Ultrasound can be routinely used in the diagnosis of the carpal tunnel syndrome?: Electrophysiological and ultrasonographic study of the of carpal tunnel

B Bicerol, M Keceli, E Copcu, N Kiylioglu, O Bolukbasi, A Akyol

INTRODUCTION
Compression of the median nerve is the most common peripheral nerve pathology [1]. It is the primary factor in constriction of the carpal tunnel; i.e. carpal tunnel syndrome (CTS). The syndrome results from increased tunnel contents or a decreased tunnel size [1]. Among its etiological factors is hereditary predisposition (hereditary pressure sensitive neuropathy), trauma, (dislocation, fracture, forearm-wrist hematoma), occupational factors (recurrent percussion to the wrist or recurrent flexion and extension of the wrist), infection-inflammation (tenosynovitis, sarcoidosis), metabolic disorders (amiloidosis, gout), endocrine conditions (acromegaly, diabetes, hypothyroidism, pregnancy), neoplastic conditions (ganglion cyst, lipoma, myeloma), collagen vascular diseases (rheumatoid arthritis, scleroderma) degenerative disorders (osteoarthritis) and iatrogenic factors (stent insertion for dialysis, radial artery puncture) [1,3].

As to the pathophysiology of CTS, compression on the median n. in the tunnel or its ischemia first causes segmental demyelinization and then axonal degeneration [1]. There have been many studies on features of the carpal tunnel in CTS [1,2]. Electrophysiological investigations, the most frequent method for diagnosis of CTS, reveal the real-time severity of median n. involvement some time after the syndrome appears. Ultrasonographic, tomographic or magnetic resonance imaging of the carpal tunnel will offer
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Information on anatomy of the tunnel. There have been a lot of studies on ultrasonographic features of the carpal tunnel; however, there have been few studies on changes in the tunnel depending on physiological positions and on features of median nerve conduction. Therefore, we aimed to investigate ultrasonographic features of the carpal tunnel and electrophysiological changes in median n. conduction during various positions of the wrist.

MATERIALS AND METHODS

The study was performed on 35 healthy volunteers. The subjects gave oral informed consent. The exclusion criteria were problems with peripheral nervous system, fracture or the history of fracture in the forearm and the wrists, hereditary or acquired neuropathy, metabolic disorders such as diabetes mellitus, diseases of thyroid, gout, and collagen diseases such as rheumatoid arthritis, dialysis and pregnancy. All subjects had physical and neurological examinations.

Electrophysiological investigations were done by one physician using Nihon Kohden Neuropack II (MEB 7102K). The points on which electrodes were attached were determined and measurements of the distances between them were done on neutral position. Then, median nerve sensorimotor conduction were determined on the first finger of the right hand when the extremity was in the neutral position, flexion and extension. Electrophysiological examinations were carried out at the constant room temperature and at the extremity temperature of 34°C. Sensorial conductions were stimulated on the wrist and recorded on the first finger with a ring electrode and antidromic method, while motor conduction were stimulated on the wrist and elbow and recorded on the thanar region with superficial bar electrode.

Antero-posterior and medio-laterally diameters of the carpal tunnel were measured on ultrasonography during neutral position, flexion and extension. Ultrasonography was performed in all subjects by the same physician using superficial linear transducer (7.5-10 MHz, Hitachi EUB 450). Grey scale images on axial and longitudinal axes were examined on US when the wrist of the right hand was in neutral position, flexion and extension.

Statistical analyses were done with SPPS 10.0 package program. Descriptive statistics were performed. Data from measurements made on the wrist of the right hand on each position were compared with ANOVA. Tukey HSD was used for Post hoc tests.

RESULTS

The study included 35 healthy volunteers, of whom 24 were females and 11 males. The mean age of the females and males were 37.5 ± 2.4 years (range:19-4 years) and 42 ± 1.9 years (range:20-56 years) respectively. All subjects were right handed. Median nerve motor neuron amplitude and speed, median nerve sensorial distal latency and speed and antero-posterior and medio-laterally diameters of the carpal tunnel on neutral position, flexion and extension are shown in Table 1.

Figure 1

Table 1: Electrophysiological and ultrasonographic features of the motor and sensorial median nerves during neutral, flexion and extension positions of the wrist.

<table>
<thead>
<tr>
<th>Study</th>
<th>Position</th>
<th>Mean ± SD</th>
<th>P</th>
<th>Groups</th>
<th>t test</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median Nerve Motor</td>
<td>N</td>
<td>11.0 ± 2.7</td>
<td>0.0001</td>
<td>N, F</td>
<td>0.828</td>
<td>0.28</td>
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<tr>
<td>Amplitude</td>
<td>F</td>
<td>10.9 ± 3.4</td>
<td>0.0001</td>
<td>N, E</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>E</td>
<td>9.8 ± 2.2</td>
<td></td>
<td>E, F</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td>Median nerve motor</td>
<td>N</td>
<td>3.1 ± 0.3</td>
<td>0.155</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>distal latency</td>
<td>F</td>
<td>3.2 ± 0.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>E</td>
<td>3.1 ± 0.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median Motor Rate</td>
<td>N</td>
<td>69.7 ± 5.3</td>
<td>0.005</td>
<td>N, F</td>
<td>0.077</td>
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<tr>
<td></td>
<td>F</td>
<td>62.3 ± 6.8</td>
<td></td>
<td>N, E</td>
<td>0.148</td>
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<td></td>
<td>E</td>
<td>59.7 ± 2.2</td>
<td></td>
<td>E, F</td>
<td>0.002</td>
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<td>Median nerve sensorial</td>
<td>N</td>
<td>30.8 ± 1.2</td>
<td>0.127</td>
<td></td>
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<tr>
<td>amplitude</td>
<td>F</td>
<td>31.2 ± 2.4</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>E</td>
<td>32.5 ± 1.3</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Median nerve sensitive distal latency</td>
<td>N</td>
<td>2.4 ± 0.2</td>
<td>0.0001</td>
<td>N, F</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>2.2 ± 0.2</td>
<td></td>
<td>N, E</td>
<td>0.281</td>
<td></td>
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<tr>
<td></td>
<td>E</td>
<td>2.2 ± 0.2</td>
<td></td>
<td>E, F</td>
<td>0.0001</td>
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<tr>
<td>Median nerve sensitive rate</td>
<td>N</td>
<td>58.6 ± 5.3</td>
<td>0.0001</td>
<td>N, F</td>
<td>0.01</td>
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<tr>
<td></td>
<td>F</td>
<td>56.2 ± 5.3</td>
<td></td>
<td>N, E</td>
<td>0.638</td>
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<tr>
<td></td>
<td>E</td>
<td>56.4 ± 5.9</td>
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<td>E, F</td>
<td>0.63</td>
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<tr>
<td>Antero-posterior</td>
<td>N</td>
<td>4.0 ± 0.6</td>
<td>0.0001</td>
<td>N, F</td>
<td>0.0001</td>
<td></td>
</tr>
<tr>
<td>diameter of the</td>
<td>F</td>
<td>4.8 ± 0.7</td>
<td></td>
<td>N, E</td>
<td>0.145</td>
<td></td>
</tr>
<tr>
<td>carpal tunnel</td>
<td>E</td>
<td>5.0 ± 0.7</td>
<td></td>
<td>E, F</td>
<td>0.0001</td>
<td></td>
</tr>
</tbody>
</table>
| Median n. motor neuron amplitude decreased on flexion and extension (p<0.0001 and p< 0.0001), while median nerve motor neuron conduction speed decreased only on extension (p< 0.005). Median nerve sensorial conduction speed
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decreased (p<0.0001) and distal latency was prolonged (p<0.0001) on flexion. The medio-laterally diameter of the carpal tunnel increased on flexion and extension, but the anterio-posterior diameter decreased on flexion (p<0.0001).

DISCUSSION

It has been reported that carpal tunnel syndrome (CTS) is the most common nerve entrapment syndrome [13]. It appears in 50-100 of every 100,000 people and two and a half times as frequent in females as in males [17, 19]. It is characterized by paresthesia and pain in the first three fingers of the hands extending to the proximal part of the forearm and increasing in severity at nights. Presence of nocturnal pain which causes patients to wake up may be more sensitive than clinical tests (Phalen maneuver, Tinel sign) in the diagnosis of CTS [1].

The carpal tunnel is made of the wrist bones at the distal site and a tendon package of the forearm flexors above the wrist bones. The median n. is above the tendons. There is a thick cover which is not much flexible on the median nerve. It is called transverse carpal ligament [13]. Although the proximal and distal parts of the carpal tunnel are open, it is surrounded by strong ligaments and bony structures and therefore increased pressure inside the tunnel may have a negative effect on the median nerve. [10].

Damage to the median n. can be demonstrated by clinical findings of sensorial changes in the regions innervated by the median n., decreased conduction along the carpal tunnel, lengthened distal latency and decreased amplitude. The tunnel contents and the soft tissue around the tunnel can be examined on US, CT and MRI [13].

Fornage was the first to define peripheral nerves of the extremities on radiological images [13]. Callege et al used US to determine the anatomy of the carpal tunnel [13]. Buchberger et al examined changes in median n. on US in patients with CTS [17]. US is a dynamic examination. In fact, movements of the fingers and tendons and their effects on the median nerve can be imaged simultaneously. In addition, how flexion and extension of the wrists affect the median n. and the size and shape of the carpal tunnel can be viewed easily.

Most of the etiological factors cause edema in the carpal tunnel, which in turn leads to compression on the median nerve. The size and configuration of the median n., coiling of retinaculum and volume of the carpal tunnel all play an important part in CTS [17]. Several studies have revealed that the pressure in the carpal tunnel varied widely with various positions of the wrist. Nuzumlalı et al in their studies on healthy individuals and patients with CTS reported that the highest pressure was in dorsal flexion of 90° [1]. There have been studies to show that the medio-laterally diameter of the median n. increases in patients with CTS [13].

We observed a decrease in median n. motor amplitude on flexion and extension of the wrist and a prolongation in median nerve sensorineural distal latency and a decrease in sensorineural conduction speed on flexion. US showed that these changes were also accompanied by a decrease in the diameter of the carpal tunnel during flexion.

As to electrophysiological findings, changes in median nerve conduction were more significant on flexion than on extension and neutral position of the wrist. Lengthened latency in sensorineural conductions and in turn decreased conduction speed may be that sensorineural fibrils are exposed to more pressure on flexion.

The results of the present study lend support the idea that changes in positions of the joints cause a micro trauma, which in turn increases the likelihood of entrapment neuropathy in individuals with no metabolic, endocrine or collagen disorder. Therefore, we think that changes in positions of the joints can be an etiological factor in patients with nerve conduction pathologies. However, nerve conduction investigations, which are frequently used in the diagnosis of CTS, allow only evaluation of fibrils with thick myelin sheaths. As the sense of pain is conducted via nerve fibrils with thin myelin sheaths, electrophysiology may not help to diagnose the condition in its sub-clinical stage.

Electrophysiology is a golden method in the diagnosis of CTS in some professionals (tailors, carpenters, musicians etc.) exposed to the only risk of micro trauma due to overuse.
Electrophysiological stage in CTS. Ultrasound also reflects close correlations between the ultrasonographic findings and electrophysiological stage in CTS. Ultrasound might be a new diagnostic tool for disorders affecting the SSCT, especially carpal tunnel syndrome [10, 22]. At present, it is required that US, CT and MRI be used so that an accurate diagnosis can be made and an appropriate treatment alternative can be selected. However, the standards of these methods should be determined. This is one of the pioneering studies in determination of electrophysiological and ultrasonographic standards to be used in the diagnosis of CTS. US will help to differentiate CTS from anastomoses such as Martin-Gruber and Riche-Cannieu and congenital anomalies such as Struther's ligament. CT and MRI will also be useful for differential diagnosis of CTS from cervical cord injuries, cervical hernia nucleus pulposus, thalamic lesion, syringomyelia and cervical spinal cord injuries, all of which may give rise to similar symptoms to CTS, but result from etiological factors different from those of CTS [12, 17, 25].

The subsynovial connective tissue (SSCT) is the most characteristic structure in the carpal tunnel and is substantially affected in cases of carpal tunnel syndrome. Color Doppler imaging can identify and track SSCT motion separately from that of its associated tendons. Analysis of SSCT motion characteristics by color Doppler imaging may be useful for studying its function clinically [13].

Fibrosis of the SSCT is the most consistent pathological finding in patients with carpal tunnel syndrome. Ettema et al. investigated the anatomy and gliding characteristics of the flexor digitorum superficialis tendon and its adjacent SSCT with high-resolution ultrasound (15 MHz). Their hypotheses were that tendon and SSCT are distinguishable by ultrasound and that their velocities during tendon excursion are different. Qualitative ultrasound analysis of a flexor tendon and its SSCT was performed on five cadaver wrists and correlated to respective findings after anatomical study of the same cadavers. They concluded that noninvasive assessment of the thickness and velocity of the tenosynovium in carpal tunnel syndrome by high-resolution sonography might be a new diagnostic tool for disorders affecting the SSCT, especially carpal tunnel syndrome [23].

Finally, according to the study of Bayrak et al, there are close correlations between the ultrasonographic findings and electrophysiological stage in CTS. Ultrasound also reflects the reduction in the number of axons estimated by the motor unit number estimation method. Therefore, they concluded that the ultrasonographic findings reflect the severity of disease in patients with CTS [27].

As a result, it should be kept in mind that treatment outcome depends on whether a diagnosis is accurate and that a firm diagnosis can be made providing that an appropriate diagnostic tool is used.

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