THE NEURAL TISSUE PROVOCATION TEST
AS A DIAGNOSTIC TOOL IN THE ASSESSMENT OF CERVICOBRACHIAL PAIN DISORDERS:
A CRITICAL APPRAISAL

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DECLARATION

This work has not previously been accepted in substance for any degree and is not being concurrently submitted in candidature for any degree.

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STATEMENT 1

This dissertation is being submitted in partial fulfilment of the requirements for the degree of MSc in Pain Management.

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This dissertation is the result of my own independent work/investigation, except where otherwise stated. Other sources are acknowledged in brackets giving explicit references. A reference list and bibliography is appended.

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SUMMARY

This thesis will analyse research that is concerned with assessing the reliability and validity of the neural tissue provocation test for the upper quadrant as a diagnostic test. The neural tissue provocation test is a technique that is used to identify the presence of sensitised peripheral nerves. It consists of a sequence of multiple joint movements that may provoke sensory responses in individuals with sensitised neural tissue by elongating the length of the nerve bedding.

In clinical practice patients frequently present with diffuse symptoms in their neck and upper extremity of unknown aetiology, and report of positive symptoms associated with peripheral nerve injury. Peripheral nerves with relative minor damage to their nerve fibres are characterised by an increased mechanosensitivity and may react to mechanical stimulation with sensory responses and with impaired compliance to movement. Assessing the mechanosensitivity of the neural structures by neurodynamic tests is a relatively new but increasingly important technique among orthopaedic physical therapists. In response to contemporary calls for the use of evidence-based-medicine and for professional accountability a growing body of scientific evidence has emerged creating the grounds for the use of this test in clinical practice.

On the basis of the reviewed anatomical and clinical evidence the median biased neural tissue provocation test can be proposed as a useful diagnostic test within the assessment of cervicobrachial pain disorders. However, reports on low reliability are due to handling irregularities when performing this complex multiple joint test, which calls for clearer operational definitions and for a standardisation in its execution.

ACKNOWLEDGEMENTS

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LIST OF ABBREVIATIONS

ANOVA	Analysis of variance
BPTT	Brachial Plexus Tension Test
CBD	Cervicobrachial Pain Disorder
CLF	Cervical lateral flexion
CCLF	Contralateral cervical lateral flexion
CSF	Cerebrospinal fluid
CTS	Carpal Tunnel Syndrome
C5	Fifth cervical spinal nerve root
Cx	Cervical spine
DF	Dorsal flexion
EBM	Evidence based medicine
EMG	Electromyography
G/H	Glenohumeral joint
IASP	International Association for the Study of Pain
ICLF	Ipsilateral cervical lateral flexion
ICC	Intraclass correlation coefficient
IVF	Intervertebral foramina
L5	Fifth lumbar segment
mm	Millimetres
Mr	Moment of stretched tissue
M1	Onset of muscle activity
NTPT	Neural tissue provocation test
P1	Onset of pain
P2	Maximum pain tolerance
RCT	Randomised clinical trial
ROM	Range of motion
RSI	Repetitive strain injury
R1	Onset of resistance
R2	Maximum resistance
SBK	Screen based keyboards
SD	Standard deviation
SEM	Standard error of measurement
SLR	Straight leg raise
S1	First sacral segment
TPT	Thermal pain threshold
T1	First thoracic spinal nerve root
ULNT1	Upper limb neural test base test
ULNT2a	Upper limb neural test N. medianus bias
ULNT2b	Upper limb neural test N. radialis bias
ULNT3	Upper limb neural test N. ulnaris bias
ULTT	Upper limb tension test
VAS	Visual analogue scale

1. INTRODUCTION

Pain is central to the practice of physiotherapy being the most frequent symptom for which patients seek healthcare and the most common cause of physical dysfunction (Simmonds 1999). Therefore an adequate assessment of pain and its underlying pathology is essential for an efficient therapy. In this respect providing an adequate assessment depends on the accuracy of the physical examination and on the efficacy of diagnostic tests.

Physiotherapists have been specialising in assessing movement disorders of the neuro-musculoskeletal system. It is still common belief that the rehabilitation of physical dysfunctions mainly involves treatment of muscles and joints. However, the examination for normal compliance to movement and abnormal responses to mechanical provocation of the neural tissue has been an integral part of the experienced physiotherapist's assessment (Elvey 1986, Kenneally *et al.* 1988, Butler 1991). The former theory of considering the examination and rehabilitation of neural tissue disorders from a biomechanical perspective (Elvey 1979b/1986/1995, Butler 1989), which has now developed into a more neurophysiological approach (Shacklock 1995, Wright 1999, Butler 2000), has moved into focus and is currently the subject of investigation. Since many treatment approaches in physiotherapy have been developed empirically, the need to scientifically validate assessments and treatment regimes has long been recognised.

When patients present with pain conditions in the upper extremities and neck, in which mechanosensitive neural tissue is considered to be the primary feature, the term cervico-brachial pain disorder has been advocated (Allison *et al.* 2002). During patient assessment a physiotherapist develops hypotheses about possible causes or diagnoses for the presenting problem. These hypotheses are then tested in the physical examination in which special

diagnostic tests are used (Davidson 2002). A diagnostic test seeks to determine whether a person has a particular condition or whether this condition can be ruled out. In correspondence to the straight leg raise (SLR) of the lower quarter a testing procedure for the neural tissue has been developed for the upper quarter, the neural tissue provocation test. When Elvey (1979b) first conceptualised the physical examination of the neural tissue, in the investigation of arm pain and regional upper quarter pain syndromes, the predominant thought behind the concept was for a better understanding and differential diagnostics of upper arm pain. Until then reliable diagnostic procedures for the interpretation of somatic referred pain to the shoulder and arm had not been defined.

One of these neuromusculoskeletal dysfunctions then under investigation was the whiplash syndrome, which at that time appeared to be a relatively minor trauma that may progress into arm pain (Hammacher & van der Werken 1996). Standard clinical examination for possible referral of pain from the cervical spine comprised muscle power testing, reflex and sensory testing, as well as nerve conduction testing. If, however, the standard clinical examination failed to reveal definite positive signs, such as positive neurological deficits or reproducibility of pain by cervical spine tests, confusion arose as to the probable medical explanation for the symptoms. Hence, aim was to overcome the difficulty in "determining the primary pathology" (Elvey 1979a, p.113).

The same difficulty of identifying the primary disorder holds true for minor nerve disorders, in which normal nerve conduction is not impeded and therefore conduction abnormalities on electromyographical recordings are not necessarily evident (Dyck 1990), as for example in the early stages of the Carpal Tunnel Syndrome (CTS). However, it has been shown that minor nerve disorders, as in injured or inflamed peripheral nerves, are characterised by an

increased sensitivity to mechanical load (Greening & Lynn 1998). It is this mechanosensitivity that provides the means of clinical assessment that is utilised by physiotherapists by the application of neural tissue provocation testing.

Since the original description of the 'brachial plexus tension test' by Elvey (1979b) the test has evolved and is now better known under the terms 'upper limb neural test' (Butler 1991), 'neurodynamic test' (Shacklock 1995), or 'neural tissue provocation test' (Elvey 1995, Hall *et al.* 1998, v. der Heide *et al.* 2001). The provocation tests for the cervicobrachial and equally for the lumbosacral plexuses have been described in the past and have been used in clinical practice for the past 20 years (Elvey 1979b, Maitland 1986, Butler 1991), however most tests had been developed by empirical methods and are now scrutinized for their scientific and clinical validity.

In response to contemporary calls for the use of evidence-based practice and for professional accountability an increasing number of randomised clinical trials (RCTs) are appearing in the literature, which assess the efficacy of manual therapy procedures (Jull & Moore 2002). Evidence based medicine (EBM) is the conscientious, explicit, and judicious use of current best evidence in making decisions (Sackett *et al.* 1996), by integrating individual clinical practice with the best available external clinical evidence from systematic research. One of the goals of EBM is to evaluate the accuracy and precision of diagnostic tests, the power of prognostic markers, and the efficacy and safety of therapeutic, rehabilitative and preventive regimens (Rosenberg & Donald 1995). Furthermore, there is an ethical responsibility to recognise and consider the current scientific evidence as it relates to the therapeutic techniques and interventions we are using on a daily basis (Turner & Whitfield 1997, Matheson 2000). This way, knowledge of the current literature can help clinicians to avoid

the act of performing treatment techniques out of tradition without questioning the rationale behind their treatment decisions.

In general there is an urgent need to further investigate the effects of manual therapy both to validate its clinical application, as well as to develop a basis for a neurophysiological model explaining pain relieving effects (Zusman 1992). Cervicobrachial pain sufferers frequently seek manual therapy treatment for relief despite little evidence to date to substantiate its effects or to determine its efficacy (Zusman 1994/1995, Aker *et al.* 1996, Dreyer & Boden 1998, Gross *et al.* 2002, Hoving *et al.* 2002). In this respect there has been an effort in the last years to find scientific proof for these methods (Zusman 1994, Wright & Vicenzino 1995, Vicenzino *et al.* 1995/1996, Gifford & Butler 1997, Sterling *et al.* 2001, Zusman 2002).

In the past years an increasing amount of scientific evidence has emerged that investigated the validity of the neural tissue provocation test in the assessment of patients with neural tissue disorder in cervicobrachial pain. The aim of this thesis is to present a critical appraisal of this research. This work does not claim to be a complete state of the art review but tries to give an overview on the effort physiotherapy has made to verify this method. The first part of this paper portrays the relevant neuroanatomical and neurophysiological features that play a role in the development of cervicobrachial pain disorders. Then a short introduction into the history of neural tension testing is given followed by the main body of this paper, which is concerned with the scientific evidence for the use of neural tissue provocation testing for the upper limb as a diagnostic tool in cervicobrachial pain disorders.

For this purpose the initial proposition that the upper limb neural test can selectively load the nervous system (Elvey 1986, Selvaratnam *et al.* 1989), and that limitations in movement are

caused by the nervous system as a continuous structure (Yaxely & Jull 1993, Coppieters *et al.* 2001b), will be under investigation. Finally, the scientific support for the use of neural tissue provocation tests in clinical practice and implications will be discussed. To prevent confusion through different terminologies that are found throughout the literature I will refer to all upper quarter neural tissue provocation tests in chapter 5 as the Upper Limb Neural Test (ULNT) except where stated otherwise.

The computerised searches have been conducted via MEDLINE, Winspirs, PeDro, and the Cochrane Data Base. Additionally conference proceedings, and references from primary articles were checked. Through the kind help of a co-student I was also able to access material on early ULNT research, which is dated but was perceived to include key papers. Inclusion criteria for the literature were German and English language, textbooks and reviews were accepted for the anatomy section but were kept to a minimum for the ULNT evaluation. To better relate the research to current practice normative ULNT studies on asymptomatic and symptomatic subjects were limited to be no older than 1990. The key search words used singularly and in combination were 'upper limb tension test', 'brachial plexus', 'cervicobrachial pain', 'neurogenic pain', and 'manual therapy'. For this literature review the critical analysis framework by Rees (1997, Appendix A) was used to quantitatively analyse the literature, although it was not possible to describe all aspects of the framework due to word allowance. A more detailed description of the single studies discussed in this thesis is presented in the tables in Appendix B.

The time scale used to select the main body of the literature ranges from 1990-2003, but more dated work was included to insure the development of neural tissue examination in physiotherapy would be covered adequately. Seminal works from Hromada (1963), and

Sunderland (1961/1968) were included as they laid the foundation to most of the neuroanatomical research today. The greatest part of the literature has been acquired through the medical and surgical libraries of the University Clinic Charité of the Humboldt-University Berlin, the state library of Berlin, the UWCM library in Cardiff, and with the help of colleagues. A limitation to the search presented itself through the fact that most studies were published in physiotherapy and physiotherapy related journals, which can be valued as a form of publication bias (Dickersin 1990).

2. EPIDEMIOLOGY OF CERVICOBRACHIAL PAIN

Neural tissue provocation testing of the upper limb has been developed to assess the presence of neural tissue involvement in cervicobrachial pain disorders (v. der Heide *et al.* 2002). Patients frequently seen in physiotherapy praxis often present with diffuse pain in the arm and neck without recalling any previous trauma, and first tend to wait for the pain to regress by itself. From my experience patients finally seek medical treatment after several weeks of persistent sometimes increasing pain, and are subsequently sent to physiotherapy after some weeks of initial treatment by their general practitioners.

Due to the lack of population-based studies the precise incidence of cervicobrachial pain is not known (Hall *et al.* 1997). The Task Force on Epidemiology of the International Association for the study of Pain (IASP) has so far published individual studies on neck pain and shoulder pain (Crombie *et al.* 1999), however, to my knowledge no epidemiological study has investigated cervicobrachial pain as such. This may be partially due to the wide array of terms that have been used to describe populations of patients with neck pain and related disorders, such as 'upper extremity disorders', 'cervical osteoarthritis', 'tension neck syndrome', 'cervical spondylosis' and 'occupational cervicobrachial disorders' (Ariëns *et al.* 1999). As neck pain and cervicobrachial pain (CBP) are closely related, and may even have been used synonymously in research, some information will be given on the prevalence and rehabilitative measures of neck pain to help establish a clearer picture on cervicobrachial pain disorders.

Neck disorders and upper extremity pain are not only common in the general population, but can be disabling and costly (Dreyer & Boden 1998, Gross *et al.* 2002). An investigation on the cost-of-illness of neck pain in The Netherlands in 1996 revealed that the share of these

costs were about 1% of the total health care expenditures and 0.1% of the Gross Domestic Product (Borghouts *et al.* 1999). There are increasing reports of pain, discomfort and dysfunction of the neck and upper extremities associated with repetitive physical work and stress (McPhee & Worth 1988, Karjalainen *et al.* 2000ab). By now it is well accepted that upper extremity and neck disorders are seen in workers who undertake light repetitive work in fixed postures, as in keyboard operators, which requires continuous stabilising around the shoulder girdle, that may even lead to repetitive strain injuries (Quintner & Elvey 1993, Grant *et al.* 1995). Even though neck pain is usually not life threatening for the patient, it may cause pain and restrictions in daily activities. The socioeconomic consequences for those taking prolonged sick leave or receiving disability pension may also lead to substantial personal suffering (Jensen *et al.* 1995, Ariëns *et al.* 1999).

In general, relatively few consistent data are available on the prevalence of neck pain in the general population, ranging from as low as 9.5% to 35% (Ariëns *et al.* 1999). The large variation in the prevalence estimates of research may be explained by the differences in definitions and inclusion criteria. In some studies acute and chronic neck pain were included, in others only chronic pain patients. Also the source of the samples and their age distribution varied considerably (Ariëns *et al.* 1999). In the Norwegian population about one third of adults will experience neck pain in the course of one year (Bovim *et al.* 1994) with a prevalence for chronic pain of 13.8%.

Other authors report of a prevalence of neck pain in the general population of The Netherlands ranging from 10% to 15% (Borghouts *et al.* 1999). A prospective longitudinal study of a general population sample initially free of neck pain in the United Kingdom reported that the 1-year cumulative incidence of neck pain was 17.9% (Croft *et al.* 2001).

Epidemiological studies that have investigated the occurrence of neck pain in the general population have also shown that women have a higher chance of developing neck pain than men and that prevalence rises with age (Bovim *et al*. 1994, Borghouts *et al*. 1998/1999, Croft *et al.* 2001).

Since upper extremity pain often occurs in conjunction with cervical disorders it is difficult to differentiate and categorise the symptoms into separate clinical disorders. Dwyer *et al*. (1990) noted that neck pain is a poorly understood symptom and clinical interpretations most often ascribe it to putative 'disc disease' or 'soft tissue injury'. In the cervical region, neck pain arising from the zygapophysial joints, ligaments, muscles and intervertebral discs may be accompanied by pain perceived in the head, shoulder girdle, upper limb, and the chest (Dwyer *et al.* 1990, Bogduk 1994, Selvaratnam *et al.* 1994), thereby providing a more complex diagnostic problem because pain can arise locally or can be somatically referred pain. Referred pain is defined as pain perceived in a region separate from the location of the primary source of pain (Bogduk 1994).

Also muscular pain (Travell & Simons 1983) and pathology from deep somatic tissue may elicit referred pain obscuring the clinical picture. It is therefore imperative to undertake differential diagnostics to rule out any serious underlying pathology such as Pancoast tumour, coronary insufficiencies, fractures after trauma, or systematic inflammatory disorders before referring patients to physiotherapy. The aim of the physiotherapist's assessment, however, is to differentiate between the potential somatic sources that can refer pain into the neck and upper extremity.

Since there are many treatments available and accepted as standard in the conservative management of cervicobrachial pain a systematic review assessed the efficacy and

effectiveness of treating mechanical neck pain (Aker *et al.* 1996). 24 RCTs that met the selection criteria were categorised by their type of intervention (9 manual therapy, 12 physical medicine, 4 drug treatment, 3 patient education). What became clear from this overview was the lack of evidence for many standard approaches used in health care today. Even for those treatments that showed some early evidence of support, such as manual treatments, implications remained inconclusive due to the small number of trials on which they were based. Apart from the low statistical power of studies differences in defining diagnostic criteria for cervicobrachial pain syndromes (Koes *et al.* 1992abc, Levoska *et al.* 1993, Ekberg *et al.* 1994) may equally impede comparison of results, thereby limiting implications that could be made for clinical practice.

Additionally a blinded review study that investigated the effectiveness of manipulation and mobilisation of the spine for back and neck complaints found that most of the 35 RCTs that were reviewed showed methodological flaws and were of poor quality (Koes *et al.* 1991). The most common problems identified were that diagnostic categories for subject inclusion were often ill defined and non-specific, treatments lacked operationalising, outcome measures were not blinded, and that small sample sizes and the lack of describing drop-outs made it difficult to detect treatment differences. Noticeably, only 5 of the 35 studies were on neck pain, which additionally had the lowest scores on methodology.

Obviously these two systematic reviews disclose the dilemma of research on the efficacy of conservative treatment of CBP disorders. First of all the definitions of inclusion criteria are much too broad and so miss out on subgroups that may profit from specific interventions. On the other side the interventions themselves are not well defined and often mix specific and unspecific treatments, such as manual therapy and exercise programmes. This way no

conclusive statements can be drawn from these kinds of studies or worse, the lack of evidence for treatment efficiency portrays the inadequacy of most research designs. Consequently, physiotherapists will have to develop their own studies designed to fit the demands of their profession.

3. PATHOBIOLOGICAL ASPECTS OF CERVICOBRACHIAL PAIN

Critical to the physiotherapeutic management of patients with upper quarter disorders is an understanding of the underlying pathophysiological mechanisms. It has been proposed that increased mechanosensitivity of upper quarter neural tissue plays an important role in the pathogenesis of cervicobrachial pain (Shacklock 1995, Hall *et al.* 1997, Greening & Lynn 1998, v. der Heide *et al.* 2002). This means that any mechanical stimulation, be it pressure or movement, can trigger a pain response in sensitised neural tissues. Since the neural tissue provocation test (NTPT) claims to transmit tension from the peripheral nerves to the cervical nerve roots (Selvaratnam *et al.* 1989, Elvey 1995, Butler 1991), thereby assessing the neural tissue as a source of pain, particular emphasis is given to the biomechanical relevant anatomy of the nerve roots and peripheral nerve trunks, as well as to the movement relationships within neural tissues and to their surroundings.

3.1. ANATOMICAL AND BIOMECHANICAL CONSIDERATIONS

One of the most frequently used interpretations of CBP is, that it may be caused by nerve root compression (Bogduk 1994). Due to the close proximity of the nerve roots within the spinal column to the discus intervertebralis and to the bony structures of the intervertebral foramina (IVF), nerve roots can be subjected to mechanical compression, deformation or stretch associated i.e. with disc herniation, osteophytes of either the uncovertebral region or the zygapophyseal joints, or with spinal stenosis.

The spinal nerve roots may be well protected against external trauma by their surrounding structures, but because they do not possess the same amount of protective connective tissue as the peripheral nerves, they are more vulnerable to interspinal mechanical deformations.

The connective tissues of the cervical nerve root can be distinguished from the peripheral nerve in two main aspects. In the peripheral nerve the perineurium, which envelopes each fascicle or nerve bundle, splits into two parts, from which most of it merges with the dura mater and only a few layers merge with the pia mater to make the sheath of the nerve root (Haller & Low 1971), a thin membranous structure which is permeable to the cerebrospinal fluid (CSF). Furthermore, the epineurium of the peripheral nerve is continuous with the dura mater, leaving only the endoneurium to continue from the peripheral nerve to the nerve root (Hasue 1993).

Insofar, the nerve root lacks both epineurium and the tough perineurial sheath exposing the constituent nerve fibres more than those of peripheral nerves, and rendering them more susceptible to mechanical deformation (Sunderland 1968, Rydevik *et al.* 1984). Secondly, nerve roots are more vulnerable to traction and compression injury because their nerve fibres are arranged in parallel non-plexiform bundles, which are loosely held together by fewer and finer collagen fibres of the endoneurium compared to those of the peripheral nerve (Sunderland & Bradley 1961b, Sunderland 1968, Hasue 1993).

The nerve root complex adapts to the movement of the extremities and spine by stretching and slackening and possibly by some sliding in the IVF (Rydevik *et al.* 1984). When approaching the IVF the nerve roots are in close relation to the pedicles (Rydevik *et al.* 1984), therefore compression of the nerve roots depends on the effective space available within the IVF, which might decrease through articular degeneration as well as through physiological movements of the vertebrae. Moses and Carman (1996) conducted a detailed investigation on the topography of the fifth, sixth and seventh cervical nerve roots in association to their structural surroundings. The study showed that significant attachments to the walls of the

IVF existed. Posteriorly, at the medial end of the foramina, the nerve roots attach to the periosteum of the inferior pedicles, and to the capsules of the zygapophysial joints. Anteriorly, they attach to the vertebral bodies and the intervertebral disc by lateral extensions of the posterior longitudinal ligament (Moses & Carman 1996).

This means that lateral displacement of the nerve root complex in the IVF is limited rendering it more vulnerable to mechanical compression within the IVF by space-occupying pathologies (i.e. osteophytes or disc herniation). If, in case of such a scenario, the nerve root becomes irritated, the neural tissue provocation test should be able to detect a sensitivity to mechanical load (more details on the testing procedure in chapter 4). However, the sensitising of neural tissue can occur without compression through inflammatory or chemical reactions. A compression type radiculopathy may however, present with paraesthesia and pain and must not necessarily lead to an impairment of normal compliance to movement (v. der Heide 2003, personal communication).

Peripheral nerves on the other hand are relatively resistant to mechanical load, which originates in the highly differentiated construction of their surrounding connective tissue. The peripheral nerve consists of sensory, motor and sympathetic nerve fibres. Within both motor and sensory nerves myelinated Aβ- and Aδ-fibres, and unmyelinated C-fibres are present in a ratio of 1:4 (Mackinnon 2002). Myelinated and unmyelinated nerve fibres are packed within endoneurial connective tissue and bundled into fascicles. These fascicles are surrounded by the perineurium, a "relatively thin but distinctive lamellated sheath of connective tissue composed of tightly packed collagenous and elastic fibres, which are arranged about the fascicles circularly, obliquely and longitudinally" (Sunderland & Bradley 1961a, p.109). The perineurium is the main component giving tensile strength and elasticity

to the nerve trunk, and also constitutes a diffusion barrier for several substances including proteins, which helps to preserve intrafascicular pressure (Sunderland 1968/1990). However, stretching might damage the perineurium and affect its permeability.

The perineurium is like a tube surrounding the fascicles, which permits some movement of the nerve fibres inside the fascicle (Sunderland 1968/1990). The connective tissue matrix that lies between the fascicles is termed the internal epineurium. The external epineurium is the outer connective tissue that supports and protects all the fascicles of a given nerve, and carries the vascular vessels (Mumenthaler & Schliak 1977). The entire nerve trunk is surrounded by the mesoneurium with its extraneural gliding surface called adventitia that permits excursion of the nerve trunk during joint motion (Sunderland 1968, Mackinnon 2002). This extraneural gliding surface together with the intraneural sliding of fascicles against each other in deeper layers, present the normal gliding mechanism during joint motion (Sunderland 1968, Rempel *et al.* 1999), which are the mechanisms under investigation when applying the NTPT.

Wilgis and Murphy (1986) reported normal gliding properties of the median and ulnar nerve trunk in fresh intact adult cadaver to be 7.3 mm and 9.8 mm respectively, during full flexion and extension of the elbow (Table 1). The extent of nerve excursion just proximal to the wrist was even more pronounced with 14.5 mm (range 11-17 mm) and 13.8 mm (range 10-15 mm) respectively. Observations on the marked median nerve excursion during upper limb movement seemed to show consistency with these findings. In an in vivo study by McLellan and Swash (1976) measurements of the deflections of a needle electrode, inserted through the skin into the median nerve at a point half way between the elbow and the shoulder, indicated a mean nerve excursion value of 7.4 mm (range 2.8-20 mm) during wrist and

finger extension (Table 1). The authors further estimated that there was 10-15 mm of excursion at the wrist during wrist and finger hyperextension, but gave no details on the exact position of the elbow.

Unfortunately, neither one of the studies reported on the position of the shoulder girdle during measurements, which has been demonstrated to influence the tension in the neural tissue (Selvaratnam *et al.* 1989, Kleinrensink *et al.* 1995b/2000, Coppieters 2002a), nor how elbow and wrist movements were combined, which leaves interpretation of the data to be incomplete. Moreover, the shrinkage and stiffness (i.e. rigor mortis) of tissues in human cadaver have to be taken into account when comparing the results between in situ and in vivo studies. Although the results seem to show some agreement, and measurements were taken at similar sites (upper arm or wrist) the position of the elbow during measurement was not clearly defined so that these data need to be corroborated by studies with exact operational definitions.

Wright *et al.* (1996) accounted for these positions and found that in 90º shoulder abduction, 10º elbow extension, and 30º forearm supination the mean total excursion of the median nerve at the wrist was 19.6 mm (Table 1). Moving the shoulder and the elbow induced marked excursion of the median nerve at the elbow of 9.1 mm and 12.3 mm respectively, but not at the site of the wrist. Recent measurements of the longitudinal median nerve motion distal to the elbow joint using spectral Doppler sonography (Hough *et al.* 2000) claim to have confirmed the values of nerve excursion reported by McLellan and Swash (1976) with a mean of 8.5 mm (range 6.2-13.7 mm). However, the remarkable large variations in the individual range of measured median nerve excursion reported by McLellan and Swash (2.8-20 mm) and Hough *et al.* (6.2-13.7 mm) have not been addressed so far.

TABLE 1: Measurements of mean nerve excursion at various sites			
Site of measurement	**N. medianus**	**N. ulnaris**	**N. radialis**
Total longitudinal excursion (*in situ)* measured <u>proximal to the elbow</u> (Wilgis & Murphy 1986)	7.3 mm (during full flexion to extension of the elbow)	9.8 mm	-
Distal longitudinal excursion (*in vivo*) measured in the <u>upper arm</u> (McLellan & Swash 1976)	7.4 mm (during wrist/finger extension to flexion)	-	-
Total longitudinal excursion (*in situ*) measured at the wrist <u>proximal to the carpal tunnel</u> (Wilgis & Murphy 1986)	14.5 mm (during full flexion to extension of the elbow)	13.8 mm	5.8 mm (with ulnar to radial deviation)
Distal longitudinal excursion (*in vivo*) measured at the <u>wrist</u> (McLellan & Swash 1976)	10 -15 mm (during wrist and finger hyperextension)	-	-
Total longitudinal excursion (*in situ*) measured at the wrist <u>proximal to the carpal tunnel</u> (Wright *et al.* 1996)	19.6 mm (from 60º wrist extension to 65° wrist flexion)	-	-
Distal longitudinal excursion (*in vivo*) measured <u>distal to the elbow </u>(Hough *et al.* 2000)	8.5 mm (during wrist extension from neutral to 60°)	-	-

An interesting finding was that when positions of the joints were combined there was a mean total of 35.4 mm of median nerve excursion (distal and proximal excursion combined) at the wrist (Wright *et al.* 1996). If nerve movement was restricted at one location this would lead to increased neural tension or stretch away from the site of compression, and could explain the diffuse symptoms frequently reported in entrapment neuropathies such as the CTS (McLellan & Swash 1976, Wright *et al.* 1996). Magnetic resonance scans on patients with work related repetitive strain injuries confirmed this, and showed reduced median nerve excursion in the carpal tunnel during wrist movement (Greening *et al.* 1999). Furthermore, the potential surgical implications of restricted nerve motion caused by adherence of the nerve to surrounding tissue have been emphasised (Wilgis & Murphy 1986). Therefore, the

restoration of the longitudinal sliding mechanism is said to be essential to ensure effective surgical treatment. These findings have led to the development of specific nerve gliding exercises in the form of gentle movement of the nerve within its nerve bed that reduce adhesions (Totten & Hunter 1991, Rozmaryn *et al.* 1998).

Another important factor in peripheral nerve anatomy is that the fascicles do not run independently along the entire length of the nerve but are repeatedly dividing and uniting to form complex fascicular plexuses (Sunderland & Bradley 1961a, Sunderland 1968) providing the nerve trunk with the capability to endure much higher loads than the nerve root. Additionally the nerve trunk runs in an undulating course within its nerve bed, as well as the fasciculi within the epineurium, and the nerve fibres inside the fasciculi (Sunderland 1968).

Under normal circumstances with increasing extension peripheral nerve trunks first straighten out their resting undulation in the nerve bed, whereby the nerve fibres, which are arranged in a spiral fashion, are able to untwist without altering the length or tension of the individual fibres inside the fasciculi (Sunderland 1968/1990). This is then followed by a slide and glide of the nerve trunk in relation to its nerve bed, adapting to positional changes of the upper limb (McLellan & Swash 1976). These observations led Zöch (1992) to investigate the lengthening properties of the median nerve in a human cadaver model with the following questions in mind:

1. What kinds of length differences exist in the nerve bed during full range movements?
2. Does a peripheral nerve adapt by stretching or by relaxing in the nerve bed?
3. How is the tension distribution in relation to the length of the nerve?

The interesting results of this morphological study showed that in maximal wrist and elbow extension and shoulder abduction the nerve bed was 4% longer than the nerve itself, whereas in maximal flexion and adduction the nerve bed was 15% shorter. Zöch confirmed that the median nerve itself adapts to full flexion of the joints by taking up a wavy pattern within the nerve bed. This procedure accounts for approximately 77% of the length difference of the median nerve from full flexion to full extension, whilst the missing 23% have to be achieved by the elastic properties of the nerve itself (Zöch 1992), bearing in mind that this process is only possible if the gliding movement within the nerve bed is not restricted by adhesions.

However, the lengthening properties of nerves have been stated in the literature with quite some discrepancy. According to Zöch (1992) the elongation is distributed evenly throughout the nerve and equals an average of 4% of its original length. The normal maximal stretching ability has been stated by Zöch to lie at 6%, however, normal daily activity usually never exceeds the value of 4% stretching. Only in the case of adhesions or fibrosis of the gliding tissue the even distribution of tension is prevented. This means the nerve distal to the adhesion has to compensate for the loss of stretching ability and might exceed the critical value of 6-8%, which in turn might lead to vascular morphological changes (Zöch 1992). As the fasciculi are stretched, their cross-sectional area is reduced (Sunderland & Bradley 1961a), which leads to an increase in intrafascicular pressure, whereby nerve fibres are compressed and microcirculation is compromised (Lundborg & Rydevik 1973).

In contrast, Sunderland and Bradley (1961a) have described the mean percentage of elongation at the elastic limit of the median nerve in 24 unembalmed human specimens to have a mean average of 14.9 ± 3.9% (range 10-22%). In this study elasticity was defined as "that property of a material which enables it to return to its original form and shape when the

external load is removed" (Sunderland & Bradley 1961a, p.108). They stated that slowly stretching the nerve leads to considerable lengthening without apparent damage. Greatest elongation at the elastic limit of a nerve trunk was reported to be in the order of 20%, whereas complete mechanical failure was observed at 30% stretch of its original length.

The significant differences in results between Zöch (1992) and Sunderland and Bradley (1961a) are partly due to the different starting lengths used, and partly due to the fact that Sunderland and Bradley conducted their research on isolated nerves subjected to progressively increasing loads to the point of mechanical failure. Zöch (1992) on the other hand used an intact nerve in situ without destroying its nerve bed and cutaneous nerve branches that presented a form of natural fixation to the surrounding, which explains the much lower values. In general, the data provided through these cadaver studies have to be regarded as approximate values when compared to the mobility and stretching capabilities of the neural tissue in living subjects, but nevertheless demonstrate the movement dynamics of neural tissue.

3.2. NEUROPHYSIOLOGICAL CONSIDERATIONS

The previous section explained the mechanisms and importance of uncompromised gliding of the nerve trunk in its adaptation to normal movement of the spine and extremities. If adhesions hinder this physiological movement and the even distribution of tension in the nerve, intraneural microcirculation could be impaired. However, nerve function as well as communication and nutritional transport systems (antegrade and retrograde transport) depend on an adequate supply of oxygen to the nerve fibres.

The peripheral nerve contains a well developed microvascular system with vascular plexuses in all of its connective tissue layers (Rempel *et al.* 1999). These vessels have a coiled configuration so that blood flow is not impaired during normal gliding of the nerve trunk (Mackinnon 2002). As the vessels reach the nerve trunk they enter the epineurial space, where there is considerable plexus formation, and run longitudinally in various layers of the epineurium. The vessels pass the perineurium obliquely to enter the endoneurium where there is only a fine network of capillaries (Mackinnon 2002). As the endoneurial space has no lymphatic vessels, oedema within the endoneurium could lead to increased endoneurial fluid pressure in the fascicles, which could interfere with the vulnerable microcirculation (Rempel *et al.* 1999). Nerve roots, ganglia, and spinal nerves on the other hand receive their blood supply from segmental arteries and medullary vessels. Nutrients may be transported to the nerve roots both by the intrinsic blood vessels and via diffusion from the CSF (Rydevik 1992, Hasue 1993).

The intraneural blood flow was investigated experimentally by Lundborg and Rydevik (1973) in a rabbit tibial nerve model. During controlled elongation of the nerve it could be demonstrated that venular stasis was induced when the nerve was stretched to about 8% (range 5-10%) over its original length, and if maintained for a longer period of time would give rise to continuous impairment of intraneural microvascular flow. The "upper stretching limit" induced a stasis of arterioles and capillaries at 15% elongation (range 11-18%), but is less important because it lies beyond the critical limit as far as long-term viability of the nerve is concerned (Lundborg & Rydevik 1973). These findings support Zöch's hypothesis (1992) that maximally stretching a nerve 6-8% of its original length would induce changes in the microvascular flow.

Within this complex microvascular system the endoneurial milieu is protected by a blood-nerve barrier (Rempel *et al.* 1999). A breakdown in the blood-nerve barrier will occur with nerve injury including entrapment or compression, resulting in the accumulation of proteins, lymphocytes, fibroblasts and macrophages within the endoneurium as a reaction to antigens in the perineurial space (Mackinnon 2002). This will further initiate inflammation and eventually scar formation in the peripheral nerve, which in turn will lead to an uneven distribution of tension during mechanical load.

In contrast, there does not seem to be any blood-nerve barrier in the nerve roots and ganglia, and intravenously injected substances can leak into the intercellular layers between the nerve fibres and cell bodies (Rydevik *et al.* 1984). This means that nerve roots and ganglia are more susceptible to chemical irritation induced by inflammatory processes of near by structures, such as the discus intervertebralis or zygapophysial joints, rather than to mechanical deformation as is commonly assumed.

Since inflammation of the nerve root or nerve trunk in the absence of space-occupying pathologies are more difficult to diagnose with standard clinical examinations (i.e. radiography, computer tomography, magnetic resonance imaging, myelography, sonography) than a radiculopathy induced by disc herniation or osteophytes, many patients with CBP disorders have pain, of which the underlying mechanisms are not known. Besides, it has been shown that magnetic resonance imaging has a high percentage of clinically false-positive findings (Boden *et al.* 1990). In asymptomatic subjects the abnormal findings, such as herniated nucleus pulposus, narrowing of disc space or foraminal stenosis, had been shown to be age related. This means that in patients abnormal anatomical findings may not necessarily be related to their symptoms. Therefore, imaging diagnostics should not be utilised by

themselves to institute a therapy without matching the findings with clinical signs and symptoms. This is where the NTPT could be used as an additional diagnostic parameter in the evaluation of the clinical findings by assessing the reaction to mechanical load of putative inflamed tissues.

3.3. EFFECTS OF COMPRESSION ON NERVE FIBRES

The correlation between systemic blood pressure and the function of the nerve has supported the hypothesis that decreased intraneural microcirculation plays an important role in nerve compression disorders (Sunderland 1968, Rydevik 1992, Rempel *et al.* 1999, Mackinnon 2002). When neural tissues are subjected to load or pressure they deform, and pressure gradients are formed that lead to the redistribution of compressed tissue towards areas of lower pressure (Rempel *et al.* 1999). Nerve compression syndromes usually occur at sites where the nerves pass through a tight tunnel formed by stiff tissue boundaries like sulcuses, muscles or fascia (Table 2).

TABLE 2: Compression syndromes of the upper extremity		
N. radialis	**N. medianus**	**N. ulnaris**
Radial palsy (Saturday night palsy)	Thoracic outlet syndrome	Thoracic outlet syndrome
Supinator tunnel syndrome	Pronator teres syndrome	Cubital tunnel syndrome
	Anterior interosseous nerve syndrome	Guyon's canal compression
	Carpal tunnel syndrome	

Animal experiments, of low magnitude extraneural compression, induced by a miniature inflatable cuff, have demonstrated that the first sign of impairment was the stasis of

epineurial vessels appearing at a pressure of 20-30 mmHg (Rydevik *et al.* 1984), and that fast as well as slow antegrade and retrograde axonal transport was inhibited (Dahlin *et al.* 1981). By the time the cuff pressure reached 60-80 mmHg the compressed segment of the nerve was completely ischaemic. This means that cell nutrition and intraneuronal communication were compromised at elevated extraneural pressures. Dyck *et al.* (1990) reported that extraneural compression of 50 mmHg applied for two minutes can alter the structure of the myelin sheaths.

These disturbances were usually rapidly reversible after short periods of compression (1-2 hours), but repeated or prolonged compression at these pressure levels may show long lasting effects. These findings are interesting when related to pressure levels recorded in patients with CTS. It was found that patients with median nerve compression had an average of 32 mmHg pressure in their carpal tunnel, while the asymptomatic control group showed an average of 2.5 mmHg pressure (Gelberman *et al.* 1981).

Intraneural blood vessels have shown to increase their permeability as a response to nerve injury. Histological examinations demonstrated rapidly increasing endoneurial pressures in nerves that had been subjected to short-duration (2 hours) low magnitude extraneural pressure (30 mmHg), probably due to increased vascular permeability of the endoneurial and epineurial vessels (Rydevik *et al.* 1984, Rempel *et al.* 1999). Also long-term effects after two hours of low extraneural compression had been shown to result in long-lasting subperineurial oedema, that later lead to degeneration (demyelination) and regeneration of nerve fibres. These events seem to be associated with the degree of endoneurial oedema, hence a dose-response relationship between the duration of compression and the degree of injury can be observed.

Furthermore, inflammation and fibrin deposits have been found to occur within hours after compression, followed by proliferation of endoneurial fibroblasts and capillary endothelia cells (Rempel *et al.* 1999). Even though fibrosis is a normal response to inflammation, fibrotic adhesions may impair normal nerve gliding, and thus would have significant consequences on the movement dynamics of the neural tissue, which could appear as a clinical relevant sign during neural tissue provocation testing.

Morphological changes such as swelling proximal and distal of the ligature, stasis of venous return, and fibrosis surrounding the nerve have also been observed in animal models of chronic constrictive nerve injury (Greening & Lynn 1998). Histological evaluations of resected nerve segments from humans suffering from nerve compression equally showed vascular sclerosis, epineurial and perineurial oedema, thickening and fibrosis at the site of injury, and evidence of degeneration and generation of nerve fibres (Rempel *et al*. 1999), thereby confirming the findings of the previous animal experiments. This cascade of biological responses to compression mainly affected large diameter myelinated fibres as unmyelinated fibres had been shown to be spared (Rydevik *et al.* 1984). A highly significant loss of Aβ fibres was observed peaking at 2 weeks post constrictive injury, however 8-10 weeks post injury Aβ fibres still appeared abnormal in respect to their diameter and overall number (Greening & Lynn 1998).

Due to ethical difficulties in carrying out experiments in humans there is obviously much less data available on neuropathological changes following nerve injury than from animal experiments. However, these observed events taking place during peripheral nerve compression in animal experiments could now be explained and related to clinical symptoms in patients. Knowledge gained from animal experiments (Lundborg & Rydevik 1973, Dahlin *et*

al. 1981, Rydevik *et al.* 1984, Dyck *et al.* 1990, Rydevik 1992, Greening & Lynn 1998, Michaelis & Jänig 1998, Rempel *et al.* 1999) has also lead to a better understanding of the pathomechanisms of entrapment neuropathies such as the CTS.

The initial symptoms in CTS are usually intermittent paraesthesia and deficits of sensation that occur primarily at night. These symptoms are probably due to impairment of the intra-neural microcirculation associated with endoneurial oedema, which disappear during the day by movement of the arm (Rempel *et al*. 1999). With increased compression more severe and constant symptoms that do not disappear during the day arise. In this stage microcirculation may be altered during the day, leading to morphological changes such as segmental demye-lination. Besides entrapment neuropathies metabolic changes as in diabetes mellitus or during pregnancy may equally cause endoneurial oedema and need to be taken into consideration during patient assessment.

Alternatively, short-term compression of intact peripheral nerves or nerve roots has been stated to cause no pain but rather paraesthesia as a result of ischaemia and not through mechanical nerve fibre deformation (Rydevik *et al.* 1992, Hasue 1993, Bogduk 1994). Whereas in the case of an inflamed or irritated nerve or nerve root, compression or minor mechanical deformation can be the cause of radiating pain (Rydevik *et al.* 1984, Hasue 1993). Inflammation can for example be caused by the breakdown products of degenerating nucleus pulposus (Rydevik *et al.* 1984, Hasue 1993, Zusman 1998), or by long-term or high magnitude compression (Dyck *et al.* 1990, Rempel *et al*. 1999). In so far irritation of the neural tissue has to be present before mechanical provocation can give rise to pain.

3.4. PAIN MECHANISMS AS IN MINOR NERVE INJURIES

In clinical practice patients frequently present with diffuse symptoms in their neck and upper extremity of unknown aetiology. Positive symptoms usually associated with peripheral nerve injury, such as paraesthesia, hyperalgesia, allodynia, spontaneous pain and impaired function may be reported. Typical symptoms reported during patient assessment that have been associated with peripherally evoked pain are listed in Table 3. It has been hypothesised that the mechanosensitivity of the neural tissue, which is heightened in the case of minor peripheral nerve damage, can be detected by the NTPT (Hall & Quintner 1996, v. der Heide *et al.* 2002), which has meanwhile been integrated into the physiotherapeutic assessment of patients with CBP disorders.

TABLE 3:	Features of peripherally evoked pain patterns (Butler 2000)
- Symptoms appear in a clear anatomic and dermatomal pattern (along the peripheral nerve) - Pain sensation described as 'burning', 'strings pulling', 'electrical feeling', 'ants on me', or 'deep and aching' - Normal axonal conduction - Widespread tenderness of nerve trunk on palpation - Paraesthesia in the peripheral neural area - Nocturnal pain - Symptoms related to vulnerable neuroanatomic sites (carpal tunnel, IVF, scalenes, first rib, proximal fibula) - Symptoms associated with tensile stress of neural tissue (i.e. antalgic postures holding arm over head to alleviate symptoms) - Pain is usually provocated mechanically, easily reproducible and similar on retesting	

It has been argued that pain and changed somatosensory thresholds may occur in the peripheral nerve following relatively minor damage of nerve fibres (Greening & Lynn 1998),

that are not necessarily associated with the loss of axonal conduction and therefore seldom show neurological deficits such as muscle weakness, depressed reflexes, decreased conduction velocity, or impaired sensation. An explanations for this may be found in minor nerve injuries of the chronic neuritis model where axons were found to be undamaged or only partially damaged and so could account for the normal nerve conduction (Greening & Lynn 1998).

Even in the case of morphologic axonal changes, the clinical limitation of nerve conduction and electromyography (EMG) in predicting neuropathological abnormalities of single nerve fibres has been demonstrated (Dyck 1990). Interestingly the pathological inferences that can be made from EMG studies of large sensory fibres were more limited than those from large motor fibres. However, patients with CBP disorders usually complain of sensory deficits that are likely not to show in EMG studies.

Peripheral neurogenic pain as in minor nerve injuries has been attributed to an increased activity in mechanically or chemically sensitised nociceptors within the nerve sheaths, and is said to be felt in the course of the peripheral nerve trunk (Hall & Elvey 1999). These mostly unmyelinated afferents that constitute the intrinsic innervation of the connective tissue sheaths are known as 'nervi nervorum' (Hromada 1963, Bove & Light 1997). These sensory C fibres of the nerve sheath have been shown to contain neuropeptides such as substance P and calcitonin gene related peptide suggesting a role in vasodilatation. It is assumed that local nerve inflammation is mediated by the nervi nervorum, especially in cases with no intrafascicular axonal damage (Bove & Light 1997).

The nervi nervorum has also been found to be sensitive to excess longitudinal stretch, but not to stretch within the normal range of motion (Bove & Light 1997), and is assumed to be

particularly vulnerable to chronic compression and friction syndromes (Greening & Lynn 1998). However, electrophysiological recordings of the nervi nervorum present considerable technical difficulties so that their contribution to sensory input remains to be evaluated.

The painful response to nerve trunk palpation has been attributed to the spread of mechano-sensitivity along the length of the nerve trunk mediated through a neurogenic inflammation via the nervi nervorum (Hall & Quintner 1995, Bove & Light 1997, Hall & Elvey 1999) and can be related to secondary hyperalgesia. Experimental studies of primary and secondary hyperalgesia suggest that there are several different mechanical hyperalgesias characterised by their location and the primary afferent fibres involved (Treede *et al.* 1992).

Mechanical hyperalgesia, which is confined to the site of injury (primary hyperalgesia), is based on the peripheral sensitisation of C fibre nociceptors, i.e. through blunt stimuli. Whereas secondary hyperalgesia, which reacts to punctuate mechanical stimuli, appears to be based on central sensitisation of A fibre nociceptor input, and occurs not only in the injured tissue but spreads to adjacent uninjured tissue (Woolf 1991, Treede *et al.* 1992). It is furthermore well known that damaged sensory nerve fibres frequently develop abnormal impulse generation, which is correlated to the presence of mechanical hyperalgesia. Paraesthesia has been shown to be induced by spontaneous ectopic sensory discharge generated from large myelinated afferent fibres that may occur at the damaged site (Nordin *et al.* 1984, Zhang *et al.* 1997, Devor & Selzer 1999).

To sum up, this section has presented the biomechanical and neurophysiological features pertaining to CBP disorders in respect to impaired movement dynamics of the neural tissue. It is known that the neural tissue is vulnerable to deformation, compression and entrapment injuries in its natural surroundings (Table 2), which may lead to restrictions of neural tissue

mobility. The neural tissue has the ability to adapt to normal joint motion through a gliding mechanism that permits movement without putting any strain on the individual nerve fibres within the fasciculi. Additionally the nerve trunk has the capability to stretch to about 6-8% of its original length, and is relatively more resistant to mechanical load than the nerve root. However, increased elongation and compression have shown to impede with neural micro-vascular circulation, which may lead to perineurial oedema, fibrosis, and degeneration of nerve fibres.

Peripheral changes caused by partial nerve injury may lead to a lowered threshold of afferent fibres for mechanical stimulation, as well as to increased firing and spontaneous activity. Abnormal input from damaged or ischaemic nerve fibres may in turn cause pain and trigger central sensitisation. Increased mechanosensitivity of neural tissue after minor nerve damage is said to play an important role in diffuse upper extremity pain that is often seen in clinical practice. In determining whether the pathology of the neural tissue is associated with clinical symptoms the NTPT utilises the sensitivity of the neural tissue to mechanical load in the assessment of CBP disorders.

However, pain should never be understood as an isolated entity, but rather as a summation of all available information from the outside world and from within our bodies, analysed by the brain in terms of what action would be appropriate (Wall 1999). Furthermore, alterations of neural, behavioural and subjective pain responses by arousal, attention and expectation, that result from the action of the central nervous system networks in modulating the transmission of nociceptive messages, should equally be taken into account (Fields & Basbaum 1999).

4. THE HISTORY OF NEURAL TISSUE PROVOCATION TESTING

The previous chapter has laid out the basic anatomical features of the neural tissue and the probable causes for the impediment of its mobility. The rational behind this section is to show how the initial medical interest on the behaviour of nerves to stretching developed into a physiotherapeutic approach to assess the neural tissue as a potential source of pain.

Observations on the behaviour of peripheral nerves subjected to stretching date from the second half of the 19th century and pertained to the therapeutic procedure for the relief of painful neuralgias of different genesis (Cavafy 1881, Symington 1882, Marshall 1883). Marshall discussed the mechanisms of pain relief attributing both symptoms and benefits to the nervi nervorum, a then hypothesised but undescribed structure (Bove & Light 1997). Apart from the therapeutic approach, investigations on the breaking points and on morphological changes of stretched peripheral nerves were conducted (i.e. Tillaux 1866, Symington 1882, Vogt 1877, Takimoto 1917, cited in Sunderland & Bradley 1961a p.102).

In the First World War studies on the elasticity of peripheral nerves came into focus due to the intensified need for nerve reconstruction. Baron and Schreiber (1918) described the importance of the surrounding tissue, in which the nerve is able to glide. Babcock (1927) studied the elasticity of peripheral nerves and found them to have less lengthening abilities in comparison to vessels. These findings were the first to illustrate the importance of an intact nerve bed for normal nerve mobility, and have set the basis for the concept of neural tissue dynamics.

The earliest references to upper limb tension testing stem from 1929, in which the most extended positions of the N. medianus, N. ulnaris and N. radialis were clearly described

(Bragard 1929). Bragard also reported, that he could establish what he called the "medianus phenomena" (tension position for the N. medianus) in patients with plexus brachialis disorders, and that the affected nerves were sensitive to pressure. Later, tests were described by Chavaney and Frykholm, who applied traction to the extended, abducted, and supinated arm, as well as simultaneously tilting the head to the contralateral side (Butler 2000). Since these early investigations the upper limb neural test has been described with emphasise on different movement sequences and under changing terminologies (Table 4).

TABLE 4: Chronology of related studies on the ULNT	
Bragard (1929)	described a series of upper limb tension tests
Chavaney (1934)	a version of the ULNT with abducted, elevated and extended arm
Frykholm (1951)	a version of the ULNT with abducted, supinated and extended arm
Cyriax (1978)	examined wrist dysfunction with extended elbow
Breig (1978)	introduced the term adverse mechanical tension
Elvey (1979)	first described the brachial plexus tension test
Kenneally *et al.* (1988)	introduced the name upper limb tension test
Butler (1991)	introduced the term mobilisation of the nervous system
Selvaratnam *et al.* (1994)	validated Elvey's brachial plexus tension test
Shacklock (1995)	introduced the term neurodynamics
Hall *et al.* (1998) v. der Heide *et al.* (2001)	used the term neural tissue provocation test

The seminal work of Elvey (1979b), who initially formulated and described the 'brachial plexus tension test' (BPTT), led to an increased physiotherapeutic interest in the neural tissue as a potential source of pathology and pain in CBP disorders. Assessing the neural tissue as part of the physiotherapeutic examination has never been presented to stand alone, but was always seen as an integral part of existing neurological and orthopaedic assessments.

In the early stages of the BPTT the idea of testing for increased neural mechanosensitivity, by increasing tension within the neural tissue through an ordered set up of joint movements, was based on the notion that 'neural tension testing' accurately reflected the mechanical function of neural structures (Elvey 1979b, Kenneally *et al.* 1988, Butler 1991). This implied that a 'normal' neural tension test meant the neural tissue moved correctly, and an 'abnormal' test meant it did not. This idea was clearly dominant when Butler published his work "Mobilisation of the nervous system" in 1991. At that time it was important to clarify which test components would have an effect on the movement of the nerve in relation to its surrounding interfacing structures (pathomechanical effect), and which components would cause 'tension' in the nerve itself and might influence its physiological function (patho-physiological effect).

Deductions like these involved only mechanical factors and were at times inaccurate because the contribution of neurophysiological factors in the production of symptoms had been over-looked (Shacklock 1995, Rempel *et al.* 1999). Therefore, Butler embraced Shacklock's concept (1995) of neurodynamics (the dynamic properties of the neural tissue), because it allowed a shift away from the pure mechanical thought to include neurophysiological issues and the inclusion of plasticity changes of the central nervous system. Having an under-standing of the basic anatomical and physiological science underlying neurodynamics is a critical part of the clinical reasoning process when treating patients with CBP disorders. Clinical reasoning is the thinking underlying clinical practice, where an initial working hypothesis is tested until sufficient information is obtained to make a diagnostic decision that leads to making a management decision (Jones 1995). Physiotherapists are now increasingly recognising the importance of integrating basic neuroanatomy and neurophysiology research

findings into their clinical practice (Zusman 1992/1994/2002, Wright 1998/1999, Matheson 2000).

So far painful conditions of the upper arm were routinely examined for possible referral of pain from the cervical spine with muscle power testing, sensation, reflex activity, and nerve conduction. However, if the standard clinical examination failed to reveal any positive signs, such as neurological deficits, or reproducibility of pain by cervical spine tests or shoulder joint tests, confusion arose as to the medical explanation for the pain (Elvey 1979b). Elvey was a pioneer in conceptualising the physical examination of the neural tissue of the upper quarter in order to investigate arm pain and regional pain syndromes in the area of the fifth and sixth cervical dermatome. Pain and movement restrictions felt in the shoulder region or lateral upper arm can be a result of glenohumeral joint or soft tissue pathology, or can be of cervical or thoracic origin (Maitland 1991). The shoulder and arm region is innervated by the C5-6 sclerotome, thus pain in this region could be regarded as referred pain from any structures supplied by the fifth and sixth nerve roots, provided that other underlying diseases (i.e. biliary colic, angina pectoris, Pancoast tumour) that may also refer pain into this region have been ruled out.

The BPTT was then propagated as a diagnostic tool, which may help "differentiate between intrinsic shoulder symptoms and shoulder symptoms which are referred from the cervical spine" (Kenneally *et al.* 1988, p.174), by placing tension on the nerve roots of the brachial plexus, and thereby testing if there was any neural tissue involvement. It was always clear that other anatomical structures would come under traction during this test, which could equally reproduce shoulder pain, therefore it was important to develop 'sensitising manoeuvres' that were able to distinguish between neural and non-neural structures. Aim

was to alter the patient's symptoms by adding sensitising manoeuvres remote from the site of pain on the condition that the impact on the nervous system passes beyond the point to which musculoskeletal structures were loaded.

The early BPTT according to Elvey (1979ab) was composed of three basic movements. The technique was performed with the patient lying supine on an examination table, head and neck supported in a resting position.

1. glenohumeral abduction behind the coronal plane, with the elbow extended
2. glenohumeral lateral rotation, and forearm supination
3. while maintaining full forearm supination the elbow was gently flexed

All manoeuvres were performed to the point of pain onset. As each of the movement components were added it was believed that a progressively greater stretch was transmitted onto the nerve trunks. The test was thought to be positive if the final movement was able to reproduce the patient's pain (Elvey 1979ab). To verify whether the pain was a reaction to loading the neural tissue or of non-neural structures to stretch, wrist extension was added to the final position to further provoke the symptoms, claiming to increase cervical root traction without implicating other structures. By only moving the wrist, muscles of the forearm or the glenohumeral joint would not be affected and so could not be held to account for the increase in symptoms.

Other sensitising manoeuvres included cervical spine movements in flexion as well as in contralateral flexion, lateral rotation and abduction of the contralateral arm, and bilateral SLR (Elvey 1979ab, Bell 1987). These manoeuvres were believed to increase symptoms on the affected side through further stressing the nerve trunks through a traction force in the

opposite direction, on the grounds that the nervous system is a continuous structure. The final reassurance of neural tissue involvement was won by taking up a BPTT position just short of the patient's pain and then adding contralateral cervical lateral flexion (CCLF), which would load the brachial plexus bringing back the patient's pain or symptoms.

All these testing components were developed by Elvey empirically in the course of his clinical experience with patients who suffered from upper quarter disorders. Elvey's postulated theory soon became popular and accepted but had yet to be validated scientifically. The development of therapeutic techniques in movement-based professions has traditionally been based on the thoughts and clinical experiences of pioneering clinicians. However, the increased demand for professional accountability and the lack of subjecting existing treatments to validation has been a well-recognised deficit. These demands are now being met by an increasing amount of physiotherapy research, which also reflect the upgrading of this profession to an academic level.

In 1986 Elvey adapted the BPTT by adding shoulder girdle depression to the starting position and by changing the final movement component from elbow flexion to extension, thus transmitting more load to the neural tissues. Keneally *et al.* (1988), who later renamed the test 'upper limb tension test', argued that the course of the major nerves in the upper limb would determine which nerves were most likely to be influenced by the movement components of the upper limb tension test, and believed the median nerve to be most affected by this test. Butler (1991) broadened the upper limb tension test into four testing procedures. The initial idea of testing the tension of a nerve was also revised, so that the basic test was now called 'upper limb neural test' (Butler 1994) namely ULNT1, ULNT2a, ULNT2b, and ULNT3 (Table 5). In Butler's ULNT base test elbow extension was employed

as the final component, this way the degree of elbow extension at the point of pain onset or maximal tolerable pain could be used as the main outcome measure (dependent variable).

TABLE 5:	The ULNT's from Butler (1994, Fig. 2-4 Appendix C)	
Name	**Main sensitising component in bold**	**Nerve bias**
ULNT1	- shoulder girdle depression (maintained) **110° G/H abduction** in the coronal plane - forearm supination with wrist/ finger extension - G/H lateral rotation - elbow extension	N. medianus (base test)
ULNT2a	- shoulder girdle depression (maintained) 10° G/H abduction in the coronal plane - elbow extension **G/H lateral rotation** - wrist/ finger extension - G/H abduction	N. medianus
ULNT2b	- shoulder girdle depression (maintained) 10° G/H abduction in the coronal plane - elbow extension **G/H medial rotation** - wrist/ finger flexion plus ulnar deviation	N. radialis
ULNT3	- wrist/ finger extension - forearm supination - full elbow flexion - shoulder girdle depression **G/H abduction** - head in lateral flexion	N. ulnaris

For a clear understanding of what a normal response is Kenneally *et al.* (1988) listed the responses to the ULNT1 as seen in 100 normal asymptomatic subjects. (1) A deep stretch or ache in the cubital fossa (99% of subjects) extending down the anterior and radial aspect of the forearm and into the radial side of the hand, with a definite tingling sensation in the thumb and first three fingers in 80% of all subjects. This response was found to be independent of age and gender of the subjects. (2) A small percentage of subjects may feel

stretch in the anterior shoulder area during elbow extension as the long head of the biceps is stretched. (3) CCLF increases the response in approximately 90% of the subjects (Figure 1).
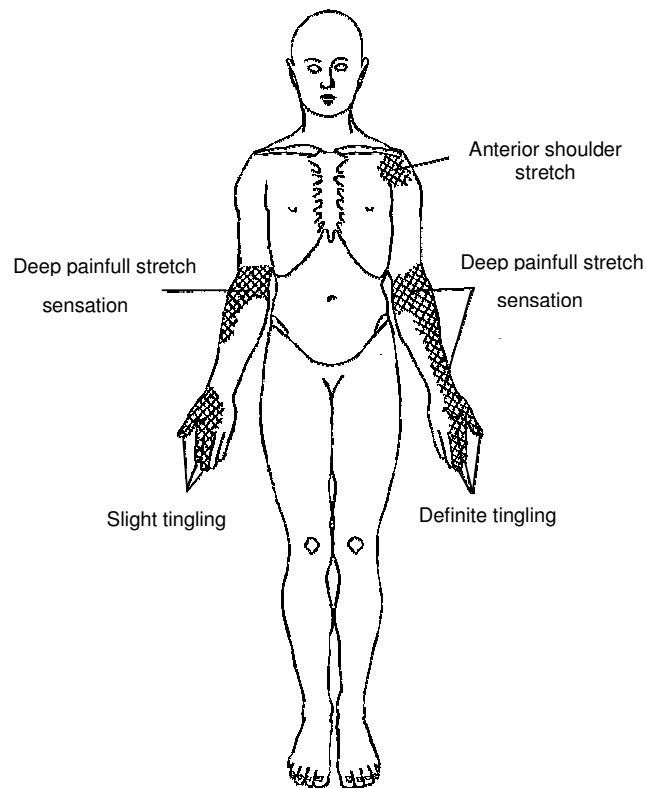
Figure 1. The normal sensory responses to the ULNT1. From Keneally *et al.* (1988) The upper limb tension test: the SLR of the arm

The close anatomical and physiological relationship between muscles, joints, neural and vascular systems sets a high demand on the specificity of these tests (Figure 2). The ULNT that may have been designed to focus particularly on neural structures, will by its nature stress tissues and elicit responses in any of these systems. Selvaratnam *et al.* (1994, Table III-Appendix B) was the first to test the ability of Elvey's BPTT (1979b) to discriminate between local pain and referred sources of pain from the cervical region. Post-surgery cardiac patients with a high probability of brachial plexus involvement (n=25) and sports-

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injury patients (n=25) were compared with an asymptomatic control group (n=25). The main finding was that cardiac patients had significantly greater loss of elbow ROM with CCLF than the two other groups. Reliability was calculated to be 82,5%, but a high percentage agreement can be attributed to chance alone and cannot in itself support a claim of high reliability (Haas 1991b). The large standard error of measurement (SEM) with 16.8° further questions whether appropriate standardisations were carried out.

Given that the cardiac group were pre-selected to have a higher probability of referred pain from the cervicothoracic region, these findings support the discriminative validity of the BPTT. The only difference between the groups was age: the cardiac group being older (mean age 55.3) than the sports injury group (mean age 26.2); thus, the results may have stemmed from age-related changes such as osteoarthrosis or possible bony proliferative changes in the lower cervical spine. In agreement with Elvey (1979b/1986) Selvaratnam *et al.* (1994) stated that the brachial plexus was stimulated by the BPTT, but object that the sole involvement of the brachial plexus cannot be fully determined. It is possible that other cervical and thoracic structures, particularly the fascia, the subclavian artery or vein (Wilson *et al.* 1994) that are affected by the surgical procedure, could be capable of referring pain to the shoulder region.

For an accurate interpretation of the test response it is therefore necessary to examine the reliability of these testing procedures. The influence of the different test components on the nervous system and its surrounding has been analysed in several human cadaver studies (Ginn 1988, Selvaratnam 1989, Wilson *et al.* 1994, Kleinrensink *et al.* 1995b/2000, Lewis *et al.* 1998), and in clinical studies that describe physical reactions and sensory responses (Bell 1987, Yaxely & Jull 1991, Edgar *et al.* 1994, Zorn *et al.* 1995, Balster & Jull 1997, Coppieters *et al.* 2001ab/2003ab, v. d. Heide *et al.* 2001/2002). Obviously clinical studies

are not able to deduce the underlying mechanisms to these responses, therefore combining the information gained from neurophysiological and biomedical research with the findings acquired from clinical studies is pertinent.
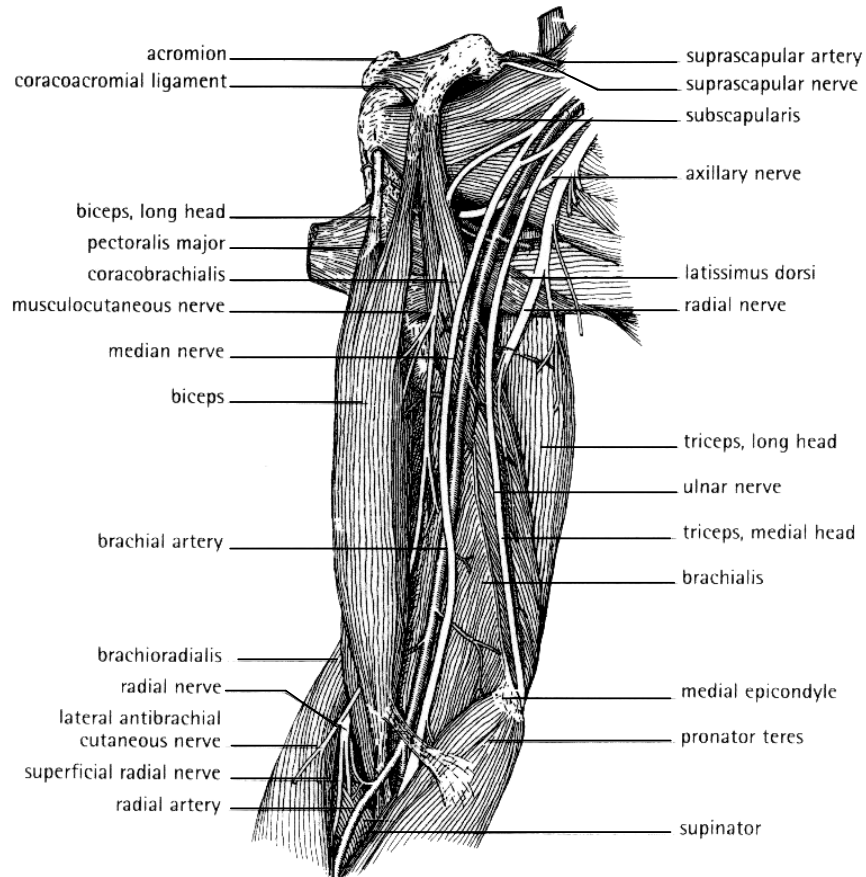
Figure 2.   The nerves, muscles and blood vessels of the forearm. From Butler DS (2000) The Sensitive Nervous System

Since the original description of Elvey's BPTT the entire concept of physically examining the neural tissue has expanded. Against what had been initially believed to be an 'adverse' mechanical response of the neural tissue to load has recently been hypothesised to be a protective muscle reaction to the provocation imparted on possibly sensitised neural tissue

(Balster & Jull 1997). However, no correlation between muscle activity and pain perception was evident in this study, therefore the hypothesised mechanisms have yet to be confirmed.

The initial concept of examining the extensibility of neural tissue was further challenged by an investigating on the normal compliance of neural tissue to the straight leg raise (SLR) test in L5/S1 radiculopathy patients. Hall *et al.* (1998) proposed that the onset of muscle activity rather than the onset of resistance seems to represent more accurately an increased neural sensitivity to mechanical load (see section 5.2.2). V. der Heide (2002) confirmed this pattern of reactive muscle activity in a case study of three subjects with cervical radiculopathy, where trapezius muscle activity was found to be in close association to the onset of pain, however, the small sample size precludes any generalising of the findings so that future investigations with larger patient populations need to be conducted that are statistically more powerful (Hicks 1999).

The original name brachial plexus tension test tends to "convey an incorrect impression of the whole thought process behind the assessment of conditions when using physical exami-nation tests of neural tissues" (Elvey 1995, p.115). The need for a correct terminology has lead to the description of the whole concept of testing manoeuvres as responses to 'neural tissue provocation testing' (Hall *et al.* 1998, Coppieters *et al.* 2001a, v. der Heide *et al.* 2001) or to neurodynamic testing (Shacklock 1995). In this sense physiotherapy has taken a step back from giving preliminary explanations of the mechanisms behind the reactions to neural tissue testing, and instead has engaged in the process of evidence-based reasoning by trying to establish a knowledge base through normative studies. This way the normal reactions of asymptomatic subjects to this test can be used as a standard against which findings in a clinical setting can be compared.

5. CRITICAL ANALYSIS OF ULNT RESEARCH

In this main section of the thesis observations and assumptions underling the NTPT will be critically evaluated. For this purpose it is necessary to first discuss some methodological criteria pertaining to the ULNT research. In the following sections, the ULNT's construct validity will be investigated by looking at biomechanical findings, and by assessing the reliability of outcome variables measured during neural tissue provocation testing in normal subjects. The last sections of this chapter will investigate scientific and clinical support for the use of the ULNT in a clinical setting. As the ULNT has been developed as a diagnostic test all research presented will be concerned with issues of its validation. The evaluation of neural mobilisation techniques will not be included as it goes beyond the scope of this study.

5.1. METHODOLOGICAL CONSIDERATIONS ON ULNT RESEARCH

In physiotherapy there is an increasing attention to the evidence-based medicine approach, partly stimulated by the demands of society to show efficacy and cost-effective physio-therapy (Koes & Hoving 1998). Many physiotherapist however, work in environments where research facilities and the support for research are lacking (Matheson 2000), impeding the desire to be part of the scientific community. Nevertheless, physiotherapy is realising this deficit and research designs are being developed that acknowledge scientific demands as well as the potentials that lie in physiotherapy. Traditionally the randomised clinical trial (RCT) is considered to be the most valid design because of its potential to control various forms of bias (Colditz *et al.* 1989, Schulz 1996, Koes & Hoving 1998). However, one limitation is that RCTs give only little insight into the proposed working mechanisms and generally only evaluate the efficacy of existing interventions compared to new ones, but play no role in the development of new treatment strategies. With regards to neural tissue testing

the ULNT is a diagnostic test and cannot be subject of an RCT. To test the validity of a test it has to be compared with a criterion standard or golden standard. The problem with the ULNT is that there is no gold standard against which it can be compared. Electrodiagnostic tests for example evaluate nerve conduction and neuromuscular diseases, but are unable to assess increased neuromechanosensitivity, a cardinal sign in minor peripheral nerve injuries.

Despite these obstacles physiotherapist have recognised the importance of investigating the diagnostic validity of the ULNT. Research from the past ten years not only reflects this increasing urge, but also reflects the evolving quality in terms of scientific merit. There has been a change in physiotherapy from an empirically based approach towards a much greater emphasis on scientifically based practice, and the neural tension tests are probably the "most thoroughly evaluated group of assessment procedures" among the physiotherapists' arma-mentarium (Wright 1998, p.1). To evaluate the subsequent research a number of methodo-logical criteria have been adopted pertaining to the ULNT as a diagnostic tool (Table 6).

TABLE 6:	Methodological criteria for the neural provocation test as a diagnostic tool

1. How reliable is the test?
   - intra-examiner, inter-examiner reliability and repeatability

2. How clear are the operational definitions in the studies?
   - starting position of the subjects (fixation of the shoulder girdle)
   - sequence of test movements
   - what end position was used and under what criteria (pain, resistance)
   - what are the quantifiable measurements (instrumentation)

3. How are the independent variables controlled for?
   - sensitising manoeuvres (wrist extension, CCLF)

4. How is the normal response in asymptomatic subjects defined?
   - sensory area of response
   - normal ROM
   - reactive muscle activity

5. How valid is the test?
   - sensitivity and specificity of the test

5.1.1. Reliability of examination procedures

Research into the use of manual examination techniques as the ULNT is essential particularly in determining if physiotherapists are successful in achieving the objectives of validity, reliability, and repeatability for these techniques. Such research requires that an accurate diagnostic test is available as a standard against which findings of the test response can be compared (Philips & Twomey 1996). This also means a diagnostic test has to show consistency in order to be reliable. Reliability has been defined as "the degree to which measurements are error-free and the degree to which repeated measurements will agree" (Rothstein *et al.* 1991, p.52). In the past there have been investigations on the reliability of manual examination techniques of the vertebral joints (Maitland 1986) that are commonly used by manual therapist in the assessment of spinal disorders.

A single-blinded crossover study by Jull *et al*. (1988) demonstrated that one experienced manipulative therapist was able to locate the symptomatic cervical zygapophysial joints, prior and post to diagnostic blocks that established the diagnosis, with a 100% sensitivity and specificity. Sensitivity meant that from the 15 patients that had zygapophysial pain syndromes all 15 were detected correctly, and reciprocally specificity related to the 5 patients without zygapophysial pain syndromes, that were equally identified correctly. The diagnostic test in this case involved manual testing of the mechanical properties of all cervical joints in search for "perceived stiffness properties". Since movement abnormalities are palpable even in asymptomatic joints, the criteria for identifying a symptomatic joint were: abnormal 'end feel', abnormal quality of resistance to motion, and reproduction of pain.

The findings of this study suggest that this set of criteria is useful in identifying symptomatic joints, but before such conclusions can be made with assurance, similar research considering other regions of the spine is necessary, and the inter-examiner reliability of these manual techniques would have to be established. Furthermore, it should be noted that since the examiner was highly skilled, results from this study are not typical for the majority of physiotherapists and cannot be generalised, but emphasise the importance of high quality manual training for orthopaedic physical therapists.

In the quest for standardised manual examination techniques, a more recent randomised crossover study by Philips and Twomey (1996) raised the question whether manual examination alone is sufficient for accurate segmental diagnosis in low back pain patients, or whether it must be accompanied by a verbal pain response. In the patient group that was examined prior to a spinal diagnostic block the manual therapist's diagnosis was correct in 16 out of 17 subjects (94.12% sensitivity) for verbal responses, and in 9 out of 17 (52.9% sensitivity) for non-verbal responses. The identification of subjects with no history of low back pain was 5 out of 5 for verbal responses (100% specificity), and 4 out of 5 for non-verbal responses (80% specificity). These results demonstrate that the patient's response was important in identifying the symptomatic segment or in identifying true-positives. These findings are concordant with the earlier propagated set of criteria (Jull *et al.* 1988) for identifying symptomatic joints, and indicate that manual examination techniques alone are not a reliable measurement, but that the reproduction of pain or/and the verbal response of the patient is necessary for an accurate diagnosis.

Percentage agreement rates were calculated to assess inter-examiner reliability for passive intervertebral movements and tissue responses, and showed a range between as low as 43%

to a 100% agreement. However, as a measure for reliability, percentage agreement does not take into account the agreement that is expected to occur due to chance alone, and since a high agreement can be attributed to chance cannot in itself support a claim of good reliability (Haas 1991b). Hence, the weak inter-examiner reliability on the passive manual techniques remains to pose a considerable problem in the attempt to standardise manual diagnostic tests.

Implicating these results to the evaluation of the ULNT's reliability obviously leads to the following considerations. In the first place, the testing procedure of the ULNT is much more complex than the spinal joint tests involving multiple joint movements and handling techniques. This results in a large number of independent variables that are difficult to control. Secondly, as with the spinal joints, a normal range of movement respectively a normal response to the ULNT has to be established against which abnormal test responses can be compared. Thirdly, the consistency of the measurement tool, in this case corresponding to the inter-examiner reliability, needs to be evaluated. Finally, as has been shown to be important for spinal manual diagnostic tests, verbal responses and reproduction of pain are equally a necessary part of neural tissue testing procedures.

It has been proposed that one of the most important steps in establishing the efficacy of any diagnostic procedure is the investigation of its reliability (Haas 1991b). In Table 7 several ULNT studies are shown, that have accounted for their test-retest reliability. Yaxely and Jull (1991), for example calculated a high inter-examiner reliability and repeatability of 94.9% and 99.9% respectively between two raters for the range of glenohumeral abduction at R2 (maximal resistance). In contrast, Hines *et al.* (1993) showed that assessing elbow extension at R1 (onset of resistance) among four raters was unreliable, which is not surprising as it is almost impossible to manually pick up on the first point of increase in tension. On the other

hand excellent intra- and inter-examiner reliability for elbow ROM at submaximal pain tolerance (Coppieters *et al*. 2001b) were shown, as well as high reliability coefficients for the intra-examiner reliability for pain onset (P1) and maximal pain tolerance (P2), and for muscle activity during elbow extension (v. der Heide *et al.* 2001). These measurements were clearly described and calculated with the one-way ANOVA intraclass correlation coefficient (ICC), which is the statistic of choice for the reliability of examiners for continuous data (Haas 1991a).

Selvaratnam *et al.* (1994) demonstrated a moderate to high intra-examiner reliability of 82.5% measuring elbow-wrist extension at P1 by correlating data acquired from two patient groups. No significant differences were found in intra-examiner repeatability for trapezius length and the ULNT, and in inter-examiner reliability for shoulder girdle depression (Edgar *et al*. 1994). Grant *et al.* (1995) computed only a fair inter-examiner reliability (0.505) between two raters for glenohumeral abduction at R2. The intra-examiner repeatability however, was calculated with a percent agreement of 2.57% and not the usual indices of –1 to +1 (Hicks 1999), which made interpretations inconclusive.

No statistical descriptions were given for the high percent agreement in the area of sensory response (Zorn *et al.* 1995), and no reports on examiner reliability were given by Quintner (1990), and Balster and Jull (1997). Obviously only weak implications can be made for the use of these parameters in clinical practice, as long as it is not ensured that only relevant outcome measures are analysed and that the appropriate statistics are being used. The significance of this will be discussed in more detail for individual studies in sections 5.2.2. and 5.2.3.

In summary, reliability measurements were made for range of motion at R1 or R2, for pain intensity and sensory area of response, and for P1 and P2. Apart from the fact that these studies differed widely in their quality for demonstrating their statistical analysis most of these studies endeavoured to establish the reliability of diagnostic procedures. However, the appropriateness of statistical methods and their interpretations of results were not always clear. Hass (1991a) stated that the ANOVA and the *t*-test are least appropriate for assessing reliability, because they test rater performance for significant differences from chance alone, which could lead to concealing large inter-examiner disagreements. In addition most studies report on both intra- and inter-examiner reliability and usually find greater concordance within raters than between them.

One reason for this is the difficulty to ensure sufficient blinding of the rater to accurately assess intra-examiner reliability. Secondly, even though there might be a strong self-consistency of each examiner, there could be disagreement between raters, which results in a lower inter-examiner reliability. However, high intra-examiner reliability does not rule out that the measurements stem from a consistency of error (Haas 1991a). Therefore inter-examiner concordance weighs more heavily in the evaluation of reliability measures, and should be included in every research evaluating diagnostic procedures or examination techniques especially if the testing procedures are not standardised.

Study	Neural test	N	Age	What was measured?	Sensitising manoeuvres	Results	Intra-, inter-examiner reliability
				TABLE 7: Test-retest accountability in ULNT research			
Yaxely & Jull 1991	ULNT2b (radial bias)	50 normal	18-30	G/H abduction and sensory response at R2	__	Normal response = 40º of G/H abduction, and stretch felt over radial aspect of forearm	High inter-examiner reliability >95% for G/H abduction at R2 (ANOVA)
Hines *et al.* 1993	ULNT1 (base test)	25 normal	19-50	onset of R1 in elbow extension	__	The range of elbow extension at R1 differed significantly between 4 raters	Low inter-examiner reliability for elbow ROM at R1 (ANOVA)
Edgar *et al.* 1994	ULNT1	60 normal	17-25	shoulder depression during ULNT, trapezius length	contralateral CLF	Lesser extensibility of neural tissue and trapezius length are related	Good inter-examiner reliability for shoulder depression and intra-examiner repeatability for trapezius length and ULNT (ANOVA)
Selvaratnam *et al.* 1994	ULNT1	25 symptomatic 25 symptomatic 25 asymptomatic	22-70 16-51 19-73	elbow-wrist extension at P1	contralateral CLF and ipsilateral CLF	BPTT is able to identify referred pain from brachial plexus in symptomatic subjects	Intra-examiner reliability 82,5% for performing the ULNT1 (ANOVA)
Grant *et al.* 1995	ULNT2b	15 symptomatic 10 asymptomatic	17-55 mean 28	G/H abduction, sensory response at P?	contralateral CLF	Patient group exhibited decreased range of G/H abduction in ULNT in comparison to normal subjects	Report of high intra- and inter-examiner reliability (p=0.004) for G/H abduction (ANOVA)

TABLE 7: Test-retest accountability in ULNT research

Study	Neural test	N	Age	What was measured?	Sensitising manoeuvres	Results	Intra-, inter-examiner reliability
Zorn *et al.* 1995	ULNT1	90 normal	18-60	Location of sensory response at P?	—	Sequencing from distal to proximal produces fewer proximal responses	93% of agreement for area of sensory response (no statistics)
Balster & Jull 1997	ULNT2a (median bias)	20 normal	18-30	Trapezius activity, elbow extension at P1	contralateral CLF	Greater magnitude of trapezius activity found in lesser extensible neural tissue group	Not stated
Coppieters *et al.* 2001a	ULNT1	35 normal	20-28	Shoulder girdle elevation force	wrist extension, contralateral CLF	Gradual increase in shoulder girdle elevation force is a normal sign during neurodynamic testing	Excellent reliability coefficient for intra-examiner (0.93-0.97) and for inter-examiner (0.93-0.96) reliability (one-way ANOVA ICC)
Coppieters *et al.* 2001b	ULNT1	35 normal	20-28	Elbow extension, sensory response at P2	wrist extension, contralateral CLF	Elbow ROM is markedly reduced when adding test components and 80% of subjects reported paraesthesia in hand	Excellent reliability coefficient for intra-examiner (0.95-0.98) and for inter-examiner (0.91-0.97) reliability (one-way ANOVA ICC)
v. der Heide *et al.* 2001	ULNT2a	20 normal	43 (mean)	P1 and P2 during elbow extension	contralateral CLF	Normal muscular response to trapezius activity at P1. CCLF increases pain and muscle response	Onset of P1 and P2 both have high intra-examiner reliability coefficients (one-way ANOVA ICC)

5.1.2. Operational definitions in ULNT research

To allow an experimental study to be replicated and results corroborated a highly repro-ducible test protocol and a detailed description of operational definitions should be provided. An operational definition is a set of procedures that guides the process of obtaining a mea-surement and includes descriptions of the attribute that is to be measured, the conditions under which the measurement is to be taken, and the actions that are taken in order to obtain the measurement (Rothstein *et al.* 1991).

There has been an effort to ensure a standardised application by operationalising the starting position of the ULNT1 in 5 out of the 8 normative studies (Edgar *et al.* 1994, Coppieters *et al.* 2001ab, Balster & Jull 1997, v. der Heide *et al.* 2001, Table II-Appendix B). The subjects were placed in a supine position with the head in a neutral position. The arm to be tested was positioned in 90° glenohumeral abduction supported by an armrest. The shoulder girdle was additionally gently depressed with an initial force of 30 Newton prior to adding the other test components to neutralise the shoulder girdle elevation that is caused by the abduction of the arm. To lessen the variation of this variable a pressure sensor pre-inflated to 20 mmHg was placed on the superior part of the subjects shoulder and used to monitor the amount of force applied by the examiner, such that a pressure increase of 40 mmHg was recorded. The inter-examiner reliability of this measurement device had been analysed with a one-way ANOVA, and indicated no significant differences (Edgar *et al.* 1994). These studies demonstrate that it is possible to employ clear operational definitions, and to use quantifiable measurements aiding the comparison and evaluation of test results.

Stating in what end position outcome measures were taken (elbow extension/flexion, G/H abduction, range of CCLF) and under what criteria (pain, resistance) is another important

operational definition. In the method section of each study the quantifiable measurements (pain, resistance, ROM) and their respective instrumentation (i.e. goniometer, VAS) should also be clearly described with indications to their validity (see 5.2.2). In addition it is important to accurately describe and operationalise the sequencing of each ULNT (Table 5), so that results of identical tests can be related to each other.

Sensory responses of different ULNT components and sequences have been investigated in 90 asymptomatic subjects (Zorn *et al*. 1995, Table II). Comparing the consequences of three different ULNT sequences disclosed that the proximal to distal build up and the middle sequence showed similar sensory responses proximal to the elbow. However, distal to proximal build up produced symptoms distal to and including the elbow. Previous studies have shown that the longitudinal excursion of peripheral nerves depends upon the location and degree of joint motion (McLellan & Swash 1976, Shaw & Wilgis 1986). In this respect the proximal or distal build up may be valuable when examining patients with a suspected proximal or distal neural component to their shoulder or upper limb pain.

5.1.3. Control for independent variables

Another important criteria for the evaluation of the ULNT is the control for sensitising manoeuvres that play a critical role in confirming neural tissue involvement in CBP disorders. Will the experimental setting provide that each testing component can be added without any deviation from the previous position? In human cadaver studies, that investigated the influence of CCLF on the tension of the brachial plexus cords, this was easier achieved as the critical parts were fixed (Selvaratnam *et al.* 1989, Lewis *et al*. 1998, Kleinrensink *et al.* 2000, Table I). In contrast, demands on in vivo studies are not only focused on rigorous measurements, but also controlling the sensitising manoeuvres plays a

decisive role when assessing neural tissue involvement in symptomatic subjects (Yaxely & Jull 1993, Selvaratnam *et al.* 1994, Grant *et al.* 1995, v. der Heide *et al.* 2002, Table III). To prevent evasive movements when adding the test components some studies have used splints to fix the head in a neutral position and/or seatbelts around the hips and thorax (Yaxely & Jull 1991/1993, Selvaratnam *et al.* 1994, Zorn *et al.* 1995), but most studies had to keep the cervical spine free for the CCLF manoeuvres.

5.1.4. Defining normal responses

Normal responses in asymptomatic subjects should be established so they can be used as a standard reaction against which test responses from symptomatic subjects can be compared. The main dependent variables that produce quantifiable measurements (see 5.2.2) are range of motion at a defined end position, type and area of sensory response, reactive muscle activity, and response to sensitising manoeuvres (Yaxely & Jull 1991, Edgar *et al.* 1994, Zorn *et al.* 1995, Balster & Jull 1997, Hall *et al.* 1998, Coppieters *et al.* 2001ab, v. der Heide *et al.* 2001, Table II).

5.1.5. Validation of diagnostic tests

Validity is defined as the degree to which a meaningful interpretation can be inferred from a measurement (Rothstein *et al.* 1991). To do so the ULNT's construct validity, which is the theoretical basis for inferring an interpretation from the measurements, has to be stated. This also includes the evaluation of the ULNT's content validity, which means 'does the test measure what we think it will measure'. In the next sections the following questions should therefore be answered :

1. What does a positive test indicate?

2. What limits the movement during testing?

3. Can the test truly discriminate between different sources of pathology?

5.2. INVESTIGATING THE VALIDITY OF THE ULNT

In the subsequent sections research investigating the ULNT's anatomical and clinical validity and reflections on some limitations will be presented. Neural tissue provocation testing was originally based exclusively on a neuromechanical construct. The nervous system was believed to respond to mechanical induced stresses by distributing forces throughout the spinal cord, meninges, nerve roots and peripheral nerves. This was based on the fact that neural tissue has elasticity and movement relationships with adjacent tissues (Sunderland & Bradley 1961ab, McLellan & Swash 1976, Wilgis & Murphy 1986, Zöch 1992). It had been demonstrated that injured or inflamed nerves, are characterised by an increased sensitivity to mechanical load (Bove & Light 1997, Greening & Lynn 1998, Rempel *et al.* 1999, Mackinnon 2002), and could react to mechanical stimulation with an impaired compliance to movement and sensory responses. The original idea of the ULNT was that an ordered set of joint movements could be used to selectively increase tension within the neural tissue and their connective tissue sheaths (Elvey 1979b), thereby testing for increased mechanosensitivity.

To test the construct validity of this original hypothesis it is necessary to quantify the amount of tension transmitted onto the neural tissues during the ULNT. Additional points needing consideration include if the test response corresponds to the presumed tissues under test or to other structures, and what is the test's predictive value. In order to test the accuracy and

reliability of a diagnostic test, research that assesses the predictive value of positive and negative tests is necessary. A good diagnostic test minimises the probability of an occurrence of either a false-positive or false-negative result. To determine the accuracy of the neural tissue provocation test in clinical practice two different conditional probabilities are relevant. Firstly the test *sensitivity*, which determines the probability of testing positive in a truly present lesion, would be greater the more tension a test caused in the intended nerve. Secondly, the *specificity*, which determines the probability of testing negative in the absence of a lesion in the intended nerve, would be determined by knowing whether the test caused significant tension in other nerves than the intended one. This would mean that pain and symptoms could be due to increased tension in other nerves, and would lead to a false-positive response. It is therefore important to investigate whether a ULNT causes the highest tension in the nerve for which it was intended.

In clinical practice, however, it is often impossible to calculate the sensitivity or specificity of a diagnostic test because of confounding factors. For example the probability that a positive test indicates a true-positive finding, determines the sensitivity or positive predictive value of the test. In pain studies, false-positive responses could arise due to central sensitisation and secondary hyperalgesia (Woolf 1989/1991, Gracely *et al.* 1992). Moreover, the probability that a negative response represents a 'true-negative response,' or the negative predictive value, can be complicated because some peripheral neuropathies can lead to decreased mechanosensitivity, thus leading to false-negative results (Shacklock 1996, Woolf & Mannion 1999).

5.2.1. Biomechanical findings during the ULNT in human cadaver studies

Initially it was believed that manipulating the distal parts of an extremity could help in differentiating between peripheral nerve trunk and nerve root lesions (Elvey 1979a, Keneally *et al.* 1988, Butler 1991). However, as it was not known how the tensile forces were distributed along the peripheral nerve, or whether they were actually transmitted all the way up to the nerve root, the interpretation of a positive test response remained difficult. To clarify these questions and to validate the diagnostic value of the ULNT, human cadaver studies investigated the distribution of tension along the median, ulnar and radial nerves. Although informative findings from cadaver studies are not likely representative of measurements from living subjects because of post-mortem tissue changes, these studies contribute to the understanding of neural tissue dynamics, and provide a framework for the clinical findings presented in the next sections.

Selvaratnam *et al.* (1989, Table I-Appendix B) was the first to test Elvey's BPTT by conducting an anatomical study on 5 unembalmed human cadavers to confirm that adding CCLF to elbow and wrist extension produced a greater strain on the brachial plexus nerve roots. The highest tensile forces were measured between markers on the spinal nerve roots and the nerve trunks. The C5 nerve root displayed the most tension followed by the C6, C7, C8, and T1 nerve roots. An important finding was that elbow extension with CCLF produced a greater strain on the nerve roots than with ipsilateral CLF, which lead the authors to recommend the use of CCLF in differentiating between disorders of the upper quarter with and without brachial plexus involvement (Selvaratnam *et al.* 1989). The robustness of the obtained data is however questionable as the authors used a camera to document the changes of spatial location of the nerve roots during the ULNT, which were then calculated into strain scores, without establishing the reliability of this method.

Lewis *et al.* (1998, Table I) later confirmed these findings in a randomised, single-blinded study of 5 human specimens that only the elbow and wrist extension components of the ULNT1 produced a significant increase in the median nerve tension, which could be further increased by adding CCLF. The ULNT sequencing was operationalised and followed the protocol described by Keneally *et al.* (1988). Buckle force transducers were used for the measurement of tension, and were prior tested in a pilot study to be highly reliable (r=0.998). Shoulder girdle depression, and glenohumeral lateral rotation in contrast have been found to not significantly increase tension in the median nerve (Lewis *et al.* 1998), although it was not clear if these components were added in the final ULNT position. Earlier Ginn (1988, Table I) even reported of a decrease in the tension of all the brachial plexus cords during glenohumeral lateral rotation. However, these findings are not representative because they were based on a single specimen, and no reliability measures of the buckle force transducer or the procedures were given, so that the study cannot be replicated to test for its rigor. The results should therefore be interpreted with caution.

Kleinrensink *et al.* (1995b, Table I) investigated the distribution of tensile forces along 5 human median nerves. Since a positive correlation had been established between the measurements of tensile forces in embalmed and unembalmed human specimens a combined analysis of the data was accepted (Kleinrensink *et al.* 1995a). The effect of 22 arm positions in the normal range of motion (ROM) on the median nerve tension was measured with buckle force transducers at the site of the axilla, elbow, and wrist. All 22 joint positions were studied three times as test and retest, and after simulating an obstruction of the gliding mechanism of the median nerve through the pronator teres muscle. The transducer had a high test-retest reliability and was calibrated after each measurement. The elbow and hand

position were clearly described, however no reference to the position of the shoulder girdle was given, which has been defined as an integral part of the ULNT (Butler 1994, Table 5).

With the elbow in full extension and hand in neutral position, the authors found that altering the position of the shoulder to maximal abduction, retroflexion and lateral rotation significantly influenced the tension of the median nerve at the level of the axilla and elbow, while tension at the level of the wrist was not influenced. With the shoulder in 90° abduction, wrist extension combined with an extended elbow increased tension in all parts of the nerve, whereas wrist extension combined with elbow flexion only caused an increase in the distal part of the median nerve. Manipulating joints in the distal part of the upper extremity can increase tension in the most proximal part of the median nerve, however, changing the shoulder joint position cannot alter the tension in the distal part of the median nerve. Thus, there is anatomical evidence that differentiating between lesions in the upper and lower part of the median nerve may be possible by manipulating specific joints, but this needs to be confirmed by clinical investigations.

A more recent anatomical and biomechanical study by Kleinrensink *et al.* (2000, Table I) clarified the specificity of the three basic ULNTs by Butler (Table 5). The outcome variables were the mechanical tension assessed with a calibrated buckle force transducer on the medial, lateral and posterior cords of the brachial plexus measured beneath the clavicle, as well as on the proximal parts of the median, ulnar and radial nerves. A fixed protocol was used for the sequence of arm positions and each measurement was taken twice to be analysed with a multiple regression analysis.

All three ULNTs caused a higher tension in the medial cord than in the lateral or posterior cords (Figure 3, Appendix C). Only the median biased ULNT1 had shown to be both

sensitive and specific for the median nerve and was recommended as a valid test by the authors. The radial biased ULNT2b caused more tension in the median nerve than in the radial nerve itself, and was not specific. Equally the ulnar biased ULNT3 was not specific since there was no significant difference between the tension caused in the ulnar, median and radial nerves (Table 8). The proposed selective stress on specific nerves and cords of the brachial plexus could therefore not be confirmed by this study.

TABLE 8 :	Mean tensile forces in Newton (±SD) caused by the ULNT (Kleinrensink *et al*. 2000)		
	ULNT1 (medianus)	ULNT3 (ulnaris)	ULNT2b (radialis)
Median nerve	10.88 (5.88)	0.59 (0.29)	8.52 (5.19)
Ulnar nerve	1.18 (0.98)	3.29 (3.04)	1.76 (1.57)
Radial nerve	1.86 (0.59)	2.94 (2.55)	5.88 (2.35)
Medial cord	9.31 (6.17)	4.21 (4.80)	8.13 (5.88)
Lateral cord	5.10 (4.21)	0.49 (0.69)	4.41 (3.42)
Posterior cord	1.57 (1.47)	2.55 (2.06)	5.00 (2.74)

Although the results by Kleinrensink *et al.* (2000) indicated the use of only the ULNT1 as a valid test, there has been a contradicting report on the specificity of the ULNT3 from a single case report of a proven ulnar neuropathy (Shacklock 1996). In this case report Shacklock described that the ULNT3 was able to reproduce the patient's symptoms while the ULNT1 was not. One reason for this discrepancy could be the fact that Kleinrensink *et al.* (2000) conducted a cadaver study where tension was measured as the outcome variable, whereas in the clinical setting the main outcome measurements were pain response and ROM.

Another difference influencing the results might be a deviation between the movement sequencing. The median ULNT1 appears to be the most specific test in transferring tensile forces to its corresponding nerve, however, the ULNT3 produces more tension in the ulnar nerve than the median biased ULNT1 (Table 8). This could explain why, despite the weaker tensile forces in total, the ULNT3 was still specific for the ulnar nerve. Clinically this means that if a patient presented with an isolated ulnar neuropathy the ULNT3 would most likely be positive, but if a patient had both ulnar and median nerve pathology the ULNT3 could not be specific in isolating the ulnar neuropathy.

A basic assumption of the ULNT is based upon the ability to selectively move neural tissue in the absence of mechanically affecting neighbouring non-neural tissue. To test this it is important to verify which components of the test move the neural tissue and which components cause tension in non-neural structures. Anatomical connections in the cervical region suggest that upper limb neural testing may equally stimulate the intervertebral discs, interspinous ligaments, zygapophyseal joints and muscles as these structures have shown to possess nociception and could therefore refer pain to the upper limb (Dwyer *et al.* 1990, Bogduk 1994, Moses & Carman 1996). During neural tissue provocation testing, especially in the end range position, many non-neural structures are stretched.

A clinical study on the subclavian artery of 2 embalmed human specimens demonstrated that CCLF with elbow extension alone or with additional wrist extension induced a stretch on the first and third segment of the subclavian artery (Wilson *et al.* 1994, Table I). The authors also stated that the strain on the lateral cord of the brachial plexus was greater than on the first part of the subclavian artery. Because no positions of the shoulder girdle or the glenohumaral joint were described no specific comparisons can be made to the ULNT.

Furthermore, these findings contradict Elvey (1979b, 1995), who observed no movement in the subclavian artery during CCLF. Even though Elvey produced no quantitative data to support his statement, the results from Wilson *et al.* (1994) may equally be flawed because the subclavian artery was prepared by freeing it from the clavicle and sternocleidomastoid muscle destroying its natural surroundings, which may have presented some form of natural fixation thereby leading to a systematic bias of the obtained data.

To sum up, the initial idea of manipulating the distal parts of an extremity to help different-tiate between peripheral nerve trunk and nerve root lesions has not been confirmed. What the cadaver studies have shown is that the main components of the ULNT capable of inducing tension changes from the proximal parts of the median nerve to the brachial plexus cords are wrist and elbow extension, and that this tension can be intensified by adding CCLF (Kleinrensink *et al.* 1995b/2000, Lewis *et al.* 1998). Selvaratnam *et al.* (1989) showed that adding CCLF to the ULNT selectively increased the tension in the brachial plexus nerve roots, with the highest tension at C5 and the lowest at T1. Interestingly the C5 and C6 nerve roots supply the upper trunk of the brachial plexus, which leads into the lateral and posterior cords (Figure 3). In contrast, Kleinrensink *et al.* (2000) found the medial cord to be under most tension when adding contralateral cervical rotation to the ULNT. This discrepancy might be due to the different sites of measurement (nerve roots vs. distal brachial plexus cords).

Unexpectedly Kleinrensink *et al.* (2000) also found that when adding contralateral cervical rotation to the ULNT the tension in the medial cord remained the same while tension in the lateral and posterior cords increased up to 50% when compared to ULNT with the head in neutral. This supports the idea that the brachial plexus plays a role in the distribution of

tensile forces (Butler 1991). Furthermore, it has been shown that the median ULNT1 is the most specific test in transmitting tension to the proximal part of the corresponding nerve, and is the only ULNT that can be recommended as a valid test (Kleinrensink *et al.* 2000). However, according to the research at hand the original idea that the ULNT can selectively isolate neural tissue from non-neural tissue has further to be questioned.

Although the information gained from these anatomical studies is valuable there are some methodological limitations that have to be considered, such as the small sample size (maximal 5 specimens), and the degenerative changes associated with the higher age of the samples. Apart from changes post mortem and the effect of conservation on the tissues, it is also not know what existing pathologies and diseases of the specimens may have influenced the measurements. As these studies are purely biomechanical investigations, they have shown that tension is transmitted to the proximal parts of the nerves and the cords of the brachial plexus; however, so far no relationship between the amount of tension produced in a nerve and its sensory response has been established. Therefore the next step in the evaluation of the test's efficacy is to investigate the normal reactions to the ULNT in asymptomatic subjects.

5.2.2. Quantifiable measurements in normative ULNT studies

To standardise the ULNT as a diagnostic test quantifiable outcome measurements, detected during the testing procedure in asymptomatic subjects, have to be determined (Table 9). These measurements also include the criteria that determine the end of the test, which can be either resistance (Yaxely & Jull 1991, Hines *et al.* 1993) or pain (Balster & Jull 1997, Coppieters *et al.* 2001b/2002b/2003ab, v. der Heide *et al*. 2001). However, determining the

end condition relies heavily on the perception and manual skills of the examiner, and on the report made by the subject.

TABLE 9: Quantifiable measurements advocated for monitoring	
Outcome Measure	**Method Of Data Collection**

1. Compliance to movement:
  ■ onset of resistance (R1)
  ■ maximal tolerable resistance (R2)
  ■ range of motion (ROM)
  ■ reactive muscle activity
  ■ shoulder girdle elevation
	1. Compliance to movement:
	  ■ manual skills of examiner
	  ■ manual skills of examiner
	  ■ standard/electro-goniometer
	  ■ EMG
	  ■ load cell

2. Sensory responses:
  ■ type and area of sensory response
  ■ onset of pain (P1)
  ■ maximal tolerable pain (P2)
  ■ reproduction of the patient's symptoms
  ■ pain intensity
	2. Sensory responses:
	  ■ verbal report of patient
	  ■ verbal report of patient
	  ■ verbal report of patient
	  ■ verbal report of patient
	  ■ numeric pain scale

3. Effects of sensitising manoeuvres:
  ■ increase of sensory responses
  ■ reproduction of the patient's symptoms
	3. Effects of sensitising manoeuvres:
	  ■ numeric pain scale
	  ■ verbal report of patient

The onset of resistance (R1) and its clinically acceptable maximum (R2) may be at any point within the normal passive range of motion (Maitland 1991). If the neural tissue is sensitised normal movement could cause a provocative mechanical stimuli that may lead to a pain response and/or to non-compliance to movement (Hall & Elvey 1999). One feature of this non-compliance is proposed to lie in the increased and reactive tissue stiffness offered by muscle spasm (Maitland 1986) caused by muscles antagonistic to the painful direction of the movement. The other feature is an increased through range tissue stiffness, which is thought to result from pathological articular or connective tissue. However, no studies have deter-mined whether a physiotherapist can differentiate between tissue resistance and muscle spasm (Hall *et al.* 1998).

Some studies have questioned whether the onset of resistance or pain truly reflects what is occurring when testing the neural tissue (Wright *et al.* 1994, Hall & Quintner 1996, Balster & Jull 1997, Hall *et al.* 1998). The following study quantified the ability of physiotherapists to determine the onset of resistance during the SLR test in an age and subject matched design of 20 subjects with no previous history of back or leg pain, and 20 subjects with validated L5/S1 radiculopathy (Hall *et al.* 1998, Table III-Appendix B). The study compared the range of SLR at R1, the electromyographical (EMG) activity of the hamstring muscles during the SLR test, and the moment of the stretched tissues (M*r*) between the two groups. An important finding was that R1 was significantly earlier in the range of SLR than the first increase of muscle activity, and that there was no significant difference between R1 in the control and the radiculopathy group, which means that R1 is not a reliable dependent variable when assessing differences in neural mechanosensitivity between symptomatic and asymptomatic subjects.

In all the subjects with radiculopathy the onset of muscle activity (M1) was significantly earlier during the SLR test than in the control group, and probably accounted for the increase in M*r* at that point rather than the increase in neural tissue tension. The authors concluded that M1 was a more accurate measurement representing neural mechanosensitivity than R1. The accuracy and reliability of the electrogoniometer and the device to measure M*r* were assessed in a pilot study and were found to be excellent (r=1.0). However, as the Pearson's *r* tends to overestimate reliability and does not account for systematic observer bias (Haas 1991b) these values need to be reassessed. The intra-examiner reliability was established for the repeated measurements of R1 and M*r* and was calculated to be good to excellent (ICC value of 0.75-0.98), but as the examiner was not blinded the result may only be a reflection

of a strong self consistency, therefore the inter-examiner reliability needs to be established before further implications can be made.

As M1 has been shown to be a clinically more significant measure than R1 the subsequent normative studies were interested in the relationship between muscle activity and neural mechanosensitivity in asymptomatic subjects. Bearing in mind that in normal subjects abnormal age-related changes of the cervical spine can be found (Boden *et al.* 1990) the following studies included only asymptomatic subjects, that had no previous history of CBP disorders or systemic diseases related to neuropathy.

Balster and Jull (1997) investigated the relationship between the ULNT1, upper trapezius muscle activity and the range of neural tissue extensibility in 20 young male asymptomatic subjects (Table II). The ULNT1 was performed according to Elvey (1986), and shoulder girdle depression was operationalised (Edgar *et al.* 1994). Reliability measures were only performed for the EMG activity and revealed no significant difference between trials, but the examiner reliability for elbow ROM was omitted. Two groups with greater and lesser neural tissue extensibility were formed by baseline measurements of their elbow extension in the final ULNT1 position.

In comparison the two groups showed no difference in perceived pain levels rated through a verbal analogue scale. The EMG measurements revealed that the lesser extensible group exhibited a significant greater trapezius muscle activity at the onset of pain (p=0.01), at the limit of elbow extension (p=0.01), and at the limit of CCLF (p=0.006). However, neither the criteria for the limit of elbow extension and CCLF were defined (pain or resistance) nor were any ranges presented in the analysis. Thus, the repeatability of these measurements as well as

comparison of the data to similar research is impeded. These methodological deficiencies therefore weaken the reliability of this study.

The hypothesised mechanisms explaining this neuromusculoskeletal interaction were that the muscle activity and pain response are either attributed to a heightened flexion withdrawal reflex elicited by afferent input upon sensitised spinal neurons (Woolf 1989, Wright *et al.* 1994) or by nociceptive input from tension sensitive neural tissue such as the nervi nervorum (Bove & Light 1997). Considering that both groups experienced the same level of pain but displayed a different muscle response indicated that the nociceptive mediated withdrawal reflex may not be the only mechanism involved in protecting the neural structures (Balster & Jull 1997).

Rather the authors suggested that stretch receptors in the neural tissue were responsible for this protective muscle action, on the grounds of recently identified stretch receptors in the phial ligaments supporting the spinal arteries. However, the existence of stretch receptors in the neural tissue has not been confirmed. Noteworthy is that no correlation was found between the increase in muscle activity and the increase in pain intensity in asymptomatic subjects (Balster & Jull 1997), which contradicts the hypothesised mechanism. If a correlation can be found in symptomatic subjects still needs to be investigated, however.

The purported theory of a nociceptive mediated withdrawal reflex has further to be reconsidered in view of the results obtained by Edgar *et al.* (1994). This correlation study investigated the relationship between the extensibility of the upper quarter neural tissue tested via the ULNT1 and the muscle length of the upper trapezius muscle in 60 asymptomatic young male volunteers (Table II). Two groups (n=30) were formed in the same manner as in the study by Balster and Jull (1997). Shoulder girdle depression was

standardised (see section 5.1.2) such that a pressure increase from 20 mmHg to 60 mmHg was performed on each subject, before adding the other test components.

No significant differences between trials were found for inter-examiner reliability of shoulder girdle depression, or for the intra-examiner repeatability of trapezius length and ULNT1 measurements. Although statistical calculations were not adequately presented, the procedure for obtaining these measurements was described in detail. The authors found that the length index of the trapezius muscle was significantly less in the subjects with lesser neural tissue extensibility independent of elbow extension or flexion, and concluded that neural tissue extensibility should be assessed prior to interpreting the results of length tests of the upper trapezius muscle in patients that present with pain in the upper quarter.

While Balster and Jull (1997) concluded that the heightened trapezius muscle activity was a protective reaction to the decreased extensibility of neural tissue, Edgar *et al.* (1994) found that in subjects with lesser neural tissue extensibility the trapezius muscle was equally lesser extensible. This is not surprising as subjects with decreased mobility will exhibit this charac-teristic in all of their tissues and not just in their neural tissue. In this respect it will be difficult to distinguish whether the nociceptive reflex is elicited to protect the less extensible neural tissue or musculature.

However, these normal findings should also be interpreted with regards to the painful reactions of the trapezius muscles in disorders of the cervicobrachial region. Patients suffering from cervical radiculopathy have been found to react with a reduced muscle tension (EMG recording), due to the decreased microcirculation of the trapezius muscle (Löfgren *et al.* 2001), whereas in chronic neck pain populations increased values of muscle

tension were shown (Larsson *et al.* 1999). In so far the trapezius length should be considered when assessing the neural tissue of chronic neck pain patients.

The second group of quantifiable measures used when testing the ULNT are the sensory responses (Table 9). In a same-subject design study by van der Heide *et al.* (2001) the correlation between pain and muscle activity responses to the ULNT1 were examined in 20 asymptomatic subjects (Table II). The starting position (Edgar *et al.* 1994) and the testing procedure were operationalised, and all statistical analyses for the measurements were clearly described. The test was performed on both arms with the cervical spine in neutral, and in CCLF as a sensitising manoeuvre. P1 and P2 were used as indicators to cease the elbow extension, which the subjects determined by using an external trigger. Elbow extension was measured with a calibrated electrogoniometer, and EMG recordings determined the muscle activity. There was no statistical significant difference for the intra-examiner reliability at P1 and P2, and the onset of pain showed a high reliability between the three trials.

The results displayed that pain responses and muscle activity of the trapezius muscle were evoked in the majority of all subjects and could be defined as a normal physiological response. A total of 59% (n=11.8) of the subjects had an onset of muscle activity at P1, but as Balster and Jull (1997) did not state the degree of elbow extension at P1 these results cannot be compared. In some subjects trapezius activity was measured before the onset of pain. Additionally adding CCLF had a sensitising effect in 18 subjects, who felt an increased intensity of their sensory responses. Of the 37.5% that had no onset of pain with the head in neutral, CCLF had a sensitising effect on the onset of pain and muscle activity.

Even though there was a strong correlation between trapezius activity and pain, approximately 16% (n=3.2) of the total study population showed trapezius activity without pain, but

experienced symptoms such as stretching sensation or paraesthesia in the arm. If muscle activity occurred as a response to symptoms other than pain, this would contradict the hypothesis of a protective motor reaction in healthy subjects, but a study with a larger sample size would be needed to confirm this. The large variation in responses may partly be due to the variation of pain responses. Pain is a sensory-emotional phenomenon and quantitative measurements do not account for the affective perception of pain that has an impact on pain intensity.

As proposed by Elvey (1979b/1986) and Keneally *et al.* (1988) an abnormal response to the ULNT may not only involve just a pain response but also the reproduction of the patient's exact symptoms. To decide when a response to the ULNT is abnormal, it is important to know the standard response to neural tissue provocation testing in asymptomatic subjects. For the therapist to accurately interpret a neurodynamic test the difference between a motion restriction indicative of a dysfunction and a limitation to movement, which can be considered as a normal response, has to be established.

Yaxely and Jull (1991, Table II) used the radial biased ULNT2b to demonstrate the normal sensory response at R2 in 50 asymptomatic young subjects (25 female, 25 male). A stretch felt over the radial aspect of the proximal forearm at 41.45° (± 4.06°) glenohumeral abduction and wrist flexion was reported in 84% of all responses (left and right arm combined). The abduction range, measured with a standard goniometer, was not influenced by the gender of the subjects nor by which side was tested. Excellent inter-examiner reliability (p=0.949) and repeatability (p=0.999) for the mean range of glenohumeral abduction in the ULNT2b were calculated.

An increase in "arm symptoms" with the addition of CCLF as a sensitising manoeuvre to the final test position was recorded, in 86% for the right arm and 90% for the left arm. However, the authors did not state if the area was identical to the initially reported stretch over the radial aspect of the arm. To what extent the sensory responses really reflect the reaction of the stretched radial nerve have further to be questioned in the light of the results by Kleinrensink *et al.* (2000, Table 8), who demonstrated that ULNT2b caused more tension in the median nerve than in the radial nerve, therefore the arm symptoms could have be elicited through stretching the median nerve. Another limitation appears to be that for the ULNT2b no standardisation for the starting position in an experimental setting has yet been defined. As in the ULNT1 the shoulder girdle was initially depressed but with what amount of force has not been stated.

To analyse the effect of different ULNT1 components on the limitation of elbow range of motion and on the provocation of sensory responses Coppieters *et al.* (2001b) investigated four test variations on 35 asymptomatic young male subjects (Table II). The starting position was operationalised (Edgar *et al.* 1994), and baseline measurements of the elbow and wrist joints were taken prior to testing. Elbow extension in a non-ULNT position was 182.6º (±3.5º). The addition of each test component resulted in a significantly reduced elbow ROM (ULNT1 in neutral 179.5º ±8.8º; ULNT1 + wrist extension 169.0 ±13.9º; ULNT1 + CCLF 154.7º ±13.2º; ULNT1 + wrist extension + CCLF 143.9 ±16.1º).

The sequence of the test variants was randomly allocated, and the analysis of variance revealed no significant difference between the three repetitions. Sensory responses were described as a (painful) stretch or paraesthesia predominantly evoked in the region of the added components. The relatively high incidence of paraesthesia (76%) suggests that at least

some responses are neurogenic in origin. The average numeric pain rating (0-10) increased progressively as the test components were added from 4.4 to 6.6. The distribution of sensory responses revealed that when CCLF was added sensory responses in the more proximal areas of the upper quarter were reported than without CCLF.

The excellent inter-examiner reliability measures (ICC 0.94-0.97) imply that elbow ROM may be used as a reliable comparable sign in the clinical evaluation of the ULNT1. The individual test components had a cumulative effect on limiting the elbow ROM when added simultaneously. Furthermore, the elbow ROM was significantly influenced by the position of the cervical spine, which could not have been caused by the mono- and biarticular structures around the elbow joint. The authors suggested that the only continuous structure that could, at least partly, influence the range of elbow extension is the nervous system, since blood vessels, skin or the lymphatic system are unlikely to distribute such restrictive forces. However, the potential role of the fascia to restrict movement will need to be investigated in the future. The correspondence between the regions of added components and the area of sensory responses implicate that reproducing symptoms in patients and altering these by changing distant components of the test such as wrist extension or CCLF, will be essential for the structural differentiation between neural and non-neural tissues.

One of the signs advocated for monitoring during ULNT1 in subjects with upper quarter disorders is the involuntary elevation of the shoulder girdle (Elvey 1994). Coppieters *et al.* (2001a) investigated the shoulder girdle elevation force during five variants of the ULNT1 in 35 young asymptomatic male subjects (Table II). In agreement to Edgar *et al.* (1994) the shoulder girdle was depressed with an initial force of 30 Newton prior to adding the ancillary manoeuvres of the wrist and/or cervical spine. Extension of the elbow and wrist measured

with two electrogoniometers were stopped when the participant reported a substantial discomfort (P2).

A calibrated load cell was used to measure the amount of shoulder girdle elevation force that had been tested reliable in a former study (Coppieters *et al.* 1999). The sequence of the test variants were randomly allocated to balance possible effects of repeated testing. A significant increase in force at the end of range for all test variants was observed, and attributed to the probable loading of neural structures and their intimate surroundings. Although the exact mechanisms causing the elevation remain unexamined, the authors support the hypothesis of a protective muscle activity (Wright *et al.* 1994, Balster & Jull 1997) as a reaction to loading the neural tissue beyond its physiological range.

In a subsequent experimental study Coppieters *et al.* (2002a, Table II) additionally investigated the influence of the shoulder position on the ULNT1 components. Operational definitions and measuring devices were the same as described in the studies by Coppieters *et al.* (2001ab). When the ULNT1 with wrist extension and CCLF was performed in 90º glenohumeral abduction the mean range of elbow extension was 180.2º (SD 6.7), but decreased significantly to 144.3º (SD 12.6) when the shoulder was additionally laterally rotated. These findings suggest that adding glenohumeral lateral rotation to the ULNT1 increases the tension on the neural tissue thereby affecting the elbow ROM.

However, contradicting opinions exist on the importance of the glenohumeral lateral rotation component in exerting tension on the median nerve. In cadaver studies by Ginn (1988) and Lewis *et al.* (1998) lateral rotation was said to have no effect on the tension of the median nerve. On the other hand Kleinrensink *et al.* (1995b) demonstrated that altering the shoulder joint to maximal abduction, retrofelxion and lateral rotation resulted in a significant increase

in the median nerve tension, but only at the level of the axilla and the elbow joint. To resolve this discrepancy future anatomical studies should be conducted under the operational definitions and the sequencing protocol that have been established by physiotherapists. This way biomechanical findings are more likely to be comparable to the findings made in clinical practice and may contribute to a better understanding of the processes under investigation.

In conclusion the response to neural tissue provocation testing is assessed by measuring the ROM (elbow extension for the median biased ULNT1 and glenohumeral abduction for the radial biased ULNT2b) at a defined end position (M1, R1, R2, P1, P2). The onset of muscle activity (M1) has been found to be a more accurate measurement representing neural mechanosensitivity than R1 (Hall *et al.* 1998). Only a few studies have actually demonstrated the presence of muscle activity during neurodynamic testing (Balster & Jull 1997, Hall *et al.* 1998, v. der Heide *et al.* 2002). The possible mechanisms for this neuromusculoskeletal interaction have not been fully understood, but are currently under further investigation. Due to weaknesses in the study design the clinical validity of the ULNT2b remains questionable, as findings did not support it to be specific for the radial nerve.

A gradual increase in shoulder girdle elevation during ULNT1 was observed and regarded as a normal sign in the interpretation of the test (Coppieters *et al.* 2001a). Sensory responses have been shown to be in the area of the (added) ULNT1 test components, and are thought to be partially of neurogenic origin (Coppieters *et al.* 2001b). CCLF and wrist extension are sensitising manoeuvres that increase the sensory responses and can be used for the structural differentiation between neural and non-neural tissues. The strong correlation between increased pain intensity and decreased ROM (Coppieters *et al.* 2001b) suggests that ROM as a comparable sign can be used as a reliable dependent variable.

The large variability between subject responses, however, challenges the suggestion of the use of an *absolute norm* of one response to define an impairment (i.e. standardised degree of elbow extension). Therefore, research needs to analyse whether the ROM of the uninvolved side can be used as a relative norm to differentiate between abnormality and normality.

Before implications can be generalised from these findings the following limitations need to be considered. 1) The small sample size (Balster & Jull 1997, Coppieters *et al.* 2001a, v. der Heide *et al.* 2001) may not have provided a sufficient power calculation. 2) Due to the lack of blinding the examiners the amount of experimenter bias is not known. 3) Most studies have incomplete pain measurements of either the quantitative (sensory/physiological) or the qualitative (emotional/affective) aspect. 4) Clinical experience has show that cervicobrachial pain syndromes are more common after the age of 40 (Persson *et al.* 1997), with a higher prevalence in women than in men (Borghouts *et al.* 1998, Croft *et al.* 2001). However, the results reflect the normal responses of a very young in the majority male population (Table II), which may be a confounding factor. It is therefore necessary to obtain data from an asymptomatic population that is gender and age matched with the patient group of interest to better account for these dependent variables.

5.2.3. The use of the ULNT in clinical practice

Sensory responses and reactions in normal subjects to the provocation of neural tissue have been described in the previous section. To standardise the application of the ULNT, testing procedures have been operationalised (Edgar *et al.* 1994) and reliability of outcome measures as P1, P2 and elbow ROM have been established (Coppieters *et al.* 2001b, v. der Heide *et al.* 2001). However, to use the ULNT as a diagnostic test the clinician has to know what the indications are for a positive test. The relevant signs monitored during diagnostic testing

should therefore have the ability to differentiate between normality and abnormality. In this last section research is presented that investigates the relevant outcome measures to neural tissue provocation testing in symptomatic subjects (Table 10).

TABLE 10:	Relevant outcome measurements advocated for the use in ULNT testing in clinical practice

	1. compliance to movement:
	    ▪ limited ROM at the point of submaximal pain (P2) in comparison to the uninvolved side
	    ▪ increased shoulder girdle elevation force in comparison to the uninvolved side (not quantifiable without load cell)
	
	2. sensory responses:
	    ▪ type and area of sensory response in comparison to the uninvolved side (recorded on a body chart)
	    ▪ pain intensities (recorded on a numeric pain scale)
	    ▪ reproduction of the patient's symptoms
	
	3. effects of sensitising manoeuvres:
	    ▪ increase of sensory responses
	    ▪ reproduction of the patient's symptoms

Neural tissue provocation tests have been used clinically to assess part of the nervous system in a number of diverse neuromusculoskeletal disorders of the upper quadrant, such as peripheral nerve entrapment (Shacklock 1996), lateral epicondylalgia, (Yaxely & Jull 1993, Vicenzino *et al.* 1996), over-use syndromes (Quintner & Elvey 1993, Clare 1994, Grant *et al.* 1995, Greening *et al.* 1999/2000), or post-traumatic neurogenic pain (Sweeney & Harms 1996). Quintner was one of the first who used the ULNT for a specific patient population and described abnormal sensory responses to Elvey's version of the ULNT in patients with neck injury after motor vehicle accidents (Quintner 1989), and in patients with persistent cervicobrachial pain (Quintner 1990). Sweeney and Harms (1996, Table III) reported that 22 of 29 patients who had mechanical allodynia after hand surgery or trauma showed a

significant difference of sensory responses and elbow ROM between the involved and uninvolved side during ULNT1 testing. More recently ultrasound imaging showed a lack of median nerve movement during wrist flexion in RSI patients to painfully limited elbow ROM during ULNT1 testing (Greening *et al.* 2000).

Investigating abnormal responses to the ULNT2b the neural tissue extensibility has been investigated in 15 screen based keyboard (SBK) operators (Grant *et al.* 1995, Table III), and in 20 patients suffering from unilateral epicondylitis (Yaxely & Jull 1993, Table III). The involved side exhibited an average of 12º lesser range in glenohumeral abduction at R2 in comparison to the uninvolved side. The inter-examiner reliability for glenohumeral abduction at R2 in asymptomatic subjects had been shown in an earlier study (Yaxely & Jull 1991). The testing sequence was performed in accordance to Butler (1991) in that the shoulder girdle was depressed to an end range and maintained during the whole procedure.

The main outcome measure, glenohumeral abduction, was measured with a standard goniometer at the point were tissue resistance limited further range. Grant *et al.* (1995) reported that all 15 female SBK operators experienced a strong stretch on the proximal radial aspect of the forearm in the final ULNT2b position. However, 90% (n=9) of the control group reported the same sensory response, which has previously been shown to be a normal response in asymptomatic subjects (Yaxely & Jull 1991). Adding CCLF as a sensitising manoeuvre increased symptoms in 73% of the SBK operators but also in 70% of the control group (Grant *et al.* 1995). In only 55% of the cases with epicondylitis (Yaxely & Jull 1993) symptoms were reproduced at R2 but no correlation between pain intensity and R2 was presented.

In both studies glenohumeral ROM was reduced in comparison to the asymptomatic group or side, and was claimed to be a sign of reduced neural tissue extensibility. This purely mechanistic approach leaves other possible limiting factors to be unaccounted for, such as the more recent findings of a possible protective motor response of the shoulder girdle musculature (Balster & Jull 1997, Coppieters *et al.* 2001a, v. der Heide *et al.* 2002). The small sample size and the inappropriate reliability calculations (Grant *et al.* 1995) further question the statistical power of these results (see section 5.1.1). While the areas of sensory responses were similar between the asymptomatic and symptomatic sides the intensities elicited by the ULNT2b were different (Yaxely & Jull 1993, Grant *et al.* 1995). However, when assessing painful conditions it is more sensible to measure limited ROM at P2 and not at the clinically insignificant point of maximal resistance.

As neurodynamic tests are often performed repeatedly within one treatment session to assess the immediate effects of treatment, the patient's ability to reliably indicate the moment of pain is an essential criterion for the clinical use of neural tissue provocation tests. The stability and reliability of the occurrence of 'pain onset' and 'submaximal pain' throughout the ROM during the ULNT1 was analysed in a laboratory and clinical setting (Coppieters *et al.* 2002b, Table III). The ULNT1 was performed in a single session on a total of 27 patients with unilateral and bilateral neurogenic CBP disorders. In addition, two examiners performed the ULNT1 with the cervical spine in neutral, with wrist extension, with CCLF, and with both wrist extension and CCLF on 10 asymptomatic subjects in laboratory conditions only. The operationalised starting position (Edgar *et al.* 1994) and a calibrated load cell for shoulder girdle depression (Coppieters *et al.* 1999) were used only in the laboratory setting. In the clinical setting the ULNT1 was performed following the operational definition by

Butler (2000), which means the shoulder girdle was controlled in a neutral position. Corresponding angles of elbow ROM were measured with an electrogoniometer in both settings.

Intra- and inter-tester reliability coefficients were greater than 0.95 (SEM $\geq$ 4.9º) for P1 and P2 in the asymptomatic group. The reliability coefficients for the symptomatic group in the laboratory setting for P1 was greater than 0.98 (SEM $\geq$ 2.8º), and in the clinical settings for P2 it was greater than 0.98 (SEM $\geq$ 3.4º). Although pain threshold and tolerance levels varied widely between subjects the study demonstrated the moment of pain can be detected reliably, an essential criterion when using neurodynamic tests in clinical practice. Furthermore, the absence of additional devices, used for operationalising the starting position in an experimental setting, can be compensated with sound operational definitions and skilful handling in a clinical setting.

The increasing importance of neural tissue involvement in minor peripheral neurogenic pain disorders has lead to the development of treatment strategies as the cervical glide technique (Elvey 1986, Vicenzino *et al.* 1994). This mobilisation technique was developed on the basis of Elvey's concept of impaired neural dynamics in upper limb disorders, and has been shown to improve pain intensity, symptom provocation and range of motion (Vicenzino *et al.* 1995/1996, Hall *et al.* 1997, Cowell & Phillips 2002, Coppieters *et al.* 2003b). According to a comprehensive review of the literature up through 1997, Elvey developed a set of clinical criteria to identify patients with minor peripheral neurogenic pain disorders amenable to physiotherapy management: (1) the presence of an active movement restriction that is related to a disorder of a specific nerve trunk, (2) a passive movement restriction that correlates with the active movement dysfunction, (3) an abnormal response to neural tissue provocation testing, (4) painful nerve trunk palpation, and (5) signs of a local musculoskeletal dysfunc-

tion responsive to physiotherapy, such as cervical segmental motion restriction indicating a cause of neurogenic disorder.

In a recent single-blinded RCT (Coppieters *et al.* 2003a, Table III) 20 patients with non-acute unilateral or bilateral neurogenic cervicobrachial pain were initially assessed with the five criteria described by Elvey (1997), and randomly allocated into two matched groups. The shoulder girdle elevation force during the ULNT1, and the effects following a cervical mobilisation technique were investigated. The operational definitions for the starting position, and the standardisation of the measurement devices established in earlier studies (Edgar *et al*. 1994, Coppieters *et al.* 1999/2001a) were used. Outcome measures were elbow ROM at the point of substantial discomfort (P2) as reported by the patients, and the shoulder girdle elevation force during ULNT1. The highest pain intensity was recorded using a numeric pain intensity rating scale (0-10).

The experimental condition consisted of a cervical contralateral glide technique at one or more segments (Elvey 1986, Vicenzino *et al.* 1994) with the involved arm in a neural preloaded position, in which several components of the ULNT1 were applied. A lateral translatory movement away from the involved side was performed, with the patient in a supine position minimising gross cervical side flexion or rotation. The technique is described to either selectively move the nerve root complex within the spinal intervertebral canal, or to move the spinal intervertebral canal in relation to the nerve root complex when it is held tense by means of shoulder girdle fixation (Elvey 1986).

The control condition consisted of pulsed ultrasound applied for 5 minutes over the most painful area, during which the arm was positioned in an unloaded position. To reduce the "therapist effect" both the experimental and the control interventions were performed by the

same therapist. The results showed a significant increase in shoulder girdle elevation force during ULNT1. However, the sudden increase in shoulder girdle elevation force occurred earlier in range on the involved side than on the uninvolved side. The immediate effect of the cervical mobilisation treatment on the involved side was that an increase in shoulder girdle elevation force occurred later in range and that the amount of end force was significantly larger than before treatment. These findings were regarded as a normalisation in force generation and were associated with the significant decrease in pain intensity and increase in ROM on the involved side. For the control group no treatment effects relating to end force, ROM, or pain intensity were observed.

The immediate hypoalgesic effect of the cervical mobilisation technique (Wright 1995, Wright & Vicenzino 1995, Vicenzino *et al.* 1998) may be a plausible explanation for a retarded nociceptive flexor withdrawal reflex proposed in earlier studies. However, future electromyographical studies need to confirm if changes in force generation correlate with changes in muscle activity. In a subsequent RCT Coppieters *et al*. (2003b Table III) used the ULNT1 to additionally assess the distribution of elicited sensory responses. The same methods and operational definitions were used as described in the former study (Coppieters *et al*. 2003a). Significant differences between the involved and uninvolved side were found for the range of elbow extension (mean difference 25.6º), for pain intensity (mean difference 3.1 points on VAS), and for the symptom area, which was approximately 2.9 times larger. These results support the use of the uninvolved side as a relative norm against which the clinical signs of the involved side can be compared.

One requirement demanded of a diagnostic test is its ability to predict pathology. Evaluating the predictive validity of the ULNT is problematic because surgical verification that neural

tissue pathology is the cause of the symptoms usually cannot be obtained. A case study by Shacklock (1996) analysed the response to the ULNT3 (ulnar bias) in a case of a surgically proven ulnar neuropathy. A 25-year-old female receptionist presented with intermittent, deep and burning pain located in her left medial elbow. There was also a cold tingling feeling extending from the medial elbow to the little finger. Physical examination revealed no abnormality of nerve conduction (light touch, pin prick, thermal sensation, vibration), and the neurological examination, ultrasound scanning and cervical spine X-rays were negative. Passive and active elbow, shoulder and neck movements were pain free and full range. The ULNT3 with glenohumeral abduction and CCLF reproduced the patient's pain, and an increased pain reaction in comparison to the asymptomatic side was seen with the ULNT1 (Shacklock 1996). Subsequent surgery revealed a tight tendinous band crossing the ulnar nerve that caused increased pressure and mechanical irritation.

The negative results from the neurological examination and ultrasound scanning may have occurred for several reasons. During the neurological examination in a comfortable position the nerve may not have been sufficiently ischaemic to show conduction abnormalities, because symptoms were only provoked under working load when the nerve was in an elongated position (typing position) unmasking the neuropathy. Hence, the neurological examination and the ULNT3 may not have tested the same parameters (nerve conduction versus mechanosensitivity). The author further stated that the specificity of the test has been demonstrated by the capability of the ULNT3 to reproduce the patient's clinical symptoms while ULNT1 did not, although the ULNT1 has been shown to produce more than double the amount of strain on the medial cord than the ULNT3 (Kleinrensink *et al.* 2000). Despite the fact that it is apparently not the strain on the medial cord that produced the symptoms,

the positive response to the ULNT3 cannot not be disputed. However, these findings need to be verified in a larger patient population, as single case studies have no statistical power.

To sum up the main points, evidence is still lacking for the use of the ULNT2b in clinical practice because glenohumeral abduction as the relevant outcome measure has not been effectively measured at a clinical relevant end point (Yaxely & Jull 1993, Grant *et al.* 1995). On the other hand excellent intra- and inter-examiner reliability for the ULNT1 provide the evidence for the use of pain onset and submaximal pain as relevant parameters in clinical practice (Coppieters *et al.* 2002b). Furthermore, significant differences exist between the involved and uninvolved side for shoulder girdle elevation force (Coppieters *et al.* 2003a), for pain intensity, elbow ROM, and for distribution of sensory responses (Coppieters *et al.* 2003b).

These parameters monitored during diagnostic testing have the ability to differentiate between normality and abnormality. In this respect a positive test would imply an abnormal response to the test on the involved side in comparison to the uninvolved side, which implies a non-compliance of mechanosensitive neural tissue to movement. However, it should be stressed that a positive test cannot identify the site of pathology or predict a diagnosis. To show its predictive validity the ULNT would need to be compared to other diagnostic test that assess the mechanosensitivity of neural tissue, or to pre-diagnosed peripheral neurogenic disorders (Shacklock 1996).

The small sample size of most studies remains a problem in achieving sufficient statistical power (Hicks 1999). However, the strength of evidence has clearly increased due to the improved quality of recent studies, and gives hope for future high quality research in physiotherapy.

6. DISCUSSION

„The nervous system was perhaps the last of the mobile tissues whose mechanical abilities and associated sensitivities had not been considered, although the fact that the neural tissue must physically respond to movement is self evident".

Butler 1998

6.1. IMPLICATIONS FOR CLINICAL PRACTICE

When the ULNT was first conceptualised the emphasis was on the mechanical aspects of the test. The hypothesis then was that an ordered set of joint movements could be used to selectively increase tension within mechanosensitive neural tissue that would react to mechanical load with non-compliance to movement and with sensory responses (Elvey 1979b). Orthopaedic physiotherapists quickly welcomed this new approach and assessment strategy because, except for the straight leg raise, there was no neurodynamic test for the upper quarter. Although at that time no scientific research was available that investigated the reliability and validity of this diagnostic test, physiotherapists accepted it as a useful tool.

After 20 years of looking into the ULNT from a clinical and scientific perspective, there is now a growing body of research from countries like the UK, the USA, Australia, Belgium and The Netherlands. The current understanding of neurodynamic tests incorporates both the basic knowledge of pathomechanics and neurophysiology. Unfortunately, although the amount of clinically-based research is increasing, physiotherapists tend to chose their treatment techniques directly from what they were taught as students or in postgraduate training courses, but seldom base their treatment on clinical research (Turner & Whitfield 1997). This might be a reflection of the more passive approach of physiotherapists to deliver

certain treatment protocols rather than to make independent decisions. Moreover, physio-therapy is still not an academic profession in countries like Germany, therefore questioning traditional treatment strategies and doing research is not very common. Nevertheless, it is the responsibility of practicing clinicians to seek evidence supporting the efficacy of new and current treatment regimes (Matheson 2000).

As a movement-based profession, physiotherapy identifies structural dysfunctions through the examination of compliance or non-compliance to active or passive movements. Patients with upper quarter pain, in whom overt neurological deficits are not present and few medical investigative tests are definitive in the diagnosis of cervicobrachial pain syndromes, are frequently referred to physiotherapy. When assessing these patients physiotherapists usually focus on finding the musculoskeletal structure 'at fault'. The routine functional assessment for upper quarter pain syndromes includes a patient interview, active and passive tests of the cervical and thoracic spine and the glenohumeral joint, palpations of the soft tissues, bones and nerve trunks, muscle function and length tests, and neurological examination of reflexes and sensibility. However, new insight gained in basic neurophysiological science has changed the physiotherapist's understanding of neurogenic pain disorders. Since the deve-lopment of the NTPT, the physical assessment of the neural tissue has been integrated into the clinical assessment of CBP disorders.

Whether the NTPT is able to discriminate between different sources of pathology, i.e. nerve root or nerve trunk pathology, has recently been investigated. While there is no scientific evidence that it is possible to identify specific structural disorders, identifying the existence of an neurogenic disorder may be possible by integrating clues and information acquired from the preceding patient interview and from findings of the clinical assessment. To do so,

the knowledge of pain mechanisms and electrophysiological investigations need to be combined with the analysis of clinical features within a clinical reasoning framework, in which an initial working hypothesis is tested until sufficient information is obtained (Jones 1995). It is the clinician's responsibility to incorporate the entire range of relevant information to produce a working hypothesis. In this respect the issue of a test's validity should be broadened to the "analysis of the degree to which a meaningful interpretation can be inferred from the integrated findings from both the patient interview and the physical examination" (Coppieters & Butler 2001c, p.520), of which the ULNT is only a part. Therefore, the ULNT in isolation will not distinguish which pathology is the cause of a positive test (Shacklock 1996), but the solution lies in the analysis of the entire information. Furthermore, in clinical practice a single test is never used in isolation to make a diagnosis especially not in patients with complex neuromusculoskeletal disorders so often referred to physiotherapy.

Designing causal treatment strategies are difficult as many different tissues and anatomical structures might be involved in a pain syndrome (Coppieters *et al.* 2003b). Pain states are currently categorised by their duration (acute/chronic), causes (whiplash, repetitive strain injury), or the body parts involved (cervicobrachial pain, epicondylitis). This method of classifying pain, however, does not help predict outcome nor does it help identify physiological subcategories. Butler (2000) proposed that pain experiences should be categorised clinically into operant mechanisms on the basis of known pathophysiology, clinical patterns and logic. This approach corresponds with the recent developments in basic pain research for a mechanism-based classification (Woolf *et al.* 1998). Pain that manifests in distinct diseases may operate through common mechanisms. On the other hand, no pain mechanism is an inevitable consequence of a particular disease process (Woolf & Mannion 1999). It is

therefore important to differentiate between central and peripheral processes (Gifford & Butler 1997) when deciding on a therapeutic strategy.

6.2. LIMITATIONS

Many non-neural structures are stretched during neural tissue provocation testing and adding sensitising manoeuvres "does not help to localise the tissue at fault because other structures are moving with the nerves during these procedures" (Di Fabio 2001, p.224). However, most clinical tests in orthopaedic physiotherapy are not able to exclusively load specific structures, i.e. testing muscle length or joint mobility. "If the utility of a treatment modality would depend on its ability to independently mobilise one structure, very few interventions in orthopaedic physiotherapy would withstand" (Coppieters & Butler 2001c, p.521). Nonetheless, the adding of remote sensitising manoeuvres to the ULNT has shown to be a valuable tool in assessing neural tissue involvement to the patient's symptoms (Selvaratnam *et al.* 1994, v. der Heide *et al.* 2001, Coppieters *et al*. 2001b). For example, if a patient's local shoulder pain was reproduced or intensified by adding wrist extension, neurogenic involvement may be reasoned as part of the disorder, as the impact of the nervous system passes beyond the point to which musculoskeletal structures are loaded.

Adding wrist extension to the extended elbow and abducted shoulder has been shown to transmit tension along the median nerve at least up to the level of the axilla (Kleinrensink *et al.* 1995b). Components like CCLF and wrist extension are thought to elongate the nerve bedding, and, when combined, the available ROM of elbow extension is markedly reduced, with sensory responses elicited through the entire arm (Coppieters *et al*. 2001b, 2002b). No doubt non-neural structures will be stressed during neurodynamic testing, but adding

sensitising manoeuvres to a final test position has shown to increase symptoms that cannot be related to the provocation of non-neural structures. With the addition of individual test components an increased pain intensity has been reported, despite a decreased elbow range of motion and decreased loading of surrounding musculature and articular structures (Coppieters *et al.* 2002a). This limited range of elbow extension is thought to be, at least partly, a result of the limited elasticity of the neural tissue; this has also been attributed to a protective motor response of the antagonistic musculature, however.

It has to be emphasised that a limitation in elbow extension does not equal limited neural tissue elasticity. Therefore, treatment cannot be aimed at 'mobilising the neural tissue.' A rather limited ROM is a clinical sign indicating a probable neurogenic contribution to the presenting symptoms. In this respect a positive test would imply aiming the treatment at improving or restoring longitudinal gliding necessary for full limb motion, reduction of adhesions, and improvement of neurophysiological processes (McLellan & Swash 1976, Wilgis & Murphy 1986, Wright *et al.* 1996, Totten & Hunter 1991, Rozmaryn *et al.* 1998).

One of the limitations in analysing the research in the current thesis was the insufficient consensus as to what principle signs should be monitored during the NTPT. This made it difficult to compare studies and identify discrepancies. Some authors consider the repro-duction of symptoms, difference in ROM, and altered through range and end-feel (resistance) as the most important signs during NTPT. Others state that the differences in ROM are insufficient and that abnormal reactions may be shown in a reactive muscle activity of the upper trapezius, the latter being regarded as a nociceptively mediated flexor withdrawal reflex to protect the respective nerve from harmful elongation (Wright *et al.* 1994, Balster & Jull 1997, Coppieters *et al.* 2001a, v. d. Heide *et al.* 2002).

Also the use of different techniques to standardise testing methods poses a problem in comparing reliability measures from individual research. It is therefore necessary, for the researchers to conduct control intra- and inter-examiner reliability studies as long as testing procedures are not standardised. Only with standardised protocols will researchers be able to compare ULNT studies and interpret the results with clarity.

7. CONCLUSION

The seminal work of Elvey (1979b) initially describing the brachial plexus tension test led to an increased interest in neural tissue as a source of pathology and pain. Presently, the neuromeningeal approach for the examination and treatment of dysfunctions and pain is widely accepted and integrated into the clinical practice of physiotherapists. The underlying mechanisms of how these effects are exerted are poorly understood, however, and they remain inadequately researched. Early research focused on the conceptual idea of testing the mechanical properties of the neural tissue by testing extensibility. This testing paradigm gradually changed as there was an increasing recognition for the important changes within the central nervous system potentially responsible for many of the signs and symptoms in patients with CBP disorders seen in clinical practice.

In reviewing the literature concerning the evaluation of the neural tissue provocation test as a diagnostic tool the following can be stated: The NTPT has been developed to assess probable neurogenic involvement in CBP disorders, and can be used as a diagnostic tool, or as an outcome measure to evaluate the effect of treatment. Minor peripheral nerve injuries, as in nerve entrapment syndromes, are characterised by an increased sensitivity to mechanical stimulation. The NTPT is a sequence of movements designed to assess the compliance of sensitised neural tissue to mechanical load by elongating the length of the nerve bedding. Biomechanical studies have only found the median biased ULNT1 to be specific in transferring tension to the corresponding nerve and recommend its use as a valid test (Kleinrensink *et al.* 2000).

A positive test implies at least a partial reproduction of the patient's symptoms, in which symptoms increase and decrease with a varying amount of nerve provocation by changing

distant test components (sensitising manoeuvres) that have no direct structural link with the symptomatic area except via the nervous system. It is not possible, however, to differentiate between proximal or distal dysfunctions by a different sequencing of the test components, or to make any inference on the type of pathology. To judge when a response to the NTPT is abnormal the uninvolved arm can be used as a standard against which the clinical findings from the involved side can be compared. Reliability of the occurrence of pain onset and submaximal pain throughout elbow ROM for the ULNT1 for both intra- and inter-examiner reliability are highly reliable (Coppieters *et al.* 2002b) and can be used as a reliable outcome measure.

Many non-neural structures are stretched during neural tension testing and may account for the elicited responses. However, the relatively high incidence of paraesthesia in asymptomatic as well as symptomatic subjects suggests that at least some of the responses are neurogenic in origin (Coppieters *et al.* 2001b). The primary factor limiting movement is believed to be the nervous system that triggers a protective muscle reaction to prevent harmful elongation; however, electromyographical studies need to confirm this in larger patient populations.

Although most measurements acquired from studies have been performed in a laboratory setting, conflicting results have been reported with regard to the reliability of the NTPT. This is partly due to the lack of standardisations of the testing procedure that in turn leads to handling irregularities when performing this complex multiple joint test. The other limiting factor lies in the variation of the criteria for the end point at which the outcome measures were taken. Onset of resistance for example is not a reliable criteria, instead the onset of muscle activity has been found to be a more relevant sign (Hall *et al.* 1998). To date, only

the starting position for the ULNT1 with an initial shoulder girdle depression has been operationalised (Edgar *et al.* 1994). Gentle glenohumeral fixation throughout the ULNT1, and glenohumeral lateral rotation are furthermore crucial components of the test that warrant the mechanical loading of the brachial plexus and the median nerve and should not be omitted (Coppieters *et al.* 2003a).

Reliability in a clinical setting was not lower than in optimal laboratory conditions (Coppieters *et al.* 2002b), suggesting that, with adequate handling and sound operational definitions, physiotherapists are able to perform these tests in a standardised and reliable way. However, it should be appreciated that subtle adverse responses monitored during NTPT in a clinical setting without any measuring devices remains a "complex task that requires multifaceted skills" (Coppieters *et al.* 2003, p. 105).

7.1. RECOMMENDATIONS

> *"We believe that we cannot totally dismiss the great clinical importance…of neural provocation testing…simply because of a lack of scientific studies addressing this topic."*
>
> *(Coppieters & Butler 2002c, p.126)*

On the basis of the analysed research, the median biased ULNT1 can be a useful diagnostic test in disorders that lack clear physical signs of nerve injury or inflammation. Moreover, the test is extremely cost-effective when compared with nerve conduction studies, and should be performed as an integral part of the physiotherapists' assessment of upper quadrant disorders. Methodological deficiencies of many studies and the incomplete presentation and analysis of the data strongly suggests more rigorous studies with standardised procedures

and evaluation criteria. Future research should provide more insight in to the possible mechanisms that lead to limited range of motion. To establish the predictive validity of the NTPT, systematic research is necessary that shows how reliable the test can predict any neurogenic contribution in comparison with traditional diagnostic methods.

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APPENDIX A

Framework for critiquing quantitative research (C. Rees 1997)

FOCUS In broad terms what is the theme of the article? What are the key words you would file this under? Are the key words in the title a clue to the focus? How important is this clue for clinical practice?
BACKGROUND What argument or evidence does the researcher provide that suggests this topic is worthwhile exploring? Is there a critical review of previous literature on the subject? Are gaps in the literature or inadequacies with previous methods highlighted? Are local problems or changes that justify the study presented? Is there a trigger that answers the question 'why did they do it then?'. Is there a theoretical or conceptual framework?
TERMS OF REFERENCE What is the aim of the research? This will usually start with the word 'to', e.g. the aim of this research was to examine/ determine/ compare/ establish/ ect. If relevant, is there a hypothesis? If there is, what are the dependent and independent variables? Are there concept and operational definitions for the key concepts?
STUDY DESIGN What is the broad research approach? Is it experimental? Descriptive? Action research or audit? Is it quantitative or qualitative? Is the study design appropriate to the terms of reference?
DATA COLLECTION METHOD What tool of data collection has been used? Has a single method been used or triangulation? Has the author addressed the issues of reliability and validity? Has a pilot study been conducted? Have limitations to the tool been recognized by the author?
ETHICAL CONSIDERATIONS Were the issue of informed consent, and confidentiality addressed? Was any harm or discomfort to individuals balanced against any benefits? Was the study considered by a local research ethics committee?
SAMPLE Who or what makes up the sample? Are there clear inclusion and exclusion criteria? What method of sampling was used? Are those in the sample typical and representative, or are there any obvious elements of bias? On how many people/ things/ events are the results based?
DATA PRESENTATION In what form are the results presented: tables, graphs, bar-charts, pie-charts, raw figures, percentages? Does the author explain and comment on these? Has the author used correlation to establish whether certain variables are associated with each other? Have tests of significance been used to establish to what extent any differences between groups/ variables could have happened by chance? Can you make sense of the way the results have been presented, or could the author have provided more explanation?
MAIN FINDINGS Which are the most important results that relate to the terms of reference/ hypothesis/ research question? Think of this as putting the results in priority order; that is the most important result followed by the next most important result, ect. There may only be a small number of these).
CONCLUSION AND RECOMMENDATIONS Using the author's own words, what is the answer to the terms of reference/ research question? If relevant, was the hypothesis accepted or rejected? Are the conclusions based on and supported by the results? What recommendations are made for practice? Are these relevant specific and feasible?
READABILITY How readable is it? Is it written in a clear, interesting, or 'heavy style'? Does it assume a lot of technical knowledge about the subject and/or research procedures (i.e. is there much jargon)?
PRACTICE IMPLICATIONS How could the results be related to practice? What is the answer to the question 'so what?' Who might find it relevant and in what way? What questions does it raise for practice and further study?

APPENDIX B

TABLE I: NORMATIVE ULNT HUMAN CADAVER STUDIES						
AUTHOR/YEAR & STUDY DESIGN	TERMS OF REFERENCE	POPULATION (n =)	METHOD (intervention)	DATA COLLECTION	VALIDITY& RELIABILITY	RESULTS/ COMMENTS
Ginn 1988 (Proceedings)	To investigate tension changes in some muscles of the shoulder region and of the brachial plexus cords	1 unembalmed male cadaver (67 years)	ULNT1 with -elimination of G/H lateral rotation and shoulder girdle depression -full G/H horizontal extension -decrease of G/H abduction to 45°	▪ buckle force transducer on pectoralis major, biceps brachii, and brachial plexus cords below the clavicle	No reliability statistics were calculated	-G/H horizontal extension and lateral rotation decrease tension in the brachial plexus cords -lateral rotation produced dramatic decrease of tension in the biceps brachii. -shoulder girdle depression had no effect on tension of muscles or cords
Selvaratnam et al. 1989 (Proceedings) experimental design (parametric data)	To examine if the ULNT1 can differentially stimulate the brachial plexus with CCLF and ICLF	5 unembalmed cadavers (5-54 hours post mortem) Exclusion: subjects with rigor mortis	ULNT1: Shoulder depression, 110° G/H abduction and max. lateral rotation, forearm supination, elbow/wrist extension with ipsilateral and contralateral CLF	▪ cable ties on nerve roots C5-T1 (upper, middle, lower trunks) ▪ cable ties on median, ulnar, radial, musculocutaneus nerves Changes in spatial location of marked points were obtained by photographs by a computer digitising pad	No reliability statistics were calculated	-elbow extension with CCLF produced greater strain on C5/6 nerve root than did with ICLF. -variation between CCLF and ICLF for C7-T1 less apparent Hypothesis: BPTT with CCLF produces selective strain on nerve roots
Wilson et al. 1994 (peer-reviewed) experimental design (parametric data)	Pilot study to examine the strain at the subclavian artery during the ULNT1 in human cadavers	2 embalmed cadavers (in their seventh decade) Clavicle and strenocleidomastoid were removed	ULNT1: Shoulder depression, 110° G/H abduction and max. lateral rotation, forearm supination, elbow/wrist extension with ipsilateral and contralateral cervical flexion	▪ changes in spatial location of marked points were obtained by photographs using a compass divider	No reliability statistics were calculated	ULNT1 with CCLF is able to produce strain on segments of the subclavian artery, but strain in the lateral cord of the brachial plexus is greater Hypothesis: arterial baroreceptors might lead to nociception

AUTHOR/YEAR & STUDY DESIGN	TERMS OF REFERENCE	POPULATION (n =)	METHOD (intervention)	DATA COLLECTION	VALIDITY& RELIABILITY	RESULTS/ COMMENTS
Kleinrensink et al. 1995b (peer-reviewed) experimental design, same-subject (repeated-measure)	To investigate the distribution of tensile forces along the median nerve of 22 positions of the arm	5 embalmed cadavers Exclusion: subjects with rigor mortis	18 positions in normal ROM, ULNT1, ULNT2a, ULNT2b, modified ULNT (Kleinrensink et al. 1993) in neutral cervical position	▪ tensile forces measured with buckle force transducer (axilla, elbow, wrist)	Transducer tested to have a high test-retest reliability, and was calibrated after each measurement	-extended elbow (0°) and DF of hand cause increased tension in all three parts of the median nerve. -G/H position does not alter tension in distal median nerve
Lewis et al. 1998 (peer-reviewed) randomised, single-blinded, same-subject protocol	To investigate tension changes in the median nerve during ULNT1 in unembalmed human cadavers	2 female, 3 male (mean age 43,4 yrs) unembalmed cadavers (10 hours after death) Exclusion: subjects with rigor mortis	ULNT1: ipsilateral G/H depression, G/H abduction to 90°, forearm fully supinated, G/H external rotation, elbow extension, wrist/finger extension as final component Sensitising manoeuvres: head CLF, contralateral arm in ULNT, ipsilateral leg and bilateral SLR (70°)	▪ buckle force transducer attached in a 90° angle to the median nerve 2 cm distal to the axilla ▪ goniometer for 90° shoulder abduction	Reliability of the equipment was tested in a pilot study. Pearson correlation was r=0.9983 (highly reliable)	-elbow/wrist extension, and CCLF significantly increased median nerve tension during ULNT1. -shoulder depression and bilateral SLR did not significantly increase tension -G/H lateral rotation does not increase tension in the median nerve
Kleinrensink et al. 2000 (peer-reviewed) experimental design, same-subject (repeated-measure)	To analyse the quantitative validity of the three ULNTs	6 arms of embalmed human specimens Exclusion: subjects with rigor mortis	ULNT for the median, ulnar and radial nerve single and combined with cervical contralateral rotation and lateral bend	▪ buckle force transducers attached to the medial, lateral and posterior cords of the brachial plexus beneath the clavicle ▪ buckle force transducers attached to the proximal part of the median, ulnar and radial nerve	Transducer tested to have a high test-retest reliability, and was calibrated after each measurement	-exclusively the ULNT for the median nerve was found to be sensitive and specific -highest tension was found in the medial cord in all three tests and additional cervical lateral rotation further increased tension

TABLE II: NORMATIVE ULNT STUDIES

AUTHOR/YEAR & STUDY DESIGN	TERMS OF REFERENCE	POPULATION (n =)	METHOD (intervention)	DATA COLLECTION	VALIDITY& RELIABILITY	RESULTS/ COMMENTS
Yaxely and Jull 1991 (peer-reviewed) descriptive study same-subject design	To investigate the response in normal subjects to the modified upper limb tension test	50 normal subjects (25 f, 25 m) right hand dominant between 18 and 30 years with no history of neck and arm pain or trauma	Modified ULNT2b: shoulder girdle depression, elbow extension, G/H internal rotation, forearm pronation, wrist-finger flexion or extension followed lastly by G/H abduction Total of 4 test per subject alternately on each arm CCLF as sensitising manoeuvre	▪ glenohumeral abduction at R2: goniometer ▪ sensory response documented 3 times during each test procedure on a body chart	Inter-tester reliability for G/H abduction 95%, and 99% inter-tester repeatability based on ANOVAs	-gender and arm side did not influence outcome -mean range of glenohumeral abduction with wrist extension 43.11° (±4.55) and for wrist flexion 41.45° (±4.06) -strong painful stretch over radial aspect of forearm and elbow is normal response in 84% of the responses Hypothesis: normal sensory sensations derived principally from increased tension in the neural tissue
Edgar et al. 1994 (peer-reviewed) correlation study, (unmatched subject group)	To investigate the influence of the ULNT1 on the upper trapezius muscle length in normal subjects	60 healthy male volunteers (17-25 yrs) Group1: lesser neural tissue extensibility Group2: better neural tissue extensibility	Upper trapezius length in ULNT1 position with cervical spine in full CCLF Shoulder girdle depression was monitored by an inflatable pressure sensor and increased from 20 mmHg to 60 mmHg	▪ standard goniometer for elbow extension ▪ vernier callipers for trapezius length ▪ inflatable stabiliser (Chattanooga, Australia) to monitor shoulder depression	Good intra-examiner reliability for the ULNT and trape-zius measurement, good inter-examiner reliability for shoulder depression	-the group with decreased neural extensibility had significantly less measured length of the upper trapezius independent of flexed or extended elbow Hypothesis: extensibility of upper trapezius and the neural tissue are related
Zorn et al. 1995 (Proceedings) correlation study, (non-parametric data)	Comparison of the symptomatic consequences of three sequences of the ULNT in normal subjects	42 healthy males and 48 females (18-60 yrs)	Test A: proximal to distal Test B: middle sequence Test C: distal to proximal (no detailed description) CCLF added to each test	▪ location of sensation was analysed with Pairwise McNemar test (method of data collection not described)	Test-retest reliability was performed with 93% agreement for area of response *no statistics	-test A and B are similar in responses evoked (proximal to the elbow), and test C produced sensations distal to and including the elbow -added CCLF produced symptoms in the hand and fingers in all three tests

AUTHOR/YEAR & STUDY DESIGN	TERMS OF REFERENCE	POPULATION (n =)	METHOD (intervention)	DATA COLLECTION	VALIDITY& RELIABILITY	RESULTS/ COMMENTS
Balster & Jull 1997 (peer-reviewed) correlation study, (unmatched subject group)	To investigate the relationship between ULNT1 (median nerve bias) upper trapezius muscle activity and range of neural tissue extensibility in asymptomatic subjects	20 male volunteers (18-30 yrs) with no history of neck or upper limb musculoskeletal injury or pain 1. group: more extensible neural tissue 2. group: less extensible neural tissue	ULNT1: shoulder girdle depression, G/H 90°abduction, G/H external rotation, forearm supination, wrist/finger extension and elbow extension in that order on the dominant arm CCLF added at end of procedure	▪ standard goniometer for elbow extension at P1 ▪ surface EMG for upper trapezius ▪ verbal analogue scale 0-10 for pain ▪ Myrin goniometer for CCLF	Repeatability of EMG signals was calculated with ANOVA and showed no significant difference between trials (SEM 5.7)	-less extensible group exhibited significantly greater upper trapezius activity than the more extensible group. -range of CCLF was less in group 1. -no difference between pain levels in the 2 groups Hypothesis: nociceptive mediated flexor withdrawal reflex through stretch receptors in neural tissue *small sample size
Coppieters et al. 2001a (peer-reviewed) experimental, same-subject, repeated-measure design	To measure shoulder girdle elevation force during the ULNT1 in asymptomatic subjects	35 asymptomatic male subjects (mean 23,5 yrs), with no previous history of cervical or upper limb symptoms. Exclusion: limited or painful G/H ROM, and diseases relate to neuropathy	ULNT1 median bias: ULNT1+ Cx in neutral ULNT1+ wrist extension ULNT1+ CCLF ULNT1+ wrist extension + CCLF	▪ calibrated load cell ▪ two calibrated electrogoniometer for ROM of elbow and wrist extension measured at P2 (report of substantial discomfort) ▪ sensory response documented on body chart	Excellent ICC for intra-and inter-tester reliability for shoulder girdle elevation force. SEM 1.59-4.73 Newton	-gradual increase in shoulder girdle elevation force with the addition of test components is a normal sign during ULNT1 testing
Coppieters et al. 2001b (peer-reviewed) experimental, same-subject, repeated-measure design	To analyse the impact of different components of the ULNT1 (median bias) on the ROM of elbow and wrist in a normal population	35 asymptomatic male subjects (mean 23,5 yrs), with no previous history of cervical or upper limb symptoms. Exclusion: limited or painful G/H ROM, and diseases relate to neuropathy	Elbow extension 1.without ULNT1 components 2.with ULNT1+ wrist extension 3.with ULNT1+ CCLF 4.with ULNT1+ wrist extension + CCLF	▪ two calibrated electrogoniometer for elbow/wrist extension ▪ numeric pain intensity scale ▪ stretch and paraesthesia recorded on body chart ▪ criteria to end elbow extension: P2 (report of substantial discomfort)	Reliability coefficient for intra-and inter-tester reliability varied from 0.91° to 0.98° SEM varied from 1.7° to 2.5°	-when nerve bedding is elongated by adding test components individually or simultaneously ROM is markedly reduced and sensory responses elicited (80% reported paraesthesia in hand) Hypothesis: sensory responses are elicited through the neural tissue as a continuous structure

AUTHOR/YEAR &	TERMS OF REFERENCE	POPULATION	METHOD	DATA COLLECTION	VALIDITY&	RESULTS/
V. der Heide *et al.* 2001 (peer-reviewed) experimental, same-subject design (multiple variables)	To investigate the presence and onset of pain and muscle activity during ULNT1 (median nerve bias) in the normal population	10 male, 10 female asymptomatic subjects (mean age 43 yrs) with no previous history of cervical or upper limb symptoms. Exclusion: limited passive range of upper limb movement, and diseases relate to neuropathy	Stage I: ULNT1 with cervical spine in neutral, P1 and P2 as indicated by the subject during elbow extension Stage II: ULNT1 with CCLF prior to elbow extension, P1 and P2 as indicated by the subject during elbow extension	▪ calibrated electro-goniometer for elbow extension ▪ surface electromyography for trapezius muscle activity ▪ all measurements at stage I and II were taken three times on both arms	90° shoulder abduction, upper arm splint and pressure sensor tested for reliability in a study by Edgar *et al.* (1994) onset of P1 showed high reliability between trials	-trapezius activity is a normal response to ULNT1 and related to pain onset. -no significant difference in the range of elbow extension at P1 between both arms. -18 subjects responded with an increase in sensory symptoms and muscular response with the addition of CCLF (not indicated at what end point) *small sample size
Coppieters *et al.* 2002a (peer-reviewed) single-blinded, same-subject repeated-measure design	To quantify the impact of different shoulder positions on the ULNT1	25 male volunteers with no history of cervicobrachial pain (mean age 23.4 years) Exclusion: limited or painful G/H ROM, and diseases relate to neuropathy	Elbow extension was performed with ULNT1 in: 1)wrist and Cx in neutral 2)wrist extension 3)CCLF 4)wrist extension + CCLF 4 ULNT1 variations were performed in 3 G/H positions: 1)abduction and lateral rotation, shoulder girdle in neutral 2)abduction + shoulder girdle in neutral 3)only abduction	▪ calibrated electro-goniometer for elbow extension ▪ numeric pain intensity scale ▪ stretch and paraesthesia recorded on body chart ▪ criteria to end elbow extension: P2 (report of substantial discomfort)	Mean intra-tester reliability for elbow ROM among 12 testers was 0.96º (SD 1.8º), mean inter-tester reliability among 12 tests was 0.93º (SEM 2.3º)	-ULNT1 plus wrist extension and CCLF both decrease elbow ROM. -G/H lateral rotation decreases elbow ROM in all 4 variations of the ULNT1. -pain intensity and paraesthesia increased when more ULNT1 components were added

TABLE III: RESULTS OF NEURAL TISSUE PROVOCATION TESTING IN SUBJECTS WITH NEURAL TISSUE PATHOLOGY						
AUTHOR/YEAR & STUDY DESIGN	TERMS OF REFERENCE	POPULATION (n =)	METHOD (intervention)	DATA COLLECTION	VALIDITY& RELIABILITY	RESULTS/ COMMENTS
Yaxley & Jull 1993 (peer-reviewed) same-subject, repeated measure design	To investigate adverse tension in the neural system in 20 subjects suffering from lateral epicondylitis	20 volunteers (11f, 9 m) mean age: 43,5 with lateral elbow pain Exclusion: any history of fractures of the neck or upper limb, central or peripheral NS disease, limited G/H mobility	ULNT2b (radialis bias) with wrist and finger extension / flexion alternating between symptomatic and asymptomatic limb (in total 4 tests) At the limit of the test position CCLF was added	▪ standard goniometer for glenohumeral abduction ▪ sensory responses (depth, distribution, quality of pain) recorded on body chart during ULNT2b ▪ pre test recording for the felt muscle stretch of wrist + finger extensors placed in full stretch	Inter-examiner reliability and repeatability for G/H abduction at R2 was previously established by Yaxely & Jull (1991)	-significant difference in the mean range of G/H abduction (12.45º) between symptomatic and asymptomatic side with wrist /finger in flexion -CCLF increased symptoms of symptomatic arm in 75% (15 subjects) *small sample size
Selvaratnam *et al.* 1994 (peer-reviewed) experimental, repeated-measure design (unmatched subject groups)	To investigate the ability of the BPTT to identify referred pain from the cervical region in patients with unilateral shoulder and upper arm pain	Cardiac group: 25 patients after open heart surgery (mean 55,3 yrs) Sports group: 25 athletes (mean 26,2 yrs) with pain after throwing activity Exclusion for both groups: cervical pain, upper limb paraesthesia, motor deficits Asymptomatic control group: 16 subjects from throwing sports (mean 37,4 yrs) and 9 with heart surgery (mean 60,3 yrs)	BPTT: shoulder abduction, external G/H rotation, elbow extension, wrist extension with cervical spine in neutral, ipsilateral CLF, and CCLF Cardiac patients examined 9 wks after surgery Sports injury subjects 30 wks after injury	▪ goniometer for BPTT manoeuvres at P1 ▪ range of movement to sensory change (ROMSC) was recorded for each added test component using the apparatus protractor scales at P1 ▪ VAS for pain and stretch discomfort at P1	95% confidence interval for differences between ROMSC during ipsilateral CLF versus CCLF was estimated by a range of 1.96º SEM Test-retest reliability was 83%, with a standard error of 16.8º	-grand mean ROMSC during CCLF was less than during ICLF (p<0.001). -cardiac group demonstrated a larger difference in ROMSC between ICLF and CCLF than did sports injury or asymptomatic group. Hypothesis: the test is able to discriminate between referred and local sources of upper limb pain, and CCLF can be used as a sensitising manoeuvres

AUTHOR/YEAR & STUDY DESIGN	TERMS OF REFERENCE	POPULATION (n =)	METHOD (intervention)	DATA COLLECTION	VALIDITY& RELIABILITY	RESULTS/ COMMENTS
Grant et al. 1995 (peer-reviewed) single blinded study (unmatched subject groups)	To investigate the response on ULNT2b (radial bias) in screen based keyboard (SBK) workers	15 female volunteers (17-55yrs) working >4 hours at SBK; 80% experiencing some discomfort in neck and arm that did not interrupt daily activity 10 female controls with an occupation that did not involve SBK (mean 28 yrs) with no discomfort in the upper quadrant	ULNT2b with CCLF added as sensitising manoeuvre at the final ULNT position	▪ verbal sensory response ▪ goniometer for G/H abduction	High intra- and inter-examiner reliability for G/H abduction (p=0.004) *inappropriate statistics	-SKB group exhibited decreased range in G/H abduction (mean 12.05°) in comparison with normal subjects -additional CCLF increased symptoms to 70% in control and 73% in SKB group *small sample size, weak statistics
Sweeney & Harms 1996 (peer-reviewed) same-subject, repeated measure design	To investigate the relationship between the ULNT1 and mechanical allodynia following nerve injury of the hand	29 patients (22 m, 7 f) aged 21-77 years, with injury to their hand. Time since injury or surgery: 4 weeks to 41 years Exclusion: limited shoulder mobility, and infection to the wound site	1. ULNT1 (Butler 1994) with CCLF added to final test position tested on the uninvolved side first 2. ULNT1 as a home mobilisation exercise for 2 weeks	▪ elbow ROM with standard goniometer at R2 or to the point of symptom reproduction ▪ area of maximum sensory response recorded ▪ VAS for deep touch	Not stated	pre mobilisation: -in 22 patients the ULNT1 reproduced their symptoms. -22 patients reported a significant difference in elbow ROM between affected and unaffected side, and 7 subjects reported normal sensory response to the ULNT1 post mobilisation: -ULNT1 evoked symptoms in 10 patients -26 subjects achieved full elbow ROM on the affected side, and 19 subjects reported normal sensory response to the ULNT1

AUTHOR/YEAR & STUDY DESIGN	TERMS OF REFERENCE	POPULATION (n =)	METHOD (intervention)	DATA COLLECTION	VALIDITY& RELIABILITY	RESULTS/ COMMENTS
Hall *et al.* 1998 (peer-reviewed) correlation study (matched subject design)	To investigate the effects of ankle dorsiflexion and cervical spine flexion on compliance of neural tissue to the straight leg raise (SLR)	20 normal subjects (no history of back pain) 20 age and sex matched subjects with a validated lumbar or sacral radiculopathy (mean duration 1.6 years)	SLR with ankle dorsiflexion SLR with cervical spine flexion SLR in neutral ankle and cervical position	▪ EMG activity of hamstrings and gluteus ▪ SLR range at onset of resistance (R1) ▪ SLR range at first increase of momentum of stretched tissue (M*r*) ▪ SLR at onset of muscle activity (M1)	Accuracy and reliability of electrogoniometer and device to measure M*r* was assessed in a pilot study Intra-examiner reliability was established	-R1 has no discriminative power or validity to measure non-compliance of neural tissue. -ankle dorsiflexion and cervical flexion had insignificant effects on M*r*. -M*r* was significantly greater in the radiculopathy group in SLR range post M1
Coppieters *et al.* 2002b (peer-reviewed) correlation study (repeated measure design within and between sessions)	To analyse the stability and reliability of P1 and P2 throughout the ROM during the ULNT1 in a laboratory and clinical setting	Experiment I: 15 (11 f, 4 m) patients with unilateral neurogenic CBP (mean age 47.7 years) Experiment II: 10 (5 f, 5 m) asymptomatic subjects with no previous history of CBP (mean age 23.4 years) Experiment III: 12 patients with CBP (10 f, 2 m) 3 patients with bilateral symptoms (mean age 41.0 years) Exclusion: limited G/H mobility	Experiment I + III for only P2: 1.ULNT1 with wrist extension Experiment II for P1 and P2: 1.ULNT1+ Cx in neutral 2.ULNT1+ wrist extension 3.ULNT1+ CCLF 4.ULNT1+ wrist extension + CCLF were added before extending the elbow	▪ Elbow extension: calibrated electrogonio-meter ▪ P1: subject pressing hand held switch ▪ P2: verbal signal of subjects	Experiment II: Intra-tester reliability for P1 0.98 and P2 0.98. Inter-tester reliability for P1 0.86 and P2 0.89 (lab.setting) Experiment I: Intra-tester reliability for P1 0.98 (lab.setting) Experiment III: Intra-tester reliability for P2 0.98. Inter-tester reliability for P2 0.98. (clin.setting)	-the reliability of the occurrence of P1 and P2 throughout the range of elbow ROM during ULNT1 testing was good to excellent in asymptomatic subjects in a laboratory and clinical setting. -in the patient group reliability was excellent and comparable to the reliability obtained in asymptomatic subjects when tested within the same session. -reliability in a clinical setting was not lower than in optimal laboratory conditions.

| AUTHOR/YEAR & STUDY DESIGN	TERMS OF REFERENCE	POPULATION (n =)	METHOD (intervention)	DATA COLLECTION	VALIDITY& RELIABILITY	RESULTS/ COMMENTS
v. der Heide *et al.* 2002 (peer-reviewed)	To investigate the reaction to the ULNT1 in patients with cervical radiculopathy	3 patients (39-41) with C6/7 radiculopathy	In a ULNT1 position starting from 90º elbow flexion elbow was extended with Cx in neutral	▪ elbow extension: calibrated electrogonio-meter at P1 and P2 as indicated by the subjects ▪ surface electromyo-graphy for trapezius muscle at P1 and P2 ▪ sensory responses reported on body chart ▪ all measurements were taken three times	High reliability between trials for onset of pain established earlier (v. der Heide *et al.* 2001)	-ULNT1 reproduced the symptoms in 2 patients -trapezius activity was evident around the point of P1 in all 3 patients -in 2 patients P1 was significantly earlier in elbow ROM than on the asymptomatic side *small sample size no statistical power
Coppieters *et al.* 2003a (peer-reviewed) single-blinded RCT	To analyse whether aberrations in shoulder girdle elevation force during ULNT1 can be demonstrated in patients with CBP, and if they can be normalised following cervical mobilisation in a laboratory setting	Experimental group: 10 patients (8 f, 2 m, mean age: 49.1) Control group: 10 patients (8 f, 2 m, mean age: 48.1) with non-acute (2 weeks to 6 months) unilateral or bilateral CBP related to the median nerve, volunteered to participate	3 repetitions of the ULNT1 Experimental condition: cervical lateral glide technique at 1 or more segments (C5-T1) with the involved arm in a neural preloaded position Control condition: 5 minutes of pulsed ultrasound over the most painful area in an unloaded position (both interventions performed by the same therapist, blinded to the patient allocation)	▪ calibrated load cell for shoulder elevation force ▪ calibrated electrogonio-meters for ROM of elbow extension measured at P2 (report of substantial discomfort) ▪ numeric pain intensity scale	No systematic changes could be observed when the amount of end force of 3 consecutive repetitions of the UNT1 was analysed	-the amount of force at the end of the test on the unin-volved side was significantly larger than on the involved side. The sudden increase in shoulder girdle elevation force on the involved side occurred earlier in range than on the uninvolved side. The immediate effect of treatment: increase in shoulder girdle elevation force occurred later in the ROM; the amount of end force was significantly larger than before treat-ment; pain intensity was significantly decreased; available ROM was significantly increased -No treatment effects observed after control intervention

| AUTHOR/YEAR & STUDY DESIGN	TERMS OF REFERENCE	POPULATION (n =)	METHOD (intervention)	DATA COLLECTION	VALIDITY& RELIABILITY	RESULTS/ COMMENTS
Coppieters *et al.* 2003b (in press) single blinded RCT	To analyse the immediate treatment effect of cervical mobilisation and therapeutic ultrasound in patients with neurogenic CBP disorders	20 patients (volunteers) with subacute (2 weeks–6 months) uni- or bilateral cervico-brachial pain Inclusion criteria formulated by Elvey (1997)	3 repetitions of the ULNT1 Experimental condition: cervical lateral glide technique at 1 or more segments (C5-T1) with the involved arm in a neural preloaded position Control condition: 5 minutes of pulsed ultrasound over the most painful area in an unloaded position (both interventions performed by the same therapist, blinded to the patient allocation)	▪ elbow ROM with electro-goniometer at P2 ▪ body chart for symptom distribution during ULNT1 ▪ numeric pain intensity scale during ULNT1 ▪ load cell was used to standardise shoulder girdle depression	Good to excellent intra- and inter-tester reliability for P1 and P2 previously established (Coppieters *et al.* 2002b)	-for the cervical mobilisation group a significant reduction in pain intensity, an improve-ment in elbow ROM, and a 43.4% reduction in area of symptom provocation was noted. -in the ultrasound group no significant changes were found. -a significant difference in available elbow ROM, elicited pain intensity, and area of symptom distribution between the uninvolved and involved side was evident.

One way-ANOVA ICC index (Coppieters *et al.* 2002b)

- 'Poor' = ICC < 0.40
- 'Fair' = 0.04 ≤ ICC < 0.70
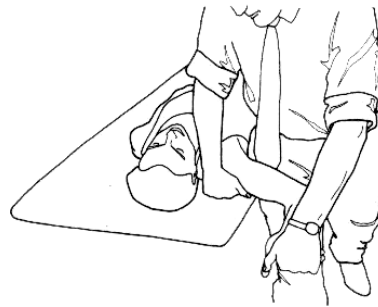- 'Good' = 0.70 ≤ ICC < 0.90
- 'Excellent' = ICC ≥ 0.90

APPENDIX C
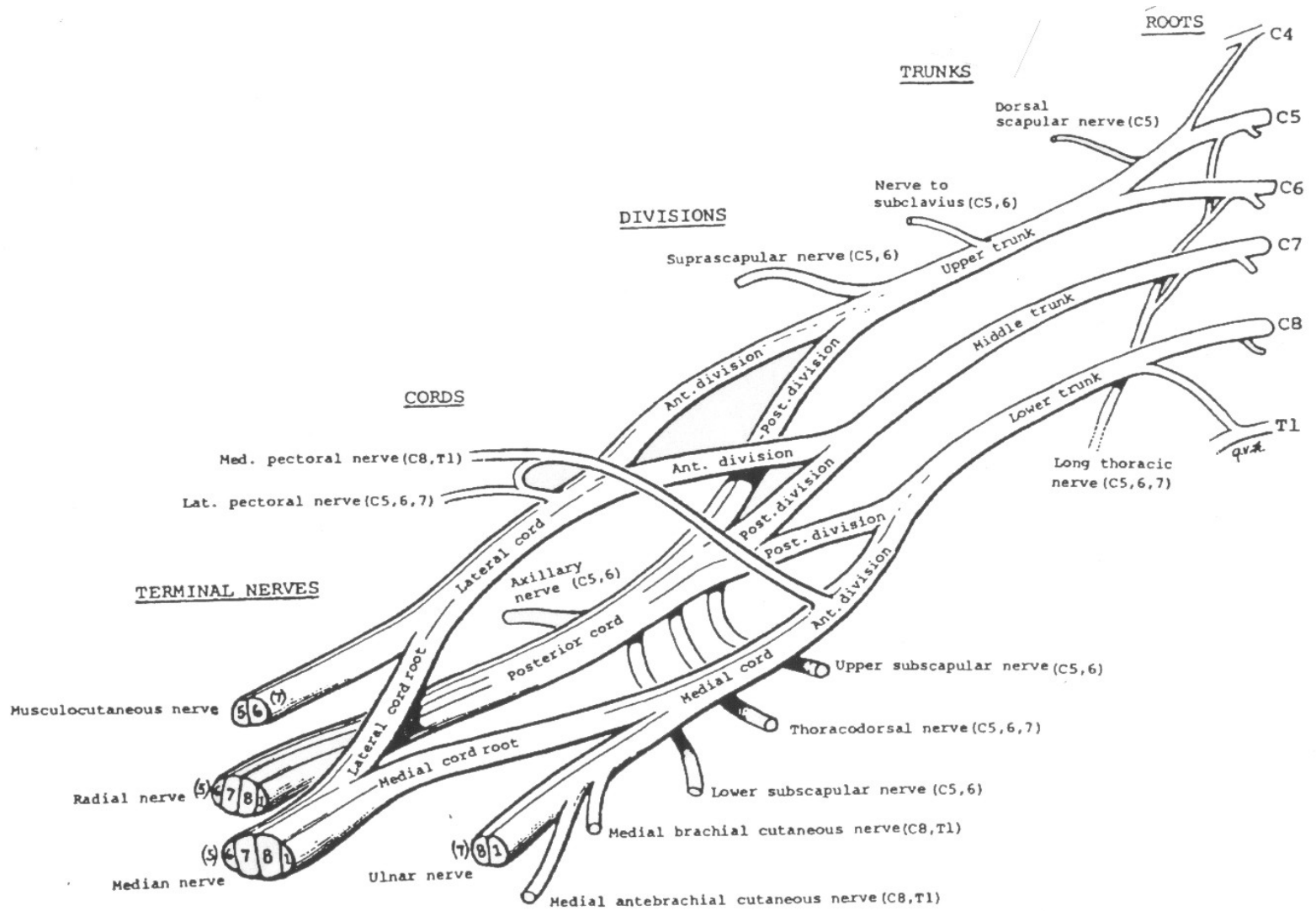
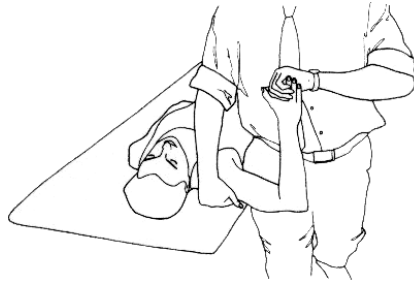**Figure 3:** Diagram of the brachial plexus. From Clemente CD (1995) Anatomy. An Atlas of the human body (4th Ed.)
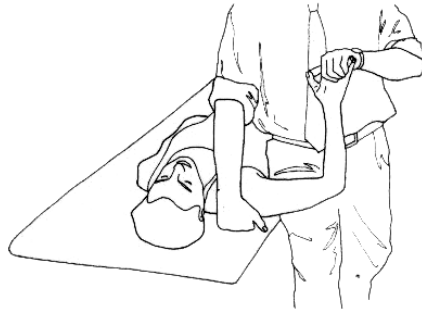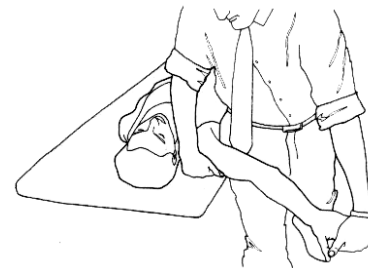
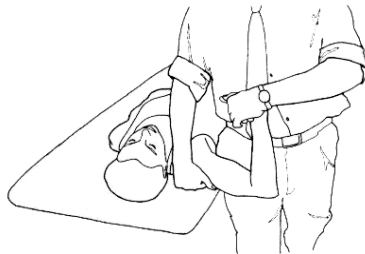ULNT1-A: Starting position

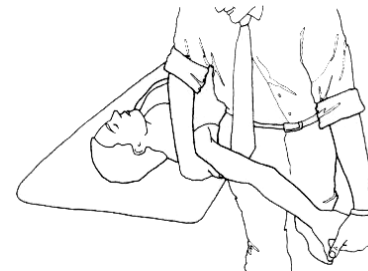ULNT1- E: Shoulder lateral rotation
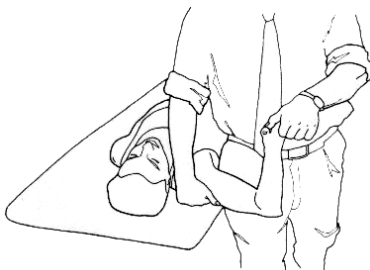

ULNT1-B: Shoulder abduction

ULNT1-F: Elbow extension

ULNT1-C: Wrist extension

ULNT1-G: Contralateral cervical lateral flexion

ULNT1-D: Forearm supination

ULNT1-H: Ipsilateral cervical lateral flexion

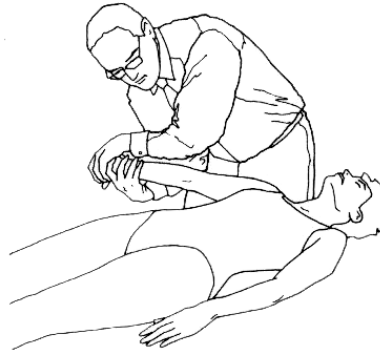*Figure 4:*   The ULNT1. From Butler DS (2000) The Sensitive Nervous System

ULNT2-A: Starting position

ULNT2-D: Whole arm internal rotation

ULNT2-B: Shoulder depression

ULNT2-E: Wrist flexion

ULNT2-C: Elbow extension

Figure 5: The ULNT2b. From Butler DS (2000) The Sensitive Nervous System

ULNT3-A: Starting position

ULNT3-E: Shoulder lateral rotation

ULNT3-B: Wrist extension

ULNT3-F: Shoulder girdle depression

ULNT3-C: Forearm pronation

ULNT3-G: Shoulder abduction

ULNT3-D: Elbow flexion

Figure 6:   The ULNT3. From Butler DS (2000) The Sensitive Nervous System