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Validation of preoperative nerve conduction studies by intraoperative studies in patients with ulnar neuropathy at the elbow

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HIGHLIGHTS

- Preoperative and intraoperative short-segment nerve conduction studies (SSNCSs) were correlated in 36 arms.
- Preoperative SSNCSs and US correctly localized ulnar neuropathy at the elbow (UNE) in all arms.
- No intraoperative SSNCSs seem to be needed to confirm cubital tunnel syndrome.

ABSTRACT

Objective: To validate the findings of preoperative motor short-segment nerve conduction studies (SSNCSs) by intraoperative SSNCSs in patients with cubital tunnel syndrome.
Methods: We prospectively recruited patients with ulnar neuropathy at the elbow (UNE) localized distal to the medial epicondyle (ME). Preoperatively, motor SSNCSs and ultrasonography (US) were performed. Immediately after surgical dissection of the humeroulnar aponeurotic arcade (HUA), intraoperative near-nerve motor SSNCSs were performed, and compared to preoperative findings.
Results: We studied 36 arms with UNE in the cubital tunnel. Preoperative US localized UNE distal to ME in all operated arms, and demonstrated ulnar nerve constriction in 19 of them. Visual inspection confirmed ulnar nerve swelling in all studied nerves, but was unreliable with regard to ulnar nerve constriction. In all 5 (14%) arms with inconclusive localization by SSNCSs, intraoperative SSNCSs confirmed the preoperative US diagnosis of cubital tunnel syndrome. Intraoperative SSNCSs confirmed the preoperative localization in 24 (67%) arms, and were non-contributive in 7 (19%) arms with intraoperatively non-recordable responses.
Conclusion: Intraoperative near-nerve SSNCSs did not change the localization in any of 36 arms with UNE distal to ME. Therefore, our data indicate that a combination of preoperative SSNCSs and US reliably localizes UNE in the cubital tunnel.
Significance: Our present study suggests that in arms with consistent preoperative SSNCSs and US studies, no intraoperative near-nerve SSNCSs are needed to confirm ulnar nerve entrapment under the HUA.

1. Introduction

Motor short-segment nerve conduction studies (motor SSNCSs, motor 5 × 2 cm studies) (Omejec and Podnar, 2016a) are regarded as the gold standard for diagnosing ulnar neuropathy at the elbow (UNE) (Visser et al., 2005). We demonstrated previously that motor SSNCSs have better diagnostic accuracy than motor 2x4 cm, standard 10-cm motor nerve conduction studies (NCSs) across the elbow (Omejec and Podnar, 2016a), and ultrasonographic (US) examination (Omejec and Podnar, 2015a). Furthermore, only SSNCSs, motor 2 × 4 cm studies and US are able to precisely localize UNE, which is needed for discrimination between the two main UNE varieties, namely ulnar nerve entrapment under the humeroulnar aponeurotic arcade (HUA), and extrinsic ulnar nerve compression in the retroepicondylar (RTC) groove (Omejec and Podnar, 2015b). This discrimination is essential for rational UNE therapy, as ulnar nerve entrapment under the HUA requires early

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surgical decompression (Campbell et al., 1988, 1992), while conservative treatment is probably first-line therapy for UNE in the RTC.

For the diagnosis and localization of UNE, motor SSNCSs and motor 2×4 cm studies are most commonly performed non-invasively using percutaneous supramaximal ulnar nerve stimulation (Azrieli et al., 2003; Simon et al., 2015; Omejec and Podnar, 2016a). However, using this approach, the extent of the measurement error remains unclear. It is possible that during supramaximal percutaneous stimulation, the nerve is excited a few mm away from the intended surface landmark, resulting in a spurious diagnosis and localization of UNE.

To resolve this issue, we performed a study directly comparing percutaneous preoperative, and invasive intraoperative near-nerve motor SSNCSs in the same arms. To date, no direct comparison between preoperative and intraoperative SSNCSs in the same ulnar nerves has been reported. Our studies were performed in arms with UNE due to ulnar nerve entrapment under the HUA (i.e., the cubital tunnel syndrome). We compared compound motor action potential (CMAP) amplitudes, conduction velocities (i.e., latency changes), diagnoses and precise UNE localizations obtained by both approaches.

2. Materials and methods

2.1. Patients

Between April 2012 and October 2014 in our secondary referral unit (i.e., the Institute of Clinical Neurophysiology, University Medical Center Ljubljana, Slovenia) we prospectively recruited consecutive patients with suspected UNE. We included patients with at least one of the following symptoms (i.e., the inclusion criteria): (1) continuous 4th and 5th finger numbness or paresthesia; (2) the ulnar-innervated muscles weakness; or (3) loss of hand dexterity. We excluded all patients with (i.e., the exclusion criteria): (1) previous elbow fracture or surgery; (2) polyneuropathy; or (3) motor neuron disorders.

The history was obtained, and the clinical neurologic, electrodiagnostic (EDx), and US examinations were performed by 4 investigators blinded to the findings of the other parts of the evaluation.

The National Ethics Committee of Slovenia approved the study, and prior to the investigation all participating patients provided written informed consent.

2.2. History and clinical examination

The first investigator took a short history, and using a focused questionnaire (Mondelli et al., 2006) collected patients' demographic and clinical data in a standardized manner. The second investigator in both arms estimated muscle bulk, assessed muscle strength using the Medical Research Council (MRC) scale (O'Brien, 2010), and graded light touch and pin prick.

2.3. Preoperative EDx and UNE pathophysiology

With the subject supine and the elbow flexed to 90°, the third investigator performed ulnar motor SSNCSs across the elbow, using a standard EMG system (Nicolet Synergy, Natus Medical Incorporated, San Carlos, USA). He stimulated the nerve at the wrist, 2 and 4 cm distal (D2, D4) to medial epicondyle (ME), at ME, and 2, 4 and 6 cm proximal (P2, P4, and P6) to ME. CMAPs were separately recorded from the ADM and FDI muscles (Omejec et al., 2015). He also recorded sensory ulnar nerve action potentials (SNAPs) from the 4th and 5th finger after nerve stimulation at the wrist. Concentric needle electromyography (EMG) of the selected forearm and hand muscles was also performed, as previously described (Omejec et al., 2015). During preoperative EDx studies, skin temperature at the elbow and wrist was checked and the limb warmed-up to remain above 30 °C.

We diagnosed and localized UNE using motor SSNCSs to a 2-cm ulnar nerve segment with maximal: (1) motor nerve conduction velocity (MNCV) slowing below the lower normative limit (<31 m/s); *or* (2) CMAP amplitude drop above the upper normative limit (>12%) (Omejec and Podnar, 2015a,b).

We diagnosed axonal UNE in arms with: (1) ulnar CMAP amplitude on D4 stimulation and ADM/FDI muscle recording below the lower normative limit (<6.5/6.6 mV, respectively), and (2) the 5th finger SNAP below the lower normative limit (<13 μV) (Omejec and Podnar, 2015a). In arms with ulnar CMAP amplitude drop > 12% (i.e., the upper normative limit) UNE with conduction block, and in arms with ulnar MNCV <31 m/s (i.e., the lower normative limit) UNE with conduction slowing were diagnosed (Omejec and Podnar, 2015a). We diagnosed axonal UNE also when additional conduction block or conduction slowing, and UNE with conduction block when additional conduction slowing was present (Omejec and Podnar, 2015b).

2.4. Ultrasonography (US)

At the wrist and at each marker across the elbow the fourth investigator measured the ulnar nerve cross-sectional area (CSA) (Pompe and Beekman, 2013) using an US device (ProSound Alpha 7, Hitachi Aloka Medical, Ltd, Tokyo, Japan), and a 4–13 MHz linear array transducer. He used a trace method for CSA measurements, and he excluded the hyperechoic epineurial rim at 13 MHz. To localize the lesions under the HUA more precisely, in all nerves with UNE distal to ME he also measured CSA at D1 and D3 markers.

Using US we diagnosed and localized UNE: (1) to the ulnar nerve constriction (i.e., $\geqslant 2$ mm^2 larger CSA just proximal and distal); (2) in ulnar nerves with maximal CSA (CSAmax) distal to ME: to the first marker distal to the ulnar nerve with CSA > our normative limit; or (3) in ulnar nerves with CSAmax at or proximal to ME: to the CSAmax (Omejec and Podnar, 2015b).

2.5. Surgery and intraoperative EDx

We selected surgical candidates from patients with: (1) typical and pronounced UNE symptoms, (2) characteristic UNE signs on neurologic examination, (3) excluded alternative diagnoses (e.g., C8 radiculopathy, distal ulnar neuropathy at the wrist, etc.), (4) UNE diagnosis confirmed by motor SSNCSs, or US, and (5) UNE precisely localized distal to ME (i.e., under the HUA) using motor SSNCSs (segments D4/D2 or D2/ME) and US (markers D4, D3, D2 or D1) studies (Omejec and Podnar, 2015b).

During surgery under general anesthesia, the plastic surgeon performed a simple ulnar nerve decompression of the cubital tunnel (Mondelli et al., 2004). The surgical procedure and intraoperative studies were performed in a bloodless operative field with a sphygmomanometer cuff applied to the upper arm and inflated to 250 mmHg for 20 min. The surgeon made an approx. 8-cm long skin incision between D4 and P4 markers along the course of the ulnar nerve. He retracted the subcutaneous tissue to clearly visualize the ulnar nerve, transected the HUA, and left the nerve in its original position.

Immediately after cubital tunnel decompression, the fourth investigator performed intraoperative ulnar near-nerve motor SSNCSs (Campbell et al., 1988). He used the EMG system, and positions of the patient and elbow identical to those used during the preoperative studies. In the last 20 arms, he also measured the intraoperative skin temperature at the elbow and wrist (Crum and Strommen, 2007). Except for stimulation at the wrist and the