Mechanosensitivity of the median nerve and mechanically produced motor responses during Upper Limb Neurodynamic Test 1

Shapour Jaberzadeh a,∗, Sheila Scutter b, Homer Nazeran c

a Department of Physiology, Adelaide University, Adelaide, SA 5005, Australia
b School of Physiotherapy, University of South Australia, Adelaide, SA, Australia
c School of Informatics and Engineering, Flinders University, Adelaide, SA, Australia

Abstract

Objectives In spite of the widespread use and recognised importance of the Upper Limb Neurodynamic Test 1 (ULNT1) in clinical practice, controversy remains about the neurophysiological basis for sensory and motor responses to the test. The aims of this study were to determine the effects of two limb positions (neutral and ULNT1) on mechanosensitivity of the median nerve, and to investigate the mechanisms underlying muscle stiffness and loss of range during the elbow extension component of the ULNT1.

Design In both limb positions, a KIN-COM® dynamometer controlled passive elbow extension at 3°/second, and recorded both elbow extension range of movement and elbow flexor resistive torque.

Setting Electrophysiology laboratory, School of Physiotherapy, University of South Australia.

Participants Twenty-six asymptomatic subjects.

Main outcome measures Range of elbow extension and elbow flexor resistive torque. Surface electrodes placed over 10 upper limb muscles recorded electromyographic (EMG) activity. Using a hand-held micro-switch, participants indicated occurrence of pain onset and pain that limited further movement.

Results The median nerve was more sensitive to mechanical longitudinal stresses during passive elbow extension in the ULNT1 position than in the neutral position, as demonstrated by increased EMG activity and increased mean elbow flexor resistive torque (neutral position, 0.8 Nm, 95% confidence interval 0.7–0.9 Nm; ULNT1 position, 3.9 Nm, 95% confidence interval 3.8–4.0 Nm). Pain onset and pain limit occurred earlier in range in the ULNT1 position (pain onset, 45°, 95% confidence interval 40–50°) than in the neutral position (3°, 95% confidence interval 0–6°). In the ULNT1 position, EMG activity was greater in muscles responsible for an antalgic posture in the upper limb, although some EMG activity was evident prior to pain onset.

Conclusions The mechanosensitivity of the median nerve was greater during elbow extension in the ULNT1 position than in the neutral position. The increased EMG activity that occurred primarily in muscles contributing to an antalgic posture with concurrent changes in resistive torque and range of movement may be explained by a flexor withdrawal response to pain that acts to reduce the stretch on the median nerve. Muscle activity prior to pain onset is probably due to mechanoreceptor activation following preferential stretch of the median nerve.

© 2004 Chartered Society of Physiotherapy. Published by Elsevier Ltd. All rights reserved.

Keywords: Upper limb tension test; Neurodynamic tension test; Mechanosensitivity; Median nerve; Flexor withdrawal reflex

Introduction

Over the last two decades, physiotherapists have developed examination techniques known as ‘neural tension tests’ to assess the mechanosensitivity of the major nerve trunks and their central connections [1–4]. The techniques for the upper limb are based on the premise that upper limb movements require neural tissues to move, adapt and extend in length [5], and use precise and sequential positioning of the limb to apply tension to selected neural components. For example, to tension the median nerve using the Upper Limb Neurodynamic Test 1 (ULNT1), the sequence and positions described by Butler [3] are as follows: shoulder girdle depression, glenohumeral abduction to 110°, forearm supination, wrist and finger extension, and glenohumeral external
rotation, with elbow extension being the final component of the test. It is theorised that the ULNT1 will move and exert longitudinal stress on the median nerve and its proximal nerve roots and the cervicobrachial plexus [6,7], with associated sensory and motor responses providing information about the mechanosensitivity of the median nerve. Anatomical studies have shown the anatomical validity of this test [8–11].

Despite the widespread use of neural tensioning techniques, little is known about whether neural tissues become more sensitive to loading in the ULNT1 position compared with a neutral position (arm resting in 30°). It is, therefore, important to explore the responses by comparing mechanosensitivity in neutral and ULNT1 positions using the final component of ULNT1, passive elbow extension. Mechanosensitivity can be assessed by monitoring changes in elbow extension range and elbow flexor resistive torque during elbow extension, and the corresponding mean electromyographic (EMG) responses of selected shoulder and arm muscles [12].

The aims of this study were to determine the effects of two limb positions (neutral and ULNT1) on mechanosensitivity of the median nerve, and to investigate the mechanisms underlying motor responses during the elbow extension component of the ULNT1. Greater mechanosensitivity would be indicated by increased EMG activity and elbow flexor resistive torque, decreased elbow extension range and pain that occurred earlier in range.

**Method**

Twenty-six asymptomatic normal subjects (13 males, 13 females), aged 21–72 years (mean: 39.6 years; S.D.: 15.5), participated in the study. All subjects were right handed and gave written consent.

A KIN-COM® dynamometer (125 AP, Chattex Corporation, Chattanooga, TN, USA) was used for measurement of elbow extension range and resistive torque. An integrated multi-channel AMLAB®-based system (AMLAB International, Sydney, Australia) was developed to simultaneously record multi-channel EMG activity and to integrate resistive torque, range of movement [13]. Bipolar surface electrodes (Duo-Trode® Myo-Tronics Inc., USA) were secured over 10 shoulder and arm muscles referenced to anatomical landmarks [14]. The raw EMG signals were differentially amplified in the AMLAB® system with a common-mode rejection ratio of 120 dB. Each amplifier was individually isolated and had a high input impedance (500 MΩ) and low noise (<1 μV). The EMG signals were band-passed filtered between 10 and 500 Hz. These signals were amplified with a gain of 3000 and sampled at 1000 Hz. The digitised data were then imported into an Excel spreadsheet. The EMG signals were full wave rectified and the mean EMG for 3 seconds before and after movement onset, pain onset and pain limit were calculated.

The muscles were classified into two groups according to their function: agonists (experimental muscles) and antagonists (control muscles) for an upper quarter antalgic posture. The antalgic posture of shoulder girdle elevation, shoulder adduction with medial rotation and some degrees of elbow and wrist flexion [15] may reduce upper limb pain by shortening the anatomic course of the median nerve and relieving tensile stress. Based on this premise, the experimental muscles selected were upper and middle fibres of trapezius, pectoralis major, biceps brachii, flexor carpi radialis and brachialialis. The control muscles were triceps, deltidoid, infraspinatus and the lower fibres of trapezius. The reference electrode was a lip clip [16].

**Subject positioning and stabilisation**

Stabilisation and control of the subjects’ position during testing was achieved using the following apparatus: (1) subject stabilisation baseboard; (2) range-of-motion-control device; and (3) adjustable seat/plinth assembly of the KIN-COM dynamometer. Subjects were positioned supine on the subject stabilisation baseboard with a chinstrap to prevent upper cervical extension. The finger and wrist splint of the range-of-motion-control device was then placed on the posterior surface of the wrist and hand with the hinge overlying the subject’s axis of wrist flexion/extension. The KIN-COM lever arm axis was then adjusted to the range-of-motion-control device elbow axis. The final position of the subject’s arm prior to testing in the neutral position was with the arm resting in 30° of abduction, 90° of elbow flexion, the forearm supinated, and the wrist and hand in neutral. The final position of the subject prior to testing in the ULNT1 position was with the arm in 110° of abduction and full lateral rotation, the elbow in 90° flexion, the forearm completely supinated, and the wrist and hand in full extension. In this position, the length of brachialialis and flexor carpi radialis are almost identical compared with the neutral position. There is some degree of stretch in the other four experimental muscles.

**Passive elbow extension**

Once in the test position (neutral or ULNT1), passive elbow extension was controlled at 3°/second by the KIN-COM dynamometer to minimise the effects of the myotatic stretch reflex [17]. In the neutral position, a single familiarisation trial of elbow extension was taken to the point of pain onset. One further trial to the point of pain limit was conducted for data collection. During this trial, passive elbow extension, elbow extension range, elbow flexor resistive torque and EMG activity of shoulder and arm muscles were recorded. Once resting EMG symptoms were recorded, the arm was placed in the ULNT1 position in the following sequence [3]: (1) application of a constant shoulder girdle depression force of 40 mmHg [18]; (2) abduction to 110°; (3) forearm supination;
(4) wrist and finger extension; and (5) glenohumeral external rotation. Subjects practiced using the hand-held microswitch to indicate pain during a familiarisation test, and were instructed to activate the switch ‘as soon as the tightening sensation changes to pain’ (pain onset) and ‘as soon as the pain changes to the maximum level of pain a physiotherapist is prepared to provoke’ (pain limit). This point is well within, and quite different from, a level representing intolerable pain for the subject [19].

Each component was performed manually to the onset of pain. The stretch was slightly reduced and the range was held by tightening the locking screws of the range-of-motion control device. This kept subjects pain free before adding the subsequent components to complete the ULNT1 position. Subjects could stop the test at any point in range by using a separate hand-held switch. To avoid a sequencing bias, the order of testing in neutral and ULNT1 positions was randomised.

Results

Data were retrieved offline. Elbow extension range and elbow flexor resistive torque at movement onset, pain onset and pain limit in the neutral and ULNT1 positions are shown in Fig. 1.

In both the neutral and ULNT1 positions, elbow flexor resistive torques at pain onset and pain limit were significantly larger than at movement onset (two-way repeated measure ANOVA, P < 0.001).

Fig. 1. Elbow extension range of motion (A) and elbow flexor resistive torque (B) at movement onset, pain onset and pain limit in the neutral and ULNT1 positions.

Fig. 2 shows resistive torque and EMG responses in the ULNT1 position in one representative subject.

There was increased EMG activity in all experimental muscles, except flexor carpi radialis, before the onset of pain that further increased after the onset of pain. With the exception of triceps, activity in the control muscles remained very low.

To allow comparison of data at equivalent ranges in the neutral and ULNT1 positions, 3-second mean EMG in the neutral position was determined at the ranges that corresponded to movement onset, pain onset and pain limit in the ULNT1 position. While most of the progressive increase in activity before pain onset to pain limit occurred in the experimental muscles, particularly the upper fibres of trapezius, a similar pattern of activity was also evident in triceps. Note that activity in all muscles declined once the stretch was removed after pain limit (two-way repeated measure ANOVA, P < 0.001). Pairwise comparison of mean differences for 3-second mean EMG before and after movement onset, pain onset and pain limit during passive elbow extension in different shoulder and arm muscles using Tukey’s Honestly Significantly Different (HSD) test supports the significant changes in experimental muscles and triceps after movement onset (Fig. 3).

Discussion

Effects of shoulder and arm position on mechanosensitivity of the median nerve

It was hypothesised that the median nerve in the ULNT1 position would be more mechanically sensitive than in the neutral position of arm and shoulder joints. This study demonstrated that the elbow extension range in the ULNT1 position is halted by pain in the middle range of joint excursion, and is associated with an increase in elbow flexor resistive torque and EMG activity of the experimental muscles. Therefore, the median nerve is more mechanically sensitive in the ULNT1 position. Elbow extension stresses the median nerve by elongating the nerve itself, the proximal nerve roots and the brachial plexus [8–10,20]. Clinically, elbow extension in the ULNT1 position has been shown to be limited when the median nerve is involved in carpal tunnel syndrome [21].

Specific analysis of the EMG responses during elbow extension in the ULNT1 position revealed a complex interaction in which the muscle activity may be involved to protect the nervous system from tensile forces. Before movement onset, the EMG activity of shoulder and arm muscles reflected relaxed muscles. Mean EMG activity after pain onset increased in all experimental muscles and in the triceps brachii. This indicated that submaximal pain and/or tensioning of continuous structures, such as the median nerve, elevated the EMG activity in the experimental muscles.
Fig. 2. Resistive torque and EMG responses in the ULNT1 position in one representative subject.
In the neutral position, during passive elbow extension, elbow flexor resistive torque and EMG activity of the experimental muscles remained unchanged, whereas in the ULNT1 position, there were significant increases at pain onset and pain limit. This suggests that continuous structures, such as the median nerve and/or the vascular system and muscular fascias [22], may be more mechanically sensitive in the ULNT1 position. This sensitivity is likely to predispose recruitment of muscles that are responsible for the antalgic posture of the shoulder and arm.

Elbow extension in the neutral and ULNT1 positions was performed very slowly (3°/second) to prevent reflex activation of the elbow flexor muscles. Therefore, through-range or end-range resistance during passive elbow extension in the ULNT1 position may be a combination of visco-elastic properties of lengthening tissues such as elbow flexor muscles, the median nerve trunk [8–10] or the subclavian artery [23].

The pattern of EMG activity in the triceps muscle was different to the other control muscles. This muscle showed a slight increase in EMG activity, especially at pain limit. This
finding may be explained by the occurrence of an inverse myotatic reflex. In this reflex, with increasing elbow extension range, the resistance in elbow flexor muscles increases, stimulating the golgi tendon organs. In the spinal cord, golgi tendon organ input via Ib afferent fibres is transmitted to interneurones that turn off the elbow flexor muscles and turn on the triceps muscle. This may serve to increase the mechanical stiffness [29] and may provide a protective function for the elbow joint [30].

In the experimental muscles, the highest and lowest EMG changes occurred in the upper fibres of trapezius and flexor carpi radialis, respectively. Sunderland [5,31] reported that shoulder girdle depression produced greater strain on the C4–C7 nerve roots and roots of the brachial plexus compared with the strain produced on these nerves by shoulder abduction, external rotation or elbow extension. Since shoulder depression is a key movement to increase the anatomic course traversed by the brachial plexus, the upper fibres of trapezius work to elevate the shoulder during elbow extension in the ULNT1 position. This action may decrease the anatomic course of the median nerve and reduce tensile stress on the affected nerve trunk and roots.

An explanation for low EMG activity of the flexor carpi radialis muscle may be that, unlike other experimental muscles which are generally prime movers of their designated movements, this muscle is not a prime mover for wrist flexion. Full wrist extension increases tension significantly in the middle and distal part of the median nerve [8,9]. Therefore, it seems that wrist flexion has less effect on relieving stress in the proximal part of the median nerve and affected nerve root during the test.

The brachialis muscles showed greater EMG activity than biceps brachii during passive elbow extension in the ULNT1 position. Basmajian and Latif [32] determined that brachialis is the primary flexor of the elbow. Compared with biceps, which can potentially act as a shoulder/elbow flexor and forearm supinator, the brachialis muscle is a pure elbow flexor regardless of forearm position. Second, brachialis is a one-joint muscle and as such is usually recruited first [33]. Third, brachialis is generally active during most elbow joint movements and postures [33], whereas biceps is more active during quick elbow flexion with an added load [30].

The relative increase of EMG activity from movement onset to 3 seconds before pain onset suggests that the increase in EMG activity of the shoulder and arm muscles during elbow extension at the ULNT1 position is not solely the result of pain. This finding is of considerable importance since it suggests that a number of mechanisms may contribute to the EMG responses of muscles during elbow extension in the ULNT1 position. Other mechanisms linking the muscle activity with neural tissue tension include mechanical sensitivity of ganglion cell bodies [34], and existence of mechanoreceptors in the thoracolumbar spinal cord [35] and peripheral nervous system [36]. Parke and Whalen [35] and Theophilidis and Kiartrizis [36] contend that the somatic nervous system has normal mechanisms that protect the nerve against tensile stresses. Therefore, muscle-pain interaction via the nociceptive-mediated flexor withdrawal reflex may not be the only mechanism responsible for EMG activity of shoulder and arm muscles during passive elbow extension in the ULNT1 position. This finding supports Balster and Jull [28] who concluded that mechanosensitivity of peripheral nerves in asymptomatic subjects is not solely mediated by pain.

The findings of this study have implications beyond neurodynamic testing of the upper limb. They provide a better understanding of the underlying mechanisms for pain and its related muscular activity as two key parameters during assessment and treatment of neural structures. Understanding of these mechanisms during neural tensioning manoeuvres has implications for the management of patients with neck and upper limb pain and dysfunctions such as peripheral nerve or brachial plexus injury, whiplash injury and cervical spondylosis. These findings do not support using techniques, such as nerve (and muscle) stretching, which only address the mechanics of the nervous system. Techniques that respect the mechanosensitive state of the nervous system would be more appropriate. Mobilisation of peripheral nerves by gentle sliding techniques [37] short of reflex muscle contraction [1] could be advocated. These gentler techniques may prevent re-inforcement of pathophysiological changes and responses in the central nervous system processing in symptomatic subjects.

Key messages

- The increase in mechanosensitivity of the median nerve in the ULNT1 position supports the premise that neural tissues are tensioned during ‘neural tension’ tests. Increased muscle activity evoked during the ULNT1 may be a flexor withdrawal response to pain that acts to indirectly protect the median nerve by preventing further tensioning via an antalgic posture.
- Activation of muscles prior to pain onset may indicate a peripheral nerve mechanoreceptor response to preferential stretching of the median nerve during the ULNT1.

Ethical approval: Human research ethics committee, University of South Australia.

Funding: Faculty of Health and Biomechanical Sciences, University of South Australia.

References


