Influence of posture and motion on peripheral nerve tension

Anatomical, biomechanical and clinical aspects

Invloed van houding en beweging op perifere zenuwspanning
Anatomische, biomechanische en klinische aspecten

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Chapter 1

Introduction

Introduction

General introduction

Posture and motion, two essential features of locomotion, should not be seen as the endpoints of a linear scale. Motion can be seen as a dynamic pattern of subsequent postures whereas posture can be seen as a state of motion 'condensed' to a level where no activity of the body can be seen. Of course never a complete state of 'no activity' is reached, unless in the deceased.

In normal daily living, we just accept posture and motion. However, in case of pain or dysfunction we need a closer inspection. Mostly, injuries to the locomotor system are self-limiting since our body has an enormous capacity of self healing. Exclusively when the situation becomes serious, assistance of an expert is needed, e.g. a general practitioner, an orthopaedic surgeon or a physiotherapist. In treating dysfunction of the locomotor system two points of view have to be discussed: the clinical-practical and the scientific-theoretical. In both views Anatomy plays an important role, although the questions asked in these disciplines are different.

Firstly, there is the clinical-practical point of view. In treating patients with pain or dysfunction of the locomotor system, the attention is often focused on the localisation of the structure causing the pain. Since the question is asked *What* structure is injured , *topographical anatomy* plays an important role in the localisation of the injured structure. For structural damage and subsequent surgical treatment this line of thought is relevant but in many cases no structural pathology can be found while the patient is complaining of pain and dysfunction, sometimes during a very long period. In other cases structural damage is found and treated but complaints recur and become chronic. In these cases questions like: *Why* is this structure injured and *How* can reinjury be prevented must be answered. Then, a more scientific- theoretical approach is needed. *Functional anatomy* and the closely related biomechanics can play an important role in finding an answer to these questions. These different approaches can be illustrated by looking at the treatment of inversion trauma and chronic instability of the ankle caused by inversion trauma.

In the clinical-practical line of thought posture and motion are often 'translated' in two closely related aspects of posture and motion: joint *stability* and joint *mobility*.

Generally ankle stability is thought to be based on intact joint capsule and ligaments. After inversion trauma stretched or torn ligaments are supposed to be the cause of the instability. Treatments, varying from tape bandages to reconstructive surgery of ligaments, focus on the healing of anatomical structures. These treatments are usually effective, but in about 30% of the cases patients keep complaining about instability of the ankle, reinjury, fear of 'giving way', and fear of reinjury[1,2,3]. Since the collagen of the ligaments is structurally healed and restored to its original strength within 6-12 weeks, there must be 'something else' causing chronicity of the injury. For dynamic stabilisation of the ankle leg muscles are better suited for the task. However, muscles have to be timely recruited. This implicates the central and peripheral part of the nervous system. Here the concept of the *arthro kinetic reflexes*[4] , also called: *joint protecting reflexes*, offers a useful model. The receptors for these reflexes are contained in capsule, ligaments and muscles (proprioceptors). The afferent and efferent links of the reflexes are contained in the nerves of the ankle joint (the peroneal nerves). Central processing occurs in the spinal cord and higher parts of the nervous system (brainstem, cerebellum, cerebral cortex etc.)

The situation is rather complex since a trauma, like an inversion trauma, has widespread effects.

As shown by alterations in the Electromyogram (EMG) of the gluteal muscles[5] and alterations of motor nerve conduction velocity (mncv) of the contralateral deep peroneal nerve (this thesis, chapter 6) other joint regions of the ipsi- and contralateral extremity have to be taken into account. All structures involved can be considered as a specific cause of pain and dysfunction but more importantly is their interaction. Before the trauma, all structures can be assumed to function in a more or less harmonious and coordinated way, creating joint stability. At a certain moment this situation obviously changed, and as a result the ligaments were damaged: inversion trauma. In inversion trauma the ligaments are the 'victims' of abnormal loading, or of a system (temporarily) out of order. The ligaments should not be seen as the primary cause of the trauma but as the weakest link in a set of structures involved in a chain of events leading to an inversion trauma. The emphasis in the question: *Why* are the ligaments injured should be transposed to: Why are *the ligaments* injured. Each structure must be analysed on its specific qualitative and quantitative contribution, otherwise there is the risk of underestimating the role of one essential link. For instance when only the ligaments are considered, adequate treatment of an inversion trauma can, nevertheless, result in chronic complaints of pain and instability .
In this thesis the concept of the arthro kinetic reflex is taken as the basis for the analysis of the structures and events relevant in establishing *posture* and *joint stability.* The attention is focused on the peroneal nerves, their role in maintaining ankle joint stability and their possible role in the origin of chronic ankle instability. Several questions will be dealt with. Firstly, an attempt is made to answer the question whether the peripheral nerves around the ankle can be injured during inversion trauma and why. For this purpose, a model is formulated of the chain of events during inversion trauma. Secondly, we wanted to know whether a relationship exists between inversion trauma and functional deficit of the peroneal nerve. For this purpose a clinical trial was performed. Finally, we attempted to find an answer to the question whether a causal relationship exists between inversion trauma and functional deficit of the peroneal nerve. For this purpose we performed a prospective clinical trial in which pre trauma motor nerve conduction velocity (mncv) values could be compared with post trauma values.
Concerning the other aspects of the locomotor system, *motion and joint mobility* the peripheral nerves also play an essential role. Again the linear way of thinking in which mobility and stability are seen as endpoints on a scale should be subject of discussion. Mobility and stability can be seen as expressions of continuously changing situations: joints being stable until the next movement. However, there must be a certain degree of stability during joint motion. In the preceding paragraph structures of the (non linear) locomotor system were discussed in the context of stability, here the same elements return. In 1965 the American Academy of Orthopaedic Surgeons (AAOS), dealing with joint mobility, proposed a method to assess the Range of Motion (RoM) of all the joints of the extremities[6]. The maximal RoM around the relevant rotational axes is defined for each joint. Nowadays their booklet is used as a worldwide 'golden standard'. Although it is realised that in some cases decrease in RoM is caused by muscle spasm or contracture, usually changes in the ligaments and joint capsule are seen as the major cause of decrease in RoM. In most cases exclusively the affected joint is taken into account. Here the wrist joint is a good example.
The AAOS defines 70° dorsal flexion and 80° palmar flexion of the wrist as the maximal RoM around the transverse axis. The maximal RoM is defined by the joint architecture, capsule, ligaments and passing muscles. However, the wrist should not be seen as an isolated entity. For example, in

normal daily activities, wrist joint motion without finger/elbow or shoulder motion is seldom seen. All joints of the upper extremity function within a complex motion system with extensive coupling of movements. This coupling has several advantages. For instance, the RoM of the extremity as a whole is much larger than that of the individual joints and the extremity can also be used more efficiently. However, coupling is not without an obvious disadvantage: the motion of the extremity can be seriously hampered by malfunctioning of just one link of the chain. There is another, less obvious problem. From spinal cord to fingers three major peripheral nerves pass all joints of the extremity. Because of their vulnerability, passage of a joint is potentially hazardous, especially in case of joints with a large RoM. For several reasons this situation seldom leads to pain or dysfunction. Sunderland[7] mentions that, in the extremities, the nerves usually are situated at the flexor side of joints; stretching of the nerve due to flexion movements is not to be expected. Secondly, on a macroscopical level the nerve has an undulating course through the extremity. On a microscopical level this also holds for the individual axons. Joint motion leads to 'unfolding the harmonica' and hence to small or no tension in the nerve. Of course these 'defense' mechanisms have their limits. When this limit is reached the nerve becomes overstretched and pain and paraesthesia occur. Here nerve length defines the limits of motion and in the example mentioned above 70° dorsal flexion and 80° palmar flexion of the wrist can not be reached without overstretching the nerve.

Several studies deal with nerve lengthening (see Chapter 1.3). These studies involve basic research as well as clinical trials. For humans *in vivo* lengthening data are not available. In the rabbit tibial nerve conduction block occurred at an elongation of 12%. At 15% elongation the conduction block was irreversible[8]. Two questions arise:

1) Which tensile forces occur in peripheral nerves due to postures used in normal daily living and
2) Can certain positions of the extremities be used to selectively stretch the major nerves.

To answer the first question, in situ studies on embalmed and unembalmed human bodies were performed. By using 'buckle' force transducers, modified for use on peripheral nerves, we assessed the effect of 22 positions of the upper extremity, within the normal RoM on tension in three segments of the median nerve. To answer the second question, we analysed the validity of 6 tension tests for the upper extremity with two different approaches. In vitro studies on embalmed and unembalmed human bodies and an in vivo study on healthy volunteers.

The principle of nerve stretch as a result of certain movements has been found to be a useful tool in the diagnosis of nerve and nerve root lesions. Already in 1864, Lasègue proposed the Straight Leg Raising (SLR) test for the diagnosis of lumbar nerve root lesions[9]. Later, based on anatomical course and relation to joint axes of motion, tests for the median, ulnar and radial nerve have been suggested analogous to the SLR[10,11,12].

We first performed an *in vitro* study with 'buckle' force transducers on the median, ulnar and radial nerves. Exclusively the two test positions suggested for the median nerve turned out to be specific and sensitive. Secondly, we performed an *in vivo* study on healthy subjects to analyse functional aspects of the two specific and sensitive tests. For this purpose we assessed Somato Sensory Evoked Potential latencies.

Literature review

This review contains a selection of relevant studies dealing with the effect of tensile forces applied to the peripheral nerve. The literature presented in table 1 is restricted to 'modern' literature. Older literature (e.g. Lasègue,1864[9] and Bragard,1929[13]) is separately discussed in the chapters 4 and 5 dealing with nerve tension for diagnostic purposes.

Review of these studies is relevant since their outcomes are confusing and sometimes contradictory. The source of this confusion and contradiction can be found by looking at the specific questions which the authors tried to answer. Frequently it turned out to be necessary to analyse the publications to reconstruct the questions. Whether such questions are still relevant at this time will not be discussed here. Scientific studies can be seen as an attempt to find answers to specific questions within the context of a current paradigm.

Just after the second world war, research focused on the mechanical aspects and on morphological changes due to elongation of nerve tissue. Relevant questions were: What is the maximal elongation which can be obtained by applying tensile loads on nerve tissue, What is the elastic limit of nerve tissue and What happens beyond this limit.

Generally the aim of these studies was to know over which distance a nerve could be sutured and again stretched without serious damage to the proximal and distal stump. The first study referred to in table 1 starts with : 'Reports of nerve injuries resulting from war wounds frequently include evidence of damage to peripheral nerves without loss of anatomic continuity' [14]. Attempts to answer the question: When (at what stresses or strains) is the nerve continuity actually lost? lead to so called 'failure tests'. Although in a limited number of studies clinical aspects of nerve function are mentioned, most early studies focus on morphological changes due to tensile stress.

The stress-strain curve of nerve tissue became a central tool in analysing the effects of tensile forces[15,16,17].

Since 1970, studies concentrated on questions like What happens to the nerve conduction due to stretch. At first, alterations in the neural micro circulation were used as a parameter[18]. Subsequently, nerve conduction parameters (Compound nerve action potential (cnap), compound muscle action potential (cmap) and nerve conduction velocity (ncv) were used to evaluate the effects of elongation[19,8,20]. The reason to perform these studies remained the same: optimising the result of surgery.

The chapters of this thesis focuss on three other questions concerning nerve stretch:

1) What is the tensile force distribution in the median nerve as a result of positions of the upper extremity within the normal RoM?
2) Can certain positions of the upper extremity be used as valid nerve stretch tests in the diagnosis of nerve and plexus lesions?
3) Can certain extreme movements (inversion trauma) cause a function deficit in peripheral nerves (peroneal nerves).

Re 1) To our knowledge, no studies have been performed to analyse tensile force distribution in peripheral nerves.

Re 2) Apart from older papers of Lasègue and Bragard, this subject has not been thoroughly studied and mostly the results have not been published in peer reviewed and/or accessible journals. Although tension tests of the nerves of the upper extremity based on their anatomical course are described in the respected textbook of 'von Lanz und Wachsmuth' as early as 1957[10], fundamental data on tensile force distribution are lacking; only empirical data have been presented[11,12].

Re 3) Several studies deal with peroneal nerve palsy related to inversion trauma[21-25]. However, the reports were on a case presentation basis and except for one single case study[24] no detailed nerve conduction studies were performed.

Outline of the thesis

Chapter 2 deals with the rationale of the use of embalmed human bodies for comparative tensile force studies on peripheral nerve tension.
In *Chapter 3* the results are presented of an anatomical - biomechanical study on embalmed human bodies. In this study a detailed description is given of the method of *in vitro* tensile force measurements on peripheral nerves. To assess tensile forces in the median nerve, 'buckle' force transducers modified for use on peripheral nerves were used. Tensile forces in the median nerve due to positions of the upper extremity within the normal RoM are analysed.
In *Chapter 4* six nerve tension tests for diagnosing nerve(root) lesions are analysed on their validity. Tensile force distribution in the median, ulnar and radial nerve as well as in the medial, lateral and posterior cord was taken as an outcome variable to judge the specificity and selectivity of these tests.
Chapter 5 concerns part two of the study 'Upper limb tension tests in the diagnosis of nerve and plexus lesions' (chapter 4 forms part one). In part one of the study it was shown that only the median nerve Upper limb tension tests (ULTT and ULTT +) were valid on the basis of tensile force distribution. Logically, in the second part of the study presented here, only the functional aspects of the median nerve tests were analysed. Latencies of Somato Sensory Evoked Potentials were taken as an outcome variable to observe whether changes in nerve function occur due to the median nerve ULTT and ULTT+.
In *Chapter 6* the effects of tensile forces on peripheral nerves beyond the limits of physiology is discussed. The results of a clinical trial are presented showing the relation between inversion trauma and function deficit of the peroneal nerves. A model is presented based on the course of the peroneal nerves with respect to the transverse and sagittal axes of the joint(s) and mechanical aspects of inversion trauma. From the model and the results of the clinical trial we hypothesised a traction lesion of the peroneal nerves due to inversion trauma. However, because pre injury mncv values were not available it was not possible to draw conclusions about a *causal* relationship between inversion trauma and function deficit of the peroneal nerve.
Chapter 7 contains the results of a prospective clinical trial on the surmised causal relationship between inversion trauma and function deficit of the peroneal nerve.
In *Chapter 8* the results of the anatomical-biomechanical studies and clinical trials which form the basis of this thesis are separately discussed. General conclusions are placed within the context of two main aspects of the human locomotor system: joint mobility and joint stability.

Table 1. Review of the literature dealing with the effect of tensile forces applied to the peripheral nerve

Author(s) Year	Method	Substrate Human/Animal	Nerve	In Vivo/Vitro	Embalmed Yes/No	Results
Denny Brown/ Doherty; 1945 (14)	The nerve was fixed between fingers. A segment of known length was stretched and the lengthening was measured on known intervals (6-31mm). Histologic assessment was performed.	10 Cats	Segment of the Peroneal nerve	In Vivo		Little force is necessary to create a 100% lengthening. In unifascicular nerves, 100% lengthening is possible without recognisable damage.
Liu, Benda and Lewey;1948 (15)	Nerve specimens of 4 cm length were stretched in fixed steps of 0.07 mm. Corresponding tension was measured on a tensiometer. Histologic assessment was performed.	Human	Segments of: Sciatic (3) Ulnar (4) Tibial (7) Peroneal (8)	In Vitro	No; 12 hours after death	Stress/Strain curves were drawn. Point where the line becomes discontinuous indicates the point of the limit of elasticity. Critical elongation is at lengthening of 4.2%: minimal ruptures in several sites of the nerve fibers. 6%: Ruptures of fasciculi. 10%: ruptures of myelinated fibers. 20%: transverse rupture of nerve fibers and endoneurium. 22%: longitudinal rupture of perineurium; herniation of fibers: pseudoneuroma
Hoen, Bracket; 1956 (26)	Stretching the sciatic nerve to bridge a gap in the nerve. Comparison of 3 methods. a) across joint, stretch of intact nerve b) idem, freshly cut nerve c) idem, after neuroma formation	Dogs, 15-20 lbs.	a) en c) intact b) cut Sciatic nerve	In Vivo		Method c) is preferred method. 25-50% lengthening leads to rupture.
Sunderland, Bradley; 1961 (16)	With a stretching apparatus nerve specimens of 17-167 mm were stretched with a velocity of 76 mm/min.	Human, 30-50 years	Segments of: Median (24) Ulnar(24) Tibial (13) Peroneal (15)	In Vitro	No; 12 hours after death	Linear relationship between force and length. Elastic limit strongly variable (8- 20% lengthening). Mechanical failure at 30%. Slowly stretching the nerve leads to considerable lengthening without apparent damage.

Haftek; 1970 (17)	a) Tibial nerves stretched with constant velocity of 5 m/min. (Instron machine) b) Abrupt, forceful stretch by weights of 300 (N=15), 1814 (N=6) and 4536 g (N=2) falling from a height of 20 m. Light and electron microscopic assessment.	Albino Rabbit	Segments of Tibial nerve	In Vitro		Elastic limit: mean 69.3 (41.7-90.5); epineurium ruptured. First part of curve: no relationship between Stress and Strain in second part: linear relationship. After reaching the elastic limit: no relation between stress/strain. Viscous behaviour. Abruptly passing elastic limit causes extensive tissue damage
Lundborg, Rydevik; 1973 (18)	Tibial nerve is severed 2 cm above the ankle. The nerve is stretched in the distal direction with a stretching apparatus.	30 Rabbits	Segments of Tibial nerve	in Vivo		At an average lengthening of 8% first signs of decreased blood flow. At an average lengthening of 15% a complete stand still in all neural vessels was observed.
Orf; 1978 (27)	Sciatic nerve is dissected between semi membranosus and semitendinosus. By looping the nerve around tubes with different diameters, the nerves were stretched 2,4,6,8,and 10% for the duration of 1day-35 weeks.	56 Rabbits	Segment of the sciatic nerve	in Vivo		Parenchymal changes in proportion to the degree of traction. Ascent of injury in the proximal portion of the nerve(degeneration spreads more proximal as tractive load increases) Impairment of neural blood flow. Retrograde cell reactions in the anterior columns of the spinal cord and ganglia. Muscular changes: first week atrophy from 2nd week on, compensatory hypertrophy.
Shaw Wilgis, Murphy; 1986 (28)	The peripheral nerve system of the arm was dissected in 15 unembalmed human bodies. At shoulder, elbow wrist, MCP joint,PIP joint and DIP joint level displacement was measured	15 unembalmed human bodies	Median, Ulnar and Radial nerve and brachial plexus.	in Vitro	No	Greatest excursion of the peripheral nerves occurred at the carpal tunnel level. Median nerve: mean:15.5 mm; Ulnar nerve: mean: 14.8 mm Least excursion in the common digital nerves prox. to DIP:ë1mm.
Klein et al; 1987 (29)	Strain in the plexus brachialis due to several arm positions was measured by means of strain gauges. Purpose was to optimize the position for arthroscopy.	5 unembalmed human bodies	Upper trunk, Lateral cord, Median nerve and Radial nerve	in Vitro	No	Minimum overall strain was in a position at 90% of flexion and 0% of abduction. The optimal position for shoulder arthroscopy (minimal strain, maximal visibility) is 45% forward flexion and 90% abduction.

Milner; 1989 (19)	The effect of slow (over 15 days) and fast (over 3 days) tissue expansion by means of a custom made silastic expander was studied.	214 female rats	Sciatic nerve	in Vivo	Nerve length increases directly proportional to the degree of expansion. Mechanical disruption did not occur even in up to 80% increase from resting length. Nerve conduction velocity (ncv) reduces to 62% of control values in nerves elongated by 10-40% and up to 45% in nerves elongated by 50%. Reduction in blood flow may account for this ncv reduction.
Rydevik et al.; 1990 (30)	At a rate of 1cm/min nerves were stretched to failure with an Instron testing machine. Histological examination was performed on stretch ed and normal control nerves.	9 rabbits	Tibial nerve	in Vivo	Rabbit Tibial nerve exhibit a non-linear stress-strain relationship. After 20% strain the curve becomes linear up to failure. The ultimate strain:38.5Â2%; the ultimate strength:11.7Â0.7 MPa. At Failure load dropped suddenly but nerve specimens remained grossly intact.
Kwan et al.; 1992 (8)	Elongation of nerve specimens of 5 cm length was analysed by means of a Video dimensional analyzer (VDA). In situ strain was calculated and stress-strain curves were drawn. In a second series of animal studies changes in nerve conduction properties (compound nerve action potential: cnap was studied), related to in situ strain was assessed.	30 rabbits	Tibial nerve	in Vitro and in Vivo	Initially the nerves were easily stretched. With further elongation, slope of the stress-strain (sts-stn) curve increased. Peripheral nerve was found to exhibit a non linear sts-stn behaviour. Sts-stn curve followed the load-elongation curve. Gross examination could show no lesions while underlying intraneural organisation could be significantly disturbed; perineurium was found to be the principal load carrying tissue component. At 6% strain cnap decreased to about 60% of baseline values after 1 hour of stretch. At 12% strain cnap decreased to complete block. After 45 min. of a load of 1MPa cnap was zero. After removal of the stress, complete recovery was attained in 15 min. After 30 min of 1,75 load cnap was zero and after stress removal, only 20% of baseline values was attained.
Van der Wey et al.; 1994 (20)	The Sciatic nerve was elongated by laser. Doppler flowmetry controlled expansion.During elongation of 2 and 6 weeks, nerve con duction parameters (cnap, cmap and ncv) were measured and analysed.	50 rabbits	Sciatic nerve	in Vivo	It showed to be possible to elongate rabbit sciatic nerve up to 40%. Gait and toe spread reflex rapidly recovered at the end of the third week.Ncv decreased in a linear relation to elongation and did not recover. Cnap amplitude was severely reduced. Under the conditions of this experiment no conduction block occurred.

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Chapter 2

Peripheral nerve tension due to joint motion

A comparision between embalmed and unembalmed human bodies

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Summary

Various joint positions of the upper extremity were used to study the tensile forces on the median nerve. To analyse the effect of embalmment, tensile forces were measured *in situ* in unembalmed and embalmed human bodies. A positive correlation was found between tensile force data from unembalmed and embalmed nerves. This finding justifies, for comparative studies, the use of embalmed human bodies, although the absolute tensile forces are higher.

Relevance

In daily activities or during certain operations, positions of the upper extremity vary, hence creating tensile forces of different magnitude on peripheral nerves. Tensile forces are also applied in the diagnosis of nerve (root) lesions of the upper extremity. To analyse these tensile forces, *in situ* experiments on unembalmed human bodies, though problematic, are supposed to be the most realistic approach. In this study, it has been shown that, in comparative studies on peripheral nerve tension, data obtained from embalmed human bodies can be used. Key words: Peripheral nerve, anatomy, mechanical stress, (normal) articular range of motion Peripheral nerve tension due to joint motion

Introduction

In the field of human anatomy and biomechanics in vivo experiments are often impossible. The use of *in vivo* animal experiments can be considered but extrapolation of conclusions to the human *in vivo* situation can be disputed. *In vitro* experiments on unembalmed human bodies *in situ* are preferred. Unfortunately, the use of unembalmed human specimens has several restrictions (pressure of time, possibility of infectious disease etc.). As a consequence embalmed human bodies are frequently used. However, little is known of the effects of embalment on the peripheral nerve itself and the tissues surrounding the nerve. Several *in situ* studies have been published on stress-strain relations, the gliding mechanism and ultimate stress leading to failure of human nerves. These studies were performed on either embalmed[1,2] or unembalmed[3,4] human bodies. Recently the effect of joint positions within the normal range of motion (RoM) on tensile forces on the median nerve of embalmed human bodies was described[5]. Since in none of these studies tensile forces in embalmed and in unembalmed specimens have been compared, in the present study such a comparison is made for tensile forces on the median nerve caused by extremity positions within the normal RoM. In the diagnosis of nerve(root) lesions of the upper extremity, nerve tension tests analogue to Lasègue's straight leg raising test[6] have been proposed[7,8,9]. In the analysis of the value of these tests, quantitative data on peripheral nerve tension are needed.

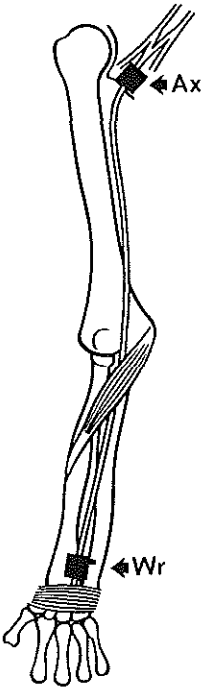

Figure 1
Position of the buckle force transducers.
Ax = two cm distal to the bifurcation (lateral and medial cord forming the median nerve), **Wr** = two cm proximal to the styloid process of the radius

Methods

Four human bodies (three females, ages: sixty-four, eighty-three and eighty-nine and one male, age: sixty-five) were embalmed by vascular perfusion between fourty-eight and sixty hours after death with a medium containing: 50g phenol 99%, 20g MgSO4, 20g Na SO4, 10g NaCl, 60ml formaldehyde 37%, 60ml glycerine, H_2O ad 1000ml. The bodies were kept in containers filled with phenol (30g/l) for six weeks. Subsequently, they were stored in phenoxy-ethanol (10ml/l) at a temperature of four degrees Celcius for three months. Measurements on eight arms were performed. Tensile force on the median nerve was measured at two sites: 1) at the axilla, about two cm distal to the bifurcation where te median nerve is formed out of the medi al and lateral cord (Ax) and 2) at the wrist, two cm proximal of the styloid process of the radius (Wr). (see fig. 1)

Two bodies (one male, age: eighty-one and one female, age: eighty-six) were stored for fourty-eight hours after death by a temperature of four degrees Celcius. Between forty-eight and fifty-six hours after death, tension measurements were performed on three arms. The measurements were performed under standardized conditions including room temperature and air humidity.

Table 1a. Eighteen standard elbow / forearm and hand positions in the normal range of motion

Elbow / forearm*		Hand*	
120° (Flexion) (I)	Maximal supination (1)	80° Palmarflexion (a)	(1)
		Neutral (0°) (b)	(2)
		70° Dorsal flexion (c)	(3)
	Maximal pronation (2)	80° Palmar flexion (a)	(4)
		Neutral (0°) (b)	(5)
		70° Dorsal flexion (c)	(6)
90° (Flexion) (II)	Maximal supination (1)	80° Palmar flexion (a)	(7)
		Neutral (0°) (b)	(8)
		70° Dorsal flexion (c)	(9)
	Maximal pronation (2)	80° Palmar flexion (a)	(10)
		Neutral (0°) (b)	(11)
		70° Dorsal flexion (c)	(12)
0° (Extension) (III)	Maximal supination (1)	80° Palmar flexion (a)	(13)
		Neutral (0°) (b)	(14)
		70° Dorsal flexion (c)	(15)
	Maximal pronation (2)	80° Palmar flexion (a)	(16)
		Neutral (0°) (b)	(17)
		70° Dorsal flexion (c)	(18)

Cervical spine: neutral (0° lateroflexion and 0° rotation); shoulder: 0° retraction and 90° abduction
The numbers 1-18 in parenthesis correspond with the position numbers of figures 2 and 3
* Definition of joint positions according to the standards of the American Academy of Orthopaedic Surgeons

Test positions

All tests were performed with the bodies in the supine position with neutral position of the cervical spine. The eighteen arm positions in the normal RoM used in this study are given in table 1a. Table 1b shows the four arm positions used for the median, ulnar and radial nerve upper limb tension test (ULTT) and a modified median nerve ULTT. In this study a modification of the ULTT was performed also, while in clinical practice there is no uniform answer to the question whether to perform the originally proposed median nerve ULTT[7] or the modified ULTT with maximal ranges of motion in all joints. The numbers 1-22 in parenthesis correspond with the position numbers of figures 2 and 3. On the unembalmed bodies the tests were performed once. In the embalmed bodies, all twenty-two joint positions were studied three times: as test and retest and after application of a tight band around the arm. To make a relevant comparison, only the results of the first measurements were taken for analysis.

Table 1b. Upper limb tension tests (ULTT) for median, ulnar and radial nerves
(for the modified median nerve ULTT maximal range of motion in the shoulder joint was used)

	Shoulder	Elbow / forearm	Hand
ULTT Median nerve (19)	110% Abduction - 10% Retroflexion - 60% Exorotation	- 0% (Extension) - Max. Supination	70% Dorsalflexion
Modified ULTT Median nerve (20)	- Max. Abduction - Max. Retroflexion - Max. Exorotation	- 0% (Extension) - Max. Supination	70% Dorsalflexion
ULTT Ulnar nerve (21)	- Max. Abduction - Max. Exorotation	- 120% (Flexion) - Max. Supination	70% Dorsalflexion
ULTT Radial nerve (22)	- Max. Abduction - Max. Retroflexion - Max. Endorotation	- 0% (Extension) - Max. Pronation	70% Palmarflexion

The numbers 19-22 in parenthesis correspond with the position numbers of Figures 2 and 3

The buckle force transducer used in this study was the tansducer described by Salmons[10] and modified according to Peters[11]. It was originally designed for mea suring stress and strain fields in ligaments. For the use on nerve tissue a further modification was needed to lock the nerve in the transducer without initial damage. For a more detailed description of the tension measurements, see Kleinrensink et al[5].

Statistical analysis
The data are summarised per position as medians and ranges (table 2). In order to quantify the similarity between data obtained from unembalmed and embalmed specimens the Spearman's Rankcorrelation test was applied to these medians.

Results

Both at the axilla and at the wrist, a great similarity exists in the data obtained from unembalmed and embalmed human bodies (figures 2 and 3). A strong positive correlation is found between tensile forces in the median nerves from unembalmed and embalmed bodies (table 2).

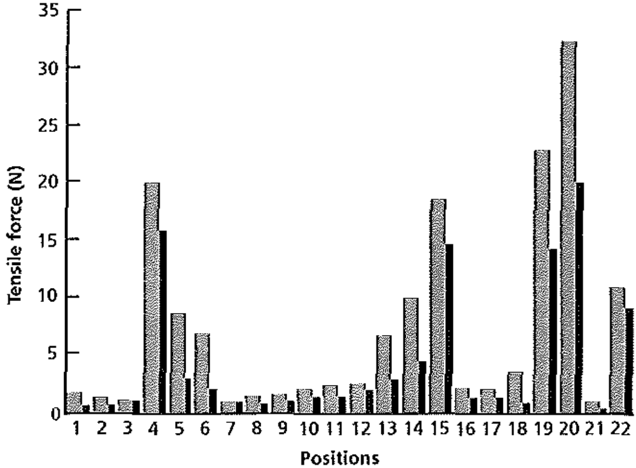

Figure 2 Medians of the tensile forces on the Median nerve measured at the Axilla. A comparison between unembalmed and embalmed Median nerves *in situ*. The position numbers correspond with position numbers in parenthesis in the tables 1a, 1b and 2. ▨ Embalmed; ▮ unembalmed

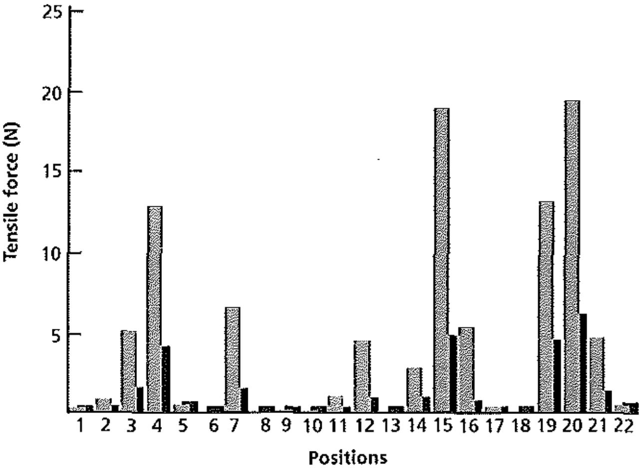

Figure 3 Medians of the tensile forces on the Median nerve measured at the Wrist. A comparison between unembalmed and embalmed Median nerves *in situ*. The position numbers correspond with position numbers in parenthesis in the tables 1a, 1b and 2. ▨ Embalmed; ▮ unembalmed

Table 2. Tension on two segments of the median nerve due to (test) positions within the normal range of motion. A comparison between embalmed and unembalmed human median nerves *In Situ*

	Axilla		Wrist	
Position[1]	Embalmed (n=8)	Unembalmed (n=3)	Embalmed (n=8)	Unembalmed (n=3)
I.1.a (1)	1.67 (0.20-3.04)[2]	0.39 (0.20-0.98)	0.29 (0.00-9.41)	0.38 (0.34-0.39)
I.1.b (2)	1.37 (0.20-3.43)	0.39 (0.20-0.98)	0.78 (0.00-6.37)	0.39 (0.38-0.39)
I.1.c (3)	0.98 (0.20-3.43)	0.98 (0.39-5.88)	5.10 (0.00-18.22)	1.57 (1.52-2.45)
I.2.a (4)	19.89 (6.57-30.28)	15.68 (11.66-19.50)	12.84 (5.78-32.93)	4.12 (2.55-5.59)
I.2.b (5)	8.62 (1.47-15.68)	2.84 (2.45-3.33)	0.49 (0.00-4.41)	0.69 (0.59-0.78)
I.2.c (6)	6.76 (0.20-13.72)	1.96 (0.39-2.16)	0.00 (0.00-0.39)	0.39 (0.34-0.49)
II.1.a (7)	0.98 (0.20-3.43)	0.88 (0.39-0.98)	6.51 (0.00-14.50)	1.47 (1.27-1.76)
II.1.b (8)	1.57 (0.10-3.43)	0.69 (0.39-0.98)	0.00 (0.00-1.47)	0.38 (0.34-0.39)
II.1.c (9)	1.67 (0.10-5.19)	0.98 (0.39-1.27)	0.05 (0.00-41.75)	0.38 (0.34-0.59)
II.2.a (10)	2.16 (0.10-5.68)	1.37 (0.59-1.96)	0.10 (0.00-10.49)	0.34 (0.29-0.39)
II.2.b (11)	2.45 (0.10-5.19)	1.37 (0.88-1.96)	0.98 (0.00-0.39)	0.34 (0.29-3.92)
II.2.c (12)	2.65 (0.10-6.08)	1.96 (1.37-2.84)	4.51 (0.88-19.11)	0.88 (0.69-0.98)
III.1.a (13)	6.76 (2.25-11.27)	2.84 (2.65-3.04)	0.00 (0.00-8.43)	0.29 (0.20-0.29)
III.1.b (14)	10.09 (1.67-18.03)	4.41 (4.02-6.66)	2.74 (1.08-17.54)	0.88 (0.69-8.23)
III.1.c (15)	18.62 (6.37-35.18)	4.60 (8.43-14.70)	18.82 (12.15-40.67)	4.70 (2.45-5.00)
III.2.a (16)	2.25 (0.20-4.90)	1.37 (0.98-1.67)	5.29 (1.37-11.17)	0.69 (0.49-0.78)
III.2.b (17)	2.16 (0.20-4.90)	1.27 (0.69-1.37)	0.29 (0.00-1.67)	0.34 (0.28-0.38)
III.2.c (18)	3.63 (0.10-11.56)	0.88 (0.69-1.27)	0.00 (0.00-0.39)	0.34 (0.27-0.39)
ULTT Med. (19)	23.03 (10.49-65.37)	14.21 (9.51-19.01)	13.03 (6.27-28.42)	4.41 (2.45-4.70)
ULTT+ Med. (20)	32.44 (12.54-71.05)	19.99 (15.09-24.89)	19.31 (9.80-40.18)	5.98 (2.55-6.37)
ULTT Uln. (21)	1.08 (0.00-4.90)	0.39 (0.00-0.88)	4.61 (0.00-12.45)	1.18 (0.98-1.57)
ULTT Rad. (22)	11.07 (0.20-23.42)	9.11 (6.96-11,17)	0.39 (0.00-2.65)	0.59 (0.39-0.69)
	r_s: 0.91 p< 0.001		r_s: 0.88 p< 0.001	

[1] The position numbers in parentheses correspond with the position numbers in figures 2 and 3
[2] Medians and (in parentheses) Ranges in newton [1]

At the axilla the medians of the tensile forces in the unembalmed human bodies tend to be lower than those in embalmed specimens (table 2 and figure 2). At the wrist, in some neutral posions and positions with palmar flexion (i.e. positions which produce little or no tension in the median nerve) this relation is less consistent (table 2 and figure 3).

Discussion

In anatomical-biomechanical research embalmed human bodies are frequently used. The relevance of the data obtained has to be discussed in the context of the experimental design used. In this study a positive correlation was found between tensile force data from unembalmed and embalmed specimens. This justifies the use of embalmed human bodies in an experimental design aimed at analysing mechanical tension in the peripheral nerves, due to changes in position of the arm[5].

A greater similarity between tension in unembalmed and embalmed nerves was found for data obtained at the axilla when compared with those of the wrist. Two explanations arise: Firstly, the difference in surroundings of the nerve at the axilla and the wrist. The surrounding tissue of the nerve in the axilla consists of loose connective tissue organised in a tubular way. In the wrist, just proximal to the level of the ligamentum carpi transversum (flexor retinaculum), a denser kind of collagen is found. The relationship of the nerve with its surroundings is here more intimate. As shown by Rath and Millesi[12], the gliding of the nerve in relation to its surroundings is an important mechanism in the dissipation of tensile forces. In the study of Kleinrensink et al.[5] and the present study, all except the four ULTT positions have the glenohumeral joint fixed at 90 degrees abduction. The variation in range of motion in the wrist is much greater than at the glenohumeral joint. Consequently, at the wrist, this makes a large demand on the gliding mechanism of the median nerve. It is to be expected that a negative effect of embalmment on this gliding mechanism would cause, at this site, a more pronounced difference in tensile forces between unembalmed and embalmed specimens.

Secondly, at the wrist, less similarity is found in positions where (very) low or no tensile forces are observed. In general, these are neutral positions and positions with palmar flexion of the wrist. In these positions the median nerve becomes slack. The buckle force transducers are constructed for registrating tensile forces. In positions where little or no tension is exerted to the nerve, values registrated with buckle force transducers become less reliable.

Since some inconsistency exists (not all positions show a smaller tensile force in the unembalmed specimens), the value of data obtained from embalmed specimens can be disputed in experimental designs with a need for direct extrapolation to the *in vivo* situation.

It can be concluded that data obtained from tensile force measurements on the median nerve of embalmed human bodies are positively related to those of unembalmed bodies. In designs intended for comparison of tensile forces on the median nerve in different positions of the arm[5], data from embalmed specimens are relevant.

Acknowledgements

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Chapter 3

Mechanical tension in the median nerve

The effects of joint positions

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Mechanical tension in the Median nerve

The effects of joint positions

Summary
Stretch tests are attractive in the diagnosis of nerve root or peripheral nerve lesion. Interpretation of the test results is often difficult since the distribution of tensile forces along the nerve, caused by the test manoeuvre is not known. In this study, the effect on median nerve tension of twenty-two positions of the arm was measured with buckle force transducers. With the elbow in full extension and the hand in neutral position, altering the position of the shoulder significantly influenced tension in the proximal part of the median nerve; tension in the distal part was not influenced. With the shoulder in 90° abduction, dorsal flexion of the hand combined with an *extended* elbow resulted in an increased tension in both distal and proximal parts of the median nerve. Dorsal flexion of the hand combined with flexion of the elbow caused an increase in tension only in the distal part. At all sites of the median nerve, the median nerve upper limb tension test caused a significantly higher tension than the radial and ulnar nerve upper limb tension tests.
This study provides insight in the normal distribution of tensile forces along the median nerve and can have clinical consequences. For differentiating nerve root from peripheral nerve lesions a specific provocative tension test for the median nerve is advocated. The results of this study provide a theoretical basis for differentiating between lesions in the proximal and distal part of the median nerve.

Relevance
In the diagnosis of nerve(root) lesions, tests in which stretching the nerve provokes the symptoms, are widely used, but no data are available on the distribution of tensile forces along nerves and nerve roots during such tests. In this study a human cadaver model is presented to analyse tensile force distribution on the median nerve; it can be an aid to evaluate the specificity of a clinical provocation test for the median nerve.
Key words: Peripheral nerve, mechanical stress, (normal) articular range of motion, diagnosis
Mechanical tension in the median nerve

Introduction
In diagnosing nerve root lesions, several tests are available in which tensile forces provoke clinical symptoms (e.g. straight leg raising test). For two reasons, the interpretation of test results is difficult. Firstly, no knowledge is available of the distribution of the tensile forces along nerve root and peripheral part of the nerve caused by normal upper extremity movements and test manoeuvres. Secondly, symptoms usually associated with nerve root lesions also can be caused by peripheral nerve lesions. Thus problems arise in differentiating between nerve root lesions and lesions in the peripheral part of the nerve and in exactly localizing the site of nerve lesion.
As an analogon of the straight leg raising test, Kenneally et al.[2] proposed the 'upper limb tension tests' (ULTT's) for the median, ulnar and radial nerve. Also for these tests, the distribution of tensile forces is not known. Thus empirical findings are clinically used without fundamental knowledge of the mechanical properties of the examined structures and knowledge of the specificity of tests.

In the present study, twenty-two positions of the arm in the normal range of motion (RoM) are used to analyze the tensile forces at three sites of the peripheral part of the median nerve. The aim of this study is to provide a basis for a correct interpretation of clinical tests for the diagnosis of peripheral nerve lesions.

Materials and methods

Forty-eight to sixty hours after death, five human bodies were embalmed by vascular perfusion with a medium containing 2.2% formaldehyde. (for a detailed description of the fixation fluid, see: Kleinrensink et al, 1995[3]) The bodies were stored for three months in containers filled with fenoxy-ethanol at a temperature of 4 degrees Celcius. After this period, measurements were performed bilaterally (n=10), at three sites on the median nerve: 1) in the axilla, about two cm distal to the bifurcation (Ax), 2) two cm proximal of the pas sage of the nerve through the pronator teres muscle (PP) and 3) at the wrist, two cm proximal to the styloid process of the radius (Wr). (Figure 1)The measurements were performed under standardized conditions including room temperature and air humidity.

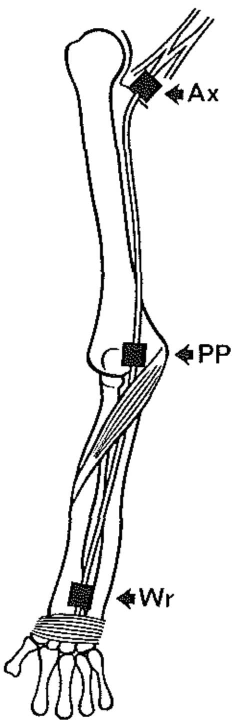

Figure 1 Position of the three buckle force transducers.
Ax = 2 cm distal to the bifurcation (lateral and medial cord forming the median nerve), **PP** = 2 cm proximal of the median nerve through the pronator teres muscle,
Wr = 2 cm proximal to the styloid process of the radius.

Test positions

For the eighteen positions in the normal RoM and the test positions used for the median, ulnar and radial nerve ULTT and a modified median nerve ULTT *see* Kleinrensink et al, 1994[3]: table 1 and 1a . All twenty-two joint positions were studied three times; as test and retest and after application of a tight band around the arm. The band was located at the site of the pronator teres muscle, simulating obstruction of the gliding mechanism of the median nerve. All tests were performed with the bodies lying supine with neutral position of the cervical spine.

Tension measurements

'Buckle' force transducers[4,5] were placed at Ax, PP and Wr. The buckle transducer of Peters[5] was designed for measuring stress and strain fields in ligaments. For attaching the transducer to the nerve, a stainless steel 'U' formed bar was constructed which locked the nerve in the transducer without initial damage. (figure 2)

The transducer was extensively tested on nerve tissue and was shown to have a high test-retest reliability. To compensate for deformation by tensile stress the nerve-buckletransducer unit was calibrated after each measurement (see: *calibration procedure*).

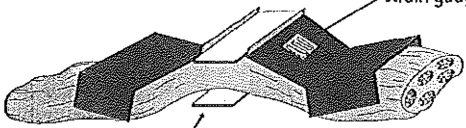

Figure 2
Buckle force transducer according to Peters[5]

The signal from the buckle transducer was amplified by a bridge amplifier (made by the Department of Biomedical Physics and Technology) and led to a four channel plotter.

Calibration procedure

To convert the transducer output from millivolt to newton, the nerve segment and transducer were simultaneously removed from the body by dissection and the transducers were calibrated for each measured nerve segment. Calibration was performed twice from 0 to 1000 g in steps of 100 g. (correlation coefficient: > 0.955; mean standard error of estimation: 0.04, range: 0.02-0.09).

Statistical analysis

All independent factors were considered fixed. Their levels are represented by dummy variables using reference coding in a multiple linear regression analysis.

Results

Normal range of movement

In relation to a reference position of 120° flexion in the elbow, a position of 90% shows no change in tension in the median nerve at the axilla (Ax), proximal to the pronator teres muscle (PP) and at the wrist (Wr).

Table 1. The effect of elbow and hand position on tension in three median nerve segments

Tension		Axilla	Pronator proximal	Wrist
Elbow reference 120°(flexion)	90°	0.20 (SEM 0.49)	0.00 (SEM 0.59)	0.00 (SEM 0.59)
	0°(extension)	8.73 (SEM 0.49)‡	8.44 (SEM 0.59)‡	4.81 (SEM 0.59)‡
Hand reference 0°(neutral)	70° dorsal flexion	3.04 (SEM 0.49)‡	3.72 (SEM 0.59)‡	8.34 (SEM 0.59)‡
	80° palmar flexion	-0.58 (SEM 0.49)	-0.59 (SEM 0.59)	-0.88 (SEM 0.59)
Reference max. supination	pronation (maximal)	-0.39 (SEM 0.39)	-1.08 (SEM 0.49)§	-1.28 (SEM 0.49)§

Mean differences and SEM (in newton) with the reference level, taking into account all other factors in the model.
‡ $p < 0.001$; § $p < 0.05$

Full extension of the elbow (0°) significantly increases tension in all three nerve segments (table 1). Tension in all three segments is also significantly higher if full extension is compared to 90° ($p < 0.01$).
The 70° (dorsal flexion) position of the hand differs significantly from the 0° (neutral) position, whereas the 80° (palmar flexion) position does not (table 1).Compared to supination, pronation decreases the tension of the median nerve at PP and Wr but not at the Ax site.

Median nerve and modified median nerve ULTT
The median nerve ULTT and the modified median nerve ULTT are compared with a reference position (see Kleinrensink et al[3] : position III.1.c, tables 1 and 1a). In performing the median nerve ULTT, 20° abduction, 10° retroflexion and 60° external rotation is added to the reference position. This results in a significant increase in tension in the median nerve at AX and PP.
Altering shoulder joint position to maximal abduction, maximal retroflexion and maximal external rotation (modified median nerve ULTT) an even larger increase is found. Tension at the distal (Wr) site is not significantly influenced by altering the shoulder position (table 2).
To analyze whether differences occur between the four testing manoeuvres, the original median nerve ULTT was taken as reference. At all three sites of the median nerve, the modified median ULTT nerve causes a significantly higher tension than the original median nerve ULTT. This in sharp contrast to the effects of the ulnar and radial nerve ULTT. (table 3).

Table 2. Differences between Upper Limb Tension Test (ULTT) / modified ULTT and a reference position, comparable in elbow and hand position (see Kleinrensink et al 1995[3], Table 1: pos.III.1.c)

Tension		Axilla	Pronator proximal	Wrist
reference: Elbow: 0°, max. supination	ULTT median nerve	8.53 (SEM 2.75)†	10.01 (SEM 3.34)†	2.94 (SEM 1.86)
Hand:70° (dorsal flexion) (table 1: pos. III.1.c)[3]	Modified ULTT Median nerve	17.00 (SEM 2.75)‡	18.44 (SEM 3.34)‡	2.26 (SEM 1.86)

Mean differences and SEM (in newton) with the reference level of the factor. ‡ $p < 0.001$; † $p < 0.01$

Table 3. Differences between the originally suggested median nerve upper limb tension test (ULTT) and the modified median nerve ULTT, the ulnar nerve ULTT and the radial nerve ULTT

Tension		Axilla	Pronator proximal	Wrist
reference: ULTT median nerve	Modified ULTT Median nerve	8.44 (SEM 2.94)†	8.44 (SEM 3.04)†	5.20 (SEM 2.16)§
	ULTT ulnar nerve	-23.15 (SEM 2.94)‡	-25.11 (SEM 3.04)‡	-12.26 (SEM 2.16)‡
	ULTT radial nerve	-14.72 (SEM 2.94)‡	-17.95 (SEM 3.04)‡	-17.17 (SEM 2.16)‡

Mean differences and SEM (in newton) with the reference level of the factor. ‡ $p < 0.001$; † $p < 0.01$; § $p < 0.05$

Differentiation between lesions in the upper and lower part of the median nerve

With the shoulder in 90° abduction and the elbow in full extension, tension is significantly increased in all three segments by 70° dorsal flexion of the hand. Dorsal flexion of the hand with 90° flexion of the elbow causes a significant rise in tension only at the Wr site of the median nerve.

In these positions no difference between pro- and supination was observed (table 4).

With a 90° flexed elbow, 70° dorsal flexion of the hand (if compared to the neutral position of the hand) causes a significant rise in tension only in the distal (Wr) part of the median nerve (table 5).

In comparing the mean values of normal range of movement tension with those of the simulated pronator teres obstruction, no significant differences were found.

Discussion

Since Lasègue[6] proposed a tension test for the sciatic nerve, this provocation test is a widely used tool in diagnosing lumbar radiculopathy. Recently, several testing positions of the upper extremity have been proposed. They are supposed to stress the cervical nerve roots and thus could be used as provocation tests[2,7,8].

The mentioned tests are based on the assumption that due to certain test manoeuvres, tension increases in a nerve root entrapped in the intervertebral foramen.

As a result of increased tension on the inflamed and irritated nerve root, the patient feels paraesthesia or pain in the sensory distribution area of the specific nerve.

Table 4. Differences between dorsal flexion of the hand combined with flexion in the elbow and dorsal flexion of the hand combined with extension (respectively with supination and pronation)

Tension		Axilla	Pronator proximal	Wrist
reference: II.1.c[1]	III.1.c[2]	14.91 (SEM 1.47)‡	16.00 (SEM 1.47)‡	12.36 (SEM 2.16)‡
reference: II.2.c[3]	III.2.c[4]	13.34 (SEM 1.37)‡	16.09 (SEM 2.75)‡	10.70 (SEM 1.86)‡

Mean differences and SEM (in newton) with the reference level of the factor
[1] elbow: 90° flexion, supination hand: 70° dorsal flexion
[2] elbow: extension, supination hand: 70° dorsal flexion
[3] elbow: 90° flexion, pronation hand: 70° dorsal flexion
[4] elbow: extension, pronation hand: 70° dorsal flexion
‡ $p < 0.001$

However, in manipulating distal parts of an extremity, the question arises whether tensile forces are actually transmitted all the way up to the nerve root. To answer this question, and to differentiate between peripheral nerve and nerve root lesions, detailed knowledge is required of the distribution of tensile forces in the test positions. Such a knowledge might explain why only half of the patients suffering from sciatica and characterised by a significant reduction of straight leg raising showed abnormal myelograms[9]. In these patients a lesion of the *peripheral* nerve, provoking symptoms generally associated with nerve *root* lesions, cannot be excluded.

Besides the question of how tensile forces are distributed along the median nerve, the aspect of the magnitude of tensile forces is of interest. Although, by our knowledge, actual tensile forces were not analysed before, there is a large number of publications concerning the relation between stretching and tensile forces in peripheral nerves. In 1961 Sunderland[10] already studied the relation between elongation and upper limit of elasticity and mechanical failure in human cadaver studies. He found the mean percentage of elongation at the elastic limit to be 14% (range 8-21%) and at mechanical failure to be 19% (range 7-31%). More recently Kwan et al.[11] in an *in vivo* experiment, found that irreversible *functional* deficit already occur red after strain of more than 12 percent of the in situ strain of rabbit tibial nerve. The present study is performed on embalmed human bodies, this can have certain influence on the tensile forces measured. Although the exact effects of embalmment on tensile forces on the median nerve have never been reported, certain changes in the collagen will take place. However, if compared to measurements on unembalmed specimens there appears to be a high positive correlation between data from unembalmed and embalmed human bodies[3]. So in the present study, which is a comparative study, the use of embalmed human bodies represents no specific problems. From table 1 it can be concluded that the position of the hand is relevant for the tension in the whole median nerve (including the proximal part). Tension in the AX part is caused by dorsal flexion of the hand only when the elbow is extended (0°).

Full extension increases tension significantly in the middle and distal part of the median nerve (table 1). Combined with the findings in table 4, this could be relevant in certain prolonged surgical procedures in which patients have to be in the supine position with fully abducted arm and extended elbow (e.g. radical mastectomy). Maintained tensile stress can be harmful to peripheral nerve[11,12,13]. The current study shows that tensile stress can be decreased by flexing the elbow to 90° or 120°. The position of the shoulder is important for the tension in the middle as well as the proximal part of the median nerve (table 2).

Table 5. Differences between dorsal flexion of the hand and neutral position, combined with flexion in the elbow (respectively with supination and pronation)

Tension		Axilla	Pronator proximal	Wrist
reference: II.1.b[1]	II.1.c[2]	0.20 (SEM, 0.29)	0.00 (SEM, 0.69)	5.40 (SEM 0.88)‡
reference: II.2.b[3]	II.2.c[4]	0.10 (SEM, 0.39)	0.49 (SEM, 0.39)	5.30 (SEM 0.98)‡

Mean differences and SEM (in newton) with the reference level of the factor
[1] elbow: 90%flexion, supination hand: 0% (neutral)
[2] elbow: 90%flexion, supination hand: 70% dorsal flexion
[3] elbow: 90%flexion, pronation hand: 0% (neutral)
[4] elbow: 90%flexion, pronation hand: 70% dorsal flexion
‡ $p < 0.001$

Manipulating joints in the distal part of the upper extremity can increase nerve tension in the most proximal part of the median nerve (table 1). However, changing the shoulder joint position does not alter nerve tension in the distal part of the median nerve (table 2).

In all three parts of the median nerve both the ulnar and radial nerve ULTT cause a significantly lower tension than the median nerve ULTT (table 3). These findings support the hypothesis that the ULTT for the median nerve is indeed specific for the median nerve. The modified median nerve

ULTT is even more effective than the original median nerve ULTT (table 3). The experiments were not performed on trunks and cords of the brachial plexus. It can be expected that tension values of the relevant cords do not significantly differ from the values of the upper part of the peripheral nerve. Preliminary data on tension in trunks and cords of the brachial plexus do indeed confirm this. With extended elbow, dorsal flexion of the hand increases tension in all parts of the nerve. With flexed elbow, the same wrist position causes increase in tension only in the Wr part (tables 4 and 5). Thus, in principle, differentiation between lesions in the upper and lower part of the median nerve is possible by manipulating specific joints. More evidence for the differentiation between lesions in the distal and proximal part of the median nerve must come from clinical investigations.

Conclusions

This study gives insight into the effects of joint positions on tensile forces of the median nerve; joint positions were compared with each other and the relative effects on tensile stress are discussed. Evidence is provided for the specificity of the ULTT for the median nerve. A rationale is offered for the manipulation of specific joints to differentiate between lesions in the proximal and distal part of the median nerve.

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Chapter 4

Upper limb tension tests as tools in the diagnosis of nerve and plexus lesions

Part one:

Anatomical and Biomechanical aspects

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Upper limb tension tests as tools in the diagnosis of nerve and plexus lesions

Part one: Anatomical and Biochemical aspects

Abstract

In this anatomical and biomechanical study, nerve tension tests used in the diagnosis of nerve(root) and plexus lesions of the upper extremity are evaluated. We studied the upper limb tension tests (ULTT) for the median, ulnar and radial nerve, as well as the ULTT combined with contralateral rotation and lateral bend of the cervical spine (ULTT+). Measurements were performed on the median, ulnar and radial nerve and the medial, lateral and posterior cord of the brachial plexus of embalmed human bodies. As the outcome variable we used mechanical tension assessed with 'buckle' force transducers. Based on the outcome variable, both the median nerve ULTT and ULTT+ turned out to be sensitive and specific tension tests. This does not hold for the ulnar and radial nerve ULTT and ULTT+. The findings justify further investigation of the median nerve ULTT and ULTT+ on its clinical validity. Tension introduced in the extremity was shown to be transmitted to both the nerves and the cords of the brachial plexus. Mechanical tension caused by the ULTT+ tended to be higher than that caused by the ULTT. The observations support the concept of the brachial plexus having a function in distributing mechanical forces, thus protecting the cervical nerve roots against excessive traction forces. It is unlikely that upper limb tension tests can be used to selectively stress cervical nerve roots.

Introduction

In 1864 Lasègue[1] proposed a simple mechanical nerve tension test for the diagno sis of lumbar disc herniations. This straight leg raising (SLR) test is assumed to provoke symptoms by stretching the entrapped and inflamed lumbar nerve root in the intervertebral foramen. Although the SLR test is widely used by clinicians, its validity has been disputed[2,3]. Difficulties with SLR test are not surprising because its validity is exclusively based on clinical, empirical data. No quantitative data are available on the tensile forces along the nerve, plexus and nerve roots generated by the test. The reason why nevertheless analogons of the SLR test for cervical nerve root lesions[4,5,6] have been sought is obvious: in clinical practice fast, easy and low cost diagnostic measures are attractive. However, since the validity of these upper limb tension tests (ULTT) is also not based on quantitative data but on nerve topography[4] or on qualitative, empirical data[5,6], the same problems as with the SLR are to be expected. To provide a quantitative basis for the use of nerve tension tests in the upper extremity, two *in vitro* studies have been performed on tensile force distribution in peripheral nerves[7,8]. Where McLellan and Swash[9] showed that, due to positions of the arm, the median nerve moves up and downward in its bed, we analysed the magnitude and distribution of tensile forces in this nerve due to joint movement. In the present study we firstly made an analysis of the validity of six ULTT: median, ulnar and radial nerve ULTT and the same testspositions with added contralateral rotation and lateroflexion of the cervical spine (ULTT+). Tensile forces (in N) were used as the outcome variable. Secondly, we wanted to know whether the tensile forces caused by the recommended tests positions, were transmitted up to the brachial plexus and if so, how they were distributed in the plexus. Thirdly, we tried to answer the question: can the results of the *in vitro* studies on tensile

force distribution be related to nerve function? Since tensile forces in peripheral nerves cannot be assessed *in vivo*, the first two questions had to be answered by performing an in vitro study. For this purpose we performed an anatomical-biomechanical experiment on embalmed human bodies (Part one of this study). In an earlier study we showed a strong positive correlation between tensile force data obtained from unembalmed and embalmed human bodies[8]. The third question could be answered by performing a neuro-physiological *in vivo* experiment (Part two of the study).
The purpose of the study (Part I and Part II) is to provide a quantitative basis for the clinical use of nerve tension tests in the diagnosis of nerve, nerve root and/or plexus lesions in the upper extremity.

Methods

Six arms of three embalmed human bodies were used. The bodies were embalmed by vascular perfusion with a medium containing 2.2% formaldehyde (see reference 8 for the composition of the fixation fluid). Tension measurements were performed on the medial, lateral and posterior cord of the brachial plexus and on the proximal part of the median, ulnar and radial nerves, two centimetres distal to the plexus cords. (Figure 1)

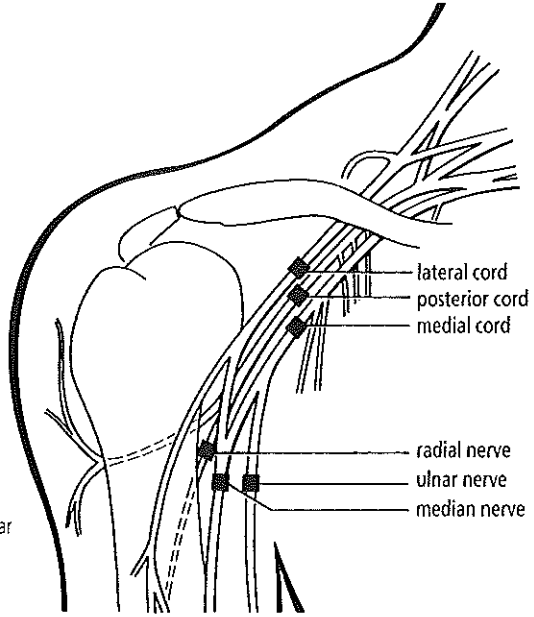


Figure 1
Position of the "buckle" force transducers for recordings at the proximal part of the Median, Ulnar and Radial nerve and the Medial, Lateral and Posterior cord of the Brachial plexus

The ULTT and ULTT+ testing manoeuvres for median, ulnar and radial nerve are summarized in table 1 and visualized in figure 2. The measurements were performed under standardized conditions including room temperature and air humidity.

Table 1. Upper limb tension tests (ULTT + ULTT+) for the median, ulnar and radial nerves

	shoulder	elbow	wrist
Median nerve ULTT	110° abduction 60° exorotation	0° (extension) max. supination	70° dorsalflexion 10° retroflexion
Ulnar nerve ULTT	max. abduction max. exorotation	120° (flexion) max. supination	70° dorsalflexion
Radial nerve ULTT	max. abduction max. pronation max. endorotation	0° (extension) max. retroflexion	70° palmarflexion

In the ULTT the Cervical Spine is in the neutral position In the ULTT+, contralateral lateral bend and contralateral rotation of the cervical spine is added to the ULTT position In all tests the fingers are in the 0° (neutral) position

To assess tensile forces 'Buckle' force transducers of small dimensions (0.8 x 2.9 cm) were used. A smaller size was not possible because of the necessity of having a full Wheatstone bridge of strain gauges to compensate for temperature changes (see Peters, 1987[10] for a description of the force transducer; for the measurement procedure see Kleinrensink et al. 1995[7]).

Calibration procedure

To convert the transducer output from millivolts to newton, the nerve segment and transducer were simultaneously removed by dissection. The transducer-nerve unit was calibrated for each measured nerve segment. Calibration was performed twice from 0 to 1000 g in steps of 100 g (correlation coefficient: > 0.983; mean standard error of estimation: 0.05, range: 0.03-0.07).

Statistical analysis

For statistical analysis, a *multiple linear regression analysis* was performed. Tension and difference between tensions were the variables to be explained. As explanatory variables dummy variables were defined to represent the fixed factors (body, arm and test position).

Results

Median, ulnar and radial nerve

In table 2 (effect of ULTT) and 2a (effect of ULTT+) the rows show the tension in the respective nerves caused by one particular test, and the columns show the tension in one nerve caused by the respective tests.

ULTT

In comparing the effects of one ULTT on three nerves (rows), exclusively the median nerve ULTT caused higher tension in the nerve for which the test is intended. The radial nerve ULTT caused more tension in the median nerve than in the radial nerve itself. In comparing the effect of the three tests on one nerve (colums), exclusively the radial nerve ULTT caused more tension in the corresponding (i.e. radial) nerve when compared with the other two tests.

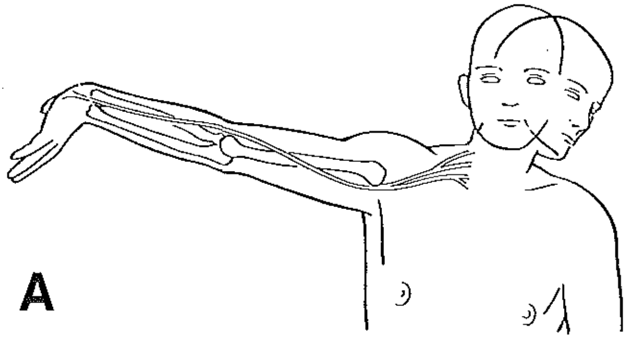
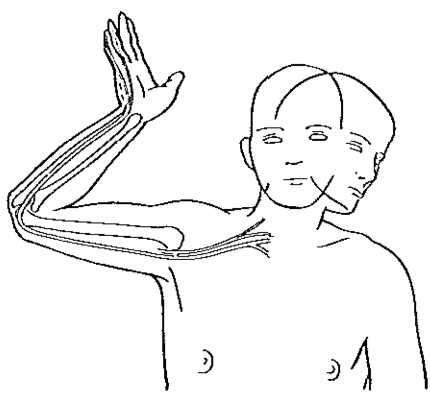


Figure 2
Joint positions of the Upper Limb Tension Test (ULTT) and Upper Limb Tension Test with contralateral lateral bend and contralateral rotation (ULTT +) for the Median, Ulnar and Radial nerve (see also table 5).
A = Median nerve ULTT and ULTT +
B = Ulnar nerve ULTT and ULTT +
C = Radial nerve ULTT and ULTT +



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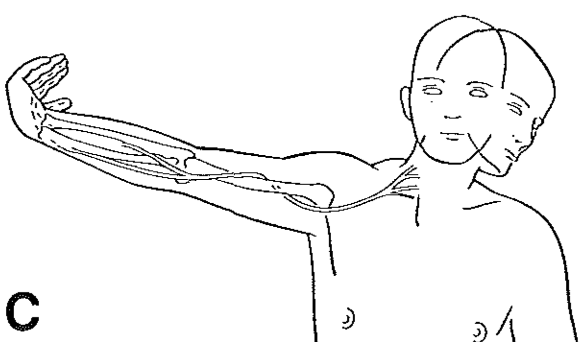


ULTT+

In comparing the effects of one ULTT+ on three nerves exclusively the median nerve ULTT+ caused higher tension in the intended nerve. In comparing the effect of the three ULTT+ tests on one nerve, exclusively the median nerve ULTT+ caused more tension in the corresponding nerve when compared with the other two tests.

Table 2. Mean tensile forces (in N ± sd) on median, ulnar and radial nerve, caused by the median, ulnar and radial nerve upper limb tension test

	A. Median nerve	B. Ulnar nerve	C. Radial nerve	p* A-B	p A-C	p B-C
I. Median nerve ULTT	10.88 (5.88)	1.18 (0.98)	1.86 (0.59)	<0.001	<0.001	n.s.**
II. Ulnar nerve ULTT	0.59 (0.29)	3.92 (3.04)	2.94 (2.55)	n.s.	n.s.	n.s
III. Radial nerve ULTT	8.53 (5.19)	1.76 (1.57)	5.88 (2.35)	<0.01	n.s.	<0.001
p I-II	<0.001	<0.05	n.s.			
p I-III	n.s.	n.s.	<0.001			
p II-III	<0.001	n.s.	<0.01			

* p-values are derived from the multiple linear regression analysis ** n.s.= not statistically significant.

Table 2a. Mean tensile forces (in N ± sd) on median, ulnar and radial nerve, caused by the median, ulnar and radial nerve upper limb tension test +

	A. Median nerve	B. Ulnar nerve	C. Radial nerve	p* A-B	p A-C	p B-C
I. Median nerve ULTT+	11.07 (3.82)	0.78 (1.08)	2.84 (1.08)	<0.001	<0.001	<0.01
II. Ulnar nerve ULTT+	0.59 (0.49)	4.61 (4.80)	5.59 (5.88)	<0.05	n.s.	n.s
III. Radial nerve ULTT+	5.19 (2.55)	1.47 (1.47)	7.55 (2.55)	n.s.	n.s.	<0.001
p I-II	<0.001	<0.05	n.s.			
p I-III	<0.01	n.s.	<0.05			
p II-III	<0.05.	n.s.	n.s.			

* p-values are derived from the multiple linear regression analysis

Medial, lateral and posterior cord

In table 3 and 3a the rows show the tension in the respective cords caused by one specific test, the columns show tension in a specific cord caused by the respective tests.

When tensile forces are introduced to the plexus via a particular nerve, the forces are expected to be transmitted to the plexus cords to which this nerve is anatomically related[4,11]. For the sake of brevity in the results section only the differences with these expectations are mentioned.

Table 3. Mean tensile forces (in N ± sd) on medial, lateral and posterior cord, caused by the median, ulnar and radial nerve upper limb tension test

	A. Medial Cord	B. Lateral Cord	C. Posterior Cord	p* A-B	p A-C	p B-C
I. Median nerve ULTT	9.31 (6.17)	5.10 (4.21)	1.57 (1.47)	<0.05	<0.001	<0.05
II. Ulnar nerve ULTT	4.21 (4.80)	0.49 (0.69)	2.55 (2.06)	<0.01	<0.05	<0.05
III. Radial ULTT	8.13 (5.88)	4.41 (3.42)	5.00 (2.74)	<0.05	<0.05	n.s. nerve
p I-II	<0.01	<0.01	n.s.			
p I-III	n.s.	n.s.	<0.01			
p II-III	<0.05	<0.05	<0.05			

* p-values derived from the multiple linear regression analysis

ULTT

All three ULTT caused a higher tension in the medial cord, than in the lateral and posterior cord. The radial nerve ULTT caused about equal tension in the lateral and posterior cords. In all three cords tension caused by the radial nerve ULTT was higher than that caused by the ulnar nerve ULTT. In the medial cord, the median nerve ULTT caused a higher tension when compared with the ulnar nerve ULTT; the radial nerve ULTT caused higher tension than the ulnar nerve ULTT. In the lateral cord the median and radial nerve ULTT caused about equal tension.

ULTT+

The median nerve ULTT+ caused higher tension in the medial cord when compared with the lateral cord. The ulnar nerve ULTT+ caused about equal tension in the medial and posterior cord. The radial nerve ULTT+ caused about the same tension in all three cords.

Table 3a. Mean tensile forces (in N sd) in medial, lateral and posterior cord, caused by the median, ulnar and radial nerve upper limb tension test+

	A. Medial Cord	B. Lateral Cord	C. Posterior Cord	p* A-B	p A-C	p B-C
I. Median nerve ULTT+	10.29 (7.64)	6.37 (4.99)	2.84 (2.06)	<0.05	<0.001	<0.05
II. Ulnar nerve ULTT+	4.51 (5.00)	0.88 (1.47)	4.21 (4.61)	<0.01	n.s.	<0.001
III. Radial nerve ULTT+	5.98 (5.59)	5.49 (3.23)	6.27 (3.52)	n.s.	n.s.	n.s.
p I-II	<0.05	<0.01	n.s.			
p I-III	n.s.	n.s.	<0.05			
p II-III	n.s.	<0.01	n.s.			

* p-values derived from the multiple linear regression analysis

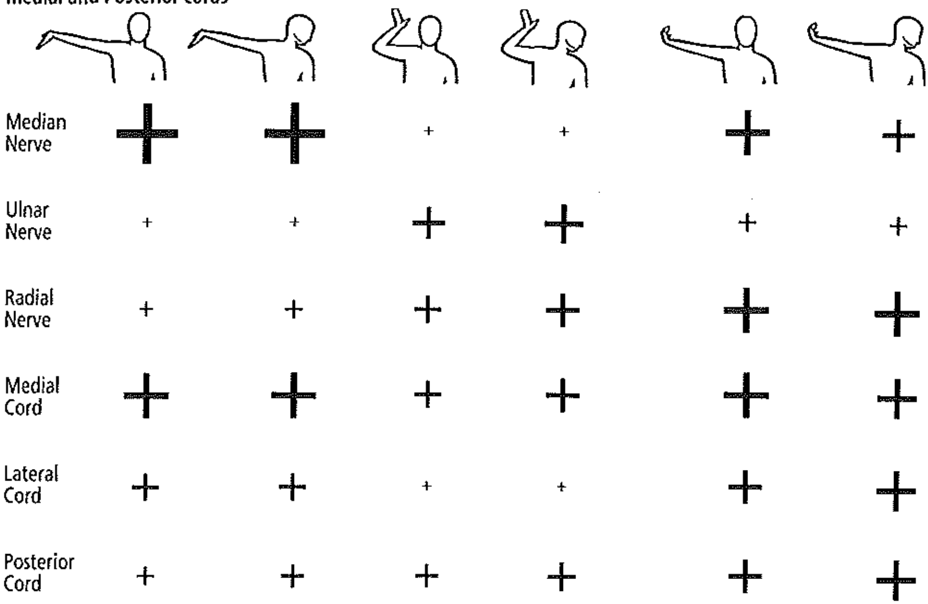
In the medial cord, the median nerve ULTT+ caused higher tension when compared with the ulnar nerve ULTT+. In the lateral cord, median and radial nerve ULTT+ caused about equal tension. In the posterior cord the radial and ulnar nerve ULTT+ caused about equal tension.
For easy reference the relation between the effects of the median, ulnar and radial nerve ULTT and ULTT+ on the median, ulnar and radial nerve and the lateral, medial and posterior cord is visualised (table 4).
Apart from four exceptions (see tables 2 and 2a and 3 and 3a), tensile forces caused by the ULTT+, tended to be higher than by the ULTT; statistical significancy was reached only in the case of the radial nerve ULTT+ vs ULTT.

Discussion

Test validity
In analysing the validity of a nerve tension test, an important question to be answered is: Does the test cause the highest tension in the nerve for which it is intended? Table 2 shows that two tests out of six do not meet this criterion (ulnar nerve ULTT+ and radial nerve ULTT). However, in clinical practice other important aspects of clinical test validity are the *sensitivity* (probability of testing positive if a nerve lesion is truly present) and the *specificity* (probability of testing negative if a nerve lesion is truly absent)[12]. Although several factors play a role in test *sensitivity*, it can be postulated that the more tension the test causes in the intended nerve, the more sensitive it will be. In this respect, the median nerve ULTT and ULTT+ are more sensitive in assessing median nerve lesions than the ulnar nerve ULTT in assessing ulnar nerve lesions and the radial nerve ULTT+ in assessing radial nerve lesions.
In determining *the specificity* of a nerve tension test it is essential to know whether the test causes significant tension in other nerves than the intended one. Otherwise test results (i.e. pain, discomfort etc.) could be due to increased tension in other nerves. Tables 2 and 2a show that the median

nerve ULTT and ULTT+ are more specific than the ulnar and radial nerve ULTT and ULTT+. The ulnar nerve ULTT is not specific since there is no significant difference between tension caused in the ulnar, median and radial nerves. Although the ulnar nerve ULTT+ caused more tension in the ulnar nerve than in the median nerve, the tension in the ulnar nerve tended to be smaller than in the radial nerve. In the diagnosis of radial nerve lesions, the radial nerve ULTT is not specific; it tended to cause less tension in the radial nerve than in the median nerve. The radial nerve ULTT+ cannot be considered specific either: the tension created in the radial nerve was not significantly higher than that in the median nerve.

Table 4 Visualisation of the effect of six tension tests on the Median, Ulnar and Radial nerves and the Lateral Medial and Posterior cords



Transmission of forces

In a previous study[7] tensile force distribution was analysed in the peripheral part of the median nerve. With respect to the transmission of tensile forces proximally, the present study was performed to answer two questions. With respect to the question whether forces caused by the ULTT and ULTT+ manoeuvres are transmitted up to the brachial plexus, the answer is positive (tables 3 and 3a). It was not possible to construct 'buckle' force transducers of such a small size that they could be placed on trunks and nerve roots. So it is not certain that forces are transmitted up to these structures. However, tension in the cords is only slightly lower when compared with the anatomically related nerves (in the case of the ulnar nerve and medial cord there is even a slight increase). Therefore, in our opinion, it can be safely assumed that due to the tests, tension is also increased in trunks and roots. Secondly we wanted to know whether the forces caused by the tension tests showed a specific pattern in the brachial plexus. As to be expected on the basis of the anatomy described in well known textbooks[4,11,13], the median nerve ULTT and ULTT+ raised more tension in the medial and lateral cord than in the posterior cord. Unexpected is the observation that significantly more tension is transmitted via the medial cord than via the lateral cord. In performing the ulnar nerve ULTT forces are, as could be expected, transmitted mainly via the medial cord. In performing the ULTT+, tension in the medial cord remained about the same but tension in the lateral and posterior cord increased with about 50% when compared with the ULTT. Apparently, forces become larger and equally distributed in the medial and posterior cord when pulling simultaneously from proximal and distal. In performing the radial nerve ULTT, the posterior cord is expected to transmit forces. Surprisingly, higher tension was observed in the medial cord than in the posterior cord. By performing the radial nerve ULTT+, tension in the medial, lateral and posterior cord becomes equal. These findings support the idea that the brachial plexus plays a role in the distribution of tensile forces[14]. Based on the present findings and combined with the topographical plexus anatomy described in the literature, it is unlikely that any of the six tests studied will selectively stress specific cervical nerve roots.

Conclusions

Based on tensile force distribution, out of the six tests studied, exclusively the median nerve ULTT and ULTT+ can be seen as valid nerve tension tests considering both sensitivity and specificity. Further clinical evaluation is justified and needed to verify its clinical value. Using the ulnar nerve and radial nerve ULTT and ULTT+, one has to be very cautious with the interpretation of test results.

It could be shown that tensile forces applied to the upper extremity are transmitted up to the cords of the brachial plexus. Considering the textbook anatomy of the brachial plexus, the transmission of tensile forces showed an unexpected pattern, especially when performing the radial nerve ULTT and ULTT+. It is unlikely that any of the studied tests can be used to stress specific nerve roots.

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Chapter 5

Upper limb tension tests as tools in the diagnosis of nerve and plexus lesions

Part two: Functional aspects

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Upper limb tension tests as tools in the diagnosis of nerve and plexus lesions

Part two: functional aspects

Abstract

The effect of mechanical tension on median nerve function was evaluated with the latencies of somato sensory evoked potentials as an outcome variable. In healthy volunteers (n=19), two median nerve upper limb tension tests were used to stretch the median nerve. Due to the test positions, the evoked potential latencies were significantly enlarged. The findings confirm the conclusions from previous *in vitro* studies, and justify the use of median nerve upper limb tension tests as clinical tools for generating tension in the median nerve.

Introduction

Nerve tension tests analogous to the straight leg raising test[1] have been proposed for the median, ulnar and radial nerves[2,3]. To analyse the validity of these nerve tension tests, tensile forces in the nerves of the upper extremity and in the brachial plexus have been measured in embalmed human bodies (Part one of this study). With mechanical tension in the nerves taken as an outcome variable, exclusively two median nerve upper limb tension tests (ULTT and ULTT+, table 1) could be considered valid[4]. However, direct extrapolation to the clinical situation was not possible. To relate the tensile force characteristics to nerve conduction function, we stretched the median nerve with the two median nerve ULTT and evaluated the effect by assessing somato sensory evoked potential (SSEP) latencies. SSEP latencies are directly related to nerve conduction velocity which in turn is linearly related to nerve elongation[5].

Methods

Subjects

The study included 19 healthy adult volunteers, 9 males and 10 females, 20-32 years of age (mean 25.7 3.3). Height of the subjects ranged from 159-193 cm (Mean 176.2 8.8). Excluded from the study were subjects with a history of a) systemic disease that could result in a neuropathy and/or b) disease of the central nervous system. Their initial SSEP latencies were within limits of a normal population used as a reference in the University Hospital Rotterdam, Dijkzigt.

Procedure

Test positions and sequence are described in table 1 (see also figure 2 A in ref. 5). Test positions in both left and right arm were assumed gradually and in painfree steps, starting with the shoulder joint and ending with the finger joints. Firstly the right arm and secondly the left arm was measured. Each completed test position was held for about eighty seconds, the duration of the SSEP's averaging. There was a one minute interval between the test positions. According to the procedure suggested by Elvey et al.[2,3], firstly the ULTT position was assumed and secondly contralateral rotation and lateral bend of the cervical spine was added (ULTT+). Since the experimenters did not want to exceed any limits of pain, the maximal joint positions were to a certain extent defined by the testpersons. The maximal joint angles used in the present study never exceeded the strictly defined joint positions of the *in vitro* study[4].

Table 1. Test positions

Test position 1: neutral position (N1)

Shoulder	Elbow	Wrist	Fingers
0%	0%	0%	0% (neutral)

(cervical spine in neutral position)

Test position 2: median nerve stretch test (ULTT)

Shoulder	Elbow	Wrist	Fingers
max. abduction max. exorotation max. retroflexion	(0% extension) max. supination	maximal dorsal flexion	0% (neutral)

(cervical spine in neutral position)

Test position 3: median nerve stretch test with cervical modification (ULTT+)

Cervical spine	Shoulder	Elbow	Wrist	Fingers
contralateral lateral bend + contralateral rotation	max. abduction max. exorotation max. retroflexion	0% (extension) max. supination	max. dorsal flexion	0% (neutral)

Test position 4: resumed neutral position (N2) (see test position 1)

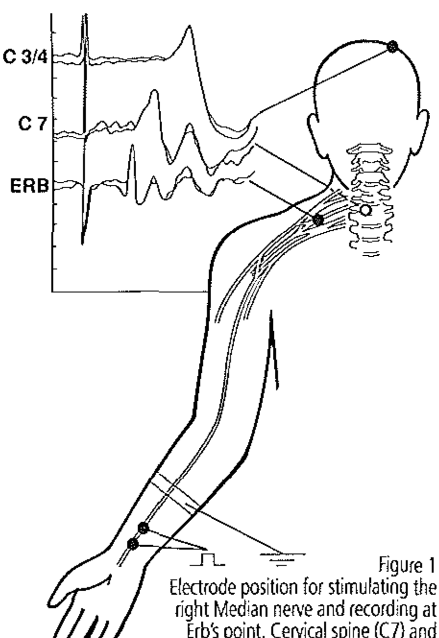

Figure 1
Electrode position for stimulating the right Median nerve and recording at Erb's point, Cervical spine (C7) and Cortex (C3) (For recordings of the left Median nerve C4 was taken)

Electrodiagnostic methods

For all tests, stimuli (square wave pulse, 0.1 ms duration) were delivered from a modular EMG system (Viking II, Nicolette). A bipolar stimulation unit was placed over the Median nerve at the wrist, between the styloid processes of the radius and ulna. The recordings of two hundred stimuli (stimulus frequency: 3.1 Hz, intensity: twice the motor treshold) were averaged. AG-AgCl surface disc recording electrodes with a diameter of 10 mm were used. The cortical SSEP's were recorded over the contralateral somato sensory cortex ('hand field'); the active electrodes were placed at C(Central)3 for the left hemisphere and C4 for the right. The reference electrode was placed at Fz (Frontal zero). Spinal SSEP's were recorded over the cervical spine. The electrode was placed at the spinous process of the seventh cervical vertebra (C7). The electrodes for brachial plexus SSEP's were situated left and right at Erb's point[6] in the supra clavicular fossa (figure1).

Upper limb tension tests as tools in the diagnosis of nerve and plexus lesions, part two The maximum impedance level was 5 kOhm, all electrodes having similar impedances. Prior to display, A/D conversion and storage, all signals were amplified (bandwith 5 Hz-1,5kHz).

Statistical analysis

Paired *t* -tests were used to determine whether the average values of SSEP latencies were altered by the ULTT and ULTT+ maneuvers when compared to the Neutral 1 position. Furthermore a comparison was made between ULTT and ULTT+ and between Neutral 1 and Neutral 2 position.

Results

In both the ULTT and ULTT+ position, all SSEP latencies recorded at Erb's point, C7 and C3/4 were significantly increased when compared to the Neutral1 position (table 2).

Table 2. The effect of ULTT and ULTT+ 1 on median nerve SSEP latencies. Standard deviations are given in parentheses

	A. Neutral1 (N1)	B. ULTT	C. ULTT+	D. Neutral2 (N2)	pA-B	pA-C	pB-C	pA-D
Erb								
R	9.90(0.62)	10.95(0.68)	10.90(0.55)	10.06(0.56)	<0.001	<0.001	n.s.2	<0.001
L	9.79(0.61)	10.73(0.81)	11.23(1.57)	10.03(0.56)	<0.001	<0.001	n.s.	<0.01
C7								
R	13.75(1.09)	14.28(0.92)	14.87(1.56)	13.82(0.78)	<0.01	<0.001	<0.05	n.s.
L	13.57(1.11)	14.59(1.69)	14.87(2.03)	13.62(0.98)	<0.05	<0.01	n.s.	n.s.
C3/4								
C3	19.27(1.03)	19.99(1.21)	20.22(1.19)	19.45(0.90)	<0.001	<0.001	n.s.	n.s.
C4	19.28(1.03)	20.43(1.80)	20.59(2.04)	19.52(0.91)	<0.05	<0.05	n.s.	<0.01

[1] For joint positions of ULTT and ULTT+ see table 1
[2] n.s. = not statistically significant

Apart from one exception, latencies due to the ULTT+ tended to be higher then those due to the ULTT (see table 2). Only in the case of the C7 recording for the right arm, statistical significancy was reached. At three sites the Neutral 2 values tended to be higher than Neutral 1 values. In both recordings at Erb's point (right and left arm) and at the C4 recording (= left median nerve: right somato sensory cortex) the differences were statistically significant.

Discussion

To know whether indeed a nerve tension test causes tension in a nerve, tension measurements should be performed *in vivo*. However, in practice useful methods are not available. Indirect methods have to be employed such as measuring tension *post mortem*. Such *in vitro* studies can answer two questions. Firstly, does a specific test position cause tension in the intended nerve? Secondly, does this test position cause tension *exclusively* in the nerve it is intended for? In previous *in vitro* studies we showed the answers to be affirmative for the Median nerve ULTT and ULTT+ but not for the ulnar and radial nerve ULTT and ULTT+[4,7,8]. However, the question could not be ans-

wered whether these tensile forces occur *in vivo* and if so, to what extent. Based on subjective data ('pins and needles', pain, 'numb feeling') patients can provide information on nerve stretch. However, one has to be careful with the interpretation since about half of the patients with a positive straight leg raising test show no objective signs of a nerve root entrapment[9]. A more reliable impression of nerve stretch can be obtained by using nerve conduction parameters[10,11,12].
In an animal model a linear relation between nerve elongation and decrease of conduction velocity has been confirmed[5]. In Part one of this study we showed that the median nerve ULTT and ULTT+ can selectively stretch the median nerve. The present study shows that performing the median nerve tension tests (ULTT and ULTT+) median nerve SSEP latencies significantly increase (i.e. conduction velocity decreases). This holds for all three recording sites.

In our study the highest increase in averaged latencies is seen when the median nerve ULTT+ is compared with the initial neutral position. At Erb's point the increase in SSEP latencies is 10%, whereas at Cervical 7 and Central 3/4 the increase is eight and five percent respectively. This indicates that due to the ULTT+ the highest decrease in conduction velocity occurs in the peripheral part of the median nerve.

Decreased conduction velocity
The observed increase of SSEP latencies due to the tests can be caused by three phenomena: 1) increase in nerve length, 2) diminished neural circulation and 3) changes in fiber diameter.

Re 1) Due to either the ULTT or the ULTT+ we found a 2,5 cm lengthening of the Median nerve with respect to a bony landmark at the distal part of the radius (unpublished data of *in vitro* experiments on unembalmed human bodies). With an average length of the arm of 72 cm (from the carpal ligament to the nerve root), this amounts to a lengthening of 3.5%. This is in accordance with the 4% stretch found by Zöch et al[13]. We found a 10% increase of SSEP latencies at Erb's point. Consequently, this increase cannot be attributed exclusively to lengthening of the nerve fibers.

Re 2) Stretching the nerve will lead to a decrease in vessel diameter and to a decrease in blood flow[14,15]. Since it has been shown that nerve function is intimately related to its circulation[16,17] the increased latencies during the test positions can be at least in part attributed to diminished bloodflow.

Re 3) For myelinated fibers a linear relationship exists between fiber diameter and conduction velocity[18]. Lengthening of the tubelike nerve fibers leads to a decrease in fiber diameter ('thinning out') and consequently to a decrease in nerve conduction velocity. According to v.d. Wey et al[12], this third possibility is the most probable explanation for the decrease in conduction velocity found due to nerve lengthening.

Restoration of normal conduction velocity

One minute after finishing the last test position (N 2 position, table 2), the latencies at Erb's point (left and right) and at C4 are still significantly higher when compared with baseline values. At C7 (left and right) and at C3 they tend to be increased. Considering the three explanatory mechanisms mentioned above, the most satisfactory explanation is that changes in the nerve fibers form the basis for the persisting increased latencies after resuming the neutral position again. After all it can be expected that in the N2 position, the nerve has assumed its original length. Therefore the persisting increase of latencies can hardly be due to nerve lengthening between the N1 and N2 position. Although nerve function rapidly deteriorates as a result of ischaemia, it recovers rapidly with restoration of blood flow[16,17]. A circulato ry deficit as the cause of the persisting increased latencies in the N2 position is also not likely.

The standard deviations of the latencies in the neutral (N1) position tended to be smaller than those in the ULTT and ULTT+ position (Neutral, range: 0,56-1,11 vs ULTT(+), range: 0,55-2,04). Probably this larger variability during the test positions is due to the relatively uncomfortable median nerve ULTT and ULTT+ test positions (see: Methods: Procedure). This holds especially for the ULTT+; about 80% of the subjects reported that the ULTT+ position was far less comfortable (paraesthesia and/or hypaesthesia) than the ULTT position.

Conclusions

Out of six tests described as specific nerve tension tests for the median, ulnar and radial nerve, exclusively the median nerve tests were shown to be valid tests. One has to be very cautious with the interpretation of results obtained with the ulnar and radial nerve ULTT and ULTT+. This was shown in an *in vitro* study, with tensile force as an outcome variable (Part one of this study). Part two of this study showed that *in vivo* the median nerve tests cause an increase in SSEP latencies (a parame ter indicative for nerve elongation). The results of both studies justify the clinical use of the median nerve ULTT and ULTT+ to stretch the median nerve and related parts of the brachial plexus.

Whether upper limb tension tests can be used as tests for selectively stretching the related nerve roots has seriously to be questioned.

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Chapter 6

Lowered motor conduction velocity of the peroneal nerve after inversion trauma

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Lowered motor conduction velocity of the peroneal nerve after inversion trauma

Abstract
To analyze the effect of inversion trauma on peroneal nerve function, motor conduction velocity was measured in twenty-two patients. In the injured leg, four to eight days post trauma motor nerve conduction velocity in the knee-caput fibulae segment of the superficial peroneal nerve was significantly smaller when compared to the contralate ral leg and the control group. Five weeks post trauma these values were normal again. For three segments of the deep peroneal nerve, the motor conduction velocity was significantly reduced, four to eight days post trauma, when compared to the control group. In the caput-ankle and knee-ankle segment, motor conduction velocity was still significantly lowered five weeks post trauma. Lowered amplitudes of the Compound Motor Action Potentials of the extensor digitorum brevis muscle were found four to eight days posttrauma. No correlation was found between motor nerve conduction velocities and subjective clinical tests (anterior drawer sign and (manually performed) talar tilt test). The results of this study support the hypothesis that inversion trauma is frequently accompanied by lesions of the peroneal nerve. Motor conduction velocity measurements can be a valuable tool in assessing more objectively functional instability of the ankle joint induced by inversion trauma.

Keywords: ankle injuries, neural conduction, reflexes, joint instability

Introduction
Inversion trauma is a very common injury to the ankle. In Finland, 7128 hospital admissions due to ankle trauma were reported, together forming 9.4% of all trauma admissions in 1985[26]. Among Dutch sports participants (N= 7909), 12.5 % of the injuries in 1985 concerned inversion trauma[7]. In The Netherlands most of these patients receive a successful non-operative treatment at a first-aid department or by a general practitioner. About one third of these patients experience a sensation of 'giving way' and instability in the injured leg, which is considered a result of torn capsule and ligaments of the lateral side of the ankle[3,9,25]. In stabilizing the ankle, joint stabilizing reflexes play an important role. They require intact superficial and deep peroneal nerves besides intact ligaments. After inversion trauma, peroneal nerve *palsy*, does indeed incidentally occur[10,18,22,27,2]. However, these reports were on a case presentation basis and detailed nerve conduction studies were done only in the single-case study of Streib[27]. The present study focuses on the short-term effects of inversion trauma (without clinical signs of peroneal nerve palsy) on motor nerve conduction velocity of the superficial and deep peroneal nerve. The purpose is to contribute to a better understanding of persistent instability of the ankle and, in addition to the subjective clinical evaluation of joint stability, create an objective way of quantifying the effects of inversion trauma. Lowered motor conduction velocity of the peroneal nerve after inversion trauma

Methods

Subjects

The study included 22 patients, 18 men and 4 women, 17-45 years of age (mean 25.4±6.0). After inversion trauma of the ankle all patients attended the Department of Traumatology of the University Hospital Rotterdam, Dijkzigt. To eliminate patients with fractures from the investigation, A-P and lateral radiographs of the ankle were obtained. Included were 7 patients with grade I inversion trauma, 3 with grade II and 12 with grade III (see next paragraph). Excluded from the study were patients with *a*) systemic disease that could result in a neuropathy and/or *b*) fracture of leg or foot and/or *c*) disease of the central nervous system.

Although it is common practice to compare the injured leg with the contralateral leg, in the case of ankle joint lesions this can be misleading. Unilateral injury of the lateral ankle ligaments can lead to functional alterations at both ipsilateral and contralateral side. There is a risk of underestimating the impairment of the injured joint[8]. Therefore the injured legs were compared to both the contralateral leg of the patients, and the ipsilateral legs (matched with the injured subjects) of 28 healthy volunteers who never suffered inversion trauma: 10 men and 18 women, 19-37 years of age, mean age 26.3±3.7.

Methods

In patients with the diagnosis: 'Isolated inversion trauma Grade I, II or III', a bandage was applied. The grading system is based on clinical testing. Stability in the sagittal plane was tested by the anterior drawer sign test (ADS). In this test the patient is in the supine position and the examiner fixates the tibia with one hand and translates the calcaneus forward. The amount of 'joint play' is interpreted subjectively and the decision normal/ abnormal is made. In the frontal plane the stability of the ankle was (manually) tested by the talar tilt test (T.T.). In this test the tibia is fixated and the calcaneus is moved medially and laterally. The amount of tilting of the talus is interpreted subjectively and judged normal/abnormal. Grade I: ADS negative, TT negative; Grade II: ADS , TT ; Grade III: ADS positive, TT positive. The patients were asked to give their written informed consent and to attend a special 'ankle consultancy' at the Department of Traumatology.

In the four to eight day interval between first visit and 'ankle consultancy', patients had to take as much rest as possible and refrain from loading the ankle. At the consultancy the patients were reexamined. Only patients assigned twice the same clinical grade of inversion trauma by two independent observers, were asked to participate in this study and to give their informed consent.

The effects of inversion trauma on the motor nerve conduction velocity of the peroneal nerve were evaluated by means of a prospective study with a consecutive series. Four to eight days post trauma, conduction velocity was measured (Recording I) and a semi rigid bandage was applied. Subsequent measurements were performed eighteen to twenty-two days (Recording II) and thirty-two to thirty-six days (Recording III) after trauma. For this purpose the bandage was removed and newly applied after the measurements. Identical measurements were performed on the unaffected (contralateral) leg. The peroneal nerve was stimulated at the knee, caput fibulae and at the ankle (figures 1 and 2). Nerve stimulation was performed with a square wave pulse of 0.3 ms duration and a voltage up to 300 V. The voltage was incrementally increased. Recordings were made using a storage oscilloscope and all measurements were printed on paper. At each recording site 4-5 trials were administered and

KCMVP **SUPERFICIAL PERONEAL NERVE**

Knee-Caput Motor Velocity Peroneus longus for DML values

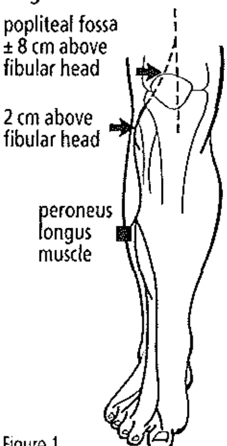

Figure 1
Superficial peroneal nerve: stimulation and recording sites (DML= distal motor latency)

stimulation site
recording site

the fastest recorded velocity was accepted as the criterion measure. Utmost care was taken to stimulate nerves supramaximally and to search for the maximal Compound Motor Action Potential above the muscle belly. A bipolar stimulation unit and Ag-AgCl surface disk recording electrodes with a diameter of 10 mm were used. To evaluate the effects of inversion trauma on the motor conduction velocity of the superficial and deep peroneal nerve, one (knee-caput) and three (caput-ankle, knee-ankle, knee-caput) segments were measured respectively (figures 1 and 2).
To evaluate the effects on the EMG waveform, peak to peak values of the amplitudes of the Compound Motor Action Potentials (CMAP) of the three muscles used for Distal Motor Latencies (extensor digitorum brevis, tibialis anterior and peroneus longus muscle) were analyzed.

DEEP PERONEAL NERVE

CAMVE
Caput-Ankle Motor Velocity Extensor digitorum for DML values

KAMVE
Knee-Ankle Motor Velocity Extensor digitorum brevis for DML values

KCMVT
Knee-Caput Motor Velocity Tibialis anterior for DML values

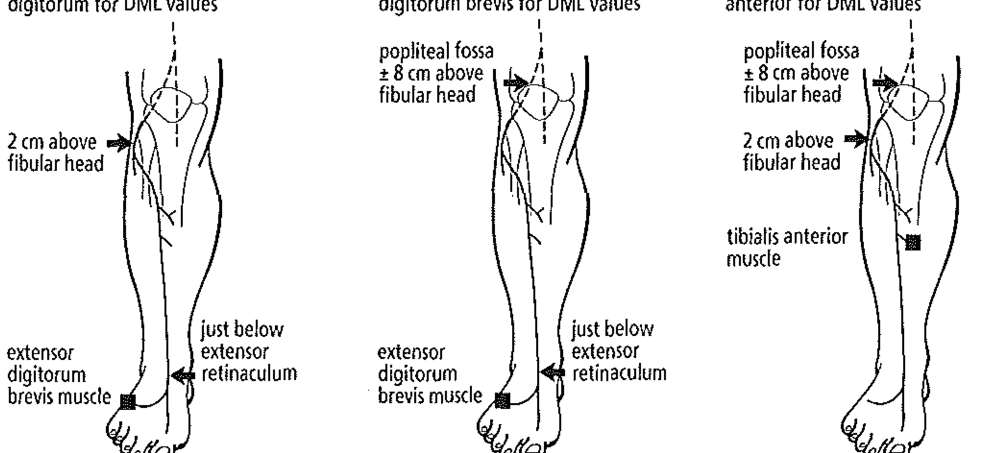

Figure 2 Deep peroneal nerve: stimulation and recording sites

The recordings in the control group were also taken with a two week interval. All measurements were performed under the same circumstances including room temperature and humidity.

Statistical analysis
For hypothesis testing a repeated measures analysis of variance (rm ANOVA) was performed. To test the differences between Recordings I, II and III, Students T-tests were used and analyses on linear trends were performed. Correlation between electrophysiological findings and clinical grading was tested by means of Spearmans rank correlation test.

Results

Superficial peroneal nerve.
Four to eight days post trauma the motor nerve conduction velocity of the superficial peroneal nerve in the injured leg is significantly reduced when compared to both contralateral leg and the *control group* values (table 1, recording I). In the control group no differences are observed between right and left leg nor between men and women; motor nerve conduction velocity values are comparable to previously reported values 21, 23. Recordings II and III (eighteen to twenty-two and thirty-two to thirty-six days post trauma) do not show significant differences (table 1).

Table 1. Superficial peroneal nerve

	Recording I (4-8 days post trauma)	Recording II (18-22 days post trauma)	Recording III (32-36 days post trauma)	Linear Trend in Recording II and III
KCMVP				
Injured	52.0 (6.3)	55.0 (10.2)	58.3 (8.9) c	Yes (p<0.05)
Contralateral leg	60.8 (8.8) a	58.0 (10.3)	60.8 (9.8)	No
Control group	60.5 (7.0) a	63.0 (10.3) b	61.0 (9.7)	No

KNMVP = knee-caput fibulae motor velocity; peroneus longus muscle used for distal motor latency (DML) values
a Compared with injured leg; P < 0.001. b Compared with injured leg; P < 0.05. c Compared with recording I; P < 0.01.

In comparing the motor nerve conduction velocity values of the injured leg on recordings I, II and III, the recording at thirty-two to thirty-six days post trauma shows a significant increase when compared with the recording four to eight days post trauma. The motor nerve conduction velocities of the three recordings show a linear trend (table 1). but the value of recording II does not differ significantly from the first recording.

For the contralateral leg differences between recordings I-II, I-III and II-III are not significant and no linear trend is observed. This also holds for the control group. When the superficial peroneal nerve was stimulated at the fibular head and the knee, the amplitude of the CMAP of the peroneus longus muscle was not significantly lower when compared to both the contralateral leg and control group.

Deep peroneal nerve.

For all recordings on the deep peroneal nerve, no differences in motor nerve conduction velocity occur between injured leg and *contralateral* leg (table 2). However, at recording I, all values of the injured leg are significantly lower than control group values; *control group* values being comparable to previously reported values [21,23].

For the proximal segment (knee-caput: KCMVT), eighteen to twenty-two days posttrauma (recording II), a difference in motor nerve conduction velocity between *injured* leg and *control* group is present. Thirty-two to thirty-six days posttrauma no difference was observed. For the distal segments (caput-ankle and knee-ankle: CAMVE and KAMVE) still significant differences between *injured* leg and *control* group values occur thirty-two to thirty-six days post trauma (recording III).

Four to eight days post trauma (recording I) the motor nerve conduction velocity of the *contralateral* leg in the distal segments (CAMVE and KAMVE) is significantly lower than that in the control group. These lower velocities are also present eighteen to twenty-two days post trauma (recording II) and even thirty-two to thirty-six days post trauma.

For the proximal segment (KCMVT) no statistically signifcant differences between contralateral leg and control group are observed. For the injured leg at CAMVE and KAMVE there is a significant increase in motor condution velocity in recording III, when compared with recording I. For the KAMVE segment there is an increase in recording II when compared to recording I. For both CAMVE and KAMVE there is linear trend when comparing the values of Reording I, II and III. For KAMVE this trend is stronger then for CAMVE ($p= 0.001$ vs $p= 0.019$). For KCMVT no such differences and no linear trend were observed. For the contralateral leg and the control group, no significant differences occur between recordings I, II and III and no linear trend was found.

Table 2. Deep peroneal nerve: mean motor nerve conduction velocities and standard deviations (m5^{1}) in the segments caput fibulae-ankle, knee-ankle, and knee-caput fibulae

Recording I	Recording II	Recording III	Linear Trend	
CAMVE				
Injured	44.6±3.0	45.7±3.4	46.6±0.0	Yes (p<0.05)
Contralateral leg	45.5±3.3	45.5±3.6	46.3±3.5	No
Control group	48.0±2.5 [a]*	49.6±3.6 [a]*	49.1±4.0 [b]•	No
KAMVE				
Injured	44.5±2.4	46.0±3.0	46.4±2.9 •	Yes (p<0.05)
Contralateral leg	45.7±3.1	45.7±3.6	46.4±2.6	No
Control group	49.0±2.8 [a]*	50.0±4.3 [a]*	49.29±4.2 [b]♦	
KCMVT				
Injured	50.1±9.2	52.5±10.5	55.1±10.0	No
Contralateral leg	55.5±8.5	55.3±9.9	56.5±8.2	No
Control group	57.0±11.8 [c]	60.4±12.0 [c]	55.3±9.2	No

CAMVE= caput fibulae-ankle motor velocity; extensor digitorum brevis used for DML values
KAMVE = knee-ankle motor velocity; extensor digitorum brevis used for DML values
KCMVT = knee-caput fibulae motor velocity; tibialis anterior used for DML values
[a] Compared with injured leg; P < 0.001; * Compared with contralateral leg: P < 0.001.
[b] Compared with injured leg; P < 0.01; • Compared with contralateral leg: P < 0.01.
[c] Compared with injured leg; P < 0.05; ♦ Compared with contralateral leg: P < 0.05.
✧ Compared with recording I: P < 0.01.
▲ Compared with recording I: P < 0.05.

While stimulating the deep peroneal nerve at the ankle, the fibular head and the knee, four to eight days posttrauma, the CMAP amplitudes of the extensor digitorum brevis (EDB) muscle were recorded. The amplitudes of the injured legs were significantly low ered when compared to the contralateral legs and control group (Ankle: resp. p=0.004 and 0.000; Fibular head: resp. p=0.037 and 0.028 and Knee: resp. p=0.045 and 0.031) When stimulated at the ankle, eighteen to twenty-two days posttrauma, the CMAP amplitudes of the EDB of the injured legs were still lowered when compared with the control group (p= 0.024) but not when compared to the contralateral legs (p=0.075). When stimulated at the fibular head and the knee, no differences with the contralateral legs and control group was observed. Thirty-two to thirty-six days posttrauma, CMAP amlpitudes of the EDB of the injured legs were similar to those of the contralateral leg and control group.
The CMAP amplitudes of the tibialis anterior muscles of the injured legs was not lowered when compared to those of the contralateral legs and control group.
To evaluate the correlation between motor nerve conduction velocity and clinical grading, the mean differences between injured leg and contralateral leg of the four motor nerve conduction velocity parameters were compared (table 3). Obviously there is no c orrelation.

Table 3. Correlation between electrophysiological findings and clinical grading

Grade I	Grade II	Grade III	r and P values	
CAMVE	-0.44 (2.7)	3.00 (1.0)	0.42 (3.5)	r 0.25 P 0.14
KAMVE	0.22 (2.7)	0.67 (0.6)	1.17 (3.5)	r 0.20 P 0.18
KCMVT	3.56 (11.1)	6.67 (14.8)	2.54 (13.4)	r -0.06 P 0.38
KCMVP	5.56 (10.8)	14.00 (3.0)	9.77 (9.9)	r 0.16 P 0.22

Mean differences and standard deviations between injured leg and contralateral leg were taken as a parameter to analyze the correlation between electrophysiological findings and clinical grading. Correlation coefficients and corresponding P-values were calculated with Spearmans rank correlation test

Discussion

Since Cohen proposed the 'arthro-kinetic reflex' as the mechanism for stabilizing joints[1], several authors accepted ankle stability to be dependent on an intact reflex mechanism[4,5,6,12]. These authors suggest that 'functional instability' is predominantly caused by injured joint receptors (disturbed propriocepsis). However, the possibility of lowered nerve conduction velocities as an explanation for functional instability is not mentioned. In our view this can be an additional factor which for instance can explain the prolonged peroneal muscle reaction time found by Konradsen and Ravn[12].

In the present study motor nerve conduction velocity is taken as a parameter to analyze a supposed peroneal nerve lesion. The motor nerve conduction velocity of the superficial and deep peroneal nerve turned out to be lower after inversion trauma. In our opinion this is due to a traction lesion.

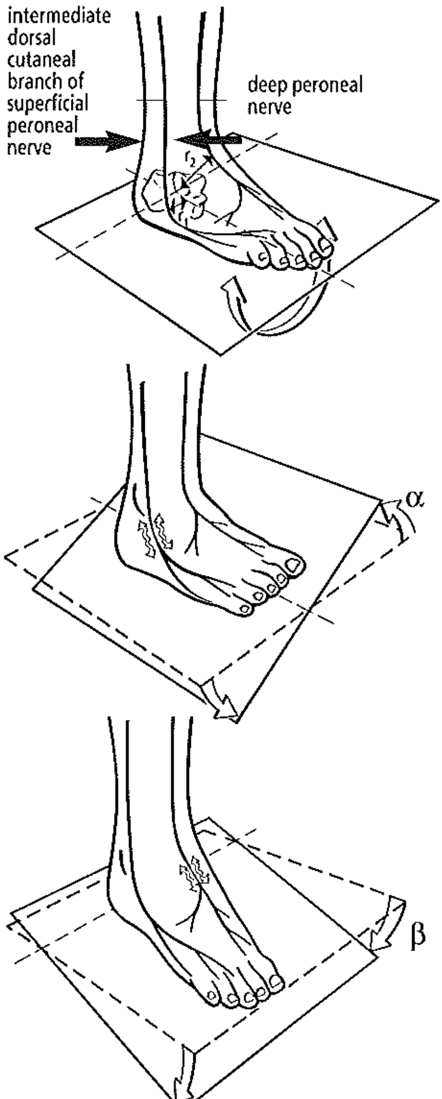

Figure 3 Location of the superficial and deep peroneal nerve and the displacement during normal inversion. The displacement can be seen as a part of a circle. According to the equation: $S = \alpha * r$, with $\alpha = 45°$ (0,8 rad.) and $r_1 =$ 4 cm in normal inversion, a 3-cm displacement of the intermediate dorsal cutaneal branch of the superficial peroneal nerve has to be compensated for in normal circumstances. For the branches of the deep peroneal nerve $\beta = 45°$ and $r_2 = 5$ cm, a 4-cm displacement has to be compensated for.

Because of the relative lateral position on the dorsum of the foot, displacement of the superficial peroneal nerve at the level of the ankle occurs especially by the supination component of inversion movement (figure 3). In normal inversion this displacement is about three centimeter. Displacement of the deep peroneal nerve is mainly due to the plantar flexion component of inversion (figure 3). In normal inversion this displacement is about four centimeters.

In inversion, damage to the peroneal nerve will normally not occur because of a variety of compensating mechanisms[29,28,17,19]. However, during inversion trauma extensive displacement can take place. Stretching a peripheral nerve beyond a certain percentage of *in vivo* length, leads to acute and long-term deficiencies in intraneural circulation and thus to alterations in the conduction properties[28,15,16]. Irreversible functional deficit has been shown after strain of more than 12 percent of the in situ strain of rabbit tibial nerve[14]. The question arises whether the inversion induced lowering of the motor conduction velocity found in the present study can be due to stretching the superficial and deep peroneal nerve beyond 'critical' values. In this respect four factors should be considered. Firstly the length of the nerve segment over which the elongation has to be compensated for. Assuming the total length of the peroneal nerve to be 100 centimeters from plexus to the end of the intermediate cutaneous nerve, a three centimeter displacement of the nerve at the lateral side of the foot results in an elongation of three percent. However, the peroneal nerve has an awkward position just distal to the caput fibulae. Here the nerve enters a musculotendinous arch in the peroneus longus muscle. In six out of fifteen anatomical preparations we found a strongly diminished gliding mechanism whereas in three this mechanism was not present at all (unpublished data). In the latter case the total length over which the elongation has to be compensated for will be about fifty centimeters, enlarging the elongation to six percent in normal inversion. Furthermore, where the nerve pierces the crural fascia an analogous entrapment can be expected[13]. With a fixation of the nerve at this site the elongation has to be compensated over about twenty centimeters; an elongation of fifteen percent is already in the critical area.

Secondly the total amount of displacement in the foot is of importance. When the entire body weight is exerted to the inverted ankle, a larger inversion angle and consequently a larger nerve displacement can be expected.

A third factor is the velocity of the traction forces. In daily life, marked dysfunction is not likely to occur, because the traction force will be exerted gradually and slowly. In inversion trauma an abrupt, fast traction will be exerted on the nerve. Such an accelerated force can be damaging to the nerve[20,24].

The last factor which has to be taken into account is the simultaneity of traction and compression forces. In moving the foot medially (adduction component of inversion), the fibular head becomes a pulley to the peroneal nerve. In rounding this pulley, an additional *transverse* force is exerted resulting in compression of the nerve. Actually the nerve is double crushed.

From the present study the question arises whether lowered motor nerve conduction velocity after inversion trauma can recover and if so at what speed?
At Recording II the motor nerve conduction velocity of the *superficial* peroneal nerve of the injured leg, is not significantly different from Recording I. However the conduction velocity is significantly smaller when compared to normal (control group) values. Thirty-two to thirty-six days post-trauma the motor nerve conduction velocity is not different from normal (control group) values; obviously recovery did occur (table 1).
In the peroneus longus muscle no lowered amplitudes of the CMAP are observed. It can be concluded that no alterations in the relationship between nerve conduction and muscle action have taken place.

For the *deep* peroneal nerve, differences in recovery time are found between the proximal segment (knee-caput: KCMVT) and the distal segments (caput-ankle: CAMVE and knee-ankle: KAMVE) and between CAMVE and KAMVE (table 2). In the KCMVT segment still a difference with control group values exists three weeks posttrauma (Recording II) but not after five weeks (Recording III). Since there is no linear trend to recovery in this respect there is some inconsistency in the recovery of the KCMVT segment. It should be noted that determining impulse conduction velocity in the (small) knee-caput segment is not very easy; inaccuracies in measurement have a relatively large influence. Consequently, both KCMVP and KCMVT values have rather large standard deviations when compared with the CAMVE and KAMVE values (table 1 and 2).

In both CAMVE and KAMVE a linear trend for recovery is found although at recording III (about five weeks post trauma) recovery to normal values is not complete.
Although no definite conclusions can be drawn about the cause of this difference in recovery time, besides inaccuracies in measurements, two mechanical explanations can be proposed: *a*) larger trac-

tion forces applied to the distal nerve segment and *b*) swelling in the ankle.
ad a) Distal to the extensor retinaculum, the motor branch to the extensor digitorum brevis muscle sharply deviates to the lateral side of the foot. In forceful inversion a distraction of this part of the deep peroneal nerve can take place between the extensor retinaculum and extensor digitorum brevis.

ad b) The motor branch to the extensor digitorum brevis muscle is located in the region where, due to the lesion, swelling occurs. A maintained compression on the distal part of the nerve (which already endured a traction lesion) could be the cause of the slower recovery of the distal segments of the peroneal nerve.

Four to eight days post trauma, the motor nerve conduction velocity values of the *contralateral* leg are significantly lower than those of the *control* group (table 2). Two possible explanations for this remarkable phenomenon arise. Firstly, the inversion trauma also affects the deep peroneal nerve of the contralateral leg. This possibility is supported by the findings of Gauffin[8] and by the tendency of motor nerve conduction velocity values to increase in the contralateral leg during the five week period of this study (table 2). Secondly, the patients are characterized by a pre-existent low motor nerve conduction velocity of the deep peroneal nerve in both legs, predisposing them for inversion trauma. To investigate this possibility a prospective study in 130 volunteering soldiers of the Royal Dutch Infantry is now performed.

In the injured leg a lowered amplitude of the CMAP was found in the recordings of the deep peroneal nerve in which the extensor digitorum brevis was used for distal motor latencies. No alterations in amplitude of the CMAP of the tibialis anterior muscle were found. These findings suggest a lesion in the extensor digitorum brevis muscle and not in the tibialis anterior muscle. This would be in agreement with the observations of Jennekens et al.[11] showing evidence of frequent denervation and slow reinnervation of the extensor digitorum brevis muscle, but not of the tibialis anterior muscle. However, it has to be born in mind that it cannot be excluded that the observed low amplitudes of the extensor digitorum brevis observed in the present study could be due to swelling over the extensor digitorum brevis; in the area in which the CMAP of the tibialis anterior muscle was recorded no such swelling occurs.

In this study there appeared to be no relation between the clinical evaluation of ankle instability and motor nerve conduction velocity of the peroneal nerve, being an important part of the ankle joint stabilizing reflex and hence of functional stability. This is in accordance with the clinically often encountered difference in clinically 'measured' joint stability and the functional stability as reported by patients.

Conclusions

The results of this study support the hypothesis of an injury to the peroneal nerve induced by inversion trauma. In the concept of the joint stabilizing 'arthro-kinetic' reflexes such a deficiency can jeopardize the functional stability of the ankle joint. When no adequate measures are taken, normal loading patterns (walking, jumping etc.) can already be hazardous to the joint, leading to secondary inversion trauma and eventually to chronic instability of the ankle joint.
No relation was found between the subjective clinical grading and electrophysiological findings. Motor nerve conduction velocity measurements, performed with surface electrodes, mean practically no extra inconvenience to the patients, do not take a lot of time and can be performed within four to eight days post trauma. These advantages can make motor nerve conduction velocity measurements (in addition to mechanical testing) an important tool in the early assessment and therapy of *functional instability* of the ankle joint.

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29 Sunderland, S. and K.C. Bradley. Stress strain phenomena in denervated peripheral nerve trunks. Brain 84: 125-127, 1961 Lowered motor conduction velocity of the peroneal nerve after inversion trauma

Chapter 7

Inversion trauma as a cause of lowered motor nerve conduction velocity of the peroneal nerve

A prospective longitudinal study

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Submitted for publication

Inversion trauma as a cause of lowered motor nerve conduction velocity of the peroneal nerve

A prospective longitudinal study

Abstract

To analyse whether inversion trauma lowers the motor nerve conduction velocity of the peroneal nerve, a prospective study was performed on 120 recruits of the Dutch Royal Army. The motor nerve conduction velocity of the deep and superficial peroneal nerve was assessed upon entering military service (baseline values) and four to eight, 18-22 and 32-36 days after inversion trauma if it occurred. Nine recruits suffered inversion trauma within 3 months follow up of whom 7 had a known post trauma follow up. Twenty five recruits were taken as controls. Four to eight days post trauma motor nerve conduction velocity of the deep peroneal nerve was significantly lowered when compared with baseline values. This holds for both the knee-ankle and caput fibulae-ankle segments of the deep peroneal nerve. Baseline values of recruits who suffered two or more inversion traumas in the past (N=9) were significantly lower than those of recruits who had suffered no or one inversion trauma (N=111). A causal relationship is shown between inversion trauma and deep peroneal nerve function deficit. The observations support the clinical importance of peroneal nerve function in ankle joint stabilizing reflexes.

Key words: Ankle injuries, nerve lesions, joint instability

Introduction

Inversion trauma of the ankle joint forms a quantitatively and qualitatively large problem in health care. In the Netherlands, the estimated number of injuries of the ankle in one year was 630.000 of which 310.000 were medically treated[1]. On average there is a 2.9 days interruption of work[2]. Furthermore, after initial treatment, about 30% of the patients continues to have feelings of 'giving way' and instability[3,4].

Generally the cause of sustained instability is considered to be torn capsule and ligaments at the lateral side of the ankle. However, another cause for persistent joint instability might be deficiency in muscular reflex activity[5-8].

Apart from intact joint capsule and ligaments, other parts of the reflex chain (among which peroneal nerves, peroneal muscles and anterior tibial muscle) are essential for ankle joint stability[9,10]. Because of its anatomical course, especially the peroneal nerve is vulnerable to traction lesions during inversion trauma. After inversion trauma, patients showed a significantly lowered motor nerve conduction velocity of the peroneal nerve when compared to the contralateral leg and to a control group[11]. In that study pre injury values of motor nerve conduction velocity were not available. Therefore it could not be established whether the low motor nerve conduction velocity was pre existent or whether it was actually caused by inversion trauma. To study this relationship, we assessed the motor nerve conduction velocity of the peroneal nerve before and after trauma.

Patients and methods

Subjects

After they gave their written informed consent, motor nerve conduction velocity (mncv) of the deep and superficial peroneal nerve was measured in both legs of 120 healthy male recruits of the Dutch Royal Army, (18-26 years of age, mean 20.4 ±2.0). Measurements were performed within the first three weeks after entering military service. During the following three month training period, 9 recruits suffered an inversion trauma. Two of them were lost for post trauma follow up due to detachment in another army camp. Seven recruits (19-23 years of age, mean 20.8 ±1.7) were available for all post trauma measurements. Previously one had suffered from two inversion traumas, five from one inversion trauma. In all cases the last trauma occurred at least one year before baseline measurements. One recruit never suffered inversion trauma before. To exclude patients with fractures from the investigation, A-P and lateral radiographs of the ankle were taken in relevant cases.

Methods

The study concerned patients with ankle trauma who consulted the medical officer. During their first visit a bandage was applied to patients diagnosed to have: 'Isolated inversion trauma Grade I, II or III.' The grading system is based on clinical testing and has been described in detail in a previous publication[11]. The patients were asked to refrain from loading the ankle, to take as much rest as possible and to consult the medical officer again four to eight days later. Then the patients were reexamined. Patients were asked to participate in this study, provided the same clinical grade of inversion trauma was assigned by an independent observer. After giving their written informed consent mncv of the peroneal nerve was measured (Recording II) and a tape bandage was applied for ten days. Bandages in all patients were applied by the same experienced physiotherapist. After this period the tape bandage was removed and the recruits were assigned to light duties for another ten days. Recording III was performed four days after removal of the tape bandage, i.e. eighteen to twenty-two days post trauma. Recording IV was performed thirty-two to thirty-six days post trauma. All mncv measurements were also performed on the contralateral leg.

To evaluate the effects of inversion trauma on the mncv of the deep peroneal nerve mncv was measured in three segments. In the Caput-Ankle(CAE) and Knee-Ankle (KAE) segment the Extensor digitorum brevis was used for distal motor latency (DML) values and in the Knee-Caput fibulae(KCT) segment the Tibialis anterior muscle was used for DML values. For the effect on the superficial peroneal nerve, mncv in one segment was measured. In this Knee-Caput fibulae segment (KCP) the peroneus longus muscle was used for DML values. For a detailed description of the nerve stimulation and recording method see ref. 11.

Comparing the injured leg with the contralateral leg can be misleading[11,12]. Therefo re, the injured legs were compared to both the contralateral leg of the patients, and the matched legs of a control group. The control group consisted of a random sample of twenty-five healthy recruits (18-25 years of age, mean age 20.5 ±1.8). Their second recording occurred eighteen to twenty-two days after the first recording (i.e. in time comparable with recording III of the experimental group). Since in our previous study[11] no time effects were observed in healthy subjects, the control group values of the second recording were also used for comparison with the experimental group at recordings III and IV. All measurements were performed under the same conditions.

Statistical analysis
Paired and unpaired samples t-tests were used to analyse pre and post injury conditions, time effects and differences between injured, contralateral leg and control group.

Results

Injured leg
Four to eight days post trauma (Recording II) the mncv in the segments CAE and KAE of the deep peroneal nerve of the injured leg was significantly lowered when compared to the baseline values of the same leg (tables 1 and 2). In the KCT segment of the deep peroneal nerve and the KCP segment of the superficial peroneal nerve of the injured leg, the mncv was also lower after trauma, but not significantly so. In all four segments measured, the mncv of the deep peroneal nerve of the injured leg was lower four to eight days post trauma when compared with both contralateral leg and control group values. This is, however also not significant. This tendency is not apparent 18-22 and 32-36 days post trauma (tables 1 and 2).

Eighteen to 22 days post trauma (Recording III), mncv values in the injured leg tended to be higher than baseline values in the segments CAE and KAE of the deep peroneal nerve and in the KCP segment of the superficial peroneal nerve.

Thirty-two to 36 days post trauma (Recording IV),mncv values in the injured leg tended to be lower when compared with the values of recording III; they become comparable to baseline values.

Table 1. Superficial peroneal nerve: mean motor nerve conduction velocities (and SEM) in m.s-1 between knee-caput fibulae; Peroneal muscle for DML values

Superficial peroneal nerve	Recording I Baseline values	Recording II 4-8 days Posttrauma	Recording III 18-22 days Posttrauma	Recording IV 32-36 days Posttrauma
KCP Injured (n=7)	56.65 (7.2)	55.83 (9.8) *p1-2 : 0.78*	59.17 (8.8)	58.16 (5.9)
Contralateral LEG (n=7)	53.06 (11.9)	59.14 (6.4)	59.73 (5.2)	57.55 (3.6)
Control group (n=25)	53.00 (7.9)		60.43 (5.9)	

KCP = segment Knee-Caput fibulae; Peroneus longus muscle for DML values

Contralateral leg and controls

During the 36 day trial period, the mncv values tended to rise both in the contralateral leg of the patients and the legs of the control group. The mncv values of the contralateral leg of the patient tended to be lower than those of the control group.

Table 2. Deep peroneal nerve: mean motor nerve conduction velocities (and SEM) in m.s-1 between caput fibulae-ankle, knee-ankle and knee-caput fibulae

Deep peroneal nerve	Recording I Baseline values	Recording II 4-8 days Posttrauma	Recording III 18-22 days Posttrauma	Recording IV 32-36 days Posttrauma
CAE				
Injured (n=7)	48.98 (2.9)	47.18 (3.1) *p1-2 : 0.00*	49.43 (2.9)	48.56 (4.2)
Contralateral leg (n=7)	47.13 (4.2)	47.67 (3.4)	48.33 (2.3)	48.73 (3.2)
Control group (n=25)	47.78 (3.6)		48.99 (3.0)	
KAE				
Injured (n=7)	49.00 (2.7)	47.25 (3.3) *p1-2 : 0.03*	49.77 (3.1)	48.60 (4.3)
Contralateral leg (n=7)	47.13 (3.7)	48.11 (3.7)	48.63 (2.9)	48.65 (3.3)
Control group (n=25)	48.47 (5.0)		49.32 (3.4)	
KCT				
Injured (n=7)	55.78 (5.2)	53.00 (7.3) *p1-2 : 0.35*	53.18 (4.5)	59.52 (7.3)
Contralateral leg (n=7)	50.83 (2.5)	56.64 (7.8)	54.45 (4.4)	57.28 (4.1)
Control group (n=25)	53.55 (8.7)		58.96 (7.1)	

CAE = segment Caput fibulae- Ankle; Extensor digitorum brevis muscle for DML values
KAE = segment Knee-Ankle; Extensor digitorum brevis muscle for DML values
KCT = segment Knee-Caput fibulae; Tibialis anterior muscle for DML values

Baseline values

Baseline mncv values (including the pre injury values of the seven patients) were not different from previously reported values[11,13,14]. In the KAE and CAE segments of the deep peroneal nerve of 9 recruits with a history of two or more inversion trauma, mncv values were significantly lower than those of the 111 recruits who suffered one or no inversion trauma in the past(KAE: 45.3±2.4 vs. 48.1±4.0; p =0.010 and CAE: 45.13.5 vs. 48.0±3.5; p=0.027).

In all segments, pre injury values of the 'injured leg to be' of the seven patients tended to be higher when compared to the baseline values of the contralateral leg. In six of the patients the 'injured leg to be' was the leg of preference (defined as the leg they used for shooting a ball and for push off in jumping).

Discussion

In several studies on joint stability, the importance of joint stabilizing (arthro kinetic) reflexes has been emphasised[5-11]. Inadequate reflexes can lead to delay in peroneal muscle reaction time and therefore can lead to (chronic) functional instability of the ankle joint. In patients suffering from chronic ankle instability, Konradsen and Ravn suggested injury to joint receptors as cause of delayed peroneal muscle reaction time and chronic ankle instability[9]. However, the same authors could find no proof for this hypothesis and therefore suggested deficiencies in muscle receptor function to be the cause of delayed peroneal muscle reaction[15]. Considering the significantly lowered mncv of superficial and deep peroneal nerves after inversion trauma, found in a previous study we proposed a traction lesion of the peroneal nerve as cause of a delayed peroneal muscle reaction time[11].

In the present study, pre injury mncv values were obtained from a group very homogenous in age, length, gender, activities and even footwear. After inversion trauma (Recording II), the mncv in the Caput-Ankle and Knee-Ankle segments of the deep peroneal nerve of the injured leg was significantly lowered when compared with pre injury values. In the (short) Knee-Caput segments of both deep and superficial peroneal nerve mncv tended to be lower but no statistical significance was reached. The lack of statistical significance on these short segments can be attributed to 1) the small number of patients (N=7) and 2) the relatively high standard deviations. The latter is due to practically unavoidable inaccuracies in assessing nerve segment length. This inaccuracy has a relatively large effect in mncv assessment in short nerve segments.

The results show that due to inversion trauma the mncv of the deep peroneal nerve is lowered. One of the muscles innervated by this nerve, the anterior tibial muscle, shows high E.M.G. activity in heel strike and initial stance phase of normal gait[10]. Lowering of the mncv, caused by inversion trauma, could lead to prolonged tibial muscle reaction time and therefore to difficulties in stabilizing the ankle in these phases of normal gait. The mean decrease in mncv in the CAE and KAE segments is relatively small (4 and 5%, respectively) but group statistics have limited value for the individual. This can be illustrated by the model presented in our previous study. The model shows that, among others, the position of the peroneal nerve in relation to the rotation axis plays a role in the percentage elongation due to trauma. Since this position is variable, the effect of the inversion trauma on nerve length varies accordingly. In the present trial two out of seven patients showed after inversion trauma an almost ten percent decrease in mncv. In these patients, larger problems in ankle joint stabilisation can be expected than in the others.

Whether lowered mncv can be related to chronic ankle instability and clinical variables like pain, fear of giving way and fear of re injury is subject of a prospective, longitudinal study with a 9 month follow-up period now performed at the trauma department of a large regional hospital.

The results of the present study do not contradict the hypothesis of a traction lesion as cause of the lowered mncv of the peroneal nerves after inversion trauma, formulated in a previous study[10]. However, it cannot be excluded that the immobilisation after the trauma during 4-8 days is the cause of the decrease in mncv, not the trauma itself. To analyse this possibility a study will be performed to analyse the effects of short (4-8 days) immobilisation on mncv of the peroneal nerves.

Eighteen to 22 days post trauma mncv values of KAE and CAE segments of the deep peroneal nerve of the injured leg tended to be higher when compared to both baseline and control values. This is not in line with the results of a previous study[10] in which 32-36 days post trauma, mncv was still significantly lower when compared with control values. Probably this discrepancy is related to a difference in treatment. In the previous study the patients were immobilised rather rigidly (semirigid bandage for four or six weeks) when compared with the patients in this trial (tape bandage for ten days followed by remobilisation). We assume that the fast recovery of mncv in this study is due to the beneficial effects of early remobilisation and exercise[16,17].

Contralateral leg

Four to 8 days post trauma, mncv values in all four segments of the contralateral leg, tended to be lower than mncv values of the control group. This is consistent with our previous findings. However, compared to their own baseline values, the mncv of the contralateral leg tended to increase. This discrepancy needs attention since normally clinicians do not have the opportunity to compare post trauma values with baseline values of the patient. They have to rely on 'normal' values.

The necessity to be cautious with comparisons of patient data with 'normal' data becomes even more poignant when realising that in this study the normal values are obtained from a group perfectly comparable with the patients and exceptionally homogenous.

The increase in the mncv of the contralateral leg seen after trauma when compared to baseline values is remarkable but not unexpected. We assume that due to trauma the contralateral legs were used more intensively. Also in this case we attribute this increase in mncv in the contralateral legs to the effect of exercise[16, 17]. In fact, during the trial period not only mncv values of the contralateral leg tended to rise but also those of the controls. Exclusively the mncv values of the injured legs show a decrease. This could indicate a general increase of the peroneal nerve due to the (military) training.

Stratifying the population of 120 recruits according to the criterion: 'suffered no or one inversion trauma' (N=111) versus 'suffered two or more inversion trauma's' (N=9), baseline mncv values of the latter group were shown to be significantly lower. This is in line with the finding that 15 patients suffering from severe chronic ankle instability measured just before reconstructive surgery show a relatively low mncv of the peroneal nerves (18% lower than control group values; Kleinrensink, unpublished results). Both findings are indicative for a relationship between low mncv of the peroneal nerve and chronic instability of the ankle. Whether there is a correlation between number of inversion traumas and the magnitude of mncv decrease is subject of a prospective longitudinal trial.

Conclusion

Four to eight days after inversion trauma mncv values in the Caput fibulae-Ankle and Knee-Ankle segments of the deep peroneal nerve were lower than pre trauma mncv values. The findings are indicative for a causal relationship between inversion trauma and f unction deficit of the deep peroneal nerve.

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Chapter 8

General discussion and summary

General discussion

This thesis deals with two aspects of the human locomotor system, mobility of joints and stability of joints in relation with biomechanical tension of peripheral nerves. In line with this division, this discussion is divided into two sections.

Section one

The effect of motion on peripheral nerve tension and the value of nerve tension tests in the diagnosis of nerve lesions.

Mobility of the upper limb is the central issue in chapter 2, 3, 4 and 5. To allow certain combinations of movements of shoulder, elbow and wrist joints, the peripheral nerves of the upper extremity have to be elastic and mobile with respect to their surroundings. During movements within the normal range of motion (RoM) minor or even no tensile forces will be generated in the nerve. However, when the movements of the extremity exceed certain limits the peripheral nerves can restrict mobility. This holds especially in case of decreased mobility and/or elasticity of the nerve. Such a decrease is not exceptional. Even complete fixation of the nerve due to adhesions between nerve and surroundings is observed (e.g. peroneal nerve behind the fibular head (see chapter 6).

The limited capacity of nerve stretch can also be used to advantage. With knowledge of the anatomical course of a peripheral nerve, specific combination of movements can be used to selectively stretch the nerve[1]. In case of nerve or nerve root injury these motion patterns can be used to provoke complaints as pain and paraesthesia and hence become a diagnostic tool. Although such fast, easy and cheap stretch tests are attractive caution is recommended. Interpretation of the test results is often difficult since the distribution of tensile forces along the nerve, caused by the test manoeuvre, is not known. In the series of studies which form the basis of the fist section of this thesis, we attempted to answer two major questions:

1) What is the distribution of forces along the median nerve due to movements within the normal RoM? and
2) Are the tension tests for the nerves of the upper extremity as suggested by Elvey and coworkers[2,3] sensitive and specific?

The first problem was to develop a device capable to quantify tensile forces in peripheral nerves. We adapted the buckle force transducer as constructed by Peters[4] and validated this instrument for the use on nerve tissue. The results of the validation experiments (unpublished data) were promising and it was decided to perform a measurement series to evaluate tensile forces in peripheral nerves. This method of assessing tensile forces in nerve tissue can not be used *in vivo*. The use of *in vivo* animal experiments could be considered but extrapolation of the outcome to the human *in vivo* situation can be disputed. Experiments on unembalmed human bodies in situ are to be preferred but because of several restrictions (pressure of time, possibility of infectious disease etc.) embalmed human bodies have been used in addition to a limited number of unembalmed human bodies. In chapter 2 this choice is justified. We compared tensile forces in the median nerve caused by extremity positions within the normal RoM. A positive correlation was found between tensile force data obtained from unembalmed and embalmed specimens. It was concluded that in comparative studies on peripheral nerve tension, data obtained from embalmed human bodies can be used, although in embalmed human nerves the absolute tensile forces are higher.

In chapter 3 the results are presented of a study in which we measured the effect of twenty-two positions of the upper extremity on median nerve tension. This study provides insight in the distribution of tensile forces along the median nerve due to positions within the normal RoM. When forces were applied to the distal part of the nerve by changing the position of the wrist (e.g. dorsiflexion of the hand), tensile forces are transmitted along the median nerve upward to the brachial plexus. It turned out that tension in the proximal part of the median nerve occurs exclusively if dorsal flexion of the hand goes together with full extension of the elbow. As a consequence we advise, for certain long surgical procedures with the patient in the supine position, fully abducted arm and extended elbow (e.g. during radical mastectomy), to chose a position of the arm with 90° flexion of the elbow. Secondly, with *extended* elbow, dorsal flexion of the hand causes increased tension in all parts of the median nerve. With the elbow in 90° *flexion*, the same wrist position causes increased tension exclusively in the most distal (wrist) part. Thus in the diagnosis and localisation of median nerve lesions, in principle, differentiation between lesions in the upper and lower part of the median nerve is possible by manipulating specific joints.

The results of the study discussed in chapter 3 form the basis for chapter 4. Here, in an anatomical and biomechanical study, we evaluate nerve tension tests used in the diagnosis of nerve(root) and plexus lesions of the upper extremity. We studied the upper limb tension tests (ULTT) for the median, ulnar and radial nerve, as well as the ULTT combined with contralateral rotation and lateral bend of the cervical spine (ULTT+). Measurements with buckle force transducers were performed on the median, ulnar and radial nerve and the medial, lateral and posterior cord of the brachial plexus of embalmed human bodies. Based on the outcome variable (tensile force in N), exclusively the median nerve ULTT and ULTT+ turned out to be sensitive and specific tension tests. Since the data of *in vitro* studies cannot be extrapolated directly to the *in vivo* situation the value of these tests in clinical situations must be confirmed by means of controlled clinical trials. Before clinical research can be performed the effect of tension tests on nerve function in healthy volunteers should be established. In chapter 4 it was shown that only the median nerve ULTT and ULTT+ could be seen as sensitive and specific tension tests. So in chapter 5 we evaluated these two tests *in vivo* to stretch the median nerve. The effect of these tests on median nerve function was evaluated with the latencies of Somato Sensory Evoked Potentials (SSEP) as an outcome variable. Due to the test positions, the SSEP latencies were significantly enlarged. In some recordings a 10% increase in SSEP latencies was found already after one minute of testing. Compared with SSEP latencies caused by the ULTT, the ULTT+ did not cause significantly larger SSEP latencies. This finding is consistent with the finding in chapter 4 in which the ULTT+ did not significantly increase tension when compared with the ULTT. The findings confirm the conclusions from the *in vitro* studies presented in chapter 3 and 4, and justify the use of the two median nerve upper limb tension tests as clinical tools for generating tension in the median nerve.

Conclusion

All three *in vitro* studies provide insight in the effects of nerve tension tests and of movements within the normal RoM on the distribution of tensile forces in peripheral nerves and cords of the brachial plexus. The *in vitro* study on the value of nerve tension tests in the diagnosis of nerve and plexus lesions provides insight in the specificity and sensitivity of these tests based on tensile force as an outcome variable. The results of the *in vitro* studies form the basis for clinical evaluation of these tests. We made a first step by evaluating the functional effects of two nerve tension tests in healthy volunteers. The results, showing significantly enlarged SSEP latencies due to the testpositions, are promising.

Section two.

Peripheral nerve lesions due to inversion trauma and the possible role in (chronic) ankle joint instability.

In the chapters 6 and 7 the effect of movements beyond the normal RoM (trauma) on peripheral nerve function is studied. During hundreds of lectures on functional anatomy and demonstrations in the dissecting room we explained the harmful effects of extreme inversion movement on the ligaments of the lateral side of the ankle. However, the effect on the peroneal nerves never occurred as an issue needing attention. This is remarkable since the nerves have a course farther away from the pronation and supination axis than the ligaments, making them in principle more vulnerable to traction lesions.

In 1992 we started preliminary experiments on embalmed human bodies which showed considerable displacement and stretch of the peroneal nerves even during normal inversion movements. Simulation of inversion trauma showed that a considerable amount of tension had to occur in these nerves. A biomechanical model with (unpublished) data obtained from 15 embalmed bodies (see chapter 6) showed that in normal inversion an elongation of 3 cm (superficial peroneal nerve) and 4 cm (deep peroneal nerve) had to be compensated for. Taking several aspects of the anatomy, pathological anatomy and trauma mechanics into account, it became clear that a lesion of the peroneal nerves had to be considered as a possible effect of inversion trauma.

In chapter 6 the results of a prospective clinical trial are presented. Of 22 patients, the motor nerve conduction velocity (mncv) of the superficial and deep peroneal nerve was measured 4-8, 18-22 and 32-36 days post trauma. As hypothesised, the mncv values were significantly lowered after trauma when compared with those of the contralateral leg and a control group. This established a more solid belief in peroneal nerve lesions due to inversion trauma. We concluded that mncv measurements can be a valuable tool in assessing functional instability of the ankle joint induced by inversion trauma more objectively.

No relation was found between clinical (mechanical) grading of the inversion trauma and decrease of mncv. It was concluded that besides the standard mechanical grading a more functional approach to the effects of this trauma is needed.

One finding of this study needs special attention. Four to eight days post trauma, the mncv values of the deep peroneal nerve of the *contralateral* leg of the patients were significantly lower than those of the *control* group. Two possible explanations for this remarkable phenomenon were considered. Firstly, an inversion trauma also negatively affects the deep peroneal nerve of the contralateral leg. Secondly, the patients are characterized by a pre existent low mncv of the deep peroneal nerve in both legs, predisposing them for inversion trauma. To investigate the last possibility pre trauma mncv values had to be compared with post trauma values.

This led us to perform a prospective longitudinal study in 120 recruits of the Royal Dutch Army of which the results are presented in chapter 7. The mncv of the deep and superficial peroneal nerve was assessed upon entering military service (baseline values) and 4-8, 18-22 and 32-36 days after inversion trauma. We could show that 4-8 days post trauma mncv of the deep peroneal nerve was significantly lowered when compared with baseline values. So a causal relationship exists between inversion trauma and deep peroneal nerve function deficit. Four to eight days post trauma, the mncv values of the deep peroneal nerve of the contralateral legs of the patients tended to be lower than those of the control group However the deep peroneal nerve mncv of the contralateral leg of the patients tended to be higher when compared with their own baseline values. From these findings we concluded that it is necessary to be cautious when comparing patient data with 'normal' data. The more so, when it is realised that in this study the normal values are obtained from a exceptionally homogenous control group, perfectly comparable with the patients.

The results of the studies presented in the chapters 6 and 7 are in line with the hypothesis of a traction lesion as a possible cause of the lowered mncv of the peroneal nerves after inversion trauma. However, it cannot be excluded that the immobilisation following the inversion trauma is the cause of the decrease in mncv. Therefore we will analyse the effects of short periods of immobilisation on the mncv of peroneal nerves.

In the prospective study, nine recruits who suffered two or more inversion traumas in the past showed a significantly lower mncv when compared with their 111 collegues who suffered no or one inversion trauma. This is in line with the finding that 15 patients suffering from severe chronic ankle instability, measured just before reconstructive surgery, show a relatively low mncv of the peroneal nerves (18% lower than control group values; Kleinrensink, unpublished results). Both findings are indicative for a relationship between low mncv of the peroneal nerve and chronic instability of the ankle. If indeed low mncv values of the peroneal nerve have a role in the origin of chronic ankle instability the question arises what therapy could be used to increase mncv values. In this respect two studies are relevant. Sale et al.[5] and Kamen et al.[6] showed that in athletes, exercise can cause an increase of mncv in peripheral nerves. This led us to perform a pilot study in which patients suffering from chronic ankle trauma (planned to have reconstructive surgery of the lateral ankle ligaments) were given a rigid program of exercises. This program was meant to increase mncv of the peroneal nerves and hence to improve functional stability of the ankle. Preliminary results show indeed an increase in mncv values of both peroneal nerves and improved stability as shown by stabilometric data.

Conclusion

Both clinical studies confirm a causal relationship between inversion trauma and a function deficit of the deep peroneal nerve and possibly the superficial peroneal nerve.

Summary

In Chapter 1 two central aspects of the locomotor system are introduced, *motion* and *posture.* The role of Anatomy in both the clinical-practical and scientific-theoretical points of view on dysfunction of the locomotor system is discussed. Inversion trauma and (chronic) ankle instability are used to demonstrate the role of topographical anatomy in the clinical approach to dysfunction. The importance of functional anatomy when combined with biomechanics is emphasised by analysing the relevant structures and the chain of events involved in (chronic) ankle instability.
The concept of the arthrokinetic reflex of the ankle is used as basis for analysing the role of the peroneal nerve in ankle joint *stability* and (chronic) ankle instability. With respect to *mobility,* the Range of Motion(RoM) is used to discuss whether combined movements of the upper extremity can lengthen the arm nerves beyond their physiological limit. Finally the value of nerve tension tests for the diagnosis of nerve and plexus lesions of the upper extremity is discussed and the experiments are introduced to analyse the validity of these tests.

In Chapter 2 we analyse the effects of embalmment on the distribution of tensile forces in the median nerve due to different joint positions within the normal RoM. To assess this distribution *in situ* experiments on unembalmed human bodies are preferred but this has practical restrictions (pressure of time, possibility of infectious disease etc.) Here, a positive correlation is shown between tensile force data from unembalmed and embalmed nerves. This finding justifies, for comparative studies on peripheral nerve tension, the use of data obtained from embalmed human bodies.

In Chapter 3 we describe the device, the 'buckle' force transducer, to assess tensile forces on peripheral nerves and the method used to analyse the distribution of tensile forces along the median nerve. Twenty-two joint positions within the normal RoM were used including two positions defined as upper limb tension tests (ULTT) for the median nerve, one position defined as ULTT for the ulnar nerve and one position defined as ULTT for the radial nerve.
It is shown that forces introduced to the distal part of the nerve for instance by dorsiflexion of the hand, can be transmitted along the median nerve upward to the brachial plexus. However, increased tension in the proximal part of the nerve exclusively occurs when dorsal flexion of the hand is combined with full extension of the elbow. The finding that tensile forces in the median nerve significantly decreases by flexing the elbow is relevant for several surgical procedures. Furthermore this finding is relevant in the diagnosis and localisation of median nerve lesions. With extended elbow, dorsiflexion of the hand increases tension in all parts of the nerve. With flexed elbow, the same wrist position causes increase in tension only in the most distal (wrist) part. So, by manipulating specific joints it is, in principle, possible to differentiate between lesions in the proximal and distal part of the median nerve.

In Chapter 4 we describe further improvement of the 'buckle' force transducers in assessing tensile forces in peripheral nerves. This chapter contains the results of part one (Anatomical and Biomechanical aspects) of the study: 'Upper limb tension tests as tools in the diagnosis of nerve and plexus lesions'. Tensile forces were measured in the median, ulnar and radial nerve as well as in the cords of the brachial plexus.

The aims of this study were twofold:

1) To analyse the validity of six ULTT's: the median, ulnar and radial nerve ULTT and the same test positions with added contralateral rotation and lateroflexion of the cervical spine (ULTT+). Tensile forces (in N) were used as the outcome variable.
2) To analyse whether the tensile forces caused by the recommended tests positions were transmitted up to the Brachial plexus and if so, how they were distributed in the plexus.

From the vast amount of test results it was concluded that:

1) Based on the outcome variable, both the median nerve ULTT and ULTT+ are sensitive and specific tension tests. This does not hold for the Ulnar and Radial nerve ULTT and ULTT+.
2) Tension introduced in the extremity is transmitted to both the nerves and the cords of the Brachial plexus.
3) The observations support the concept of the brachial plexus having a function in distributing mechanical forces, thus protecting the cervical nerve roots against excessive traction forces.
4) It is unlikely that upper limb tension tests can be used to selectively stress cervical nerve roots.

Chapter 5 is formed by part two (Functional aspects) of the study 'Upper limb tension tests as tools in the diagnosis of nerve and plexus lesions'. In part one we showed that with tensile forces as the outcome variable out of the six tests studied exclusively the median nerve ULTT and ULTT+ could be considered specific and sensitive. Therefore we analysed in part two only the effects of the median nerve ULTT and ULTT+ on nerve function. Somato Sensory Evoked Potential (SSEP) latencies were used to quantify nerve function. We showed that SSEP latencies significantly increased as a result of the two nerve tension tests. Since increase in SSEP latencies is indicative for nerve elongation, the results of both studies justify the clinical use of the median nerve ULTT and ULTT+ to stretch the median nerve and related parts of the brachial plexus.

Chapter 6 describes the method used to assess function (deficit) of the peroneal nerve after inversion trauma. In 22 patients, 4-8, 18-22 and 32-36 days after inversion trauma motor nerve conduction velocity (mncv) of both the superficial and deep peroneal nerve was assessed. In the injured leg, 4-8 days post trauma mncv in the knee-caput fibulae segment of the *superficial* peroneal nerve was significantly lower when compared to the contralateral leg and to the control group. Five weeks post trauma these values were normal again. For all three segments of the *deep* peroneal nerve which were measured, the motor conduction velocity was significantly reduced, four to eight days post trauma, when compared to the control group. In the caput-ankle and knee-ankle segment, of the deep peroneal nerve motor conduction velocity was still significantly lowered five weeks post trauma.
A remarkable finding was that 4-8 days post trauma, the mncv values of the deep peroneal nerve of the *contralateral* leg were significantly lower than those of the *control* group.
It is concluded that mncv measurements (in addition to mechanical testing) can be an important tool in the early assessment and therapy of *functional* instability of the ankle joint. The disadvantages of mncv assessment for the patients are relatively small (mncv measurements are performed with surface electrodes, mean practically no extra inconvenience to the patients, do not take a lot of time and can be performed within 4-8 days post trauma).

The study presented in Chapter 7 can be seen as an extension of the trial presented in chapter 6. To know whether the lowered mncv of the peroneal nerves after inversion trauma was indeed caused by inversion trauma, we needed pre and post trauma mncv values. In 120 recruits of the Dutch Royal Army of the superficial and deep peroneal nerve mncv was measured shortly after entering military service (baseline values). Of seven recruits who suffered inversion trauma, mncv was assessed 4-8, 18-22 and 32-36 days post trauma. Post trauma values were compared to baseline, contralateral leg and control group values. Four to eight days post trauma the mncv in the knee-ankle and caput fibulae-ankle segments of the deep peroneal nerve of the injured leg was significantly lower than baseline values. In addition we could show that baseline values of recruits who suffered two or more inversion traumas in the past (N=9) were significantly lower than those of recruits who had suffered no or one inversion trauma (N=111).
It is conluded that a causal relationship exists between inversion trauma and function deficit of the deep peroneal nerve and probably also of the superficial peroneal nerve The findings, are in line with (unpublished) results of mncv measurements in 15 patients suffering from chronic ankle instability just before reconstructive surgery showing a mean mncv of 18% lower than control group values. These findings are a strong indication for a relationship between low mncv of the peroneal nerve and chronic instability of the ankle.

Because of studies showing that mncv can be increased by exercise we performed a trial in which a program of excercises intended to improve peroneal nerve function and functional stability of the ankle in patients suffering from chronic ankle instability.

In Chapter 8 we discuss the anatomical-biomechanical experiments and clinical trials related to the influence of posture and motion on peripheral nerve tension.

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Samenvatting

Houding en beweging vormen de centrale begrippen in dit proefschrift. In hoofdstuk 1 wordt de relatie tussen deze twee begrippen toegelicht. Tevens wordt het belang van de Anatomie in zowel de klinisch-practische als de wetenschappelijk-theoretische benadering van dysfunctie van het houdings- en bewegingsstelsel besproken. Verschillende vormen van anatomie spelen in deze benaderingen een rol. Grosso modo wordt bij de practische benadering primair de vraag gesteld: Wat is op welke plaats stuk? (topografische anatomie) en bij de theoretische benadering: Waaròm is dit stuk? en Waarom juist daar? (functionele anatomie). Het belang van het combineren van praktijk en theorie wordt geïllustreerd met een analyse van structuren die relevant zijn bij (chronische) enkelinstabiliteit. Bij deze analyse wordt gebruik gemaakt van het concept van de arthrokinetische reflex. In deze reflex speelt de n. peroneus zowel afferent als efferent een grote rol.
Als uitgangspunt voor de studie naar gewrichtsmobiliteit wordt het begrip 'Range of Motion'(RoM) gebruikt. Voor het bepalen van de RoM wordt normaliter slechts gekeken naar één gewricht. Vanzelfsprekend is dit niet, want in het dagelijks leven bewegen we hoogst zelden in één gewricht. In dit proefschrift wordt aandacht geschonken aan bewegingspatronen waarbij meerdere gewrichten zijn betrokken. Het is de vraag of bij zulke bewegingspatronen de betrokken zenuwen overmatig gerekt worden.
Tot slot wordt besproken in hoeverre zenuwrektesten relevant zijn voor de diagnose van zenuw- en plexusletsels van de bovenste extremiteit. In dit kader worden de experimenten geintroduceerd die gebruikt werden voor de validiteitsanalyse van deze tests.

Het effect van gewrichtsposities op zenuwrek zou eigenlijk *in vivo* bepaald moeten worden. Hiervoor is echter geen methode beschikbaar. Als alternatief komen *in situ* metingen aan ongebalsemde lichamen in aanmerking. Er zijn echter practische bezwaren tegen het gebruik van ongefixeerd menselijk materiaal (tijdsdruk, kans op infecties etc.) Daarom wordt in hoofdstuk 2 het effect bestudeerd van balseming op de grootte en de verdeling van trekkrachten in de n. medianus ten gevolge van verschillende gewrichtsposities binnen de normale RoM. Na balseming is de trekspanning in het algemeen groter: de zenuw is stijver geworden. Echter, er wordt een sterke positieve correlatie gevonden tussen resultaten verkregen bij ongebalsemde lichamen en die bij gebalsemde lichamen. Voor vergelijkende studies naar de verdeling van trekkracht op perifere zenuwen rechtvaardigt deze bevinding het gebruik van resultaten verkregen van gebalsemde lichamen.

In hoofdstuk 3 wordt het meetinstrument, de 'buckle force transducer' beschreven waarmee de trekspanning op perifere zenuwen is gemeten. Tevens wordt de onderzoeksmethode en het onderzoeksprotocol voor de analyse van de distributie van trekspanning op de n. medianus beschreven. Tweeen twintig gewrichtsposities binnen de normale RoM werden gebruikt waaronder één positie gedefinieerd als 'Upper Limb Tension Test' (ULTT) voor de n. ulnaris, één als ULTT voor de radialis en twee posities beschreven als ULTT voor de n.medianus.
Aangetoond wordt dat krachten, ingeleid aan het distale deel van de zenuw (bijv. door dorsaalflexie van de hand), doorgeleid kunnen worden over de n. medianus tot aan de plexus brachialis. Dit gebeurt echter alleen bij volledige extensie van de elleboog. De bevinding dat flexie van de elleboog

de spanning in de n. medianus significant verlaagd is relevant voor de positie van de arm op de infuusplank gedurende een aantal chirurgische ingrepen. Verder is deze bevinding relevant voor de diagnose en localisatie van n. medianus letsels. Bij gestrekte elleboog geeft dorsaalflexie van de pols een toename van trekspanning in de gehele zenuw. Met gebogen elleboog geeft dezelfde polspositie alleen spanning in het polsdeel van de n. medianus. Het is dus in principe mogelijk om met behulp van flexie en extensie van de elleboog te differentiëren tussen lesies in het proximale en distale deel van de n. medianus.

Hoofdstuk 4 betreft de Anatomische en Biomechanische aspecten van de studie: 'Upper Limb Tension Tests as tools in the diagnosis of nerve and plexus lesions' De Nn. medianus, ulnaris en radialis werden gerekt m.b.v. rektesten voor deze zenuwen(later door Elvey et al. 'Upper Limb Tension Tests' genoemd) en de resulterende spanning in de betreffende zenuwen en de fasciculi van de Plexus Brachialis werd gemeten. Het doel van deze studie was tweeledig, nagaan of

1) de zes ULTT's valide zijn. Het betreft: medianus, ulnaris en radialis ULTT en de zelfde testposities gecombineerd met contralaterale rotatie en-lateroflexie van de cervicale wervelkolom (ULTT+). Hierbij werd de trekkracht (in N) als effectparameter gebruikt.
2) trekkrachten, veroorzaakt door de aanbevolen posities,naar proximaal (tot aan de plexus brachialis) worden doorgeleid en zo ja, hoe zij worden verdeeld in de plexus.De studie leverde een grote hoeveelheid resultaten op.

Geconcludeerd werd:

1) zowel de medianus ULTT als de medianus UlTT+ kunnen gezien worden als sensitieve en specifieke tests. Dit geldt niet voor de ULTT en ULTT+ van n. ulnaris en n. radialis.
2) spanning in de zenuwen, opgewekt door bewegingen van de pols, geeft aanleiding tot verhoogde spanning in zowel proximaal gelegen delen van de zenuwen als in de fasciculi van de plexus brachialis.
3) in de plexus brachialis vindt verdeling van trekkrachten plaats waardoor de veel kwetsbaarder wortels beschermd worden tegen overmatige trekkrachten.
4) het is zeer onwaarschijnlijk dat Upper Limb Tension Tests gebruikt kunnen worden om individuele cervicale wortels selectief te rekken.

Hoofdstuk 5 betreft deel II ('Functional aspects') van de studie 'Upper limb tension tests as tools in the diagnosis of nerve and plexus lesions'. Omdat uit deel I van deze studie bleek dat uitsluitend de medianus ULTT en ULTT+ sensitief en specifiek zijn werd in deel II alleen het effect van deze testen op de n. medianus functie bij 19 gezonde proefpersonen bepaald. 'Somato Sensory Evoked Potentials' (SSEP) latentietijden werden als parameter gebruikt om de functie te kwantificeren. Daarbij werd de tijd gemeten die verloopt tussen zenuwprikkeling ter hoogte van de pols en de reactie hierop ter hoogte van de plexus Brachialis, de zevende halswervel en de schedel. Vergeleken met de uitgangspositie werden ten gevolge van de twee testen significant verlengde SSEP latentietijden waargenomen. Verlengde latentietijden zijn in dit verband indicatief voor zenuwverlenging. Geconcludeerd wordt dat de medianus ULTT en ULTT+ gebruikt kunnen worden om de n. medianus en de daar aan gerelateerde delen van de plexus brachialis te rekken.

In Hoofdstuk 6 wordt beschreven hoe gebruik gemaakt is van de geleidingssnelheid als parameter voor dysfunctie van de n. peroneus na inversietrauma. Om vast te stellen of na inversietrauma functiestoornissen van de n. peroneus optreden, werd bij 22 patiënten de motorische geleidingssnelheid (motor nerve conduction velocity, mncv) gemeten in drie delen (segmenten) van de n. peroneus superficialis en profundus. De metingen werden verricht 4-8, 18-22 en 32-36 dagen na een inversietrauma. In het aangedane been was de mncv in het knie-caput fibulae segment van de n. peroneus *superficialis* significant lager dan die van het contralaterale been en die van een controlegroep. Vijf weken na het trauma waren deze waarden weer normaal. In de drie bestudeerde segmenten van de n. peroneus *profundus* was de mncv significant lager dan die van een controle groep. In de segmenten caput fibulae-enkel en knie-enkel was de mncv na vijf weken nog steeds verlaagd t.o.v. de controlegroep.
De conclusie is dat mncv metingen in combinatie met de mechanische stabiliteitstesten een waardevol instrument kunnen zijn in vroege diagnose en therapie van *functionele* instabiliteit van de enkel. De nadelen van mncv metingen voor de patiënt zijn relatief gering (mncv metingen worden met oppervlakte elektrodes uitgevoerd en nemen weinig tijd in beslag) en de mncv kan - in tegenstelling tot depolarisatie potentialen - snel na het trauma (4-8 dagen posttrauma) worden bepaald.
Opmerkelijk was dat 4-8 dagen posttrauma de mncv waarden van de n.peroneus profundus van het *contralaterale* been significant lager waren dan die van de *controlegroep*. Dit zou kunnen betekenen dat *a)* het trauma ook ook invloed heeft op de geleidingssnelheid van het contralaterale been en/of *b)* reeds voor het bewuste inversietrauma lage geleidingssnelheden hebben bestaan. Dit laatste zou dan zelfs de oorzaak van het trauma kunnen zijn.

Om vast te kunnen stellen of er een causaal verband is tussen inversietrauma en geleidingsstoornis van de n. peroneus is het noodzakelijk om pre én posttrauma waarden te kennen. Daarom werd bij 120 recruten van de Koninklijke Landmacht de mncv van de n. peroneus superficialis en profundus gemeten tijdens of vlak na de intrede keuring (uitgangs- of 'baseline' waarden). Zie Hoofdstuk 7. Van zeven recruten die een inversietrauma kregen werd de mncv bepaald, 4-8, 18-22 en 32-36 dagen posttrauma. Posttrauma waarden werden vergeleken met 'baseline'waarden, waarden van het contralaterale been en met waarden van een controlegroep. Vier tot acht dagen posttrauma was de mncv in het knie-enkel en caput fibulae-enkel segment van de n. peroneus profundus significant verlaagd t.o.v. 'baseline' waarden. Tevens werd aangetoond dat 'baseline' waarden van recruten die in het verleden twee of meer inversietrauma's ondergingen (N=9), een significant lagere mncv hebben van de n. peroneus profundus dan recruten die geen of een inversietrauma ondergingen in het verleden (N=111). Geconcludeerd wordt dat een causale relatie bestaat tussen inversietrauma en functiestoornissen van de n. peroneus profundus en wellicht ook van de n. peroneus superficialis. De resultaten komen overeen met (nog ongepubliceerde) resultaten van mncv metingen bij 15 patiënten met chronische instabiliteit van de enkel die al op de operatielijst stonden voor een enkelbandplastiek. Bij deze groep patiënten was de mncv gemiddeld 18% lager dan die van een vergelijkbare controlegroep. De resultaten van beide studies wijzen op een verband tussen lage mncv waarden van de n. peroneus en chronische instabiliteit van de enkel. Gesteund door studies die aangeven dat de mncv beïnvloed kan worden door training is een trial gestart waarin aan patiënten met ernstige, chronische enkelinstabiliteit gedurende vier weken protocollair oefentherapie werd aangeboden met toenemende intensiteit en moeilijkheid. De voorlopige resultaten zijn zeer bemoedigend.

In hoofdstuk 8 worden de anatomisch-biomechanische experimenten en de klinische 'trials' bediscussieerd, van commentaar voorzien en in het kader geplaatst van de invloed van houding en beweging op perifere zenuwspanning.

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Curriculum vitae

We zijn wat we denken
Dhammapada, ± 500 voor Chr.

Gerrit Jan Kleinrensink werd geboren op 1 juli 1954 te 's Gravenhage. Na de middelbare school (J.R. Snoeck Henkemansschool) werd in 1976 het diploma Fysiotherapie behaald aan de Haagse Academie voor Lichamelijke Opvoeding. Tijdens deze opleiding werd zijn interesse voor de functionele anatomie gewekt; vanaf het tweede jaar was hij studentassistent bij het snijzaalonderwijs. De stap naar de Faculteit Bewegingswetenschappen van de Vrije Universiteit was dan ook klein. Ook tijdens deze studie was hij gedurende vele jaren student assistent bij de afdeling functionele anatomie. In 1988 werd het doctoraalexamen behaald.
Naast de studie in Amsterdam werkte de auteur van 1976 tot 1986 parttime als fysiotherapeut en als docent Anatomie-Fysiologie en docent Neurologie bij de in-service opleiding tot Verpleegkundige A van de haagse ziekenhuizen Leyenburg en Westeinde en de 'haegsche' Diaconessehuizen Bronovo en Voorburg.
Van 1986 tot 1991 werkte hij vier dagen per week als docent Anatomie-Fysiologie en als coördinator van de vakgroep Anatomie-Fysiologie bij het Opleidingsinstituut Florence Nightingale, het centrale opleidingsinstituur voor Verpleegkundige A van de zeven ziekenhuizen van 's Gravenhage.
In die periode werd met vrienden, tevens collega's, *De Krant* opgericht. Met deze band waarin de auteur als 'grondtonenkoning' basgitaar speelt werd met groot succes de Nederlandse feesten en partijen scene afgereisd. Na het afronden van dit boekje wordt de band gereanimeerd en ligt de rest van Europa voor deze band open.
Sinds 1980 is de auteur betrokken bij nascholingscursussen in de klinische anatomie van het bewegingsapparaat voor fysiotherapeuten en artsen (via het bureau PAOG van de Erasmus Universiteit Rotterdam, het N.P.I., voorheen S.W.S.F. en de Freie Universität, Berlin). Naast het werk in 's Gravenhage ging hij, in 1988 met een gastvrijheidsverklaring, één dag in de week onderzoek doen aan het bewegingsapparaat bij de vakgroep Anatomie van de Faculteit Geneeskunde en Gezondheidswetenschappen van de Erasmus Universiteit Rotterdam (Hoofd: Prof. Dr J. Voogd). Vanaf oktober 1991 is hij *full time* in dienst.
De laatste drie jaar is de auteur als 'part-time Bisschop van Myra' (1 dag per jaar) verbonden aan de Koningin Beatrix basisschool te Zoetermeer.

Tot slot. 'Je weet het vaak niet', maar nu heb ik de baan die ik iedereen toe zou wensen. Na de promotie hoop ik nog lang onderzoek te combineren met het geven van onderwijs.