Preliminary Study on the Lesion Location and Prognosis of Cubital Tunnel Syndrome by Motor Nerve Conduction Studies

Zhu Liu, Zhi-Rong Jia, Ting-Ting Wang, Xin Shi, Wei Liang
Department of Neurology, The First Hospital of Peking University, Beijing 100034, China

Abstract

Background: To study lesions’ location and prognosis of cubital tunnel syndrome (CubTS) by routine motor nerve conduction studies (MNCSs) and short-segment nerve conduction studies (SSNCSs, inching test).

Methods: Thirty healthy subjects were included and 60 ulnar nerves were studied by inching studies for normal values. Sixty-six patients who diagnosed CubTS clinically were performed bilaterally by routine MNCSs and SSNCSs. Follow-up for 1-year, the information of brief complaints, clinical symptoms, and physical examination were collected.

Results: Sixty-six patients were included, 88 of nerves was abnormal by MNCS, while 105 was abnormal by the inching studies. Medial epicondyle to 2 cm above medial epicondyle is the most common segment to be detected abnormally (59.09%), \( P < 0.01 \). Twenty-two patients were followed-up, 17 patients’ symptoms were improved. Most of the patients were treated with drugs and modification of bad habits.

Conclusions: (1) SSNCSs can detect lesions of compressive neuropathy in CubTS more precisely than the routine motor conduction studies. (2) SSNCSs can diagnose CubTS more sensitively than routine motor conduction studies. (3) In this study, we found that medial epicondyle to 2 cm above the medial epicondyle is the most vulnerable place that the ulnar nerve compressed. (4) The patients had a better prognosis who were abnormal in motor nerve conduction time only, but not amplitude in compressed lesions than those who were abnormal both in velocity and amplitude. Our study suggests that SSNCSs is a practical method in detecting ulnar nerve compressed neuropathy, and sensitive in diagnosing CubTS. The compound muscle action potentials by SSNCSs may predict prognosis of CubTS.

Key words: Cubital Tunnel Syndrome; Electrophysiology; Short-segment Nerve Conduction Studies; Ulnar Nerve

Introduction

Cubital tunnel syndrome (CubTS) is the second most common peripheral entrapment neuropathy, the first one is carpal tunnel syndrome.\(^1\)\(^-\)\(^3\) Patients with CubTS often complained numbness of ulnaris side hands, paresthesia of elbow, and these symptoms can be exacerbated by flexion of elbow.\(^4\) In advanced stage, the affected limb presents weakness due to atrophy of hypothenar and Intrinsic hand muscles. Neuropathophysiological tests including traditional long-segment nerve conduction and short-segment nerve conduction studies (SSNCSs, inching test) are keys for diagnosis, illness evaluation and therapeutic effect.\(^5\)\(^,\)\(^6\) It has been discussed for a long time about the length that performed in neural electrophysiologic tests, across the elbow especially. In most of the early studies, 10 cm across the elbow has been widely accepted as the optimal distance.\(^7\) But as the technology has been developed, SSNCSs are used more and more in clinical trials. Campbell \textit{et al.} found that compared with traditional NCSs, SSNCSs have higher rate of abnormality and compressed lesions, which were proved to be correlated with operative findings.\(^8\) This study was aimed to discuss the value of prognosis, lesion location and diagnosis of these two neurophysiological studies in CubTS.

Methods

Patients group

All the patients were seen in our hospital between September 2009 and December 2012. We recruited 66 patients with CubTS, 44 male, 22 female, mean age 46.63 years old, range from 19 to 72 years, course of disease from 4 days to 8 years, all right handed. Inclusion criteria: (1) Patients with symptoms and positive physical signs of distribution region of ulnar nerve. (2) Patients suspected CubTS, and examined long segment motor nerve conduction studies (MNCSs) bilaterally. (3) Written informed consent was obtained from participants before procedure. Exclusion criteria: (1) Any cause of nervous dysfunction other than compression, such

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Address for correspondence: Prof. Zhi-Rong Jia, Department of Neurology, The First Hospital of Peking University, Beijing 100034, China E-Mail: jiazhirong@163.com

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as diabetic mellitus, hepatic and renal dysfunction, cervical spondylopathy, arm or elbow trauma history, paraneoplastic syndrome, toxicosis history (e.g., alcohol), hypothyroidism, amyloidotic peripheral neuropathy, vitamin B12 deficiency, connective tissue disease, infection, hereditary disease, and so on. (2) Abnormal upper arms that cannot perform neurophysiological tests. (3) Neurophysiological tests abnormalities found in other than ulnar nerve.

Healthy control group
Thirty healthy gender-and age-matched volunteers (12 men, 18 women) was included in healthy control group, mean age 37.43 years old, range from 24 to 58 years old, compared with patients group, P value is 0.805, the difference was not statistically significant.

Follow-up
One year later, we followed-up patients group and 22 of them were included. For those who were not convenient to come to the hospital, we called them for the chief complaint, symptoms, therapeutic regimen, and so on. Two of them had surgery, others were prescribed vitamin B12 to nourish nerves and modified bad habits which were not good for ulnar nerve, such as elbow over flexion. Risk factors for prognosis of CubTS were recorded, such as compressed lesion location, nerve conduction time and compound muscle action potential (CMAP).

Neurophysiological tests
Neurological test were performed by the key point electromyography machine (Bendimed, Denmark). Subjects were asked to lie supine position and in a warm, shielded and quiet room. Their extremity skin temperatures were at 32°C or above. The room temperature was maintained at 22°C–25°C. All the subjects were performed routine MNCS and SSNCSs bilaterally.

Motor nerve conduction studies
The routine MNCSs was conducted using a filter setting of 2 Hz to 10 kHz, a sensitivity setting of 2–10 mV/Division. Four centimeter distal to the midpoint of the medial epicondyle and 6 cm proximal to this level were stimulated, and abductor digiti minimi was for motor response. Before the measurements were performed, we ensured that supramaximal stimulation was achieved and that adequate pressure was applied to the stimulating electrodes in order to enable focal stimulation without spread. Latency was measured from stimulus to CMAP onset and amplitudes from baseline to the negative peak. We recorded every segment latency and conduction velocity.

Abnormal criteria
Practice parameter for electrodiagnostic studies in ulnar neuropathy at the elbow:[7]

Motor nerve conduction study
1. Motor nerve conduction velocity (MCV) is slower than the normal standard – 2 s, or latency is longer than the normal standard + 2 s
2. Absolute MCV from the above elbow (AE) to below elbow (BE) is <50 m/s. An AE-to-BE segment is greater than 10 m/s slower than the BE-to-wrist (W) segment
3. Decrease is >20% in CMAP negative peak amplitude from BE to AE; this is not for diagnose criteria alone.

Short-segment nerve conduction studies
1. Nerve conduction prolong of each segment is +2 s greater than the very segment of healthy control group
2. Compare with the adjacent segment, decrease more than 20% in CMAP.

Statistical analysis
The statistical package SPSS 14.0 was used to analyze the data (SPSS, Chicago, IL, USA). Mean values and standard deviations of conduction time and amplitude were calculated for each group. Independent samples t-test was used for comparison of latency and CMAP of MCV and inching test in each segment. Chi-square test was used to analyze the significance of inter-group differences in enumeration data. To find risk factors for prognosis, logistic analysis was used. A value of P < 0.05 was considered statistically significant.

Results
Healthy control group
Nerve conduction times and CMAPs of each stimulation were recorded bilaterally in this group [Tables 1 and 2], no statistically significant was found between right and left arms. The right and left arms in healthy control group had no statistically difference, so conduction time of each segment (+2 s) in the healthy group was used as the upper limit for patients group [Table 3].

Patients group
In the 66 patients, 41 subjects had unilateral lesions (41 arms, 30 left, 11 right), while 25 subjects (50 arms) had bilateral lesions. There were 41 arms without symptoms and positive physical examination. Among 91 symptomatic arms, 71 arms were presented numbness of distribution of ulnar nerve, 17 arms presented weakness of digitus minimus, 3 arms
only presented weakness of clenching fist. In 66 patients, 32 needed long-term operation of computers, 10 were manual laborers which included 4 machinery repairmen, 1 carpenter, 5 builders. Other 24 patients had no special work experience and personal history that may related to impairment of ulnar nerve.

**Motor nerve conduction study**

Eighty eight ulnar nerves were detected abnormal by routine MNCS, all were symptomatic. And the 88 ulnar nerves were abnormal in AE-to-BE segment, 4 in axilla-to-AE, 3 in BE-to-wrist. Eighteen ulnar nerves showed distal latency abnormal. There were 18 ulnar nerves had CMAP abnormality, including 10 in axilla, 18 in AE, 3 in BE, 3 in wrist [Table 4].

**Table 1: Mean and SD for latency of each segment of right and left arms in healthy control group by SSNCSs**

| Time (ms) | Left          | Right         | P       |
|----------|---------------|---------------|---------|
| TE − 3   | 0.39 ± 0.16   | 0.33 ± 0.18   | 0.139   |
| TE − 2   | 0.35 ± 0.18   | 0.37 ± 0.18   | 0.573   |
| TE − 1   | 0.43 ± 0.15   | 0.42 ± 0.14   | 0.801   |
| TE + 1   | 0.46 ± 0.15   | 0.48 ± 0.18   | 0.653   |
| TE + 2   | 0.31 ± 0.13   | 0.33 ± 0.13   | 0.460   |
| TE + 3   | 0.30 ± 0.12   | 0.29 ± 0.13   | 0.732   |

**Table 2: Mean and SD for CMAP of each segment of bilateral arms in healthy group by SSNCSs**

| CMAP (mV) | Left          | Right         | P       |
|-----------|---------------|---------------|---------|
| BE6       | 9.56 ± 1.94   | 9.97 ± 2.26   | 0.456   |
| BE4       | 9.68 ± 2.14   | 10.29 ± 2.44  | 0.309   |
| BE2       | 9.85 ± 2.13   | 10.16 ± 2.34  | 0.591   |
| E         | 9.90 ± 2.09   | 10.17 ± 2.51  | 0.645   |
| AE2       | 9.67 ± 1.96   | 10.00 ± 2.47  | 0.570   |
| AE4       | 9.65 ± 2.09   | 9.91 ± 2.57   | 0.662   |
| AE6       | 9.53 ± 2.15   | 9.91 ± 2.68   | 0.552   |

**Table 3: Upper limit conduction time of each segment in healthy group**

| Items      | TE − 3 | TE − 2  | TE − 1  | TE + 1  | TE + 2  | TE + 3  |
|------------|--------|---------|---------|---------|---------|---------|
| Mean value | 0.36 ± 0.16 | 0.36 ± 0.18 | 0.42 ± 0.14 | 0.47 ± 0.16 | 0.32 ± 0.13 | 0.30 ± 0.12 |
| Upper limit| 0.70   | 0.72   | 0.72   | 0.8     | 0.59     | 0.55     |

**Short-segment nerve conduction studies**

There were 105 arms detected to be abnormal by SSNCSs, including 88 asymptomatic arms, which diagnosed CubTS by traditional NCS, and 17 asymptomatic nerves that normal in traditional NCS. In our study, data showed that medial epicondyle to 2 cm above medial epicondyle (AE2-E) is the most common segment to be found abnormally [Table 5 and Figure 1]. The abnormality of each segment is significantly difference ($P < 0.01$). There were 24 nerves detected to be abnormal in CMAPs by SSNCSs, including 18 nerves were detected also abnormally by traditional NCS, and 6 nerves were normal by traditional NCS.

**Factors may suggest the prognosis**

In this study, we followed-up these 66 patients for 1-year. And data had been collected from 22 of them. When diagnosed CubTS for the first time, 16 patients had numbness in the distribution of ulnar nerve, 4 patients had abduction weakness of digitus minimus along with paresthesia of ulnar nerve distribution of elbow, 2 patients had atrophy of intrinsic muscle of hands.

Thirty eight nerves out of 44 (86.36%) were detected to be abnormal in segment of medial epicondyle to 2 cm above medial epicondyle (AE2-E). Four arms out of 44 (9.09%) had more than one compressed lesions. All 44 arms were abnormal in motor nerve conduction time by SSNCSs, while 16 arms were abnormal in CMAP at the same time.

Two patients with atrophy of intrinsic muscle of hands had surgery, others were prescribed vitamin B12 to nourish nerves and modified bad habits which was not good for ulnar nerve. In the 22 patients, 17 patients’ symptoms were improved, and modified bad habits which was not good for ulnar nerve.

Logistic regression was used to analyze factors may relate to CubTS’ prognosis, such as compressed lesion location, nerve conduction time, percentage of decreased CMAP, chief complaint, job character which may impair ulnar nerve, and number of compressed lesions. Nerve conduction time and percentage of decreased CMAP of six different segments had no statistical significance ($P > 0.05$), implied that lesions’ location may have no influence in prognosis. Absolute value of nerve conduction time of each segment also had no effect ($P = 0.247$). While, absolute percentage of decreased CMAP was statistical significance (odds ratio = 2.68, $P < 0.01$), suggested that percentage of decreased CMAP between segments close by may have a role in prognosis. However, larger sample sizes were needed for further study.
**Discussion**

Cubital tunnel syndrome is caused by compression of the ulnar nerve as it passes around the elbow joint. The most common compressed locations are struthers arcade, the medial intermuscular septum, medial epicondyle of humerus, the ligament of Osborne of elbow, place between the humeral head of flexor carpi ulnaris muscle and the ulnar head and deep flexor pronator teres tendon, especially Osborne ligament and the medial epicondyle of the humerus. Epicondyle of the humerus is where the ulnar nerve at the most superficial location and adjacent to bone structures, and hence ulnar nerve is vulnerable to external compression, traction and friction here. Osborne ligament compresses the ulnar nerve when elbow is in flexion or extension, and causes ulnar nerve dysfunction.\[9,10\]

Probable pathogenesis of CubTS includes: (1) When elbow is in flexion, the fibrous aponeurosis becomes thicker between the two heads of musculi flexor carpi ulnaris, which is the lateral wall of cubital tunnel, narrowing the cubital tunnel lacuna, making ulnar nerve compressed;\[11\] (2) Compression of ulnar nerve across the elbow by hyperostoeogny or osteophy, which aggravates the tunnel’s narrowing;\[11\] (3) Induced by soft tissue adjacent to the ulnar nerve in the tunnel;\[11\] (4) Compressed by intraluminal mass;\[11\] (5) When flex the elbow, cubital tunnel volume decreased, elbow support ligament contracted, and ulnar nerve compressed.\[10\] At the same time, increasing pressure in cubital tunnel and tension of ulnar nerve, could reduce the blood supply of the ulnar nerve, leading to ischemic necrosis, demyelination and Wallerian degeneration.\[12,13\]

So far for the Chinese patients who are clinical suspicion of CubTS, routine MNCS is used to detect ulnar nerve dysfunction. But it is short of discovering early or mild impairment, which can be covered by compensation. And it tests a 10 cm long distance, the result can only reflect general function of the ulnar nerve, hardly to locate the very lesion. SSNCSs can detect lesions in 2 cm distance, fix the lesions’ location precisely [Figure 2].\[14-16\] It is valuable for diagnosing CubTS, and help surgeries.

In this study, 78 nerves out of 132 (59.09%) were detected to be abnormal in segment AE2-E, and 26 nerves (19.70%) in segment E-BE2. It showed that 2 cm above medial epicondyle to medial epicondyle was the most common place ulnar nerve compressed, where the ulnar nerve goes into the ulnar groove of humerus, is superficial, and the ulnar nerve is adjacent to bony structures. It was vulnerable to the force of oppression caused by over flexation of elbow. The results kept a consistency with early studies. Visser et al.\[17\] reported that in 53 patients who were diagnosed CubTS by short-segment conduction study, 27% lesions were in the medial epicondyle, 20% in the digital of medial epicondyle. In the Herrmann et al.\[18\] report, 62% compression was located in proximal medial epicondyle of humerus, and 23% in the medial epicondyle, 15% was in the distal end of the medial epicondyle.

In this study and our previous study,\[11\] compressed lesions which detected by SSNCSs were also demonstrated by gross anatomy and surgery, and the most common place was consistent with anatomy feature. So the SSNCSs can provide accurate lesion locations for surgery. And 2 cm

### Table 4: Distribution of abnormal motor nerve conduction study

| Items            | DL | CMAP | MCV |
|------------------|----|------|-----|
| Wrist            | 18 | 3    | -   |
| BE-to wrist      | -  | 3    | 3   |
| AE-to-BE         | -  | 18   | 88  |
| Axilla-to-AE     | -  | 10   | 4   |
| Number           | 18 | 18   | 88  |

DL: Distal latency; CMAP: Compound muscle action potentials; MCV: Motor nerve conduction velocity; AE: Above medial epicondyle; BE: Below medial epicondyle.

### Table 5: Abnormality of each segment by SSNCSs

| Items | BE4-BE6 | BE2-BE4 | E-BE2 | AE2-E | AE4-AE2 | AE4-AE6 |
|-------|---------|---------|-------|-------|---------|---------|
| Number of abnormal nerves | 6       | 10      | 26    | 78    | 18      | 10      |
| Abnormality (%)          | 4.55    | 7.58    | 19.70 | 59.09 | 13.63   | 7.58    |

BE4-BE6: Segment of 6 cm below medial epicondyle to 4 cm below medial epicondyle; BE2-BE4: Segment of 4 cm below medial epicondyle to 2 cm below medial epicondyle; E-BE2: 2 cm below medial epicondyle to medial epicondyle; AE2-E: Medial epicondyle to 2 cm above medial epicondyle; AE4-AE2: 2 cm above medial epicondyle to 4 cm above medial epicondyle; AE4-AE6: 4 cm above medial epicondyle to 6 cm above medial epicondyle; SSNCSs: Short-segment nerve conduction studies.
above medial epicondyle should be paid highly attention in diagnose and evaluation of therapeutic effects of CubTS. It helps clinician in diagnosing, prognostic and evaluation of therapeutic effects of CubTS.

In this study, all the patients diagnosed CubTS by routine motor nerve conduction test, had symptoms such as limb numbness. 41 asymptomatic limbs were normal by routine motor nerve conduction testing, while 17 limbs of which were abnormal by inching test. It indicated that SSNCSs may be more sensitive in diagnosing CubTS, and can find minor and subclinical lesions [Figures 2 and 3]. In the Azrieli et al. study sensitivity of SSNCSs was 81%, the routine MNCS was 24%. Omejec et al. reported sensitivity of SSNCS was 76% to 90%, higher than the routine long-segment NCS. On the contrary, some researchers reported that the false positive results of SSNCSs across the elbow were due to the possibility of technical error induced by ulnar nerve dislocation. The key for nerve dislocation and measurement error of nerve distance was the position of elbow. American Association of Electrodiagnostic Medicine, American Academy of Neurology, American Academy of Physical Medicine and Rehabilitation recommended 70°–90° mild elbow flexion from horizontal, which was the least disproportion. So we adopted 70° in our study and hope to minimize the technical error.

Our study showed that percentage of decreased CMAP was statistical significance related to CubTS’ prognosis. It suggested that compressed lesions with CMAP severely decreased would have a higher risk of poor prognosis than the ones without or with less CMAP decreased in 1-year follow-up. The degree of decreased CMAP may also indicate the prognosis, the greater CMAP decreased the worse prognoses might got. The percentage of CMAP decreased may indicate to the degree of ulnar nerve impairment.

Our former study found that electrophysiological results correlated with pathogenesis. The ulnar nerves whose neurophysiological examination was normal, was compressed by congestive dilatation of the venous plexus, the degree of compression was mild and the nerve was normal in appearance. Cases with CMAP decreased and reduction in MCV of the ulnar nerve greater than 50%, were compressed by accurate ligment, hyperostoeogeny, or two heads of flexor carpi ulnaris. Mild pathogenesis always had normal CMAP, while severe ones often had CMAP decreased. It was in concert with the common sense in neurophysiology that the decreased velocity suggested demyelination, while decreased CMAP suggested axonal degeneration. And in the early stage of CubTS, pathology of ulnar nerve was mainly demyelination, and ischemia of compressed lesions. The impairment was reversible. In the terminal stage of CubTS, pathology turned to be axonal degeneration with demyelination. Decreased CMAP indicated ulnar nerve may have axonal degeneration. Compared with the ones only with demyelination, had a higher risk of poor prognosis, and harder to recover its function.

In our study, the lesion locations had no significant relationship with prognosis. But our cases showed a lesion preference in 2 cm above medial epicondyle. And almost all the patients had only one lesion. Whether lesions distribution had influence in prognosis or not, is still under discussion, and more studies are needed.

According to our study, the latency and nerve conduction time of SSNCSs may indicate lesions’ location, and CMAP may probably indicate prognosis.

Figure 2: Result of routine motor nerve conduction studies (MNCSs). A cubital tunnel syndrome (CubTS) clinical suspected patient had a routine MNCS. Before the measurements were performed, we ensured that supramaximal stimulation was achieved and that adequate pressure was applied to the stimulating electrodes in order to enable focal stimulation without spread. As the record showed that conduction velocity and compound muscle action potential decreased in segment of above elbow (AE) to below elbow (BE), so the compressed lesion located in the 10 cm segment of AE to BE. And diagnosed CubTS by neurophysiological standard, lesion can be located in this 10 cm segment.

Figure 3: Result of short segment nerve conduction studies (SSNCSs). The same cubital tunnel syndrome clinical suspected patient had a SSNCS. Before the measurements were performed, we ensured that supramaximal stimulation was achieved and that adequate pressure was applied to the stimulating electrodes in order to enable focal stimulation without spread. There was a significant change between 2 cm below medial epicondyle to medial epicondyle, latency had been prolonged and compound muscle action potential decreased. So the lesion was precisely located in this 2 cm distribution, and there was no evidence of multifocal compressed neuropathy in this patient.
In summary, we suggest that patients with symptoms that indicate the impairments of ulnar nerve, such as pains and numbness of the forearm and finger, weakness of hands and muscles atrophy, should have neural electrophysiological examination. Patients who highly suspected CubTS in clinic, and normal results of routine long segment nerve conduction, should have SSNCS, which can detect the nerve impairment in early stage and mild lesions, and avoid further development of the disease. Patients who are diagnosed CubTS by routine long segment MNCS, also should have SSNCSs, to ensure the locations of the ulnar nerve compressed lesions, and to discover whether there is multiple compressed lesions or not. It provides information in detail for surgeries. And physicians should give patients individual and comprehensive advices, such as correction of bad posture, which can recover the nerve function as soon as possible.

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