Taipei, Taiwan
[c] Institute of Clinical Medicine National Yang-Ming University, Taipei, Taiwan

ARTICLE INFO

Article history:
Accepted 20 September 2008
Available online 30 October 2008

Keywords:
Forearm median mixed nerve
Carpal tunnel syndrome
CTS
Retrograde axonal atrophy
Retrograde conduction slowing

ABSTRACT

Objective: A decrease of forearm median motor conduction velocity (CV) is a common electrophysiological finding in carpal tunnel syndrome (CTS), ascribed to two possible mechanisms: either conduction block or slowing of the fastest myelinating fibers in the carpal tunnel, or retrograde axonal atrophy (RAA) with retrograde conduction slowing (RCS). We hope to utilize both direct and derived forearm median mixed nerve conduction studies to clarify the mechanism of the decrease of forearm median motor CV in CTS.

Methods: Seventy-five CTS patients and 75 age-matched control subjects received conventional motor and sensory nerve conduction studies of median and ulnar nerves and forearm median mixed nerve conduction techniques. First, direct measurement of forearm median mixed conduction velocity (Forearm mixed CV) and nerve action potential amplitude (Forearm mixed amplitude) was determined with recording at elbow and stimulation at wrist. Then, stimulating electrode was placed over palm and recording at elbow and then at wrist to calculate the derived Forearm mixed CV. Electrophysiological parameters, including direct Forearm mixed CV and amplitude and derived Forearm mixed CV, were compared between CTS patients and controls.

Results: CTS patients had significantly prolonged wrist–palm sensory and motor conduction, significantly decreased forearm median motor CV, and normal ulnar nerve conduction. The direct Forearm mixed amplitude was significantly decreased in CTS patients. The direct Forearm mixed CV was similar in CTS patients and controls, but there was a significant decrease in derived Forearm mixed CV in CTS group. The difference between direct and derived Forearm mixed CV was significantly greater in the CTS, suggesting that direct and derived Forearm mixed CV represent CV from different nerve fibers, one passing outside carpal tunnel without undergoing RAA or the other through the carpal tunnel with occurrence of RAA.

Conclusion: A decrease of direct Forearm mixed amplitude really occurs in CTS, implying that RAA and RCS will develop over proximal median nerve at distal nerve injury and the decreased forearm median motor CV is best ascribed to RAA and RCS. Furthermore, in CTS, the direct Forearm mixed CV measures the CV from undamaged nerve fibers without passing through carpal tunnel, resulting in the misinterpretation of the cause of proximal conduction slowing secondary to conduction block or slowing over the wrist.

Significance: We provide a direct evidence of the occurrence of RAA and RCS that would explain the cause of proximal median nerve conduction slowing. However, the clinical significance of RAA and RCS is uncertain.

1. Introduction

Median wrist–palm conduction study typically demonstrates a decrease in conduction velocity (CV) or prolonged conduction time in patients with carpal tunnel syndrome (CTS) (Buchthal and Rosenfalck, 1971; Kimura, 1978; Mills, 1985). Occasionally, some patients show decreased forearm median motor CV (Buchthal et al., 1974; Chang et al., 2000; Uchida and Sugioka, 1993; Wilson, 1998), which is commonly ascribed to two possible mechanisms. One is a conduction block of the fastest myelinating fibers in the carpal tunnel, leaving only the slower and smaller axons to be measured (Stevens, 1997; Thomas and Fullerton, 1963; Wilson, 1998). The other is retrograde axonal atrophy (RAA) with retrograde conduction slowing (RCS), which may result in increased electrical resistance and therefore a decreased

* Corresponding author. Address: Section of Neurology, Taichung Veterans General Hospital, No. 160, Chung-Kang Road, Section 3, Taichung 40705, Taiwan. Tel.: +886 4 23741302; fax: +886 4 23584403.
E-mail address: cmh50@ms10.hinet.net (M.-H. Chang).

1388-2457/$34.00
doi:10.1016/j.clinph.2008.09.014

CV (Chang et al., 2002a, 2003a, 2004; Devor and Govrin-Lippmarn, 1986; Dyck et al., 1981). The real mechanism may be disclosed by measurement of the forearm median mixed nerve conduction velocity (Forearm mixed CV) (Pease et al., 1990; Uchida and Sugioka, 1993; Wilson, 1998). If the forearm median motor CV is slow with a decrease in Forearm mixed CV, RAA with RCS would be suggested (Pease et al., 1990; Uchida and Sugioka, 1993), and if with a normal Forearm mixed CV, conduction block or slowing would be indicated (Wilson, 1998). The measurement of Forearm mixed CV in previous studies all utilized a direct method with recording at elbow and stimulation at wrist. However, some electromyographers argued that the direct Forearm mixed CV might not reflect the actual CV of mixed nerve fibers passing through the carpal tunnel, because some median nerve fibers in the forearm may not pass through the carpal tunnel and these fibers do not undergo RAA and the reduction of CV (Chang et al., 2002b, 2003b; Hansson, 1994). Therefore, direct measurement of Forearm mixed CV is not appropriate for assessing the cause of decrease of the forearm median motor CV (Chang et al., 2002b, 2003b). However, if the direct forearm median mixed nerve amplitude (Forearm mixed amplitude) comprises two fiber components and RAA really occurs over the nerve fibers through carpal tunnel, Forearm mixed amplitude would be decreased. If direct Forearm mixed amplitude were not decreased, the conduction block and slowing would be concluded.

In an attempt to clarify the mechanism of decreased forearm median motor CV in CTS, we conducted this study to compare direct Forearm mixed amplitude between CTS and controls and to compare direct and derived measurement of Forearm mixed CV with the hope of solving this conundrum.

2. Subjects and methods

The patients and controls consented and understood the detailed electrophysiological protocol.

2.1. Study population

Seventy-five consecutive patients with mild CTS symptoms were enrolled in the study. CTS was diagnosed clinically based on the presence of at least one of the following primary symptoms: (1) numbness, tingling pain or paresthesia in the median nerve distribution, (2) precipitation of these symptoms by repetitive hand activities which could be relieved by resting, rubbing and shaking the hand, and (3) nocturnal awakening by such sensory symptoms. The diagnosis was often supported by a positive Tinel's sign. All patients with clinically diagnosed CTS demonstrated median neuropathy at the wrist confirmed by the presence of one or more of the following standard electrophysiological criteria: (1) prolonged distal motor latency (DML) to the abductor pollicis brevis (APB) (abnormal $\geqslant$ 4.4 ms, stimulation over the wrist, 8 cm proximal to the active electrode); (2) prolonged anti-dromic distal sensory latency (DSL) to the second digit (abnormal $\geqslant$ 2.9 ms; stimulation over the wrist, 14 cm proximal to the active electrode); and (3) prolonged anti-dromic wrist–palm sensory conduction time (W-P SCT) at a distance of 8 cm (W-P SCT, abnormal $\geqslant$ 1.9 ms). Advanced CTS patients were not included as it is extremely difficult to elicit the mixed or sensory response. For example, patients with a complaint of fixed or continuous sensory impairment (numbness or pain) or with an abnormal pin-prick sensation over median nerve distribution were excluded before electrophysiological studies. Furthermore, patients with muscle atrophy or weakness of APB upon neurological examination were excluded. In addition, patients with a history of or physical examination results suggestive of a neuromuscular disorder other than CTS, for example, polyneuropathy or hereditary neuropathy, were also excluded. The clinical diagnosis of CTS was confirmed by at least two experienced neurologists.

2.2. Electrophysiological protocol

The studies were performed using a Nicolet Viking IV or Dantec Key Point 4 electromyograph. Surface recording after stimulation was performed for all studies. Recording electrodes were two 1-cm-diameter stainless steel disk electrodes for motor and mixed nerve studies, and saline-soaked velcro ring electrodes for anti-dromic sensory studies. The surface temperature of the hand was measured and maintained at or above 32 °C.

2.2.1. Motor nerve conduction studies of median and ulnar nerves

Median and ulnar motor studies were performed by recording compound muscle action potentials (CMAPs) from the APB and abductor digiti minimi (ADM), and detailed methods were described in the previous studies (Chang et al., 2006). DML, CMAP amplitudes from the baseline to the negative peak, the median and ulnar forearm motor CV, median wrist–palm motor CV (W-P MCV) were determined and calculated. Furthermore, the ratio of wrist/palm CMAP amplitudes was calculated. Conduction block was defined as 30% reduction of CMAP amplitude at the wrist stimulation compared with that at palm stimulation.

2.2.2. Sensory nerve conduction studies of median and ulnar nerves

Median and ulnar sensory nerve action potentials (SNAPs) were obtained by producing anti-dromic stimulation at the wrist and palm. DSL, W-P SCT as well as SNAP amplitudes from the baseline to the negative peak were determined.

2.3. Measurement of direct forearm median mixed nerve amplitude and conduction velocity (direct Forearm mixed amplitude and CV)

Measurement of direct Forearm mixed amplitude and CV was performed by stimulation at the wrist with the recording electrode placed at the elbow (wrist–elbow). Supramaximal responses were obtained, and latencies to the onset as well as amplitudes from the baseline to the negative peak were measured and so direct Forearm mixed amplitude and CV could be determined.

2.3.1. Measurement of derived forearm median mixed nerve conduction velocity (derived Forearm mixed CV)

The stimulating electrode was placed at palm and recording electrode was at wrist first, 8 cm proximal to the stimulating electrode, and then over the elbow. Latency was measured from the onset or takeoff and after the subtraction of conduction time of palm–elbow and palm–wrist, whereupon the derived measurement of Forearm (wrist–elbow) mixed CV was calculated and determined.

2.4. Statistical analysis

Comparison of median DML, DSL, W-P SCT, W-P MCV, forearm median motor CV, direct Forearm mixed amplitude, direct and derived Forearm mixed CV, ulnar DML and forearm ulnar motor CV was made between controls and CTS patients. Furthermore, the difference between direct and derived Forearm mixed CV was determined by the direct measurement of Forearm mixed CV minus the derived measurement of Forearm mixed CV and was then compared between controls and patients using the Mann–Whitney test to characterize the significance of the results. A p value of <0.05 was considered significant.