Original article

Upper limb neurodynamic test 1 and symptoms reproduction in carpal tunnel syndrome. A validity study

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A B S T R A C T

The aim of this study was to estimate the validity of the Upper Limb Neurodynamic Test 1 (ULNT1) for the diagnosis of Carpal Tunnel Syndrome (CTS) with blind comparison to a reference criterion of a compatible clinical presentation and abnormal nerve conduction. 47 subjects with suspected CTS were enrolled. All patients were tested with nerve conduction studies and ULNT1. Considering results as positive in the presence of reproduction of symptoms on affected upper limb, or side-to-side differences in elbow extension, or symptoms modified by lateral neck side-bending, we estimated sensitivity as 91.67%, specificity as 15%, positive likelihood ratio as 1.0784, negative likelihood ratio as 0.5556, and post-test probability for negative test as 40%. Using a new criterion, i.e. the reproduction of symptoms only in the first three digits of the affected hand, we estimated sensitivity as 54.17%, specificity as 70%, positive and negative likelihood ratios as 1.8056 and 0.6548, respectively, and post-test probability for positive test as 68%. Our investigation suggests that the reproduction of the typical current CTS symptoms in the affected hand during ULNT1 testing, improves estimation of the probability of the presence of this condition, even if this test alone cannot be used to diagnose CTS.

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1. Introduction

Carpal tunnel syndrome (CTS) is one of the most common nerve compression disorders, and mainly affects women aged 55 and older (Atroshi et al., 1999). According to the Consensus Criteria for the Classification of CTS published by Rempel et al. (1998), the typical symptoms of CTS include pain associated with numbness, tingling or burning in at least one of the first, second or third digit.

Although the disagreement between CTS clinical symptoms and median nerve conduction studies (NCS) abnormalities is widely reported both in the general population (Redmond and Rinver, 1988; Glowacki et al., 1996) and in working populations (Homan et al., 1999; Violante et al., 2004), a report of the American Association of Electrodiagnostic Medicine (AAEM, 2002) recommended nerve conduction studies to confirm a clinical diagnosis of CTS, with a high degree of sensitivity and specificity (Jablecki et al., 1993, 2002). Therefore, NCS are still the reference standard for the diagnosis of CTS.

Although several tests for diagnosing CTS on physical examination such as the Tinel, Phalen, Reverse Phalen, Compression Tests, and Tethered Median Nerve Stress tests have been presented in the literature and adopted into clinical practice, studies of their reliability and diagnostic accuracy have yielded conflicting results (Aroori and Spence, 2008). To date, no single clinical test has been judged sufficient to identify this disorder (de Krom et al., 1990; MacDermid and Doherty, 2004; Descatha et al., 2010).

A clinical hypothesis that a patient has a nerve compression disorder begins with the patient’s report of specific symptoms such as pain, dysesthesia, paresthesia, often accompanied by signs of nerve impairment (e.g., weakness, anaesthesia, hypoesthesia) (Wainner et al., 2003). Presumably, nerve compression results in increased...
mechanosensitivity, although there may be differences between acute and chronic presentations. Plausibly, acute compression causes inflammatory change and development of neural irritation (Kobayashi et al., 2005). In particular, nerve injury due to acute compression increases intra-carpal canal pressure and causes changes in microcirculation up to complete ischemia (Dawson et al., 1999; Rempel and Diao, 2004). Functional abnormalities of nerve fibers, such as transient alteration of membrane excitability, and a conduction block if local compression is severe, have been observed under experimental conditions as a result of both ischemic and probably mechanicochemical mechanisms (Lundborg et al., 1982; Dawson et al., 1999).

Moreover the biological effects of short-term nerve compression were studied in the animal model showing several subsequent changes: endoneurial edema, inflammation and fibrin deposits, proliferation of endoneurial fibroblasts and capillary endothelial cells, up to proliferation of fibrous tissue (Powell and Myers, 1986; Dyck et al., 1990; Rempel and Diao, 2004). Local acute ischemia seems to be the cause of the acute and intermittent paresthesias. These symptoms reflect ectopic impulse generation in large myelinated sensory fibers due to transient disruption of membrane excitability (Rosenbaum and Ochoa, 1993; Dawson et al., 1999). Furthermore, Ochoa separates the pathophysiology of the common paresthesias from the less common genuine pain. In fact, if the paresthesias resulted from the spontaneous generation of neural activity in entrapped myelinated fibers induced by local ischemia, pain would due to direct mechanical irritation of nerve (Ochoa, 1994; Dawson et al., 1999).

We do not know for sure if the inflammatory changes occur in chronic and progressive compression (Schäfer et al., 2009). However experiments of chronic nerve compression have shown that the biological response of the nerve was similar to that found in the cuff experiments, i.e. early perineurial edema followed by a short-term macrophage recruitment and fibrosis (Mackinnon et al., 1994; Rempel and Diao, 2004). Furthermore in patients treated with release surgery for CTS the synovium surrounding the nerve contains more edema and fibrosis when compared to healthy controls (Rempel and Diao, 2004).

As a consequence it should be hypothesized that neural irritation, and subsequent increased mechanosensitivity, also occurs in chronic conditions, especially when acute episodes of nerve compression and ischemia are superimposed on chronic nerve compression.

Clinical indications of increased mechanosensitivity of the nervous system can be elicited through neurodynamic assessment (Elvey, 1979; Butler, 1991; Elvey and Hall, 1997; Shacklock, 2005). The theoretical hypothesis for neurodynamic testing is that a mechanical, electrical and chemical continuum exists in the nervous system (Butler, 2008). Neural containers or mechanical interfaces place stretch or compressive forces upon nerve (Butler, 2000; Shacklock, 2005), that can affect blood flow, produce inflammation, influence ion channels (Sunderland, 1978), or favor the development of mechanosensitive abnormal impulse generating sites (AIGS) (Devor and Seltzer, 1999). If the nerve has lost its ability to strain and glide, despite the lack of high strain values, forces that may have normally been displaced cannot be dispersed and more mechanical forces are placed on the AIGS (Butler, 2000).

In CTS ‘impaired nerve movement may cause increases in local strains sufficient to impair nerve conduction and blood flow and possibly cause abnormal firing and symptoms of pain and paresthesia’ (Dilley et al., 2003). This suggests a link between mechanics and metabolism of the nerve.

Neurodynamic tests evaluate the ability of the nervous system to elongate along a continuum of strain. In CTS a local compression as well as tension across other segment/s of the median nerve will reproduce patient’s known symptoms. As a consequence, the addition of nerve tension through elbow, shoulder and neck movements (Coppieters et al., 2009), may potentially modify the accuracy of a segmental provocation test. Moreover, connections between the nerves are common and the brachial plexus may be involved in CTS (Butler, 2000). Minor serial impingements along the median nerve (double crush hypothesis) also could provoke the nerve for an additive effect and trigger a distal neuropathy (Upton and McComas, 1973). According to this theoretical perspective, a double crush can be revealed through tension tests which involve several segments. However, it should be noted that several mechanisms, both central and peripheral, are involved in neuropathic pain and compression of nerves does not always cause pain (Schäfer et al., 2009). As a consequence, there is no scientific consensus on the role of nerve strain on painful responses and on the usefulness of neurodynamic tests in compression syndromes, in place of or together with a segmental provocation test.

Upper Limb Neurodynamic Tests (ULNTs), as described by Butler (2000), move the neural tissues and stimulate them mechanically, in order to gain an impression of their mobility and sensitivity to mechanical stresses (Shacklock, 1995). Among ULNTs, the ULNT1 emphasizes tension on the median nerve as shown by anatomic studies (Kleinsreink et al., 1995; 2000; Wright et al., 1996; Lewis et al., 1998) and is the most widely adopted test in the clinic.

Nevertheless, there is no complete consensus in the literature on the criteria for a positive neurodynamic test and on the movement sequence. The test is often considered positive when there is a reproduction of patient’s symptoms (e.g. pain, dysesthesia or paresthesia) and “if a movement of a body segment remote from the location of symptoms provoked in the neurodynamic test position alters the response,” modifying the symptoms or the elbow extension, often referred to as ‘structural differentiation’ (Coppieters et al., 2006). A further proposed criterion for test positivity consists in the presence of ‘differences in the test response between the involved and uninjured sides or variations from what is known to be a normal response in asymptomatic subjects’ (Nee and Butler, 2006). Moreover, there is no universally accepted procedure for this test with respect to the sequence of movements (i.e., from proximal to distal, or vice-versa).

In order to reduce false positives due to mechanical stimulation of other upper limb nerves (e.g. radial or ulnar nerve), Bickel (2010) recommends limiting the reproduction of symptoms for judging a test as positive only to the reproduction of neurogenic symptoms in the median sensory field of the hand (ie, first, second or third digit). As a consequence, a general lack of agreement across results in the literature has obstructed consensus on the validity and diagnostic utility of positive findings when the standard proximal to distal sequence of the ULNT1 described by Butler (2000) elicits the patient’s symptomatology.

With the aim to further investigate the usefulness of ULNT1 in the diagnosis of CTS, we conducted this prospective diagnostic test study to explore the validity of this test when used on adults with a suspected diagnosis of CTS using two different criteria sets.

2. Methods

2.1. Examiners

The team was composed of four people, an electrodiagnostic tester, a physician and two physiotherapists.

2.2. Subjects

Consecutive subjects with suspected CTS referred to the Clinic of Occupational Medicine of the University of Bologna (Italy) for median NCS were included in this study. Exclusion criteria were: upper limb joint pathologies which could significantly limit the range of motion (ROM) of the left or right upper limb, inflammatory, infective or systemic pathologies, history of surgical procedure...
for CTS, cervical radiculopathies, cognitive deficits. The more symptomatic limb was considered the involved limb for subjects with bilateral symptoms. This trial was approved by the Ethical Committee of the University Hospital S. Orsola-Malpighi of Bologna (Italy). All subjects signed an informed consent before commencement of the clinical tests.

2.3. Measurements

History and symptoms: Demographic and anthropometric data, current and previous medical history, location, quality and 24-h behaviour of symptoms were collected using an assessment form. Moreover, patients filled in the self-administered hand diagram developed by Katz and Stirrat (1990).

NCS: NCS were performed using Technique A on all patients following the previously described recommendations of the AAEM. Motor and sensory median NCS were conducted bilaterally using the segmental palmar technique (Kimura, 1978). Because NCS are affected by body temperature (Denys, 1991), both hands were warmed by immersion in warm water until they reached a palmar temperature of at least 32°C. All NCS were performed in a warm room (22–25°C). For the purpose of this study we used the combination of typical current symptoms (i.e. numbness, tingling, burning or pain in at least one of the first, second or third digit) and the reduction of SCV-WP as the referent case definition of CTS (Descatha et al., 2010). SCV-WP was considered “normal” when falling within the lower 99% confidence limit of the reference value described in an adult population between the ages of 15 and 50 years. i.e. >43.8 m per second (Kimura, 1979).

Cervical Provocation Tests and Cervical ROM measurement: The Spurling Test (Spurling and Scoville, 1944), the Neck Distraction Test (Cleland, 2005) and the goniometric measurement of cervical ROM (left and right rotation) were used to exclude cervical radiculopathies, according to the method proposed by Wainner et al. (2003). The goniometric measurement of cervical ROM was carried out using the G314S inclinometer, by Plasti, Milan (Italy).

Upper limb passive movement tests: Passive movement tests were performed in order to identify possible joint stiffness which could affect the available ROM during ULNT1 testing.

ULNT1: A biaxial electrogoniometer (model SG110, Penny and Giles Biometrics Ltd Gwent, UK) was used to measure the elbow ROM. The arms of the electrogoniometer were positioned on the lateral sides of the upper limb (arm and forearm, respectively). The cables connecting the electrogoniometer arms to the display unit were positioned proximally. Both arms and connecting cables were attached to the upper limb using adhesive tape and foam underwrap. The electrogoniometer was reset before each session.

2.4. Procedure

First, history and symptoms were collected by a physician and NCS were performed by an experienced electrodiagnostic tester. After obtaining informed consent, Spurling Test and Neck Distraction Test were performed by a physician, while the goniometric measurement of cervical ROM and passive movement tests were carried out by a physiotherapist trained in these procedures. A 20–30 min break was required between NCS and the ULNT1 in order to reduce the sensitization of the median nerve following electrodiagnostic studies.

Before the ULNT1 was performed, participants were informed about its characteristics and how to communicate symptoms to the examiner. Subjects used the verbal signal “stop” when numbness, tingling, burning or pain occurred. Subjects also described the type (i.e. stretch, tingling or pain) and location of symptoms and the possible modifications provoked by contralateral and/or ipsilateral cervical side-bending using the verbal signals ‘better’, ‘worse’ or ‘the same’. In response to questions during the medical history, subjects informed the physiotherapist only about their symptomatic or more symptomatic upper limb. Subjects were positioned in supine, without a pillow, close to the edge of a high/low plinth, with legs extended and the untested upper arm in neutral position.

The ULNT1 was performed as described by Butler (2000), first on the unaffected or less affected upper limb and subsequently on the affected or more affected one. The shoulder girdle of the tested arm was stabilized in a neutral position without any external device to control it. After this, the shoulder was abducted to approximately 110°, the wrist and fingers were extended, the forearm was supinated, the shoulder was laterally rotated, the elbow was extended, and the cervical spine was actively contralaterally and ipsilaterally flexed. Movements were performed to the end of range or until symptoms were produced. If symptoms were elicited during any step of the ULNT1, the test was stopped and structural differentiation was performed. If no symptoms were elicited, the test was done throughout the full available range. A second physiotherapist recorded the angular degree of the elbow at the final step, the location and type of provoked symptoms in both upper limbs, and the possible modifications produced by cervical movements.

All the examiners were blinded. The professional who collected historical data from subjects and performed the Spurling Test and Neck Distraction Test was unaware of NCS results. Likewise, the professional performing the upper limb passive ROM, cervical rotation ROM and ULNT1 was unaware of the subject’s medical history, clinical findings, NCS results, and goniometric measures. Moreover, the subjects were blind to their NCS findings.

2.5. Statistical analysis

All data were recorded in an electronic database. Confidentiality was ensured by keeping identifying data separated from all other data. Statistical analysis compared positive ULNT1 with the case definition of CTS. First, analysis was conducted according to Wainer’s criteria. The ULNT1 was considered positive in presence of at least one of the following: (1) reproduction of patient’s symptoms; (2) side-to-side differences (>10°) in elbow extension on completion of all motion sequences; (3) symptomatic limb side: contralateral neck side-bending increased symptoms or ipsilateral side-bending decreased symptoms (Wainner et al., 2005).

Second, analysis was repeated considering the same (2) criterion, but (1) and (3) criteria as positive only in presence of symptoms reproduction in the first, second or third digit of the affected arm. This choice was aimed at limiting the reproduction of positive symptoms only in the typical location of CTS.

Two-by-two contingency tables for ULNT1 results and CTS diagnosis were constructed: sensitivity, specificity, and likelihood ratios

<table>
<thead>
<tr>
<th>Characteristics of participants</th>
<th>Overall (n = 44)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No. females (%)</strong></td>
<td>33 (75.0)</td>
</tr>
<tr>
<td><strong>Age (yrs), mean (SD)</strong></td>
<td>46.3 (10.8)</td>
</tr>
<tr>
<td><strong>Height (cm), mean (SD)</strong></td>
<td>164.3 (8.1)</td>
</tr>
<tr>
<td><strong>Weight (kg), mean (SD)</strong></td>
<td>68.9 (14.3)</td>
</tr>
<tr>
<td><strong>BMI (Kg/m²), mean (SD)</strong></td>
<td>25.5 (4.5)</td>
</tr>
<tr>
<td><strong>No. Smokers (%)</strong></td>
<td>15 (34.1)</td>
</tr>
<tr>
<td><strong>No. work (%)</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Blue collar</strong></td>
<td>33 (75.0)</td>
</tr>
<tr>
<td><strong>White collar</strong></td>
<td>7 (15.9)</td>
</tr>
<tr>
<td><strong>Not employed</strong></td>
<td>4 (9.1)</td>
</tr>
<tr>
<td><strong>No. symptom duration (%)</strong></td>
<td></td>
</tr>
<tr>
<td>&lt; 1 month</td>
<td>1 (2.3)</td>
</tr>
<tr>
<td>1–3 months</td>
<td>5 (11.4)</td>
</tr>
<tr>
<td>&gt; 3 months</td>
<td>38 (86.3)</td>
</tr>
</tbody>
</table>
(LRs) with 95% confidence intervals were calculated. Sensitivity and specificity were calculated using the Wilson Method (Altman et al., 2000). The acceptable levels were set between 50% (unacceptable level) and 100% (perfect test) (Wurff Van der et al., 2000). In order to estimate diagnostic accuracy, +LR and −LR and their associated 95% confidence intervals were calculated using the score method.

The accuracy of a tested diagnostic procedure is commonly expressed in terms of positive and negative predictive values; however these values are dependent on the prevalence of the disease in the population being studied. Sensitivity and specificity are measures of test performance able to discriminate between subjects with disease and those who are free of disease. The sensitivity and specificity of an index test are always defined in comparison to the reference standard test that is considered correct according to clinical experience. Although the sensitivity and specificity of a test do not depend on the prevalence, they depend on the spectrum of patients across which the test is being evaluated. Consequently the LRs, rather than sensitivity and specificity, are the best way to compare two or more test in order to determine which one is the best for ruling in and for ruling out the disease. LRs can be also used to understand the relationship between pre-test probabilities, sensitivity and specificity, and the resulting probability of the disease after obtaining a positive or negative result.

A +LR from 2 to 5 yields small increases in the post-test probability of the target condition, from 5 to 10 moderate increases, and above 10 large increases (Grimes and Schulz, 2000). The −LR can vary from 1 to 0, with smaller values “better” in terms of the likelihood that an individual does not have the target condition (Riegelman, 2005). The +LR was calculated as sensitivity/(1−specificity) and the −LR was calculated as (1−sensitivity)/specificity (Altman et al., 2000). The diagnostic accuracy of the ULNT1 was considered satisfactory, thus affecting the probability of disease, with +LR 2.0 or −LR 0.50. Calculation of post-test probability was made by applying the likelihood ratio to the pre-test probability (prevalence in our sample) of disease. The software package used was STATA version 9.0 (Stata Corporation, TX).

### 3. Results

#### 3.1. Subjects

Our sample was composed of 47 subjects, 35 females and 12 males. 37 subjects reported bilateral symptoms. The characteristics of the sample for gender and age reflected the epidemiology of CTS in the general population; most subjects were female, adult, employed as blue collar workers and complaining of chronic symptoms. In three cases, ULNT1 results were not usable (one subject showed equally bilateral symptoms and signs, one subject did not complete the test, one showed symptoms and signs affecting the ulnar nerve) leaving 44 subjects available for study (see Table 1).

#### 3.2. Tests results

The results found on the basis of the case definition and those obtained from ULNT1 testing (using Wainner’s criteria as well as the additional criterion of reproduction of symptoms in the first, second or third digit of the affected arm) are illustrated in Table 2. Using Wainner’s criteria for a positive test, sensitivity of the ULNT1 was 91.67%, specificity was 15%, the +LR was 1.0784, and the −LR was 0.5556. When considering only the reproduction of symptoms on the first, second or third digit of the affected arm, results changed considerably. Sensitivity was sharply reduced to 54.17%, but specificity increased to 70%, while the +LR was 1.8056 and the −LR was 0.6548. Based on the prevalence or pre-test probability for CTS of 54% in this sample, the post-test probability of the target condition using the +LR, increased marginally to 56.4% using only Wainner’s criteria but increased to 68.4% using the new criterion. Using −LR to assess the probability of a patient having CTS if result of index test is negative, we found that post-test probability decreased to 44% under the condition of using the new criterion, and decreased to 40% using Wainner’s criteria (see Table 3).

#### 4. Discussion

We compared sensitivity, specificity, pre-test and post-test probability and LRs of two different application of the ULNT (index test) in the diagnosis of CTS using the combination of typical current symptoms of CTS (i.e. numbness, tingling, burning or pain in at least one of the first, second or third digit) and the reduction of SCV-WP as the referent case definition.

The sample population of the present study was similar for age, symptoms duration, rate of bilateral CTS, and severity of clinical presentation to that studied by Wainner et al. (2005), who found sensitivity = 0.75, specificity = 0.13, −LR = 1.9, and +LR = 0.86. Adopting Wainner’s diagnostic criteria (2005), our results appeared to follow the same trend (high sensitivity and low specificity). In the present study, we actually found a slightly higher sensitivity and better LRs. In particular, we obtained −LR value = 0.5556.

Adopting the other criterion to define positivity of the test, i.e. considering only the reproduction of symptoms in the first, second or third digit of the affected arm, results were inverted, showing low sensitivity and high specificity. However these LRs did not reach values of sufficient magnitude to establish the probability of the disease with any degree of certainty. In our sample the pre-test probability was 54% before any clinical testing. Using Wainner’s criteria (+LR = 1.07) the probability of a patient with positive ULNT1 having CTS increased up to 56.4%; after adopting the second criterion (+LR = 1.80), the probability of a patient with positive ULNT1 having CTS increased up to 68% (see Fig. 1).

Thus, the primary conclusion of our study relative to using the ULNT1 in clinical practice was that adding the new criterion improved +LR, but LRs remain unsatisfactory, because +LR is less

### Table 2

<table>
<thead>
<tr>
<th>Symptoms 1st, 2nd or 3rd digit</th>
<th>Positive CTS</th>
<th>Negative CTS</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wainner’s criteria</td>
<td>Positive</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>22</td>
<td>17</td>
<td>39</td>
</tr>
<tr>
<td>Negative</td>
<td>2</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>24</td>
<td>20</td>
<td>44</td>
</tr>
</tbody>
</table>

### Table 3

<table>
<thead>
<tr>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Likelihood ratio for positive test</th>
<th>Post-test probability for positive test (%)</th>
<th>Likelihood ratio for negative test</th>
<th>Post-test probability for negative test (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wainner’s criteria</td>
<td>0.9167 (0.741, 0.977)</td>
<td>0.15 (0.052, 0.360)</td>
<td>1.0784 (0.377, 3.083)</td>
<td>56.4</td>
<td>0.5556 (0.194, 1.588)</td>
</tr>
<tr>
<td>Symptoms 1st, 2nd or 3rd digit</td>
<td>0.5417 (0.351, 0.721)</td>
<td>0.70 (0.481, 0.854)</td>
<td>1.8056 (1.132, 2.879)</td>
<td>68.4</td>
<td>0.6548 (0.411, 1.044)</td>
</tr>
</tbody>
</table>
than 2.0 and $-LR$ is greater than 0.50. The results obtained using the new criterion were similar to those reported in previous diagnostic studies on other clinical tests for the diagnosis of CTS, which showed higher specificity than sensitivity (Aroori and Spence, 2008). However, neither our results, nor Wainner’s are in line with the findings of Coveney et al. (1997), who reported high sensitivity (82%), specificity (75%), and Positive Prognostic Value ($PPV = 93\%$). We suggest that the differences in results can be attributed to differences in exclusion criteria, recruitment sources, sample characteristics, or the test procedure.

Based on the fact that prevalence of CTS was 54% in our sample, we found that the post-test probability was lowered to 40% when using the Wainner criteria. Therefore the probability that CTS is absent after obtaining a negative result is 60% (see Fig. 1). Alternatively, using the second criterion i.e. only reproduction of the known symptoms in the first, second or third digit of the affected arm is a positive test, we obtained a post-test probability of CTS of 68%. Compared to the results reported by Wainner et al. (2005), our results show a slight improvement, but equally limited clinical usefulness.

Our study also offers some ideas for research on this clinical syndrome with an elusive definition. In the present study, the ULNT1 was performed adopting the standard sequence, from proximal to distal. The “reverse” test could in fact provoke a sensitization of the presumed source of the problem at the first step of the test and this early provocation of symptoms may obviate test completion. Future research could verify whether a distal initiation of the testing sequence alters the test accuracy.

The present investigation has some limitations including the relatively small sample, the potential bias in our recruiting strategies, and the sample’s specific characteristics. Most subjects had bilateral symptoms, thus failing to represent the general clinical population affected by CTS.

Furthermore, in our study population the vast majority of subjects reported long lasting symptoms, consequently our results could be applied only at chronic CTS patients, and they don’t provide information on the response to ULNT1 in cases of acute CTS. However the acute form, intended as a rapid and sustained increase of pressure in the carpal tunnel, is relatively uncommon (Aroori and Spence, 2008). Another issue that should be taken into account is the temporal placement of NCS prior to the clinical tests, that might have influenced the results of ULNT1 testing through a possible sensitization of the nervous system. For this reason, it might be interesting to repeat the study performing ULNT1 followed by NCS, which would be the more typical pattern of clinical examination and testing, followed by more definitive diagnostic laboratory work to confirm or reject a working hypothesis about the target condition.

5. Conclusions

Our investigation suggests that, based on our data, reproduction of the typical current CTS symptoms in the affected hand (or in the more affected one, in case of bilateral symptoms) during ULNT1 testing, slightly improves estimation of the probability of the presence of this condition. If we reproduce the typical current CTS symptoms in the affected hand (or in the more affected one, in case of bilateral symptoms) during ULNT1 testing, the probability of presence of this syndrome is about 70%. Moreover, if no symptoms are reproduced in the affected upper limb, no differences in the elbow ROM are observed, or cervical lateral flexions do not modify symptoms, the probability of absence of this syndrome is about 60%. Although our study may assist the interpretation of the ULNT1 in case of suspected CTS, the weak accuracy of this test suggests that it may not be used in isolation and must be interpreted in combination with other clinical findings and diagnostic studies. Future research to revised clinical prediction rules on CTS may be strengthened by using both criteria for a positive test.

Due to the actual inconsistency of findings reported in the literature regarding both the best ULNT1 procedure and the best...
criteria for positive and/or negative results, further investigations are needed with a clear interpretation and standardization of neurodynamic sequences to allow a comparison across several investigations.

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