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Diagnostic sensitivity of electrophysiology and ultrasonography in ulnar neuropathies of different severity

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HIGHLIGHTS

- Sensitivity of electrophysiology and ultrasound in ulnar neuropathy was highly dependent on clinical severity.
- Ultrasound increased the diagnostic yield in very mild and mild neuropathies.
- Ultrasound localized all the ulnar neuropathies with abnormal non-localizing electrophysiology.

ABSTRACT

Objective: To assess the diagnostic performance of electrophysiology and nerve ultrasound in ulnar neuropathies of varying clinical severity in 135 consecutive patients.
Methods: Clinical severity of ulnar neuropathy was graded on a 4 point scale from *very mild* (symptoms only) to *severe* (marked atrophy of intrinsic hand muscles). Sensitivity and localization ability of electrophysiology and nerve ultrasound were assessed for each point of the scale.
Results: Ultrasound had higher sensitivity than electrophysiology in clinically *very mild* (20% and 3% for ultrasound and electrophysiology, respectively) and *mild* (62% and 47% for ultrasound and electrophysiology, respectively) neuropathies, had greater localizing ability in axonal ulnar neuropathies, and identified nerve hypermobility.

Ultrasound nerve cross-sectional area had strong positive correlation with both clinical and electrophysiological severity scores, but with significant overlap across the severity groups.
Conclusion: The diagnostic work-up of ulnar neuropathies was improved by using both electrophysiology and ultrasound at all levels of clinical severity. Ultrasound increased the diagnostic yield in very mild and mild neuropathies, localized all the ulnar neuropathies with abnormal non-localizing electrophysiology and identified nerve hypermobility.
Significance: This is the first detailed analysis of the diagnostic performance of electrophysiology and ultrasound in ulnar neuropathies of varying severity.

1. Introduction

Ulnar neuropathy is the second most common mononeuropathy after carpal tunnel syndrome. It usually results from focal nerve abnormality around the elbow, with an estimated incidence of 24.7/10^5/year (Mondelli et al., 2005). It presents with paraesthesia or sensory loss in the little and ring fingers and weakness of ulnar innervated muscles. Untreated ulnar neuropathy can progress to cause severe disability. However, despite its relatively high incidence, diagnosis and management of ulnar neuropathy at the elbow can be still challenging. Diagnosis is usually based

Abbreviations: ADM, Abductor Digiti Minimi; CMAP, Compound Muscle Action Potential; CSA, Cross Sectional Area; CSA Max, Maximal Cross Sectional Area; EDX, Electrophysiology; EMG, Electromyography; FDI, First Dorsal Interosseous; MRC, Medical Research Council; SNAP, Sensory Nerve Action Potential; UNE, Ulnar neuropathy at the elbow.

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on clinical findings supported by electrophysiological tests. Electrophysiology can confirm an abnormality and localize the lesion by demonstrating focal conduction slowing, with or without temporal dispersion and conduction block across the elbow (AAEM, 1999a). However, the diagnostic accuracy of these tests is not as high as in carpal tunnel syndrome, with sensitivity ranging from 38% to 89% in different studies (AAEM, 1999b), and problems associated with measurement errors (Landau et al, 2002; Maynard and Stolov, 1972). In addition, in a subgroup of patients with axonal ulnar neuropathies, electrophysiology is abnormal but non-localizing (Jabre and Wilbourn,1979; Shady Abuaisha and Boulton, 1998; Wilbourn, 1987).

In recent years, high-resolution nerve ultrasound has emerged as a reliable and sensitive tool for investigating ulnar neuropathies, particularly helpful when electrodiagnostic tests are either non-localizing (Beekman et al., 2004, 2011; Omejec et al, 2015; Pelosi et al, 2018) or falsely negative (Beekman et al., 2004; Yoon et al, 2010). However, a recent study reported higher sensitivity of electrophysiology than ultrasound (Omejec et al, 2015).

Over the past 4 years, we have routinely used nerve ultrasound alongside electrophysiology in the investigation of all ulnar neuropathies referred to our institution. In this study, we sought to assess the diagnostic performance of each method in ulnar neuropathies of varying clinical severity and evaluate the benefit of using both methods in the diagnostic work-up in our patient cohort.

2. Methods

All procedures were in accordance with the Helsinki Declaration of 1975 and were part of routine clinical practice for the evaluation of ulnar neuropathy at our institution.

2.1. Patients

Clinical inclusion criteria were: numbness and/or paraesthesia in the little and ring fingers with or without weakness and atrophy of ulnar-innervated hand muscles and medial elbow pain. Patients with previous ulnar nerve decompression, medial cord/lower trunk plexopathy or C8/T1 radiculopathy or distal ulnar neuropathy confirmed by electrophysiological tests (see ***Electrodiagnostic tests*** for electrophysiology protocol) were excluded. Three patients with diabetes were included, as their ulnar nerve was disproportionately affected and it was unclear if this was ulnar neuropathy at the elbow or a complication of the systemic condition. Findings in these three patients were also discussed separately.

2.2. Clinical severity

Clinical severity of ulnar neuropathy was graded using a 4 point scale adapted from previous classifications (Bartels et al., 1998; McGowan, 1950) as 1. *Very Mild*: symptoms only; 2. *Mild*: reduced sensation (light touch and pin-prick) in the ulnar-innervated hand regions, motor function either normal or very mild weakness of abductor digiti minimi (ADM) and/or first dorsal interosseous (FDI), Medical Research Council (MRC) >4; 3. *Moderate*: sensation reduced (as per *Mild*), ADM/FDI atrophy and weakness, MRC 4, and 4. *Severe*: sensation reduced (as per *Mild*) or absent, ADM/FDI atrophy and weakness, MRC $\leq$ 3.

2.3. Electrodiagnostic tests and severity

American Association of Electrodiagnostic Medicine recommendations were followed for electrophysiological evaluation and diagnosis of ulnar neuropathy at the elbow and exclusion of mimicking conditions such as C8/T1 radiculopathy and medial cord/lower trunk plexopathy (AAEM, 1999a). A single investigator (LP, clinical neurophysiologist >25 years) performed the electrophysiological studies using the Nicolet Synergy EDX EMG System, (Natus Medical Incorporated, San Carlos, USA). Sensory and motor nerve conduction studies were performed with surface stimulation and recording under controlled limb temperature (>31 °C). The ulnar antidromic sensory nerve action potential (SNAP) was recorded from the little finger with stimulation at the wrist. The ulnar compound muscle action potential (CMAP) was recorded from the abductor digiti minimi (ADM) and first dorsal interosseous (FDI) with stimulation at the wrist, below elbow (BE) (3 cm distal to the medial epicondyle), above elbow (AE) (10 cm proximal to BE) and axilla. Abnormality was defined (from prior values obtained in our laboratory) by sensory baseline-to-peak amplitude <10uV, sensory peak latency >3.1 ms (distance 11 cm), CMAP amplitude <5.0 mV for ADM and <6.0 mV for FDI, and motor conduction velocity (MCV) <50 m/s. Motor studies were performed with the elbow in 70–90° of flexion.

According to the AAEM criteria, the following suggested a focal ulnar nerve lesion at the elbow: absolute MCV from AE to BE < 50 m/s; an AE-to-BE segment > 10 m/s slower than BE-to-wrist MCV; a decrease in CMAP negative peak amplitude from BE to AE > 20%, suggestive of conduction block or temporal dispersion (assuming that anomalies of innervation i.e. Martin-Gruber anastomosis were not present); a significant change in CMAP configuration at the AE site compared to the BE site (assuming that anomalies of innervation i.e. Martin-Gruber anastomosis were not present). In cases of severe neuropathy associated with conduction slowing over the BE-to-Wrist segment – presumably secondary to Wallerian degeneration – a comparison of the MCVs over the AE-to-BE and axilla-to-AE segments were included.

When motor conduction studies with the above protocol provided inconclusive evidence for a focal lesion at the elbow, an inching study was also obtained, looking for abnormal changes in the CMAP amplitude, area or configuration or abnormal changes in latency over 2 cm increments. We used a 2 cm $\times$ 5 protocol with two stimulations below the epicondyle, two above, and one at the epicondyle. However, accurate measurements of the onset (or peak) latency of the CMAP over short segments were not always possible when the CMAP amplitude was markedly reduced and the CMAP morphology was abnormal. In these situations, the inching study was deemed unreliable.

In non-localizing ulnar neuropathies, electromyography (EMG) of FDI and/or ADM, flexor carpi ulnaris (FCU) and, where appropriate flexor digitorum profundus (FDP 4–5), was done to assess severity of denervation. Additional non-ulnar muscles (flexor pollicis longus and extensor indicis proprius and, if required, cervical paraspinal) were also examined to exclude C8/T1 radiculopathy and medial cord/lower trunk plexopathy.

Electrodiagnostic severity was graded on a 4 point scale as 1. *Normal*; 2. *Mild*: reduced SNAP with normal CMAP amplitude with stimulation at the wrist and/or motor conduction slowing; 3. *Moderate*: reduced SNAP *and* CMAPs or absent SNAP and normal or mildly reduced CMAP with abnormal EMG, and 4. *Severe*: absent SNAP and absent or markedly reduced CMAPs with stimulation at the wrist. This grading emphasises electrodiagnostic findings of axonal loss.

2.4. Ultrasound

Ultrasound was obtained on the same day as the electrophysiology using SonoSite Edge (Fujifilm SonoSite Inc., Bothel, WA, USA) with nerve imaging software and a 6–15 MHz linear array transducer. The ulnar nerve was studied with the arm abducted and the elbow in flexion (70–90°). The nerve was first scanned in

the transverse plane from wrist to mid-humerus to detect diffuse or localized pathology. The maximal cross sectional area (CSA Max) was measured at the site where the nerve was thickest, using the trace function of the ultrasound device with manual tracing within the hyperechoic rim surrounding the nerve. The probe was held perpendicular to the nerve at the angle in which the nerve was the brightest with the best discernible rim. The mean value of three measurements was taken. With CSA Max at the elbow, cross sectional area (CSA) measurements were also taken for comparison at mid-forearm and mid-humerus (upper limit for mean +2 SDs, obtained from prior data in our laboratory, are: elbow 8.7 mm^2 , mid-forearm 7.8 mm^2 and mid-humerus 7.9 mm^2).

Ultrasound examination at the elbow also included a dynamic test to identify ulnar nerve hypermobility with or without snapping triceps, during active and passive movement of the elbow from extension to full flexion. We used the Okamoto et al (2000) classification to distinguish subluxation (the nerve moves on to the tip of the epicondyle) from anterior dislocation (the nerve moves anteriorly beyond the tip of the epicondyle).

2.5. Statistical analysis

Relationships between clinical severity scores, electrophysiology severity scores and CSA Max values were studied with Spearman's correlation coefficient.

The mean CSA Max values from each clinical severity group were compared by the Mann-Whitney U test.

3. Results

One hundred and forty-one consecutive ulnar neuropathies in 135 patients (M:F = 71:64, mean age 52.6 years) who were referred to our institution over a two year period from May 2014 to April 2016, met the inclusion criteria.

3.1. Sensitivity and localising performance

Table 1 summarizes the number and percentage of abnormal studies on electrophysiology and ultrasound according to clinical severity. The overall combined sensitivity of the two techniques to identify an abnormality across all 4 clinical severity groups was 60% (84/141).

The overall sensitivity was higher for ultrasound (83/141 = 58%) than electrophysiology (67/141 = 47%). This was due mainly to higher sensitivity of nerve ultrasound in the clinically *very mild* group (11/54 = 20% and 2/54 = 3% for ultrasound and electrophysiology, respectively) and, to a lesser extent, also in the clinically *mild* group (25/40 = 62% and 19/40 = 47% for ultrasound and electrophysiology, respectively) (Fig. 1). Sensitivity in the clinically *moderate* group was 100% (24/24) for ultrasound and 96% (23/24) for electrodiagnostic tests (Fig. 1). In the clinically *severe* group, sensitivity was equally high, 100% (23/23), for both tests (Fig. 1).

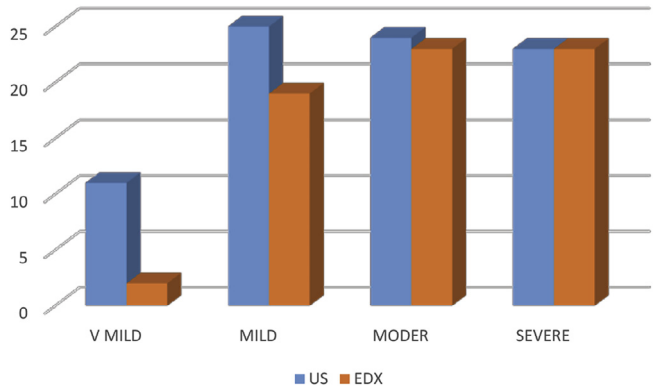

Fig. 1. Number of abnormal tests on ultrasound (US) and electrophysiology (EDX) (y axis) by clinical severity (x axis). The total number of nerves by clinical class were 54, 40, 24 and 23 for very mild, mild, moderate and severe, respectively.

If the clinically *very mild* group was excluded, the overall combined sensitivity of the two techniques to identify an abnormality was 82% (72/87 ulnar neuropathies). Sensitivity was higher for ultrasound (72/87 = 82%) than electrophysiology (65/87 = 74%), due to higher sensitivity of nerve ultrasound in the clinically *mild* group.

In 25 ulnar neuropathies (representing 17% of the total referrals and 37% of the neuropathies with abnormal electrophysiology), electrophysiology was abnormal but non-localizing, showing features of axonal degeneration with reduced amplitude sensory and/or motor responses, but no clear focal abnormality at the elbow. Eighteen of these (72%) were in the clinically *severe* and *moderate* groups (9 arms in each), and 7 were in the clinically *mild* group. These 25 ulnar neuropathies included our 3 patients with diabetes. Ultrasound was abnormal in all 25 of these electrophysiologically non-localisable neuropathies, showing increased CSA Max at the elbow in 22, and diffuse nerve enlargement in all 3 patients with diabetes. Sixteen of these 25 patients were included in a previous study (Pelosi et al, 2018).

3.2. Nerve hypermobility

Dynamic ultrasound study showed nerve hypermobility with subluxation or full anterior dislocation (Okamoto classification, Okamoto et al., 2000) in 24 ulnar nerves (24/141 = 17%). More than half (14 out of 24 = 58%) were in the clinically *very mild* group and another quarter (6/24 = 25%) in the *mild* group. Two of the remaining 4 nerves with hypermobility were in the clinically *moderate* and 2 in the clinically *severe* group. The latter two were in association with snapping triceps syndrome. The percentage of nerves with hypermobility in each clinical severity class is shown in Fig. 2. Hypermobility was present in a majority (63%) of the nerves with abnormal ultrasound in the *very mild* group and in one patient with normal ultrasound and reduced amplitude SNAP who also belonged to the *very mild* group.

Table 1
Number and percentage of abnormal studies on electrophysiology and ultrasound according to clinical severity.

Clinical severity	V mild	Mild	Moderate	Severe	All 4 severity	%
Number of nerves	54 (38%)	40 (28%)	24 (17%)	23 (16%)	141 (100%)	100
EDX & US abnormal	1	18	23	23	65	46
EDX abnormal	2	19	23	23	67	47
US abnormal	11	25	24	23	83	58
Total abnormal	13	25	24	23	84	60
Total normal	41	15	0	0	57	40
(sub)luxation	14	6	2	2	24	17

EDX = Electrophysiology; US = Nerve ultrasound.

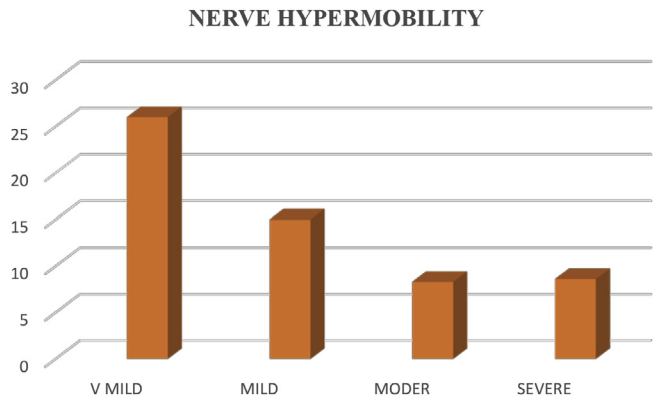

Fig. 2. Percentage of nerves with hypermobility (y axis) in each clinical severity class (x axis).

3.3. Statistical analysis

There was a strong positive correlation between the clinical severity scores and the electrophysiologic severity scores (R = 0.87).

The best agreement between the two scales was in the clinically *severe* group, with 21 out of 23 patients scoring severe on electrophysiologic tests and the remaining 2 scoring moderate. In the clinically *moderate* group, electrophysiological abnormalities were moderate in a majority (16/24 = 58%), severe in 2 and mild in 5. In the clinically *mild* group, electrophysiological abnormalities were mild in most (15/19 = 79%) and moderate in 4.

There was strong positive correlation between the CSA Max values and both the clinical (R = 0.78) and the electrophysiologic (R = 0.82) severity scores (Figs. 3 and 4).

The CSA Max increased with increasing clinical severity, with highly significant differences between the mean CSA Max of the 4 clinical severity groups (*Very Mild* = 8.36 ± 2.4 mm^2, *Mild* = 10.93 ± 2.9 mm^2, *Moderate* = 14.55 ± 2.7 mm^2 and *Severe* = 19.3 ± 5.7 mm^2. All group differences were significant at p < .001). However, there was significant overlap in the CSA Max values, especially between the *mild* and *moderate* groups, which means that an estimate of disease severity in individual patients, based on nerve enlargement, could be inaccurate.

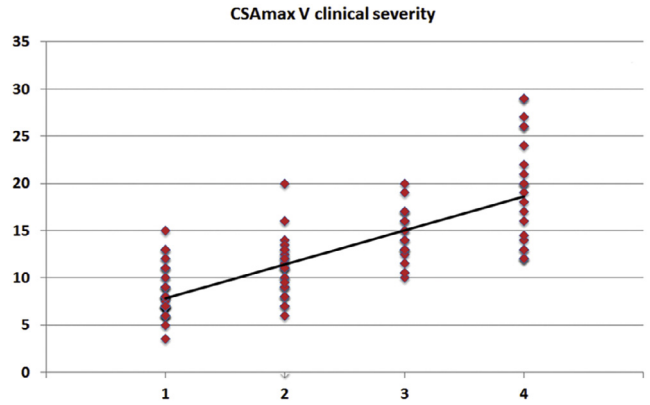

Fig. 3. CSA Max (maximal cross-sectional area in mm2) (y axis) by clinical severity scores (x axis).

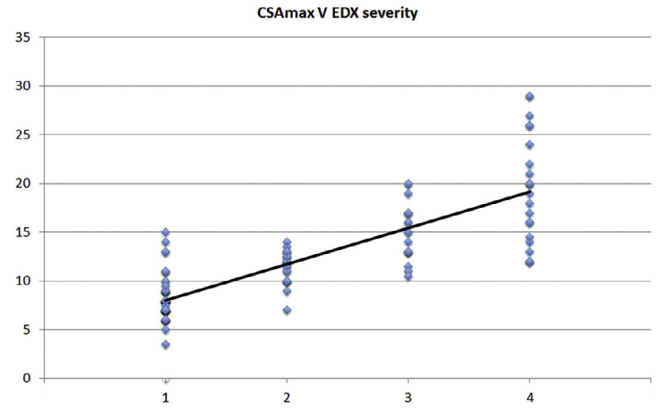

Fig. 4. CSA Max (maximal cross-sectional area in mm2) (y axis) by EDX (electrophysiology) severity scores (x axis).

4. Discussion

This analysis of our practice with combined electrophysiology and ultrasound over a 23 month period shows that the diagnostic work-up of ulnar neuropathy is significantly improved by the addition of nerve ultrasound at all levels of clinical severity.

In our cohort, the sensitivity of both electrophysiology and ultrasound was highly dependent on the clinical severity, being very high in clinically severe and moderate neuropathies, but relatively poor in mild and even poorer in very mild neuropathies. Ultrasound outperformed traditional electrodiagnostic tests (AAEM recommendations in our practice) in the detection of abnormalities when the clinical severity was mild or very mild, and increased the overall diagnostic yield by 11%. This is consistent with previous reports of abnormal nerve ultrasound in patients with clinical signs of ulnar neuropathy, but normal electrophysiology (Beekman et al, 2004; Yoon et al, 2010). A systematic analysis of the performance of either method in relation to clinical severity has not previously been done, and there are large variations in reported sensitivities of each technique for the detection of abnormalities in clinical ulnar neuropathy, ranging from 38 to 89% for electrophysiology (AAEM 1999b; Omejec et al, 2015) and 46–100% for ultrasound (Bayrak et al., 2010; Beekman et al., 2004; Beekman et al., 2011; Gruber et al., 2010; Mondelli et al., 2008; Omejec et al, 2015; Volpe et al., 2009; Wiesler et al, 2006; Yoon et al., 2008). Our findings suggest that this may be partly due to differences in the clinical severity of the populations being studied. For example, Omejec et al. (2015), who reported higher sensitivity of electrophysiology compared to nerve ultrasound in clinically definite ulnar neuropathy at the elbow, studied a population in whom 89% of patients had moderate or severe clinical ulnar neuropathies (compared to 34% in our study). In our study, the overall sensitivity of both techniques was significantly higher when the clinically very mild group was excluded from the analysis (82% and 72% for ultrasound and electrophysiology, respectively, compared to 58% and 47% when the very mild group was included). However, we feel it is important to include these clinically *very mild* patients, as they represented almost 40% of the referrals for ulnar neuropathy in our 'real-life' clinical neurophysiology practice. The sensitivity of both tests was 100% in the clinically severe group.

A major contribution of ultrasound to the diagnostic work-up was its impressive performance in patients with abnormal, but non-localising electrophysiologic studies. This scenario was present in 17% of the total referrals and over a third of the neuropathies with abnormal electrophysiology, most of which were

in the clinically moderate or severe group. Ultrasound was able to localise the abnormality in all of these patients, with most having focal ulnar neuropathies at the elbow and a minority (all patients with diabetes) showing diffuse nerve enlargement. This ability of ultrasound to define the underlying abnormality (by discriminating ulnar neuropathy at the elbow from the much less common truly diffuse ulnar mono-neuropathy associated with systemic disease) significantly altered clinical management. The superiority of ultrasound over electrophysiology in localizing the ulnar neuropathy in this subgroup is consistent with previous studies (Beekman et al, 2004; Omejec et al, 2015; Pelosi et al, 2018; Scheidl et al, 2013). In addition, our study shows that this scenario occurs particularly with ulnar neuropathies in the clinically severe and moderate groups.

Ultrasound enabled us to identify ulnar nerve hypermobility in 17% of the referrals. The significance of nerve hypermobility in ulnar neuropathy remains unclear. It is plausible that repetitive friction of the nerve against the epicondyle might give rise to neuropathy in some patients (van den Berg et al., 2013). Equally, some studies report asymptomatic ulnar nerve subluxation in up to 27% of limbs, and anterior dislocation in 20% (Okamoto et al., 2000), and others have shown a similar incidence of ulnar nerve (sub)luxation in patients with ulnar neuropathy at the elbow and healthy controls (van den Berg et al., 2013). In our study, hypermobility was significantly more frequent in the clinically very mild (i.e. patients with symptoms but no clinical signs) than in the other three groups combined, which is consistent with the original observation by Sunderland (1972). In addition, more than half of the dislocating nerves in our study were enlarged at the medial epicondyle level on ultrasound, suggesting mild nerve damage. It was reported previously that ulnar nerve (sub)luxation might cause mild, but probably not severe ulnar nerve damage (Omejec and Podnar, 2016). Ultrasound may be a useful tool to identify those patients in whom dislocation is contributing to nerve damage, and who may benefit from follow-up to exclude disease progression. Our finding that dislocation was less common in patients with moderate and severe neuropathy is in agreement with recent literature suggesting a protective effect against severe nerve injury, possibly by reducing tension on the nerve during elbow flexion by acting as a 'natural' transposition. (Leis et al, 2017). The exception to this in our study were two patients in whom nerve hypermobility was associated with snapping triceps, both of whom had severe clinical deficits. Again, the presence of snapping triceps was easily identified by ultrasound.

There was a strong positive correlation between nerve enlargement and electrophysiologic severity scores. This is in keeping with previous studies showing that nerve enlargement correlates with the severity of nerve conduction study abnormality (Bayrak et al., 2010; Beekman et al., 2004; Mondelli et al., 2008; Volpe et al., 2009; Yoon et al, 2008) and is greater in axonal neuropathies (Omejec et al, 2015; Scheidl et al., 2013). As the electrophysiological severity in our classification primarily reflects axonal loss, our findings confirm a relationship between the severity of axonal loss and the degree of nerve enlargement.

We also found a strong positive correlation between nerve cross-sectional areas and clinical severity scores and highly significant differences in the mean cross-sectional areas between the 4 clinical severity groups. However, there was significant overlap in the individual ultrasound values, especially between the mild and moderate clinical groups, suggesting that ultrasound alone is inadequate to estimate clinical severity in individual patients.

In this study, we did not examine the utility of ultrasound in identifying the causative mechanism of ulnar neuropathy at the elbow, but this has already been reported in previous studies (Beekman et al., 2011; Omejec et al, 2015). The fundamental role of electrodiagnostic tests, including electromyography, was in defining the severity of axonal loss, and the presence and extent of active denervation in clinically mild, moderate and severe neuropathies.

Our findings suggest that some predictions can be made based on the clinical severity at presentation. Severe and moderate clinical deficits are highly predictive of abnormal electrophysiological and ultrasound tests. However, in a significant proportion of these patients (over 1/3 in our cohort) electrophysiology alone will be unable to determine the site of the lesion, whereas ultrasound will identify a lesion at the elbow in the majority. Normal electrophysiological and ultrasound tests can be expected in approximately half and 40% respectively of clinically mild neuropathies, and almost all and 20% respectively of symptomatic ulnar neuropathies without clinical signs. A quarter of the very mild neuropathies will be associated with nerve hypermobility.

The lack of a diagnostic gold standard in ulnar neuropathy at the elbow (UNE) is an issue, particularly for patients with very mild presentations. Some of these patients may not have UNE. This may explain the low sensitivity of testing in this group. On the other hand, those patients who do have early presentation of UNE, could progress to more significant disability if not identified. This study suggests that the addition of nerve ultrasound to the diagnostic tests is helpful to identify these patients.

The lack of blinding is the main limitation of this study. However, all data was collected during real-life clinical practice, where blinding was not possible.

Overall, this review of our practice shows a definite improvement in the diagnostic work-up of ulnar neuropathy when both electrophysiology and ultrasound are used, as compared to the use of a single method, at all levels of clinical severity. Ultrasound has a definite role by increasing the diagnostic yield in the very mild and mild neuropathies, localizing the lesion in ulnar neuropathies with abnormal non-localizing electrophysiology, and identifying nerve hypermobility with or without snapping triceps and possible damage associated with hypermobility.

Conflict of interest

None of the authors has potential conflicts of interest to be disclosed.

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