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## Compound muscle action potential amplitude predicts the severity of cubital tunnel syndrome

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A commentary by Christopher J. Dy, MD, MPH, is linked to the online version of this article at [jbsj.org](http://jbsj.org).

# Compound Muscle Action Potential Amplitude Predicts the Severity of Cubital Tunnel Syndrome

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**Background:** Cubital tunnel syndrome has a spectrum of presentations ranging from mild paresthesias to debilitating numbness and intrinsic atrophy. Commonly, the classification of severity relies on clinical symptoms and slowing of conduction velocity across the elbow. However, changes in compound muscle action potential (CMAP) amplitude more accurately reflect axonal loss. We hypothesized that CMAP amplitude would better predict functional impairment than conduction velocity alone.

**Methods:** A retrospective cohort of patients who underwent a surgical procedure for cubital tunnel syndrome over a 5-year period were included in the study. All patients had electrodiagnostic testing performed at our institution. Clinical and electrodiagnostic variables were recorded. The primary outcome was preoperative functional impairment, defined by grip and key pinch strength ratios. Multivariable regression identified which clinical and electrodiagnostic variables predicted preoperative functional impairment.

**Results:** Eighty-three patients with a mean age of 57 years (75% male) were included in the study. The majority of patients (88%) had abnormal electrodiagnostic studies. Fifty-four percent had reduced CMAP amplitude, and 79% had slowing of conduction velocity across the elbow (recorded from the first dorsal interosseous). On bivariate analysis, older age and longer symptom duration were significantly associated ( $p < 0.05$ ) with reduced CMAP amplitude and slowing of conduction velocity across the elbow, whereas body mass index (BMI), laterality, a primary surgical procedure compared with revision surgical procedure, Disabilities of the Arm, Shoulder and Hand (DASH) questionnaire scores, and visual analog scale (VAS) scores for pain were not. Multivariable regression analysis demonstrated that reduced first dorsal interosseous CMAP amplitude independently predicted the loss of preoperative grip and key pinch strength and that slowed conduction velocity across the elbow did not.

**Conclusions:** Reduced first dorsal interosseous amplitude predicted preoperative weakness in grip and key pinch strength, and isolated slowing of conduction velocity across the elbow did not. CMAP amplitude is a sensitive indicator of axonal loss and an important marker of the severity of cubital tunnel syndrome. It should be considered when counseling patients with regard to their prognosis and determining the necessity and timing of operative intervention.

**Level of Evidence:** Prognostic Level IV. See Instructions for Authors for a complete description of levels of evidence.

Cubital tunnel syndrome is the second most common compression neuropathy<sup>1,2</sup>. It often presents insidiously because the initial symptoms can be intermittent and vague. The sensory symptoms, being limited to the ulnar side of the hand, may not be as bothersome as the critical sensory loss seen in carpal tunnel syndrome. It is therefore not unusual

for patients to present with advanced symptoms such as atrophy of the intrinsic muscles and weakness<sup>3</sup>.

The classic constellation of ulnar compression symptoms is the result of a complex interplay of mechanical and ischemic forces on the ulnar nerve at the cubital tunnel<sup>4,5</sup>. Initially, the compressed nerve develops focal areas of demyelination that may

**Disclosure:** There was no source of external funding for this study. The **Disclosure of Potential Conflicts of Interest** forms are provided with the online version of the article (<http://links.lww.com/JBJS/F182>).

TABLE I Patient Demographic Characteristics

Characteristics	Values
No. of patients	83
Age* (yr)	57.2 ± 14.1
Male sex†	62 (75%)
Dominant hand affected†	47 (57%)
Duration of symptoms* (mo)	34.5 ± 30.6
Bilateral symptoms†	5 (6%)
BMI* (kg/m <sup>2</sup> )	29.3 ± 5.7
Smoking†	16 (19%)
Diabetes mellitus†	13 (16%)
Hypothyroidism†	12 (14%)
Osteoarthritis of cervical spine†	10 (12%)
Complex regional pain syndrome†	1 (1%)
Simultaneous carpal tunnel syndrome†	16 (19%)
Prior cubital tunnel surgery†	24 (29%)

\*The values are given as the mean and the standard deviation.  
†The values are given as the number of patients, with the percentage in parentheses.

progress to axonal loss over time<sup>6</sup>. Patients presenting with severe or long-standing symptoms often have a component of axonal injury and are less likely to fully recover despite surgical intervention<sup>5,7,8</sup>. This is in contrast to carpal tunnel syndrome, which is predominantly a demyelinating neuropathy, from which the majority of patients recover following surgical intervention<sup>9</sup>.

The diagnosis of cubital tunnel syndrome is made with physical examination and often the support of electrodiagnostic studies<sup>10</sup>. Typically, patients will have slowing of conduction velocity across the elbow or a conduction block, which is a reflection of the degree of demyelination present. Sensory nerve action potential amplitudes and compound muscle action potential (CMAP) amplitudes are proportional to the number of functional axons present. The amplitudes are reduced in patients with axonal loss from severe or long-standing disease<sup>11</sup>. Axonal loss is also evident on electromyography (EMG), with spontaneous activity in the acute setting (e.g., fibrillations and positive sharp waves) and changes in the configuration and recruitment of motor unit potentials in both the acute and chronic settings<sup>12</sup>.

Traditionally, the severity of cubital tunnel syndrome is classified on the basis of clinical signs and symptoms, such as the McGowan-Goldberg and Dellon classifications<sup>13-15</sup>. The Akahori and Gu classifications include electrodiagnostic criteria, but are limited to conduction velocity<sup>16-19</sup>. To date, the surgical literature has focused on conduction velocity to guide surgical decision-making in cubital tunnel syndrome<sup>2,10,18-23</sup>. In contrast, our colleagues in neurology and physiatry use conduction velocity to localize the site of compression and use CMAP amplitude as an indicator of disease severity<sup>11,24-26</sup>.

We sought to correlate electrodiagnostic parameters with preoperative functional impairment in patients with cubital

tunnel syndrome. We hypothesized that reduced CMAP amplitude would predict greater functional impairment and be a more useful indicator of disease severity than conduction velocity in this patient population.

## Materials and Methods

A retrospective study was performed of adult patients who underwent a surgical procedure for cubital tunnel syndrome by the senior author over a 5-year period (2013 to 2017). Institutional approval from the Human Research Ethics Board was obtained. Patients were included if they

TABLE II Symptoms, Signs, and Electrodiagnostics of the Patient Cohort

Variable	Value
Symptoms*	
Pain	55 (66%)
Paresthesias to ring and small finger	74 (89%)
Numbness	32 (39%)
Weakness	45 (54%)
Clinical signs	
Small finger static 2-point discrimination >6 mm*	54 (65%)
Intrinsic atrophy present*	57 (69%)
Froment sign positive*	45 (54%)
Tinel sign at cubital tunnel*	56 (68%)
Scratch collapse at cubital tunnel*	51 (61%)
Grip strength† (kg)	23.1 ± 12.7
Grip strength ratio†	0.66 ± 0.25
Key pinch strength† (kg)	4.7 ± 2.6
Key pinch ratio†	0.61 ± 0.28
Scores‡	
DASH score‡	37.8 ± 21.7
VAS score for pain‡	4.3 ± 3.2
Electrodiagnostics	
Conduction velocity across elbow†§ (m/s)	37.7 ± 18.5
CMAP amplitude above elbow†§ (mV)	5.2 ± 4.3
CMAP amplitude at wrist†§ (mV)	6.0 ± 4.6
CMAP absent*§	9 (11%)
Conduction block present*§	27 (33%)
Sensory nerve action potential amplitude†# (µV)	11.4 ± 18.2
Sensory conduction velocity†# (m/s)	24.3 ± 25.4
Sensory nerve action potential absent*#	46 (55%)
Abnormal EMG in first dorsal interosseous and/or abductor digiti minimi*	57 (69%)
Martin-Gruber anastomosis present*	3 (4%)

\*The values are given as the number of patients, with the percentage in parentheses. †The values are given as the mean and the standard deviation. ‡The mean pain in the affected extremity over the past month. §Recorded from the first dorsal interosseous. #Recorded from the small finger.

**TABLE III Correlation Coefficients for Electrodiagnostic Parameters\***

	First Dorsal Interosseous		Abductor Digiti Minimi		Sensory Nerve Action Potential Amplitude§	Sensory Conduction Velocity#
	Amplitude†	Conduction Velocity‡	Amplitude†	Conduction Velocity‡		
First dorsal interosseous						
Amplitude†	—	0.49 (0.29 to 0.65)	0.86 (0.79 to 0.92)	0.51 (0.33 to 0.65)	0.64 (0.52 to 0.77)	0.68 (0.51 to 0.81)
Conduction velocity‡	0.49 (0.29 to 0.65)	—	0.53 (0.32 to 0.69)	0.89 (0.78 to 0.96)	0.42 (0.22 to 0.61)	0.38 (0.14 to 0.57)
Abductor digiti minimi						
Amplitude†	0.86 (0.79 to 0.92)	0.53 (0.32 to 0.69)	—	0.63 (0.50 to 0.74)	0.68 (0.54 to 0.81)	0.61 (0.43 to 0.76)
Conduction velocity‡	0.51 (0.33 to 0.65)	0.89 (0.78 to 0.96)	0.63 (0.50 to 0.74)	—	0.52 (0.39 to 0.65)	0.55 (0.41 to 0.68)
Sensory nerve action potential amplitude§	0.64 (0.52 to 0.77)	0.42 (0.22 to 0.61)	0.68 (0.54 to 0.81)	0.52 (0.39 to 0.65)	—	0.72 (0.63 to 0.81)
Sensory conduction velocity#	0.68 (0.51 to 0.81)	0.38 (0.14 to 0.57)	0.61 (0.43 to 0.76)	0.55 (0.41 to 0.68)	0.72 (0.63 to 0.81)	—

\*The correlations are given as the r value, with the 95% CI in parentheses. †This is the CMAP amplitude with stimulation at the wrist. ‡This is the motor nerve conduction velocity across the elbow segment. §This is the sensory nerve action potential amplitude for the small finger. #This is the antidromic sensory nerve conduction velocity for the small finger.

had clinical and electrodiagnostic evidence of cubital tunnel syndrome and had undergone a surgical procedure. Patients were excluded if they had their electrodiagnostic testing performed at an outside institution, brachial plexopathy, or confounding neurologic conditions (except carpal tunnel syndrome). All patients underwent transmuscular ulnar nerve transposition by the senior author as previously described<sup>17-29</sup>.

### Clinical Data

The following clinical data were extracted from patient charts: demographic characteristics, comorbidities, symptoms, duration, a previous surgical procedure for cubital tunnel syndrome, clinical examination, nerve conduction study, and EMG. The presence or absence of pain, paresthesias, numbness, and atrophy were recorded. Static 2-point discrimination

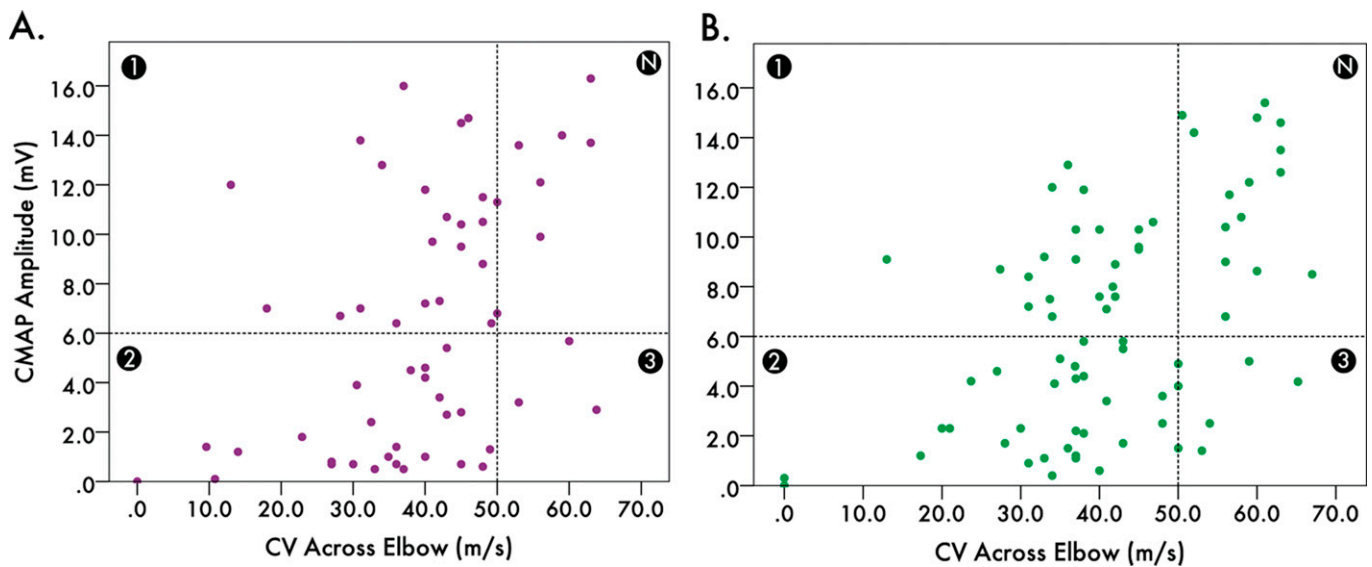


Fig. 1  
**Figs. 1-A and 1-B** Scatterplot of CMAP amplitude at the wrist compared with conduction velocity (CV) across the elbow. The dotted lines represent normal values for CMAP amplitude and CV. Quadrant 1 represents normal CMAP amplitude and reduced CV. This reflects focal demyelination of the nerve in the absence of substantial axonal loss. Quadrant 2 represents reduced CMAP amplitude and CV, as seen with demyelination and axonal loss. Quadrant 3 represents reduced CMAP amplitude and preserved CV. This uncommon scenario occurs when there is axonal loss but preservation of the fastest conducting nerve fibers. Values may differ between the first dorsal interosseous and abductor digiti minimi recordings. N = normal values. **Fig. 1-A** First dorsal interosseous. **Fig. 1-B** Abductor digiti minimi.

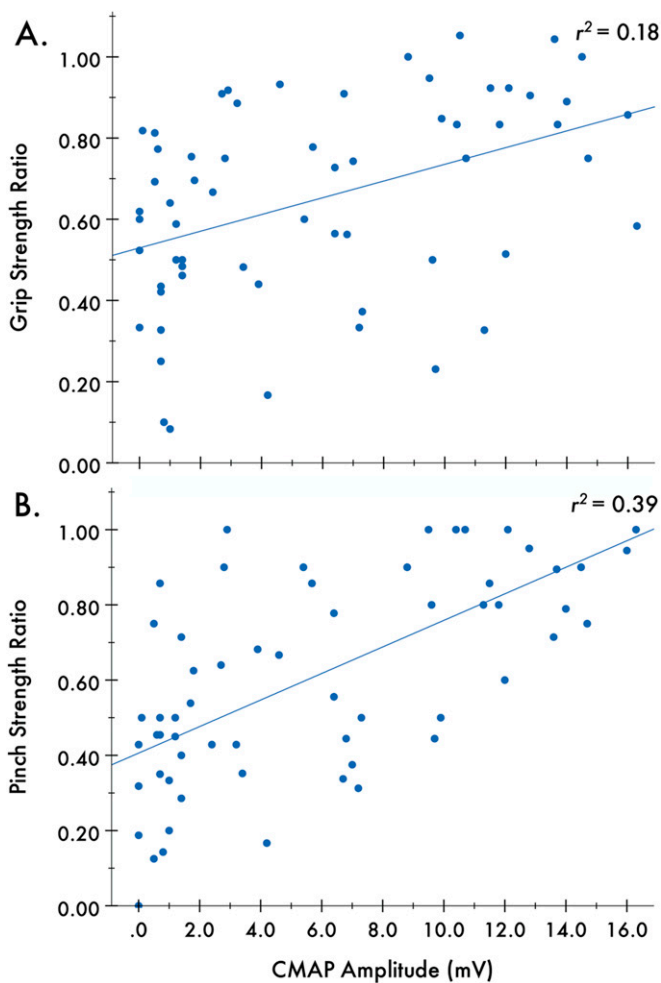


Fig. 2  
Correlation of the first dorsal interosseous CMAP amplitude at the wrist with the preoperative grip strength ratio (Fig. 2-A) and with the preoperative key pinch strength ratio (Fig. 2-B).

was recorded for the small finger using the Disk-Criminator (North Coast Medical). Grip strength was measured using a Jamar hydraulic hand dynamometer (model 5030J1; Sammons Preston) and key pinch strength was measured using a pinch gauge (model PG-30; B&L Engineering). The presence of a Tinel sign at the cubital tunnel and a Froment sign were recorded. A scratch collapse test was used to localize the site of compression along the ulnar nerve<sup>30,31</sup>. Upper-extremity impairment was evaluated with the Disabilities of the Arm, Shoulder and Hand (DASH) questionnaire<sup>32,33</sup>. A 10-cm visual analog scale (VAS) score was recorded for mean pain in the affected extremity over the past month.

The primary functional outcomes were grip and key pinch strength ratios because they provide objective measures of extrinsic strength (through the grip strength ratio) and intrinsic strength (through the key pinch strength ratio). These were calculated as the grip or pinch strength on the affected side divided by the strength on the unaffected side<sup>34</sup>.

### Electrodiagnostic Testing

All patients underwent electrodiagnostic testing by 1 of 4 subspecialized neurologists at our institution. To minimize variability in electrodiagnostic technique, patients were excluded if they had testing completed at an outside institution. All examinations were completed according to the guidelines of the American Association of Electrodiagnostic Medicine for ulnar nerve compression<sup>35</sup>. Standardized skin temperatures and room humidity were maintained. A motor nerve conduction study was performed with recording over the abductor digiti minimi and first dorsal interosseous. The following parameters were recorded: latency, CMAP amplitude, and conduction velocity from above-the-elbow, below-the-elbow, and wrist segments. Throughout this current study, we report CMAP amplitude with wrist stimulation to minimize the effects of a concurrent conduction block at the elbow. For patients with Martin-Gruber anastomoses ( $n = 3$ ), we report amplitude from the below-the-elbow segment. A sensory nerve conduction study was recorded using antidromic techniques with stimulation at the wrist and recording over the small finger. Peak latency, sensory nerve action potential amplitude, and conduction velocity were recorded. EMG of the first dorsal interosseous and abductor digiti minimi was assessed for the presence of spontaneous activity and the configuration and recruitment of motor unit potentials. The following normative values were used:  $>6$  mV for CMAP amplitude, and  $>5$   $\mu$ V for sensory nerve action potential amplitude. A conduction block was deemed present if there was  $>20\%$  drop in CMAP amplitude across the elbow<sup>35</sup>. Slowing of conduction velocity was defined as  $<50$  m/s across the elbow segment.

### Statistical Analysis

Descriptive statistics were summarized via established methods. The Pearson correlation was used to assess the correlation between electrodiagnostic variables, with 95% confidence intervals (CIs) calculated by the bootstrap method. Generalized linear regression using ordinary least squares was created to calculate the independent effect of each exposure variable of interest on the main outcomes. Because the primary outcomes (grip and key pinch strength ratios) are proportions, an inverse logistic (logit) transformation was performed to the outcome variable, and then multivariable fractional logit regression was applied<sup>36</sup>. Bivariate comparison was performed to assess the uncontrolled effect of each exposure variable against the primary outcomes for reference. The multivariable model was created on the basis of the combination of exposure variables that produced the maximum value of adjusted  $R^2$  to produce the model with the best overall fit<sup>37</sup>, with a maximum of 1 predictor per 10 outcome events to prevent overfitting. Final associations were reported as beta coefficients ( $\beta$ ) with 95% CIs, and  $\alpha < 0.05$  indicated significance in all tests.

### Results

Over the study period, 215 patients underwent ulnar nerve transposition; 83 patients met inclusion criteria and 132

**TABLE IV Bivariate Regression Showing Uncontrolled Effect of Exposure Variables on Preoperative Grip and Pinch Strength\***

Predictor Variables	Key Pinch Strength Ratio		Grip Strength Ratio	
	$\beta$ †	P Value	$\beta$ †	P Value
Age	0.00031 (−0.018 to 0.018)	0.97	−0.0066 (−0.024 to 0.010)	0.45
Sex	0.090 (−0.57 to 0.75)	0.79	−0.48 (−1.02 to 0.058)	0.080
Laterality	0.42 (−0.12 to 0.96)	0.13	0.45 (−0.060 to 0.96)	0.083
BMI	0.0070 (−0.041 to 0.055)	0.78	0.0036 (−0.054 to 0.061)	0.90
Symptom duration	0.00019 (−0.00019 to 0.0057)	0.32	0.00031 (−0.000015 to 0.00063)	0.061
Revision surgery	−0.21 (−0.75 to 0.33)	0.44	−0.035 (−0.55 to 0.48)	0.90
Abductor digiti minimi				
CMAP amplitude	0.12 (0.056 to 0.18)	<0.001‡	0.076 (0.021 to 0.13)	0.007‡
Conduction velocity across elbow	0.020 (0.0072 to 0.033)	0.002‡	0.010 (−0.0023 to 0.022)	0.11
Conduction block present	−0.0050 (−0.58 to 0.57)	0.99	−0.24 (−0.81 to 0.32)	0.40
First dorsal interosseous				
CMAP amplitude	0.16 (0.11 to 0.21)	<0.001‡	0.093 (0.039 to 0.15)	0.001‡
Conduction velocity across elbow	0.039 (0.025 to 0.054)	<0.001‡	0.017 (0.0040 to 0.030)	0.009‡
Conduction block present	−0.25 (−0.92 to 0.41)	0.45	−0.11 (−0.72 to 0.49)	0.72
Small finger sensory nerve action potential absent	0.29 (−0.26 to 0.83)	0.30	0.21 (−0.31 to 0.74)	0.43
EMG abnormal§	−0.93 (−1.60 to −0.25)	0.007‡	−0.74 (−1.32 to −0.16)	0.013‡

\*The multivariable (controlled) predictors of preoperative grip and pinch strength are in Table V. †The values are given as the standardized regression coefficient, with the 95% CI in parentheses. ‡Significant. §There is a presence of spontaneous activity or changes in motor unit recruitment and/or morphology in the first dorsal interosseous and/or abductor digiti minimi.

patients were excluded. Fifty-four patients had their electrodiagnostic testing performed at an outside institution, and 78 patients were excluded for confounding diagnoses (e.g., brachial plexus injury). Patient demographic characteristics are summarized in Table I. Clinical and electrodiagnostic findings are shown in Table II.

#### Motor Nerve Conduction Study

CMAP amplitude and conduction velocity across the elbow exhibited moderate correlation at both the first dorsal interosseous ( $r = 0.49$  [95% CI, 0.29 to 0.65]) and abductor digiti

minimi ( $r = 0.63$  [95% CI, 0.50 to 0.74]) (Table III). Figure 1 demonstrates the distribution of CMAP amplitude and conduction velocity across the patient cohort. The majority of patients had abnormal preoperative electrodiagnostic studies (88%). Values recorded from the abductor digiti minimi and first dorsal interosseous were disparate in numerous patients: 54% of patients had reduced abductor digiti minimi amplitude and 72% had slow abductor digiti minimi conduction velocity. When recording from the first dorsal interosseous, amplitudes were reduced in 54%, and conduction velocity was slow in 79%.

**TABLE V Controlled Predictors of Preoperative Grip and Pinch Strength in Multivariable Regression Analyses\***

Predictor Variables	Key Pinch Strength Ratio		Grip Strength Ratio	
	$\beta$ †	P Value	$\beta$ †	P Value
BMI	—	—	−0.022 (−0.085 to 0.041)	0.50
First dorsal interosseous CMAP amplitude	0.16 (0.090 to 0.24)	<0.001‡	0.10 (0.032 to 0.17)	0.004‡
First dorsal interosseous conduction velocity§	0.0091 (−0.015 to 0.033)	0.47	—	—
Abductor digiti minimi conduction block present	0.50 (−0.068 to 1.21)	0.28	0.44 (−0.20 to 1.08)	0.18

\*For each outcome, the multivariable model with the best overall fit (highest adjusted  $R^2$ ) is displayed. †The values are given as the ratio, with the 95% CI in parentheses. ‡Significant. §This is the motor nerve conduction velocity across the elbow segment.

### *Sensory Nerve Conduction Study*

Sensory nerve action potential amplitude and sensory conduction velocity exhibited moderate correlation ( $r = 0.72$  [95% CI, 0.63 to 0.81]) (Table III). The majority of patients had an abnormal sensory nerve action potential amplitude (66%) and abnormal sensory conduction velocity (63%). No recordable ulnar sensory response was found in 55% of patients.

### *Comparison of Clinical and Electrodiagnostic Variables*

Bivariate analysis failed to demonstrate a significant relationship of body mass index (BMI), laterality, a primary surgical procedure compared with a revision surgical procedure, the DASH score, and the VAS pain score with electrodiagnostic parameters including the CMAP amplitude, conduction velocity across the elbow, sensory nerve action potential amplitude, sensory conduction velocity, and an abnormal EMG (all  $p > 0.10$ ). However, advanced age and longer duration of symptoms were associated with reduced CMAP amplitude and slowed conduction velocity across the elbow, whether recorded from the first dorsal interosseous or abductor digiti minimi (all  $p < 0.05$ ).

### *Association of Patient and Electrodiagnostic Variables with Muscle Strength*

The correlation of CMAP amplitude with grip and key pinch ratios are shown in Figure 2. Bivariate regression identified a significant relationship of CMAP amplitude, conduction velocity across the elbow, and an abnormal EMG with preoperative grip and key pinch strength ratios (Table IV). When multivariable regression was performed for preoperative key pinch strength, only first dorsal interosseous amplitude was a significant independent predictor ( $p < 0.001$ ), and first dorsal interosseous conduction velocity ( $p = 0.47$ ) and the presence of an abductor digiti minimi conduction block ( $p = 0.28$ ) were not. Similarly, for preoperative grip strength, only first dorsal interosseous amplitude was a significant independent predictor ( $p = 0.004$ ), and BMI ( $p = 0.50$ ) and the presence of an abductor digiti minimi conduction block ( $p = 0.18$ ) were not. Regression coefficients ( $\beta$ ) and 95% CIs are shown in Tables IV and V.

### **Discussion**

In a cohort of 83 patients with cubital tunnel syndrome, we demonstrated that reduced first dorsal interosseous amplitude predicted preoperative functional impairment (i.e., weakness in grip and key pinch strength). Slowing of conduction velocity across the elbow failed to independently predict preoperative weakness. First dorsal interosseous amplitude correlated most strongly with pinch strength, which is likely secondary to its critical importance in the key pinch maneuver. In contrast, abductor digiti minimi amplitude failed to predict preoperative weakness, which we suspect is due to its relative unimportance in grip and pinch strength. An abnormal EMG also failed to predict preoperative weakness on multivariable analysis, which is not surprising given that it provides a qualitative assessment of muscle innervation.

The optimal treatment of cubital tunnel syndrome remains controversial, with numerous studies failing to demonstrate a

superior surgical technique<sup>2,38-40</sup>. This may be due in part to heterogeneous preoperative severity of disease. The senior author prefers ulnar nerve transposition because it offers the lowest risk of persistent and recurrent symptoms, in her experience. However, our findings are relevant to whichever technique the surgeon prefers.

The current clinical classifications for cubital tunnel syndrome are simple to apply but are imprecise and fail to accurately account for the degree of axonal injury present in more severe cases. The McGowan-Goldberg and Dellon classifications do not utilize electrodiagnostic parameters at all, whereas the Akahori and Gu classifications refer to changes in conduction velocity only<sup>13-16,18,19</sup>. Many studies in the surgical literature rely on changes in conduction velocity across the elbow to guide surgical decision-making<sup>2,18-22</sup>. From this study and our clinical experience, we believe that increased attention should be placed on CMAP amplitude values.

### *Classification of Nerve Injury in Cubital Tunnel Syndrome*

The nerve conduction study provides critical information that allows one to classify patients according to the Sunderland degree of nerve injury<sup>8</sup>. This is immensely helpful in counseling patients with regard to their prognosis. The Sunderland classification was originally described for acute nerve injuries. We believe that this classification is also useful for chronic compression injuries, as the pathophysiology described for each Sunderland grade (I to IV) parallels that of chronic nerve compression<sup>5,6,41</sup>. A patient who presents with slowed conduction velocity across the elbow and normal CMAP amplitude has focal demyelination (i.e., Sunderland first-degree injury) and should have a favorable expeditious recovery postoperatively. This patient may benefit from surgical intervention, but the urgency is diminished. In contrast, a patient with slowed conduction velocity and reduced CMAP amplitude has a component of axonal injury (i.e., second-degree or third-degree injury). On EMG, spontaneous activity and changes in the configuration and recruitment of motor unit potentials provide further evidence of axonal injury. Recovery is dependent on the number of remaining functional motor units and the status of the denervated motor end plates<sup>5</sup>. The surgeon should consider early definitive surgical intervention to maximize recovery and should preserve the remaining motor units. It is important to note that the presence of objective weakness does not necessarily imply the presence of an axonal injury. In these patients, the CMAP amplitude is an important consideration to predict who will have a favorable recovery.

### *Quantification of Axonal Injury*

A patient presenting with reduced amplitude has sustained some degree of axonal injury<sup>42</sup>. Gordon et al. demonstrated that function is preserved until a loss of >80% of the motor unit pool because the remaining healthy axons will collaterally sprout to innervate territories up to 5 times their original size (Fig. 3)<sup>43</sup>. CMAP and sensory nerve action potential amplitudes provide a quantitative estimate of the overall number of functional axons and are uniformly reported on all nerve conduction studies<sup>11,12</sup>. They are reproducible and can be tracked over



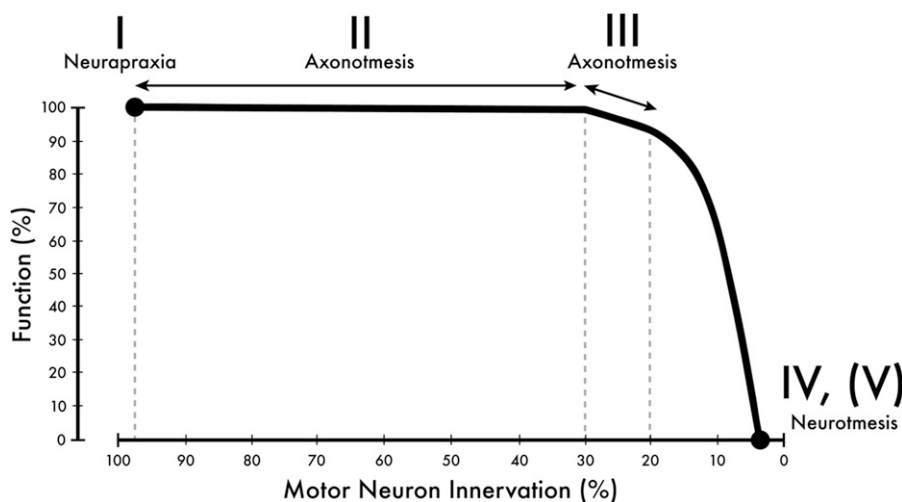


Fig. 3  
Relationship between muscle function and percentage of intact motor neurons. Although the relationship between function and motor neuron innervation has been recognized by researchers for some time<sup>43</sup>, its correspondence to the Sunderland and Seddon classifications of nerve injury<sup>8</sup> has not been linked to this physiologic relationship. For example, a Sunderland first-degree injury (I) has no axonal loss and therefore should recover full function. A second-degree injury (II) has variable axonal loss but full recovery of function, whereas a third-degree injury (III) has substantial axonal loss with functional recovery that is variable and incomplete. The Sunderland fourth-degree (IV) and fifth-degree (V) injuries usually have no functional recovery as the axonal loss is complete. Fourth-degree injuries occur rarely with compression neuropathy, and fifth-degree injuries occur only with traumatic or iatrogenic peripheral nerve injuries. Note that this figure depicts the anticipated functional recovery following a nerve injury and not the function immediately following nerve injury.

time. Electromyographers use changes in conduction velocity to localize the site of compression and amplitude to quantify axonal loss and classify severity<sup>11</sup>.

#### Role of Electrodiagnostic Studies in Surgical Planning and Determining Prognosis

Electrodiagnostic studies confirm the clinical diagnosis of cubital tunnel syndrome and can also provide prognostic information. This may direct the decision to proceed with conservative therapy or a surgical procedure. Patients presenting to our clinic with mild or intermittent symptoms are offered a trial of conservative therapy. It is our practice to obtain electrodiagnostics on all patients presenting with moderate to severe symptoms (e.g., McGowan-Goldberg II to III) and those who remain symptomatic after conservative therapy. A surgical pro-

cedure is offered to patients in the setting of normal electrodiagnostic testing when conservative therapy fails to improve symptoms and clinical examination supports the diagnosis of cubital tunnel syndrome of a dynamic-ischemic nature.

Electrodiagnostic results can vary between laboratories and it is important for surgeons treating compression neuropathies to develop a relationship with a qualified neurologist or physiatrist. The scatterplots in Figure 1 show the variability in conduction velocity and demonstrates why using it alone to guide surgical intervention is problematic. The consideration of changes in both amplitude and conduction velocity are important because each reflects a different aspect of nerve function (Table VI).

Numerous studies have attempted to use electrodiagnostics to predict prognosis in cubital tunnel syndrome, although all have relied on subjective postoperative outcomes<sup>25,26,44,45</sup>. Shi et al. studied 73 patients who underwent ulnar nerve transposition and found that higher CMAP amplitude predicted greater postoperative improvement in self-reported symptoms, assessed by the patient-rated ulnar elbow evaluation (PRUNE) score<sup>25</sup>. Friedrich and Robinson correlated the electrodiagnostics of 59 patients with their subjective postoperative outcome<sup>26</sup>. Those patients were more likely to make a complete recovery if they had a conduction block across the elbow to the first dorsal interosseous and normal abductor digiti minimi amplitude.

Differential changes in the abductor digiti minimi and first dorsal interosseous in cubital tunnel syndrome have been previously reported, and our study confirms this finding<sup>24</sup>. The etiology remains uncertain. In addition to ulnar nerve compression at the cubital tunnel, the deep motor branch may be compressed in the Guyon canal. Distal compression is primarily a clinical diagnosis because it is not reliably

**TABLE VI Summary Points with Regard to Electrodiagnostic Testing**

Decreased conduction velocity (e.g., <50 m/s) localizes the site of nerve compression
Changes in conduction velocity do not constitute a conduction block
Decreased CMAP amplitude (e.g., <6 mV at the wrist) implies axonal loss and allows determination of disease severity
EMG provides additional evidence of axonal injury and allows determination of the chronicity of compression (i.e., fibrillations and positive sharp waves are seen in acute injury, and changes in motor unit recruitment and morphology are seen in acute and chronic axonal injuries)

investigated on a routine nerve conduction study. Alternatively, fascicles of the first dorsal interosseus may be more susceptible to demyelinating injury and the abductor digiti minimi may be more prone to axonal injury because of the fascicular topography and the compressive forces applied<sup>26</sup>. Finally, the importance of the first dorsal interosseus to the key pinch maneuver may mean that patients notice symptoms sooner and present before compression has progressed to axonal injury.

### Study Limitations

Our cohort only includes patients who underwent a surgical procedure because patients treated conservatively were less likely to have had the electrodiagnostic testing completed at our institution. Electrodiagnostic testing was performed by 1 of 4 subspecialized neurologists rather than 1 individual. However, all testing was performed in a single laboratory using standardized protocols. There were a high number of patients undergoing a revision surgical procedure following outside referrals in the cohort (29%). However, the goal of this study was to correlate electrodiagnostic parameters with preoperative clinical examination for the purpose of determining disease severity and, by including revision cases, we were able to increase the spectrum of disease severity assessed.

### Future Directions

This study promotes the need for future investigations to delineate the prognostic utility of CMAP amplitude as a predictor of postoperative functional outcome. The current literature relies on subjective improvement and nonvalidated clinical symptom scores<sup>26,44,45</sup>. Data that correlate preoperative CMAP amplitude with objective functional measures of postoperative recovery and validated patient-reported outcomes will be useful in advancing our understanding of the disease process and its management.

### Conclusions

We have shown that reduced first dorsal interosseus amplitude predicts loss of preoperative grip and key pinch strength

and isolated slowing of conduction velocity across the elbow does not. CMAP amplitude is an important indicator of the disease severity in cubital tunnel syndrome and should be considered when counseling patients with regard to prognosis and determining the necessity and timing of surgical management. Patients with isolated slowing of conduction velocity should anticipate favorable recovery within months. In contrast, patients with reduced CMAP amplitude should be advised that recovery will be slow and, depending on the degree of axonal loss, potentially incomplete. ■

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## References

- Mondelli M, Giannini F, Ballerini M, Ginanneschi F, Martorelli E. Incidence of ulnar neuropathy at the elbow in the province of Siena (Italy). *J Neurol Sci*. 2005 Jul 15; 234(1-2):5-10. Epub 2005 Apr 20.
- Zlowodzki M, Chan S, Bhandari M, Kallianen L, Schubert W. Anterior transposition compared with simple decompression for treatment of cubital tunnel syndrome. A meta-analysis of randomized, controlled trials. *J Bone Joint Surg Am*. 2007 Dec; 89(12):2591-8.
- Mallette P, Zhao M, Zurakowski D, Ring D. Muscle atrophy at diagnosis of carpal and cubital tunnel syndrome. *J Hand Surg Am*. 2007 Jul-Aug;32(6):855-8.
- Dellon AL, Mackinnon SE. Human ulnar neuropathy at the elbow: clinical, electrical, and morphometric correlations. *J Reconstr Microsurg*. 1988 Apr;4(3): 179-84.
- Mackinnon SE. Pathophysiology of nerve compression. *Hand Clin*. 2002 May; 18(2):231-41.
- Mackinnon SE, Dellon AL, Hudson AR, Hunter DA. Chronic nerve compression—an experimental model in the rat. *Ann Plast Surg*. 1984 Aug;13(2):112-20.
- Tapadia M, Mozaffar T, Gupta R. Compressive neuropathies of the upper extremity: update on pathophysiology, classification, and electrodiagnostic findings. *J Hand Surg Am*. 2010 Apr;35(4):668-77. Epub 2010 Mar 11.
- Sunderland S. A classification of peripheral nerve injuries producing loss of function. *Brain*. 1951 Dec;74(4):491-516.
- Kronlage SC, Menendez ME. The benefit of carpal tunnel release in patients with electrophysiologically moderate and severe disease. *J Hand Surg Am*. 2015 Mar; 40(3):438-44.e1.
- Hutchinson RL, Rayan G. Diagnosis of cubital tunnel syndrome. *J Hand Surg Am*. 2011 Sep;36(9):1519-21. Epub 2011 May 7.
- Landau ME, Campbell WW. Clinical features and electrodiagnosis of ulnar neuropathies. *Phys Med Rehabil Clin N Am*. 2013 Feb;24(1):49-66. Epub 2012 Oct 25.
- Preston DC, Shapiro BE. Electromyography and neuromuscular disorders clinical-electrophysiologic correlates. 3rd ed. New York: Elsevier; 2013. Ulnar neuropathy at the elbow;p: p 298-318.
- McGowan AJ. The results of transposition of the ulnar nerve for traumatic ulnar neuritis. *J Bone Joint Surg Br*. 1950 Aug;32-B(3):293-301.
- Goldberg BJ, Light TR, Blair SJ. Ulnar neuropathy at the elbow: results of medial epicondylectomy. *J Hand Surg Am*. 1989 Mar;14(2 Pt 1):182-8.
- Dellon AL. Review of treatment results for ulnar nerve entrapment at the elbow. *J Hand Surg Am*. 1989 Jul;14(4):688-700.
- Akahori O. Cubital tunnel syndrome grade of palsy and prognosis, and selection of operation. *Seikei Geka*. 1986;29:1745-51.
- Watanabe M, Arita S, Hashizume H, Honda M, Nishida K, Ozaki T. Multiple regression analysis for grading and prognosis of cubital tunnel syndrome: assessment of Akahori's classification. *Acta Med Okayama*. 2013;67(1):35-44.

18. Qing C, Zhang J, Wu S, Ling Z, Wang S, Li H, Li H. Clinical classification and treatment of cubital tunnel syndrome. *Exp Ther Med*. 2014 Nov;8(5):1365-70. Epub 2014 Sep 22.
19. Gu YD. Current status and suggestion of clinical classification of carpal and cubital tunnel syndromes. *Chin J Orthop*. 2011;31(7):818-9. Chinese.
20. Gaspar MP, Kane PM, Putthiwara D, Jacoby SM, Osterman AL. Predicting revision following in situ ulnar nerve decompression for patients with idiopathic cubital tunnel syndrome. *J Hand Surg Am*. 2016 Mar;41(3):427-35. Epub 2016 Jan 16.
21. Charles YP, Coulet B, Rouzaud JC, Daures JP, Chammas M. Comparative clinical outcomes of submuscular and subcutaneous transposition of the ulnar nerve for cubital tunnel syndrome. *J Hand Surg Am*. 2009 May-Jun;34(5):866-74.
22. Kong L, Bai J, Yu K, Zhang B, Zhang J, Tian D. Predictors of surgical outcomes after in situ ulnar nerve decompression for cubital tunnel syndrome. *Ther Clin Risk Manag*. 2018 Jan 4;14:69-74.
23. Krogue JD, Aleem AW, Osei DA, Goldfarb CA, Calfee RP. Predictors of surgical revision after in situ decompression of the ulnar nerve. *J Shoulder Elbow Surg*. 2015 Apr;24(4):634-9. Epub 2015 Feb 3.
24. Shakir A, Micklesen PJ, Robinson LR. Which motor nerve conduction study is best in ulnar neuropathy at the elbow? *Muscle Nerve*. 2004 Apr;29(4):585-90.
25. Shi Q, MacDermid J, Grewal R, King GJ, Faber K, Miller TA. Predictors of functional outcome change 18 months after anterior ulnar nerve transposition. *Arch Phys Med Rehabil*. 2012 Feb;93(2):307-12.
26. Friedrich JM, Robinson LR. Prognostic indicators from electrodiagnostic studies for ulnar neuropathy at the elbow. *Muscle Nerve*. 2011 Apr;43(4):596-600. Epub 2011 Feb 11.
27. Mackinnon SE, Yee A. Transmuscular ulnar nerve transposition. 2013 Aug 23. <https://surgicaleducation.wustl.edu/transmuscular-ulnar-nerve-transposition-traumatic-medial-cord-injury/>. Accessed 2019 Jan 17.
28. Davidge KM, Boyd KU. Ulnar nerve entrapment and neuropathy. In: Mackinnon SE, editor. *Nerve surgery*. New York: Thieme; 2015. p 262-71.
29. Davidge KM, Ebersole GC, Mackinnon SE. Pain and function following revision cubital tunnel surgery. *Hand (N Y)*. 2017 Nov 1;1558944717743593. [Epub ahead of print].
30. Davidge KM, Gontre G, Tang D, Boyd KU, Yee A, Damiano MS, Mackinnon SE. The "hierarchical" scratch collapse test for identifying multilevel ulnar nerve compression. *Hand (N Y)*. 2015 Sep;10(3):388-95.
31. Kahn LC, Yee A, Mackinnon SE. Important details in performing and interpreting the scratch collapse test. *Plast Reconstr Surg*. 2018 Feb;141(2):399-407.
32. Beaton DE, Katz JN, Fossel AH, Wright JG, Tarasuk V, Bombardier C. Measuring the whole or the parts? Validity, reliability, and responsiveness of the Disabilities of the Arm, Shoulder and Hand outcome measure in different regions of the upper extremity. *J Hand Ther*. 2001 Apr-Jun;14(2):128-46.
33. Ebersole GC, Davidge K, Damiano M, Mackinnon SE. Validity and responsiveness of the DASH questionnaire as an outcome measure following ulnar nerve transposition for cubital tunnel syndrome. *Plast Reconstr Surg*. 2013 Jul;132(1):81e-90e.
34. Beumer A, Lindau TR. Grip strength ratio: a grip strength measurement that correlates well with DASH score in different hand/wrist conditions. *BMC Musculoskelet Disord*. 2014 Oct 6;15:336.
35. American Association of Electrodiagnostic Medicine. Practice parameter for electrodiagnostic studies in ulnar neuropathy at the elbow: summary statement. American Association of Electrodiagnostic Medicine, American Academy of Neurology, American Academy of Physical Medicine and Rehabilitation. *Muscle Nerve*. 1999;22(3):408-11.
36. Baum CF. Stata tip 63: modeling proportions. *Stata J*. 2008;8(2):299-303.
37. Lindsey C, Sheather S. Variable selection in linear regression. *Stata J*. 2010;10(4):650-69.
38. Caliendo P, La Torre G, Padua R, Giannini F, Padua L. Treatment for ulnar neuropathy at the elbow. *Cochrane Database Syst Rev*. 2012 Jul 11;7:CD006839.
39. Macadam SA, Gandhi R, Bezuhly M, Lefavre KA. Simple decompression versus anterior subcutaneous and submuscular transposition of the ulnar nerve for cubital tunnel syndrome: a meta-analysis. *J Hand Surg Am*. 2008 Oct;33(8):1314.e1-12.
40. Gervasio O, Gambardella G, Zaccone C, Branca D. Simple decompression versus anterior submuscular transposition of the ulnar nerve in severe cubital tunnel syndrome: a prospective randomized study. *Neurosurgery*. 2005;56(1):108-17; discussion 117.
41. O'Brien JP, Mackinnon SE, MacLean AR, Hudson AR, Dellon AL, Hunter DA. A model of chronic nerve compression in the rat. *Ann Plast Surg*. 1987 Nov;19(5):430-5.
42. Dy CJ, Mackinnon SE. Ulnar neuropathy: evaluation and management. *Curr Rev Musculoskelet Med*. 2016 Jun;9(2):178-84.
43. Gordon T, Yang JF, Ayer K, Stein RB, Tyreman N. Recovery potential of muscle after partial denervation: a comparison between rats and humans. *Brain Res Bull*. 1993;30(3-4):477-82.
44. Shi Q, MacDermid JC, Santaguida PL, Kyu HH. Predictors of surgical outcomes following anterior transposition of ulnar nerve for cubital tunnel syndrome: a systematic review. *J Hand Surg Am*. 2011 Dec;36(12):1996-2001.e1: 6.
45. Tong J, Dong Z, Xu B, Zhang C, Gu Y. Predictors of surgical outcomes for severe cubital tunnel syndrome: a review of 146 patients. *Acta Neurochir (Wien)*. 2018 Mar;160(3):645-50. Epub 2017 Dec 7.