Acta Neurol. Belg., 2010, **110**, 78-83

Short segment incremental study in ulnar neuropathy at the wrist: report of three cases and review of the literature

Pınar YALINAY DIKMEN[1], A. Emre ÖGE[2] and Jale YAZICI[2]

[1]Department of Neurology, Acıbadem University, Faculty of Medicine, Istanbul, Turkey; [2]Department of Neurology, Istanbul University, Istanbul Faculty of Medicine, Çapa, Istanbul, Turkey

Abstract

Ulnar nerve lesions may occur at different sublocations at wrist and may involve various branches of the nerve. Standard neurophysiological studies are generally insufficient in revealing these lesions. Demonstration of conduction block and/or focal slowing of nerve conduction is the most definitive electrodiagnostic evidence for the localization of segmental demyelination. Short-segment incremental study (SSIS) is a sensitive technique for detecting the ulnar neuropathy at the wrist (UNW).

We report 3 cases of UNW caused by ganglion cysts in Guyon's canal which were studied by using SSIS across the wrist. Even though SSIS is a time-consuming and technically demanding method, it increases the electrodiagnostic potential of detecting segmental demyelination in this location.

Key words: Short-segment incremental study; ulnar neuropathy at the wrist; ulnar neuropathy; inching; Guyon's canal.

Introduction

Ulnar neuropathy at the wrist (UNW) which was first reported by Hunt in 1908, is an uncommon entrapment neuropathy (1, 2). It might be difficult to localize the UNW by standard neurophysiological studies because of variable involvement of the different branches of the ulnar nerve (3). If sensory complaints are absent, the diagnosis may be more difficult (4). It is frequently confused with ulnar neuropathy at the elbow (UNE) or motor neuron disease (5).

UNW has five major clinical and electrodiagnostic presentations (6, 7). The neurophysiological studies should be planned to detect the involved branches and the nature of the lesion. SSIS is a sensitive method for verifying UNW by localizing the segmentally demyelinating lesion with the demonstration of focal slowing and conduction block (8).

Here, we report 3 cases of UNW, which were evaluated by SSIS across the wrist, and review relevant literature.

Methods

All nerve conduction studies (NCS) were performed with surface stimulation (by using stimulators with inter-electrode distances of 2.5 cm) and recording. Surface recording electrodes were placed according to belly-tendon technique for motor NCS. Orthodromic sensory NCS were performed by stimulating the fingers with ring electrodes and recording at the wrist. Concentric electrodes were used for needle electromyography.

In SSIS study, performed while recording from first dorsal interosseus muscle (FDI), one-centimeter increments were marked from 2-3 cm above and 3-4 cm below to the the distal wrist crease (DWC). Supramaximal stimulations of the ulnar nerve was made at each consecutive incremental point by careful localization of the nerve beneath the stimulator and by avoiding the distal spread of the depolarisation caused by superfluous stimulation intensity and durations (usually $\leq$ 0.1 ms durations were used).

Cases

Case 1 was a 58-year-old right-handed female teacher who presented with a 1.5 month history of progressive weakness and clumsiness in her left hand and fingers. There was a mild weakness of the abduction of the second and fifth fingers on the left side. FDI was moderately atrophied. Sensory examination was normal.

Table 1 summarizes the NCS. Low amplitude compound muscle action potentials (CMAP) with

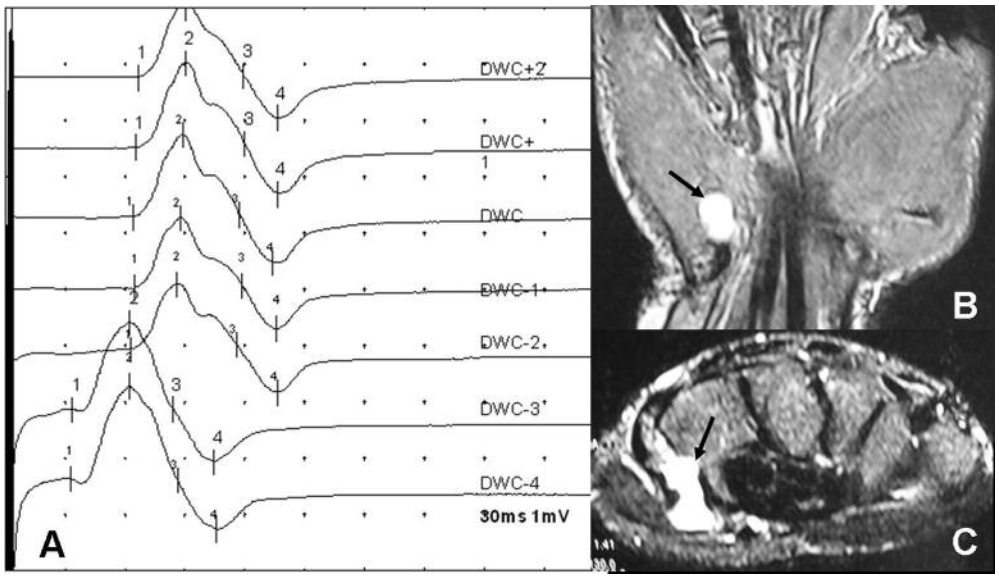

FIG. 1. — A) Case 1. Compound muscle action potentials (CMAPs) recorded from the first dorsal interosseus muscle with stimulation at 1 cm incremental points between 2 cm above and 4 cm below to the the distal wrist crease (DWC). Note the focal prolongation of the latency alongwith a CMAP amplitude drop between the 2 and 3 cm points distally to the DWC. Coronal (B) and transversal (C) MRI studies reveal the cystic mass lesion (arrows).

prolonged latencies were recorded from adductor digiti minimi (ADM) and FDI muscles with stimulation of the ulnar nerve at left wrist. CMAPs elicited by stimulation at the wrist (recorded from the FDI) had markedly reduced amplitudes (more than 50%) as compared to those elicited by palm stimulation, signifying a conduction block between these stimulation points. Wrist-to-palm nerve conduction velocity (NCV) was calculated as 19.5 m/s.

In SSIS, a focal prolongation of the latency along with a CMAP amplitude drop were encountered between the 2 and 3 cm points distally to the DWC (Fig. 1).

Needle EMG showed fibrillation potentials and positive sharp waves with mild to moderate increased amount of large-amplitude, long-duration, polyphasic Motor Unite Action Potentials (MUAPs) with decreased recruitment in the left ADM, FDI, fourth lumbrical and third volar interossei.

According to the classification of UNW by Wu *et al.*, lesion localization was categorized as type 3 (6).

A magnetic resonance imaging (MRI) of the wrist demonstrated a cystic mass with well defined margins (ganglion cyst) compressing the ulnar nerve, with its greater diameters nearly corresponding to the location in SSIS (Fig. 1).

Case 2 was a 51-year-old right-handed housewife who presented with a 3 –week history of progressive left hand weakness, pain at the palm and numbness in digits 4 and 5. On her neurological examination there was weakness of adduction and abduction of the digits 4 and 5. Sensory examination did not reveal an objective sensory loss.

Ulnar sensory nerve action potential (SNAP) was low in amplitude on the left as compared to the other side (6 mV vs 12 mV) (Table 1). The ulnar motor NCS study revealed low amplitude-prolonged latency CMAPs recorded from ADM and FDI with wrist stimulation.

In SSIS, a clear prolongation of the CMAP latency difference was observed between the stimulation point at DWC and the one 1 cm proximal to it (Fig. 2). Although it seems that there was an amplitude drop at the same location, it could not be interpreted as a partial conduction block due to the low amplitude CMAP with distal stimulation (Table 1).

Needle EMG showed fibrillation potentials and positive sharp waves with moderate amount of large-amplitude, long-duration, polyphasic MUAPs with decreased recruitment in the left ADM and FDI.

According to the classification of UNW, the lesion of the patient was categorized as a type 1 lesion (6).

On the second examination performed 1 month later, left FDI was weaker with moderate atrophy. Sensation was diminished on palmar ulnar aspect of the hand and in digits 4 and 5.

The MRI of the wrist demonstrated a well defined cystic mass, consistent with a ganglion cyst, compressing the ulnar nerve (Fig. 2).

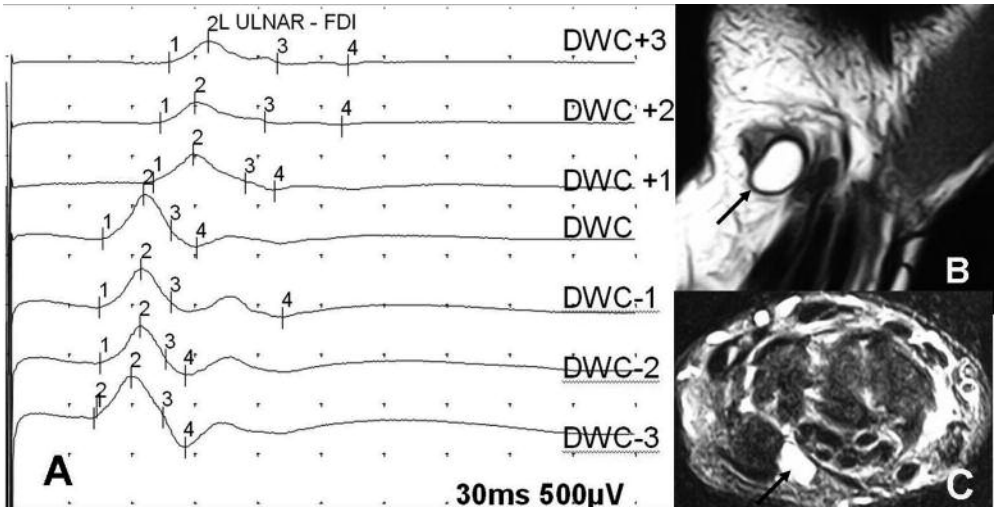

FIG. 2. — Short-segment incremental study in Case 2. A clear prolongation of the compound muscle action potential latency difference was observed between the distal wrist crease and the stimulation point 1 cm proximal to it (A). Coronal (B) and transversal (C) MRI studies show the cystic mass (arrows).

Case 3 was a 40-year-old right-handed housewife who presented with a 2 weeks history of progressive left hand weakness and numbness in digits 4 and 5. On her neurological examination there were adduction and abduction weakness of the digits and reduced sensation on the ulnar side of the palm and digits 4 and 5.

The ulnar SNAP was markedly low in amplitude on the involved left side (5 mV vs 21 mV on the contralateral side). In ulnar motor NCS, low amplitude-prolonged latency CMAPs were recorded from ADM and FDI muscles. SSIS performed with recordings from FDI showed a manifest prolongation of distal latency with a mild amplitude loss between the stimulation points at DWC and 1 cm proximal to it (Fig. 3).

Needle EMG showed fibrillation potentials and positive sharp waves with mildly increased large-amplitude, long-duration, polyphasic MUAPs with decreased recruitment in the left ADM, FDI and adductor pollicis muscles.

According to the classification of UNW the lesion localized as type 1 lesion (6).

The MRI of the wrist revealed findings of a well demarcated cystic lesion, (ganglion cyst), compressing on the ulnar nerve (Fig. 3).

SUMMARY OF THE SSIS FINDINGS

In our 3 patients, focal latency changes caused by 1 centimeter increments in the proximal and distal intact segments of the ulnar nerves and those studied in 1 patient asymptomatic side were between 0.10 and 0.40 ms (mean ± 0.25 ms). Whereas, the latency changes at the lesion sites were found to be very conspicuous (2.90, 2.35 and 4.5 ms) and were located in good accordance with the cystic lesions in MRI studies. In Case 1, there was also a considerable amount of CMAP amplitude drop at the lesion site, implying the presence of a partial conduction block (Fig. 1).

Discussion

In 1861, Felix Guyon gave a description of a thight oblique canal at the wrist containing the ulnar nerve, artery and vein with some fatty tissue (9). Gross and Gelbberman dissected 40 cadaver limbs to better identify the anatomy of this canal (10). Guyon's canal is 4 to 4.5 cm long and located between the pisiform bone and the hook of hamate. Its floor is formed by combination of the thick transverse carpal and pisohamate ligaments. The roof is made up by volar carpal ligament and palmaris brevis muscle.

In Guyon's canal, the ulnar nerve divides into the deep palmar motor branch and the superficial sensory branch. Before exiting through the pisohamate hiatus, the deep motor branch provides motor innervation to the four hypothenar muscles (ADM, flexor digiti minimi, oppens digiti minimi and palmaris brevis). After the hiatus, the superficial sensory branch supplies sensory innervations to the fifth and medial side of the fourth digit, and the deep motor branch provides motor innervation to the other ulnar intrinsic hand muscles including dorsal interossei,

Table 1

Motor and sensory nerve conduction studies (excluding SSIS) in the three patients

	Nerve	Stimulation	Recording	Latency (ms)	Amplitude (mV/μV)	NCV (m/sn)	Normal Latency	Normal Amplitude	Normal NCV
Patient	1								
	L Median	Wrist	APB	2.95	10.3		≤ 4.4	≥ 4	≥ 49
		Elbow		6.80	10.3	59.7			
	L Ulnar	Wrist	ADM	5.55	0.4		≤ 3.3	≥ 6	≥ 49
		BE		8.15	0.3	76.9			≥ 49
		AE		9.95	0.3	61.1			
	L Ulnar	Palm	FDI	2.75	**3.1**				
		Wrist		**6.85**	**1.6**	**19.5**	≤ 4.5	≥ 7	≥ 50
		BE		10.55	1.5	56.8			≥ 50
		AE		12.0	1.3	75.9			
	L Median	III finger	Wrist	2.10	29.5	54.0	≤ 3.5	≥ 20	≥ 50
	L Ulnar	V finger	Wrist	2.95	19.8	50.0	≤ 3.1	≥ 17	≥ 50
Patient	2								
	L Median	Wrist	APB	2.65	8.10		≤ 4.4	≥ 4	≥ 49
		AF		6.60	7.70	57			
	L Ulnar	Wrist	ADM	**4.05**	**0.2**		≤ 3.3	≥ 6	≥ 49
		BE		7.85	0.3	52.6			≥ 49
		AE		9.90	0.3	53.7			
	L Ulnar	Palm	FDI	3.85	0.8				
		Wrist		**6.80**	**0.2**		≤ 4.5	≥ 7	≥ 50
		BE		10.80	0.2	52.5			≥ 50
		AE		12.55	0.2	62.9			
	L Median	III finger	Wrist	2.0	18.5	62.5	≤ 3.5	≥ 20	≥ 50
	L Ulnar	Vfinger	Wrist	2.85	**6.3**	**33.5**	≤ 3.1	≥ 17	≥ 50
Patient	3								
	L Median	Wrist	APB	3.40	10.5		≤ 4.4	≥ 4	≥ 49
		AF		7.45	10.3	55.6			
	L Ulnar	Wrist	ADM	8,05	1,8		≤ 3.3	≥ 6	≥ 49
		BE		12,05	1,7	52,6			≥ 49
		AE		13,95	1,6	52,5			
	L Ulnar	Wrist	FDI	**8,90**	**2,5**		≤ 4.5	≥ 7	≥ 50
		BE		11,90	2,4	50.0			≥ 50
		AE		14,30	2,2	50.0			
	L Median	III finger	Wrist	2.40	33.4	50.0	≤ 3.5	≥ 20	≥ 50
	L Ulnar	V finger	Wrist	2.0	**5.0**	**45**	≤ 3.1	≥ 17	≥ 50

L: Left, APB: Abductor pollicis brevis, BE: Below elbow, AE: Above elbow, ADM: Adductor digiti minimi, DWC: Distal wrist crease. Values of the inching studies are presented in the relevant figures.

the third and the fourth lumbricals, adductor pollicis and FDI (11).

UNW is obviously an uncommon entrapment neuropathy compared to the carpal tunnel syndrome and the ulnar neuropathy at the elbow (UNE). Patients usually complain about sensory and motor symptoms of ulnar nerve damage. Since the dorsal cutaneous branch arises from the nerve in the distal forearm, the dorsal medial aspect of hand is spared (12). On inspection, the hand intrinsic muscles may be wasted. Tinel's sign may present over Guyon's canal.

Many different etiological factors have been reported to be associated with UNW. According to the literature review by Shea and Mc Lain (13) the most frequent cause is ganglion cysts (28.7%). Twenty three percent of cases are caused by recurrent blunt trauma, 23% by single acute traumatic events, 10% by other surgically removable lesions, and 8.1% by vasculitic disorders. Among other reported causes are fibrous bands, bicycle riding and other sources of chronic external compression, playing video game, anomalous muscles and infarction.

According to the classification established by Wu *et al.*, there are five types of UNW in relation to lesion locations and the subsequent clinical presentations (6) (Fig. 4). Type 1 lesion is just outside or in the proximal end of Guyon's canal. It refers to a mixed motor and sensory neuropathy. Type 2 lesion is limited to the superficial sensory branch and consequently is a pure sensory neuropathy. Type 3 is a pure motor neuropathy caused by the lesion of the

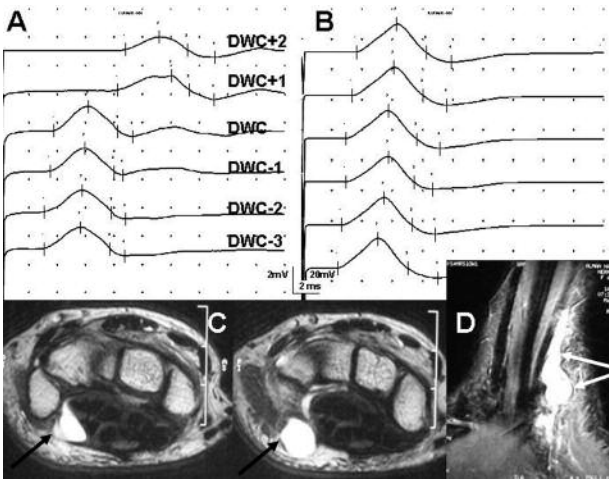

FIG. 3. — Case 3. Clearly increased latency difference between the stimulation points at the distal wrist crease and 1 cm proximally (A). B shows the same Short-segment incremental study (SSIS) perfomed on the contralateral side. Transversal (C) and coronal (D) MRI studies show the cystic lesion which reached its greatest diameter nearly at the same location as SSIS found the focal latency prolongation (arrows).

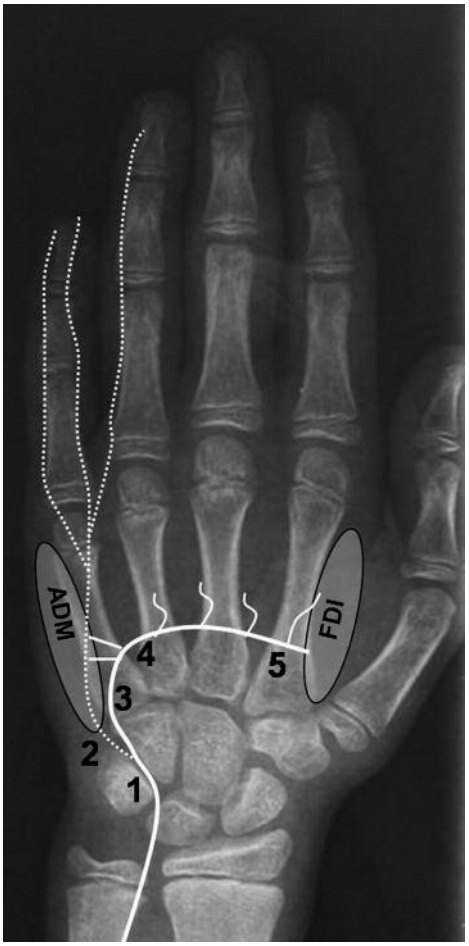

FIG. 4. — Classification of lesion localizations according to Wu *et al.* Refer to the paragraph 5 of Discussion.

deep branch just distal to the separation of the superficial branch but proximal to the branch for hypothenar muscles. Type 4 is also a pure motor neuropathy with sparing of hypothenar muscles with the lesion distal to the origin of both the superficial branch and the branch going to the hypothenar muscles. Type 5 is a motor neuropathy limited to the most distal muscles, FDI and adductor pollicis. According to Wu *et al.*, the most common UNW sites are type 1, type 3, and type 4, respectively (6). Type 1 and type 3 are also the most common patterns according to Cowdey *et al.* (14).

A review of the literature reveals that the majority of reports on UNW are found in the journals of orthopaedic and plastic surgery and generally consist of small number of cases. There is only one remarkably large electrophysiological study which includes 20 consecutive patients with clinically defined UNW and 30 asymptomatic normal control subjects (14). In this study, the authors prospectively compared 4 electrophysiological methods: 1&2) ulnar motor study recording FDI, stimulation at the palm and wrist, looking for conduction block and conduction slowing across the wrist ; 3) recording from lumbrical and interossei, comparing ulnar versus median distal latencies; 4) ulnar motor studies recording from FDI and ADM, comparing their respective distal latencies. In five patients, SSIS across the wrist were done. Slow wrist-palm FDI conduction velocity (< 37 m/s) was found in 16 UNW patients (80%), definite or probable conduction block in 14 (70%), prolonged DL to FDI (> 4.5 milliseconds) in 12 (60%), and to ADM in 11 (55%) and an abnormal lumbrical-interossei latency difference in 12 (60%). They concluded that, only the CB and slow wrist-palm conduction velocity, recorded from FDI, were specific for UNW. Of the 5 patients in whom SSIS studies were performed, all demonstrated definite CB.

In patients suspected to have UNW, ulnar NCS with recording from FDI must be performed as well as the more conventional ulnar NCS with hypothenar recording, because of the sparing of ADM in type 4 and type 5 lesions (7). Palmar stimulation of the ulnar nerve in addition to the stimulation at wrist increases the probability of UNW diagnosis from 60% to 95% (14). However, the stimulation at a single location can't detect the exact site of the lesion. SSIS can pinpoint the focal latency prolongation and conduction block caused by the segmentally demyelinating lesion located between the

two consecutive stimulation points nearly one-centimeter apart (3). McIntosh *et al.* reported in 1998 the first SSIS across the wrists of 2 patients with UNW and 10 normal subjects (3). They stimulated the ulnar nerves at seven subsequent 1 cm-intervals between 3 cm proximal and 4 cm distal to the DWC while recording from FDI. The authors ascertained the abnormality as a latency prolongation of more than 0.5 ms/cm and/or an amplitude change greater than 120% between the two stimulation points( ). In the same year, by using the inching method, Padua *et al.* reported a patient who had a neurapraxic block at Guyon's canal (15).

SSIS can provide a fairly precise localization of the segmentally demyelinating UNW lesions as were found in our patients. This might particularly be helpful for clinicians dealing with UNW cases who do not have readily identifiable imaging lesions similar to those of our cases. Even though, SSIS is more time-consuming and technically demanding as compared to the conventional NCV studies, it is very sensitive and specific for an ulnar nerve compression at the wrist. We suggest adding it into the electrophysiological test batteries for searching the UNW.

REFERENCES

1. Eckman PB, Perstein G, Atrocchi P. Ulnar neuropathy in bicyle riders. Arch Neurol. 1975 Feb;32(2):130-132.
2. Hunt JR. Occupation neuritis of deep palmar branch of ulnar nerve: Well defined clinical type of professional palsy of hand. J Nerv Ment Dis. 1908;35:673-689.
3. McIntosh KA, Preston DC, Logigian EL. Short-segment incremental studies to localize ulnar nerve entrapment at the wrist. Neurology. 1998;50(1):303-306.
4. Erkin G, Uysal H, Keleş I, Aybay C, Özel S: Acute ulnar neuropathy at the wrist: a case report and rewiew of the literature. Rheumatol Int. 2006;27:191-196.
5. Preston DC, Shapiro BE. Electromyography and Neuromuscular Disorders, 2 nd edition. Elsevier, Philadelphia, 2005;313-326.
6. Wu JS, Morris JD, Hogan GR. Ulnar neuropathy at wrist: case report and rewiev of literature. Arch Phys Med Rehabil. 1985;66:785-788.
7. Olney RK, Hanson M. AAEE Case report # 15: Ulnar neuropathy at or distal to the wrist. Muscle Nerve. 1988 Aug;11(8):828-832.
8. Kothari MJ, Preston DC, Logigian EL. Lumbrical-interossei motor studies localize ulnar neuropathy at the elbow. Muscle Nerve. 1996;19:170-174.
9. Guyon F. Note sur une disposition anatomique propre a la face anterieure de la region du poignet et non encore decrite. Bull Soc Anat Paris 1861;6:184-186.
10. Gross MS, Gelberman RH. The anatomy of the distal ulnar tunnel. Clin Orthop. 1985;196:238-247.
11. Stewart JD. Focal peripheral neuropathies. 3rd edition 2000. Lippincott Williams&Willkins, 2000: 241-271.
12. Venkatesh S, Kothari MJ, Preston DC. The limitation of the dorsal ulnar cutaneous sensory response in patients with ulnar neuropathy at the elbow. Muscle Nerve. 1995;18:345-347.
13. Shea JD, Mc Lain EJ. Ulnar-nerve compression syndrome at and below the wrist. J Bone Joint Surg. 1969;51A:1095-1103.
14. Cowdey SR, Preston DC, Hermann DN, Logigian EL. Electrodiagnosis of ulnar neuropathy at the wrist: conduction block versus tradational tests. Neurology. 2002 aug;13;59(3):420-427.
15. Padua L, Insola A, LoMonacao M, Denaro FG, Padua R, Tonali P. A case of Guyon syndrome with neuroapraxic block resolved after surgical decompression. Electroencephalography and clinical Neurophysiology. 1998;109:191-193.

Pınar Yalınay Dikmen,
Department of Neurology,
Acıbadem University,
Faculty of Medicine,
34390 Istanbul (Turkey).
E-mail: pinar.yalinay@acibadem.edu.tr
: pinarya@hotmail.com