THE IMPACT OF MUSCULAR VARIATION ON THE NEURODYNAMIC TEST FOR THE MEDIAN NERVE IN A HEALTHY POPULATION WITH LANGER'S AXILLARY ARCH

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ABSTRACT

Objective: The neurodynamic test of the median nerve (ULNT1) is frequently used to assess the mechanics and physiology of the brachial plexus and median nerve. The present study looks for a positive ULNT1 in a healthy population with Langer’s axillary arch (LAA) and analyzes whether LAA affects the elbow extension range of motion (EE-ROM) of the ULNT1.

Method: Of 640 volunteers screened, 26 LAA sides were finally included. Additional history taking revealed “minor symptoms” in some subjects. Minor symptoms do not qualify as a disorder because there is no interference with daily activities and no medical advice is sought. This study investigates whether the ULNT1 can (re)produce minor symptoms or abnormal responses in subjects with LAA. The EE-ROM was compared between the subjects’ left and right side, and the subtraction angle—which is the effect of placing the cervical spine in contralateral lateral flexion—was compared between LAA sides and controls.

Results: Langer’s axillary arch sides showed a significant increase in the occurrence of minor symptoms and positive ULNT1, but no influence was observed on the EE-ROM.

Conclusions: These findings suggest that LAA may be capable of transiently provoking the axillary neurovascular bundle. The unaffected EE-ROM may be the consequence of a vascular origin of the minor symptoms or the consequence of an ulnar nerve/medial cord response to the ULNT1. (J Manipulative Physiol Ther 2008;31:474-483)

Key Indexing Terms: Regional Anatomy; Brachial Plexus; Median Nerve; Neurovascular Syndrome; Diagnosis; Rehabilitation

Langer’s axillary arch (LAA) is a muscular arch in the axilla closely related to the underlying neurovascular bundle. It is an anomalous muscular variant that extends from the lateral edge of the latissimus dorsi across the axillary vessels and the distal brachial plexus into the tendon of insertion of the pectoralis major (Fig 1),1,2 but it

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can also attach to the coracoid process.\textsuperscript{1,3} This arch, which is the principal anatomic variation in the axilla, is present in approximately 7\% of the general population, although a varying incidence ranging from 0.25\% to 27\% has been reported.\textsuperscript{3,4}

Because of its anatomical features, LAA is tightened by abduction-external rotation and elevation of the shoulder, as has been shown during axillary surgery and on cadavers.\textsuperscript{5-8} When this occurs, LAA compresses the underlying neurovascular bundle and may cause neurologic and/or vascular symptoms.\textsuperscript{1,9} For this reason, it has been hypothesized that the arch can cause compressive disorders, which can be suggestive of thoracic outlet syndrome and should therefore be considered in the differential diagnosis of this syndrome.\textsuperscript{4,10-12} However, not all LAAs cause symptoms. Many are encountered during routine axillary surgery in patients with no history of upper limb neurovascular symptoms.\textsuperscript{13,14}

Little attention has been paid to the relationship between neurodynamic testing and congenital variations in the muscular anatomy of the upper limb. Therefore, in this study, we investigate the impact of LAA on the results of the upper limb neurodynamic test of the median nerve (ULNT1) in a group of healthy volunteers.

A first aim was to investigate the sensory responses of the ULNT1 in a healthy population with LAA. The purpose was to look for a positive ULNT1 that could indicate a possible interaction of LAA with the axillary neurovascular bundle followed by a transient disturbance of normal functioning of the latter.

Additional history taking after the initial screening process revealed that some of the included subjects showed “minor symptoms.” Minor symptoms are of such nature that they do not interfere with daily activities, that neither medical advice nor treatment is sought, and that they are not qualified as a disorder and therefore do not fulfill the exclusion criteria. Thus, the present study investigates in the subjects with LAA exhibiting minor symptoms whether these minor symptoms can be reproduced by the ULNT1. In the subset of subjects with LAA without minor symptoms, it will be investigated whether the ULNT1 produces abnormal sensory responses. In the early subclinical stage of nerve compression, positive responses to provocation testing such as the ULNT1 may be the only positive finding.\textsuperscript{15}

As well as for these sensory responses, there is a special interest in the elbow extension range of motion (EE-ROM). Despite the fact that “symptom reproduction” clinically is valued as a criterion of overriding importance in scoring the test, limited EE-ROM is also one of the identifying features of a positive ULNT1.\textsuperscript{16} The EE-ROM becomes a decisive factor when the reproduction of the patients’ complaints after the ULNT1 is absent and when a restricted EE-ROM is the only sign detected on the affected side as a possible “covert abnormal response.”\textsuperscript{17} Literature and clinical reports indicate the general agreement that there is no difference in EE-ROM between left and right sides in healthy populations, thus making it possible to compare to the contralateral side to detect asymmetries in EE-ROM.\textsuperscript{17-20} Byng,\textsuperscript{21} however, has found significant restrictions in EE-ROM in a healthy group of keyboard users, thus questioning the symmetry of EE-ROM in healthy individuals, particularly in specific subgroups. The participants with LAA comprise such a subgroup. Therefore the present study focuses as well on the EE-ROM, particularly in relation to the presence of a potentially harmful muscular variant (LAA). Hence, a second aim was to analyze whether LAA is capable of affecting the EE ROM.

METHODS

Screening

A total of 640 healthy students were screened for the presence of LAA (Fig 2). Each arm was placed consecutively on the examiner’s shoulder with extended elbow, neutral wrist, and shoulder at 90° flexed. Subsequently, the subject adducted the shoulder isometrically with submaximal force while the examiner palpated the medial border of latissimus dorsi toward the humeral insertion of the pectoralis major.\textsuperscript{22} In most of the cases, any LAA was clearly visible as an anomalous axillary mass.

Participants thus diagnosed as having LAA were subjected to an ultrasound investigation conducted by an experienced physician to assess the authenticity of the anomaly. A Sonoline Sienna ultrasound machine (Siemens, Erlangen, Germany) with a 7.5-MHz linear probe was used, and LAA was imaged in cross-sectional and longitudinal view, both contracted and relaxed.
Excluded from the study were subjects with nonmuscular arches or in whom there was doubt concerning the authenticity of LAA, subjects with a history of a musculoskeletal disorder in the upper quadrant within the previous 6 months, and subjects who were aware of having diseases closely associated with neuropathy or who had central or peripheral nervous system disease. Each subject underwent a physical examination and to be included in the study had to show a pain-free and full ROM of the upper limb joints, including the cervical spine. With the same criteria, a matched control group was created, composed of subjects without LAA as determined by manual examination of the axilla.

Of the 640 volunteers, 30 initially screened positively for LAA. Eleven of these were subsequently excluded after the ultrasound procedure because LAA was not confirmed. Of the 19 subjects remaining, none were excluded from the study on the criteria described above. In the control group, one participant was excluded because of medical treatment with a possible effect on the nervous system. Thus, 19 asymptomatic volunteers participated in the LAA group (8 males, 11 females; age [mean ± 1 SE], 20.55 ± 0.65 years; body mass index, 21.87 ± 0.52) and 17 asymptomatic participants volunteered for the control group (5 males, 12 females; age, 22.44 ± 0.33 years; body mass index, 21.40 ± 0.34). The Ethics Committee of the Ghent University approved this study and all 640 volunteers signed an informed consent form.

**Test Description**

The ULNT1 was performed bilaterally on each subject in 2 different test positions: cervical spine in neutral position (NL) and cervical spine in submaximal contralateral lateral flexion (CLLF), that is, maximal passive range of lateral flexion without provoking discomfort. For each position, the test was repeated 3 times. Before the test, the wrist was submaximally extended (total wrist extension minus 10°) through a thermoplastic splint.

The head and cervical spine were gently placed in position, and, with a custom-designed device, a vertical pad was positioned against the side of the head to prevent lateral flexion of the cervical spine toward the limb being tested. The shoulder was then abducted until the scapula started elevating. At this point, a horizontal adjustable pad was placed on top of the acromion, without applying any additional caudal pressure to prevent further scapular elevation. Subsequently, shoulder abduction was continued to end range, the forearm was supinated, and the shoulder laterally rotated, finally followed by elbow extension, the ROM of which was the principal measurement of the test (Figs 3 and 4).

Elbow extension was terminated by the examiner when maximal end resistance (R2) was reached. The participants were well informed about having the possibility to interrupt and determine the end of the test whenever “submaximal discomfort” occurred, which was defined according to Coppieters et al as “the maximal tolerance level for the test in view of the subject’s knowledge that the test has to be performed repeatedly.”

**Procedure**

The VICON optoelectronic system (ViDeo CONvector; Oxford Metrix, Oxford, UK) measured end ROM of elbow extension as the angle between the longitudinal axis of the forearm and the extension of the longitudinal axis of the upper arm. This angle was subsequently converted into the complementary angle and further referred to as the elbow extension ROM (EE-ROM).

To compare the EE-ROM between subjects, we used the subtraction angle which is the deficit of EE-ROM between both test positions; as a result, it basically expresses the effect of adding a sensitizing maneuver (ie, CLLF). Selvaratnam et al showed larger subtraction angles in a group with brachial plexus involvement compared to controls, suggesting that differences in these subtraction angles could be attributed to neural tissue tension. Besides, the subtraction angles are normalized values suitable for analysis between groups of individuals which would be difficult to achieve with EE-ROM because of its large natural intersubject variability (normal EE-ROM deficit, 16.5°–53.2°). Despite the fact that this procedure has been used successfully in previous studies, the authors believe that the characteristics of the subtraction angle need further investigation.

The order of the starting position was randomized between NL and CLLF to prevent any test bias. A 1-minute and a 3-minute interval were imposed between the test.
repetitions and change of the test position, respectively. All tests were administered by the same tester who was blind to the EE-ROM but not to the presence of the LAA.

Before starting the ULNT1 tests, the participants were subjected to additional history taking with special attention to vascular and neurologic signs and symptoms related to activities and sustained postures in daily life and sports. Because participants included in the study had no upper quadrant disorder, this additional history was focused on depicting minor but relevant symptomatology. The term minor symptoms was defined as nondisturbing signs or symptoms of vascular or neurologic nature, which nevertheless do not interfere with daily activities and sports to such an extent that no medical advice or treatment is sought. Minor symptoms are elicited in specific activities or postures—mostly including a glenohumeral abduction-external rotation component—and are characterized by low irritability, which means they almost immediately subside when the trigger is discontinued.

During each ULNT1, the elicited sensory responses were recorded on a body chart and afterwards compared to the results of the additional history. For participants with minor symptoms, the ULNT1 was scored as positive if these minor symptoms were partly or completely reproduced or when abnormal sensory responses were elicited in one or both test positions, and for “asymptomatic” participants if abnormal sensory responses were recorded in one or both test positions. Abnormal sensory responses are classically defined as those differing from normal responses to the ULNT1, which are (1) a stretch in the anterior shoulder region; (2) a deep stretch in the cubital fossa, extending down the anterior and radial aspects of the forearm into the radial aspect of the hand; and (3) a definite tingling sensation in the thumb and the first 3 fingers. Consequently, in this study a positive ULNT1 was determined independently of the EE-ROM. This also allows, if required, to analyze the interrelation of these 2 parameters which may further elucidate the underlying mechanisms of neurodynamic testing.

**Statistical Analysis**

The intraclass correlation coefficients were calculated as a measure of the intra-examiner reliability, using the 3 repetitions in each test position. For analysis, a linear mixed model was performed using subjects as a random factor. The intraclass correlation coefficient is defined as the ratio of intersubject variance over total variance. To analyze the categorical data, which is the occurrence of minor symptoms and positive ULNT1, a χ² test was used.

We analyzed the EE-ROM in 2 ways. The symbol (a) indicates analysis between both sides, intraindividually; (b) indicates analysis between LAA sides and control sides,
interindividually; and (a) and (b) further appears in the section “statistical analysis” to emphasize that this particular sentence (passage) refers to or is related to the intra- or interindividual approach. To resemble the ULNT1 standard procedure of comparing the patient’s EE-ROM to that of the least or nonaffected side, we analyzed the EE-ROM between both sides (a) with particular interest to the participants with unilateral LAA. Subsequently, the EE-ROM was also analyzed between LAA sides and controls (b): as mentioned above, for this purpose we used the subtraction angle.

We investigated whether EE-ROM between sides differed between subgroups (ie, controls, bilateral, and unilateral LAAs) and whether “hand dominance” influenced the EE-ROM. Our full model therefore included interactive effects of side and subgroups and the main effect of hand dominance. The level of significance was determined using a linear mixed model corrected for taking multiple measures on the same individual (a). Subsequently, an analysis of variance model was used to analyze the differences in subtraction angle between control sides and LAA sides showing minor symptoms and a positive ULNT1 (b). The EE-ROM was always assessed in both test positions, that is, NL and CLLF. The results below are presented as mean angles (±1 SE) and the level of significance was set at $P < .05$.

**RESULTS**

The participants were given the opportunity to end the test whenever they felt “submaximal discomfort.” However, in spite of this, not a single participant interrupted the test. Hence, the EE-ROM was measured at end resistance determined by the examiner. The mean intra-examiner reliability for measurement of EE-ROM among the 4 tests was 0.97 (range, 0.96-0.98), with a mean standard error of measurement of 1.9° (range, 1.6°-2.3°). A significant effect of hand dominance on EE-ROM was found and adjusted in

<table>
<thead>
<tr>
<th>Subdivisions</th>
<th>LAA</th>
<th>Distribution</th>
<th>Nature</th>
<th>LEFT Minor symptoms</th>
<th>LEFT ULNT1</th>
<th>Distribution</th>
<th>Nature</th>
<th>RIGHT Minor symptoms</th>
<th>RIGHT ULNT1</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Bilateral</td>
<td>Complete hand</td>
<td>Heaviness, numbness, pins and needles</td>
<td>0/0</td>
<td>Complete hand</td>
<td>Heaviness, numbness, pins and needles</td>
<td>0/0</td>
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<td></td>
<td>Bilateral</td>
<td>Ulnar/medial</td>
<td>Pins and needles</td>
<td>0/0</td>
<td>Ulnar/medial</td>
<td>Pins and needles</td>
<td>0/0</td>
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<tr>
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<td>Legacy</td>
<td>Complete hand</td>
<td>Coldness, numbness</td>
<td>0/0</td>
<td>Complete hand</td>
<td>Coldness, numbness</td>
<td>0/0</td>
<td></td>
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<tr>
<td>2</td>
<td>Right</td>
<td>–</td>
<td>–</td>
<td>0/0</td>
<td>Ulnar/medial</td>
<td>Numbness, coldness, heaviness, pins and needles</td>
<td>0/partial</td>
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<td>Bilateral</td>
<td>Ulnar</td>
<td>Pins and needles</td>
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<td></td>
<td>Left</td>
<td>Ulnar</td>
<td>Numbness, pins and needles</td>
<td>0/partial</td>
<td>Ulnar</td>
<td>Pins and needles</td>
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<tr>
<td></td>
<td>Left</td>
<td>Ulnar</td>
<td>Pins and needles</td>
<td>Complete</td>
<td>Partial</td>
<td>–</td>
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<td></td>
<td>Left</td>
<td>Median</td>
<td>Numbness</td>
<td>Partial</td>
<td>Complete</td>
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<tr>
<td></td>
<td>Left</td>
<td>Ulnar</td>
<td>Coldness, pins and needles</td>
<td>Complete/partial</td>
<td>–</td>
<td>–</td>
<td>0/0</td>
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<tr>
<td>3</td>
<td>Bilateral</td>
<td>–</td>
<td>–</td>
<td>Abnormal ulnar/abnormal ulnar</td>
<td>–</td>
<td>–</td>
<td>Abnormal ulnar/abnormal ulnar</td>
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<tr>
<td></td>
<td>Left</td>
<td>–</td>
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<td>Abnormal ulnar/abnormal ulnar</td>
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<td>–</td>
<td>Abnormal ulnar/abnormal ulnar</td>
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<tr>
<td>4</td>
<td>(n = 9)</td>
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<td>–</td>
<td>0/0</td>
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</table>

Ulnar indicates ulnar nerve cutaneous distribution; median, median nerve cutaneous distribution; abnormal ulnar, abnormal responses in the ulnar distribution; partial, partial reproduction of minor symptoms; complete, complete reproduction of minor symptoms; 0, negative ULNT1 test; NL/CLLF, neutral and contralateral lateral test position. Subdivision 1, minor symptoms without positive ULNT1; subdivision 2, minor symptoms with positive ULNT1; subdivision 3, no minor symptoms with positive ULNT1 based on abnormal ulnar; subdivision 4, no minor symptoms nor positive ULNT1.

* Subdivision 4 consists of 2 bilateral, 3 left, and 2 right LAAs (n = 9).
the statistical models used (mean restriction of 2.6° ± 1.7° on the dominant side, \( P < .006 \)).

**Minor Symptoms and Positive ULNT1**

Of the 19 participants with LAA, 12 had unilateral and 7 bilateral distribution, which results in a total of 26 LAA sides (Table 1). Figure 5 illustrates that 54% (14/26) of the LAA sides showed minor symptoms in comparison to 6% (2/34) of the control sides (\( \chi^2 = 11.23, P = .0008 \)). A positive ULNT1 was recorded in 42% (11/26) of LAA sides, which obviously differed significantly from the controls (\( \chi^2 = 11.62, P = .0007 \)); in the control group, no positive ULNT1 tests were registered (Fig. 5).

Hence, the sensitivity of the ULNT1—based on provocation of minor symptoms or abnormal sensory responses—in the present study’s population with LAA is 42%, whereas the specificity amounts to 96%. Of course, this high level of specificity has to be interpreted with care because it is to a large extent the result of using a control group consisting only of asymptomatic individuals.30 Of LAA sides showing minor symptoms, 57% (8/14) produced a positive ULNT1.

When the distribution and nature of the minor symptoms and the ULNT1 results were analyzed, 4 distinct patterns emerged, which could be classified into 4 subdivisions (Table 1). The first subdivision comprises LAA sides with minor symptoms but without positive ULNT1; the second subdivision contains LAA sides with both minor symptoms and positive ULNT1. In the third subdivision, LAA sides exhibit no minor symptoms but they do record a positive ULNT1 based on abnormal sensory responses, and in the fourth subdivision, LAA sides show neither minor symptoms nor a positive test.

**Elbow Extension Range of Motion and Subtraction Angle**

Table 2 summarizes the mean EE-ROM for both test positions in both sides. The mixed model showed no interaction effect for EE-ROM between side and subgroups tested in NL and CLLF (NL: \( F_{2,32} = 1.63, P = .21 \); CLLF: \( F_{2,32} = 2.63, P = .088 \)) (Table 2). More specifically, in the subgroup with unilateral LAAAs (Table 2, line 3), no significant difference was found for EE-ROM between LAA sides and their contralateral non-LAA sides (NL: \( t_{12} = 0.99, P = .32 \); CLLF: \( t_{12} = 1.32, P = .77 \); adjusted for multiple comparison by Tukey test).

Because no interaction was found between sides and subgroups in the previous mixed model (Table 2), no effect of LAA was expected on the subtraction angle. To verify and illustrate this inference, an analysis of variance was conducted post hoc which revealed no differences for the subtraction angle between LAA sides and controls (left side: \( F_{1,9} = 0.26, P = .61 \); right side: \( F_{1,23} = 2.06, P = .17 \)) (Table 3, rows 1 and 2).

Additional analyses were focused on LAA sides showing both minor symptoms and a positive ULNT1 to investigate whether only in this subset of LAA sides would the EE-ROM be affected.

The Wilcoxon signed rank test for paired samples showed no significant differences for the EE-ROM between unilateral LAA sides with minor symptoms and a positive ULNT1 and their contralateral “asymptomatic” non-LAA side with negative ULNT1 (NL: \( V_e = 4.00, P = .17 \); CLLF: \( V_e = 9.00, P = .34 \)) (Table 3, row 3).

Finally, a Wilcoxon-Mann-Whitney test revealed no significant differences for the subtraction angle between controls and LAA sides with minor symptoms and a positive ULNT1 (\( U = 126, P = .47 \)) (Table 3, row 3).

**DISCUSSION**

The main finding of the present study is that LAA is associated with increased occurrence of minor symptoms and positive ULNT1 without affecting the EE-ROM. Minor symptoms were found in 54% of LAA sides which differed significantly from controls (6%). Many cases have been described in which different anatomic variants can cause compression neuropathies in the median nerve. Among these variants are Struthers’ ligament, aberrant muscles in and around the carpal tunnel, and, of course, LAA itself.31–35 In most of these reports, the variants are encountered during

![Figure 5](image-url)

Fig 5. Significantly more minor symptoms and positive ULNT1 tests occur at LAA sides compared to controls. The “*” indicates a significant result (\( P < .05 \)).
surgical procedures releasing the nerve from compression. In the present study, however, subjects with LAA were selected from a healthy population. To the authors’ best knowledge, no previous reports exist of anatomical variants being related to minor symptoms in subjects selected from a large healthy population in a similar study design.

A positive ULNT1 was found in 42% of LAA sides in contrast with not one control side showing a positive test (Fig 5); the EE-ROM of LAA sides, however, showed no restrictions in comparison to controls or to the other side (Table 3). The present study is therefore also the first to report positive ULNT1 based on the (re)production of minor symptoms without limited EE-ROM in subjects with a muscular variant. Byng’s study is the only other finding of a positive ULNT1 in a group selected from a healthy population. In that study, the EE-ROM was found to be restricted in a healthy group of keyboard users when compared to healthy nonkeyboard controls. The fact that in the present study the EE-ROM was unaffected together with positive ULNT1s can possibly be attributed to the features of LAA.

Several hypotheses can be introduced as possible mechanisms which may be responsible for minor symptoms or “abnormal sensory response” (re)production. Because of its close relationship with the underlying neurovascular bundle, LAA may compress and induce friction to the neural tissues when it slides to accommodate upper limb movement. This process can irritate and sensitize the nerve, which actually increases its mechanosensitivity. According to the clinical features of the subjects with LAA presenting with minor symptoms in the current study, it is likely for the neural tissue to be only mildly sensitized. Such condition of mildly increased mechanosensitivity may be responsible for the occurrence and reproduction of minor symptoms but only when the peripheral nervous system is maximally loaded, that is, at fully extended EE-ROM regarding the ULNT1.

Based on the distribution pattern of the minor symptoms, especially in the second subdivision (Table 1), it is more likely that the ulnar nerve or medial cord is responsible instead of the median nerve. Kleinrensink et al reported that the ULNT1 caused higher tension in the medial cord than the “ulnar nerve ULNT” did. Shacklock reported abnormal responses elicited by the ULNT1 in a surgically proven ulnar neuropathy, which supports the results of Kleinrensink et al of neural force distribution in the upper limb. These findings can possibly explain why in the present study minor symptoms occurring in the ulnar distribution are reproduced by the ULNT1: if the median nerve is really not involved, it will not be restrained from maximal neural loading as was shown by the present results showing no restriction of EE-ROM. The median nerve can thus generate maximal tensile stress which is transmitted proximally along the nerve to the distal part of the medial cord. This part of the medial cord is also most likely the location where LAA applies compression, as a result of the distal slide of the neural tissue during this final phase of the test. In this way, tension and compression may interact to produce minor symptoms originating from the medial cord/ulnar nerve. This study confirms the reports that anatomical specificity of the ULNT1 is not absolute, as illustrated by sensory responses elicited in the ulnar nerve distribution.

Another possible explanation may be that the symptoms are of vascular origin. Wright describes a mechanism in which shoulder hyperabduction compromises the axillary arterial flow. Hyperabduction and glenohumeral abduction-external rotation are extreme positions of the shoulder adopted in daily life activities; as for example occupations that involve working with the arms overhead. Such positions can impair the circulation and, if maintained, lead to both neurologic disturbances and vascular symptoms which are intermittent but recur when the arm is taken into and maintained in this specific position.

The “elevated arm stress test,” consisting of 90° glenohumeral abduction-external rotation and scapular depression, is known to be a provocative maneuver for the neurovascular bundle in thoracic outlet syndrome. These positions are substantial components of the ULNT1: the starting position comprises the glenohumeral abduction-external rotation and the scapula is fixed in NL, which means that it is prevented from elevating while moving toward the end glenohumeral abduction which results in a scapular depression force. A hypothesis may be that a possible effect of the ULNT1 starting position together with the effect of LAA, which is tightened in this position, already results in impaired circulation leading to minor symptoms. This indicates that the remaining ULNT1 components (ie, forearm supination, wrist extension, and, finally, elbow extension, which are movements further loading the neural tissue) will probably have no contributory effect to the provocation of vascular minor symptoms and

### Table 3. Mean (±1 SE) EE-ROM° and mean (±1 SE) subtraction angle as a result of different analyses

<table>
<thead>
<tr>
<th></th>
<th>EE-ROM° NL</th>
<th>EE-ROM° CLLF</th>
<th>Subtraction angle°</th>
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<tbody>
<tr>
<td></td>
<td>LAA</td>
<td>Non-LAA</td>
<td>LAA</td>
</tr>
<tr>
<td>Right sides</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAA</td>
<td>8.5 (2.7)</td>
<td>4.9 (1.6)</td>
<td></td>
</tr>
<tr>
<td>Non-LAA</td>
<td>5.6 (2.6)</td>
<td>2.3 (2.1)</td>
<td></td>
</tr>
<tr>
<td>Left sides</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minor symptoms</td>
<td>160.3 (2.0)</td>
<td>162.3 (3.9)</td>
<td>156.1 (4.5)</td>
</tr>
<tr>
<td>and positive ULNT1</td>
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more importantly will not be affected. This may also explain
the present findings of minor symptom reproduction
without limited EE-ROM in subjects with LAA.

In minor peripheral neuropathic disorders, some exami-
nation procedures may need to be combined to reproduce
symptoms or abnormal responses. This principle occurs
when sustained compression is exerted on the nerve in a
neurodynamically loaded position, for example, carpal
compression test with wrist flexion.\textsuperscript{15,48} This cumulative
process of different mechanical parameters reaching symp-
tomatic threshold may happen when the ULNT1 is
performed in the presence of LAA, concerning both the
neurogenic and the vascular hypothesis.\textsuperscript{18,49,50} When the
possible effect of LAA’s compression is added to the effect
of maximal tension of ULNT1 in full EE-ROM, minor
symptoms may be (re)produced. Another example is the
compression of the third portion of the axillary artery by the
humeral head during 90° glenohumeral abduction-external
rotation.\textsuperscript{46,51} If this is cumulated with compression of LAA,
which is tightened in this position, circulation may become
impaired and symptoms occur.

Differentiating arterial symptoms from manifestations of
neurologic compression depending on the nature and
distribution of minor symptoms can be difficult especially
in the early stage. In this stage of vascular entrapment,
symptoms are mild and frequently intermittent in line with
the presence of LAA, concerning both the neurogenic and the vascular hypothesis.\textsuperscript{18,49,50} When the
possible effect of LAA’s compression is added to the effect
of maximal tension of ULNT1 in full EE-ROM, minor
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Differentiating arterial symptoms from manifestations of
neurologic compression depending on the nature and
distribution of minor symptoms can be difficult especially
in the early stage. In this stage of vascular entrapment,
symptoms are mild and frequently intermittent in line with
the results of the present study.\textsuperscript{52,53} Different observers
report numbness, coldness, paresthesia, color change of the
skin, and dull diffuse pain as the main early vascular
symptoms occurring in one or more fingers or in the
complete hand.\textsuperscript{46,54,55} Nevertheless, interpretation of these
symptoms can be very confusing as coldness, numbness,
and heaviness have also been reported to be of neural origin.\textsuperscript{52}
The clinical presentation in the present study is highly
variable, and, therefore, both systems may be responsible,
depending on which parts of the brachial plexus are involved
and to what extent the circulatory system is involved.

Finally, the present study shows that LAA, with or
without minor symptoms, tends to have no impact on EE-
ROM. Therefore, the hypothesis that LAA could induce
asymmetry in EE-ROM has to be rejected. At the same time,
symmetry in EE-ROM has to be interpreted critically if only
because the present study found a significant effect of hand
dominance. The EE-ROM of the dominant side is restricted
to 2.6° in comparison to the nondominant side. This effect of
hand dominance on EE-ROM is important clinically but
even more important from a research perspective and should
be corrected for in the statistical models used.

Given that LAA is found in approximately 7% of the
general population, approximately one of 15 patients visiting
the physiotherapist for all kinds of complaints has LAA
probably without even knowing it. Langer’s axillary arch can
be superimposed to other neurogenic or nonneurogenic
disorders and in that way be presented to the physiotherapist.
Besides, it is possible that LAA presents at the nonaffected side
on which the ULNT1 is also performed as standard procedure.
Therefore, whenever the ULNT1 shows results similar to
the present study it is advisable to screen the axilla for LAA.

There are some limitations in our methodology which
need to be mentioned. First, the tester was not blind to the
presence of LAA. This is a possible source of bias during
additional history taking and in judging the ULNT1. Secondly, if the present minor symptoms are considered to
be of neural origin it is more likely that the ulnar nerve is
responsible instead of the median nerve according to the
symptom distribution pattern which became clear during
additional history taking. The physical examination during
screening did not include glenohumeral instability testing
which may have been a possible source of bias because the
ULNT1 starting position could produce signs and symptoms
in unstable shoulders similar to those in the present study.

The results of the present study show an association
between the presence of LAA and the occurrence of minor
symptoms. There were significantly more positive ULNT1
tests in LAA sides (42%) than in controls (0%) while EE-
ROM was not restricted.

These results suggest that LAA may be capable of
transiently provoking the axillary neurovascular bundle. The
unaffectected EE-ROM may be the result of a possible vascular
origin of the minor symptoms or, in the scope of a
neurogenic theory, may be a typical consequence of an
ulnar nerve/medial cord response to the ULNT1, which is
essentially median nerve biased.

When ULNT1 results are encountered similarly as in the
present study, it is advisable to screen the axilla for LAA
which, if present, can explain otherwise confusing results.
Future research should focus on LAA in relation to the ulnar
nerve assessed with the ulnar nerve neurodynamic test
(ULNT2). The possible association between LAA and the
axillary artery should be further explored by means of Doppler ultrasound.

### Practical Applications

- Langer’s axillary arch may cause neurologic (and
vascular) disorders in the upper limb; however,
each arch may not be symptomatic.
- The elbow extension range of motion (EE-ROM) of
the neurodynamic test of the median nerve (ULNT1) is not influenced by Langer’s axillary
arch in healthy subjects.
- There is a significant increase in the occurrence of
minor symptoms and positive ULNT1 tests (based
on minor symptom reproduction or abnormal
sensory responses) in healthy subjects with Lan-
ger’s axillary arch compared to controls.
- The occurrence and the (re)production of minor
symptoms by ULNT1 may be of vascular and/or
neurologic origin.
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REFERENCES


