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Mixed Nerve Conduction Study of Brachial Plexus and Its Application on the Diagnosis of Brachial Plexus Lesion

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This study demonstrates a technique to measure the mixed nerve conduction velocity (MNCV) of the ulnar and median component of the brachial plexus and its application on the diagnosis of brachial plexus lesion. Twenty patients with clinically confirmed pectoralis minor syndrome (Group I) and ten patients with traumatic brachial plexus injury due to traffic accident (Group II) were studied. The ulnar nerve was stimulated at the ulnar groove and the median nerve stimulated at the antecubital fossa of the elbow. The averaged nerve action potentials were simultaneously recorded from both axilla and Erb's point with surface recording electrodes. MNCV of the ulnar and median components of the brachial plexus between the Erb's point and axilla (clavicular segment) were calculated. It was found that MNCV of the ulnar component of brachial plexus was 40.31 ± 2.75 m/sec (35.3-44.3 m/sec) on the affected side, and 60.43 ± 4.67 m/sec (54.2-69.4 m/sec) on the unaffected side in group I patients. In group II patients, there was no significant difference in MNCV between the affected and unaffected sides for either ulnar or median component of brachial plexus. It is concluded that measurement of MNCV of the brachial plexus is a valuable technique in the diagnosis of brachial plexus entrapment lesion, such as pectoralis minor syndrome, but less sensitive to detect a traumatic brachial plexus lesion with axonal loss. (J Rehab Med Assoc ROC 1997; 25(1): 57-63)

Key words: brachial plexus, nerve conduction, thoracic outlet syndrome

INTRODUCTION

The brachial plexus lesions may include direct nerve injury due to acute trauma, such as traction, contusion and compression, chronic entrapment, such as thoracic outlet syndrome (sclenus anicus syndrome, costoclavicle syndrome, first rib syndrome, and pectoris minor syndrome), or neoplastic compression, and brachial neuritis [3].

The electrodiagnostic study is an useful tool in the diagnosis of brachial plexus lesion. These techniques include proximal median and ulnar motor nerve conduction velocity (NCV) study [24], F wave response [30], somatosensory evoked responses [27], and sensory nerve conduction studies through brachial plexus [8].
proximal motor NCV study and F wave response study are painful for the patients. The F wave and SSEP studies evaluate the NCV of a long loop. The conduction slowing of the short segment of brachial plexus may be masked when the conduction time is measured at a long segment which includes the distal portion of the normally conducting nerve. Axillary F-loop latency \(^{[8,9,10,11]}\) was recommended to measure the relatively short segment of brachial plexus. However, only motor fibers were assessed in these studies, and it is a painful procedure.

In a recent case study of pectoralis minor myofascial pain syndrome after whiplash, Hong and Simons \(^{12}\) described a new technique to measure the mixed nerve conduction velocity (MNCV) of the brachial plexus by stimulating ulnar or median nerve at elbow and recording nerve action potentials from two sites (Axilla and Erb’s point) simultaneously. The advantages of this technique include: 1). recording of a short segment of brachial plexus, 2). elimination of the artifact from motor responses (antidromic conduction of motor fibers), and 3). less number of stimulation required (simultaneous recording from two sites by applying stimulation at one site).

The purpose of our study is to apply the techniques of mixed nerve conduction study on the median and ulnar components of brachial plexus as described by Hong and Simons \(^{12}\) to detect brachial plexus lesions. This study reports 20 patients with pectoralis minor syndrome who had positive findings in pectoralis minor (or hyperabduction) maneuver, and 10 patients with traumatic brachial plexus injury confirmed by the routine electromyographic (EMG) studies.

### METHODS

#### Subjects

Group I: Twenty patients (11 males and 9 females with average age of 37 ± 10 years) with clinical evidence of pectoralis minor syndrome were included in this group. All of them complained pain and tingling in the involved arm (mostly in the ulnar aspect) episodically or continuously. Every patient had a painful and tight pectoralis minor muscle in the involved side. The hyperabduction maneuver was positive in every patient.

In performing the hyperabduction maneuver, the arm in the affected side was brought up to about 120° flexion followed by backward stretch (abduction and extension of shoulder), so that the pectoralis minor muscle was stretched to create a compression force to the underlying neurovascular bundles. A positive test was based on the reduction or absence of radial pulse and aggravation of sensory complaints. The chronically tight pectoralis minor muscle was due to chronic myofascial trigger point (TrP) \(^{12}\). There was at least one TrP found in the pectoralis minor muscle of the affected side. The diagnosis of TrP was based on a tender spot in a palpable taut band with typical referred pain patterns as described by Travell and Simons \(^{13}\). These patients had no confirmed diagnosis of cervical radiculopathy or myelopathy based on neurological examination, radiological and EMG findings. EMG study showed no evidence of axonal lesion in any muscle of all patients. They also had no evidence of bone fracture or dislocation in radiological studies. These patients later received standard myofascial pain therapy and showed significant improvement of the symptoms.

Group II: Group II subjects consisted of 10 patients (9 males and 1 female with average age of 28 ± 11 years) with traumatic brachial plexus injury. These patients had pain, numbness and weakness of the upper extremity after traffic accidents. Obvious muscle atrophy of the affected limb was noted in 9 patients. Routine electromyographic study confirmed the diagnosis of brachial plexus lesion. Multiple root avulsion was noted in 2 patients, trunk lesion in 5 patients (upper trunk:2, upper and lower trunk:1, lower trunk:1, middle and lower trunk:1) and cord lesion in 3 patients (lateral and posterior cord:1, posterior cord:1, medial cord:1). Concomitant fracture was noted in two patients (fib fracture:2, Smith-Barton fracture:1), and rotator cuff tendinitis in one patient. Nerve graft and tendon transfer had been done in three patients before they receiving our examination.

### Nerve conduction study

An EMG machine (TECA MyoStar, Nicolet Viking, or Cadwell 5200A) was used for the nerve conduction studies. During the test, the subject was positioned supine, the elbow was extended and the arm fully supported at
the lateral side. The arm was abducted to around 75 degrees and the forearm was supinated.

The compound action potentials of the mixed nerve fibers were recorded from 2 sites: in the axilla and at the Erb's point. The active axillary recording electrode was placed in the axillary fossa near the brachial artery and the reference recording electrode was placed along the pathway of brachial artery, 3 cm proximal to the active electrode. The active Erb's recording electrode was placed in the supraclavicular fossa at the Erb's point and the reference electrode was placed along the pathway of the brachial plexus, 3 cm proximal to the active electrode. The ground electrode was placed centrally on the deltoid muscle. The ulnar nerve was stimulated at the ulnar groove and the median nerve stimulated at the antecubital fossa of the elbow (Fig. 1). The sweep speed was set at 2 msec/division. The sensitivity was set at 20 μV/division. The stimulus was a rectangular impulse with a duration of 0.1 msec at a frequency of 1 Hz. The voltage was increased progressively to gain an action potential of maximal amplitude. In the majority of cases, the evoked response recorded from the axilla could be obtained with no difficulty. However, the amplitude of nerve action potentials recorded from Erb's point in most cases, and from the axilla in a few cases, were too small to be recorded. In these cases, the responses of 100-500 consecutive stimuli were averaged.

The latencies were measured to the onset of the initial negative wave. The distance between the two active electrodes of Erb's point and axilla (D_{Erb-A}) was measured by a caliper. The tape meter was used to measure the distance between the stimulating electrode at elbow and the recording electrode at the axilla. The NCV can be calculated by the formula:

$$\text{NCV (m/sec) of clavicular segment} = \frac{D_{Erb-A} \text{ (mm)}}{L_{Erb} - L_A \text{ (msec)}}$$

Where D_{Erb-A} is the distance between Erb's point and axilla, and L_{Erb} and L_A represent the latencies recorded.

**Figure 1.** Mixed Nerve Conduction Study on the Median and Ulnar Components of Brachial Plexus. S1: Median nerve stimulation at the antecubital fossa. S2: Ulnar nerve stimulation at the ulnar groove. R1: The active axillary recording. R2: The active Erb's recording.

from Erb's point and from axilla respectively (Fig. 2). The skin temperature on the medial side of the forearm was measured with an electric thermometer and ranged from 32 to 34 °C.

In group I patients (pectoralis minor syndrome), mixed nerve conduction study was performed only on the ulnar component of the brachial plexus (stimulation of the ulnar nerve at elbow) since the clinical complaints (pain or tingling) only limited in the distribution of the ulnar sensory nerve supplied area, and it was done on both the ulnar and median components of brachial plexus (stimulation of both ulnar and median nerves at elbow) in group II subjects (traumatic brachial plexus lesion).

The nerve conduction velocity was measured bilaterally for comparison. We compared mean values of MNCV of the affected brachial plexus to that of the unaffected one (opposite side) by using student's two-sample t test. A value of p<0.05 was considered as statistically significant.

## RESULTS

Table 1 shows the mixed nerve conduction velocities of brachial plexus between Erb's point and axilla (clavicular segment).

Group I: The conduction velocity of the ulnar component of brachial plexus in the affected side was 40.31±2.75 m/sec (range 35.3-44.3 m/sec), and 60.43±4.67 m/sec (range 54.2-69.4 m/sec) in the unaffected side. There was significant difference of the MNCV of the ulnar component of brachial plexus between the affected and unaffected sides in Group I patients (p<0.05). However, there was no significant difference in amplitude of nerve action potential between the affected and unaffected sides.

Group II: The conduction velocity of the median component of brachial plexus in the affected side was 83.26±7.21 m/sec (range 70.2-99.2 m/sec), and 89.36±9.96 m/sec (range 74.0-109.6 m/sec) in the unaffected side. The conduction velocity of the ulnar component of brachial plexus in the affected side was 79.41±8.44 m/sec (range 64.5-91.7 m/sec), and 82.12±9.64 m/sec (range 66.7-96.5 m/sec) in the unaffected side. There was no significant difference in mean MNCVs between the affected and unaffected sides in either ulnar or median component of brachial plexus. There was also no significant difference in mean amplitudes of nerve action potentials between the affected side (35±16 µV for ulnar component and 31±18 µV for median component, recorded from axilla; 15±12 µV for ulnar component and 9±5 µV for median component, recorded from Erb's point) and unaffected side (39±13 µV for ulnar component and 34±15 µV for median component, recorded from axilla; 18±9 µV for ulnar component and 11±4 µV for median component, recorded from Erb's point).

## DISCUSSION

The findings in our study showed that the mixed nerve conduction velocities of ulnar component of brachial plexus between Erb's point and axilla of the

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Case No.</th>
<th>Onset Mon. ago</th>
<th>NCV (m/sec): Affected side</th>
<th>NCV (m/sec): Normal side</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Median</td>
<td>Ulnar</td>
</tr>
<tr>
<td>PMS</td>
<td>20</td>
<td>14.9±12.6</td>
<td>40.3±2.7*</td>
<td></td>
</tr>
<tr>
<td>Traumatic injury</td>
<td>10</td>
<td>20.6±9.9</td>
<td>83.2±7.2</td>
<td>79.4±8.4</td>
</tr>
</tbody>
</table>

PMS: Pectoralis Minor Syndrome
Median: Median component of brachial plexus
Ulnar: Ulnar component of brachial plexus
* p<0.05 as compared with the unaffected side
patients with pectoralis minor syndrome (Group I) were significantly different between the affected side and unaffected side. However, there was no significant difference in the mixed nerve conduction velocities of ulnar or median component of brachial plexus (clavicular segment) between the affected and unaffected sides in the patients with traumatic brachial plexus injuries (Group II).

There is a great difference in the pathophysiology between the thoracic outlet syndrome and traumatic brachial plexus injury. In pressure palsies of the brachial plexus, such as thoracic outlet syndrome, chronic demyelination is the predominant manifestation in a mild form or in an early stage. The amplitude of the motor and sensory nerve action potentials are reduced with temporal dispersion, and the conduction is slow across the segment of compression. The EMG findings may be normal in the mildly or moderately injured patients. In traumatic lesions of the plexus, axonal loss were often predominant after traction injury or mechanical destruction. EMG studies are generally more valuable than conduction studies [1].

In Group I patients, the unaffected side might also have some degree of involvement (subclinical), since TrPs (either active or latent) and palpable taut bands of the pectoralis muscles were also found on that side in almost every case. There was also a tendency that patients with more latent TrPs had slower mixed nerve conduction velocity of the ulnar component of the brachial plexus on the unaffected side. Though these patients had no active symptoms of the unaffected side, there might be some degree of demyelination happened, which was similar to the situation in the case of early carpal tunnel syndrome. This would explain why the mixed nerve conduction velocity of the ulnar component of the brachial plexus on the unaffected side in Group I patients was significantly slower than that on the unaffected side in Group II patients.

In our study, all the 20 cases in Group I were pectoralis minor syndrome. In other types of thoracic outlet syndrome such as scalenus anticus syndrome, costoclavicle syndrome, cervical rib syndrome, and first rib syndrome, the site of lesion may be proximal to Erb’s point or on the Erb’s point. Therefore, this technique may not be sensitive to detect such lesions. In fact, seven of our ten patients in Group II with traumatic brachial plexus injury had lesion at the trunk or root level, which may be proximal to the Erb's point. This may make our method also less sensitive for such cases. If we divided the patients of Group II into two subgroups (above Erb’s point and below Erb’s point) and calculated the mixed nerve conduction velocity of the ulnar component and the median component of brachial plexus respectively, it was found that the mixed nerve conduction velocities of both the ulnar and the median components of brachial plexus in the affected side are slower in the patients with lesion below the Erb’s point (cord lesions) than in the patients with lesion above the Erb’s point (root and trunk lesions). However, this difference was not statistically significant probably due to small sample size. The fibers in the median component of the brachial plexus include fibers through both medial and lateral cords. If a lesion involves only medial cord or lateral cord, and MNCV is measured by recording action potential from Erb’s point, it would be unable to detect a demyelination lesion of medial cord or lateral cord. Furthermore, there was only one patient in Group II had a lesion involving only medial cord. This was probably the reason why there was no significant difference in the mixed nerve conduction velocity of the ulnar component between the affected side and the normal side in Group II. It is unclear why there was no significant difference in amplitudes of nerve action potentials between the affected and unaffected sides in Group II patients. The amplitude of the evoked nerve action potential was not a reliable measurement due to the fact that the depth of the nerve is variable from person to person. The more valuable method may be to change the site of the recording electrode to the area near the cervical root. However, this technique is more difficult to performed and needle recording may be required.

Compared to the previous studies, our method is less painful and easily accepted by the patients. It evaluates a shorter segment and thus can localize the lesion in the brachial plexus more precisely. Moreover, both motor and sensory fibers were studied in the mixed nerve conduction studies. It is concluded that measurement of brachial plexus MNCV is a valuable technique in the diagnosis of brachial plexus lesion in the patients with pectoralis minor syndrome, but less sensitive to detect a traumatic brachial plexus lesion. Further study on
brachial plexus MNCV, including the comparison of the differences among lesions at different sites (lesions proximal or distal to Erb’s point, root, trunk, or cord lesions, etc.) and the assessment of improvement after therapy, would be very helpful to intensify the value of this special technique.

REFERENCE

臂神經叢正中神經部分及尺骨神經部分混合神經傳導測量以及其在上臂神經叢傷害診斷上之應用

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國立成功大學醫學院附設醫院  復健部  神經部*

本實驗主要是要測定臂神經叢傷害的病人，其臂神經叢正中神經部分 (median component) 及尺骨神經部分 (ulnar component) 混合神經傳導速率 (mixed nerve conduction velocity, MNCV)。實驗的對象是二十位臨床上診斷為胸小肌症候群 (pectoralis minor syndrome) 的病人 (第一組) 及十位外傷性臂神經叢傷害 (traumatic brachial plexus injury) 的病人 (第二組)。我們於肘關節處，分別電刺激尺神經及正中神經，並使用表面記錄電極於腋窩 (axilla) 及阿氏點 (Erb’s point) 處記錄其神經平均動作電位，且計算出腋窩及阿氏點間臂神經叢 (正中神經部分及尺骨神經部分) 混合神經傳導速率。實驗結果顯示：於第一組受試者，其患側臂神經叢尺骨神經部分混合神經傳導速率为 40.31±2.75 m/sec (35.3-44.3 m/sec)，對側則為 60.43±4.67 m/sec (54.2-69.4 m/sec)，二者具統計學上之差異 (p<0.05)。於第二組受試者，其患側臂神經叢尺骨神經部分混合神經傳導速率为 79.41±8.44 m/sec (64.5-91.7 m/sec)，對側則為 82.12±9.64 m/sec (66.7-96.5 m/sec)；其患側臂神經叢正中神經部分混合神經傳導速率为 83.26±7.21 m/sec (70.2-99.2 m/sec)，對側則為 89.36±9.96 m/sec (74.0-109.6 m/sec)。於患側及對側間，臂神經叢尺骨神經部分及正中神經部分混合神經傳導速率均沒有顯著差異。本實驗因此結論：測定臂神經叢尺骨神經部分混合神經傳導速率，對於診斷臂神經叢慢性壓迫傷害 (chronic entrapment) 是一相當有價值的方法；而對於外傷性的臂神經叢傷害且伴有神經軸損傷 (axon loss) 者，則較不具有診斷意義。（中華復健醫誌 1997; 25(1): 57-63）

關鍵詞：臂神經叢 (brachial plexus)，神經傳導 (nerve conduction)，胸小肌症候群 (thoracic outlet syndrome)