International Journal of Physical Medicine & Rehabilitation

Nobuta et al., Int J Phys Med Rehabil 2018, 6:6
DOI: 10.4172/2329-9096.1000494

Research Article Open Access

Electrophysiological Features of Ulnar Tunnel Syndrome Caused by Ganglion – A Descriptive Study

Shingo Nobuta[1*], **Kazuaki Sonofuchi**[2] **and Eiji Itoi**[3]

[1]*Department of Orthopaedic Surgery, Tohoku Rosai Hospital, Sendai, Japan*

[2]*Goto Orthopaedic Clinic, Sendai, Japan*

[3]*Department of Orthopaedic Surgery, Tohoku University School of Medicine, Sendai, Japan*

***Corresponding author:** Shingo Nobuta, MD, PhD, Department of Orthopaedic Surgery, Tohoku Rosai Hospital 4-3-21 Dainohara, Aoba-ku, Sendai, Miyagi 981-8563, Japan, Tel: +81-22-275-1111; Fax: +81-22-275-7541; E mail : nobutays@jc5.so-net.ne.jp

Received date: October 31, 2018; **Accepted date:** November 20, 2018; **Published date:** November 22, 2018

Abstract

Objective: Ulnar tunnel syndrome (UTS) is an uncommon ulnar neuropathy. Clinical and electrophysiological diagnosis of UTS is difficult. The purposes of this study were to assess the diagnostic value of the nerve conduction measurements for UTS caused by ganglion, and to investigate the electrophysiological features of UTS.

Methods: The subjects were five patients with UTS. Before surgery, all patients had motor weakness and intrinsic muscle atrophy with positive Froment's sign, and three patients had numbness and hypesthesia in the ulnar nerve distribution. In all patients, a magnetic resonance imaging (MRI) of the wrist demonstrated soft tissue mass at the ulnar tunnel. The compound muscle action potential (CMAP) from the abductor digiti minimi (ADM) muscle and the first dorsal interosseous (FDI) muscle, and sensory nerve action potential (SNAP) from the little finger were recorded and analyzed. All patients underwent surgery of ulnar tunnel release and excision of the ganglion. Static 2 points discrimination tests (2PD) on the little finger, pinch strength were assessed before and after surgery.

Results: ADM-CMAP and FDI-CMAP were recorded in all five patients and all of them showed abnormality in ADM- and FDI-CMAP. Delayed latency (mean: 5.4 msec) and/or low amplitude (mean: 1.4 mV) were seen for ADM-CMAP and for FDI-CMAP (mean: 7.1 msec, 2.6 mV). SNAP was recorded in four patients and it all showed normal latency and amplitude. After surgery, all patients obtained complete recovery of motor function and sensation. Mean 2PD improved from 7.8 mm to 5.0 mm, and mean pinch strength increased from 1.8 kg to 4.8 kg postoperatively. Postoperative ADM-CMAP and FDI-CMAP showed the shortening of latency and the increase of amplitude, but they did not recover to the normal range.

Conclusion: Both ADM-CMAP and FDI-CMAP were important for definite electrophysiological diagnosis of ulnar tunnel syndrome caused by ganglion. Residual delayed latency and low amplitude were seen after surgery regardless of complete recovery of intrinsic muscle.

Keywords: Ulnar tunnel syndrome; Ganglion; Electrophysiology; Abductor digiti minimi; First dorsal interosseous

Introduction

Ulnar tunnel syndrome (UTS) is an uncommon ulnar entrapment neuropathy. Guyon [1] described the anatomy of the area in 1861, and Hunt [2] reported three patients of neuritis of the deep palmar branch of the ulnar nerve. Seddon [3] described ulnar nerve palsy due to carpal ganglion, and Richmond [4] reported four cases of ulnar nerve compression caused by a carpal ganglion. In 1965, Dupont et al. [5] described the term ulnar tunnel syndrome and reported four cases. UTS can be caused by a variety of factors, and many previous reports described that one of the most common causes of UTS was a ganglion [3-10,13,15,16]. Magnetic resonance imaging (MRI) provides valuable information for UTS caused by ganglion [15,17]. However, the exact clinical diagnosis is not always easy, and electrophysiological diagnosis is essential to localize the lesion [11-13,15-21]. A few electrophysiological studies for UTS caused by ganglion have been reported [11-17], and they described the delayed conduction at the wrist. Furthermore, there are few electrophysiological reports for UTS caused by ganglion examined before and after surgery [11,15]. The purposes of this study were to assess the diagnostic value of the nerve conduction measurements for UTS caused by ganglion, and to investigate the elctrophysiological features of UTS by the data examined before and after surgery.

Patients and Methods

Between December 2010 and July 2016, five upper limbs from five patients with UTS caused by ganglion were reviewed after a mean follow-up of 8 months (range 4-12). Details of five cases are shown in Table 1. The ages of the patients at surgery ranged from 45 to 66 years, with a mean age of 55 years. There were four females and one male.

								FollowUp		
Case	Age	Gender	side	Duration of symptoms	2PD mm	Pinch kg	Zone (size) of ganglion	Mo	2PD	Pinch
1	54	F	R	1 m	10	3.2	1 (10/12/10)	12	5	4.6
2	45	F	R	5 m	12	2	1 (22/14/10)	4	5	4
3	56	F	L	3 m	7	0.5	1 (7/7/6)	7	5	3.6
4	66	F	L	2 m	5	1.3	2 (8/7/5)	10	5	4.2
5	56	M	R	1 m	5	2.1	1 (8/8/11)	6	5	7.4

Table 1: Details of five cases of ulnar tunnel syndrome caused by ganglion (F: Female; M: Male; R: Right; L: Left; m: Month(s); 2PD: 2 Points Discrimination Test on Little Finger; Pinch: Pulp Pinch Strength; Zone: Site of Compression; Size: in coronal (length/width/) and axial (thickness) MRI (mm); Mo: Months).

The affected side was right in three and left in two, the dominant extremity was involved in three. Mean duration of symptoms was 2.4 months (range 1-5). Written informed consent was obtained from each patient. UTS caused by ganglion was diagnosed based on clinical symptoms, MRI findings and electrophysiological evaluation. All patients had motor weakness and intrinsic muscle atrophy with positive Froment's sign, and three patients had numbness and hypesthesia in the ulnar nerve distribution. Intrinsic muscles include the abductor digiti minimi (ADM) muscle and the first dorsal interosseous (FDI) muscle. Tinel's sign at the ulnar tunnel was not seen in any of these patients. Static 2 points discrimination test (2PD) on the little finger ranged from 5 to 12 mm, with a mean of 7.8 mm (SD: standard deviation 3.1). Pulp pinch strength revealed from 0.5 to 3.2 kg, with a mean strength of 1.8 kg (SD: 1.0). In all patients, MRI of the wrist demonstrated soft tissue mass at the ulnar tunnel with the size varied from 5 to 22 mm (Table 1).

Electrophysiological evaluation included motor and sensory nerve conduction measurements were performed before and after surgery. The compound muscle action potential (CMAP) from ADM and FDI, and sensory nerve action potential (SNAP) from the little finger were recorded and analyzed. Observation was made using a Nicolet Viking electromyography system (Nicolet instruments, Madison, WI, USA). Palmar skin temperatures were not allowed to fall below 32. ADM- and FDI-CMAP were recorded with surface electrodes by supramaximal stimulation of the ulnar nerve at the wrist (stimulus duration 0.1-0.2 ms: milliseconds), and SNAP was recorded with ring electrodes by minimal stimulation of the ulnar nerve at the wrist. By the measurement of twenty healthy subjects, mean 2SD indicate the normal value of the latency and the amplitude in our institute. Normal values were ADM-CMAP latency<2.9 ms, amplitude>5.3 mV (millivolts), FDI-CMAP latency<4.2 ms, amplitude>6.6 mV, SNAP peak latency<3.5 ms, and amplitude>3.4 μV (microvolts). According to these criteria, we found delayed latency and low amplitude for ADM-, FDI-CMAP and SNAP. Furthermore, to rule out cubital tunnel syndrome, ADM- and FDI-CMAP were recorded by stimulating the ulnar nerve at the elbow with no conduction delay at the cubital tunnel.

All patients underwent surgery of ulnar tunnel release and excision of a ganglion. At surgery, we identified the site of compression by a ganglion within the ulnar tunnel in each case, and classified them into three zone [10]. Zone 1 is the area proximal to the bifurcation of the ulnar nerve, Zone 2 compress the motor branch of the nerve after it has bifurcated, and Zone 3 carries the superficial or sensory branch of the ulnar nerve. Static 2 points discrimination (2PD) test on the little finger, pinch strength and Froment's sign were assessed after surgery. Complications including hematoma, infection and nerve injury were also examined. The data were analyzed with the Student's t-test and the Mann-Whitney U test. A p-value less than 0.05 was considered statistically significant.

Results

Before surgery, ADM-CMAP and FDI-CMAP were recorded in all five patients and they all showed abnormality in ADM- and FDI-CMAP. Delayed latency (mean: 5.4 ms, SD: 3.8) and or low amplitude (mean: 1.4mV, SD: 1.4) were seen for ADM-CMAP and for FDI-CMAP (mean: 7.1 ms, SD: 3.2, mean: 2.6 mV, SD: 4.6). Specifically, case 2 with a large ganglion showed largely delayed latency (11.8 ms) and very low amplitude of ADM-CMAP (0.2 mV) and FDI-CMAP (12.6 ms, 0.1 mV). SNAP were recorded in four patients and they all showed normal latency and amplitude (Table 2).

At surgery, after releasing the ulnar tunnel by division of the volar carpal ligament, compression in Zone 1 was seen in four patients, in which a ganglion was compressing the motor branch (to ADM, FDI) and the sensory branch of the ulnar nerve proximal to the fibrous arch, and Zone 2 was in one, in which a ganglion was mainly compressing the motor branch (to FDI) at the fibrous arch (Table 1). There was no case in Zone 3. A ganglion rose from the triquetrohamate joint in all patients, and fibrous arch was also released. The ganglion was traced to its origin and excised. Histologic examination confirmed the diagnosis of ganglion in all patients. There were no complications including hematoma, infection and nerve injury.

				Parameter measurements before / after surgery		
				ADM-CMAP		SNAP

Case	Age	Gender	side	Lat.	Amp.	Lat.	Amp.	Lat.	Amp.
1	54	F	R	6.0/4.1	2.1/2.0	5.6/5.0	10.7/10.3	2.5/1.7	25/40
2	45	F	R	11.8/3.9	0.2/4.4	12.6/5.3	0.1/4.0	2.6/2.5	10/12
3	56	F	L	3.1/3.1	3.6/5.6	6.5/4.9	0.6/6.8	N.D	
4	66	F	L	3.5/2.9	0.5/1.2	4.4/3.7	0.1/0.9	2.0/1.9	8/12
5	56	M	R	2.7/2.6	0.6/6.6	6.4/4.2	1.5/12.2	2.6/2.6	20/25

Table 2: Details of electrophysiological data of five cases (ADM: Abductor Digiti Minimi; CMAP: Compound Muscle Action Potential; FDI: First Dorsal Interosseous; Lat.: Latency (msec); Amp.: Amplitude (mV,μV); SNAP: Sensory Nerve Action Potential; N.D.: Not Done; _: Normal range).

After surgery, all patients obtained complete recovery of motor function and sensation. Mean 2PD improved from 7.8 mm to 5.0 mm (SD: 0, no significance), and mean pinch strength increased from 1.8 kg to 4.8 kg (SD: 1.5, $p<0.02$), with negative Froment's sign. Postoperative ADM- and FDI-CMAP showed the shortening of latency (mean: ADM- 3.3 ms, SD: 0.6, FDI- 4.6 ms, SD: 0.6, no significance) and the increase of amplitude (mean, ADM- 4.0 mV, SD: 2.3, FDI- 4.7 mV, SD: 4.9, no significance). However, they did not recover to the normal range, except ADM-latency in one, ADM-amplitude in two, FDI-latency in one, and FDI-amplitude in three (Table 2).

Illustrative case

A-56-year old right hand dominant male presented with one month history of onset and intrinsic weakness of his right hand (case 5). Right hand showed intrinsic muscle atrophy and weakness with positive Froment's sign, and clawing of the little finger, without sensory loss. 2PD was 5 mm, and pinch strength 2.1 kg. Axial T1 weighted MRI showed cystic mass lesion at the ulnar tunnel (Figure 1).

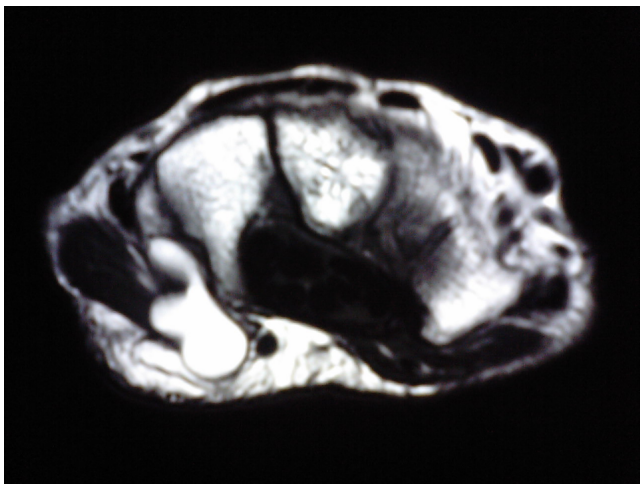

Figure 1: Axial T1 weighted MRI showed cystic mass lesion at the ulnar tunnel (arrow) in case 5.

ADM-CMAP revealed normal latency (2.7 ms) and low amplitude (0.6 mV) (Figure 2A), and FDI-CMAP showed delayed latency (6.4 ms) and low amplitude (1.5 mV) (Figure 3A), nevertheless SNAP indicated normal latency and amplitude. Intra-operatively a 8/8/11 mm ganglion was mainly compressing the motor branch of the ulnar nerve in Zone 1 (Figure 4). The ganglion was excised and histology confirmed diagnosis. Six months after surgery, complete recovery of motor function was obtained with pinch strength 7.4 kg, ADM- and FDI-CMAP showed the shortening of latency and the increase of amplitude (Figure 2B and Figure 3B).

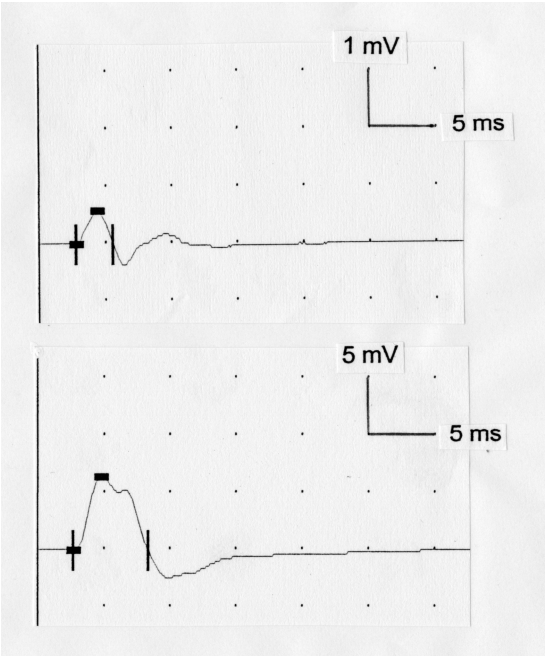

Figure 2: A. ADM-CMAP latency was 2.7 ms and amplitude 0.6 mV. B. Six months after surgery, latency was 2.6 ms and amplitude 6.6 mV.

Discussion

Ulnar tunnel syndrome (UTS) is distinctly uncommon ulnar entrapment neuropathy. UTS can be caused by a variety of intrinsic and extrinsic factors [7,11,14,17], and the literature reported that a ganglion was one of the most common causes of UTS [3-10,13,15,16]. MRI provides valuable information for UTS caused by ganglion [15,17]. However, the exact clinical diagnosis and the location of lesion can be difficult, and electrophysiological diagnosis may help to confirm the diagnosis and to localize the level of neuropathy [11-13, 15-21].

Electrodiagnostic data should include conduction velocity across the elbow as well as the wrist to rule out cubital tunnel syndrome [17], therefore, we performed the nerve conduction measurements including ADM-CMAP, FDI-CMAP and SNAP to rule out the cubital tunnel syndrome and to confirm the diagnosis of ulnar tunnel syndrome.

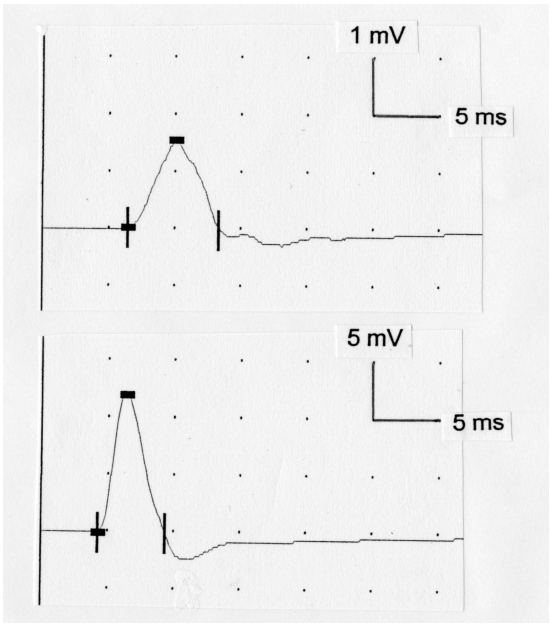

Figure 3: A. FDI-CMAP latency was 6.4 ms and amplitude 1.5 mV. B. Six months after surgery, latency wazs 4.2 ms and amplitude 12.2 mV.

Anatomically, ulnar tunnel is classified into three zone [10]. Zone 1 is the area proximal to the bifurcation of the ulnar nerve Zone 2 affects the motor branch (except the branch to ADM) of the ulnar nerve after it has bifurcated, and Zone 3 compress the superficial or sensory branch of the ulnar nerve. Depending on the site of compression the entrapment may be motor, sensory or mixed clinically [16]. In our series, Zone 1 was in four cases and Zone 2 was in one, however, case 5 with Zone 1 showed no sensory loss.

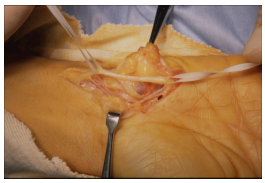

Figure 4: Intraoperative photograph of a ganglion which arising from triquetrohamate joint, mainly compressing the motor branch of the ulnar nerve (arrow).

According to the anatomical Zones, ADM-CMAP, FDI-CMAP and SNAP indicate following data theoretically. In Zone 1 compression, ADM-latency and FDI-latency are delayed and SNAP amplitude are diminished, in Zone 2 lesion, FDI-latency is delayed, according to the site of compression, ADM-latency may be normal, in Zone 3 compression, SNAP amplitude are diminished and ADM- , FDI-CMAP are normal [17]. In our series of five cases, four cases of Zone 1 showed delayed ADM-latency in three, low ADM-amplitude in four, delayed FDI-latency in four, low FDI-amplitude in three, and SNAP was normal in three. Case 4 with Zone 2 lesion indicated delayed latency and low amplitude in ADM- and FDI-CMAP, and normal SNAP (Table 2). Therefore, both ADM- and FDI-CMAP were important for definite diagnosis of UTS and SNAP was not so valuable to confirm the diagnosis. On the other hand, there have been several reports concerning the short segment incremental study (SSIS, inching method) of FDI-CMAP [18-21], and described that SSIS was valuable for diagnosis of precise localization of UTS. However, SSIS was somewhat time-consuming and technically difficult, particularly palmar sites of stimulation [20], therefore we performed traditional nerve conduction measurements of FDI-CMAP and ADM-CMAP.

A few electrophysiological studies for UTS caused by ganglion have been reported [11-16], and they described the delayed conduction at the wrist. Furthermore, there are few electrophysiological reports for UTS caused by ganglion examined before and after surgery [11,15]. Ebeling et al. [11] reported one case with a ganglion among nine cases of UTS, and described that ADM-latency shortened from 4.4 ms to 2.9 ms, and FDI- latency shortened from 20.5 ms to 4.4 ms postoperatively. Erkin et al. [15] reported a case with a ganglion, and stated that FDI-latency shotened from 3.5 ms to 3.2 ms (normal value<3.2), and FDI-amplitude increased from 2.1 mV to 5.4 mV (normal value>6.0) postoperatively. In our series, ADM- and FDI-CMAP did not recover to the normal range postoperatively, except ADM-latency in one, ADM-amplitude in two, FDI-latency in one, and FDI-amplitude in three (Table 2). Regardless of complete recovery of intrinsic muscle after surgery, residual delayed latency and low amplitude were seen. In these cases, to explain neurophysiologically, myelinization and axonal regeneration of fibers in ADM and FDI branch would not be sufficient after a mean follow-up of 8 months.

There were several limitations with this study. First, number of subjects was small (five patients). Second, we could not clear the relationship between the size of ganglion and the electrophysiological data or the recovery time for intrinsic muscles. Third, mean follow-up period of 8 months was not so long, therefore, long-term follow-up would be needed to detect the recovery of ADM-CMAP and FDI-CMAP.

Conclusion

Both ADM-CMAP and FDI-CMAP were important for definite electrophysiological diagnosis of ulnar tunnel syndrome caused by ganglion. Residual delayed latency and low amplitude were seen after surgery.

Acknowledgement

The authors wish to thank Ms. Yumi Watabe and Ms. Hiromi Takeda, Tohoku Rosai Hospital, for assisting in the manuscript preparation.

References

1. Guyon F (1861) Note sur une disposition anatomique propre a la face anterieure de la region du poignet et non encore decrite. Bull Soc Anat Paris 6: 184-186.
2. Hunt JR (1908) Occupation neuritis of the deep palmar branch of the ulnar nerve. J Nerv Ment Dis 35: 676

3. Seddon HJ (1952) Carpal ganglion as a cause of paralysis of the deep branch of the ulnar nerve. J Bone Joint Surg Br 34: 386-390.
4. Richmond DA (1963) Carpal ganglion with ulnar nerve compression. J Bone Joint Surg Br 45: 513-515.
5. Dupont C, Cloutier GE, Prevost Y, Dion MA (1965) Ulnar-tunnel syndrome at the wrist. J Bone Joint Surg Am 47: 757-761.
6. Vanderpool DW, Chalmers J, Lamb DW, Whiston TB (1968) Peripheral compression lesions of the ulnar nerve. J Bone Joint Surg Br 50: 792-803.
7. Shea JD, McClain EJ (1969) Ulnar-nerve compression syndromes at and below the wrist. J Bone Joint Surg Am 51: 1095-1103.
8. Hayes JR, Mulholland RC, O'Connor BT (1969) Compression of the deep palmar branch of the ulnar nerve. Case report and anatomical study. J Bone Joint Surg Br 51: 469-472.
9. Uriburu IJ, Morchio FJ, Marin JC (1976) Compression syndrome of the deep motor branch of the ulnar nerve. (Piso-hamate hiatus syndrome). J Bone Joint Surg Am 58: 145-147.
10. Gross MS, Gelberman RH (1985) The anatomy of the distal ulnar tunnel. Clin Orthop 196: 238-247.
11. Ebeling P, Gilliatt RW, Thomas PK (1960) A clinical and electrical study of ulnar nerve lesions in the hand. J Neurol Neurosurg Psychiat 23: 1-9.
12. Kleinert HE, Hayes JE (1971) The ulnar tunnel syndrome. Plast Reconstr Surg 47: 21-24.
13. Kuschner SH, Gelberman RH, Jennings C (1988) Ulnar nerve compression at the wrist. J Hand Surg Am 13: 577-580.
14. Murata K, Shih JT, Tsai TM (2003) Causes of ulnar tunnel syndrome: a retrospective study of 31 subjects. J Hand Surg Am 28: 647-651.
15. Erkin G, Uysal H, Kele IL, Aybay C, Ozel S (2006) Acute ulnar neuropathy at the wrist. A case report and review of the literature. Rheumatol Int 27: 191-196.
16. Inaparthy PK, Anwar F, Botchu R, Jahnich H, Katchburian MV (2008) Compression of the deep branch of the ulnar nerve in Guyon's canal by a ganglion: two cases. Arch Orthop Trauma Surg 128: 641-643.
17. Chen SH, Tsau TM (2014) Ulnar tunnel syndrome. J Hand Surg Am 39:571-579.
18. Hatori M, Sakurai M, Miyasaka Y, Nobuta S (1989) Electrodiagnosis of ulnar tunnel syndrome by inching technique. J Jpn Soc Surg Hand 6:346-350.
19. McIntosh KA, Preston DC, Logigian EL (1998) Short-segment incremental studies to localize ulnar nerve entrapment at the wrist. Neurology 50: 303-306.
20. Cowdery SR, Preston DC, Herrmann DN, Logigian EL (2002) Electrodiagnosis of ulnar neuropathy at the wrist: conduction block versus traditional tests. Neurology 59: 420-427.
21. Seror P (2013) Electrophysiological pattern of 53 cases of ulnar nerve lesion at the wrist. Neurophysiol Clin 43: 95-103.