UNIVERSITY OF SOUTHAMPTON

FACULTY OF MEDICINE, HEALTH AND LIFE SCIENCES

School of Health Sciences

Superficial Cervical Muscle Activation in Chronic Neck Pain

by

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<u>ABSTRACT</u>

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Chronic neck pain can occur in a proportion of individuals who have suffered a whiplash injury and also in individuals that have not experienced a trauma to the neck. The mechanisms that cause chronic pain are unclear, and whether they differ in traumatic or non-traumatic onset is unknown.

A review of the background literature identified differences in muscle activation for individuals with chronic neck pain, following a whiplash injury and from a non-traumatic onset, compared to healthy controls. However, differences in the combined action or synergy of superficial neck muscles in these chronic neck pain groups, during non-forced activities, had not been widely reported. A new methodology was developed to address this area of research. A pilot study was undertaken to establish the reliability of the method and to identify areas for refinement.

The main study employed the refined methodology to determine possible differences in activation and synergies of the upper trapezius and sternocleidomastoid muscles, alongside correlations of subjective pain and fatigue with surface electromyographic measures, using linear array electrodes.

Some differences in muscles activation and synergy were observed between the groups. Individuals showed different strengths of relationships between subjective and objective measures and different proportions of significant correlations were shown between groups.

To Simon

for feeding me

To Kathy and Lizzy

for growing so beautifully beside me

Contents

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Abbreviations and acronyms

5-HT   5-hydroxytryptamine

ACh   Acetylcholine

A/D   Analogue to digital

A&E   Accident and Emergency

Ag/AgCl   Silver/silver chloride

ANOVA   Analysis of variance

AROM   Active range of motion

ARV   Average rectified value

ATP   Adenosine triphosphate

BK   Bradykinin

BMI   Body mass index

C1-C7   Cervical vertebrae 1 - 7

Ca^{2+} Calcium ion

CBT   Cognitive behavioural therapy

CCFT   Cranio-cervical flexion test

cm   Centimetre

CNS   Central nervous system

CRPS   Complex regional pain syndrome

CV   Conduction velocity

CWT   Continuous wavelet transform

DOMS   Delayed onset muscle soreness

EMG   Electromyography

FFT   Fast fourier transform

FM   Fibromyalgia

FPS	Frequency power spectrum
gm	Gram
GMDF	Global median frequency
GRMS	Global root mean square
HC	Healthy controls
Hz	Hertz
IASP	International Association for the Study of Pain
ICC	Intraclass correlation coefficient
ICF	International classification of function
ISVR	Institute of Sound and Vibration Research
IZ	Innervation zone
JPE	Joint position error
Kg	Kilogram
KS	Kolmogorov-Smirnov
LSCM	Left sternocleidomastoid
LUTRP	Left upper trapezius
Max	Maximum
MDF	Median frequency
µV	Microvolt
Min	Minimum
ml	Millilitre
MMDF	Motor unit action potential median frequency
mm	Millimetre
mmHg	Millimetres of mercury
MMP	Mixed musculoskeletal pain

MMPI	Minnesota multiphasic personality inventory
MN	Motor neurone
MNF	Mean frequency
MR	Motor unit action potential rate
MRI	Magnetic resonance imaging
MRMS	Motor unit action potential root mean square
ms	Milliseconds
MSb	Mean square between groups
MSw	Mean square within groups
MU	Motor unit
MUAP	Motor unit action potential
mV	Millivolts
MVC	Maximal Voluntary Contraction
NDI	Neck disability index
NHP	Natural head repositioning
NIOSH	National Institute for Occupational Safety and Health
NRS	Numerical rating scale
NTNP	Non-traumatic neck pain
NTO	Non-traumatic onset
pH	Puissance of hydrogen
PSD	Power spectral density
Q-Q	Quantile - Quantile
QTF	Quebec Task Force
r	Reliability coefficient
RMS	Root mean square
ROM	Range of motion

RSCM	Right sternocleidomastoid
RSI	Repetitive strain injury
RUTRP	Right upper trapezius
SCM	Sternocleidomastoid
sEMG	Surface electromyography
SENIAM	Surface EMG for Non-Invasive Assessment of Muscles
SP	Substance P
Std	Standard deviation
TO	Traumatic onset
UEMD	Upper extremity muscle disorder
UK	United Kingdom
UTRP	Upper trapezius
VAS	Visual analogue scale
VRS	Verbal rating scale
WAD	Whiplash associated disorder
WHO	World Health Organisation

Glossary

Bilateral arm raise	An activity where both arms are raised outstretched to the side, level with the shoulders
Cervical	The region of the upper spine/neck consisting of 7 cervical vertebrae
Chronic pain	Pain that has lasted longer than the expected time taken for the related tissue damage to heal
Dynamic muscle activation	Activation of muscles in the performance of a movement or contraction
Habituation	A learned behaviour
Hyperflexion/hyperextension of the neck	Flexion and extension beyond the normal physiological range of movement
Isometric contraction	A contraction during which the muscles involved stay the same length
Motor control strategy	A neuromuscular coordinated action employing subdivisions of a muscle and/or more than one muscle
Muscle fatigue	The loss of strength and energy that results from exercise and causes a decline in the ability of a muscle to maintain a contraction
Neck extension	A rearward movement of the head, extending the neck
Neck flexion	A forward movement of the head, flexing the neck.
Protraction	A non-angular forward movement
Synergy	The combined action of muscles

DECLARATION OF AUTHORSHIP

I, Sally Anne Curtis, declare that the thesis entitled

Superficial Cervical Muscle Activation in Chronic Neck Pain

and the work presented in the thesis are both my own, and have been generated by me as a result of my own original research. I confirm that:

- this work was done wholly or mainly while in candidature for a research degree at this University;

- where any part of this thesis has been submitted for a degree or any other qualification at this University or any other institution, this has been clearly stated;

- where I have consulted the published work of others, this is always clearly stated;

- where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work;

- I have acknowledged all main sources of help;

- where the thesis is based on work done by myself jointly with others, I have made clear exactly what was done by others and what I have contributed myself;

- parts of this work have been published as:

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Signed: ....

Date: ………………………………………………………………………..

Acknowledgements

Chapter One – Introduction and overview of the study

1.1 Introduction to the research problem

Chronic neck pain is the second most common musculoskeletal complaint following chronic back pain and poses a major problem for the healthcare sector. It is estimated that the lifetime prevalence of neck pain is 67 - 80% and the point prevalence is 15 – 20%. As a result chronic neck pain has a huge impact on the economy in healthcare costs and lost working days (Cote et al., 1998; Ferrari & Russell, 2003; Feyer et al., 2006) .

Establishing how and why chronic neck pain develops has proved to be difficult due to the diverse nature of this condition, such as the causes of onset and the resulting symptoms. The International Association for the Study of Pain (IASP) has identified over 60 causes of neck pain including fractures, infections and tumours, however these causes are uncommon and more easily diagnosed (Merskey & Bogduk, 1994). The underlying causes of non-traumatic neck pain and pain which results from a trauma can be difficult to determine. The problematic nature of this area is illustrated by whiplash injuries. Up to 60% of individuals who experience a whiplash injury will develop chronic pain, this figure also shows that 40% of this population will not (Scholton-Peeters et al., 2003). These figures indicate that it is not simply the trauma, as a result of the injury, that causes chronic pain.

It is accepted that pain can cause alterations to muscle control strategies (Vlaeyen & Linton, 2006), which are possibly employed to minimise the use of painful muscles and thus minimise pain. Such alterations could be detrimental to the muscles that become under-used and to those where their use is increased in compensation (Nederhand et al., 2006; Vollenbroek-Hutten et al., 2006).

An increase in muscle fatigue in individuals who experience neck pain has been shown (Falla et al., 2003a; Falla et al., 2004c). It is possible that the use of superficial muscles could increase as a compensatory mechanism in order to avoid using painful, deeper structures. It is also possible that overuse of superficial muscles could itself result in pain. Fatigue could be a consequence of underuse and overuse of muscles.

Electromyography (EMG) is used to observe muscle activity including muscle fatigue (Dimitrova & Dimitrov, 2002). Using EMG to measure superficial neck muscle activity in individuals with chronic pain may enable identification of common patterns of muscle use. It may also be possible to establish whether altered patterns are common to all chronic neck pain cases or whether these patterns differ depending on the type of onset.

A relationship has been reported between perceived exertion and measures of fatigue in muscles in healthy control participants (Hummel et al., 2005). Relationships between subjective pain and fatigue with muscle activity have yet to be elucidated. Subjective associations are hard to establish for many reasons, such as the involvement of psychosocial factors, which can affect an individual's perception of pain, and variations in pain processing. A direct comparison between a muscle's activity and subjective pain and fatigue ratings could indicate the involvement of that muscle in the individual's perception of pain and fatigue.

There are many causes of onset and factors involved in the development of chronic neck pain. In the absence of accurate predictive techniques for this condition, identification of common or distinct features in chronic neck pain arising from different causes could greatly aid preventative and rehabilitative interventions.

1.2   Overview of the study

Review of informing literature

⬇

Methods (Pilot study)

⬇

Results (Pilot study)

⬇

Discussion (Pilot study)

⬇

Rationale for main study

⬇

Methods (Main study)

⬇

Results (Main study)

⬇

Discussion and Conclusion

Research questions – Pilot study

Is the methodology reliable for;

Determining differences in cervical muscle activation in people who have suffered a whiplash injury, individuals with non-traumatic neck pain and the normal population?

Determining relationships between muscle activation, subjective pain and fatigue?

⬇

Refinement of methodology

⬇

Research questions – Main study

1. Do cervical muscle activation patterns differ in people who have suffered a whiplash injury, individuals with non-traumatic neck pain and the normal population?

2. Is there a difference in the relationship between subjective measures of pain, fatigue and objective measures of muscle activity in people who have suffered a whiplash injury, individuals with non-traumatic neck pain and the normal population?

Chapter two – A review of informing literature

This chapter presents the relevant background literature to this study. It will take the reader from the anatomy of the neck and a detailed evaluation of skeletal muscle and function through to neck pain and whiplash injury. Acute and chronic pain mechanisms are then explored, followed by a comparison of studies between non-traumatic neck pain and neck pain from a whiplash injury. Finally, a review of electromyography (the technique proposed for this research) will be undertaken.

2.1   Anatomy of the Neck

The neck contains from deep to superficial, cervical vertebrae with intervertebral discs and ligaments joining them; deep muscles that act on the vertebrae and produce movements of the cervical spine; muscles producing movement of head on the neck; muscles producing movements of the scapula; and large blood vessels and nerves passing to and from the body, plus the visceral structures of the larynx and pharynx (Standring, 2008).

The vertebrae articulate via sympheses between the vertebral bodies, synovial joints between the facet (zygopophyseal) joints and the fibrous joints between the laminae, transverse and spinous processes (Standring, 2008).The muscles in the neck are arranged posterior, anterior and lateral to the cervical spine. The posterior group include spinotransverse muscles, semisplenalis, splenius, and muscles connecting to the upper limb, i.e. trapezius and levitator scapulae. The anterior and lateral group include longi colli and capitus, recti capitus anterior and lateralis and the scalenes. Sternocleidomastoid muscles pass from the temporal bone to the sternum and clavicle.

The two muscles of interest in this study are the upper trapezius and the sternocleidomastoid, which are superficial cervical extensors and flexors respectively. The superficial nature of these muscles permits recording of surface electromyographic signals. The origins, insertions and actions of these muscles are summarised in Table 2.1 (Standring, 2008). The accessory nerve innervates the sternocleidomastoid and trapezius muscles. It emerges through the jugular

foramen of the skull where the spinal root branches to innervate various structures. The accessory nerve is thought to be solely responsible for innervation of the sternocleidomastoid and primarily responsible for innervation of the upper trapezius. In some cases the cervical plexus also contributes to motor control of the upper trapezius (Standring, 2008).

Table 2.1. The origins, insertions and actions of the sternocleidomastoid and the upper trapezius muscles (Adapted from Standring, 2008).

Muscle	Origin	Insertion	Action
Sternocleidomastoid	Manubrium of the sternum and medial third of the clavicle	Mastoid process of the temporal bone and superior nuchal line of the occciput	Flexion, ipsilateral lateral flexion and contralateral rotation of the head on the neck*
Upper Trapezius	External occipital protuberance and superior nuchal line of the occiput ligamentum nuchae, and the spinous process of C7	Acromion process and the lateral third of the clavicle	Extension, ipsilateral flexion and contralateral rotation of the head and neck

* Possible extension of the head at the atlanto-occipital joint

The cervical muscles stabilise and position the head in relation to the position of the body. These include the sternocleidomastoid muscles (SCM) and the upper trapezius muscles (UTRP), which are two of the largest and most superficial muscles in the cervical spine. Although the SCM is commonly associated with flexion and rotation, an early study using fine wire electrodes showed SCM activity in resisted extension against a head weight in the prone position (Vitti et al., 1973).

The trapezius muscle laterally rotates, elevates and retracts the scapula. However, if the scapula is fixed, the upper fibres of the trapezius will extend and enable lateral flexion of the neck. When lying supine, the SCM muscles can be recruited to lift the head. By contracting one SCM muscle, the head will rotate and tilt towards the ipsilateral shoulder. When activated bilaterally, the SCM muscles assist the longus coli in producing flexion of the neck. The SCM muscles are

commonly recruited in side-to-side rotation of the head and in a lateral, upward movement of the head.

The trapezius muscle has 3 divisions, upper, middle and lower. Traditionally the upper division (cranial) is defined as originating from external occipital protuberance and superior nuchal line of the occiput ligamentum nuchae, and the spinous process of C7. The middle division of the trapezius originates from the spinous process of 1st to the 5th thoracic vertebrae and the lower division (caudal) originates spinous process of 6th to 12th thoracic vertebrae (Standring, 2008). However, there has been some controversy regarding the anatomical location of the upper trapezius. Johnson et al., (1994) proposed that the middle trapezius originates from the spinal process of C7. This has implications for electrode placement in sEMG recordings of the upper trapezius. Electrode placement for the upper trapezius muscle based on the traditional description of this muscles anatomical location is 2 cm lateral to the midpoint between C7 and the acromion process. According to the description by Johnson et al., (1994) this would result in the electrode being placed over the middle division of the muscle (see sections 6.7.1 & 6.7.2 for electrode placement).

Afferent information from the neck is received from mechanoreceptors, which respond to deformation e.g. pressure and touch, and nociceptors (which are free nerve endings) that are responsive to painful or irritating stimuli. These sensory receptors are found in the connective tissues, around the joints and in the tendons and ligaments (Gutyon, 1991d).

Active movement which is produced by the muscles does not normally result in damage to the structures of the neck. However, when movement is beyond the normal physiologic range e.g. a whiplash injury, it can result in pain caused by damage to the muscles themselves and their associated connective tissues (Bogduk & Yoganandan, 2001; Brault et al., 2000a; Vasavada et al., 2007).

For further detail on the physiology of pain see section 2.6

2.2 Anatomy of skeletal muscle

Contraction of skeletal muscle results in mechanical energy that creates heat, movement and postural stabilisation. Skeletal muscle fibres are derived from a syncitium of fused myoblasts. The contractile proteins of these original cells are actin and myosin which overlap to form a repeating unit of skeletal muscle, the sarcomere, which is the basis of each myofibril. Groups of myofibrils are surrounded by a sarcolemma and this forms a muscle fibre. Invaginations of the sarcolemma perpendicular to the myofibrils occur at regular intervals; this forms transverse tubules (T-tubules), which are juxtaposed to the sarcoplasmic reticulum of the muscle fibre. Mitochondria are found between the myofibrils that form each muscle fibre and the nuclei are peripheral to them beneath the sarcolemma. Satellite cells (stem cells for skeletal muscle) are found outside the sarcolemma. Muscle fibres (also called extrafusal fibres) are connected to external structures by their junctions with the adjacent endomysium and myotendonous junctions where the myofibril Z-discs join the sarcolemma and the connective tissue matrix of tendons (Wigley, 2008).

2.2.1 Electrical activity of muscles fibres

Muscle fibres are innervated by motor nerve endings at specialisations of the sarcolemma termed neuromuscular junctions. A terminal bouton from the nerve ending (motor end plate) is situated directly above a highly folded portion of the sarcolemma, which forms the sole plate. An action potential passing along the motor axon causes voltage-gated calcium channels to open. The subsequent influx of calcium initiates the release of Acetylcholine (ACh) from vesicles in the presynaptic neurone at the motor end plate. ACh diffuses across the neuromuscular junction cleft and binds with post synaptic ACh receptors on the sarcolemmal sole plate. This binding causes the opening of ligand-dependant sodium channels enabling an influx of the positively charged sodium ions. In skeletal muscle the potential difference across the sarcolemma is normally about -70mV (Guyton, 1991a).The electrical impulse then propagates across the sarcolemma, including the T-tubules, causing calcium release deep within the myofibrils, this fuels the movement of actin and myosin filaments to slide over each

other. The coordinated movement along all sarcomeres results in contraction of the muscle fibres (Guyton, 1991a).

2.2.2   Muscle fibre types

During muscle contraction, sensory information is gained from afferent nerve endings which entwine around nuclear bag and nuclear chain fibres. These fibres are contained within connective tissue sheaths which are collectively called a muscle spindle. Intrafusal fibres also receive motor innervation.

Extrafusal muscle fibres are classified into types based on their contraction speed (termed twitch speed) and metabolism substrate (Uhlig et al., 1995). Those fibres with abundant mitochondria and myoglobin use fat as an energy source via aerobic metabolism. Such fibres are called type I fibres. These fibres are generally smaller in size, with a relatively small cross sectional area. Other fibres have fewer mitochondria and little myoglobin, however they contain more glycogen used for anaerobic metabolism. These fibres have a fast contraction speed and are termed type II fibres, which are generally larger with a greater cross sectional area than type I fibres. Within this category there are type IIA fibres which are fast-twitch and fatigue resistant with oxidative metabolism and type IIB which are fast-twitch, susceptible to fatigue with glycolytic metabolism. Whereas some animals have segregated fibre types, in humans all muscles contain mixed fibre types which can be identified by specialized histological techniques. There is a relationship between motor innervation and muscle fibre type and evidence of fibre type transition if the innervation changes (Wigley, 2008).

There is a predominance of motor units that contain type I fibres in muscles that maintain tension and are active for long periods of time. Conversely, motor units with type IIB fibres are predominant in muscles that generate short-term powerful contractions. The actions of type IIA fibres are to "cope with routine activity against a background of more continuous postural tension" (Salmons, 1995, p757). This level of motor unit organisation is aimed at reducing the possibility of fatigue in a muscle. Type IIC fibres are those undergoing transition from one fibre type to another. Deeper muscles tend to have a higher proportion of type I fibres and superficial muscles have a higher proportion of type II fibres

Muscle fibres are adaptable and versatile, all fibre types are capable of hypertrophy with increased use, such as with an increase in exercise. Muscle fibres can also undergo atrophy with decreased use, which can be the result of ageing or injury (Standring, 2008). Gender differences are also apparent in the proportions and the size of muscle fibres. Males tend to have a higher proportion of type II fibres and show a larger cross sectional area for all fibre types than females. However, females have been shown to have larger type I fibres than type II in the vastus lateralis muscle (Simoneau and Bouchard, 1989; Staron et al., 2000). These studies show that the size of a muscle fibre cannot necessarily be assumed due to its classification of type.

2.2.3   Innervation of skeletal muscle

Skeletal muscles require innervation to remain differentiated and will atrophy otherwise. Skeletal muscle responds quickly to alterations in frequency and duration of use (Musacchia et al., 1988). Increased muscle activity builds up and maintains muscle mass and inactivity causes tissue degeneration. Long-term inactivity of skeletal muscle results in disuse atrophy (Booth, 1982; Jackman & Kandarian, 2004) and is characterised by a reduction in the cross-sectional area of the affected muscle fibres with decreased protein content, fatigue resistance and production of force. The resting tensions that result from contraction of a few muscle fibres give the muscle its tone. A single motor neurone (MN) and a group of muscle fibres that it innervates are called a motor unit (MU). In large muscles, each motor unit may contain 1200 muscle fibres whereas in smaller muscles, especially those capable of fine discriminative movement, the motor unit may contain 12 fibres or less. Each motor unit contains the same fibre type; MUs that consist of type I fibres are generally smaller than those containing type II fibres (Wigley, 2008).

Whereas some small muscles may be innervated by a single cranial or spinal nerve, larger muscles can be innervated by two or three spinal nerves. The group of motor neurones and their distal branches that innervate a whole muscle are collectively known as a motor neurone pool. The firing frequency of the individual

motor neurones and the proportion of the MN pool that is activated determine the force of contraction (Michael-Titus et al., 2007). Two types of motor neurones are distinguished; α motor neurones (α-MN) which innervate the extrafusal muscle fibres and γ motor neurones (γ-MN) which innervate the intrafusal fibres of the muscle spindles. Of the α- MN population, small α-MNs innervate MUs mainly composed of slow-twitch fibres. Large α-MN innervate MUs mainly composed of fast-twitch fibres. The main response to α-MN innervation is generation of force for movement and balance. γ-MNs are smaller than α-MNs; they are part of the reflex arc to the muscle spindles (Guyton, 1991b).

Skeletal muscle is closely bound to its surrounding connective tissue layers which play a significant part in the transmission of muscle contractile forces. This close relationship between muscle and its connective tissue is also reflected in the innervation of each tissue which feeds into segmental reflex arcs (Huijing, 1997).

Golgi tendon organs consist of small bundles of collagen fibres surrounded by a capsule of concentric collagen sheets. They are activated by a passive stretch of the tendon but are more sensitive to active contraction of muscle. The spinal reflex arc sends afferent information from the intrafusal muscle fibres and the tendon organs. The afferent neurones synapse on spinal interneurones, which in turn synapse on the α-MNs and γ-MNs. These motor neurones send excitatory signals to the extra and intrafusal muscle fibres and also inhibitory signals to the antagonistic muscle groups (Guyton, 1991d).

The reflex arc is responsible for the maintenance of muscle tone. If muscle tone decreases, causing the muscle to lengthen, the spindles become stretched and trigger increased impulse firing in the spindle afferents. This in turn increases the firing rate of the alpha motor neurones to that same muscle and causes it to contract (Guyton, 1991b).

Resting muscle tone is required to produce effective movements. Insufficient tone would prevent a muscle from responding quickly and efficiently to a stimulus. Too much muscle tone (hypertonicity) would not provide muscle fibres with sufficient rest in between contractions and could result in muscle fatigue. Excessive muscle

tone can also cause unnecessary contraction around joints, which can cause impairment of movement.

2.2.4   Muscle contraction

There are three main classifications of active muscle contraction. Concentric contractions occur in movements where the muscle shortens, eccentric contractions are where the muscle actively lengthens and during isometric contractions the muscle length remains unchanged (Wigley, 2008).

2.2.4.i   Rate coding

Motor units contract in response to action potentials from the motor neurons and these can occur at various firing frequencies. Rate coding is the frequency of action potentials that are fired in a motor neuron. The firing rate of action potentials is dependent upon the force required for a particular muscle contraction. During voluntary contractions, the lowest firing rate of neuronal action potentials to a muscle is approximately 8 -10 action potentials or pulses per second (pps). An average rate during an isometric contraction is 30-50 pps (based on soleus and biceps brachii muscles) (Duchateau et al., 2006). A wide variety of maximal firing rates have been recorded and are dependent upon the fibre type composition of that muscle, e.g. muscles largely composed of slow-twitch fibres have lower rates of action potentials in the motor neurons that innervate them. Maximal values of 100 – 200 pps have been recorded during rapid contractions (Duchateau et al., 2006).

2.2.4.ii Motor unit recruitment

The Henneman size principle states that there is an orderly recruitment of MU during a contraction (Henneman et al., 1965). Smaller MUs are recruited first and in the result of an increase in muscle force, more of the larger MUs are recruited. Initially the smaller MUs containing slow-twitch type I fibres produce a relatively weak but fatigue resistant force. When a greater force of contraction is required, the larger MUs containing more fast–twitch type IIA fibres are recruited. These fibres are more powerful than the slow-twitch fibres and are relatively fatigue

resistant. When a maximal or near maximal contraction is required the largest and most powerful fibres (Type IIB) are recruited, but due to their glycolytic metabolism they fatigue rapidly and can only exert short bursts of power (Miles, 1994).

Submaximal isometric contractions can be maintained for a longer duration, sometimes up to many hours. However, to maintain force output an increasing voluntary effort is required (Taylor et al., 2000). In less powerful, submaximal contractions there will be an initial activation of the type I fibres. As these fibres begin to fatigue type II fibres will be recruited to maintain the contraction. This will result in an increase in motor unit recruitment as the exercise progresses.

It has been proposed that during continuous contractions, some MUs fire while others recover from activation. This alternating recruitment provides an integral recovery period for MUs; rest gaps are said to occur when there is very low activity in muscle fibres during a contraction (Nordander et al., 2000).

2.2.4.iii Motor control and posture

Movement and posture are largely dependent upon the simultaneous contraction and relaxation of skeletal muscle. Muscle contraction has various levels of control, from spinal cord reflexes to conscious control of movement initiated by the motor cortex (Figure 2.1). Inputs from the brain stem, cerebellum, basal ganglia and thalamus are also involved in the coordination and execution of movement (Constanzo, 2006).

A recent review looked at several studies which investigated the effect of posture on elbow flexor muscle fatigue (Enoka & Duchateau, 2008). It was reported that the position of the shoulder and trunk could impact on the subjects' performance of upper limb tasks. It was reported that certain postures resulted in increased fatigue of the elbow flexor muscles. This implies that adjusted posture in response to pain, as stated by the fear avoidance model (see page 37), could cause activity of other muscles to become compromised resulting in increased muscle fatigue.

```
                    ┌─────────────────────────────────┐
                    │         Cerebral cortex         │
                    │          Motor cortex           │
                    └─────────────────────────────────┘

                         ┌──────────┐
                         │ Thalamus │
                         └──────────┘

              ┌──────────┐
              │  Basal   │
              │ ganglia  │
              └──────────┘

         ┌────────────┐              ┌────────────┐
         │ Cerebellum │              │ Brain stem │
         └────────────┘              └────────────┘

                    ┌─────────────────────────────┐
                    │         Spinal cord         │
                    └─────────────────────────────┘

              ┌──────────┐       ┌──────────────┐
              │ Sensory  │       │    Muscle    │
              │receptors │       │ contraction  │
              └──────────┘       │     and      │
                                 │   movement   │
                                 └──────────────┘
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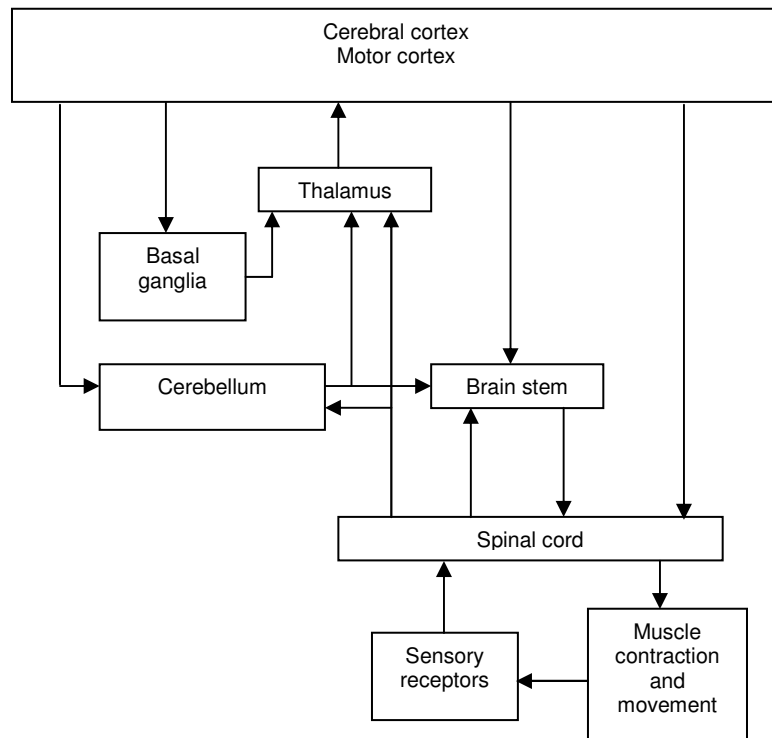
Figure 2.1 The hierarchy of motor control.

2.2.5 Muscle fatigue

Muscle fatigue is the temporary failure of a muscle to maintain or develop a certain expected force or power. The causes of fatigue can be due to physiological and psychological factors (Jones et al., 2004). Physiological factors can be peripheral and central in origin and Figure 2.2 shows the levels at which fatigue could occur during a muscle contraction. However it is unlikely that any fatigue process occurs in isolation it seems more probable that fatigue occurs simultaneously at central and peripheral levels e.g. pain arising from fatigued muscle fibres could decrease the motivation to continue with a muscle contraction.

It has been suggested that the major peripheral factors in fatigue are intracellular ionic changes that cause alteration of membrane excitability and decrease contractile force (Zwarts et al., 2008).

The Cinderella hypothesis, first proposed by Hägg in 1991, suggests that the smaller low-threshold MUs (those MUs recruited at lower forces and the first to be recruited in a muscle contraction) are also more liable to be damaged in a sustained activation of the muscle (Hägg, 1991). Such muscle fibre damage could cause them to become chronically fatigued, possibly due to long-term activation with inadequate rest gaps (Kallenberg et al., 2006).

Motivation

Motor cortex

Spinal cord

Motorneurone

Peripheral nerve

Neuromuscular junction

Muscle cell membrane

T-tubules

Calcium release

Cross bridge activity

Consequence of contraction
E.g. metabolites, heat
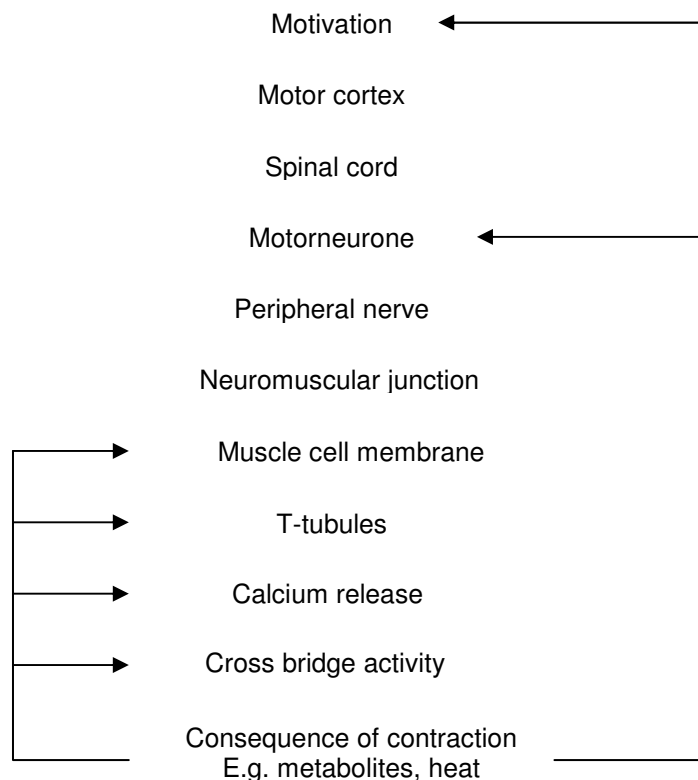
Figure 2.2 The levels at which fatigue could occur during a muscle contraction. The arrows indicate where the consequence of muscle contraction could feed back affecting further muscle activity resulting in fatigue. Reproduced with permission from: Skeletal muscle from molecules to movement (Jones et al. 2004).

Reduced recruitment MUs containing type I fibres and increased recruitment of MUs containing type II fibres would result in greater muscle fatigue due to the increased fatigability of type II fibres. This hypothesis could be integral in the development of chronic pain by overuse (or inappropriate use) of muscles as a result of a single traumatic impact or repetitive non-traumatic impacts.

The changes in MU activation during fatigue result in changes to electrical frequencies elicited from active muscles. Fatigue results in a shift of the frequency spectrum towards the lower frequencies (see section 2.9.8). These changes are assumed to relate to a progressive decrease in muscle fibre conduction velocity as a result of fatigue (Houtman et al., 2003). The changes recorded during fatigue may be associated with, but not implicit in, the cause of fatigue.

For detail on detection of electrical muscle activity and fatigue see section 2.9.

2.2.6   Blood flow in Muscles

An important consideration in muscle activity is circulation. Optimal muscle contraction is dependant not only on the muscle innervation but also on the continuous flow of oxygen and nutrients to the muscle and the removal of carbon dioxide and metabolites through extensive capillary plexuses. The rate of skeletal muscle blood flow is approximately 3 to 4 millilitres (ml) per minute per 100 grams (gm) of muscle in its resting state. This can be increased to 50 – 80 ml per minute per 100 gm during exercise (Guyton, 1991c).

In an exercising muscle, the blood flow will decrease during contraction due to compression of the vessels and increase between contractions (Guyton, 1991c). Blood pressure ensures the flow of blood to the muscles, and contraction of the skeletal muscle against fascia partitions (particularly in the limbs) along with the presence of venous valves, promotes the flow of blood into the venous circulation.

The blood flow within a muscle is controlled by local regulation and sympathetic nerves. Local regulation is the primary cause of increased flow, reduction in oxygen and an increase in vasodilator substances. These include nitric oxide, hydrogen ions, carbon dioxide and lactic acid which cause the arterioles to dilate.

Sympathetic activity can decrease blood flow in muscles by stimulating vasoconstriction in response to the release of noradrenaline, but it can also cause vasodilation with the release of adrenaline. This allows control of blood flow to

specific muscles (Guyton, 1991c). A study by Hansen et al. in 2000 showed that local tissue hypoxia attenuated sympathetic vasoconstriction in the forearm. This study used two methods to elicit hypoxia, firstly by exercise using a hand grip and secondly by exerting pressure on the forearm to reduce blood flow. The attenuation of the sympathetic response was seen in both methods of generating hypoxia. This indicates that local factors can override central mechanisms to enable an adequate circulation within a muscle.

Other afferent nerve endings within muscles and the surrounding connective tissues convey information concerning the local pH, which is altered in hypoxia. They also convey information regarding the extent of local tissue damage noted by the release of muscle fibre elements, enzymes and adenosine triphosphate (ATP), and cytokines released from inflammatory cells subsequent to injury. The sensory reception of these stimuli results in the perception of pain.

2.3   Epidemiology of neck pain

Neck pain is a widespread problem that affects a large percentage of the population. It has been reported that 10-17% of adults at any one time and as many as 71% of adults during a lifetime experience neck pain (Walker-Bone et al., 2004). A study by Marshall et al., (1995) of hospital workers in the UK found that there was a neck pain incidence of 34% in participants that had never experienced a neck injury. In addition 80% of participants who had suffered a neck injury reported neck pain (Marshall et al., 1995). It is interesting to see the difference in prevalence of neck pain between those who had experienced a neck injury and those that had not, however this study has limitations. For example, the participants were recruited from a health care setting and the sample is therefore not representative of the population.

A Swedish survey questioned half of all men born in 1914 about neck pain. It was found that 5% had daily neck pain (Isacsson et al., 1995). No differences were found in relation to workload or job strain but significantly lower levels of pain were reported in participants who had informational support regarding pain.

A review of worldwide literature reported great variation in methodologies of chronic neck pain studies. Variation was evident in sample groups, definitions and terminology used (Feyer et al., 2006).These factors make it very hard for any firm comparisons and conclusions to be drawn between research studies. However, two outcomes of this review were that chronic neck pain is a common symptom within a population, and that generally women experience more neck pain than men (Feyer et al., 2006).

2.4   The causes of non-traumatic neck pain

Excluding underlying pathologies, neck pain has been shown to arise from several factors such incorrect or awkward posture and repetitive use of muscles (Walker-Bone & Cooper, 2005). The relationship between conditions in the workplace and musculoskeletal disorders was first described by Bernardino Ramazzini in 1700. He noted that prolonged stationary postures and irregular movements; seen in bakers, weavers and scribes, could lead to "disease" (Franco & Fusetti, 2003).

In 1985 The World Health Organisation (WHO) described work-related conditions as multifactorial to incorporate the numerous risk factors that can contribute to these conditions (WHO, 1985). These factors include; physical, psychosocial, individual, social and cultural. The relevant importance of the wide range of contributory factors has caused controversy in this area of research. In order to address this problem a classification of musculoskeletal disorders and recommendations for research were published in 1997 by the National Institute for Occupational Safety and Health (NIOSH) (Bernard, 1997).

Despite the report by NIOSH there are still a plethora of terms used to describe muscle pain arising from work. These include repetitive strain injury (RSI), work-related upper limb disorder, work-related musculoskeletal disorders, cumulative trauma disorder and occupational overuse syndrome (Lynn, 2006). RSI is a term used to describe disorders that can result from repeated exposure to ergonomic hazards. Such hazards include: repetitive and forceful motions, awkward posture, static muscle load, extremes of temperature, excessive vibration, poorly organised work schedules and psychosocial factors (Barbe & Barr, 2006; Yassi, 1997).

An increase in the use of computer workstations both at work and at home has corresponded with an increase in the rate of reported upper limb/neck disorders (Yassi, 1997). A review of the relationship between neck disorders and occupation reported that it is difficult to compare the relationship due to the varying methodologies employed. However, the consensus was: "Abnormal posture, repetitive tasks, lack of support from colleagues and poor control over work may lead to neck pain" (Walker-Bone & Cooper, 2005, p1393).

A model for the development of RSI was developed, based on the relationships between factors from the workplace (external loads, organisational factors and social context) and the person (loading, internal tolerance and outcomes) (Lynn, 2006). Another model that included non-workplace factors stated that stressful environments at home can be as pertinent as those in the workplace (Barbe & Barr, 2006). These studies illustrate the difficulties in this area of research. For a complete picture the workplace, individual and non-workplace factors all need to be taken into consideration.

A new aspect of RSI research is focussed on minor peripheral nerve damage caused by damage to the nerve itself or changes in the microenvironment of particular peripheral nerves (Barbe & Barr, 2006; Lynn, 2006). A rat model of chronic nerve compression was used end there was found to be an increase in intraneural inflammatory cytokines, fibrosis and declines in electrophysiological function, including a decrease in conduction velocity of the nerve (Barbe & Barr, 2006). Although pro-inflammatory cytokines have been linked to "sickness behaviours", they have not been studied in humans with RSI. Therefore a firm link between physiological and psychological factors has yet to be established.

2.5   Whiplash injury

There have been many descriptions of injuries to the soft tissues of the cervical spine as a result of a rear impact. "Railway spine" was a term used at the turn of the century when accidents involving trains were more common than automobile accidents (Eck et al., 2001). The term whiplash or whiplash syndrome was first

used by H. E. Crowe in 1928 to describe a soft tissue injury to the cervical and upper thoracic spine due to a sudden acceleration/deceleration force as the result of a rear-impact from a moving object e.g. a car (cited in Foreman & Croft, 1995). Cervical acceleration/deceleration syndrome was described to try to specify the nature of the injury (Foreman & Croft, 1995).

Whiplash is a term that is often used loosely with no universally accepted definition. In 1995 the Quebec Task Force (QTF) produced a more specific definition of whiplash as "an acceleration-deceleration mechanism of energy transfer to the neck which may result from a rear-end or side impact, predominantly in motor-vehicle accidents, and from other mishaps." (Spitzer et al. 1995, p22S).

To classify injuries resulting from whiplash incidents the QTF formulated the term Whiplash Associated Disorder (WAD) and defined the clinical presentation of patients in five grades:

  WAD-0 = No complaint about neck pain, no physical signs
  WAD-I = Neck pain, stiffness, or tenderness only, but no physical signs
  WAD-II = Neck problems and musculoskeletal signs
  WAD-III = Neck problems and neurological signs
  WAD-IV = Neck problems and fracture or dislocation

A more recent classification system has been proposed by Sterling in 2004. This model includes more detail and outcome measures than the classification system proposed by the QTF and is described in Table 2.2.

It can be seen from the classification systems that a whiplash injury can frequently result in soft tissue injury. The injury can then develop with a variety of symptoms to cause acute and chronic pain (Atherton et al., 2006; Banic et al., 2004; Cailliet, 1991; Nachemson & Jonsson, 2000; Obelieniene et al., 1999).

Table 2.2 A proposed new classification system for whiplash associated disorders-implications for assessment and management. Source of text: Sterling (2004), Manual Therapy 9: 2; 60-70.

Proposed grade	Physical and psychological impairments present
WAD-0	No complaint about neck pain no physical signs
WAD-I	Neck complaint of pain, stiffness or tenderness only no physical signs
WAD-II A	Neck pain motor impairment: decreased range of motion (ROM), altered muscle recruitment patterns sensory impairment: local cervical mechanical hyperalgesia
WAD-II B	Neck pain Motor impairment: decreased ROM, altered muscle recruitment patterns sensory impairment: local cervical mechanical hyperalgesia psychological impairment: elevated psychological distress
WAD-II C	Neck pain Motor impairment: decreased ROM, altered muscle recruitment patterns Increased joint position error (JPE) Sensory impairment: local cervical mechanical hyperalgesia, general sensory hypersensitivity. May show some sympathetic nervous system (SNS) disturbance Psychological impairment: psychological distress, elevated levels of acute post-traumatic stress
WAD-III	*As WAD-II C* with neurological signs of conduction loss including: decreased or absent deep tendon reflexes, muscle weakness and sensory deficits
WAD-IV	Fracture or dislocation

2.5.1   Epidemiology of whiplash

There have been many studies to show the incidence of whiplash. Scholton-Peeters (2003) stated that 39 per 100,000 of the German population have suffered a whiplash injury, 70 per 100,000 in Quebec and 188 per 100,000 in the Netherlands. An approximate incidence in Western societies has been stated as 1 per 1000 of the population (Barnsley et al., 1994). Another study indicated that there are approximately 300,000 new incidences of whiplash per year in the UK which translates to 500 cases per 100,000 of the population (Moore et al., 2005).

A more recent report that was based upon a systematic review of whiplash related articles between 1980 and 2006 states that in the last 30 years Western countries

have seen the incidence of reported WAD increase (Holm et al., 2009). In this review incidence in North America and Western Europe was suggested to be 300 per 100,000 inhabitants, with females being at a slightly increased risk. It also stated that there was preliminary evidence that younger individuals and those with prior neck pain were at a higher risk of seeking further health care and compensation following a whiplash injury.

The large differences in these statistics can be attributed to many factors, including methods by which these data were collected and analysed, concentration of traffic, levels of vehicle safety and the prevalence of litigation within a particular country. A study undertaken in Lithuania looked at post-traumatic neck pain after a rear-end collision (Obelieniene et al., 1999). Although 48% of respondents reported acute pain a week after the incident, only 4% had frequent neck pain (>7 days/month) after 1 year. This compared to 6.25% of control subjects. The author suggested that the lack of persistent symptoms was due to cultural differences, mainly within the legal-medical system. For example the absence of personal insurance cover or financial assistance for individuals taking sick-leave from employment in Lithuania appears to have led to a low expectation of long-term symptoms or disability.

Litigation has also been implicated as a factor in the development of chronic pain (Busse et al., 2004). This study reported that participants in receipt of compensation had higher levels of self-reported disability. However, this study recruited subjects from a chiropractic clinic and the findings may be subject to sample bias as individuals may seek private health care in order to assist their claim. It would be necessary to establish if participants were seeking compensation to try to obtain a more representative sample.

The information in Table 2.3 shows several examples of studies using differing designs and population samples. This illustrates some of the problems that can arise when drawing conclusions of the incidence of whiplash injury while extracting data from epidemiological studies.

Table 2.3 Examples of epidemiological studies of whiplash injury.

Adapted from Nachemson and Jonsson (2000) Neck and back pain, pp 180-181

Study	Country	Design	Sample	Results	Comments
Barnsley et al (1995)	Australia	survey of cervical facet joints post whiplash	50 consecutive patients referred to neck research clinic	painful facet joints identified in 54% of patients	small sample size, every other patient did not identify pain
Borchgrevink et al (1996)	Norway	retrospective study of injuries resulting from car accidents	426 patients from A&E department. No patient contact data collected from social security files	58% displayed sustained symptoms linked to the accident	data collected of limited value due to lack of patient contact
Deans et. al (1987)	Great Britain	follow up from neck pain study and control group	137 patients	two times more likely to sustain injury from rear impact that front impact	participants questioned 1-2 years after incident
Spitzer et. al. (1995)	Canada	compensation claims followed for 6 years	all reported whiplash collisions in 1987 (Canada)	70/100000 inhabitants suffer whiplash incident	sample limited due to their claim for compensation
Partheni et al. (2000)	Greece	prospective study of victims presenting at A&E	180 patients	90% recovered after 4 weeks. No cases of chronic disability.	participants with previous neck pain included
Richter et al. (2004)	Germany	prospective study of victims presenting at A&E	32 patients diagnosed with WAD-I & II	66% reported no problems at 6 months	small sample size

2.5.2 Economic impact of whiplash injury

When looking at the problem of whiplash injury as a whole, the majority of reported symptoms resolve within 2-3 months, 14 - 42% of patients endure ongoing symptoms of some description and 10% suffer from persistent and severe pain (Barnsley et al., 1994). This can have a dramatic affect on quality of life, including the ability to return to work.

The development of improved safety measures such as headrests and heightened awareness of correct seating positions have helped to reduce the numbers of those sustaining a whiplash injury following a motor vehicle collision. Interestingly, one study showed that some headrests reduced whiplash injury by 37% in female drivers with very little effect on males (Farmer et al., 2003). An explanation was not given for these findings, it was stated that the headrests were designed for taller males and the results were unexpected. However, this study only tested the headrests of two cars and these results cannot be inferred on headrests generally. New seat designs in some cars can be effective at reducing whiplash injury in both males and females - 43% and 55% respectively (Farmer et al., 2003).

The healthcare and rehabilitation costs, as well as lost working time, means that whiplash injury is significant and results in a substantial burden on a Nation's economy (Magnusson et al., 1999). It is reported that the consequence of whiplash injury has a total cost of $3.9 billion annually in the United States (Eck et al., 2001).

In Canada the approximate cost to the economy per patient is $2500 (Spitzer et al., 1995). According to a study in 2006 by the insurers Norwich Union, rehabilitation from whiplash injury costs the UK economy £1.05 billion a year in terms of work related output. The study also states that the UK economy would save approximately £29 million a year if working days lost following a whiplash injury were reduced by one day (http://www.aviva.com; date accessed 18/09/ 2006). However, these data should be interpreted with caution as it is extracted from a survey financed by an insurance company.

2.5.3   Biomechanics of the neck in a whiplash injury

A definition of biomechanics is: "The mechanics of movement in living creatures" (Schwartz et al., 1988, p142). However, it has also been described in relation to injury as the mechanical forces that affect or alter living organisms (Rosenquist & Rook, 2003).

The hyperextension/hyperflexion theory describes the acceleration and deceleration of the body and head from a biomechanical aspect (Yoganandan & Pintar, 2003). In normal voluntary movement extension and flexion of the neck can be actuated by many muscles (Cailliet, 1991; Foreman & Croft, 1995; Yoganandan & Pintar, 2003).

The hyperextension/hyperflexion theory states that when the body is accelerated the head does not move as quickly as the body due to inertia. Therefore the muscles in the neck do not react quickly enough to give sufficient support which result in the neck being extended beyond physiological limits. When the body stops the neck continues to move due to the momentum of the forces applied, and the neck goes into hyperflexion (Cailliet, 1991).

The hyperextension/hyperflexion model of whiplash injury has now been superseded. Studies have determined that following a rear impact, C6 goes into extension before any movement of the upper cervical vertebrae (Bogduk & Yoganandan, 2001;,Brault et al., 2000b). This causes the lower cervical vertebrae to be in extension whilst the upper vertebrae are in flexion (Eck et al., 2001; Klein et al., 2001). This results in an "S" shape in the cervical spine, shown in Figure 2.3.

The "S" shaped curvature to the cervical spine can be seen during the first 50-110 ms of a whiplash event. This can result in damage to intervertebral discs, facet joints and nerve roots. Soft tissue damage may also occur and cause compression of nerve roots. Contraction induced or muscle lengthening injuries can also occur following this type of trauma (Brault et al., 2000b; Eck et al., 2001).

A whiplash injury can result in a range of pathologies including fractures of the vertebral bodies, zygopophyseal joint and joint capsule damage, lesions to the ligaments and intervertebral discs, nerve tissue injury and muscle damage (Bogduk & Yoganandan, 2001; Jonsson et al., 1994; Taylor & Taylor, 1996). This review will focus on minor damage resulting from a whiplash injury and the pain resulting from such damage.
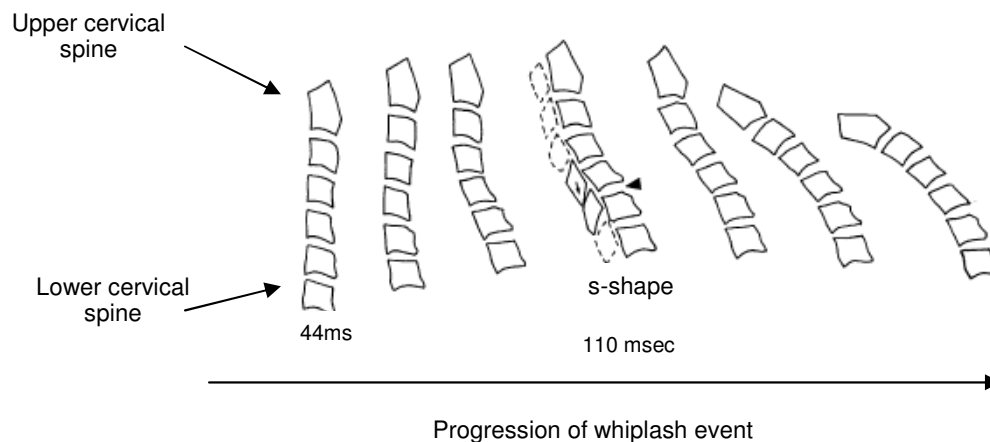
Figure 2.3 A transient S-shaped curvature of the spine.
A diagrammatic representation of a series of radiographs of the cervical spine taken during a rear-end impact (4kph) in the absence of a head rest. 44ms post-impact the spine straightens and at 110ms it produces an S shaped curve. At this point the lower cervical spine is in extension and the upper cervical spine is in flexion. Lastly the cervical spine assumes a C shaped curve due to extension of the upper cervical spine. Reproduced with permission, (Bogduk & Yoganandan, 2001).

2.6 Pain

Following the review of non-traumatic neck pain and whiplash injury, the mechanisms involved in the development of pain are presented in this section. Pain is a complex phenomenon with no overarching definition. It is an individual and subjective experience that can have many contributing factors including age, gender, emotion, cognition and motivation. The physiology and psychology of pain are inextricably linked.

2.6.1 The physiology of pain

Pain is detected through nociceptors (free nerve endings) found in most tissues of the body and transmitted through sensory or afferent neurones to the central

nervous system (CNS). It is in the CNS that the sensation of pain is perceived and responses to the painful stimuli are elicited.

There are four distinct types of primary sensory afferents (Aguggia, 2003; Guyton, 1991d; Wall & Melzack, 1994). A α fibres are the largest diameter fibres with a conduction velocity (CV) of 80 – 120 m/sec. These afferents respond to changes in proprioceptive information. A β fibres respond to light and heavy mechanical stimulus and have a CV of 35-75 m/sec. (A β fibres are now implicated in sensitisation - changes in the processing of input in the spinal cord are reported to cause signals from the A β fibres to be perceived as pain signals.) A δ fibres are the "fast pain" primary afferents and are associated with acute and well localised pain sensation and have a CV of 6-30 m/sec. C fibres are small diameter neurones associated with longer lasting pain that have a slightly longer onset than acute pain. These fibres are associated with motivational and affective aspects of chronic pain. They have a CV of 0.5-2 m/sec.

It is not simply the painful stimulus that determines an individual's perception of pain. Three systems have been identified to interact in the production of pain. There is the sensory or discriminative system, which includes temporal, spatial, strength and intensity components; the motivational/affective system in which learned and conditioned responses are integrated into an individual's response to pain and the cognitive/evaluative system that determines an individual's interpretation and an appropriate behavioural response to pain. The pain experience may be further influenced by factors such as gender, culture and experience (Huether & Leo, 2002).

2.6.1.i Nociception

Peripheral pain arises from muscles, tendons and other tissues including peripheral nerves (Pain arising from peripheral nerves is termed neuropathic pain and caused by damage to the nerves themselves). Peripheral pain occurs in response to nociceptive stimuli. The stimuli that cause nociception are chemical, mechanical and thermal. Chemicals released in response to tissue damage include bradykinin (BK), histamine, serotonin (5-HT) and potassium ions (Guyton,

1991d). Inflammatory mediators such as cytokines, chemokines, interleukins and substance P (SP) are also released (McMahon & Jones, 2004).

The electrical response to the release of these mediators is then conducted along afferent nerve fibres to the CNS for processing in order for an appropriate response to elicited. This afferent input occurs via a four neurone pathway, the cell bodies of the first order or primary neurones are situated in the dorsal root ganglion. These neurones enter the dorsal horn of the spinal cord and synapse with the second order or secondary neurones. These then ascend through the spinothalamic tract of the spinal cord, to the thalamus. From the thalamus, the third order neurones project to the postcentral gyrus which is located in the cerebral cortex. These then synapse with the fourth order neurones that are located in the sensory regions of the cortex (Riedel & Neeck, 2001).

Nociceptors also have efferent activity that can originate both centrally and peripherally. Inflammatory mediators are released through the peripheral axon reflex (Meyer et al., 2006). Peptides such as substance P, neurokinin A and K and calcitonin gene-related peptide (CGRP) can induce the flare response to injury upon release. Such antidromic activity originating from the spinal cord (also referred to as the dorsal root reflex) has been implicated in chronic inflammatory conditions such as arthritis (Willis, 1999). It has been suggested that this activity contributes to inflammatory processes by causing the release of neuropeptides from afferent terminals in the periphery (Meyer et al., 2006).

2.6.1.ii Inflammatory pain

One possible consequence of a whiplash injury is inflammation, which involves a complex cascade of reactions at the peripheral receptor terminal. These reactions can be divided into two entities, neurogenic and non-neurogenic inflammation. Neurogenic inflammation includes the release of neuropeptides (e.g. substance P and CGRP) from C fibre terminals whereas non-neurogenic inflammation involves the release of inflammatory substances (e.g. histamines, prostaglandins, cytokines, leukotrienes, bradykinin, etc) from the blood vessels and connective tissue. Inflammation occurs in response to muscle and other soft tissue damage (Meyer et al., 2006).

2.6.1.iii Neuropathic pain

"Neuropathic pain syndromes (pain after a lesion or disease of the peripheral or central nervous system) are clinically characterised by spontaneous and evoked types of pain, which are underpinned by various distinct pathophysiological mechanisms in the peripheral or central nervous system" (Baron 2006, p 95).

There are two key concepts that are essential for the understanding of neuropathic pain: abnormal nociceptor activity from the injured neuron and changes in central sensory processing as a result of abnormal nociceptor activity (Meyer et al., 2006).These concepts encompass the pain arising from damage to the nerves and the resultant sensitisation of the CNS.

The mechanisms of neuropathic pain are not distinct from the pain mechanisms that have already been discussed. Hyperalgesia resulting from peripheral and central sensitisation is seen in inflammatory pain as well as in neuropathic pain. However, it is the underlying cause of hyperalgesia that differs. In neuropathic pain the nociceptors themselves are damaged, whereas in inflammatory pain the nociceptors respond to damage in surrounding tissues. Both neuropathic pain and inflammatory pain are possible outcomes of a whiplash injury.

2.6.1.iv Somatic referred pain

Pain felt distant to the source of pain is known as referred pain. Somatic referred pain that arises from muscle and associated skeletal structures. An example of this is damage to the tissues of the cervical spine can cause pain to be referred to the shoulder and upper limb. This pain results from activation of nerves originating from the same spinal segment (Newell, 2008).

2.6.1.v Central and peripheral sensitisation

A painful stimulus can result in a sensitised response due to changes in pain processing. Allodynia occurs when a normally non-painful stimulus results in a

painful response. Hyperalgesia is an increase in the perception of pain without an increase in strength of stimulus (Wall & Melzack, 1994).

Primary hyperalgesia occurs at the site of injury and secondary hyperalgesia occurs in uninjured tissue around the site of injury. Both allodynia and hyperalgesia are conditions of hypersensitivity (Aguggia, 2003).

Hypersensitivity is a neuronal response that can occur peripherally and centrally. Sensitisation of nociceptive neurones occurs when nociceptors increase their response to a stimulus without change in the intensity of that stimulus (Wall & Melzack, 1994). Sensitisation is known to be an underlying mechanism in hyperalgesia   (Aguggia, 2003; Riedel & Neeck, 2001; Wall & Melzack, 1994).

It has been suggested that peripheral hypersensitisation results from the repetitive release of prostanoids and other inflammatory mediators as a result of tissue damage. This can change the ion channel properties of nociceptor membranes therefore increasing their reactivity to a stimulus (Baron, 2006; McMahon & Jones, 2004).

Central sensitisation is characteristic of chronic pain and occurs largely through increased input from the C-fibres onto neurones in the dorsal horn. With continued stimulation, functional and structural changes to the neurones can occur which can alter the efficacy of neurotransmission. This can result in new synaptic connections being formed and is known as synaptic plasticity (McMahon & Jones, 2004;,Wall & Melzack, 1994).

New synaptic connections can cause the sensitivity of spinal neurones to incoming stimuli to increase. The characteristics of central sensitisation are a decrease in the stimulation threshold, an increase in cutaneous receptive fields and hyperactivity of spinal neurones (Herrero et al., 2000).

Peripheral and central sensitisation can result from tissue damage, such as that seen in a whiplash injury. Peripheral sensitisation can occur when inflammation of the tissues causes an increase in primary afferent output, which in turn can cause

an alteration in the function of the dorsal horn neurones. Both of these mechanisms can result in central sensitisation.

Subjects showing a generalised hypersensitivity to pressure, temperature, movement and skin blood flow following a whiplash injury have been shown to be more likely to develop chronic pain than subjects that showed normal sensitivity (Sterling et al., 2003a).

2.6.2   Muscle pain

For a detailed description of the anatomy of skeletal muscle see section 2.2

There have been many terms used to describe muscle pain including "muscular rheumatism", "idiopathic myalgia" and "fibrositis" (Travell & Simons, 1983).Two terms used to describe pain of muscular origin are currently popular: fibromyalgia and myofascial pain. Fibromyalgia is defined as "a form of nonarticular rheumatism characterised by musculoskeletal pain, spasms, stiffness, fatigue and severe sleep disturbance." (Anderson, 2002, p676). The definition of myofascial pain is: "Pain pertaining to muscle and its sheath of connective tissue or fascia." (Anderson, 2002, p1145). Both of these terms refer to muscles, associated fibrous tissue and pain.

In general, wear-and–tear of skeletal muscle myofilaments is repaired on an ongoing basis which is similar to all body tissues. In unaccustomed exercise, changes in the sarcomere arrangement by way of cytoskeletal disruption can be seen within 15 minutes of cyclic eccentric exercise (Lieber et al., 1996). Type IIB fibres are damaged more frequently than type I fibres. Subsequent damage is linked to inflammatory processes and change in excitation-contraction coupling within the muscle fibre. Macrophages migrate in and phagocytose defective myofibrils. This is accompanied by an increase in inflammatory markers in the injured muscle and later in the blood.

Delayed onset muscle soreness (DOMS) occurs 1-2 days after exercise while peak muscle damage is seen after 3 days. It is thought that the damage to the

connective tissue layers around the muscle causes an influx of fluid and raises the intramuscular pressure. This, alongside the release of inflammatory mediators, is thought to be the cause of the soreness (Herbert & de Noronha, 2007).

Ischaemia or stretch against a contraction (as can be seen in a whiplash injury) can result in intrinsic degeneration of the muscle fibre (Brault et al., 2000b; Proske & Morgan, 2001). When damage occurs the cell produces mitogenic factors that promote regeneration. Satellite cells situated under the basal lamina are activated where they then increase in size. The myofibrils break down into individual sarcomeric units, myonuclei die and the cellular contents are removed by leukocytes (mainly macrophages) which also release mitogenic factors. When all cellular debris has been removed the activated satellite cells combine with remaining basal lamina and a rapid regeneration of the muscle cell takes place (Carlson & Faulkner, 1983; Morgan & Partridge, 2003).

Muscle pain is assumed to be the result of increased sensitivity of local muscle nociceptors and/or central sensitisation. This is in accordance with established nociceptive processes in cutaneous tissue (Farasyn, 2007). Referred muscle pain can then result from central hyperexcitability, which reflects the mechanisms of central sensitisation due to facilitation of the A β-fibres and increased excitability of the motor neurone pool. The application of pressure to painful areas of the muscle, known as trigger points, can cause an increase in muscle nociceptor activity, possibly resulting in the spread of pain to other areas of the body (Staud et al., 2005).

Central pain modulation in fibromyalgia sufferers during exercise is different to that in normal controls, with the fibromyalgia participants displaying a lack of central pain inhibition during isometric exercise (Staud et al., 2005). It was proposed that the fibromyalgia participants either had a decreased efficiency in central inhibition or increased facilitation that overrides the normal inhibitory processes. However, trigger points do not need to have pressure applied to them to cause referred muscle pain. Travell and Simons (1983) describe myofascial pain as being a spontaneous pain that is referred from a trigger point to specific, distant reference zones.

Both myofascial pain and fibromyalgia are linked to non-traumatic neck pain and neck pain resulting from a whiplash injury (Banic et al., 2004; Bendsten, 2000; Dommerholt, 2003; Janda, 1988; McLean et al., 2005; McLean & Clauw, 2005; Vollenbroek-Hutten et al., 2006). It has been suggested that trigger points could be the source of the prolonged peripheral nociceptor input and could result in central sensitisation, a common phenomenon in persistent pain (Dommerholt, 2003).The discovery of high levels of nociceptive substances such as bradykinin and substance P as compared to control subjects in active trigger points supports Dommerholt's suggestion (Shah et al., 2005).

A review of the pathophysiology of upper extremity muscle disorders (UEMDs) states that possible causes of UEMDs are intra-cellular Ca^{2+} accumulation and impaired blood flow (Visser & van Dieen, 2006). Long-term, low-frequency stimulation has been shown to increase Ca^{2+} in rat muscle, especially in muscles with a predominance of Type II (fast-twitch) fibres. Increased levels of Ca^{2+} have been associated with membrane damage, which could result in pain due to the release of intracellular substances.

Long-term, low-frequency activity could be the result of muscle hyperactivity and an impaired ability to relax a muscle after use, as seen in the study by Nederhand et al. in 2002. Long term, low level contractions may result in a reduction in blood supply by increased intramuscular pressure. This is because blood flow can be impeded at pressures above those of systemic blood pressure. Differing pressures have been seen in the trapezius muscle, with higher pressures in areas of higher motor unit activity. It has been shown that prolonged pressure at low levels (for 8 hours at a pressure of 30 mmHg) resulted in damage to some muscle fibres at normal blood pressure (Visser & van Dieen, 2006). This review did not address the question as to whether these symptoms are a cause or a consequence of the UEMDs.

An effective and responsive circulation within muscle tissue enables optimal muscle function due to adequate oxygenation and removal of metabolites. Impairment of muscle blood flow could cause ischaemic pain due to the build-up of metabolites, exacerbated by lack of blood flow, which in turn could result in

avoidance of use of the painful muscle and lead to altered muscle use. This could be a contributory factor in the maintenance of chronic neck pain as explained by the fear avoidance model (Section 2.6.4.iii).

2.6.3   Initial pain after a whiplash injury

See section 2.5 for grading of a whiplash injury according to the presented pathology.

Reported symptoms following a whiplash incident vary in severity. The most frequent complaint is neck pain and headache, followed by shoulder pain and paresthesia in the arms and hands. Although more serious injuries can occur, such as fracture of the articular pillar or haemarthrosis of the zygapophysial or facet joint, these are much less common (Barnsley et al., 1994; Ferrari & Russell, 2003; Nederhand et al., 2003).

Zygapophysial or facet joint damage has been linked to chronic pain. However it has been reported that only 1 percent of whiplash patients suffering from chronic pain displayed abnormalities in the structures of the cervical spine when investigated with a variety of imaging techniques (Malanga & Nadler, 2002). In contrast, a study by Barnsley in 1995 showed that 54% of patients had painful zygapophysial joints following a whiplash injury. This study used nerve blocks at the zygapophysial joints to determine the origin of pain. Although this was a relatively small study of 50 patients, the results were replicated in a further placebo controlled trial by Lord in 1996, therefore increasing the reliability of the former study (Barnsely et al., 1995; Lord et al., 1996).

Soft tissue injury has been implicated in whiplash associated disorder (Barnsley et al., 1994; Eck et al., 2001; Spitzer et al., 1995). It is known that eccentric exercise when carried out vigorously enough can result in sore muscles. Muscles can be forcibly lengthened during this type of exercise and this has been shown to cause disruption to sarcomeres. The excitation-coupling system can also be damaged (Proske & Morgan, 2001). Muscle injury due to forcible lengthening has been shown to occur when participants were exposed to low-speed rear-end impacts: "The initial rearward retraction of the head relative to the torso resulted in

lengthening of the activated sternocleidomastoid, consistent with a contraction-induced injury" (Brault et al. 2000b, p426). When studying the effect of a rear impact on cervical spine specimens, it was found that following a whiplash-type trauma there was a significant decrease in ligament strength (Tominaga et al., 2006). Although this study was carried out on specimens, it highlights the potential for damage to ligaments in a whiplash injury in vivo. This could result in pain and a lack of stability of the cervical spine.

Despite evidence of soft tissue trauma due to a whiplash incident, it is debated whether this may be a contributing factor to the development of chronic or persistent symptoms. A study observed muscle pain and tender points in individuals for a year following a whiplash injury (Kasch et al., 2008). It was reported that individuals who developed chronic pain had more tender points and were more responsive to pressure induced pain in the masseter and trapezius muscles, than those who had recovered. Tenderness was also recorded in the recovered patients in the neck region and areas remote to the neck. However, it was not possible to differentiate between pain and tenderness that arose from normal activities and as a result of the injury. These results do shed some doubt to the extent that superficial muscle tenderness and pain are involved in the chronicity of pain following a whiplash injury.

It has been proposed that there is an interaction between pain and muscle activity in correspondence to the pain adaptation model (see section 2.6.4 for further detail on models of pain). This states that an increase in antagonistic muscle activity reduces movement and stabilises the area around the damage. This type of muscular adaptation has been observed following whiplash injury (Johansen et al., 1999; Nederhand et al., 2000; Nederhand et al., 2002; Sterling et al., 2003b)

Generalised hypoesthesia was identified in individuals with acute WAD, irrespective of localised hypersensitivity or pain and disability levels (Chein et al., 2008b). This study categorised high risk and low risk groups for poor recovery and persistent symptoms based upon sensory hypersensitivity and the neck disability index (NDI). Both groups showed similar patterns of hypersensitivity to pressure tests, as compared to the control group, along with elevated psychological distress

levels. Both groups demonstrated a decreased sensitivity to cold and electrical stimuli and to the ability to detect vibration at sites along the C6-C7 dermatomes. The authors implicate adaptations to central nervous system processing and suggest that raised levels of psychological distress may also be involved. However, this study observed patients 4 weeks after the injury and the findings can only be related to an acute injury. It would have been interesting to have seen if these effects were also present in individuals that developed chronic pain and those that did not.

A similar study which compared individuals with WAD and those with cervical radiculopathy reported that both groups demonstrated generalized sensory hypersensitivity and hypoesthesia (Chein et al., 2008a).These findings show that peripheral nerve damage could be a factor as well as abnormal central pain processing.

Spinal cord hypersensitivity and muscular hyperalgesia have been seen both in whiplash injury and fibromyalgia. This suggests that these conditions show similar pain mechanisms, which could arise from damage to the muscles or the neuronal response to a painful stimuli (Banic et al., 2004; Johansen et al., 1999).

It is unclear whether muscle pain causes hypersensitivity or whether the central hypersensitivity perpetuates muscle pain. It is possible that pain results from a vicious cycle of events caused by interaction between the motor and sensory systems. Chronic pain develops when this vicious cycle continues after tissue has healed.

2.6.4   Chronic pain

There are several terms used to describe long-term pain, including chronic and persistent pain. Recently, the term persistent is becoming more widely used than chronic as the latter term is said to imply (with the change of the use of language) "bad" pain. However, for the purposes of this study the term chronic pain will be used. Chronic pain is described as pain that has lasted longer than the expected

time for the damaged tissue to heal. This has been classified from 1 month to more than 6 months (Verhaaka et al., 1998).

Models of pain have been proposed to explain the transition of acute pain to chronic pain and these are explored in the following sections. The basis of these models is alteration of muscle activity and motor control due to painful stimuli.

2.6.4.i   The pain-spasm-pain model

The pain-spasm-pain model was first suggested by Travell in 1942 (cited in van Dieen et al., 2003), it proposed that muscle spasm can occur in response to a trauma with the purpose of partial immobilisation of the injured area. The increased muscle activity could then result in a raised pain threshold.

Two theories with different underlying neural mechanisms have been proposed to form the basis of the pain–spasm–pain model. The first theory states that muscle and its associated connective tissue are innervated by sensory afferents that transmit information to the brain and also synapse with interneurons, which then synapse with α motorneurons at the spinal cord level. Activation of α motorneurons in response to pain may result in reflexive hyperactivity of muscles in response to pain (Wyke, 1987).

The second theory proposes that muscle spindles are affected by nociceptor activity by way of direct excitation of the γ motorneurons. Increased muscle spindle activity results in hyperexcitability of the α motorneuron pool (Johansson & Sojka, 1991).

These theories both describe hyperexcitability of the α motorneuron pool in response to pain, which could result in increased muscle activity. The increased activity could then result in further pain through accumulation of metabolites and changes in the pH of the environment. However, these theories do not have to exist in isolation and it is quite reasonable that both of the proposed mechanisms occur simultaneously. One limitation of this model is that it does not take activity of

other muscles into account, which may be affected by the altered activity of the muscle in question.

There have been many studies conducted on both animals and humans to verify the pain-spasm-pain model with mixed results. The majority of animal research has been undertaken on decerebrate or anaesthetised animals which would seriously impair any higher nervous system input (van Dieen et al., 2003).

Individuals who had suffered a whiplash injury and had subsequently developed persistent pain were less able to relax the upper trapezius muscle after exercise. WAD participants showed higher baseline levels of muscle activity during rest. This could be a result of muscle spasm or hypertonicity that occurs in response to pain (Nederhand et al., 2000).

Altered activation of muscles may affect the fibre type proportions of a muscle. With increased low-level use, fibres can differentiate into the more fatigue resistant fibre type (Uhlig et al., 1995).

2.6.4.ii   The pain-adaptation model

The pain adaptation model disputes that muscular pain following trauma or injury is maintained by an increase in muscular activity. Conversely, it proposes that the activity of the affected muscle decreases in the presence of pain and that pain can cause an increase, albeit small, in activity levels of the antagonist muscle. This suggests that muscle dysfunction observed in chronic and painful musculoskeletal conditions is a normal protective adaptation and not a direct cause of pain (Lund et al., 1991).

A study by Nederhand et al. (2003) explored the muscle activation patterns of the upper trapezius muscle in patients following a whiplash injury. This group was divided into four subgroups dependent on the level of recovery. They found a greater reduction of muscle activity in the upper trapezius in the patients showing the lowest levels of recovery (established using the neck disability index). It was proposed that the alteration in the use of this muscle could be explained by the

pain adaptation model whereby use of the painful muscle was reduced (Nederhand et al., 2003).

Incorporation of the pain adaptation model in movement was also demonstrated with the use of hypertonic saline injections to induce experimental muscle pain. Saline was injected into the trapezius muscle and resulted in decreased activity during low load repetitive tasks (Madeleine et al., 1998). This decrease in muscle activity is in accordance with the pain adaptation model. Experimental pain is an immediate insult as opposed to chronic pain, which means that it will not involve the changes in neuronal plasticity seen in chronic pain, this makes comparisons problematic.

Gender differences have been reported in the reorganisation of muscle activation, as a result of experimental pain, whereby men show a more efficient reorganisation of muscle than women (Falla et al., 2008; Ge et al., 2005). Men appear to have a more efficient muscular adaptation to experimental pain, reducing the activity in painful areas and increasing activity in the non-affected subdivisions of the upper trapezius, this is in accordance with the pain adaptation model. Women do not show such efficient reorganisation and this could be related to the increased incidence of chronic neck pain in women.

2.6.4.iii The fear avoidance model

Acknowledgement of the importance of psychosocial factors in the development of chronic pain has recently increased (Cook et al., 2006; Croft et al., 2003; Ferrari & Schrader, 2001; Kerns et al., 2006; Nederhand et al., 2004; Nederhand et al., 2006;,Vlaeyen & Linton, 2006).

The fear avoidance model, which incorporates the physiological aspects of the pain response as well as psychological components of pain, was developed to explain how chronic musculoskeletal pain arose after the apparent recovery from tissue trauma. The model proposed that when pain resulting from an injury is perceived to be threatening, which is also known as pain catastrophising, a pain-related fear can develop leading to avoidance behaviours of the affected muscles.

Such perceptions and resulting actions can then be followed by disability, disuse and depression (Vlaeyen & Linton, 2006).

Such responses to a situation can cause a vicious cycle of fear and avoidance to develop. However, individuals who show non-catastrophising behaviour e.g. not showing fear in response to pain and confronting daily activities, show a faster recovery (Vlaeyen & Linton, 2006). This model was based upon self reports of pain and disability, attention to bodily sensations, guarded movement and muscle activity. However, this model did not consider those individuals who do not show fear avoidance but continue to present with chronic pain. Figure 2.4 shows the original fear avoidance model proposed in 2000 by Vlaeyen et al.

Figure 2.4 The fear-avoidance model of chronic pain. Vlaeyen, J. W. S. & Linton, S. J. 2006, Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain*, vol. 85, pp. 317-332.This figure has been reproduced with permission of the International Association for the Study of Pain® (IASP®). The figure may not be reproduced for any other purpose without permission.

According to the fear avoidance model deep muscle pain in the neck may lead to a reduction in use of those muscles with a subsequent increase in use of the superficial neck muscles (see section 2.2.3 on innervation of skeletal muscle). Impairment of the deep cervical flexor muscles was reported in subjects with insidious neck pain and whiplash injury performing the craniocervical flexion test (CCFT) (Jull et al., 2004). The neck pain groups also showed a significant increase in sternocleidomastoid muscle activity as compared to healthy controls. A pressure

test was used to determine deep muscle activity rather than a direct measure from muscle, which makes direct associations between pain and deep flexor muscle activity difficult. A study be Fernandes-de-las-Penas et al., (2008) reported that females with bilateral chronic neck pain had smaller cross-sectional areas of the cervical multifidus muscles as compared with healthy females. Fatty infiltration of deep cervical muscles in individuals that have suffered a whiplash injury has also been reported (Elliott et al., 2006). This could indicate a lack of use, therefore innervation, of the deep cervical muscles following an injury. This is in accordance with the fear avoidance model and could perpetuate problems due to weakening of the deep muscles.

A study that recorded simultaneously from deep and superficial flexor muscles during the CCFT reported that participants with neck pain showed a decrease in deep flexor activity (longus colli) and a trend for increased sternocleidomastoid activity (Falla et al., 2004b).These findings support the fear avoidance model, however, the methodology used maximum voluntary contraction (MVC) to normalise the subsequent contractions. The ability of individuals with neck pain to be able to or to want to perform MVC is questionable; pain may prevent a true maximal response. The contraction was against a force and is not representative of everyday neck activity; as such it may not be reflective of changes in more natural activities.

The fear avoidance model has been applied to individuals with both persistent non-traumatic neck pain (Linton & Ryberg, 2001) and in neck pain following a whiplash injury (Nederhand et al., 2004; Nederhand et al., 2006). Linton and Ryberg (2001) compared cognitive behavioural therapy (CBT) with a group receiving physical therapy of their choice. Although CBT was shown to improve the chances of not developing disability, and therefore supporting the fear-avoidance model, there were several aspects in the design of this study that could affect the reliability of the findings. The participants were recruited from the general population, as opposed to a pain clinic, this meant that although the participants were quite representative of the normal population, their reported pain and disability was minimal. Therefore the results of this study cannot be inferred on the more serious chronic pain cases. The physical therapy received was not controlled and therefore

assumptions of the effectiveness of treatment cannot be made. Finally, it would have been interesting to see the effect of controlled treatment in conjunction with the CBT to determine the efficacy of combined treatment.

In further support of the fear avoidance model Nederhand et al., (2004) used the Neck disability index (NDI) and the Tampa scale of Kinesophopbia to measure perceived disability and fear of movement respectively. The questionnaires were undertaken 1 week after sustaining a whiplash injury and then again 24 weeks after the accident. The study reported that it was possible to predict the development of chronic pain from the initial questionnaire scores.

A follow on study (Nederhand et al., 2006) looked at participant responses, post whiplash injury, at 1, 4, 8, 12 and 24 weeks. The NDI, Tampa scale of Kinesophopbia, visual analogue scale (VAS), for pain intensity, and EMG for muscle activity were used to determine the possible development of chronic pain. It was shown that both the pain intensity (VAS) and fear of movement were associated with decreased muscle activity. This supports both the fear avoidance model and the pain adaptation model where movement of painful muscles is reduced. However, a clearer picture of fear avoidance would have been shown with identification of the location of the pain. The participants reported pain in the neck and shoulder region, but not specifically from the muscles that the recordings were made.

There appears to be some degree of cross over between the pain adaptation model and the fear avoidance model. The pain-adaptation model looks at the neuronal response to pain and the fear-avoidance model looks at the behavioral response. It may be possible that both models apply in the same instance and that the reorganisation of motor strategies shown in the former could be a result of the behavioral response of the latter.

Further evidence in support of the fear avoidance model was reported when subjects received 4 weeks of myofeedback training; whereby muscle activation was visualized to enable participants to see the effects of relaxation of muscle activity. Following training, a decrease in perceived pain in the upper trapezius

muscle, both at the time of testing and 3 months training after stopped, was reported (Vollenbroek-Hutten et al., 2006). However, this study reported that the decrease in perceived pain was not correlated with a decrease in muscle activity during computer tasks. The study also reported a trend between decreased pain and increased ability to relax the upper trapezius muscles. This ability to relax could result in muscle activation being less painful due to decreased muscle fibre fatigue and decreased levels of metabolites. The results could also suggest that psychological factors could have been an influence on pain perception and when subjects felt they had some control of their pain, the self perception of pain decreased. This study did not have a control group as it was interested in the effect of training in individuals with pain. It would have been interesting to see if pain-free individuals also responded to the myofeedback training, and whether an inability to relax muscle may not be accompanied by chronic pain.

Similar results were seen in individuals with chronic pain resulting from a whiplash injury (Voerman et al., 2006). The study reported a significant decrease in self reported pain and disability following 4 weeks of myofeedback training. A decrease in muscle activity at rest, after performing computer tasks, and during relaxation was also shown. However this study had a small sample size and no control group which makes inference on a wider population difficult.

The effects of myofeedback training and ergonomic counseling were studied in female computer workers to determine the effectiveness of these treatments on self-perception of pain and disability (Voerman et al., 2007). Participants were either placed in the myofeedback or ergonomic counseling group. The results showed that after four weeks of treatment self-reported pain and disability in both groups was significantly reduced. However, there were no differences reported in the efficacy of both methods of treatment. These results imply that fear avoidance can be overcome by increased relaxation and improvements in posture.

2.6.4.iv The avoidance - endurance model

An extension of the fear avoidance model, the avoidance-endurance model, was proposed by Hasenbring in 2000. This model states that endurance-related

responses to pain, where pain is ignored and painful muscles become over used, sits alongside the fear avoidance response. It is suggested that individuals in pain can cognitively suppress pain and its effect on daily life; this is deemed reflective of a positive outlook. However, overuse of muscles and tissues that are painful, due to existing damage, can cause a further increase in pain. Alternatively, fear avoidance can lead to frequent pain-related thoughts; this can lead to a sense of failure and possibly depression (Hasenbring, 2000). This adds a third dimension to the fear avoidance model, in addition to pain arising from disuse and recovery, another possible outcome is pain in response to overuse.

A questionnaire for avoidance-endurance behaviour has been developed, piloted and shown to be valid but has yet to be used in longitudinal studies (Hasenbring et al., 2009). This could prove to be a very useful tool as it assesses pain, disability as well as coping strategies and could be used in conjunction with objective measures of muscle activity to provide further insight into the development of chronic pain.

By observing synergistic flexor and extensor muscle activity (such as the sternocleidomastoid and upper trapezius muscles) during exercise it may be possible to see whether chronic neck pain affects motor control strategies in accordance with models of pain.

2.6.5   Chronic pain after a whiplash injury

Studies into the development of chronic pain following a whiplash injury show that 19 to 60% of whiplash injuries result in complaints of chronic pain after 6 months (Klein et al., 2001; Scholton-Peeters et al., 2003). Different types of chronic pain have been classed as one subgroup known as functional somatic syndromes (Croft et al., 2003). These syndromes are characterised by the level of distress and symptoms rather than apparent tissue damage. They are associated with pain catastrophising, which has been reported to be a major factor in long term pain and disability. This occurs when an individual has very negative thoughts about their situation and perceives minor difficulties as major adverse events (Flor &

Turk, 2006). This is in accordance with the fear avoidance and the avoidance-endurance models.

Separation of the constructs that form the biopsychosocial model, placing pain catastrophising in the "psychologic" category is thought to be incorrect. All of the factors involved appear inextricably linked and pain catastrophising may affect the biologic processes and vice versa. The "neuroendocrine, neuroimmune, psychophysiologic and functional neuroanatomic effects of catastrophising of the pain experience" have been implicated in the development of chronic pain. (Campbell & Edwards 2009, p100). This demonstrates a truly holistic approach to pain and shows that the potential for pain catastrophising can in turn influence physiologic pain mechanisms. Psychosocial factors alongside physiological responses, which are involved in pain processing and the perception of pain, are important considerations when observing subjective, self-reports of pain.

Hypersensitivity, due to plasticity changes in the central nervous system, has been demonstrated by a reduction of spinal reflex thresholds in patients suffering from chronic pain after a whiplash injury and fibromyalgia as compared to healthy controls (Banic et al., 2004). The use of spinal reflexes as an outcome measure is more quantifiable than palpation, which has cortical involvement and indicates an increase in spinal cord sensitivity. Banic et al., (2004) also performed personality trait and psychological profile tests and found that all groups scored in the normal range for personality traits but the two pain groups showed significantly elevated levels of psychological distress compared to the controls. These findings reinforce that pain catastrophising may affect the physiology of the nervous system; although it could be that the chronic pain results in psychological changes as described by the fear avoidance model.

A study by Johansen et al. (1999) has shown that not only local, but also generalised hypersensitivity (in the form of muscular hyperalgesia) in large referred pain areas occurs in chronic whiplash patients. This was demonstrated using pressure and pin prick threshold. The results of this study showed that patients with chronic pain had a significantly greater sensitivity to experimentally induced pain than healthy control subjects (Johansen et al., 1999). Although this study

used experimental pain, the results could indicate wider changes in neuronal sensitivity in the CNS or an increase in pain catastrophising in individuals with chronic pain resulting from a whiplash injury. The use of psychological questionnaires may have discriminated between these two possible causes.

In a review of whiplash pathology, doubt was expressed as to whether muscle damage could be involved in the development of chronic pain. This is because muscle injuries should heal within a matter of weeks, leaving the patient with no lasting pain (Barnsley et al., 1994). However, it is apparent that muscle damage and the consequent reactions to the initial damage could be a major factor in the development of chronic pain. Muscle injury has been reported to lead to myofascial pain or trigger point development, which could be involved in the development of chronic or persistent symptoms (Dommerholt, 2003).

Home exercise programmes which used a group of 59 symptomatic patients with an acute whiplash injury were compared to gauge their effectiveness. One group were given a standard treatment plan and the other group were given additional exercise as part of their treatment. Although there was very little difference in levels of improvement between the two groups, those participants that were asymptomatic at 6 months initially showed higher self-efficacy characteristics and different patterns in coping strategies (Soderland et al., 2000). In agreement with the fear avoidance model, the results of this study imply that early identification of those patients with poor coping strategies could enable specific treatment plans that are designed to address such psychological factors, thereby increasing the chances of a more successful recovery.

A series of studies investigated psychological changes following whiplash injury. It was initially found that psychological distress was presented in all participants that had experienced a whiplash injury. However, it only persisted in those participants whose symptoms continued (Sterling et al., 2003c). This could be explained by the continuation of symptoms causing the distress, or it could be the personality type lending itself to the perpetuation of the condition. In a subsequent study, Sterling et al., in 2005 found that 20-25% of participants had developed sensory and pain processing changes, loss of movement and higher levels of reported pain following

a whiplash injury. This study concluded that those participants who showed poor recovery had experienced a more complex injury that led to the aforementioned changes (Sterling et al., 2005). The results of the studies by Sterling et al. could show that some individuals develop coping strategies post-injury which minimise future distress. This could also support the association between psychological and physiological symptoms as previously suggested by Campbell and Edwards (2009). The most recent study by this group found that at 2-3 years post injury, only NDI scores given at the initial assessment were a significant predictor of the development of chronic pain (Sterling et al., 2006). In accordance with the fear avoidance model, this study also reported sensory, motor and psychological impairment of those participants with chronic pain following a whiplash injury, this reinforces the findings of the earlier study by Sterling et al. in 2005.

High initial pain intensity scores have been shown to be significant as a prognostic factor for chronic pain. Evidence that age, gender, psychological response and compensation did not serve as predictors for development of chronic pain was also presented. Factors such as coping strategies, anxiety, educational level and cognition were not sufficient to be able to draw conclusions regarding pain prognosis (Scholton-Peeters et al., 2003).

Acute stress reactions at the time of injury have been linked to the development of chronic neck pain in individuals with WAD (Kongstead et al., 2008).This corresponds with Soderlund (2000) and implies that the way an individual copes immediately after the accident is an important consideration for early management and treatment of a whiplash injury. The manner that an individual responds to and copes with injury and pain can be affected by many factors. The wealth of material available to the public regarding the potential for chronic pain after a whiplash injury may produce a pre-conscious expectation that leads to symptom amplification. This study also identified the feeling of a lack of control arising from patient fear, generated by initial reaction of paramedics (application of neck collar and immobilisation). In addition they also identified the language used by doctors as a major factor in the development of chronic pain (Ferrari 2002). These findings relate to the findings of Vollenbroek-Hutton et al. (2006) who found patients that felt in control reported less pain.

Psychosocial factors have been used to predict the outcome of long-term pain and disability. Sterling et al. (2003) measured psychological distress for 6 months post-injury and found that those participants with ongoing moderate to severe pain had higher levels of psychological distress. However, the study did not indicate whether it was chronic pain that caused psychological distress or if the individual's distress resulted in the perpetuation of pain. Unfortunately, unless psychological profiles are undertaken before an injury occurs it is almost impossible to state whether the psychology of an individual affects the pain response or vice versa.

Cultural expectation has been forwarded as a contributory factor for the development of chronic pain following a whiplash injury. This is a result of the fact that in some countries ongoing symptoms do not appear to be such a common phenomenon. In Lithuania, for example, where only 6-11% reported developing chronic pain, there is little acknowledgement within the general population that such an injury can lead to chronic pain and disability (Obelieniene et al., 1999; Schrader et al., 1996). In Germany there is little expectation of the development of chronic symptoms following whiplash injury. When the symptoms of a group undertaking physiotherapy were compared with those in a control group who received no treatment, the reported symptoms of both groups were equal at 6 weeks (Ferrari & Schrader, 2001). In Greece, a study of 130 patients with acute whiplash injury found that 91% had recovered in 4 weeks with the remainder showing neck pain symptoms on a par with the general population by 3 months (Partheni et al., 1999). In contrast to these studies, it has been reported that only those patients that presented with neck pain on palpation, muscular pain and paraesthesia had a longer course of recovery compared to those patients with few or milder symptoms (Suissa et al., 2001).

These findings cast doubt as to whether the chronic symptoms reported in some countries are related to the injury sustained at the accident or whether it could be due to cultural expectation, treatment, attitude, litigation matters or a combination of these factors. It appears to be accepted that chronic pain as a result of a whiplash injury is not a fictitious condition. However, it is possible that an individual's response to the original injury can have an effect on the incidence of chronic pain as seen in the research cited in this section.

Support for the holistic biopsychosocial approach to the treatment and management of neck pain was recently reported (Jull & Sterling, 2009). Pain is often a presenting complaint but often it is not possible to image or determine the pathoanatomy. It was stated that there should not be a singular line of management but that a multimodal approach to the pain state and to recovery should be used.

2.6.6 Models of disability

Models of disability have been proposed in order to account for all aspects of the development of pain which have been discussed in the previous sections. These models provide a more holistic approach to the treatment of chronic pain and its effect on an individual's life.

2.6.6.i The Simmonds model of disability

The management and measurement of musculoskeletal pain is shown in the Simmonds bidirectional model of disability (Figure 2.5). This model gives a broad overview of the effects of musculoskeletal pain on the individual and the interdependence of the relevant components, showing that functional limitation can affect disability, pathology and impairments. Therefore, improved function can aid improvements in pain. The Simmonds Model of disability is directed towards the therapist, incorporating a multidimensional, or biopsychosocial approach to treatment (Gifford et al., 2006).

2.6.6.ii The model of International Classification of Function (ICF)

The International Classification of Function (ICF) developed a model of disability (Figure 2.6) that incorporates the medical model, which sees the disability that results from disease or trauma as a feature of the individual It also incorporates the social model which views disability as a problem created by social response rather than an aspect of the person (Ustun, 2007). This model incorporates three levels of functioning in the person: the level of the body or body part, the level of the whole person and the level of the person in a social context.
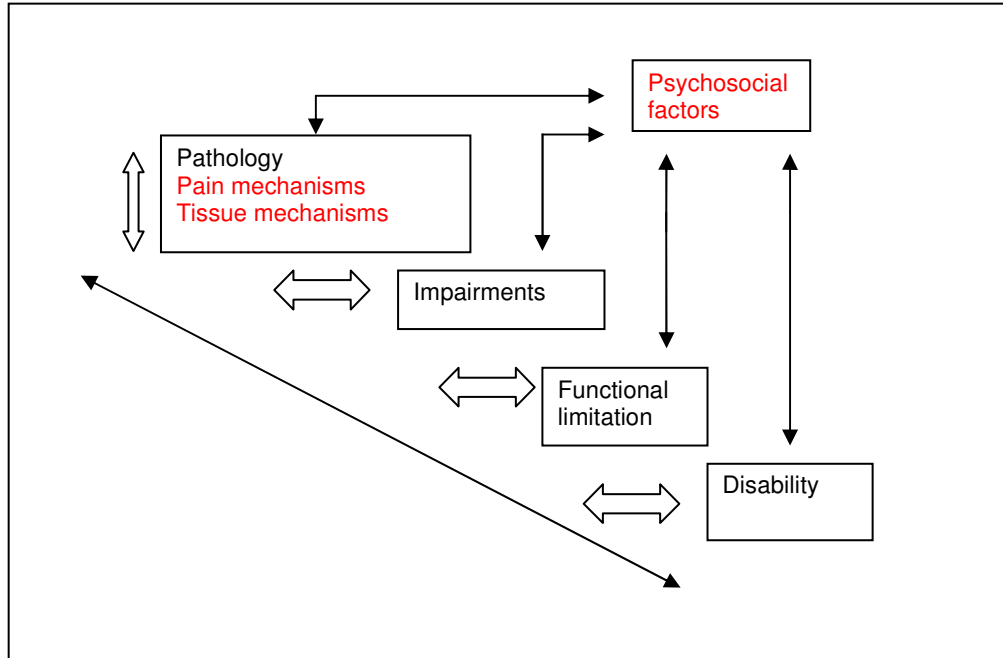
Figure 2.5 The adapted Simmonds bidirectional model of disability. *The addition of pain mechanisms, tissue mechanisms and psychosocial factors (in red type) to the Simmonds bidirectional model of disability.* Reproduced with permission from: Gifford, L., Thacker, M., & Jones, M. A. 2006, "Physiotherapy and pain," in *Wall and Melzaks textbook of Pain*, 5th edn., Elsevier Churchill Livingstone.

A fundamental difference between the Simmonds and the ICF model its focus. The focus of the Simmonds model is the disability or the level of dysfunction, whereas the focus of the ICF model is the level of an individual's capacity and how they perform in their normal environment (Ustun, 2007). It is possible that by focusing on the disability, rather than on a person's ability, pain catastrophising could be induced.

This could be related to the fear avoidance model in the development of chronic pain, whereby pain causes avoidance of use of a particular muscle which could lead to disuse atrophy of that muscle and over reliance on other muscles. The models of pain suggest that early identification of pain and a patient's perception of that pain may help to establish the most appropriate course of treatment for an individual.

Health Condition
(Disorder of disease)

Body Functions and Structures

Activity

Participation

Environmental Factors

Personal Factors

Contextual Factors
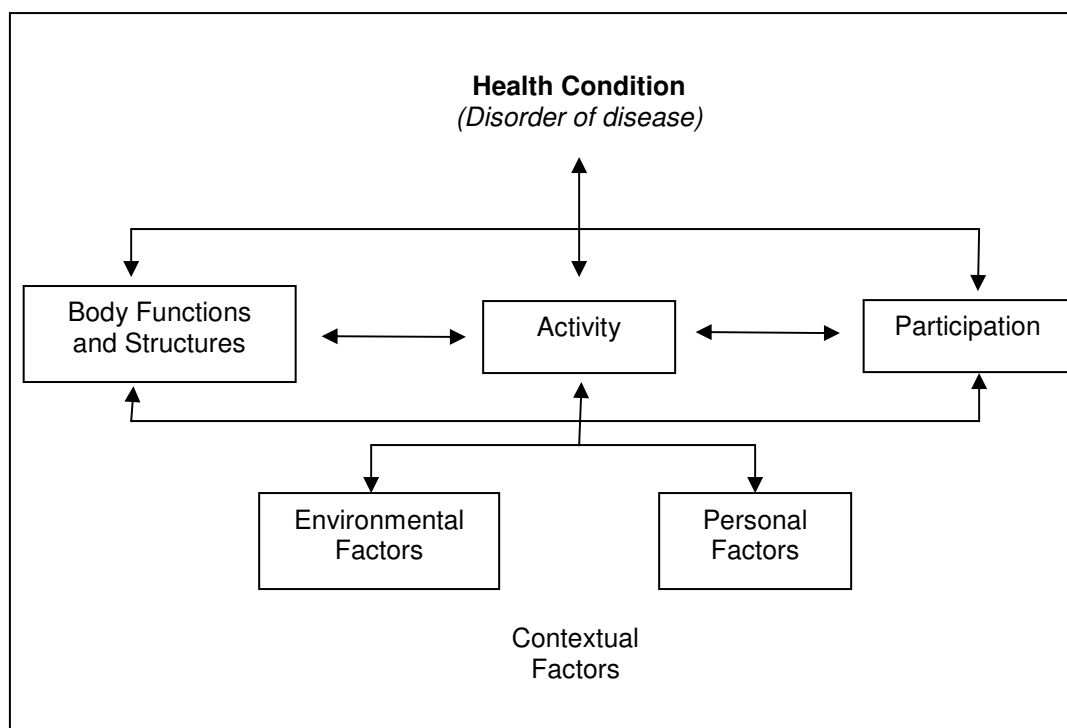
Figure 2.6 The International Classification of Function - Model of Disability. Reproduced with permission from URL (http://www.who.int/classifications/icf/site/beginners/bg.pdf) (date accessed 01/02/2007)

2.6.7 Methods of pain and disability assessment

Initially it would be advantageous to the patient and the practitioner to be able to identify and measure levels and intensity of pain. However, there is no reliable objective method of pain assessment as the experience of pain is personal and subjective. Common subjective methods of pain assessment include visual analogue scales (VAS), numerical rating scales (NRS) and verbal rating scales (VRS) (Melzack & Katz, 2006). More detailed measures of pain and disability include the Neck Disability Index (NDI) and the McGill Pain Questionnaire.

2.6.7.i The visual analogue scale (VAS)

Use of the VAS is widespread and due to its simple, continual line, construction it can be used to assess several dimensions of pain, e.g. intensity or discomfort. This scale has a numerical basis, with 0 being no pain and 10 being worst possible

pain. This type of scale allows inter and intra subject comparisons and monitoring of symptom progression. There are no graduations on this scale, therefore avoiding practice effects, i.e. individuals remembering a previous score.

A recent study investigated the reliability and validity of the VAS for measuring pain and disability in musculoskeletal pain patients (Boonstra et al., 2008). It concluded that it test retest reliability was moderate to good. However, because of a weak correlation with other disability measures they found the validity of this method questionable.

To use this method during exercise the participants would have to be able to physically indicate on the visual analogue scale their pain intensity. This may not always be practical as some activities may not be conducive to writing scores while performing them. This is a measure suited to longitudinal studies that compare physical outcome measures with the pain felt by the subject.

2.6.7.ii The verbal and non-verbal numerical rating scale (NRS)

The NRS (Appendix A) requires participants to indicate a number on a scale between 0 – 10 that reflects their pain intensity (with 0 being no pain and 10 being worst possible pain). This can be written or spoken, so it is not problematic during exercise when participants would be unable to give written answers. The use of the visual analogue scale was compared to the numerical rating scale and both formats showed good reliability and validity (Hollen et al., 2005).

2.6.7.iii The neck disability index (NDI)

The NDI (Appendix B) is an adaptation of the Oswestry Low Back Pain Index (Vernon & Mior, 1999). It is widely used to assess the effect of pain on day-to-day living and quality of life. It is used as a tool for assessing the functional status of the neck and has been used in the investigation of coping strategies in patients with whiplash injury (Nederhand et al., 2002; Nederhand et al., 2003; Soderland & Lundberg, 2003; Vernon & Mior, 1999). It is a questionnaire that contains 10 sections describing aspects of daily living. Each section is rated out of 6, with a

maximum score of 60, and could be used to investigate correlations with other pain measures such as the VAS and the NRS. This enables possible links between pain and disability to be established.

2.6.7.iv The McGill pain questionnaire

The McGill pain questionnaire employs verbal descriptors to describe pain felt by individuals and has been used to describe chronic pain following a whiplash injury (Johansen et al., 1999). Verbal descriptors provide an insight into how subjects' perceive pain, however they may be influenced by the subject's attitude to their pain and ability to verbally describe how they feel. This could in turn influence the potential validity of the results. However, repeated testing has shown this questionnaire to have a high degree of reliability and is useful in assessing the initial status of subjects. Although it is useful in assessing an individual's perception of pain, it may not be the best method to monitor the progression of pain in correlation with other methods of assessment.

Assessment of pain and disability can provide information regarding the perception of pain and the effect of pain on an individual's life. However, the information is subjective in nature and can therefore be subject to limitations associated with self-report measures. These include social desirability and researcher bias. This can lead to false reporting of scores due to participants giving responses which are seen to be socially acceptable, or to please the researcher. These factors may affect the validity of results. However, self-report methods of pain and disability assessment have been shown to be effective (Hollen et al., 2005; Vernon & Mior, 1999). Such information can be used alongside other outcome measures to assess potential differences and consequence of onset in the development of chronic pain.

2.7   A comparison of traumatic and non-traumatic neck pain

The purpose of the following section is to detect differences in outcome measures for studies involving neck pain from a traumatic onset (whiplash injury) or from

non-traumatic onset. This may enable identification of responses in chronic pain development in order to establish possible relationships between them.

Studies have been identified which include both whiplash (traumatic onset - TO (WAD)) and insidious (non-traumatic onset - NTO) groups. These studies have identified similarities and differences in outcome measures (Tables 2.4.1, 2.4.2, 2.5.1 and 2.5.2). The revised abbreviations in this section are used to clearly identify different sub groups within the insidious or non-traumatic groups.

Similarities and differences have been acknowledged in pain measures. TO (WAD) and NTO showed decreased pressure pain thresholds over the area of the cervical spine (Scott et al., 2005). However, this study also reported that TO (WAD) showed significant increases in remote hypersensitivity to temperature as compared to the NTO group. This could indicate that the mechanisms of central hypersensitivity differ between the two groups. Increased spinal cord hypersensitivity was reported in both TO (WAD) and NTO (Fibromyalgia (FM)) groups compared with the control group, yet no significant differences in pain thresholds between the groups were found (Banic et al., 2004). No significant differences were reported in lower back pain accompanying neck pain between three groups (TO (WAD), TO and NTO) but these groups did report a higher rate of lower back pain than the normal population (Guez et al., 2006).

Differences in cerebral blood flow, as measured with magnetic resonance imaging (MRI) scans, were observed (Sundstrom et al., 2006). The NTO group displayed a decreased flow in the right temporal region and an increase in flow in the right insula as compared to the TO (WAD) group. The insula receives afferent input from the thalamus and the temporal region is involved in memory formation. Increased blood flow in these areas could indicate a difference in higher pain processing and pain perception.

These studies implicate increased sensitisation and response to pain in individuals with chronic pain and that in some circumstances this may differ with type of onset. Such differences could be due to the amount of, or cause of, tissue damage. This could also affect the response of the individual to their pain.

Analysis of psychological profiles and self reports of pain showed that TO (WAD) had a significantly higher number of males than the NTO (FM) group and that they reported higher levels of disability. This study also showed no differences in a number of measures, such as body mass index, marital status, education levels, physical activity and psychological work situations (Guez et al., 2003). Psychological profile tests between TO (WAD) and NTO (mixed musculoskeletal pain, MMP) groups showed that both of these groups had elevated levels in the somatisation, depression, obsessive-compulsive and psychotism scales (Peebles et al., 2001).

Minnesota multiphasic personality inventory tests (MMPI) were also used to ascertain possible differences in personality traits between TO (WAD) and NTO groups. No significant differences were found between the groups but both showed higher scores in comparison to a healthy control group, which indicates exaggerated psychological complaints (Guez et al., 2005). However, it was reported that the NTO group showed better scores for coping strategies, possibly implying that the traumatic nature of neck injury impacts negatively on how an individual responds to that injury and subsequent development of pain.

These studies provide examples of the many aspects that can be tested by the psychological tests available. Firm conclusions can be difficult to make due to the many different patient groups and study designs together with self-report limitations. Biomechanical outcome measures have been used to identify differences between the TO and NTO chronic neck pain groups. Differences have been identified with increased angle of cervical flexion, increased segmental motion, decreased natural head repositioning, decreased active range of motion, increased sway, increase jerk index and repositioning error in the TO groups (Dumas et al., 2001; Kristjansson et al., 2003a; Kristjansson et al., 2003b; Kristjansson & Jonsson, 2002;,Sjolander et al., 2008).

Table 2.4.1. Studies that reveal no differences between traumatic onset and non- traumatic chronic neck pain groups				
Authors	Subjects	Outcome measures	Results	Comments
Elert et.al 2001	23 TO (WAD) 36 NTO (FM) 27 HC	Muscle activity and muscle tension EMG	TO (WAD) and NTO (FM) groups showed elevated muscle activity compared to HC.	This study identified identified that not all chronic pain patients exhibited increased muscle tension
Dumas et al. 2001	(headache of cervical origin) 20 TO (WAD) 24 NTO 16 migraine* 17 HC	Strength and endurance of the cervical flexors Dynomometer recordings	TO (WAD) and NTO showed a significant decrease in flexor strength and endurance compared to HC	This study also identified differences between TO and NTO groups (Table 2.5.1)
Peebles et al. 2001	67 TO (WAD) 91 NTO (MMP)	SCL-90-R psychological profiles	No significant differences in psychological profiles between TO (WAD) and NTO (MMP) groups	Both groups showed elevated levels in Somatization, depression, obsessive- compulsive and psychoticism scales. Not all MMP participants had neck pain.
Nederhand et al. 2002	19 TO (WAD) 18 NTO 18HC	Muscle reactivity or pre and post exercise muscle activity EMG	No significant differences between TO (WAD) and NTO groups	This study showed that WAD patients had a tendency for higher muscle reactivity.
Guez et al. 2003	249 TO (WAD) 565 NTO (FM)	Sociodemographic data survey KARASEK questionnaire	No difference in BMI, Marital status, educational level, smoking habits, psychosocial work situation or levels of physical activity	Differences identified between TO and NTO groups (Table 2.5.1)
TO Traumatic onset, NTO non-traumatic onset, FM fibromyalgia, WAD whiplash associated disorder-II, HC healthy control MMP Mixed musculoskeletal pain * this study also included a migraine group that is not discussed here.				

Table 2.4.2. Studies that reveal no differences between traumatic onset and non- traumatic chronic neck pain groups				
Authors	Subjects	Outcome measures	Results	Comments
Banic et al. 2004	27 TO (WAD) 22 NTO (FM) 29 HC	Reflex thresholds pain thresholds	TO (WAD) and NTO (FM) showed significantly decreased reflex thresholds compared with HC	No difference in pain thresholds between WAD, FM and controls
Jull et al. 2004	25 TO (WAD) 25 NTO 25 HC	Cranio-cervical flexion test EMG	TO (WAD) and NTO showed significantly increased muscle activity compared to HC	Control groups obtained expected pressure targets unlike both TO (WAD) and NTO groups during the test
Scott et al. 2005	29 TO (WAD) 20 NTO 20 HC	Central hypersensitivity	TO (WAD) and NTO groups showed decreased pressure pain thresholds over the cervical spine compared to HC	Differences between TO (WAD) and NTO groups also seen (Table 2.5.2)
Guez et al. 2005	21 TO (WAD) 21 NTO	MMP1-2 neuropsychological tests	NTO and TO (WAD) showed no difference in the neuropsychological tests	NTO showed better coping scores that TO (WAD). Both groups scored higher than HC reference group
Sjolander et al. 2008	7 TO (WAD) 9 NTO 16 HC	ROM ROM variability Jerk index	No Significant differences in ROM between groups. Significant differences in Jerk index and repositioning error between chronic pain (TO and NTO) and HC	The author note differences that were not statistically significant and suggests this may be due to the small number of participants
Guez et al. 2006	81 TO (WAD) 105 TO 566 NTO	Pain questionnaire	No differences seen in prevalence of low back pain between TO (WAD), TO and NTO groups	This study states that there are significantly higher rates of low back pain in chronic neck pain patients than in the general population
Woodhouse and Vasseljen 2008	59 TO (WAD) 57 NTO 57 HC	ROM variability JPE Conjunct motion	Reduced conjunct motion seen in the TO (WAD) and NTO as compared to HC group. No group differences in ROM variability and JPE	Differences between TO (WAD) and NTO groups also seen (Table 2.5.2)

Table 2.5.1. Studies that reveal differences between traumatic onset and non- traumatic chronic neck pain groups				
Authors	Subjects	Outcome measures	Results	Comments
Dumas et al. 2001	(headache of cervical origin) 20 TO (WAD) 24 NTO 16 migraine* 17 HC	Cervical AROM	TO (WAD) showed significant decrease compared to NTO and HC groups for AROM in the transverse and sagittal plane also showed increased extensor strength	This study also identified similarities between TO and NTO groups (Table 2.4.1)
Kristjansson et al. 2002	41 TO (WAD) 39 NTO 40 HC	Angle of cervical flexion at C4-C5 Ratio of upper and lower cervical lordosis	TO (WAD) had significantly greater angle of flexion than NTO and HC groups	Although not significant WAD showed a decreased ratio of upper to lower cervical lordosis compared to NTO and HC
Kristjansson et al. 2003	41 TO (WAD) 39 NTO	Sagittal and translational segmental motion	Significantly more TO (WAD) subjects had abnormal increased segmental motions than NTO	These results included translational and rotational when only translational were analysed no significant differences were found
Kristjansson et al. 2003	22 TO (WAD) 20 NTO 22 HC	Natural head posture (NHP) repositioning	TO (WAD) showed a trend for greater deficit in repositioning sense than NTO and HC	4 other tests were used with no differences found between all groups. These test were not established unlike NHP
Guez et al. 2003	249 TO (WAD) 565 NTO	Sociodemographic data survey KARASEK questionnaire	TO (WAD) had significantly more males and showed higher levels of disability than NTO group	Sick leave was used as a measure of disability and could be influenced by many other factors. No differences were found in other data (Table 2.4.1)
TO Traumatic onset, NTO non-traumatic onset, FM fibromyalgia, WAD whiplash associated disorder-II, HC healthy control AROM Active range of motion * this study also included a migraine group that is not discussed here.				

Table 2.5.2. Studies that reveal differences between traumatic onset and non- traumatic chronic neck pain groups				
Authors	Subjects	Outcome measures	Results	Comments
Michaelson et al. 2003	9 TO (WAD) 9 NTO 16 HC	Sway posture	TO (WAD) group showed larger sway, reduced balancing ability and reduced head stability than NTO and HC	The author suggests that alterations in proprioception and motor control rather than chronic pain may be responsible for the differences
Falla et al. 2004a	10 TO (WAD) 10 NTO 10 HC	Cranio-cervical flexion test EMG	TO (WAD) showed significantly increased muscle activity compared to NTO and HC groups	NDI had a significant positive correlation with EMG amplitude in both groups
Scott et al. 2005	29 TO (WAD) 20 NTO 20 HC	Hypersensitivity to temperature	TO (WAD) group showed significant remote hypersensitivity compared to NTO and HC groups	Similarities between TO (WAD) and NTO groups also seen (Table 2.4.2)
Sundstrom et al. 2006	27 TO (WAD) 18 NTO 15 HC	Regional cerebral blood flow MRI scan	Significant difference between NTO and TO (WAD) and NTO and HC in areas of blood flow in the brain	NTO showed decreased flow in right temporal region and increased flow in right insula
Sjolander et al. 2008	7 TO (WAD) 9 NTO 16 HC	ROM ROM variability Jerk index	Significant increases in Jerk index and repositioning error between chronic pain (TO and NTO) and HC	No differences were also found (Table 2.4.2)
Woodhouse and Vasseljen 2008	59 TO (WAD) 57 NTO 57 HC	Maximal cervical ROM	TO (WAD) groups showed significantly decreased maximal ROM as compared with NTO and HC group	No differences were also found (Table 2.4.2)
TO Traumatic onset, NTO non-traumatic onset, FM fibromyalgia, WAD whiplash associated disorder-II, HC healthy control AROM Active range of motion * this study also included a migraine group that is not discussed here.				

However, no differences in range of motion (ROM) and muscle strength between TO and NTO groups have been reported (Dumas et al., 2001; Sjolander et al., 2008). No differences in ROM variability or joint positioning error (JPE) between TO (WAD), and NTO groups were also reported (Woodhouse & Vasseljen, 2008). However, this study also stated that the TO (WAD) group showed a significantly decreased maximal cervical ROM compared to the NTO and HC groups. The HC group showed the greatest ROM and that the TO (WAD) and NTO groups displayed reduced conjunct motion as compared to the HC group.

There do appear to be clear differences between individuals with chronic neck pain of a traumatic and non-traumatic onset. However, the variability in results seen in the biomechanical outcome measures illustrates the importance of clear reporting of methods and the use of distinct subject groups. It could also reflect the individual variation that occurs in response to pain and trauma.

Electromyography (EMG) measures the electrical activity of muscles and has been used to identify abnormal muscle responses (see section 2.9 for a more detailed discussion of electromyography). Chronic pain has been related to significantly greater signal amplitude in cervical flexor muscles in both TO (WAD) and NTO groups compared to the HC groups during the cranio-cervical flexion test (Jull et al., 2004). However, during a repetitive arm movement test, significantly higher EMG amplitude in the TO (WAD) group was seen as compared to the NTO and HC groups in the anterior scalenes, sternocleidomastoid and in the right upper trapezius muscles (Falla et al., 2004a). Elert et al. (2001) also found that TO (WAD) and NTO (FM) participants had an elevated level of superficial cervical muscle activity compared to controls. However, the author did note that not all chronic pain participants exhibited increased muscle tension.

The studies described show that muscle activity can be affected by pain. However, it is not possible to ascertain whether these results correspond to a particular model of pain as the location of pain was not always specified.

Despite some conflicting results, there are reported differences between TO (WAD) and NTO groups (Tables 2.4.1, 2.4.2, 2.5.1 and 2.5.2). The biomechanical studies showed the greatest differences between the groups but differences were also seen in pain, psychological and demographic studies. The development of chronic pain appears to display similarities, although some differences in central sensitisation have been identified. These responses to pain could be affected by the initial injury (where appropriate) and psychological factors such as coping strategies that may also be affected by the cause of the pain. It is possible that a whiplash injury may result in biomechanical alterations to the cervical spine that are not seen in NTO participants. Therefore, both neck pain groups may employ altered motor control patterns for movement of the cervical spine but these altered strategies may differ in relation to the cause of onset.

2.8   Motor control strategies

In accordance with the models of pain, altered patterns of muscle activation or motor control strategies may be employed to avoid using painful areas. Altered trunk muscle recruitment, while performing arm movements, has been shown in individuals with low back pain as compared to pain-free individuals (Hodges & Richardson, 1999). This indicates that the experience of pain may alter the motor control strategies used, not only in movement of the painful areas but also in those areas distant to it.

In order to ascertain whether pain is the cause of altered motor control strategies or whether the altered strategies already exist, a study using experimental pain in the lower back was undertaken (Hodges et al., 2003). The results showed that feedforward responses of the lower back muscles were altered when performing arm movements. However, the altered responses were variable between the participants. Although this study used experimental pain, which cannot be directly compared to chronic pain, the results do indicate that changes on motor control strategies do occur in response to pain. These findings support both the pain adaptation and the fear avoidance models.

Induced pain also resulted in the absence of relaxation in the erector spinae muscles in full flexion, which resulted in reduced velocity and range of movement

in the muscles of the lower back (Zedka et al., 1999).Interestingly, these altered responses were transient and the normal range of movement returned when pain subsided. Following saline injections in the biceps brachii, an increase in upper trapezius muscle activity occurred during isometric contractions (Schulte et al., 2004). This shows that motor control strategies in the neck and shoulder were altered to avoid using painful muscles distant to them.

These studies suggest the use of guarded movement, as described by the pain adaptation and fear avoidance models. However, it is possible that pain could also be a consequence of altered use of muscle control strategies themselves, with extra demand placed upon the non-painful muscles. Abnormal use of pain-free muscles could result in hypertonicity and altered blood flow, which could then cause pain with further use of these muscles.

An investigation into the effects of altered motor control strategies following a whiplash injury and whether these strategies were reversible was undertaken (Stapley et al., 2006). This study observed whether individuals with a whiplash injury showed decreased postural control when the neck muscles were fatigued and if rehabilitation affected these outcome measures. Individuals with neck pain and self-reported dizziness performed forced isometric contractions against a band placed on their forehead to activate the dorsal neck muscles and induce fatigue. Sway was measured before and after the fatiguing contractions. The same procedure was then undertaken following two weeks of physiotherapy, including resistive training. Of the thirteen participants, seven showed muscle fatigue following the sustained isometric contractions accompanied by an increase in sway. These alterations were not present after physiotherapy treatment.

These results imply that underuse of painful muscles can make them more susceptible to fatigue and reduce postural balance. It appears that resistive training can strengthen muscles and increase postural balance. This study did employ a small sample size and only just over half the participants showed abnormalities of posture via sway. This implies that whiplash injury does not necessarily affect posture; it could be dependent upon the tissue damage incurred or the response to pain. The differences seen in the participants of this study could be explained by the avoidance-endurance model, where some individuals avoided

using the painful muscles, thereby causing weakness. The others continued to use painful muscles normally, which perpetuates the pain with little or no loss of function.

A model proposing possible factors involved in the development of chronic neck pain, involving a traumatic (WAD) group and a non-traumatic onset group has been presented to illustrate the potential similarities and differences in the mechanisms involved in this condition (Figure 2.7). Whether the cause of chronic neck pain is traumatic or non-traumatic in origin there appears to be similarities in the pattern of initial physiological responses and the secondary responses/outcomes. Trauma can result in soft tissue damage, specifically to muscles and connective tissue. This damage could lead to initial peripheral and central sensitisation, which in turn could cause altered muscle recruitment due to the avoidance of using damaged and painful muscles. The alteration in muscle use could itself cause further sensitisation due to the stress placed on muscles not normally employed. Muscle hypertonicity may then occur and a consequent decrease in blood flow may lead to ischaemia. Pain arising from the muscles could trigger avoidance mechanisms, leading to altered muscle recruitment. If the resulting behaviour is habituated, chronic pain could develop. However, if the response to the hypertonicity is not to habituate the altered muscle use, but to reemploy the correct motor control strategies, recovery may follow.

As discussed in the previous sections, there are many measurable aspects of neck pain, such as range of motion, head repositioning and psychosocial factors. Muscle activity and synergy can be detected using electromyography (EMG); this recording technique will be explored in the following sections. Using EMG alongside subjective measures of pain and disability may enable identification of similar or different superficial neck muscle activity in individuals with chronic neck pain, of a traumatic or non-traumatic onset, as compared to the normal population. It may also be possible to identify differences or similarities in altered motor control strategies between the groups by observing neck flexor/extensor synergy. This could help to determine whether the type of pain onset affects the possible development of altered motor control strategies.

Figure 2.7 Model of the development of chronic neck pain
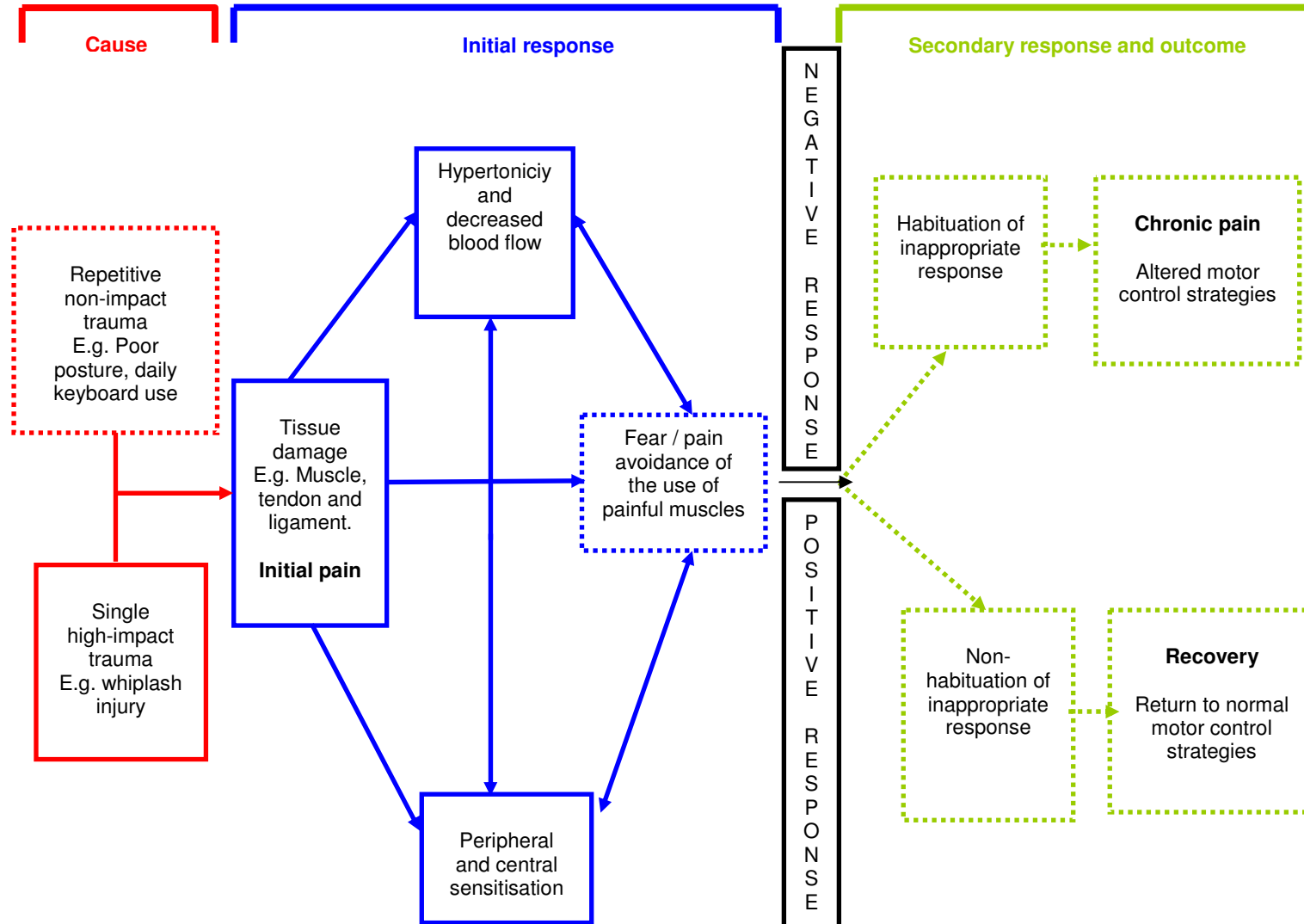
Cause

Initial response

Secondary response and outcome

N E G A T I V E R E S P O N S E

P O S I T I V E R E S P O N S E

Repetitive non-impact trauma E.g. Poor posture, daily keyboard use

Single high-impact trauma E.g. whiplash injury

Tissue damage E.g. Muscle, tendon and ligament.

Initial pain

Hypertoniciy and decreased blood flow

Fear / pain avoidance of the use of painful muscles

Peripheral and central sensitisation

Habituation of inappropriate response

Chronic pain

Altered motor control strategies

Non-habituation of inappropriate response

Recovery

Return to normal motor control strategies

Negative response: Contributing factors include pain-catastrophising, a lack of a sense of control over pain, unsatisfactory employment, unsatisfactory employment status, lack of self-awareness, fear of pain. **Positive response**: Contributing factors include no pain-catastrophising, a sense of control over pain, satisfactory employment, satisfactory employment status, self-awareness, lack of fear of pain. *Main areas for the effects of contributory factors (dashed line)*

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2.9 Electromyography (EMG)

This section of the literature review aims to introduce and discuss the muscle recording technique of EMG. Building from the basic muscle structure, function and control presented in section 2.2, a description of how the electrical activity of muscles can be detected, recorded, processed and analysed is presented. This will be followed by a discussion of studies involving electromyographic recordings of superficial neck muscles.

2.9.1 History of electromyography

EMG is the recording of the electrical signal in muscle fibres which is generated by neurotransmitter release at the neuromuscular junction. The relationship between muscle contraction and electricity was first established by Galvani in the 1790s. He reported that static electricity evoked contraction of muscle tissue in frog leg specimens. The 1800s saw the advent of the galvanometer which provided evidence of electrical activity during the contraction of muscles in humans. Techniques for measuring muscular activity progressed and by the early 1900s it was established that the magnitude of electrical response of muscle was due to the recruitment of muscle fibres as opposed to the neural input. Developments in EMG saw its clinical application begin in the 1930s for the study of dynamic movement, relaxation and the effects of pain (Cram et al., 1998).

2.9.1.i Electromyographic recording techniques

There are two main types of EMG - surface EMG (sEMG) and intramuscular EMG. sEMG: "comprises the sum of the electrical contributions made by the active MUs as detected by the electrodes placed on the skin" (Farina et al., 2003b, p1486). This method is thought of as a global measure of muscle activity and this technique records all electrical activity under the electrodes. This method is suitable for measuring superficial muscle activity. However, traditional bipolar electrodes only provide a limited view of muscular activity as it is the sum of all MUs activated at a point in time and is therefore unable to detect of individual MU activity.

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Intramuscular EMG is used to detect activity directly from individual muscle fibres by inserting a needle or a fine wire electrode directly into the muscle itself. This is a more appropriate method for assessing the activity of single MUs and has the advantage of greater specificity of placement than sEMG. It can also be used for recording the deep muscles that cannot be measured using sEMG. A disadvantage of using this method is that it is invasive and is potentially painful.

Due to the invasive nature of needle and fine wire electrodes and the potential for participants to experience pain and discomfort, sEMG is the method that was employed in this study. The pilot study used global sEMG to assess muscle activity. The linear array system became available after the pilot study commenced and was used for the main study. This method enabled the global signal to be recorded, as in the pilot study, but also allowed the observation of MU activity enabling a more detailed analysis to be undertaken.

sEMG has been used to observe the behaviour of superficial neck muscles, both in healthy individuals and in those with chronic neck pain resulting from a whiplash injury and from a non-traumatic onset. This method has also been employed to observe changes in muscle fatigue, recruitment and synergy as well as activity before, during and after exercise. It is a tool used by researchers to ascertain, amongst many other parameters, whether muscle activity differs in individuals that are experiencing pain, compared to individuals in a pain free state (Falla et al., 2004c; Falla et al,. 2004b; Falla et al., 2003b; Johnston et al., 2008b; Johnston et al., 2008a; Jull et al., 2004; Nederhand et al., 2000; Nederhand et al., 2002;,Nederhand et al., 2003; Sommerich et al., 2000).

2.9.2   Generation of the sEMG signal

During a voluntary muscle contraction, the electrical activity generated from the points of muscle fibre depolarisation is detected as a voltage difference by the electrodes, and results in the sEMG signal.   This signal is a result of the contributions of all active MUs under the electrodes at that time (Cram et al., 1998).

Refer to section 2.2.4.ii for motor unit recruitment

The MUAP is the summation of the action potentials from all the muscle fibres contained within a MU during a single firing. The MUAP train is the sum of all MUAPs of an MU and the sEMG is the sum of all MUAP trains. MUAP characteristics result from the morphological properties of the fibres that are active as well as the active and passive bio-electrical factors. MU interdependence and firing patterns is termed the interference pattern. sEMG records multiple MUAPs in a muscle in a given time, which is detected by the electrodes. The resulting signal is a reflection of the motor output in the CNS. Therefore changes in the sEMG signal can indicate how multiple MU activity changes during a sustained contraction. It can also allow comparison of sEMG recordings between pain-free participants and those experiencing chronic pain (Merletti, 2005).

2.9.3.i   Bipolar sEMG signal detection

sEMG electrodes are placed on the skin above the muscle of interest. The tissue that separates the source from the electrodes is collectively said to act as a volume conductor. The volume conductor allows signals to be obtained a distance away from the source (Stegeman et al., 2000). Its properties can also affect the resulting signal e.g. the depth of subcutaneous fatty tissue can affect the amplitude of the signal and lower the frequency content (Farina et al., 2004).

A monopolar signal is the electrical potential detected by a single electrode compared to a reference electrode which is placed at an area which is unrelated to the muscle under investigation. A bipolar signal results from the difference in electrical potential between the two electrodes as compared to the reference electrode (Figure 2.8). When a bipolar signal has been detected by the electrodes, it is passed through a differential amplifier which amplifies the difference between the two inputs. This process enables recognition of any common-mode input signal e.g. 50Hz electrical noise emitted from household mains electrical signal.
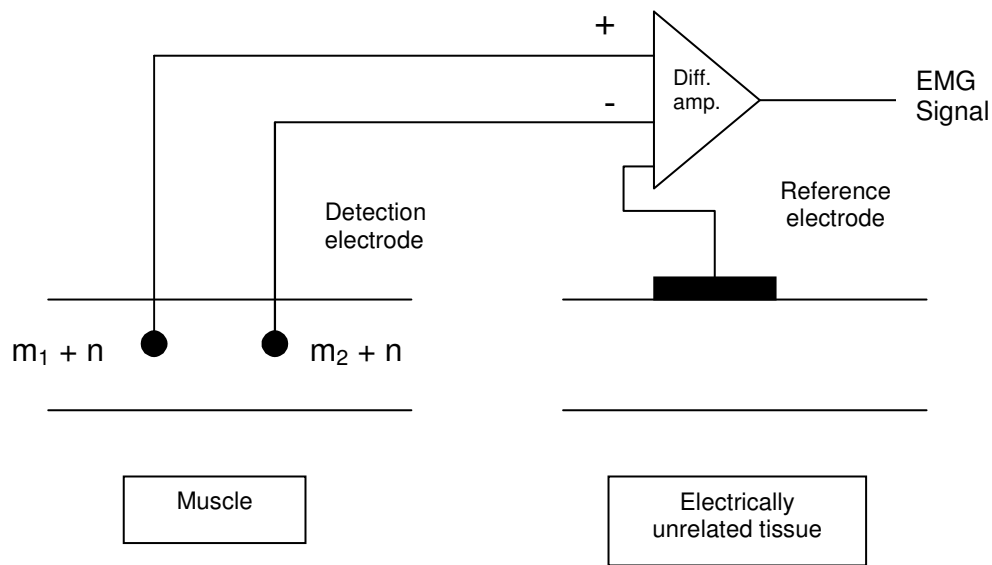
Figure 2.8 Detection of a surface EMG signal. reproduced with permission from: (De Luca 2006) Encyclopaedia of instrumentation and medical devices. John Wiley and Sons.

2.9.3.ii   Linear array electrodes and signal detection

A relatively new, more spatially selective method of sEMG detection has been developed using linear array electrodes (Merletti et al., 2003; Zwarts & Stegeman, 2003). Linear array electrodes are a series of electrodes with short interelectrode distances that can be placed over a muscle to provide more detailed topographical information such as the orientation of muscle fibres, the innervation zone and the position of tendons (see figure 2.9).   This information allows a more accurate electrode placement and therefore a more accurate sEMG signal. This technique measures the global signal as with traditional bipolar electrodes, but can also provide information on individual MUs through computerised signal decomposition. Myoelectric manifestations of fatigue can be ascertained using global and motor unit action potential (MUAP) parameters. Global EMG parameters reflect the activity of the whole muscle as detected by electrodes, which includes crosstalk and field potentials from other muscles. MUAP parameters can be extracted from superficial motor units situated directly under the electrodes, thereby excluding electrical activity from other muscles. Monopolar and bipolar sEMG recordings cannot distinguish individual MUAPs, however, the spatial arrangement of the linear array

electrodes enables detection of individual MUAPs. This is due to their smaller detection area, which results from the shorter inter-electrode distance and the smaller diameter of the electrodes. This recording method allows the observation of changes to a distinct population of MUs during exercise.

Figure 2.9 represents a multichannel recording from linear array electrodes which show the time delay of MUAP propagation on the consecutive channels as a result of the position of the muscle fibre. The attenuated signal in the centre is where the electrodes are placed over the innervation zone (IZ). The signal radiates in both directions from this point, which can be seen by the reversal in polarity of the signals either side of the IZ. The signals recorded over the tendon are also attenuated.
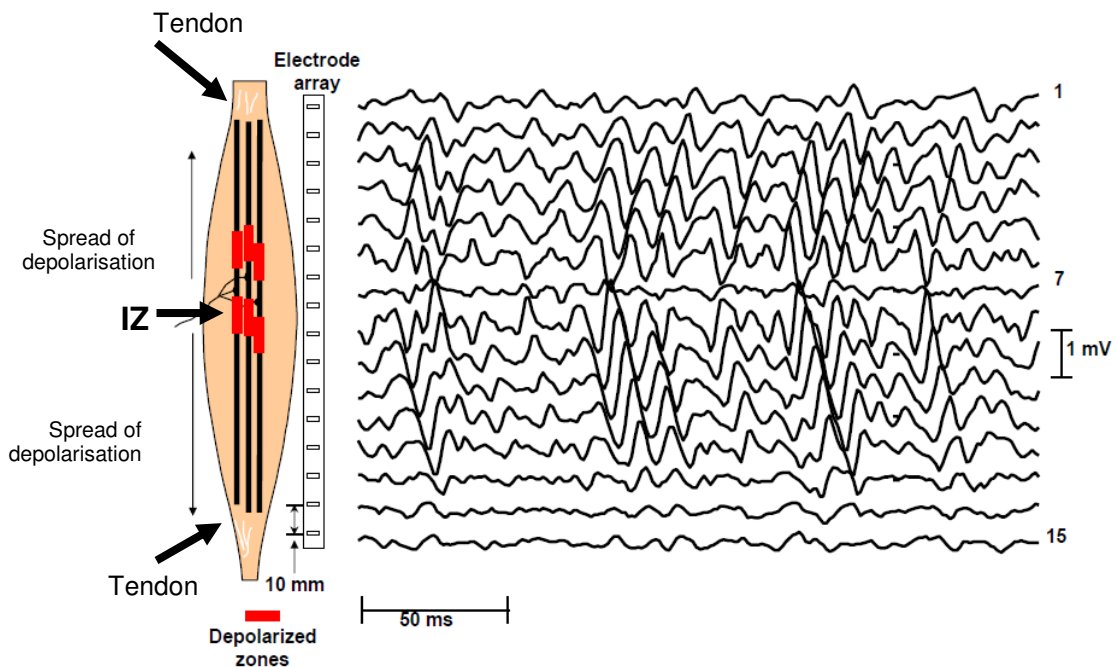
Figure 2.9 A schematic diagram of the signals detected by a 16 channel linear array electrode. Reproduced with permission from: (Merletti 2005) Surface EMG and detection. International Summer School on non invasive electromyography. Moncalieri, Torino, Italy. Courtesy of LISiN.

Decomposition of the interference pattern into its constituent MUAP trains provides more detailed information regarding the detected MUAPs. This includes an estimation of the number of MUAPs per second (MUAP rate or MR) and the MUAP

shape, which is related to the properties of the muscle fibres (Kallenberg 2007). Alterations in MUAP rate and shape are a reflection of changes, both centrally and peripherally, that can occur during a muscle contraction. Linear array signals with appropriate signal processing can be used to detect changes in muscle activation during a fatiguing exercise.

2.9.4 Applications of the sEMG signal

The relationship between force and signal amplitude has been described as both a relatively linear relationship with the amplitude of the EMG signal increasing with the force of contraction and a non-linear relationship, with the amplitude of the EMG signal showing a greater increase than the force (De Luca, 1997). In small muscles where the firing rate of the motor units has a wider range and motor unit recruitment is limited to a lower force range, the relationship is relatively linear. Whereas, in larger muscles where motor unit recruitment extends into the upper end of the force range and the firing rate has a smaller range, the relationship is relatively non-linear. As a general rule, the amplitude of a signal increases during a sustained contraction, reflecting the increase in MU firing and recruitment of MUs containing larger type-II fibres that are required to maintain the contraction.

2.9.5 Normalisation of the sEMG signal

Muscle contraction is frequently measured against a maximal voluntary contraction (MVC). A MVC is considered to be 100% of muscle activity. The MVC is then used to determine percentages of subsequent signals to normalise the responses (Farina et al., 2004). The loss of voluntary force in MVC occurs after only a few seconds. Although this method does provide a level of standardisation, it is questionable whether the participant is in fact achieving a MVC, especially in those individuals experiencing pain. The usefulness of MVC has also been questioned because most people do not perform MVC or high levels of contraction in their everyday life (Stapley et al., 2006). It is therefore not considered to be a reflection of the normal activity of the muscle.

Normalisation may also be undertaken by using the sEMG signal before activity begins as a baseline value. Subtracting the baseline value from the greatest amplitude of the signal during muscle activity can give a sEMG value of 100%. All sEMG values can then be presented as a percentage of that maximal value. However, normalisation is not always required for sEMG analysis, for example if relationships between successive values are used (i.e. β regression coefficients), rather than analysing absolute values.

By analysing sEMG signals under more natural non-forced fatiguing conditions, where the subject is only using the force required to sustain a posture as opposed to contractions against a force, it may be possible to investigate whether a whiplash injury and/or chronic pain affects the fatiguing characteristics of sEMG signals.

2.9.6   Problems associated with sEMG signals

There are many problems associated with obtaining an accurate EMG signal (De Luca, 1997). One of the major factors is signal to noise ratio. This is the ratio of the energy in the EMG signal to the energy in the noise signal. Electrical noise is defined as electrical signals that are not a part of the EMG signal. This can originate from electrical equipment or from the ambient atmosphere such as electromagnetic radiation. The problem of noise can be addressed with good quality electrodes, good skin contact and signal processing. A second problem is muscle crosstalk, which is defined as signals that are detected by the electrodes but that are not originating from the muscle of interest. This is a particular problem with small muscles. Crosstalk can be minimized by using small electrodes and a small interelectrode distance (Winter et al., 1994). Also ensuring electrodes are placed correctly over the muscle of interest by using established electrode placement guidelines (Hermens and Freriks, 1999). Analysis of signals by decomposition, which have been obtained from linear array electrodes, can directly observe individual motor units situated under the electrodes. Together with the use of spatial high-pass filters, the use of linear array electrodes removes the chance of cross talk affecting the results of signal analysis (Disselhorst-Klug et al., 2000).

Another issue faced when trying to obtain an accurate sEMG signal is electrode position. It is vital that the electrode is situated correctly over the muscle. It must not be placed over or near tendons as propagation of the action potentials extinct due to the depletion of muscle fibres in those areas (see figure 2.9). They must not be placed over the muscle motor point or IZ as this will detect signals traveling in opposite directions out from the IZ. The action potentials, when detected by a differential configuration, will effectively cancel each other out within the signal. Optimal electrode position can be gained using established guidelines. Signals from linear array electrodes can also be used to identify the IZ (Falla et al., 2002b; Hermens & Freriks, 1999). Electrode orientation is another factor which affects the quality of signal. The longitudinal axis of the electrodes should be parallel with the muscle fibres. This ensures that both of detection surfaces will intersect most of the same muscle fibers reflecting signals from a fixed set of fibres. This can also be determined using linear array electrodes.

Skin impedance, which decreases with the presence of sweat and increases with dry skin, has a very high variability and can affect the quality of a sEMG signal. The measurement of skin impedance requires the injection of a known current on the skin, so that the response can be characterised. Modern preamplifiers reduce the effect of impedance. However, it is important to ensure there are not large differences between the electrodes (Hermens & Freriks, 1999). Impedance can be reduced by abrading and cleaning the skin but not all factors can be compensated for.

One final issue is subcutaneous fat. Differing depths of subcutaneous fat mean that there can be differing distances between the muscle of interest and the electrodes. The distance between the origin of the signal and the electrodes affects the size and shape of the signal. Like skin impedance, the amount of subcutaneous fat affects the amplitude and has a slight low-pass filtering effect. The effect of subcutaneous fat can be taken into account in statistical analysis by factoring in BMI measurements as a covariate.

By using a method that takes the above factors into consideration and by referring to established protocols, the potential problems identified in this section can be

minimised. Using sub-maximal contractions, which are not measured against a 100% MVC, avoids the potential for causing unnecessary pain and discomfort to participants with existing neck pain and enables direct comparisons of absolute values.

2.9.7   sEMG signal processing

In order to obtain reliable and meaningful data from sEMG signals, it is vital that appropriate signal processing is undertaken. The following sections provide a brief introduction into the main aspects of signal processing.

2.9.7.i   Pre-processing

Analogue to digital (A/D) conversion allows computer analysis of a signal by converting it into a numerical format. The accuracy of the information that is digitally extracted from an analogue signal is related to the sampling frequency and the number of bits of the A/D converter. The greater the number of samples used, the greater the accuracy in the reproduction of the original signal. A sinusoidal wave has to be sampled at no less than twice its frequency to ensure a correct digital recreation. This is known as the Nyquist Theorem. By not adhering to the Nyquist Theorem an incorrect or false reconstruction of the signal can occur, which is known as aliasing (Merletti, 2005).

2.9.7.ii   Time domain variables

Once detected and amplified, sEMG signals undergo processing techniques to enable analysis of muscle activity. The raw signal is biphasic so signals are rectified or squared, making the whole signal positive. The rectifier permits current flow in one direction only and it transposes the signal's negative content across the zero axis (Thompson, 1998). The mean value of a rectified EMG signal over a given time is known as the average rectified value (ARV). This can then be smoothed by applying a low pass filter.

The rectified signal (ARV or smoothed) can be averaged over time to provide an estimate of the EMG amplitude. The amplitude of an EMG signal is stochastic or random. The range of signal amplitude is 0 to 10 mV (peak-to-peak) or 0 to 1.5 mV (RMS). The RMS corresponds to the square root of the average power of the EMG signal over a given time (Merletti 2005).

The RMS value is commonly used in sEMG studies of muscle contraction and fatigue to estimate both the degree of force produced by a muscle and the degree of activation (Hermens & Freriks, 1999). (Merletti, 2005).(Thompson, 1998). (Merletti 2005). (Dimitrova & Dimitrov, 2002; Falla et al., 2004c; Falla et al., 2003b; Farina et al., 2004; Gerdle et al., 2000; Kallenberg, 2007; Thompson, 1998).

The linear envelope is used to represent EMG patterns and is obtained by taking the absolute EMG value and applying a low-pass filter. The frequency range of human muscle is approximately 10 - 350 Hz. By applying a high pass filter, low frequencies e.g. movement artefact, can be eliminated. Notch filters can be applied to eliminate particular narrow range frequencies e.g. 50 Hz electrical noise.

2.9.7.iii Frequency domain variables

A continuous signal can be expressed as an infinite sum of different sinusoids or harmonic components. Figure 2.10 shows the harmonic components of an analogue signal. The original signal is shown by the red line. The superposed blue signal is the mathematical (digital) summation of the 10 harmonics illustrated above it. To create an exact reconstruction of the red signal an infinite number of harmonics would be needed, but a high level of accuracy can be achieved with 10 harmonics. The Fast Fourier Transform (FFT) is an algorithm that transforms time-domain data into frequency-domain data (Figures 2.10 & 2.11). Applying the FFT to the EMG signal allows the frequency spectrum to be determined. Analysing changes of the frequency spectrum can give information about the fatiguing properties of a muscle (see section 2.9.9).
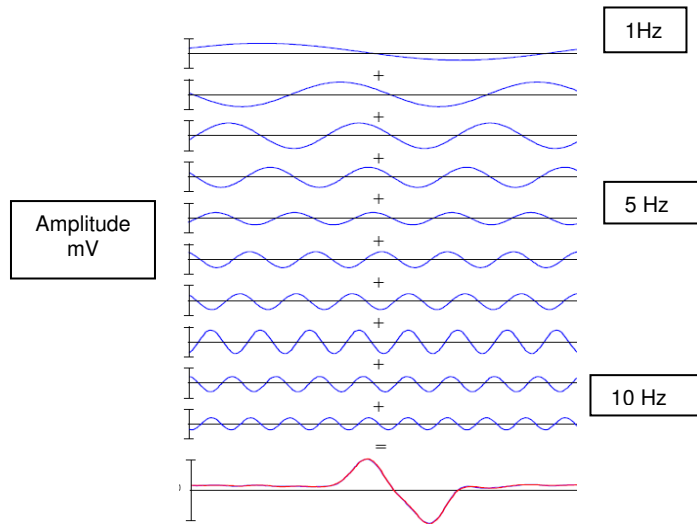
Figure 2.10 Fourier decomposition of a sample motor unit action potential (MUAP)
Reproduced with permission from URL (http://www.delsys.com/Attachments_pdf/WP_SEMGintro.pdf)
date accessed (02/07/2007).
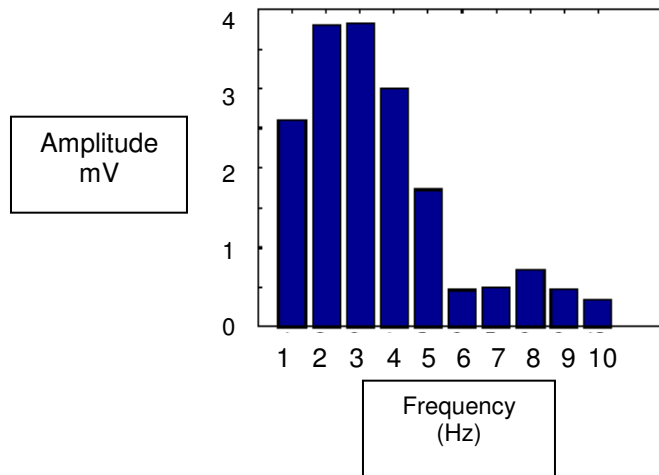
Figure 2.11 An amplitude histogram of the 10 harmonics depicted figure 1.5.3.
Reproduced with permission from URL (http://www.delsys.com/Attachments_pdf/WP_SEMGintro.pdf)
date accessed (02/07/2007)

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2.9.8   The frequency spectrum of the sEMG

The frequency spectrum of the activated muscle fibre is dependent on the conduction velocity of the fibre. The large diameter fast-twitch fibres have higher frequency content, whereas the smaller slow-twitch fibres display lower frequency content when active. The normal range for the electrical activity of human muscles is 10 – 350 Hz, with the majority of activity occurring within the 50 - 150 Hz range. A higher frequency will be observed in a strong contraction due to the recruitment of greater numbers of MUs containing fast-twitch fibres whereas a contraction of lesser force will show a lower frequency upon activation. The frequency spectrum is a reflection of all the activated MUs within a muscle at any given time (Duchateau et al., 2006).

In order to divide the EMG signal into its constituent frequencies, it can be processed and displayed as a frequency power spectrum (FPS). This shows the distribution of signal power in the frequency domain and therefore indicates the strength of each component frequency in the EMG signal (Figure 2.12).
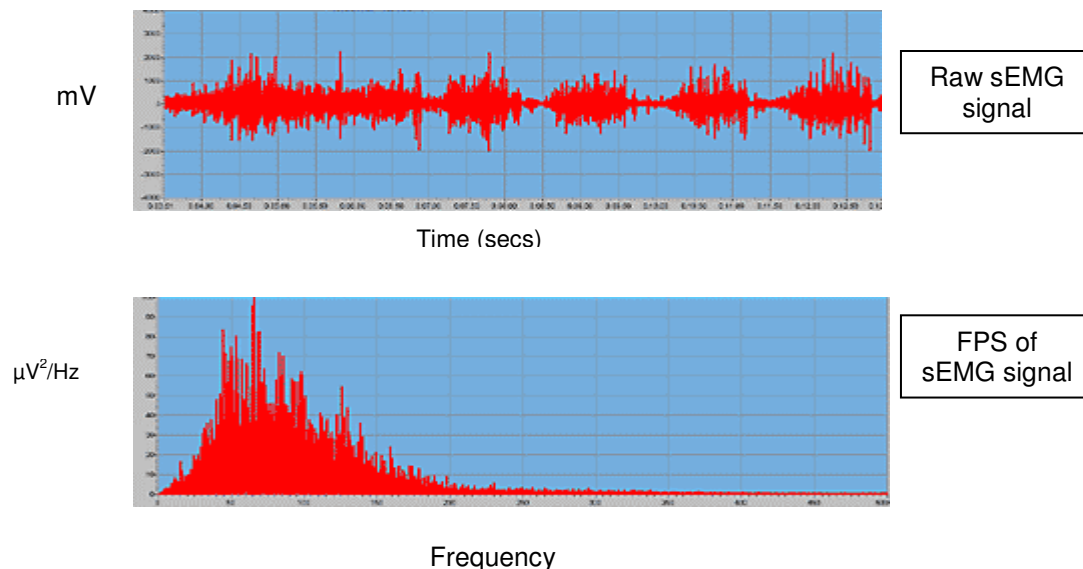
Figure 2.12 An example of a raw sEMG signal and its corresponding frequency power spectrum (FPS). Reproduced with permission from URL: http://www.nexgenergo.com/ergonomics/biodatasoft.html (date accessed 13/03/2009)

As changes in MU recruitment occur, corresponding changes will be apparent in the FPS. Modification of the frequency spectrum (due to varying MU activation) is

continuous during a muscle contraction and can be quantified by analysing the changes in the median frequency (MDF) and mean frequency (MNF) as measures of fatigue. MDF is less sensitive to outlying signals, which originate from electrical noise and biochemical and physiological changes within muscles during contraction. Therefore MDF is a preferable measure than the MNF (De Luca, 1997).

2.9.9   Myoelectric manifestations of fatigue

See section 2.2.5 for detail on muscle fatigue

Increased RMS with decreased MDF are established myoelectric signs of fatigue (De Luca 1984; Merletti et al., 1990; Dimitrova & Dimitrov, 2002). In accordance with the size principle, the smaller MUs, containing type I fibres, are recruited first during a contraction. When type I fibres are unable to maintain the contraction or more force is required, increased numbers of MUs containing type I, and/or MUs comprised of larger type II fibres, are recruited (Henneman et al., 1965). Larger fibres have a higher conduction velocity and produce signals of larger amplitude with a longer duration (Kupa et al., 1995). Recruitment of these fibres would manifest as an increase in RMS for both global (GRMS) and MUAP (MRMS) parameters.

As muscle fatigues, e.g. in a rapid forced contraction or a longer duration non-forced contraction, the conduction velocity of the recruited MUs decreases (Enoka & Duchateau, 2008). This is reflected in increased duration of MUAPs and a consequent decrease in MDF for both global (GMDF) and MUAP (MMDF) parameters.

MDF changes also reflect the number and type of MUs recruited. These factors regulate the required force of a contraction. In a low-level contraction of a non-fatigued muscle, an adequate number of motor units will be recruited to enable the desired force to be produced. This may be achieved with little or no involvement of larger MUs comprised of fast-twitch fibres. When the MUs containing slow-twitch fibres (recruited to maintain the low-level contraction) become fatigued other, smaller MUs are recruited to maintain the required force production whilst the fatigued MUs rest.

In a contraction that requires a greater level of power the fast-twitch fibres are recruited, as well as the fatigue resistant slow-twitch fibres to produce a greater force. However, the fast twitch fibres are less fatigue resistant and during a more powerful contraction the fast-twitch fibres fatigue more rapidly than the slow-twitch fibres (Kupa et al., 1995). This is reflected by a greater decrease in the MDF in more powerful contractions. This is in accordance with the findings of Gerdle et al. (2000), who found that the mean frequency of the power spectrum clearly correlated with the proportions of type I fibres in muscles acting on the knee. These findings were established by correlating sEMG signals which were obtained during fatigue whilst performing dynamic knee extensions, with histological samples of the muscle (Gerdle et al., 2000).

2.9.9.i   Linear array electrodes and muscle fatigue

(see section 2.9.3.ii for linear array electrodes and signal detection)

Linear array electrodes demonstrate a greater spatial selectivity and therefore an increased ability to identify individual MUs directly under electrodes compared to traditional bipolar electrodes (Disselhorst-Klug et al., 2000). The same myoelectric manifestations of fatigue (decreasing MDF and increasing RMS), as detected using bipolar electrodes, are seen when using linear array electrodes. However, a more detailed analysis of the effects of fatigue on MUs is possible with the linear array system. Additional parameters, such as conduction velocity (Houtman et al., 2003) and MUAP firing rate (Kallenberg & Hermens, 2006), can also be incorporated into the analysis. Alongside these factors, observation of the RMS and MDF of selected MUs (MRMS and MMDF) during fatigue provides a more detailed insight into the effect of fatigue on MUs.

Falla et al., (2003) observed sEMG signals from the sternocleidomastoid and anterior scalene muscles in individuals with and without chronic neck pain whilst performing MVCs. Although linear array electrodes were used, MUAP parameters were not analysed. This study looked at global mean frequency (MNF), global average rectified value (ARV) and conduction velocity (CV) of MUAPS as measures of fatigue to determine if the muscles of individuals with chronic neck pain showed

different fatigability than healthy controls. The study reported significantly greater values for the sEMG initial value and slope of the mean frequency at 25% and 50% MVC in the chronic neck pain subjects. These results indicate that individuals with chronic neck pain show greater fatigability of these muscles than the control subjects. However, there were no significant differences between the groups for conduction velocity values.

Falla et al., (2004d) repeated the protocol used in their 2003 study to determine if there was a relationship between the duration of pain and fatigue in the sternocleidomastoid and scalene muscles for individuals with chronic pain of up to 5 years duration. This study reported no significant correlation between duration of pain and measures of fatigue at 25% and 50% MVC. However, the authors suggest that the changes occur in the first year of neck pain and that adaptation after this time may consequently reduce signs of fatigue. They suggested that a further study that observes individuals with a shorter duration of pain may provide more insight into the manifestations of fatigue in chronic pain.

A more detailed investigation into MUAP parameters was reported by Kallenberg and Hermens in 2006. This study observed MUAP rate (MR) as well as global and MUAP RMS and MDF values in individuals with work-related chronic pain and healthy controls during 5 computer based tasks. The results showed that individuals with chronic pain showed higher MR and MUAP MDF, with a trend for higher MUAP RMS. The authors suggested that the individuals with chronic pain use higher threshold or larger MUs, with a higher CNS input, than the healthy controls. It must be considered that this study used low level activity and comparison with more demanding, fatiguing tasks must be undertaken with caution.

A further study by Kallenberg et al., in 2007 studied the effects of fatigue on the upper trapezius muscle during sustained low level contractions in individuals with and without chronic neck pain. The results showed that chronic pain subjects showed less pronounced muscle fatigue than the healthy control subjects. The chronic pain subjects showed a reduced increase in global and MUAP RMS values compared to the healthy controls. Also, the healthy controls showed a decrease in global and MUAP MDF and CV values whereas the chronic pain subjects' values

remained fairly constant. The author suggested that the chronic pain subjects' muscles showed less signs of fatigue as they were already fatigued prior to the contractions as a result of the chronic pain. This may be due to hypertonicity and an increase in muscle use resulting from pain avoidance behaviour. This would cause higher threshold, larger MUs to be recruited earlier in the activity. The type of contraction used in this study may affect the behaviour of the muscle. A more powerful, fatiguing contraction would result in the higher threshold, larger MUs fatiguing more rapidly and therefore a different myoelectric manifestation of fatigue would result.

In conclusion, the rate of fatigue is dependent upon the type of contraction elicited together with the innervation and the properties of the muscle.

2.9.10   The effect of pain on sEMG of neck muscles

Muscle activity can change in response to pain; therefore changes in sEMG recordings may be associated with pain. Altered motor control strategies have been shown to occur in cases of chronic pain (Hodges et al., 2003; Hodges & Richardson, 1999; Schulte et al., 2004; Stapley et al., 2006; Zedka et al., 1999). The sEMG signal from superficial neck muscles could reflect changes in activity in response to pain occurring at deeper levels, possibly in the deep neck muscles (Falla et al., 2003a). The activity of the deep neck muscles is undetectable using sEMG, but the activity of the superficial muscles may reflect alterations occurring at a deeper level. The following section discusses studies that have investigated sEMG of the superficial neck muscles in chronic pain participants.

Anterior scalene and sternocleidomastoid muscles showed greater levels of fatigue in participants with chronic neck pain, measured by the rate of change of the MNF (Falla et al., 2003b). Reasons suggested for this response include a reduction in smaller MUs comprised of type I fibres, with an increase MUs containing type II fibres (due to alteration in muscle use), and differences in recruitment from the motor unit pool.

Individuals with unilateral chronic neck pain showed increased fatigue on the painful side (by way of the slope of the MNF and initial MNF values) (Falla et al., 2004c).

The muscles investigated were the sternocleidomastoid and the anterior scales and recordings were made during sub-maximal isometric contractions.

The greater fatigability may not be due to a decrease in type I fibres, and corresponding transition of muscle fibres to an increased number of type II fibres as suggested by Falla et al. (2004c). Changes in muscle use is known to cause transition of fibre types, either from type I to type II or from type II to type I (Uhlig et al.,1995; Weber et al., 1993). The former of these two transitions in a postural muscle will result in increased fatigability. However it seems unusual that muscle fibres, if their use for maintaining posture is increased, would transform in direction of type I to type II. It would seem more logical for the fibres to transform from a more powerful muscle fibre type to a fatigue resistant fibre type, to meet the increased demand on the muscle. However, it is possible that with increased muscle use, i.e. a muscle normally used for rapid movement being used in a more postural role, that type I fibres could have been partially fatigued due to damage from overuse before the exercise began. This is in accordance with the Cinderella hypothesis and would result in greater recruitment of type II fibres earlier in a muscle contraction, therefore such muscles would demonstrate greater myoelectric manifestations of fatigue.

Upper trapezius activity was observed during monotonous work in women with and without neck and shoulder pain (Sandsjo et al., 2000). The findings showed that the pain group had fewer muscle rest gaps in both dominant and non-dominant sides compared to the pain-free participants. There was also increased EMG activity (amplitude) in both dominant and non-dominant sides of the pain group, but not in the pain-free group. This suggests that the pain participants increase their upper trapezii activity to stabilise and limit movement in this area to prevent further pain. This is in accordance with the fear-avoidance model. The lack of muscle rest gaps could also indicate increased low-level contraction in the upper trapezius. However, it is difficult to establish whether the increased muscle use is a cause or a consequence of the shoulder pain experienced by the participants in this study.

sEMG was used to observe the muscle activity of three groups in static postures, during dynamic manual exercise and after cessation of exercise. The two groups consisted of WAD-II participants and healthy controls. The WAD-II group had

increased muscle activity in the upper trapezius compared to the healthy control group both during dynamic exercise and after exercise. This group also displayed a decreased ability to relax the muscles after exercise, and a higher co-activation of the upper trapezius in the resting side (Nederhand et al., 2000).This could suggest increased stabilisation by the upper trapezius that also results in reduced muscle relaxation.

In a further study, Nederhand et al. (2002) investigated the EMG activity of the upper trapezius in chronic pain. This study compared a WAD-II group, a non-specific neck pain group and a healthy control group before, during and after exercise. They found no significant differences between the pain groups, but reported a tendency for increased muscle reactivity (as measured by the mean pre-exercise EMG subtracted from the mean post-exercise EMG) in the WAD-II group (Nederhand et al., 2002). Both groups showed higher muscle reactivity than the control group. These results suggest that a whiplash injury itself may not be a cause for development of chronic pain but a risk factor. The inability to relax muscles, which is seen in both pain groups, indicates that increased stabilisation by inappropriate muscles can occur in pain avoidance irrespective of the cause of the pain. However, it is debatable as to whether the alteration in muscle use perpetuates or increases the pain state by inducing further muscle pain.

To further investigate EMG activity before, during and after exercise, a longitudinal study by Nederhand et al. measured sEMG during dynamic and isometric arm raise exercises in WAD-II participants. These recordings were made during the acute pain phase of the injury and during several follow-up sessions in the 6 months after the original injury. Interestingly, this study showed that increased muscle reactivity did not occur during the acute phase or in the follow-up period; in fact there was a decrease in reactivity of the upper trapezius muscles. It was found that higher levels of pain intensity correlated with decreased muscle activation and that muscle activity continued to decrease after the initial trauma (Nederhand et al., 2003).

The series of investigations by Nederhand et al. show that there are differences in sEMG activity of the upper trapezii in individuals with chronic pain compared to individuals that are pain free. A factor that has not been discussed is whether WAD-

II participants could have sustained damage to the upper trapezius muscle during the whiplash injury. Another factor is whether the muscle could be compensating for damage to deeper structures of the neck, such as muscles, tendons and ligaments.

Damage to the upper trapezii during rear-end whiplash injuries has been shown to occur. It has also been shown that SCM muscles could be damaged during rear-end impact, but it was concluded that the posterior superficial muscles were more likely to be damaged in a rear-end impact than the anterior muscles (Vasavada et al., 2007).

In a study that compared WAD-II and insidious onset chronic neck pain participants, increased SCM activation was observed in cervical flexion when compared to healthy controls. No significant differences were identified between the two pain groups (Jull et al., 2004). These findings indicate that the muscle activity and pain felt by the WAD-II participants may not be directly related to SCM damage caused by the whiplash injury. However, the SCM muscles may act in a compensatory manner in cases of chronic neck pain.

Individuals with chronic neck pain showed an increase MUAP rate (MR) and MMDF for the UTRPs as compared to healthy controls while performing work-related computer tasks. A trend for higher MRMS values was seen in the pain groups but there were no reported differences in GRMS (Kallenberg et al., 2007; Kallenberg & Hermens, 2006). The authors suggest that this could be due to the increased recruitment of high threshold MUs in chronic neck pain.

A study which used linear array electrodes reported chronic pain participants had reduced myoelectric manifestations of fatigue in the dominant upper trapezius muscle during low-level sustained contractions. As compared to healthy controls, individuals with chronic pain showed a smaller increase in GRMS, MRMS and MR. GMDF and CV decreased in the control group but remained constant in the chronic pain cases (Kallenberg et al., 2007). It is suggested these results could be reflective of pre-existing fatigue in the upper trapezii of the chronic pain participants.

This review of the literature has shown differences in superficial neck muscle activity in participants with chronic neck pain as compared to the healthy population. These alterations could be in response to damage or from a response employed to avoid the use of painful muscles. However, it is possible that chronic pain may result from the alteration of muscle activity itself rather that the original injury. This would correspond with individuals that have not experienced a neck trauma but who have developed chronic neck pain.

2.9.11 Overview of the pilot and main study

A pilot study investigated the reliability of the methodology employed for measuring activation of the upper trapezii and the sternocleidomastoid muscles using bipolar sEMG. The methodology was developed to determine whether it was possible to observe differences in individuals that have experienced a whiplash injury and those who developed chronic neck pain from a non-traumatic onset, during sustained, non-forced fatiguing tasks. It was also used to determine if changes in the fatiguing properties of these muscles were associated with subjective reports of pain and fatigue.

The results of the pilot study informed the methodology and rationale of the main study (see section 5.9 for further details of the rationale for the main study). The main study was undertaken using linear array electrodes to investigate whether superficial neck flexor and extensor muscle synergy during fatiguing tasks differed between the chronic pain groups and healthy controls, which have not been previously reported. The results of the pilot study were also used to determine if changes in myoelectric manifestations of fatigue were associated with subjective reports of pain and fatigue.

Chapter Three – Pilot study methods

This chapter presents the methodology for the pilot study. The aim of this study was to assess the reliability of the methodology employed for measuring activation of the upper trapezii and the sternocleidomastoid muscles using bipolar sEMG.

3.1 Research questions

Is the methodology reliable for investigating cervical muscle activation patterns in order to:

Detect differences in cervical muscle activation among people who have suffered a whiplash injury, individuals with non-specific neck pain and the normal population?

Identify relationships between subjective pain, fatigue and objective measures of muscle fatigue in the subject groups?

3.2 Overall pilot study design

The pilot study used a cross-sectional design to assess the superficial cervical muscle function and chronic pain of participants with whiplash associated disorder grade 2 (WAD II), participants with non-traumatic neck pain (NTNP) and normal, healthy controls (HC). Conventional surface electromyography (sEMG) was used to assess muscle function during two isometric fatigue tests. Participants completed questionnaires and pain and fatigue scales were used to enable the investigation of relationships between muscle activity, pain, fatigue and disability. Each participant underwent the procedure three times (on three separate days) over a period of no more than two weeks to assess the reliability of the data.

3.3   Objectives of the pilot study

To refine the methodology this pilot study was undertaken to:

1.   Assess the reliability of sEMG recording method

2.   Estimate the sample size for the main study.

3.   Assess the availability of potential subjects.

4.   Test the feasibility of the proposed outcome measures and make changes
          if necessary.

5.   Determine the most appropriate methods for analysing the sEMG signals.

3.4   Ethical Approval

Ethical approval for this study was granted by the Southampton and South West Hampshire Local Research Ethics Committee.   Ethics number 04Q17024/34 (appendix C).

3.5   Selection criteria

Upon initial enquiry to advertisements, participants were sent an information sheet (appendix D). Upon reply from the individual, a meeting was arranged to confirm screening criteria and the contents of the information sheet. Individuals were asked the type of impact (if relevant), symptoms of injury (if relevant) and duration of pain. If this information was judged by the researcher to match the screening criteria and the individuals agreed to take part in the study, they were then asked to give informed consent (appendix E) to take part in the study.

3.5.1   Inclusion criteria for WAD-II participants

Participants were eligible for inclusion in this group if:
- They had experienced neck pain (lasting 8 weeks or more in the last 2 years*) resulting from a WAD-II injury (neck problems and musculoskeletal signs only).
- The injury was a consequence of a rear impact collision.

3.5.2   Inclusion criteria for NTNP participants

Participants were eligible for inclusion in this group if:
- They had previously experienced chronic neck pain (for more than 8 weeks in the last 2 years*) without traumatic onset (this was subjectively measured).

3.5.3   Inclusion criteria for HC participants

Participants were eligible for inclusion in this group if:
- They had not suffered from chronic neck pain (for more than 8 weeks) or a neck   injury
- They were pain free in cervical region at the time of the activity

3.5.4   Exclusion criteria

Participants were excluded if they had experienced any of the following:
- head contact trauma
- cervical instability
- retrograde or posttraumatic amnesia
- trauma related neurological signs
- trauma related orthopaedic signs
- pre-existing neck, head or shoulder pain prior to the WAD injury
- WAD injury resulting from a side-on or front impact collision

The point where pain becomes chronic is not clear. A review of literature defined chronic as being from 1 month to more than 6 months (Verhaaka et al., 1998). To optimise recruitment a period of 8 weeks or more was used to determine chronic pain participants in this study.

3.6   Experimental procedure - Participants

Participants were staff and students from the University of Southampton and were recruited through posters and email requests. The 15 participants were placed in 3

groups: WAD II, N=5, NTNP, N=5 and HC, N=5, (see section 4.1 for participant demographics).

3.6.1   Participant demographics

Participants were informed of the experimental procedure and were then shown the questionnaires and the equipment to be used. Following this, participants were then asked for demographic information regarding age, medication and duration of pain. Gender was also recorded.

3.6.2   Disability measurement

WAD-II and NTNP Participants were asked to fill in the Neck Disability Index (NDI) questionnaire (appendix B) (Vernon & Mior, 1999) before the activities began The NDI scores were calculated out of a maximum score of 50. In subsequent sessions participants were asked if the response to the original questionnaire had changed.

3.6.3   Participant activities

The study consisted of two separate activities, one for measurement of the upper trapezii muscles during an arm raise activity and another for measurement of the sternocleidomastoid muscles during a head raise activity. The two activities were verbally described to the participants before the recording began. They were also offered the opportunity to observe the activities on a pre-recorded video.

The two exercises were undertaken in an order, with the arm raise activity followed by the head raise activity. It was decided not to randomise the order of the exercises as the head raise is a difficult and sometimes unpleasant activity to complete, especially for those participants with neck pain. Performing the head raise exercise first may have affected the performance of the following arm raise exercise. The impact of undertaking the arm raise exercise first was deemed less likely to affect the outcomes of the head raise exercise (see section 8.3 for limitations of the study).

3.6.3.i   Arm raise activity

Prior to the activity, the subjects were informed that during the activity they would be asked to verbally rate both the pain and fatigue they were feeling at 30 second intervals on the numerical rating scale (appendix A). They were informed that pain was an unpleasant sensory experience and fatigue was a feeling of tiredness due to the effort of the activity.

The participants were seated in a chair with their hips and knees at approximately 90^o. They were asked not to cross their legs or feet or to press down with their feet during the exercise. A horizontal line, which was approximately 5 cm deep, was projected onto a wall directly in front of them by an overhead projector. The participants were asked to raise their arms laterally and the line on the wall was adjusted to be in line with top of the shoulders and forearms (see figure 3.1). The participants were then asked to place their hands in their laps and await the instruction to raise their arms laterally, so that the shadow of the top of their shoulders and arms was touching the line projected onto the wall. They were asked to maintain that position for as long as possible. The activity finished when participants could no longer maintain the position and the shadow of their arms fell below the projected line. The depth of the projected line allowed arm movement of approximately 5cm during the fatiguing activity before a gap appeared between the shadow of the top of the arm/shoulder and the projected line, when the gap appeared the exercise was stopped. This allowed for small natural movements that occurred before participants were unable to maintain the position.

Please note that figure 3.1 shows the projected line below the arms rather than above the arms as described. This picture was taken during the development of the method and the position of the line subsequently changed. The corrected position is shown in black.
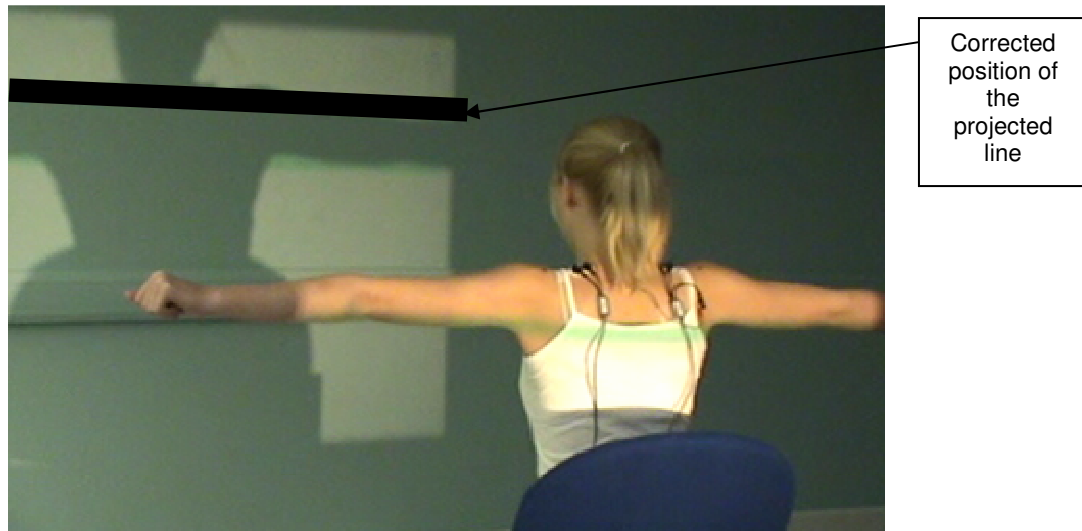
Figure 3.1 Arm raise activity for upper trapezius activation

Recording of sEMG began when the instruction to raise the arms was given. 5 seconds was given to assume the position and then every 30 seconds after that the participants were asked to give pain and fatigue ratings, which were recorded on the participant data sheet (appendix F). Recording was stopped when the shadow of the arms fell below the projected line. The participants then rested while the electrodes were removed from the upper trapezii muscles and placed on the sternocleidomastoid muscles. This period of time was no less than 10 minutes.

3.6.3.ii   Head raise activity

Before the activity, the subjects were informed that during the activity they would be asked to verbally rate both the pain and fatigue they were feeling at 10 second intervals on the numerical rating scale. They were informed again that pain was an unpleasant sensory experience and fatigue was a feeling of tiredness due to the effort of the activity.

Participants were then asked to lie in a supine position on an exercise mat with their head placed on a pressure mat. The pressure mat contained a buzzer, that when switched on, sounded when pressure was applied to the mat. They were informed that the mat would be activated and they would hear a buzzing sound.

They would then be asked to lift their head until the sound stopped and maintain this position for as long as possible (Figure 3.2). When it was not possible to maintain the position any longer, they were instructed to lower their head onto the pressure mat, reactivating the buzzer.
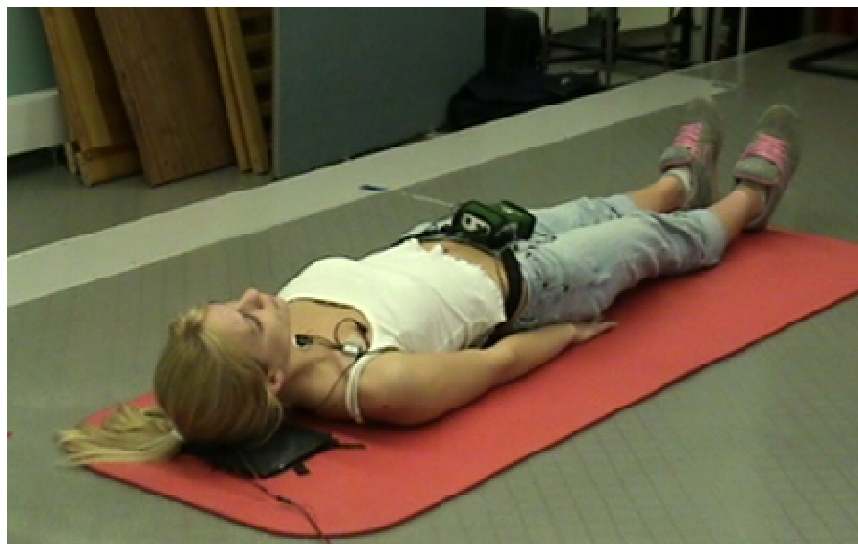
Figure 3.2 Head raise activity for sternocleidomastoid activation

Recording of sEMG began when the instruction to raise the head was given. 5 seconds was given to assume the position and then every 10 seconds following that the participants were asked to give pain and fatigue ratings, which were recorded on the participant data sheet (appendix F). Recording was stopped when the head was placed on the pressure mat and the buzzer was reactivated. The participants were asked to lie and rest until they felt comfortable enough to move, they were then asked to take a seat and the electrodes were removed and the skin was cleaned and dried.

3.6.4.   Pain and fatigue ratings during activities

Verbal pain and fatigue ratings were given during the activities using a numerical rating scale with 0 = no pain and 10 = worst pain imaginable (appendix A) (Hollen et al. 2005). During the activities the scores were given every 10 seconds when raising the head and every 30 seconds when raising the arms. The investigator waited for 5 seconds after initiation of the activity, where the participant's position

was assumed, to begin the timing for the ratings. These rating scores were recorded on the participant data sheet.

The duration of the arm raise activity was much longer than the head raise activity therefore taking rating scores every 30 seconds gave an appropriate number of scores. Taking scores every 10 seconds provided an appropriate number scores for the head raise activity, which was of a shorter duration.

3.7   sEMG recordings – equipment

sEMG signals were obtained using the Noraxon wireless system (Noraxon telemyo 2400T wireless EMG). Recordings were obtained from the upper trapezii muscles bilaterally and the sternocleidomastoid muscles bilaterally using the Noraxon myoresearch XP v1.03.04. software. The signals were recorded with Noraxon silver/silver chloride (Ag/AgCl) duotrodes, with 2 cm inter-electrode distance. The signal was amplified (gain 1000). The signal was 1^{st} order high pass-filtered of 10 Hz with an 8^{th} order Butterworth/Bessels low-pass filter of 500 Hz. The sampling frequency was 3000 Hz. The signal was recorded for the duration of the exercise and these data were stored on a laptop computer. To ensure good electrode contact the skin was prepared by gentle abrasion with fine emery paper and cleaned with 70% alcohol wipes.

3.7.1   Electrode placement for the upper trapezus muscles

The electrodes were positioned in accordance with the SENIAM recommendations (Hermens & Freriks 1999). Ag/AgCl electrodes were placed 2 cm laterally from the midpoint of the line between the spinous process of the vertebrae C7 and the acromion process. A tape measure was used to determine the midpoint. The electrodes were placed parallel to the line (parallel to the muscle fibres), with an inter-electrode distance of 2 cm. The reference electrode was placed over the bony prominence of the C7 vertebrae (Figure 3.3).
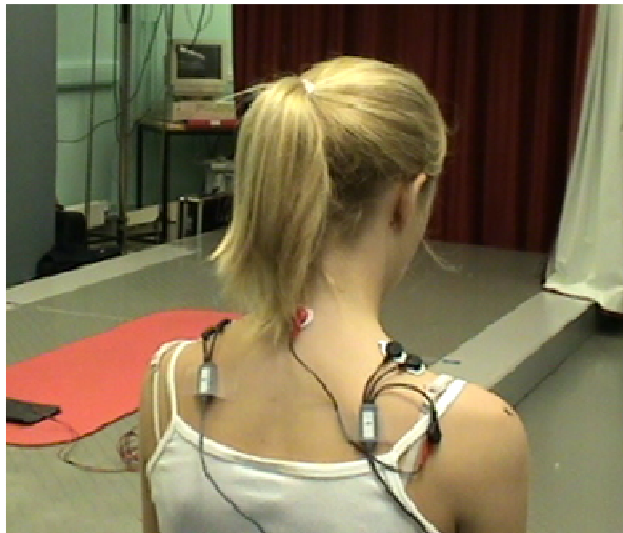
Figure 3.3 Electrode placements for the upper trapezius muscles

3.7.2   Electrode placement for the sternocleidomastoid muscles

The electrodes were positioned according to the recommendations described by Falla et al. (2002) to avoid placement over the innervation zone. The electrode position was determined by measurement of the muscle, with a tape measure, from the mastoid process to the sternal notch. Participants were asked to turn their heads to the side so the muscle became visible. The electrodes were placed on the lower third of the muscle above the tendon, on the muscle belly of the sternal head (Figure 3.4). The electrodes were placed parallel to the muscle fibres, with an inter-electrode distance of 2 cm. The reference electrode was placed over the bony prominence of the C7 vertebrae.

Figure 3.4 Electrode placements for the right sternocleidomastoid muscle

3.8 sEMG signal processing

The filtered sEMG signals were processed using programs written in the application software MATLAB by Dr A. De Stefano. The programs removed the first and last 5 seconds of the signal to exclude any possible movement artefact. In both programs the signal was full wave rectified and the root mean square was used to analyse the signals.

The power spectral density (PSD) of the signal was then computed using the segment averaging method. The parameters for this process were: number of points for the FFT = 4096 and window size = 1024 points. The median frequency was computed using the cumulative sum function. Figure 3.5 shows an example of the median frequency of the power frequency spectrum from the program used.
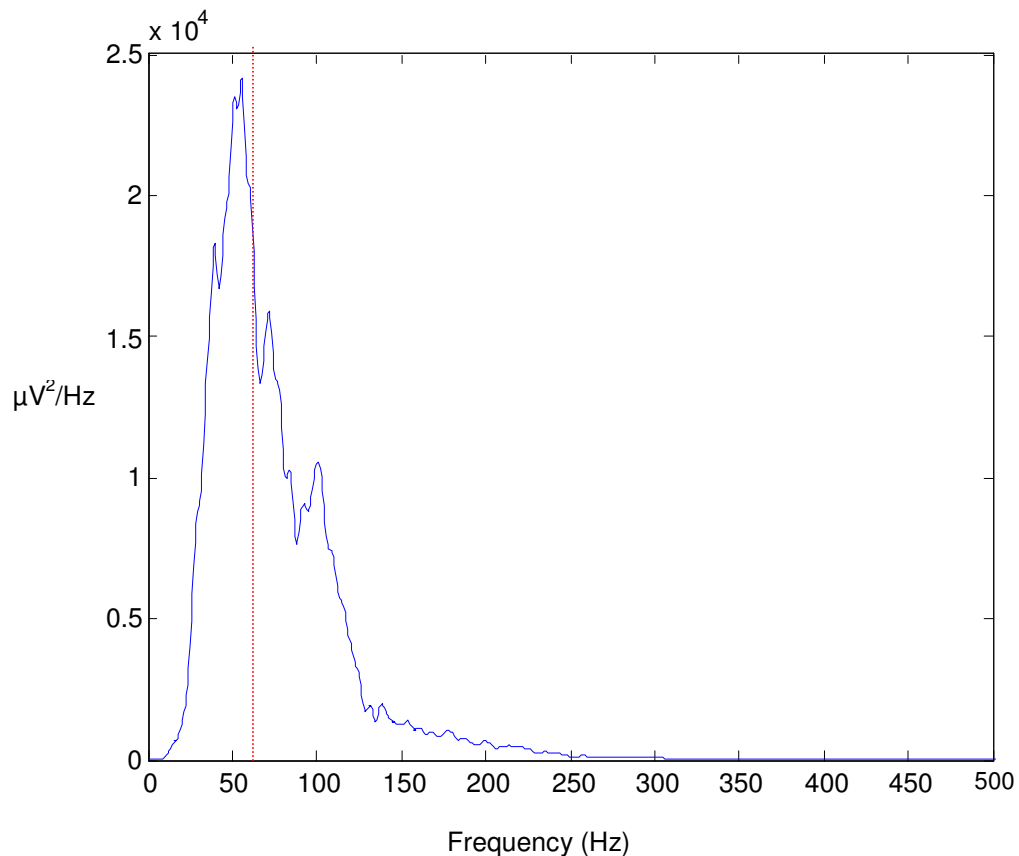
Figure 3.5 An example of the power frequency spectrum. The distribution of frequencies of a 10 second epoch, from a recording of the left upper trapezius muscle during exercise, is shown. The frequencies are displayed in the power frequency spectrum. The median frequency (MDF) of this epoch is 64 Hz (red dashed line).

The first program normalised these data in the time domain whereby the total duration of the signal was classed as 100%. This enabled comparison of the progression of activity in signals irrespective of the signal length. 10 second epochs were taken for the trapezii recordings and 5 second epochs for the sternocleidomastoid recordings at 5 equal locations along the signals.

The epoch duration was determined by the duration of the activities. The longer the duration of epoch, the more accurate reflection of the EMG activity is provided. The long duration of the arm raise activity allowed 5 x 10 seconds epochs to be taken. However, the shorter duration of the head raise exercise meant that signals may not be sufficiently long enough to extract 5 x 10 second epochs. Therefore 5 second epochs were used.

The second program took 10 second epochs for the trapezii recordings and 2 second epochs for the sternocleidomastoid recordings. These were taken at 30 second and 10 second intervals from the beginning of the signal, corresponding with the pain and fatigue ratings given during the exercises. The shorter epoch time for the head raise activity reflected the increased number of epochs taken using this program.

3.9   Statistical analysis

Demographic data were presented as means along with standard deviations and ranges. Median frequency values were presented as box and whisker plots to display the median values and distribution of these data.

3.9.1   Reliability of the median frequency data

For each participant, a sEMG signal was recorded on 3 separate days over a period of 2 weeks to establish the reliability of the sEMG signals. To ascertain the reliability of the median frequency data, the Intraclass correlation coefficient 1,1 (ICC 1,1) was applied. The ICC 1,1 is used to assess test-retest reliability and can be interpreted as the proportion of subject variance that is associated with differences within the subject group scores. (Garson, 2007).

The ICC 1,1 is calculated using the formula:

$$r = \frac{MSb - MSw}{(k\text{-}1)\,MSw}$$

Where:
- MSb = Mean square between the group
- MSw = Mean square within the group
- k = number of repeats

3.9.2   β regression coefficients

To obtain summary data for measures of fatigue, a linear regression coefficient was applied to the MDF values taken from the 5 epochs of the signal normalised in the time domain.

The linear regression coefficient is derived from the best fit of a straight line through a series of data points. The coefficient predicts the relationship between x and y (Campbell & Machin, 2005).

The linear regression line is derived from the equation $y' = bx + a$
where:

x is the independent variable or score used as the predictor (Epoch)
y' is the predicted score of y based on a known value of x
b is the slope or direction of the line
a is the y intercept

The alpha (α) regression coefficient represents the regression intercept and the beta (β) regression coefficient represents the slope.

3.9.3   Statistical tests

Statistical analysis was undertaken on the advice of Dr R. Pickering, Research Support and Development Unit, School of Medicine, University of Southampton.

β regression coefficients were explored and examined for normality using quantile-quantile (Q-Q) plots and the Kolmogorov-Smirnov (KS) test.

The one-way analysis of variance (ANOVA) was used to explore differences of the β regression coefficient means between groups. The one-way ANOVA is a robust test that can be applied when the distribution is only approximately Gaussian and the majority of these data were of a normal distribution.

Wilcoxon's signed-rank test was used to observe side to side differences for each parameter.

The relationships between pain scores and MDF, fatigue scores and MDF, pain scores and fatigue scores, NDI and MDF were estimated for each group and for each, repeat using Spearman's Correlation coefficient (r).

Statistical analysis was performed using SPSS version 14.0 for Windows (SPSS Inc., Chicago, Illinois, USA) and Microsoft Excel 2000 (Microsoft Corporation, Redmond, WA, USA). A probability level of P=0.05 was set as the minimum criterion of statistical significance for all tests.

Chapter Four – Results of the pilot study

This chapter presents the results of the pilot study which were used to inform the methodology of the main study. The demographic data are presented first, followed by the reliability of the median frequency data. β regression coefficients were calculated to provide a summary measure of the slopes of fatigue. The distribution of the slopes and analysis of differences between the groups are then shown. Finally, correlations between subjective scores and MDF are presented.

4.1   Participants

15 volunteer participants were recruited from Southampton University through email and poster advertisements. There were 5 participants in each group (healthy controls (HC, 3 female, 2 male), whiplash-associated disorder grade 2 (WAD-II, 3 female, 2 male), and non-traumatic neck pain (NTNP, 3 female, 2 male). The mean age, weight, height and duration of pain of the participants are shown in Table 4.1. All participants in this study were right-handed.

Table 4.1 Mean age, height, weight and duration of pain of the 3 groups

Group	HC (N=5) Mean (std) Range Min -Max	NTNP (N=5) Mean (std) Range Min -Max	WAD-II (N=5) Mean (std) Range Min -Max
Age in years	35.8 (8.9) 26 - 50	34.6 (11.7) 24 - 52	42.4 (12.7) 30 - 62
Height in cm	173 (9.1) 162.5 - 183	171.3 (7.3) 163 - 180	166.9 (8.8) 157.5 - 178
Weight in Kg	72.6 (15) 49 - 87	70.1 (16.8) 56 - 89	80.6 (7.7) 69 - 89
Duration of pain in months	N/A	20.4 (3.3) 18 - 24	18 (8.1) 8 - 30

4.2   Participants neck disability scores

The participants in the WAD-II and NTNP groups completed the neck disability index (NDI) questionnaire before each session. The results of the NDI questionnaires are shown in table 4.2.

Table 4.2 Participants' neck disability index (NDI) scores

Participant	repeat 1	repeat 2	repeat 3	mean
NTNP				
1	21	21	21	21
2	11	11	11	11
3	5	5	5	5
4	4	4	4	4
5	3	3	3	3
WAD-II				
1	23	23	22	22.67
2	11	9	11	10.34
3	11	11	19	13.67
4	6	6	6	6
5	10	10	10	10

4.3   Median frequency of the power frequency spectrum

An example of a raw sEMG signal is presented in Figure 4.1. The onset and end of the arm raise activity are shown.
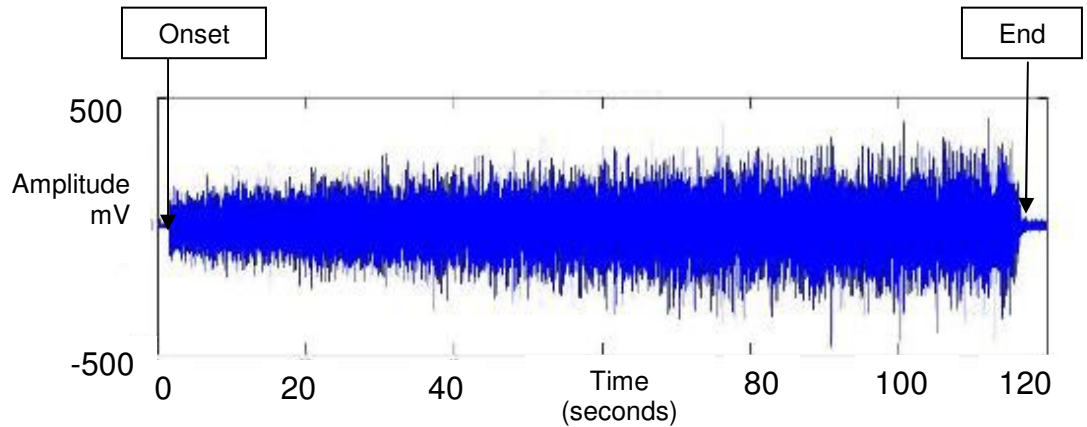
Figure 4.1 Example of a raw sEMG signal of the upper trapezius before processing.

The areas at beginning and the end of the Figure 4.1, where the amplitude is much smaller, are the signal pre- and post-exercise. 5 second sections of the recordings were removed from the beginning and the end of signal before processing. This prevented movement artefact affecting the processed signal as a result of participants assuming the activity positions and resuming the resting position when the activity finished.

By taking the MDF of the 5 epochs at regular intervals from a signal (that has been normalised in the time domain) changes in the frequencies can be observed in figure 4.2 (see section 3.8).
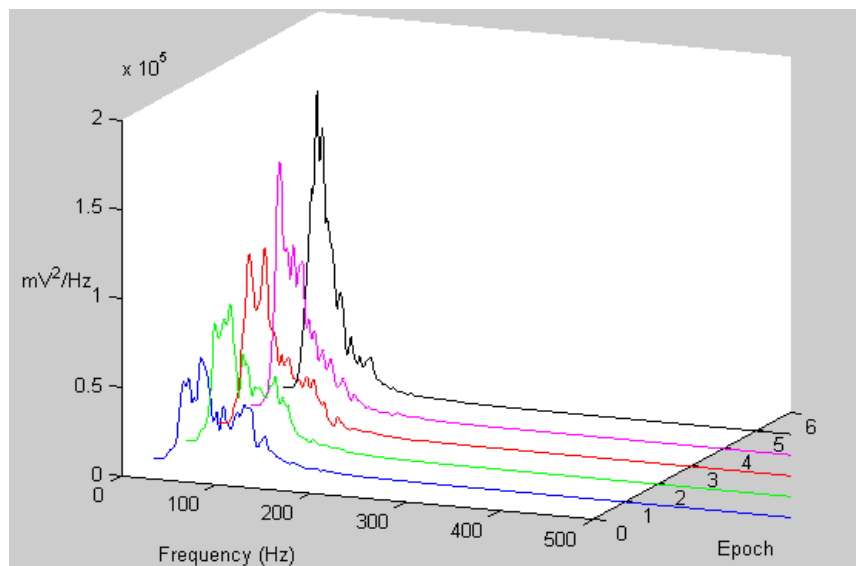
Figure 4.2 Changes in power frequency spectrum of the upper trapezius during exercise.

The frequencies were obtained by processing the raw EMG signal. The MDF values in Figure 4.2 decease from epoch 1 – 5.

4.4   Reliability of the MDF epochs

Reliability coefficients were determined using the intraclass correlation coefficient 1,1 (ICC 1,1). The coefficients of both sternocleidomastoid and trapezii muscles were calculated for each epoch (Table 4.3).

Table 4.3 Reliability coefficients (ICC 1,1) for the median frequency values.

Muscle	RELIABILITY COEFFICIENTS (ICC 1,1)				
	Epoch 1	Epoch 2	Epoch 3	Epoch 4	Epoch 5
LUTRP	0.37 (P)	0.39 (P)	0.51 (G/F)	0.43 (G/F)	0.46 (G/F)
RUTRP	0.83 (E)	0.76 (E)	0.62 (G/F)	0.74 (G/F)	0.73 (G/F)
LSCM	0.59 (G/F)	0.49 (G/F)	0.57 (G/F)	0.61 (G/F)	0.64 (G/F)
RSCM	0.55 (G/F)	0.62 (G/F)	0.70 (G/F)	0.83 (E)	0.83 (E)

Reliability coefficient categories - > 0.75 = Excellent (E), 0.75 – 0.4 = Good to Fair (G/F) , < 0.4 = Poor (P) (Fleiss 1986). 18 of the 20 reliability coefficients were at least good to fair.

4.5   Median frequency values

The median values and distribution of the median frequency, which included the 3 repeats for each epoch, are presented as box and whisker plots (Figures 4.3, 4.4, 4.5, 4.6).
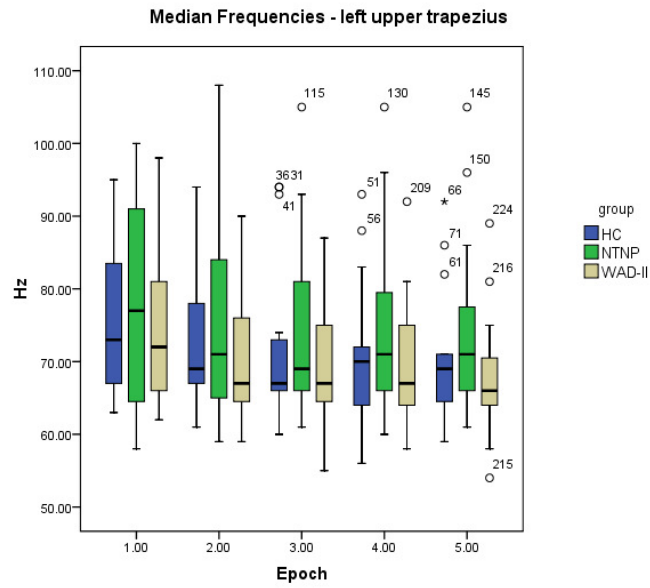
Figure 4.3 Median frequency values for the left upper trapezius muscle

As shown in Figure 4.3 the median MDF values for the left upper trapezius muscle are similar for all groups. There are a high number of outlying data points which indicate variation in the individual responses.
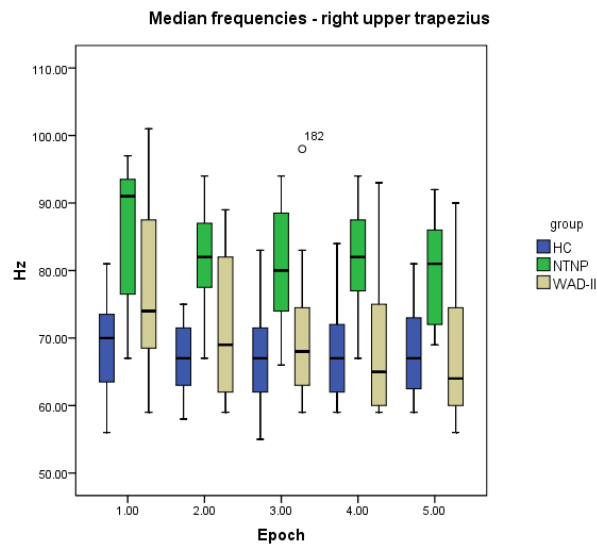
Figure 4.4 Median frequency values for the right upper trapezius muscle

A trend for higher median MDF values in the NTNP group over the 5 epochs can be seen in Figure 4.4.
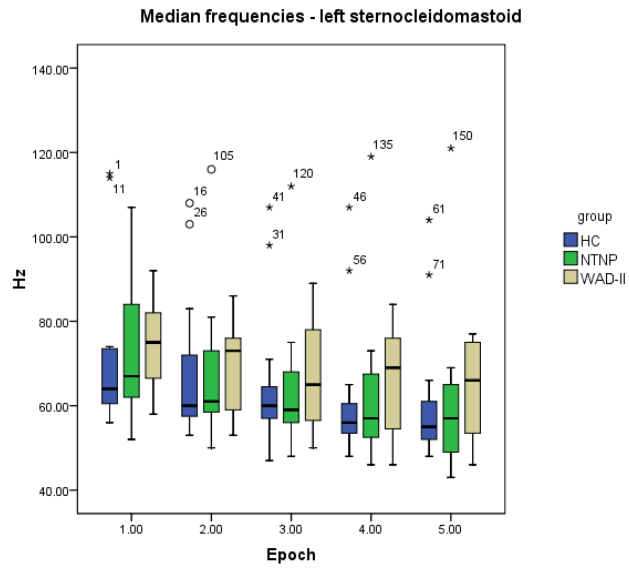
Figure 4.5 Median frequency values for the left sternocleidomastoid muscle

As shown in Figure 4.5, a slight trend for higher median MDF values in the WAD-II group, as compared to the HC and NTNP groups, is seen in the left sternocleidomastoid muscle. There are a high number of outlying data points which indicate variation in the individual responses.
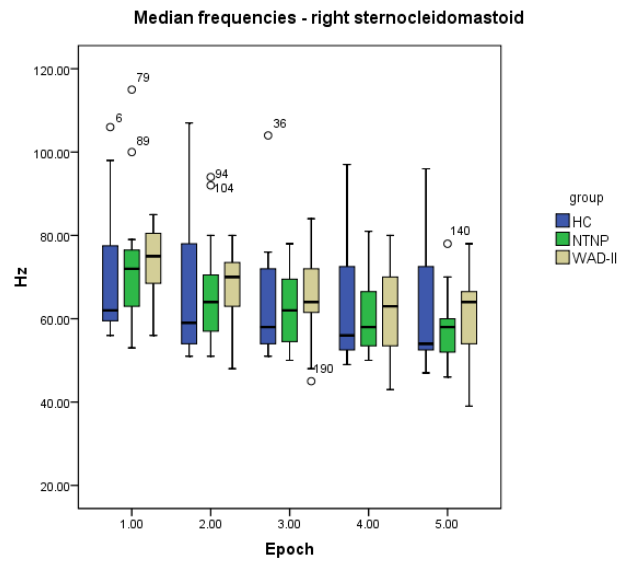
Figure 4.6 median frequency values for the right sternocleidomastoid muscle

Trends for lower median MDF values in the HC group and higher median MDF values in the WAD-II group are shown in Figure 4.6 for the right sternocleidomastoid muscle.

4.6   Distribution of the β regression coefficients

To ascertain the distribution of the β-regression coefficients, Q-Q plots and the Kolmogorov-Smirnov test were applied. The Kolmogorov-Smirnov test showed that the sternocleidomastoid muscles and the left upper trapezius muscle exhibited a normal distribution for the mean β-regression coefficients. The one-way ANOVA was used as the majority of these data were of a normal distribution.

4.7   Group differences of the β regression coefficients means

The β regression coefficients were calculated from the 5 MDF epochs for each participant and for each repeat. The mean for each participant was then calculated. The participant means were used to determine the mean β regression coefficient for each group (Figure 4.7). To observe possible differences in the means of the β-regression coefficients between groups, a one-way analysis of variance was applied (Table 4.4).
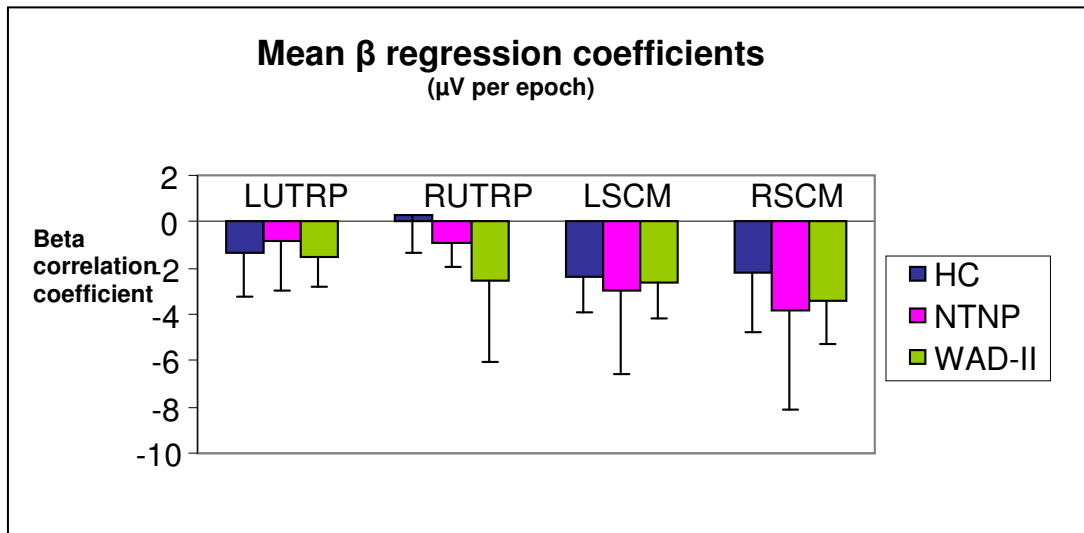
Figure 4.7 Mean β regression coefficients of the fatigue slopes (N=5 for each group).

As seen in Figure 4.7, the mean β regression coefficients of the sternocleidomastoid muscles show a trend for being more negative compared to the upper trapezii. Trends can be seen between the groups in these data, with the more negative slopes of fatigue seen in the SCM muscles for the NTNP group. The WAD-II group show the more negative slopes of fatigue in the UTRP muscles.

Table 4.4 Results of a one-way ANOVA of the mean β-regression coefficients.

	F value	p value
LUTRP	0.288	0.755
RUTRP	1.640	0.235
LSCM	0.051	0.950
RSCM	0.409	0.673

There were no significant differences in mean β-regression coefficients between the groups as shown in Table 4.4.

4.8 Side to side differences of the β regression coefficients

To investigate differences between the groups for fatigue in the left and right UTRP and SCM muscles, the Wilcoxon's signed rank test was applied to the mean β-regression coefficients (Table 4.5).

Table 4.5 Wilcoxon's test P values for side to side differences within groups

	HC	NTNP	WAD-II
LUTRP- RUTRP	0.080	0.715	0.686
LSCM-RSCM	0.500	0.35	0.040*

* p < 0.05

There were no significant differences between the mean β-regression coefficients of the left and right upper trapezii for all groups. The WAD-II group does show a

significant difference between left and right sternocleidomastoid, with the slope of fatigue being greater in the right SCM.

4.9   The relationship between pain scores and MDF

Correlations of pain scores and MDF values were only possible for the UTRP muscles. The head raise activity was not of a sufficient duration in many cases to obtain sufficient data to perform correlations of these parameters for the SCM muscles.

During the exercises, participants scored their pain on a numerical rating scale at 30 second intervals for the upper trapezii activity. The MDF values were calculated from real time epochs which corresponded with the times that the pain scores were given during the activity. Figure 4.8 provides an example of the NTNP group for the correlation of real time MDF values and pain scores given during the exercise.
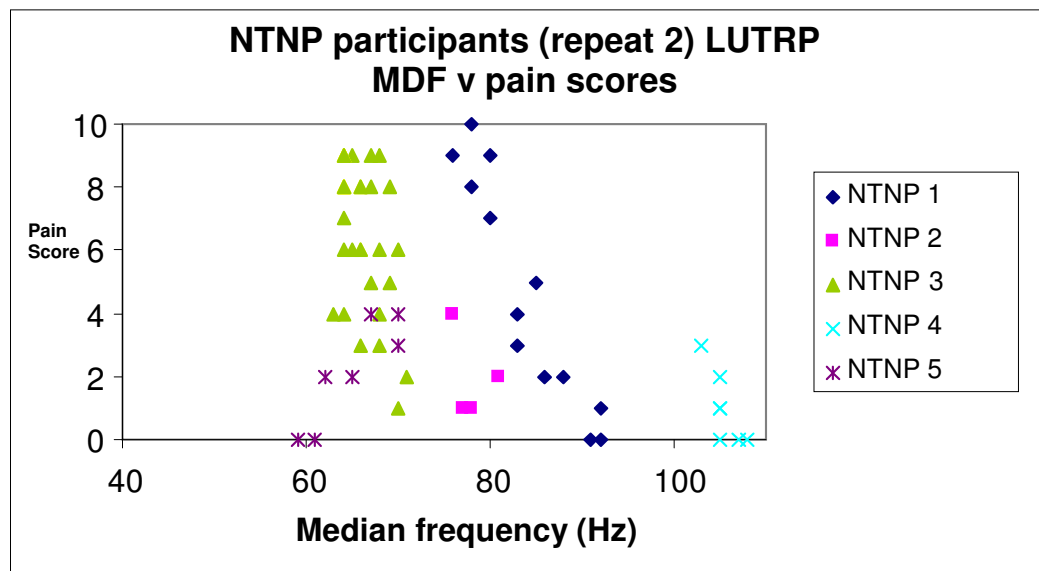
Figure 4.8 An example of correlation between MDF (real time) and pain scores for the left upper trapezius in the NTNP group.

Table 4.6 Spearman's correlation coefficients (*r*) of real time MDF epochs and pain scores for the upper trapezii (N=5 for each repeat).

	Repeat	HC	NTNP	WAD-II
LUTRP correlation coefficient for MDF and pain score	1	-0.300	-0.008	0.545**
	2	-0.141	-0.454**	-0.349*
	3	0.083	-0.350*	-0.530
RUTRP correlation coefficient for MDF and pain score	1	-0.290	-0.367**	0.454**
	2	-0.035	-0.698**	-0.061
	3	0.262	-0.202	0.102

$p < 0.05$ ** $p < 0.01$

Lower MDF values with corresponding higher subjective pain scores would result in a negative correlation coefficient. The NTNP and WAD-II groups showed significant negative coefficients as seen in Table 4.6.

4.10 The relationship between fatigue scores and MDF

During the exercises participants rated their fatigue on a numerical rating scale at 30 second intervals for the upper trapezii activity. The MDF values were calculated from real time epochs which corresponded with the times that the fatigue ratings were given. Figure 4.9 provides an example of the NTNP group for the correlation of real time MDF values and fatigue ratings given during the exercise.
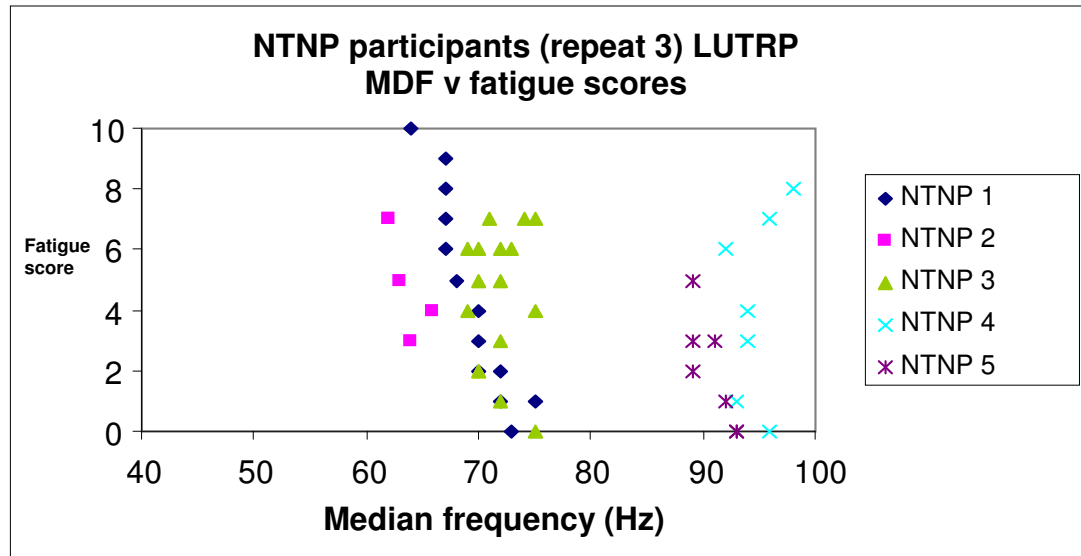
Figure 4.9 An example of correlation between MDF (real time) and fatigue scores for the left upper trapezius in the NTNP group. A variation in individual correlations can be seen.

Table 4.7 Spearman's correlation coefficients (r) for real time MDF epochs and fatigue scores for the upper trapezii (N=5 for each repeat).

	Repeat	HC	NTNP	WAD-II
LUTRP correlation coefficient for MDF and fatigue rating score	1	-0.275	-0.123	0.330*
	2	-0.368*	-0.263*	-0.102
	3	0.028	-0.408**	-0.140
RUTRP correlation coefficient for MDF and fatigue rating score	1	-0.367*	-0.044	0.194
	2	0.000	-0.524**	0.061
	3	0.209	-0.073	-0.125

- $p < 0.05$ ** $p < 0.01$

Lower MDF values with corresponding higher subjective fatigue scores would result in a negative correlation coefficient. The NTNP group showed the highest number of significant negative correlation coefficients as seen in table 4.7.

4.11 The relationship between NDI scores and MDF

Subjective neck disability was assessed using the NDI questionnaire. The mean NDI scores from the 3 repeats of activities were correlated with the mean β-regression coefficients to establish if there was a relationship between subjective disability scores and β regression coefficients (Figure 4.10).
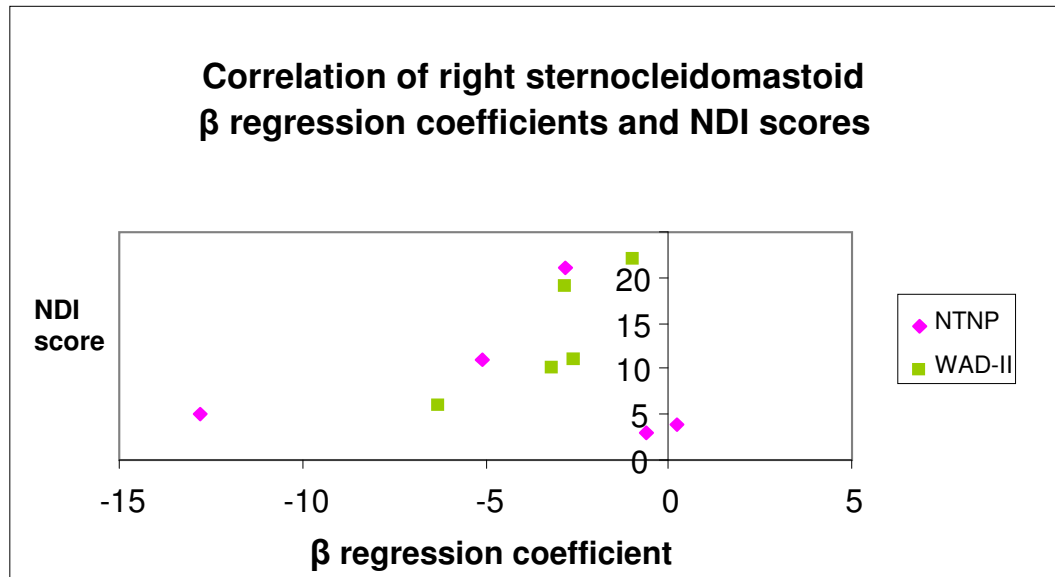
Figure 4.10 The correlation of mean NDI scores and mean β-regression coefficients for the WAD-II and NTNP groups (N=5)

Table 4.8. Spearman's Correlation coefficient (*r*) of mean β-regression coefficients and mean NDI scores for sternocleidomastoid and trapezius muscles (N=5 for each group).

Correlation coefficient	WAD-II	NTNP
LUTRP and NDI	0.600	-0.900*
RUTRP and NDI	0.200	0.300
LSCM and NDI	0.900*	-0.100
RSCM and NDI	1.00**	-0.500

* $p < 0.05$ ** $p < 0.01$

More negative β regression coefficients with increased NDI scores would result in a negative correlation. β regression coefficients showing a lesser slope of fatigue with

an increased NDI score would result in a positive correlation. Table 4.8 shows that the NTNP group had one significant negative correlation. The WAD-II group showed 2 significant positive correlations.

4.12   The relationship between pain and fatigue scores

Pain and fatigue scores were given together during both exercises. To see if the relationship between the two scores differed in the 3 groups, a Spearman's correlation was undertaken with corresponding pain and fatigue scores for the three sessions (Table 4.9).

Table 4.9 Spearman's correlation coefficient (*r*) of pain and fatigue scores.

	Repeat	HC	NTNP	WAD-II
Correlation coefficient for pain and fatigue scores	1	0.853**	0.837**	0.750**
	2	0.899**	0.882**	0.701**
	3	0.942**	0.784**	0.311

- p < 0.05 ** p < 0.01

The correlations were strongest for the HC group, with all coefficients being significant (p> 0.01). The coefficients for the NTNP group were also all significant (p> 0.01). Two of the three coefficients were significant (p>0.01) for the WAD-II group.

4.13   Estimation of sample size for the main study

Power calculations were undertaken using the mean MDF epoch values from the pilot study to ascertain the appropriate sample size for the main study (Research 2006). A sample size for each epoch was calculated. The power calculations for 3 of the muscles resulted in an average sample size of 19 participants per group. The results for the LSCM resulted in an average sample size of 100. The anticipated sample size for the main study was estimated at N= 20 for each group which results in the LSCM being underpowered.

Chapter Five - Discussion of Pilot Study Results

The aim of this pilot study was to develop and test the methodology for the main study. The results of the pilot study were used to refine the original research design, with a view to the development a useful assessment tool. A brief discussion of the findings is presented in order to provide a background for the rationale of the refined methodology.

5.1   Reliability of MDF data

The reliability of the median frequency values ranged between 0.37 and 0.83 (ICC 1,1). This shows the reliability to be generally good to fair. However, the range of ICC values identified potential problems with this method. There were several variables that could have affected the reliability; these included the equipment used, the investigator and the small sample size. Problems with the study design have been identified during this investigation and are discussed in more detail in section 5.9. With a larger sample size, more accurate recording equipment and refinements to the study design it may be possible to establish a more accurate method for recording sEMG variables. This variation in the reliability of these data indicates that the obtained results should be interpreted with caution.

5.2   β regression coefficients

There were no significant differences in mean β regression coefficients between the groups. The sternocleidomastoid muscles in both chronic neck pain groups showed a trend for more negative mean slopes of fatigue than the HC group. These results are in agreement with the findings of Falla et al. (2006) who reported a greater slope of mean frequency in chronic neck pain patients during isometric cervical flexion, compared to healthy controls.

For the upper trapezius muscles, the WAD-II group showed a trend for more negative slopes of fatigue than the HC and NTNP groups.

Conversely, Kallenberg et al. (2006) reported a slight decrease in MDF of the upper trapezius muscles in control subjects and a constant MDF in chronic pain cases during a sustained contraction. However, Kallenberg's study used force induced contractions, albeit low-level, which could alter the recruitment of MUs within a muscle. The reduction in fatigue in the control group does reflect the smaller fatigue slopes for three muscles seen in the HC group.

The results of the pilot study may have been affected by the study design, for example the arm raise activity may not have been sufficient in fatiguing the muscles. Increased encouragement to maintain the position for longer could have resulted in greater signs of muscle fatigue.

5.3   A comparison of β regression coefficients between the left and the right side

The results indicate a difference between the mean fatigue slopes of the left and right sternocleidomastoid muscles in the WAD-II group. These results showed that the non-dominant side appeared more fatigue resistant as the participants in this group were right handed and the fatigue was greatest in the right sternocleidomastoid. Contrary to these findings, an investigation of EMG activity in pain-free participants by Farina et al. in 2003 reported that upper trapezius muscles on the side of hand dominance were less fatigable than those on the non-dominant side in healthy control participants (Farina et al., 2003a). This suggests that muscle fibre types may adapt to an increase in use. However, the mechanisms involved in these changes may be different to those seen in chronic pain.

5.4   The relationship between pain and fatigue scores and MDF

The majority of the correlation coefficients for MDF and subjective pain scores were negative. This shows an increase in fatigue, correlated with an increase in subjective pain. The NTNP group showed significant negative correlations for four of the six repeats. The WAD-II group had two significant positive and one significant negative correlations and the control group no significant correlations. A possible explanation for these results is that the fatigue in the upper trapezius muscles had a

greater involvement in the pain felt in the NTNP group, whereas the other groups may have felt pain from a broader spectrum of muscles involved in the exercise. If there are no specific painful muscle groups then the activity in any one muscle group may not be directly representative of pain and fatigue felt. However, it is difficult to show firm relationships, even with significant correlations, due to the lack of fatigue shown in the upper trapezius muscles.

Subjective fatigue scores, correlated with MDF values resulted in 3 significant negative correlations for the NTNP group, 2 significant negative correlations for the WAD-II group and 1 significant positive correlation for the WAD-II group. As with the pain scores, it could be that the activity in the upper trapezius muscles had a greater involvement in the fatigue felt in the NTNP group than the other groups. In the WAD-II group the correlations, both positive and negative, were not particularly strong. This could suggest that the perceived pain and fatigue could originate from a wider range of muscles or that the pain and fatigue could be resulting from deeper areas of injury that are not related to superficial muscle activity.

A close relationship between the subjective perception of exertion in the neck and muscle fatigue of the upper trapezii has previously been reported (Hummel et al., 2005). The pilot study found similar results despite the fact that perceived exertion was assessed rather than fatigue. However, the results are comparable as these two terms are similar. Hummel's study also used the Borg scale rather than a numerical rating scale to assess subjective exertion. Although the results of the study show similar findings to Hummel et al., both the parameters measured and the measurement scales that were used differ, making it more difficult to draw concrete comparisons.

5.5   The relationship between NDI scores and MDF

An increase in fatigue (shown by more negative β regression coefficients) and higher NDI scores would result in a negative correlation. A significant negative correlation for NDI and fatigue for the left upper trapezius was seen in NTNP group. The WAD-II group showed significant positive correlations for both sternocleidomastoid muscles, which could suggest that higher perceived disability

was related to lower levels of fatigue. This could be a result of fear avoidance whereby other muscles are activated to avoid use of the sternocleidomastoid muscles, possibly due to pain. Therefore the muscles of the WAD-II group did not fatigue as much as the NTNP group.

NDI scores have been identified as possible predictors in the development of chronic pain following a whiplash injury (Nederhand et al., 2004). Patients with high NDI scores have been shown to develop increased EMG activity in the CCFT and increased hypersensitivity (Nederhand et al., 2004; Sterling et al., 2006). The fact that high NDI scores result from pain catastrophising cannot be discounted. It is possible that individuals with a greater fear of pain and a more negative outlook might themselves induce chronic pain by altered muscle use in pain avoidance.

5.6 The relationship between subjective pain and fatigue ratings

In order to establish whether there was a relationship between subjective pain and fatigue scores, a correlation was undertaken for each of the sessions. The HC and NTNP groups displayed entirely significant positive correlations and the WAD – II group had 2 significant positive correlations. These results were as expected due to the sensations of pain and fatigue being closely related.

A possible explanation for these results is that the pain the HC group were experiencing was directly related to their fatigue. It is possible that both pain and fatigue originate from the same source - the muscles involved in the exercise. The WAD-II group may be experiencing pain which originates more from other sources, such as deeper structures of the neck. Therefore, the pain that the WAD-II group experience does not correlate particularly well with the perceived fatigue of the exercise. The correlations in the NTNP group were not as strong as the HC group but were stronger than the WAD-II group. This could suggest that the pain and fatigue were from the same source but pre-existing pain had reduced the relationship shown in the HC group. A relationship between chronic pain and fatigue has been identified and recommendations have been made to assure that healthcare professionals, involved with treating chronic pain patients, are aware of the high prevalence of fatigue associated with pain. (Fishbain, 2006).

5.7　　　Summary of results

The results of the pilot study indicated that the reliability of the sEMG measures is generally fair to good. The results also indicated trends showing slight differences in muscle fatigue and significant differences in bilateral activation between individuals who have suffered a whiplash injury, individuals with non-specific neck pain and the normal population. The relationship between subjective pain and fatigue scores, and the relationship between these scores and fatigue slopes, also demonstrate potential differences between groups. However, due to the small sample size and the variation in reliability coefficients, these results should be viewed with caution. To ascertain the validity of these findings it will be necessary to repeat this study with groups of 20 participants (as estimated using sample size calculations, section 4.13) with a refined methodology to improve the accuracy of the results. *The statistical analysis of the correlations was subsequently found to be inappropriate and is amended in the main study.*

5.8　　　Methodological considerations

The pilot study identified potential problems with the methodology. It was realised that the position of the hands in the arm raise exercise could be affecting the level of activity in the upper trapezii (Hermens and Kallenberg, 2006, personal communication). The reference position employed during previous studies involves the hands being relaxed with the fingers pointing to the floor. In this study the hands were outstretched. In the main study participants will be asked to assume the lateral arm raise position with relaxed hands.

Through observations of the head raise activity, it was noted that some participants did not maintain a steady head position and on occasions it appeared to be raised more than 1 cm. To correct this, a head frame has been developed with a lighting system to indicate the correct height of the head lift (Figure 6.2).

The position of the feet during the arm raise activity could also have affected the recruitment and use of the trapezii muscles. The participants' feet were touching the floor which could mean that force could be applied through them during the fatiguing activity altering the general muscle response. This has been addressed in the new

methodology by using a chair where participants' feet will not be in contact with the floor.

Skin impedance was not measured which may have affected the accuracy of the sEMG signals. Obtaining acceptable skin impedance in the neck region can be problematic due to the sensitivity of this area. When undertaking analysis in the main study BMI values will be factored in as an estimate of subcutaneous fat, which affects impedance, to minimise its effect of the sEMG values.

Accurate identification of the acromion process was problematic with some of the participants. This may have affected accurate electrode positioning. This manual skill improved throughout the course of the study and it is felt that this will not be problematic in the following study.

By not encouraging participants to maintain the activity for as long as possible a true muscle fatigue may not have been achieved. it has been stated that encouraging participants to maintain a fatiguing position has resulted in greater myoelectric manifestations of fatigue (Hermens & Kallenberg 2006, personal communication). To address this point, a clock will be visible to the participants during the activity and verbal encouragement will be given.

All of the above factors could have affected the sEMG recording and caused insufficient muscle fatigue as reflected by the results of the pilot study. To improve the study design these factors have been addressed in the new methodology (Chapter 6). This includes increased sample size within groups and including WAD-I participants. In addition, the use of linear array electrodes to simultaneously record the upper trapezii and sternocleidomastoid muscles and the new protocol may provide a clearer picture of cervical flexor and extensor muscle synergy.

Reliability of the new linear array recording technique will not be undertaken as this has been previously ascertained. The test retest reliability of MUAP properties of recordings of the SCM and UTRP using linear array electrodes has previously been established during performance of 3 tasks (Kallenberg et al., 2009). The tasks were ironing, head turning and shoulder abduction and showed good test retest reliability. For the ironing and shoulder abduction the upper trapezius the ICC values for global

MDF were 0.95 and 0.98, and global were RMS 0.91 and 0.97, for the respective tasks. The ICC values for MUAP MDF were 0.93 and 0.97, and MUAP RMS were 0.78 and 0.95, for the respective tasks. For the sternocleidomastoid muscle during the head turn task the ICC value for global RMS was 0.96, global MDF was 0.91, MUAP RMS was 0.95 and MUAP MDF was 0.95. However, the reliability of MUAP rate was questioned due to the high level of muscle activity, which can cause super imposition of signals and make MUAP detection problematic. Training in the use of the software for analysis was undertaken by the author by Dr L. Kallenberg to optimise operator judgement in line with the reliability study by Kallenberg et al., in 2009 (see section 6.8.2).

5.9   Rationale for the new methodology

The pilot study observed the activity of the upper trapezius muscles and the sternocleidomastoid muscles separately. To obtain more information of motor control strategies simultaneous recording of upper trapezii and sternocleidomastoid muscle activation during a non-forced fatiguing exercise were undertaken. Recording from both muscles during these tasks may provide a better insight into possible altered motor control strategies compared to the investigation of one muscle pair alone. The methodology for two activities, one in which the upper trapezii muscles were predominantly activated, and another in which the sternocleidomastoid muscles were predominantly activated, have been refined. The refinements include a standardised hand position to ensure a more accurate recruitment and employment of the muscles involved in the head raise activity. A new head raise measure was also developed to standardise the head height during the head raise activity. A new seating position, where the feet were not in contact with the floor, was used to reduce the recruitment of and reliance on other muscles to stabilise and support the maintenance of the arm raise position. Verbal encouragement to maintain the positions was also included to try to ensure the positions were held as long as possible to maximise the effects of fatigue.

Studying two chronic pain groups, where the pain results from non-traumatic and traumatic origins, may elucidate the relationship between altered muscle control strategies in these two groups.

During the pilot study the opportunity arose to obtain a multi-channel linear array electrode system. The use of this system, with appropriate signal decomposition software, allowed a more detailed analysis of the parameters of muscle activity. (see sections 2.9.3.ii & 2.9.9.i for more detail on linear array techniques and application). These parameters of muscle activity included central nervous system output (estimated by motor unit action potential rate), which is not possible using the bipolar electrode system employed in the pilot study. The linear array electrodes record activity from individual motor units and the MUAP rate (MR) and MUAP RMS (MRMS) and MUAP MDF (MMDF) can be calculated. As opposed to the bipolar system, analysis of the signals obtained from the linear array electrodes identified individual motor units, separate to field potentials and cross talk that emanate from the muscle of interest and from muscles situated close to the muscle of interest respectively. Analysis of those MUs directly under the electrodes allowed a clearer picture of how they were affected by fatigue, as opposed to the global muscle recordings. The MR is directly related to input from the central nervous system (Kallenberg, 2007; Kallenberg & Hermens, 2006) and can be compared to the MRMS and MMDF for a more detailed picture of the changes in muscle activity and where these may emanate from. These factors, along with the global measures of MDF and RMS, provide a more comprehensive picture of muscle activation and synergy during non-forced fatiguing activities in the different participant groups.

Chapter Six – Methods for the Main Study

This chapter presents the revised protocol for the main study, which has been informed by the outcomes of the pilot study. By incorporating the new linear array recording equipment, the amended procedure and new analysis techniques the following protocol was developed.

6.1   Overall study design

This study used a cross-sectional, exploratory, observational design to assess the superficial cervical muscle function and chronic pain of participants with whiplash associated disorder grade 1 and 2 (WAD I & II), participants with non-traumatic neck pain (NTNP) and normal, healthy controls (HC). Multichannel surface electromyography (sEMG) was used to assess muscle function during two isometric fatigue tests. Participants completed questionnaires and used pain and fatigue scales to enable the relationships between muscle activity, pain, fatigue and disability to be investigated.

6.2   Research questions

1. Do cervical muscle activation patterns – MUAP rate (MR), global RMS (GRMS) global MDF (GMDF), MUAP RMS (MRMS) and MUAP MDF (MMDF) – and synergy differ in WAD, NTNP and HC participants?

2. Is there a difference in the relationship between subjective measures of pain and fatigue and objective measures of muscle activity in WAD, NTNP and HC participants?

6.3   Objectives of the study

To determine if there are differences between the subject groups in:

- Activation and synergy of UTRP and SCM muscles
- CNS input to the UTRP and SCM muscles
- Fatigue of the UTRP and SCM muscles
- Correlations of subjective ratings of pain and fatigue with amplitude and frequency of muscle activity.

6.4   Ethical approval

Ethical approval for this study was granted by the Ethics Committee of the School of Health Professions and Rehabilitation Sciences, University of Southampton. Ethics number PO7/04-01 (appendix G).

6.5   Selection criteria

Upon initial enquiry to advertisements, participants were sent an information sheet (appendix H). Upon reply from the individual, a meeting was arranged to confirm screening criteria and the contents of the information sheet. Individuals were asked the type of impact (if relevant), symptoms of injury (if relevant) and duration of pain. If this information was judged by the researcher to be appropriate to the screening criteria and the individuals agreed to take part in the study, they were then asked to give informed consent (appendix I) to take part in the study.

6.5.1   Inclusion criteria for WAD participants

Participants were eligible for inclusion in this group if:
- They had experienced neck pain resulting from WAD-I or WAD-II injury (neck problems and musculoskeletal signs only).
- The injury was a consequence of a rear impact collision.
- They have had neck pain resulting from the injury for more than 8 weeks in the last 2 years*

6.5.2   Inclusion criteria for NTNP participants

Participants were eligible for inclusion in this group if:
- They had previously experienced chronic neck pain (for more than 8 weeks in the last 2 years*) without traumatic onset (subjectively measured).

* The point where pain becomes chronic is not clear. A review of literature defined chronic as being from 1 month to more than 6 months (Verhaaka et al., 1998). To optimise recruitment a period of 8 weeks or more was used to determine chronic pain participants in this study.

6.5.3 Inclusion criteria for HC participants

Participants were eligible for inclusion in this group if:
- They had not suffered from chronic neck pain (for more than 8 weeks) or a neck   injury
- They were pain free in cervical region at the time of the activity

6.5.4 Exclusion criteria

 Participants were excluded if they had experienced any of the following:
- head contact trauma
- cervical instability
- retrograde or posttraumatic amnesia
- pre-existing neck, head or shoulder pain in the WAD group
- trauma related neurological signs
- trauma related orthopaedic signs
- a WAD injury from a side-on or front impact collision

6.6 Experimental procedure – Participants

The participants were placed in 3 groups (WAD, NTNP and HC). Individuals were staff and students from the University of Southampton, recruited through poster advertisements

6.6.1 Participant demographics

Participants were informed of the experimental procedure and were then shown the questionnaires and the equipment to be used. Following this, participants were then asked for demographic information regarding age, gender, weight, height, medication, handedness and duration of pain.

6.6.2   Pain and disability measurement

WAD and NTNP Participants were asked to fill in the Neck Disability Index (NDI) questionnaire (Vernon & Mior, 1999) before the activities began (appendix B). The NDI scores were calculated out of a maximum score of 50. The participants were also asked to identify the intensity of pain they were experiencing prior to the activities on a numerical rating scale with 0 = no pain and 10 = worst pain imaginable (appendix A).

6.6.3   Participant activities

In order to observe the characteristics of muscle activation patterns and muscle activity during fatigue, two activities were undertaken. These activities involved the activation of superficial neck muscles: one in which the upper trapezii muscles were predominantly activated and another in which the sternocleidomastoid muscles were predominantly activated.   The two activities were verbally described to the participants before the recording began. During the activities, sEMG recordings of both trapezius muscles and both SCM muscles and qualitative pain and fatigue ratings were collected.

The two exercises were undertaken in an order, with the arm raise activity (predominantly trapezius muscles) followed by the head raise activity (predominantly SCM muscles). It was decided not to randomise the order of the exercises as the head raise is a difficult and sometimes unpleasant activity to complete, especially for those participants with neck pain. Performing the head raise exercise first may have affected the performance of the following arm raise exercise. The impact of undertaking the arm raise exercise first was deemed less likely to affect the outcomes of the head raise exercise.

6.6.3.i   Arm raise activity

Participants were seated with their hips and knees at approximately 90^o with their hands resting in their laps, they were asked not to cross their legs or feet. The chair was raised so that participants' feet were not in contact with the floor.

A projector was placed behind the participant so that an image of the arm-positioning target could be seen on a screen in front of them. The arm-positioning target consisted of a level line that was adjusted to the height of the top of the shoulders (Figure 6.1).The participants were then asked to raise their arms laterally, with the top of shoulders and forearms level with the target line on the wall in front of them and with their hands relaxed. They were asked to maintain this position until the onset of fatigue, which was determined by the moment that their either or both arms lowered below the target line or the participant could no longer maintain the position. This was based on the judgement of the experimenter. A clock was visible to the participant and was used to encourage continuation on the exercise for as long as possible. Verbal encouragement was also given.
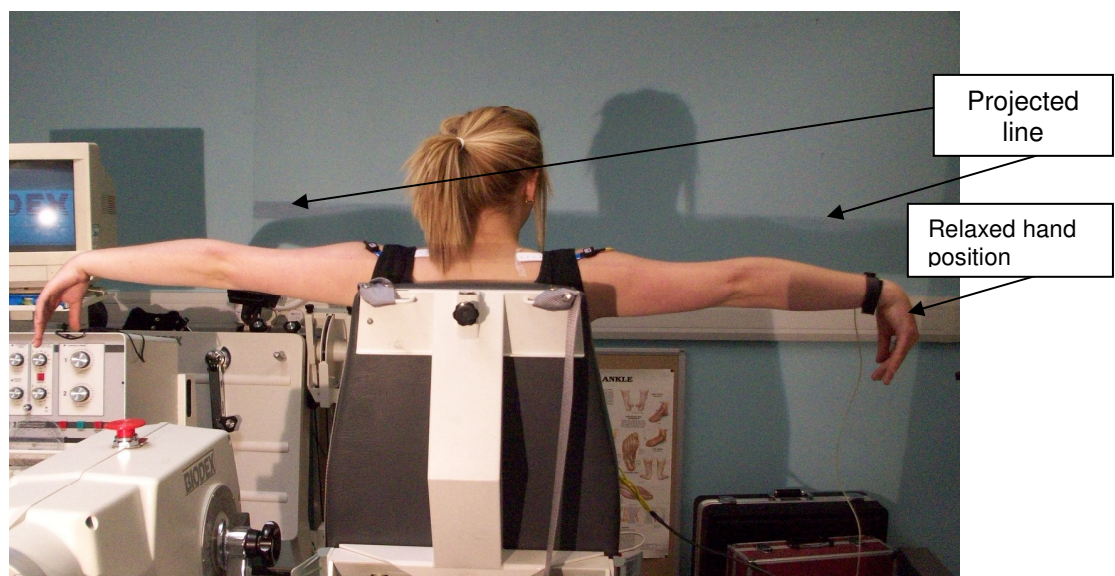
Figure 6.1 Arm raise activity

During the time the arms were raised, the participants were asked to verbally rate both fatigue and pain on a scale of one to ten every thirty seconds. sEMG of both trapezii and SCMs were continuously recorded during the activity.

When the participant could no longer hold the position (maintaining contact of the shadow of their shoulders and arms with the projected line) the activity was stopped.

For accuracy of the procedure, subjects were given the opportunity to practice the movement. This practise consisted of maintaining the position for 10 seconds prior to recording followed by a five minute recovery period before the recording began. A 10 minute rest period followed before the head raise exercise began.

6.6.3.ii   Head raise activity

For the second activity the participants were asked to lie in a supine position, with a frame designed to monitor the distance of the head raise placed around their head (Figure 6.2). The frame contained an electronic sensor attached to a head plate that was placed upon the participants' forehead. When contact was made between the forehead and the plate a red light was activated in the view of the participant.
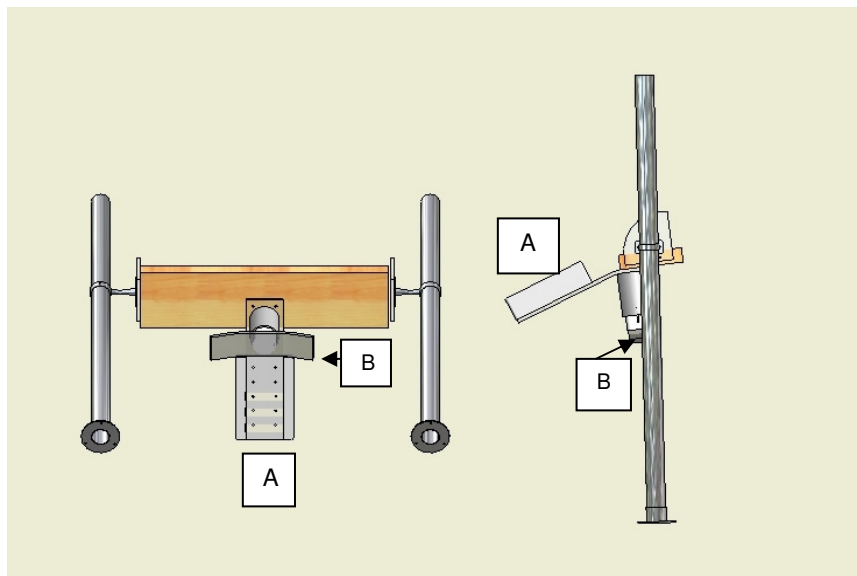
Figure 6.2 An illustration of the system to monitor head raise height. A = light box containing red and green light. B = head plate that the participants have placed on their forehead to raise and activate the lighting system.

When the head was raised by 1 cm, a green light was activated indicating that their head had been raised to the required level (Figure 6.3). If a distance of more than 1.25 cm occurred a red light was activated, indicating to the participant that they had raised their head too far (Figure 6.4)

Green light indicating that the head raise position is within the correct height range

Figure 6.3 Head raise position within the 1.0 – 1.25 cm range

Red light without the green light indicating that the head raise position has exceeded the correct height range

Figure 6.4 Head raise position exceeding the 1.0 – 1.25 cm range

The participants were asked not to rotate their head and to tuck their chin into their chest. They were then asked to raise their head to activate the green light but not the red light. They were asked to maintain this position for as long as possible. During this time the participants were asked to verbally rate both fatigue and pain on a scale of one to ten every ten seconds. sEMG of both trapezii and SCMs were continuously recorded during the exercise.

The exercise ended when the green light disappeared and the red light was activated. For accuracy of the procedure, subjects were given the opportunity to practice the movement by maintaining the position for 10 seconds prior to recording, followed by a two minute recovery period before recording began.

6.6.4 Pain and fatigue ratings during activities

Prior to the commencing activities, the subjects were informed that they would be asked to verbally rate both the pain and fatigue they were feeling at regular intervals on the numerical rating scale during the activities. The participants were given standard definitions of pain and fatigue. These definitions were *"pain is an unpleasant experience that may cause, or result in a feeling of moderate to severe discomfort"* and *"fatigue is the loss of strength and energy that results from exercise and causes a decline in the ability of a muscle to maintain a contraction".*

The same numerical rating scale that was used to assess the intensity of pain prior to the activities was used for the verbal pain and fatigue scores given while performing the activities. During the activities the scores were given every 10 seconds when raising the head and every 30 seconds when raising the arms. The investigator waited for 5 seconds after initiation of the activity, where the participants' position was assumed, to begin the timing for the ratings. The pain and fatigue rating scores were recorded on the participant data sheet (appendix F).

The duration of the arm raise activity was much longer than the head raise activity therefore taking rating scores every 30 seconds for the arm raise activity gave an appropriate number of scores for analysis. Taking scores every 10 seconds provided an appropriate number scores for the head raise activity, which was of a shorter duration.

6.7 sEMG recordings – equipment

The signals were recorded with linear arrays of 4 electrodes with 10 mm inter-electrode distance (OT-Bioelettronica, Torino, Italy). The signals were recorded for the duration of the exercise and data were stored on a laptop computer. Bipolar electromyographic signals were obtained using the EMG-16, (16 channel amplifier LISiN, Politecnico di Torino and OT-Bioelettronica, Torino, Italy).The myoelectric

signals were amplified with a gain of 1000 and sampled at 2048 Hz. The use of 4x4 electrodes allowed simultaneous recording from both of the sternocleidomastoid and upper trapezius muscles.

To ensure good electrode contact the skin was cleansed with 70% alcohol wipes and prepared with an abrasive paste (NuPrep abrasive skin prepping paste, D.O. Weaver & Co., USA). Twenty microlitres of conductive gel (acqua gel, Italy) was delivered into each electrode well with a dispenser (Eppendorf Multipipette Plus) to ensure good electrode to skin contact was made.

6.7.1 Electrode placements for the upper trapezius muscles

The electrodes were positioned in accordance with the Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) project recommendations (Hermens & Freriks 1999). The centre of the linear arrays (between the middle 2 electrodes) were placed 2 cm laterally from the midpoint of the line between the spinous process of the C7 vertebra and the acromion process. A tape measure was used to determine the midpoint. The electrodes were placed parallel to the muscle fibres (Figure 6.5). The reference electrode was placed around the participant's wrist. A sample signal was then obtained and visually scrutinised to ensure the electrode was not placed over the innervation zone (see figure 2.9 section 2.9.3). If the innervation zone was identified, the electrode placement was adjusted until the innervation zone was no longer visible.
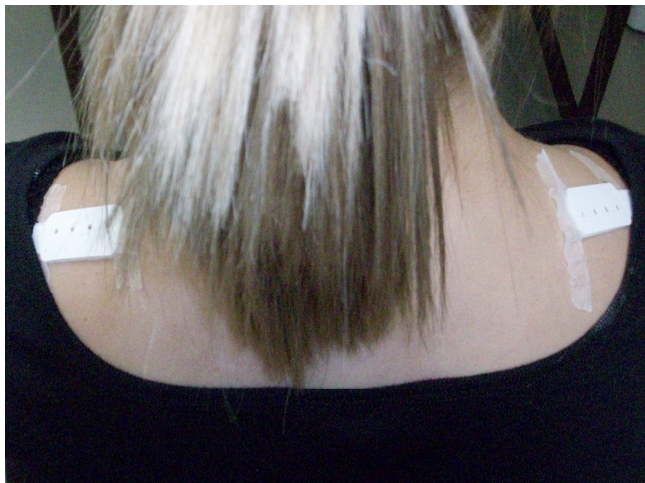
Figure 6.5 Electrode placements for the upper trapezius muscles

6.7.2   Electrode placements for the sternocleidomastoid muscles

The electrodes were positioned according to the recommendations described by Falla et al. (2002). The position of the linear arrays of 4 electrodes was determined by measurement of the muscle, with a tape measure from the mastoid process to the sternal notch. Participants were asked to turn their heads to the side so the muscle became visible. The centre of the linear arrays (between the middle 2 electrodes) were placed a third of the way up the muscle from the sternal notch, above the tendon, on the muscle belly of the sternal head. The electrodes were placed parallel to the muscle fibres (Figure 6.6).

The reference electrode was placed around the participant's wrist. A sample signal was then obtained and visually scrutinised to ensure the electrode was not placed over the innervation zone. If the innervation zone was identified, the electrode placement was adjusted until the innervation zone was no longer visible when possible. The shape of the muscle and the nature of the head raise exercise caused the electrodes to lose the position of the initial placement. Skin and electrodes frequently moved over the muscle during the exercise, which could result in the electrodes recording over an innervation zone.
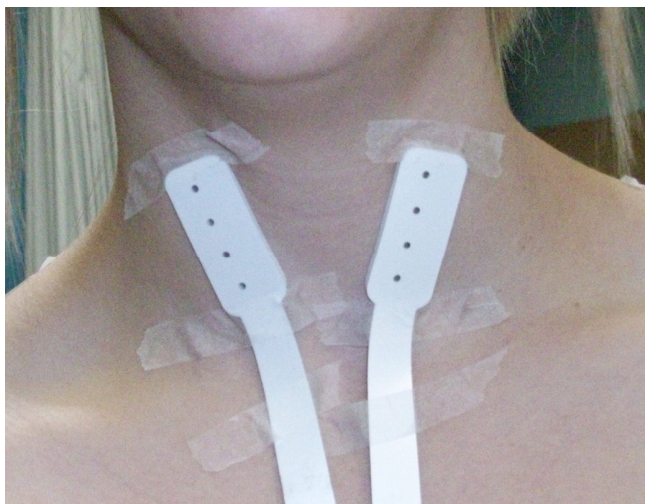
Figure 6.6 Electrode placements for the sternocleidomastoid muscles

6.8 sEMG signal processing

The sEMG signals were visualised and processed using programs written in the application MATLAB.

6.8.1 Identification of signals for processing

4 linear arrays with 4 electrodes were used. One linear array was placed on each of upper trapezius muscles and one linear array on each of sternocleidomastoid muscles. Bipolar recordings were made, i.e. the signal on channel 1 was the result of the difference in electrical potential between electrode 1 and 2, as compared to the reference electrode. The recordings on channels 4, 8, 12 and 16 were discarded because the bipolar recordings from these channels were the result of two separate arrays and therefore electrically unrelated tissue (e.g. channel 4 from the RUTRP with channel 5 from the LUTRP). The raw signals (Figure 6.7) were visually inspected for any obvious anomalies prior to processing.
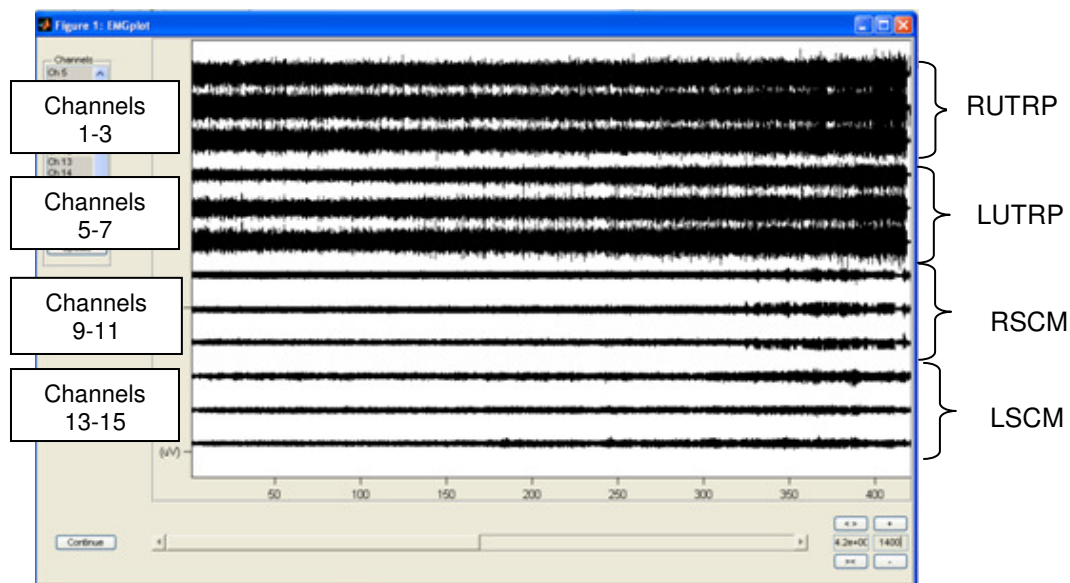
Figure 6.7 An example of a sEMG recording prior to processing with the discarded channels removed.

6.8.2   Signal processing programs

Three separate programs were written to process the sEMG signals.

Program 1 was written by Dr L. Kallenberg and provided by Roessingh Research and Development, Enschede, The Netherlands. This program calculated the start and end times of five epochs at equal points along the signal, which had been normalised in the time domain. The program allowed the start and end time of the complete signal to be entered along with the duration of the required epochs for the calculation. This allowed sections of the signal, recorded before or after the exercise, to be discarded and provided the time points for the epochs which were used to obtain the relevant data from program 2.

Program 2 was written by C. Smit and provided by Roessingh Research and Development, Enschede, The Netherlands. This program allowed visual inspection of the EMG signals (Figure 6.7) and identification of individual motor unit action potentials (Figure 6.8). The visual inspection also provided the opportunity to detect anomalous signals. After an initial inspection the channels that displayed signals with a poor signal-to-noise ratio or artefacts were discarded. The signals were filtered using a 4^{th} order Butterworth band pass filter, 10 – 400 Hz. A 50 Hz filter was available but not used as it could have adversely affect the processing of certain high activity signals.

A 3 day training course was undertaken by the author under the instruction of Dr L. Kallenberg at Roessingh Research and Development, Enschede, The Netherlands. The author was trained in the use of the software then performed analysis on specific signals. The results were compared to those obtained by the instructor. When the author and the instructor achieved consistently comparable results, the training was complete. To confirm that the author continued with a good standard of analysis, sample signals from the main study were sent to Dr L. Kallenberg who analysed them independently. The author's and instructor's results continued to be comparable.

5 epochs of each signal were analysed. The program allowed selection of the start and end point of each epoch, determined using program 1. Program 2 then extracted the global parameters (GRMS and GMDF) and exported the values to an excel spreadsheet.

The program also allowed the identification of MUAPs from the EMG signals through a process of EMG decomposition (Figure 6.8). MUAP detection was performed by a process of segmentation, based on Continuous Wavelet Transform (CWT) template matching. A wavelet shape (a first derivative of a Gaussian) was used as a template to match with, and identify, individual motor unit action potentials within each epoch of EMG signal.
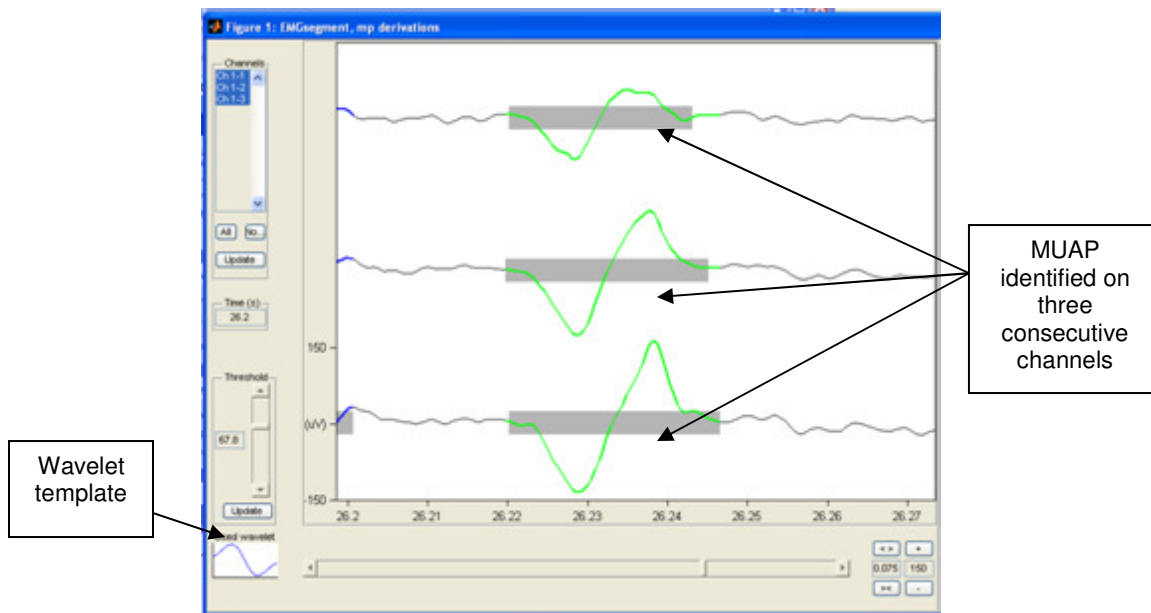
Figure 6.8. An example of a motor unit action potential (MUAP) detected using Continuous Wavelet Transform template matching.

There were sections of the signals that demonstrated multiple, superimposed MUAPs, which needed to be separated into individual MUAPs where possible (Figure 6.9). This was achieved by adapting a user-defined threshold. The grey blocks highlight where the activity of the signal has reached the threshold. MUAPS detected on all channels form the blue and green sections of the signal.
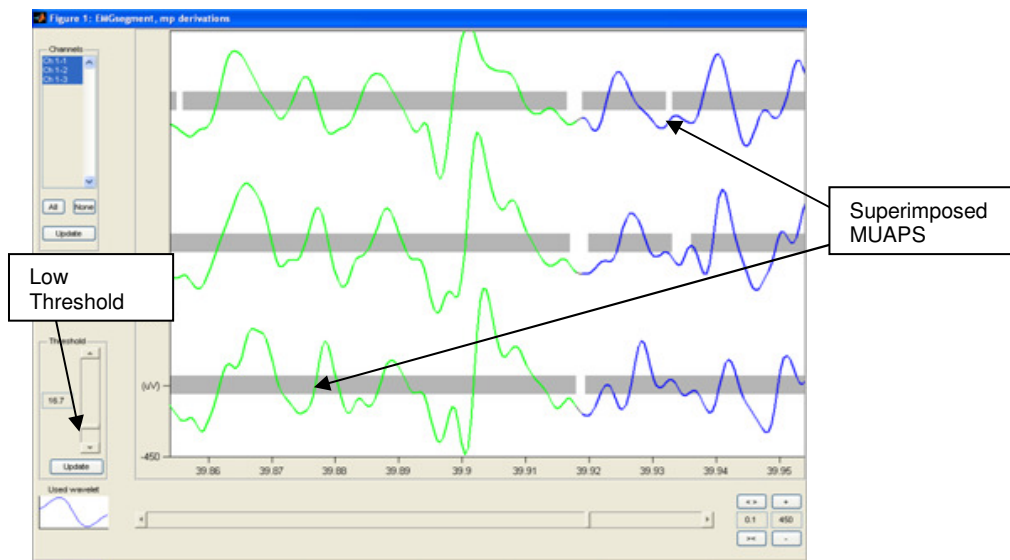
Figure 6.9 An example of superimposed MUAPs at a low detection threshold.

However, the lack of sensitivity of the software meant that separation of the multiple MUAPs could result in smaller, individual MUAPs not being detected (Figure 6.10). The same signal with different threshold values is shown in Figure 6.9 and 6.10. Figure 6.10 shows a higher sensitivity and reduced superimposition of MUAPs
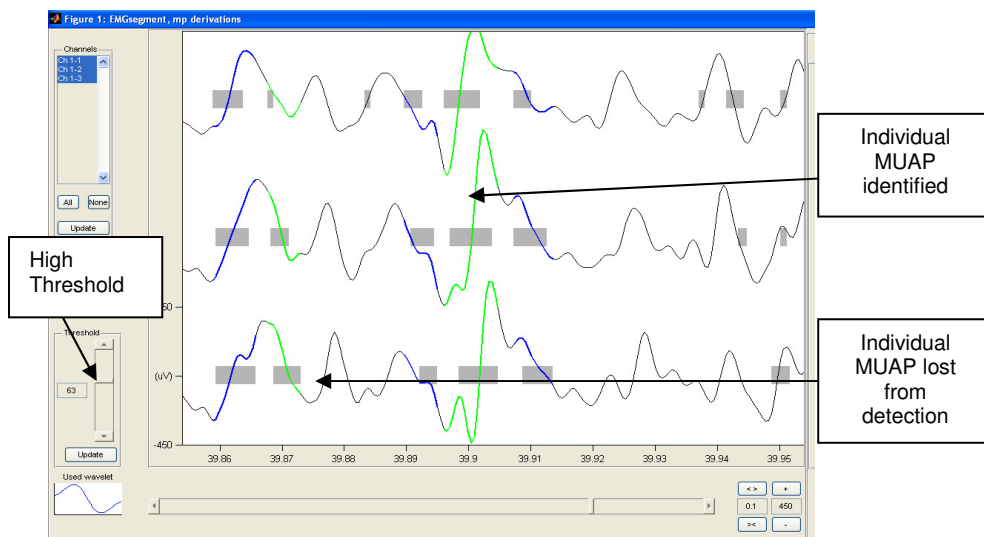
Figure 6.10. An example of individual MUAPs at a high detection threshold.

A threshold value was determined for each signal using the software operator's judgement. This allowed separation of superimposed MUAPs while retaining as

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many of the smaller individual MUAPs. This had to be undertaken for each of the 5 epochs for all signals. A uniform threshold value could not be used because the optimal value varied with differing levels of EMG activity both within and between subjects. The EMG activity of a signal could vary greatly along its duration and so it was not reliable to use the threshold value determined for an epoch at the beginning of a signal for an epoch at the end. The program then averaged the results from the remaining channels. In MUAP parameters a minimum of 2 and maximum of 3 channels were used for each muscle recording. For global parameters a minimum of 1 and a maximum of 3 channels were used for each muscle recording. The MDF and RMS parameters of the identified MUAPs were then automatically exported to an excel spreadsheet.

From the 5 epochs β regression coefficients were determined for global and MUAP MDF (as a measure of muscle fatigue), global and MUAP RMS (as a measure of muscle activity and fatigue) and MUAP rate (as a measure of central nervous system input). These analyses were used to address whether cervical muscle activation patterns differed in WAD, NTNP and HC participants.

Program 3 was written by Dr S. Notley, ISVR, University of Southampton. This program filtered signals using a 4^{th} order Butterworth bandpass filter, 10 – 400 Hz. It allowed extraction of MDF and RMS parameters of global EMG signals at regular epochs that corresponded to the timing of the subjective pain and fatigue ratings. This programme allowed the start time, the duration of epoch and time of each epoch to be entered. One channel per muscle was processed and suitable channels were determined through visual inspection of the signals in Program 2. Ten second epochs, which had been extracted every 30 seconds, were processed for the arm raise exercise. The duration of these signals was generally long, therefore 10 second epochs were deemed to be an appropriate length to provide representative signal samples. The head raise exercises were generally of a shorter duration, therefore 2 second epochs extracted every 10 seconds were used to provide representative signal samples. 5 seconds of the signal were removed from the beginning and end of the signal to correspond with the verbal rating scores.

These data were used to perform correlations of objective global sEMG measures (MDF and RMS) with subjective measures of pain and fatigue. This analysis

addressed whether there was a difference in the relationship between subjective measures of pain and fatigue and objective measures of muscle activity in WAD, NTNP and HC participants

6.9   Statistical analysis

The sEMG parameters GRMS, MRMS, GMDF, MMDF and MR data were presented as descriptive statistics by way of means and standard deviations. Box and whisker plots were used to display the medians and ranges. The data was explored and examined for normality using histograms with normal curves, Q-Q plots and the Kolmogorov-Smirnov (KS) test.

sEMG parameters were explored as epochs and β regression coefficients (for detail see section 3.9.2). The sample sizes were small and not all data demonstrated a normal distribution. Wilcoxon's signed-rank test was used to observe side to side differences for each parameter. These tests and data allowed any differences or trends to be identified. The Kruskal-Wallis test was applied to ascertain differences in the β regression coefficients between the groups.

In order to determine whether factors such as age, BMI and duration of pain affected these data collected, a parametric model was used. The repeated measures ANOVA was appropriate for use as it is a robust model that can be used when the data are of a near normal distribution. Age, duration of pain and NDI did not have a significant effect on these data; however BMI did have a significant effect.

The relationships between subjective scores and sEMG parameters for each individual were estimated using Spearman's correlation coefficient (*r*).

Statistical analysis was performed using SPSS version 16.0 for Windows (SPSS Inc., Chicago, Illinois, USA) and Microsoft Excel 2000 (Microsoft Corporation, Redmond, WA, USA). A probability level of $P < 0.05$ was set as the minimum criterion of statistical significance for all tests.

Chapter Seven – Results of Main Study

This chapter presents the results of the main study, which has been undertaken to observe the characteristics of muscle activation patterns and muscle synergy during non-forced fatiguing activities. The results also aim to determine possible differences in activation and synergy between the participant groups.

7.1   Participants

35 volunteer participants were recruited from the University of Southampton through poster advertisements. These participants were placed into 3 groups: healthy controls (HC, N=18, 5 males, 13 females) non-traumatic neck pain (NTNP, N=13, 3 males, 10 females) and whiplash associated disorder – grade 1 and 2 (WAD, N=4, 1 male, 3 females).

The mean age, weight, height, body mass index (BMI) and duration of pain of the participants are shown in Table 7.1.

Table 7.1 Mean age, weight, height, body mass index (BMI) and duration of pain of the HC, NTNP and WAD groups.

		Age in years	Height in cm	Weight in Kg	BMI	Duration of pain in months
HC (N=18)	mean (std)	39.4 (13.6)	171.7 (9.6)	70.2 (12.2)	23.5 (2.5)	N/A
	range (min-max)	18 - 63	155-191	52-108	19-23.5	N/A
NTNP (N=13)	mean (std)	42.1 (11.2)	170.8 (6.8)	72.9 (14.2)	24.7 (4.4)	110.9 (84.9)
	range (min-max)	22 - 59	106-182	51-91.2	17-30	14-300
WAD I & II (N=5)	mean (std)	33.5 (12.4)	176 (5.7)	76 (6.5)	24.4 (2.6)	38.3 (32.4)
	range (min-max)	22 - 47	170 - 183	67-82	22-28	9-72

As shown in Table 7.1 the NTNP group has the highest mean age and the HC group has the greatest range. The WAD group has the lowest mean age and the smallest range. Mean BMI is very similar for all 3 groups with the NTNP group showing the greatest range. The NTNP group has the longest mean and greater range for duration of pain than the WAD group.

32 participants were right-handed and 3 participants were left-handed. The left-handed participants stated that their left hand was dominant for writing and not for other tasks. Data were analysed for dominant non-dominant sides and no difference in the results were seen when compared to left to right side results. Therefore right and left side comparisons were made.

Participants in the NTNP and WAD groups completed the Neck Disability Index (NDI) questionnaire before each session and initial pain rating before each session. The mean results are shown in Table 7.2.

Table 7.2   Participants' mean pre-test pain ratings and NDI scores. The maximum pretest pain score was 10 and the maximum NDI score was 50.

	Pretest pain scores		NDI scores	
	Mean (std)	range min-max	mean (std)	range min-max
NTNP (N=13)	1.9 (1.19)	0 - 4	10.7 (3.54)	5 - 16
WAD (N=4)	3 (2.83)	1 – 7	7.7 (1.91)	5 - 9

The WAD group showed the highest mean pre-test pain scores and the NTNP group showed the highest mean NDI scores. All HC participants stated that they were not experiencing any pain at the time of the activities. Individual scores for NDI, BMI and the duration of pain are included in table 7.2

Table 7.3 Duration of arm raise and head raise activities.

	Arm raise (seconds)		Head raise (seconds)	
	mean (std)	range min-max	mean (std)	range min-max
HC (N=18)	323.3 (122.0)	150 - 570	72.9 (30.6)	30 - 150
NTNP (N=13)	390.0 (241.9)	120-1110	95.4 (57.8)	30 - 220
WAD (N=4)	300.0(54.8)	240 - 360	75.0 (31.1)	50 - 120

Table 7.4. Kruskal-Wallis test for duration of activities between groups.

	Arm raise	Head raise
p value	0.560	0.343

Although the NTNP group showed a slightly longer mean duration for both the arm raise and the head raise activity (Table 7.3), there were no statistically significant differences in duration between the groups (Table 7.4)

One participant in the NTNP group maintained the arm raise position for 1110 seconds, which was much longer than any other participant. Exclusion of this participant's data from analysis did not greatly affect the mean value and the NTNP group still showed the longest mean duration for the arm raise exercise.

Table 7.5 Individual participant demographic information, sex, age handedness, BMI, NDI, duration of pain and a description of the location of pain.

Participant	Sex	Age	Handedness	BMI	NDI	Duration of pain (months)	Location of pain
control 1	M	26	L	21	N/A	N/A	N/A
control 2	F	36	R	23	N/A	N/A	N/A
control 3	F	34	R	23	N/A	N/A	N/A
control 4	F	43	L	24	N/A	N/A	N/A
control 5	F	42	R	22	N/A	N/A	N/A
control 6	F	21	R	25	N/A	N/A	N/A
control 7	F	40	R	22	N/A	N/A	N/A
control 8	F	50	R	24	N/A	N/A	N/A
control 9	F	63	R	18	N/A	N/A	N/A
control 10	F	18	R	23	N/A	N/A	N/A
control 11	M	45	R	29	N/A	N/A	N/A
control 12	F	45	R	21	N/A	N/A	N/A
control 13	M	25	R	21	N/A	N/A	N/A
control 14	F	28	R	28	N/A	N/A	N/A
control 15	M	53	R	25	N/A	N/A	N/A

Participant	Sex	Age	Handedness	BMI	NDI	Duration of pain (months)	Location of pain
control 16	M	25	R	23	N/A	N/A	N/A
control 17	F	58	R	22	N/A	N/A	N/A
control 18	F	57	R	24	N/A	N/A	N/A
NTNP 19	M	53	R	16	10	66	neck / front L neck / rear L shoulder / front L shoulder / rear L & C
NTNP 20	F	35	R	19	14	216	neck / rear L & R & C shoulder / rear L & R head / rear L behind left eye
NTNP 21	F	27	R	20	16	168	neck / rear L & R shoulder / rear L&R head / front and rear top
NTNP 22	F	33	L	24	11	24	neck / front L & R neck / rear L & R shoulder / front L & R shoulder / rear L & R & C
NTNP 23	F	33	R	21	10	96	neck / rear L & R & C shoulder / rear L & C
NTNP 24	M	44	R	25	9	24	neck / front L & R shoulder / front L & R arms / front L & R
NTNP 25	F	59	R	25	11	96	neck / front L shoulder / front L shoulder / rear L & C
NTNP 26	F	47	R	28	6	14	neck / rear R shoulder / rear R & C arm / rear R
NTNP 27	M	43	R	20	10	192	neck / rear L & R & C head / rear L & R & C head / top
NTNP 28	F	48	R	30	16	66	neck / rear C
NTNP 29	F	22	R	30	14	96	neck / rear L & R & C shoulders / rear L & R
NTNP 30	F	48	R	28	7	84	neck / front L & R neck / rear L & R & C shoulders / front L & R shoulders / rear L, R & C
NTNP 31	M	55	R	28	5	300	neck / rear R
WAD 32	F	24	R	24	9	18	neck / front L & R neck / rear L & R
WAD 33	F	22	R	22	9	12	neck / rear C shoulder / rear L & R
WAD 34	M	47	R	23	7	60	neck / rear R
WAD 35	F	41	R	28	5	72	neck / rear C shoulder / rear L & R
KEY: L – Left, R – Right, C – Centre, M – Male, F – Female							

Table 7.5 show 71% of NTNP and WAD participants have pain in both the neck and shoulders. 65% of NTNP and WAD participants have pain in both the left and right sides. 4 NTNP and 1 WAD subjects had side specific pain.

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7.2 Arm raise activity

This section presents the results of the arm raise activity with a detailed analysis of individual muscle activity. Box and whisker plots for; amplitude: global root mean square (GRMS), motor unit action potential root mean square (MRMS), frequency: global median frequency (GMDF), motor unit action potential median frequency (MMDF) and motor unit action potential rate (MR) are included to show the distribution of the data over the 5 epochs. Tables of mean β regression coefficients for each parameter are also presented.

Post Hoc tests were not undertaken due to the small and uneven group sizes, which would make the reliability of these tests questionable. By referring to the relevant graphs it is possible to observe differences between the groups. In the arm raise activity, the upper trapezius muscles were predominantly activated.

The results of all muscles are presented, whether or not differences were reported, to provide a complete picture of muscle activation and synergies. It should be noted that the box and whisker plots scales vary and this should be taken into account when comparing the figures

7.2.1 Right upper trapezius

The Kruskal-Wallis test was applied to β regression coefficients for all parameters of the right upper trapezius. No statistically significant differences were found.

Table 7.6 Mean β regression coefficients of GRMS and MRMS (µV per epoch) for the right upper trapezius

	Mean β regression coefficients of GRMS (std)	Mean β regression coefficients of MRMS (std)
HC (N=16)	8.04 (8.37)	5.82 (6.37)
NTNP (N=12)	7.62 (6.01)	4.79 (5.06)
WAD (N=3)	6.55 (5.91)	3.03 (5.34)

Table 7.6 shows a trend for higher mean GRMS and MRMS β regression coefficients in HC group and lower values in the WAD group. The WAD group had the largest difference between the GRMS and MRMS values.
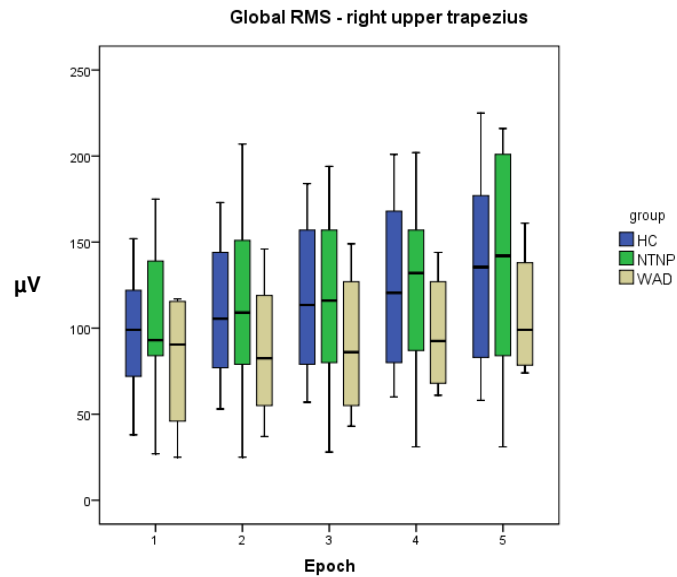
Figure 7.1 Distribution of GRMS values for the right upper trapezius.

Figure 7.1 shows the data range for the 3 groups was similar, with no statistically significant differences seen in values between the groups over the 5 epochs (Table 7.10). However, a trend for lower median GRMS values is seen in the WAD group.
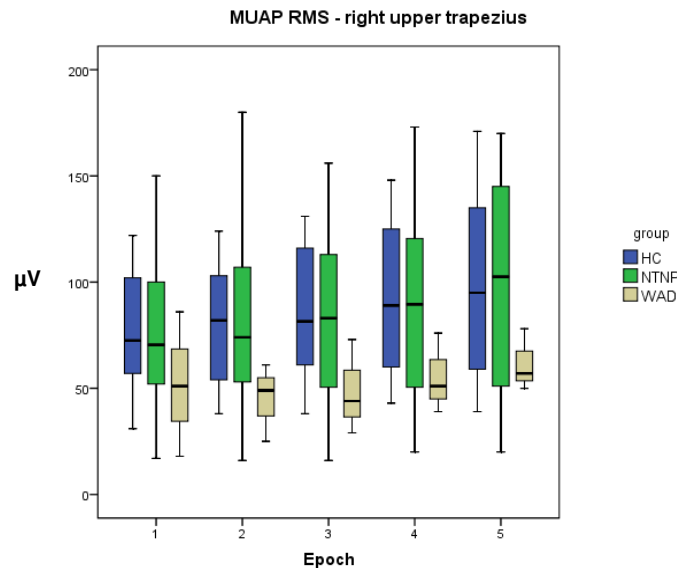
Figure 7.2 Distribution of MRMS values for the right upper trapezius.

Statistically significant differences (p=0.042) between the groups were observed between the groups for MRMS values over the 5 epochs (Table 7.10). Figure 7.2 shows that the WAD group consistently had the smallest range of data with the

lowest median values and that the NTNP group had the greatest distribution of data for MRMS.

Table 7.7 Mean β regression coefficients of GMDF and MMDF (Hz per epoch) for the right upper trapezius.

	Mean β regression coefficients of GMDF (std)	Mean β regression coefficients of MMDF (std)
HC (N=18)	-2.02 (2.18)	-1.82 (1.49)
NTNP (N=12)	-1.52 (1.08)	-1.61 (1.44)
WAD (N=4)	-0.75 (0.98)	-1.73 (1.66)

Table 7.7 shows that the HC group showed a slight trend for more negative mean β regression coefficient values for both GMDF and MMDF.
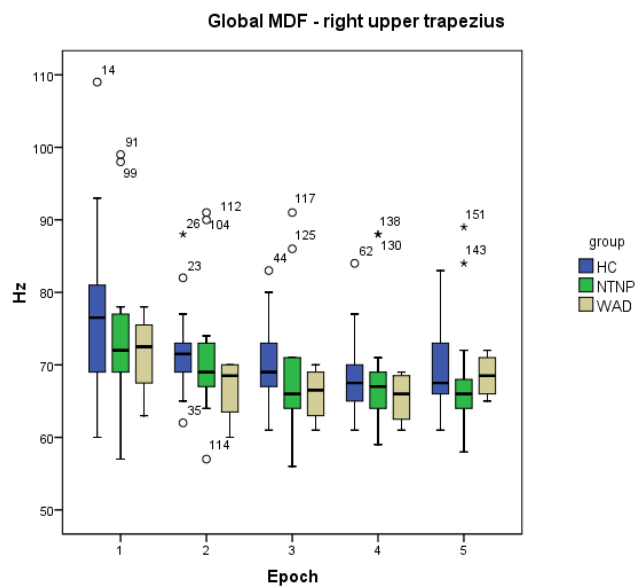
Figure 7.3 Distribution of GMDF values for the right upper trapezius.

Figure 7.3 shows that the median GMDF values for the groups were similar with no statistically significant differences in values over the 5 epochs (Table 7.9). The outlying data points indicate the individual variation in GMDF values.
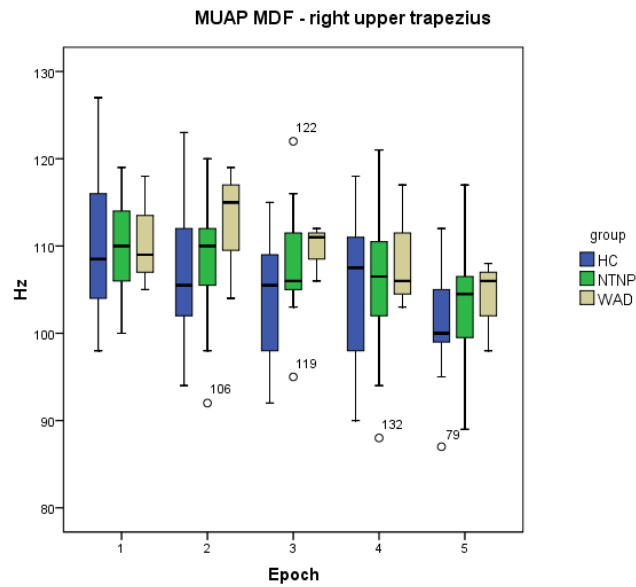
Figure 7.4 Distribution of MMDF values for the right upper trapezius.

The median MMDF values decreased from epoch 1 - 5 for all groups and showed no statistically significant differences, as shown in Figure 7.4 and Table 7.10.

Table 7.8 Mean MR (MUAPs per second) for the right upper trapezius.

Epoch	Mean MR		
	HC (N=16) mean (std)	NTNP (N=12) mean (std)	WAD (N=3) mean (std)
1	28.3 (2.9)	27.5 (2.1)	28.0 (1.8)
2	29.4 (3.0)	27.7 (1.3)	29.7 (2.0)
3	30.3 (2.9)	28.1 (3.3)	30.8 (1.7)
4	28.9 (3.5)	27.8 (2.6)	31.1 (3.0)
5	29.5 (2.3)	29.4 (3.2)	33.3 (2.0)
Difference between epoch 1 and 5	1.2	1.9	5.3

The WAD group showed more of an increase in the mean MUAP rate over the 5 epochs than the HC and NTNP groups, as seen in Table 7.8.

Table 7.9 Mean β regression coefficients of MR (MUAP/second per epoch) for the right upper trapezius.

	Mean β regression coefficients of MR (std)
HC (N=16)	0.29 (1.08)
NTNP (N=12)	0.22 (0.47)
WAD (N=3)	-0.56 (1.46)

Table 7.9 shows that the mean β regression coefficient values for MR were slightly positive for the HC and NTNP groups and slightly negative for the WAD group.
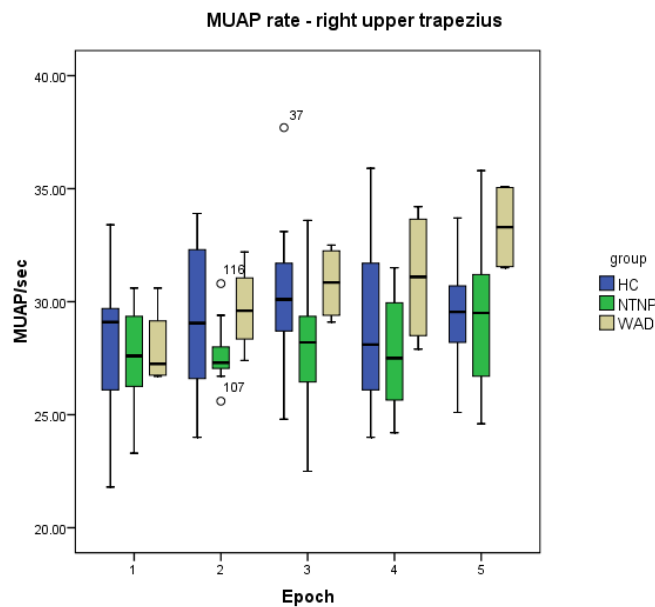
Figure 7.5 Distribution of MR values for the right upper trapezius.

Although differences in the MR values between the groups were not statistically significant, Figure 7.5 and Table 7.8 show a trend for a greater increase in median and mean MR values over the 5 epochs in the WAD group.

Table 7.10 Repeated measures ANOVA between groups (with BMI as a covariate) for the right upper trapezius.

Repeated measures ANOVA - RUTRP	
	Test of between subjects effects (p value)
GRMS	0.256
MRMS	0.042*
GMDF	0.697
MMDF	0.638
MR	0.075

As shown in Table 7.10, the test of between subjects' effects showed a statistically significant difference between the groups in mean MRMS values over the 5 epochs

7.2.2 Summary of right upper trapezius activity

Statistically significant differences were seen between the groups for MRMS. The WAD group showed the lowest values. The WAD group also showed a trend for a greater increase in MR over the 5 epochs. The HC and NTNP groups showed similar GRMS, MRMS and MR. All groups had similar initial values for all parameters and all groups showed similar MDF values.

7.2.3 Left Upper Trapezius

The Kruskal-Wallis test was applied to β regression coefficients for all parameters of the left upper trapezius. No statistically significant differences were found.

Table 7.11 Mean β regression coefficients of GRMS and MRMS (μV per epoch) for the left upper trapezius.

	Mean β regression coefficients of GRMS (std)	Mean β regression coefficients of MRMS (std)
HC (N=17)	11.09 (10.2)	8.09 (7.74)
NTNP (N=12)	7.86 (8.70)	5.45 (6.42)
WAD (N=4)	10.2 (5.3)	5.97 (3.95)

Table 7.11 shows that the HC group had the most positive β regression coefficients means for GRMS and MRMS. The WAD group had the greatest difference between the mean GRMS and MRMS slopes.
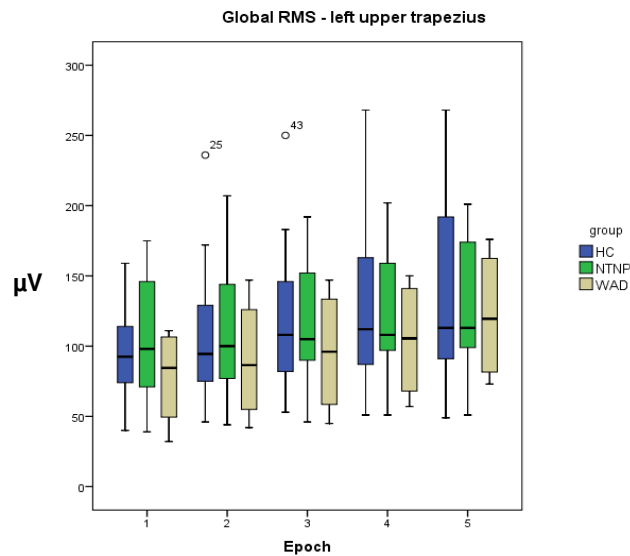
Figure 7.6 Distribution of GRMS values for the left upper trapezius.

There were no statistically significant differences between the groups for GRMS values over the 5 epochs (Table 7.15). Median values were similar for all groups as shown in Figure 7.6.
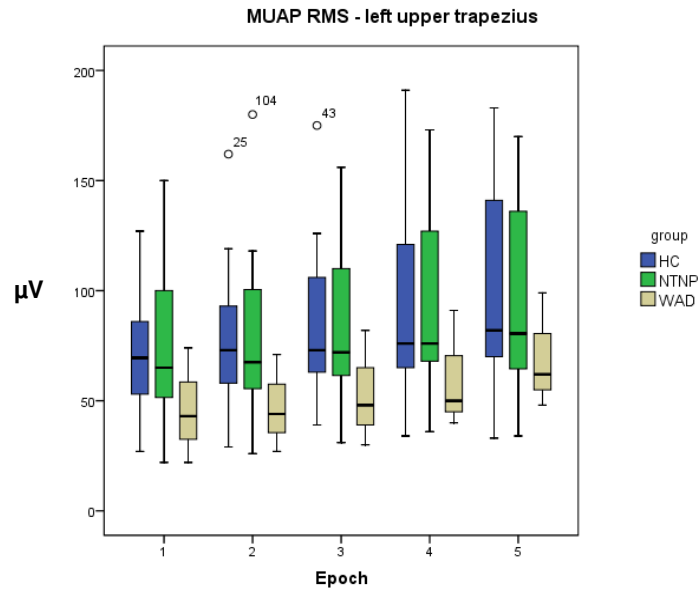
Figure 7.7 Distribution of MRMS values for the left upper trapezius.

Although not statistically significant, the WAD group showed a trend for lower median values and a smaller range of data compared to the HC and NTNP groups over the 5 epochs, as seen in Figure 7.7 and Table 7.15. This pattern of activity between groups is very similar to that seen in the right upper trapezius (Figure 7.2).

Table 7.12 Mean β regression coefficients of GMDF and MMDF (Hz per epoch) for the left upper trapezius.

	Mean β regression coefficients of GMDF (std)	Mean β regression coefficients of MMDF (std)
HC (N=17)	-2.54 (2.69)	-2.23 (1.64)
NTNP (N=12)	-2.19 (1.98)	-1.90 (2.09)
WAD (N=4)	-0.80 (1.54)	0.25 (1.20)

Table 7.12 shows that the HC and NTNP groups had more negative mean regression coefficients for both GMDF and MMDF the WAD group.
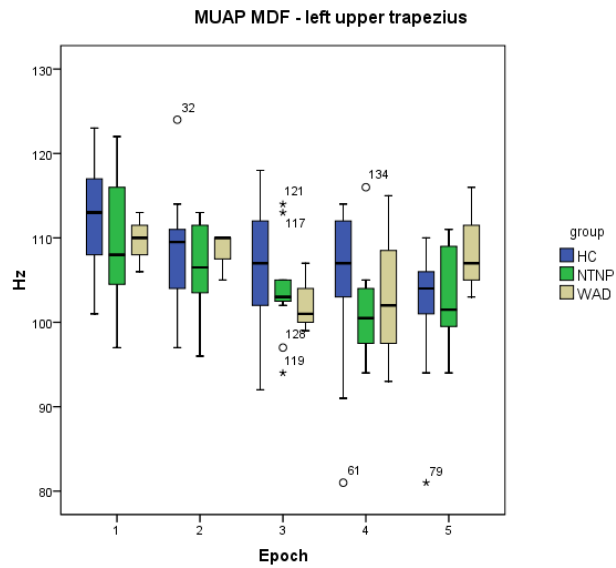
Figure 7.8 Distribution of GMDF values for the left upper trapezius.

As seen in Figure 7.8, all groups showed similar median values for GMDF. Although not statistically significant, the WAD group showed a trend for the smallest range of data over the 5 epochs (Table 7.15).
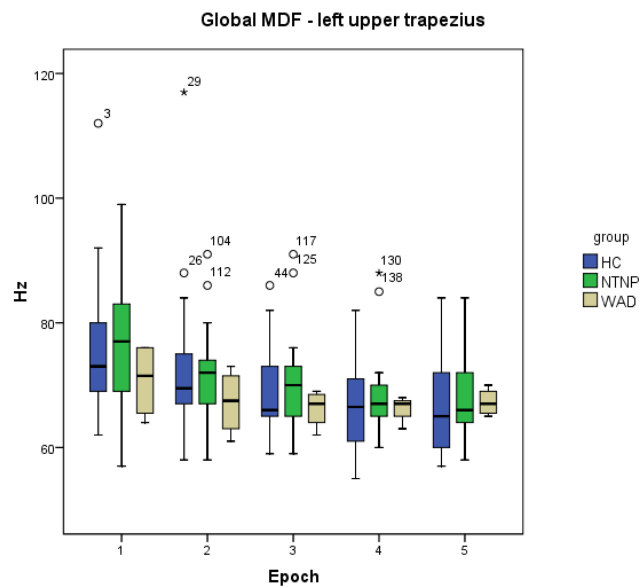
Figure 7.9 Distribution of MMDF values for the left upper trapezius.

Figure 7.9 and Table 7.15 shows no statistically significant differences and no consistency of data distribution and median values were seen for any of the groups over the 5 epochs.

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Table 7.13 Mean MR (MUAPs per second) for the left upper trapezius.

Epoch	Mean MR		
	HC (N=17) mean (std)	NTNP (N=12) mean (std)	WAD (N=4) mean (std)
1	27.7 (2.9)	28.1 (2.1)	31.8 (3.2)
2	29.1 (2.6)	28.9 (2.4)	32.3 (0.9)
3	29.3 (2.5)	29.0 (2.6)	35.4 (3.5)
4	28.9 (3.4)	28.7 (2.9)	32.7 (2.1)
5	28.5 (2.4)	28.8 (3.6)	33.3 (0.8)
Difference between epoch 1 and 5	0.8	0.7	1.5

The WAD group showed a trend for higher MR values than the HC and NTNP groups. All groups showed a slight increase from epoch 1 to 5, as seen in Table 7.12.

Table 7.14 Mean β regression coefficients of MR (MUAP/second per epoch) for the left upper trapezius.

	Mean β regression coefficients of MR (std)
HC (N=17)	0.05 (0.72)
NTNP (N=12)	-0.26 (0.81)
WAD (N=4)	-0.20 (0.96)

Table 7.14 shows that there were no statistically significant differences in mean β regression coefficient values for MR between the groups. The mean β regression coefficient values for MR were negative for the NTNP and WAD groups. The HC group showed a very slight positive value.
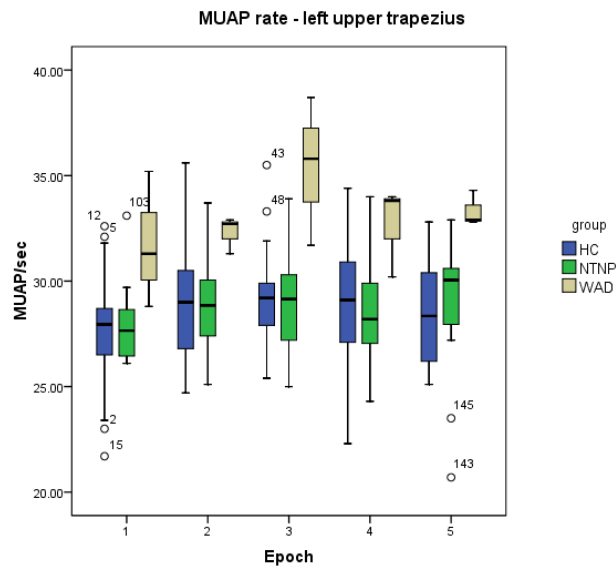
Figure 7.10 Distribution of MR values for the left upper trapezius

Table 7.15 Statistically significant differences in the progression of MR between the groups were observed. Consistently higher values for the WAD group over the 5 epochs are shown in Figure 7.10. The HC and NTNP groups show similar values.

Table 7.15 Repeated measures ANOVA between groups (with BMI as a covariate) for the left upper trapezius

Repeated measures ANOVA - LUTRP	
	Test of between subjects effects (p value)
GRMS	0.575
MRMS	0.134
GMDF	0.482
MMDF	0.649
MR	0.011

As shown in Table 7.15, the test of between subjects' effects with BMI as a covariate showed statistically significant differences in MR between the groups.

7.2.4   Summary of left upper trapezius activity

No statistically significant differences were seen between the groups for any parameter in the left upper trapezius (Table 7.15). The WAD group showed a trend for the lowest GRMS and MRMS values and the highest values for MR over the 5 epochs. The HC and NTNP groups showed similar GRMS, MRMS and MR. All groups showed a similar progression of GMDF and MMDF values.

7.2.5   Right Sternocleidomastoid

(MDF values were unobtainable for the sternocleidomastoid muscles during the arm raise activity due to a poor signal to noise ratio)

The Kruskal-Wallis test was applied to β regression coefficients for all parameters of the right sternocleidomastoid muscle. No statistically significant differences were found.

Table 7.16 Mean β regression coefficients (µV per epoch) for the GRMS and MRMS of the right sternocleidomastoid muscle.

	Mean β regression coefficients of GRMS (std)	Mean β regression coefficients of MRMS (std)
HC (N=17)	3.27 (4.93)	2.68 (4.18)
NTNP (N=12)	2.84 (5.36)	1.41 (2.74)
WAD (N=3)	0.76 (0.74)	0.70 (0.72)

Table 7.16 shows the β regression coefficient means for both GRMS and MRMS were more positive for the HC group and less positive for the WAD group.
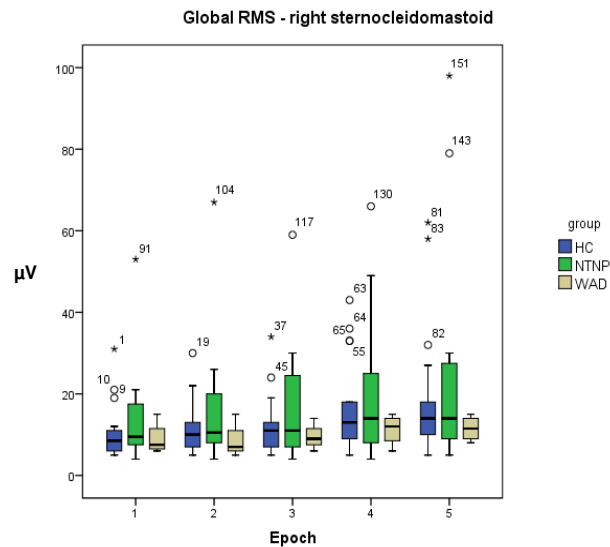
Figure 7.11 Distribution of GRMS values for the right sternocleidomastoid.

Statistically significant differences (p=0.027) between the groups were seen in GRMS values over the 5 epochs (Table 7.19). The NTNP group showed the greatest distribution of data. The number of outlying data points in Figure 7.11 illustrates the amount of individual variation in activation of this muscle.
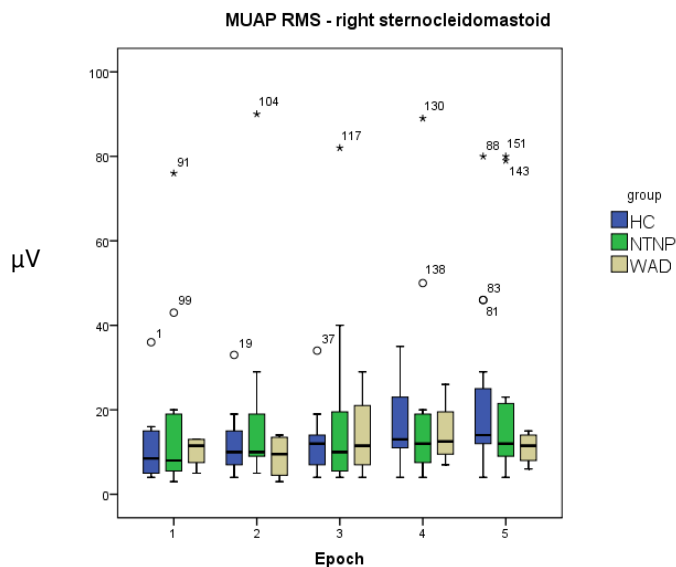
Figure 7.12 Distribution of MRMS values for the right sternocleidomastoid.

Statistically significant differences between the groups were seen in MRMS activity over the 5 epochs (p=0.042) (Table 7.19). The NTNP groups showed the greatest distribution of data. The number of outlying data points in Figure 7.12 illustrates the amount of individual variation in activation of this muscle.

Table 7.17 Mean MR (MUAPs per second) for the right sternocleidomastoid.

Epoch	Mean MR		
	HC (N=17) mean (std)	NTNP (N=12) mean (std)	WAD (N=3) mean (std)
1	5.8 (10.4)	9.6 (10.4)	4.2 (5.5)
2	8.4 (10.8)	12.7 (12.0)	3.6 (5.7)
3	10.5 (12.9)	12.6 (11.6)	7.3 (6.4)
4	13.1 (12.3)	16.7 (13.2)	10.1 (6.7)
5	13.7 (11.5)	17.2 (11.7)	11.4 (7.1)
Difference between epoch 1 and 5	7.9	7.6	7.2

All groups showed a similar increase in mean MR over the 5 epochs, as seen in Table 7.17. The WAD group had consistently lower values and the NTNP had consistently higher values.

Table 7.18 Mean β regression coefficients of MR (MUAP/second per epoch) for the right sternocleidomastoid

	Mean β regression coefficients of MR (std)
HC (N=17)	1.07 (2.32)
NTNP (N=12)	0.32 (0.71)
WAD (N=3)	0.18 (0.62)

All groups showed positive regressions coefficients. Table 7.18 shows that the HC group show the most positive slope for increase in MR.
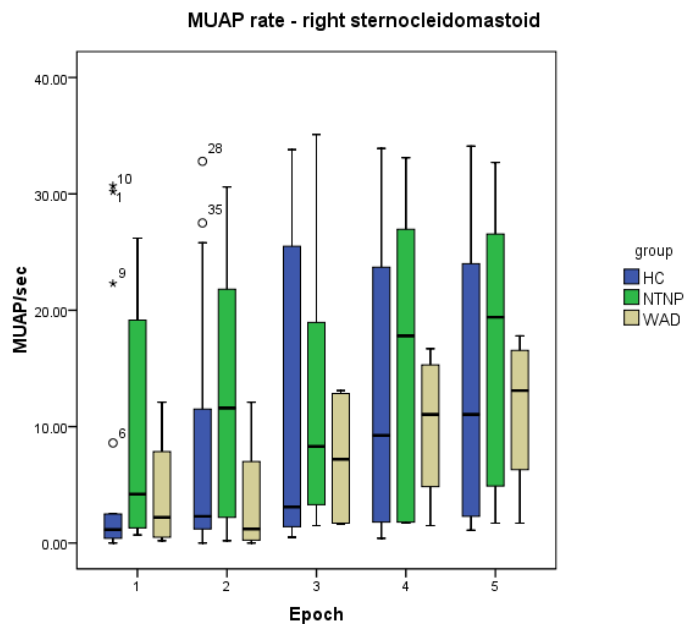
Figure 7.13 Distribution of MR values for the right sternocleidomastoid

Figure 7.13 and Table 7.19 show that although there were no statistically significant differences, a trend for higher median MR values was seen in the NTNP group. With the exception of epoch 2, a trend for lower median MR values was seen in the HC group.

Table 7.19 Repeated measures ANOVA between groups (with BMI as a covariate) for the right sternocleidomastoid

Repeated measures ANOVA - RSCM	
	Test of between subjects effects (p value)
GRMS	0.027*
MRMS	0.042*
MR	0.184

Table 7.19. The test of between subjects' effects with BMI as a covariate showed a statistically significant difference in both mean GRMS and MRMS when comparing the 3 groups over the 5 epochs.

7.2.6   Summary of right sternocleidomastoid activity

Statistically significant differences were seen between the groups for GRMS and MRMS in the right sternocleidomastoid muscle (Table 7.18). The WAD group showed the lowest GRMS and MRMS values. The NTNP group showed the highest GRMS and MRMS with a trend for the highest MR values. The HC groups showed a trend for the lowest MR values. All groups showed a similar increase in MR over the 5 epochs.

7.2.7   Left Sternocleidomastoid

The Kruskal-Wallis test was applied to β regression coefficients for all parameters of the left upper trapezius. No statistically significant differences were found.

Table 7.20 Mean β regression coefficients of GRMS and MRMS (μV per epoch) for the left sternocleidomastoid.

	Mean β regression coefficients of GRMS (std)	Mean β regression coefficients of MRMS (std)
HC (N=17)	2.73 (4.00)	2.33 (3.24)
NTNP (N=12)	4.60 (5.40)	2.10 (2.2)
WAD (N=3)	5.00 (7.40)	0.96 (0.95)

As seen in Table 7.20, the WAD group showed the more positive mean β regression coefficient for GRMS and the HC group showed the more positive mean β regression coefficient for MRMS. The WAD group had the greatest difference between the mean β regression coefficients for GRMS and MRMS.
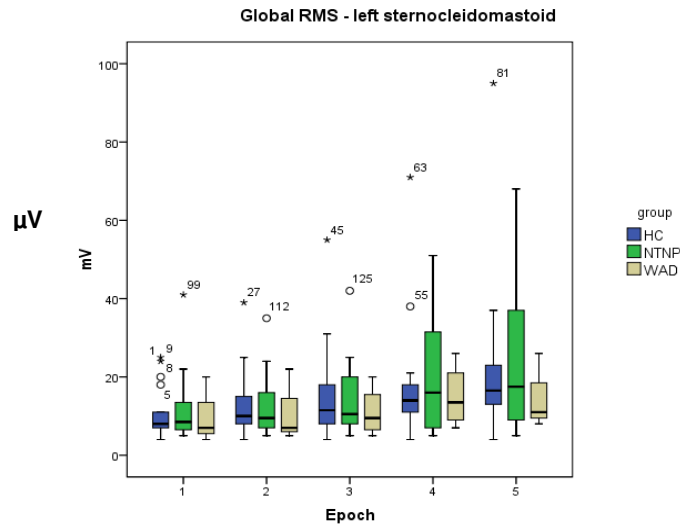
Figure 7.14 Distribution of GRMS values for the left sternocleidomastoid.

There were no significant differences in GRMS values between the groups over the 5 epochs (Table 7.23). Figure 7.14 shows similar median values for all groups over the 5 epochs. The NTNP groups show a slight increase in distribution of data.
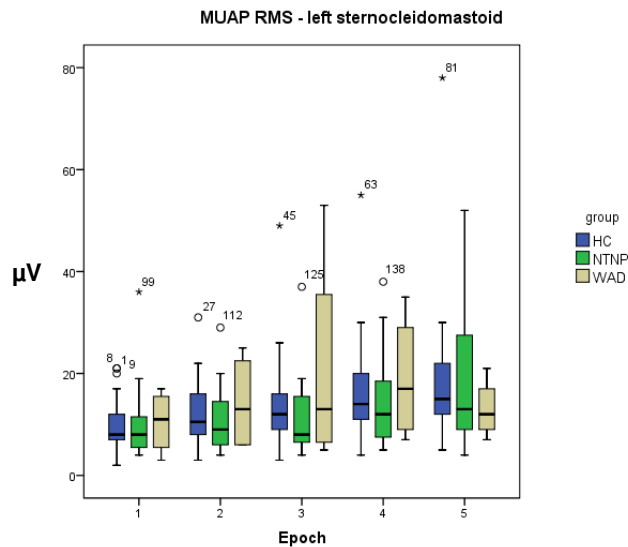
Figure 7.15 Distribution of MRMS values for the left sternocleidomastoid.

With the exception of epoch 5, Figure 7.15 shows the WAD group had slightly higher median MRMS values. There were no significant differences between the groups over the 5 epochs (Table 7.23).

Table 7.21 Mean MR (MUAPs per second) for the left sternocleidomastoid.

Epoch	Mean MR		
	HC (N=17) mean (std)	NTNP (N=12) mean (std)	WAD (N=3) mean (std)
1	7.9 (11.7)	9.8 (11.6)	6.3 (12.1)
2	11.6 (12.3)	11.6 (12.4)	5.4 (9.9)
3	13.8 (13.1)	14.1 (11.7)	9.4 (10.2)
4	14.7 (10.7)	16.5 (13.6)	11.4 (10.3)
5	20.1 (9.5)	15.8 (11.4)	13.1 (12.2)
Difference between epoch 1 and 5	12.2	6.0	6.8

Table 7.21 shows a trend for the highest increase in MR in the HC group over the 5 epochs.

Table 7.22 Mean β regression coefficients of MR (MUAP/second per epoch) for the left sternocleidomastoid.

	Mean β regression coefficients of MR (std)
HC (N=17)	-0.09 (1.99)
NTNP (N=12)	-0.44 (1.10)
WAD (N=3)	0.56 (0.69)

Table 7.22 shows that the mean β regression coefficients of MR for the HC and NTNP groups were slightly negative. The WAD group showed a positive mean β regression coefficient.
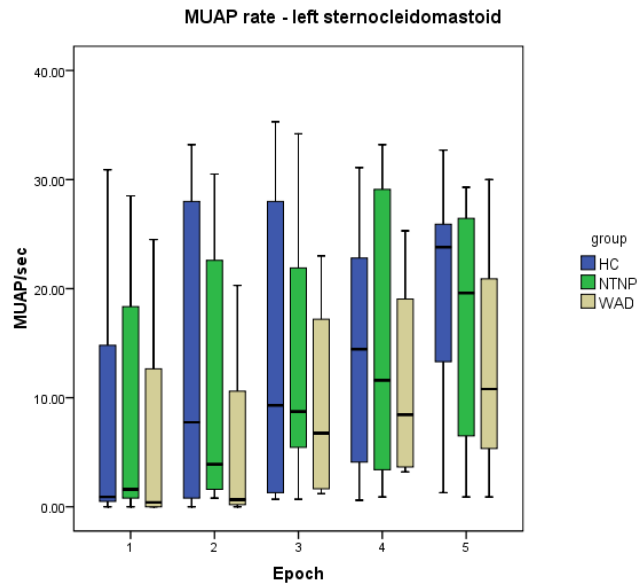
Figure 7.16 Distribution of MR values for the left sternocleidomastoid.

No statistically significant differences were seen between the groups over the 5 epochs (Table 7.23). A trend for higher median MR values over the 5 epochs is seen in the HC group, as shown in Figure 7.16.

Table 7.23 Repeated measures ANOVA between groups (with BMI as a covariate) for the left sternocleidomastoid

Repeated measures ANOVA - LSCM	
	Test of between subjects effects (p value)
GRMS	0.231
MRMS	0.657
MR	0.332

Table 7.23 the test of between subjects' effects with BMI as a covariate showed no statistically significant difference in GRMS, MRMS or MR when comparing the 3 groups.

7.2.8   Summary of left sternocleidomastoid activity

There were no statistically significant differences between the 3 groups, for any parameter in the left sternocleidomastoid muscle over the 5 epochs (Table 7.23). The WAD group showed a trend for the lowest GRMS and MRMS, with the lowest MR. The HC and NTNP groups showed similar GRMS, MRMS and similar MR. The HC group showed the highest increase in MR over the 5 epochs.

7.2.9   A summary table of muscle synergy in the arm raise activity

Table 7.24 A summary of muscle activation in the arm raise activity. The changes are based on the differences between epochs 1 and 5. Cross-referencing to the relevant results and discussion sections are indicated.

Muscle	Parameter	HC	NTNP	WAD	Results reference (Figure no.)	Discussion reference
RUTRP	GRMS	↑↑	↑↑	↑	7.1	8.1.1
	MRMS *	↑↑	↑↑	↑	7.2	8.1.1
	GMDF	↓	↓	↓	7.3	8.1.1
	MMDF	↓	↓	↓	7.4	8.1.1
	MR	↔	↔	↑	7.5	8.1.1
LUTRP	GRMS	↑↑	↑↑	↑↑	7.6	8.1.1
	MRMS	↑	↔	↑	7.7	8.1.1
	GMDF	↓	↓	↔	7.8	8.1.1
	MMDF	↓	↓	↔	7.9	8.1.1
	MR*	↔	↔	↑	7.10	8.1.1
RSCM	GRMS*	↑↑	↑↑	↑	7.11	8.1.2
	MRMS*	↑↑	↑↑	↔	7.12	8.1.2
	MR	↑↑	↑↑	↑↑	7.13	8.1.2
LSCM	GRMS	↑↑	↑↑	↑	7.14	8.1.2
	MRMS	↑↑	↑↑	↑	7.15	8.1.2
	MR	↑	↑	↑	7.16	8.1.2

Key to muscle activity over the 5 epochs: ↑↑large Increase, ↑ increase, ↓↓ large decrease ↓ decrease, ↔ No / very little change, * significant difference between groups (P<0.05).

7.2.10 Side to side differences

Side to side differences were explored for all sEMG parameters in the upper trapezii and sternocleidomastoid muscles during the arm raise activity. No trends or statistically significant differences were identified.

7.2.11 Correlations between subjective measures of pain and fatigue with objective measures of muscle activity

During the arm raise activity subjective scores of pain and fatigue were given every 30 seconds. Individual scores were correlated with corresponding GRMS and GMDF values. The number of individual significant correlations is expressed as both number and percentages within each group. Groups that contain more than 50% significant correlations are highlighted.

Table 7.25 Number of subjects and percentage per group of significant positive (p<0.05) Spearman's correlations of individual pain or fatigue scores with GRMS values.

		RUTRP GRMS number of correlations (%)	LUTRP GRMS number of correlations (%)	RSCM GRMS number of correlations (%)	LSCM GRMS number of correlations (%)
Pain	HC (N=18)	10 (56)	10 (56)	9 (50)	8 (44)
	NTNP (N=12)	8 (66)	9 (75)	6 (50)	7 (58)
	WAD (N=4)	1 (25)	1 (25)	1 (25)	4 (100)
Fatigue	HC (N=18)	11 (61)	10 (56)	8 (44)	10 (56)
	NTNP (N=12)	9 (75)	10 (83)	6 (50)	8 (67)
	WAD (N=4)	1 (25)	3 (75)	2 (50)	4 (100)

An increase in pain and fatigue scores with an increase in GRMS values result in positive correlations. In Table 7.25, the NTNP group showed the highest number of significant positive correlations for pain and fatigue with the UTRP muscles.

Table 7.26 Number of subjects and percentage per group of significant negative (p<0.05) Spearman's correlations of individual pain and fatigue scores with GMDF values.

		RUTRP GMDF number of correlations (%)	LUTRP GMDF number of correlations (%)
Pain	HC (N=18)	6 (33)	6 (33)
	NTNP (N=12)	3 (25)	4 (33)
	WAD (N=4)	0 (0)	1 (25)
Fatigue	HC (N=18)	7 (39)	6 (33)
	NTNP (N=12)	4 (33)	5 (42)
	WAD (N=4)	1 (25)	1 (25)

Increasing pain and fatigue scores with decreasing GMDF values result in negative correlations. Table 7.26 shows that no group achieved over 50% significant subject correlations.

Table 7.27 Number of subjects and percentage per group of significant positive (p<0.05) Spearman's correlations of individual pain and fatigue scores

	HC (N=18) number of correlations (%)	NTNP (N=12) number of correlations (%)	WAD (N=4) number of correlations (%)
Pain v Fatigue	18 (100)	12 (100)	4 (100)

Table 7.27 shows that all subjects from the three groups showed significant positive correlations of pain and fatigue scores.

There were no identifiable patterns or trends in participants with side specific pain.

Correlations were undertaken for each muscle of pre-test and NDI scores with mean β regression coefficients for GRMS and GMDF. There were no trends seen for the NTNP and WAD groups for any parameter.

7.3   Head raise activity

The results of the head raise activity are presented in this section, with a detailed analysis of individual muscle activity. Box and whisker plots for; amplitude, global RMS (GRMS) and frequency, global MDF (GMDF) are included to show the distribution of the data over the 5 epochs. Tables of mean β regression coefficients for each parameter are also presented.

As with the arm raise activity Post Hoc tests were not undertaken due to the small and uneven group sizes, which would make the reliability of these tests questionable. By referring to the relevant graphs it is possible to observe differences between the groups that have been identified by the repeated measures ANOVA. In this activity the sternocleidomastoid muscles were predominantly activated.

(MUAP values were unobtainable for all muscles due to electrode movement over the skin. MDF values were unobtainable for the upper trapezii muscles during the head raise activity due to the poor signal to noise ratio).

7.3.1   Right upper trapezius

The Kruskal-Wallis test was applied to β regression coefficients for GRMS (µV per epoch) in the right upper trapezius. No significant differences were found between the groups.

Table 7.28 Mean β regression coefficients of GRMS (µV per epoch) for the right upper trapezius.

	Mean β regression coefficients of GRMS (std)
HC (N=17)	1.41 (1.72)
NTNP (N=13)	1.23 (2.60)
WAD (N=3)	0.20 (0.20)

The WAD group showed the least positive mean β regression coefficient of the 3 groups (Table 7.28).
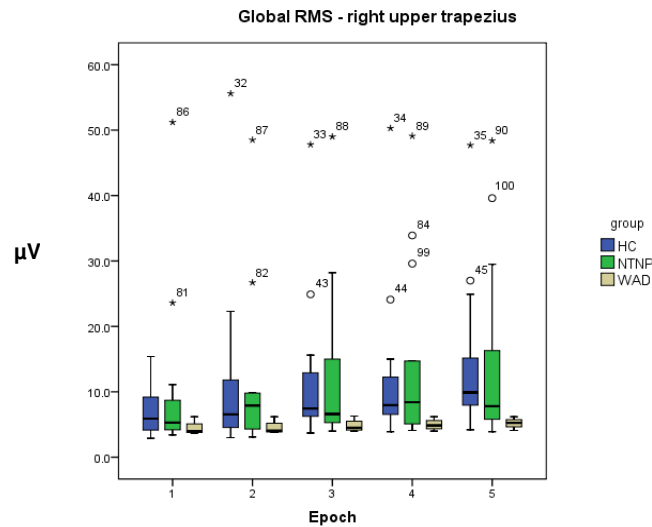
Figure 7.17 Distribution of GRMS values for the right upper trapezius.

No significant differences in GRMS were seen between the groups over the 5 epochs. However, the WAD group shows a trend for the lowest median values and the smallest distribution of data for all 5 epochs (Table 2.28 and Figure 7.17). The number of outlying data points illustrates the individual variation in this muscle.

Table 7.29 Repeated measures ANOVA between groups (with BMI as a covariate) for the right upper trapezius

Repeated measures ANOVA - RUTRP	
	Test of between subjects effects (p value)
GRMS	0.285

As can be seen in Table 2.29, the test of between subjects' effects with BMI as a covariate showed no statistically significant difference in GRMS when comparing the 3 groups.

7.3.2   Left upper trapezius

The Kruskal-Wallis test was applied to β regression coefficients (µV per epoch) of GRMS for the left upper trapezius No statistically significant differences were found between the groups.

Table 7.30 Mean β regression coefficients of GRMS (µV per epoch) for the left upper trapezius.

	Mean β regression coefficients of GRMS (std)
HC (N=17)	1.40 (2.42)
NTNP (N=13)	1.41 (2.62)
WAD (N=3)	-0.025 (0.12)

Table 7.30 shows that the HC and NTNP groups have similar β regression coefficients. The WAD group showed a very slight negative value.
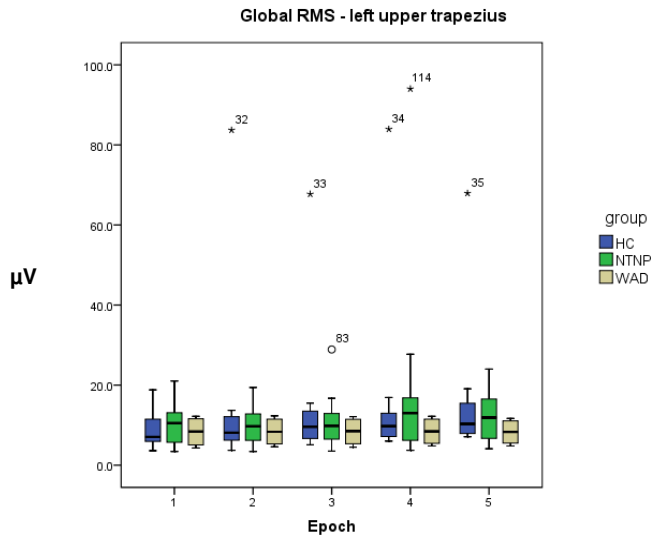
Figure 7.18 Distribution of GRMS values for the left upper trapezius.

There were no significant differences between the groups over the 5 epochs (Table 7.31). A slight increase in the distribution of data in the NTNP group as compared to the HC and WAD groups can be seen in Figure 7.18. All groups showed very similar median values.

Table 7.31 Repeated measures ANOVA between groups (with BMI as a covariate) for the left upper trapezius

Repeated measures ANOVA - LUTRP	
	Test of between subjects effects (p value)
GRMS	0.729

The test of between subjects' effects with BMI as a covariate showed no statistically significant difference in GRMS when comparing the 3 groups, as seen in Table 7.31.

7.3.3   Right Sternocleidomastoid

The Kruskal-Wallis test was applied to β regression coefficients of GRMS and GMDF for the right sternocleidomastoid, no statistically significant differences were found between the groups.

Table 7.32 Mean β regression coefficients of GRMS (µV per epoch) for the right sternocleidomastoid.

	Mean β regression coefficients of GRMS (std)
HC (N=17)	8.89 (7.17)
NTNP (N=13)	15.11 (13.68)
WAD (N=3)	10.39 (7.49)

Table 7.32 shows the NTNP group have the more positive β mean regression coefficient and the HC group have the least positive mean β regression coefficient.
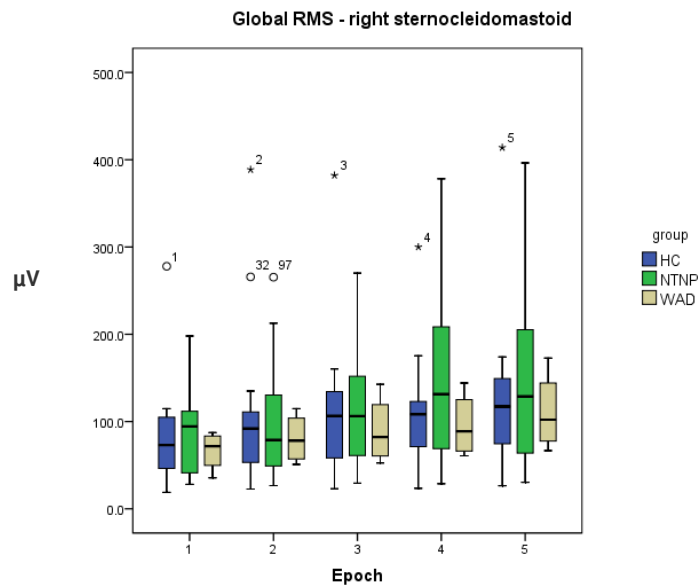
Figure 7.19 Distribution of GRMS values for the right sternocleidomastoid.

There were no statistically significant differences between the groups over the 5 epochs (Table 7.33). Figure 7.19 shows the NTNP group displays a trend for the greatest distribution and the WAD group shows a trend for the smallest distribution of data over the 5 epochs.

Table 7.33 Mean β regression coefficients of GMDF (Hz per epoch) for the right sternocleidomastoid.

	Mean β regression coefficients of GMDF (std)
HC (N=17)	-5.03 (2.15)
NTNP (N=13)	-5.33 (4.30)
WAD (N=3)	-4.33 (1.80)

Table 7.33 shows similar negative β regression coefficient of GMDF for all groups.
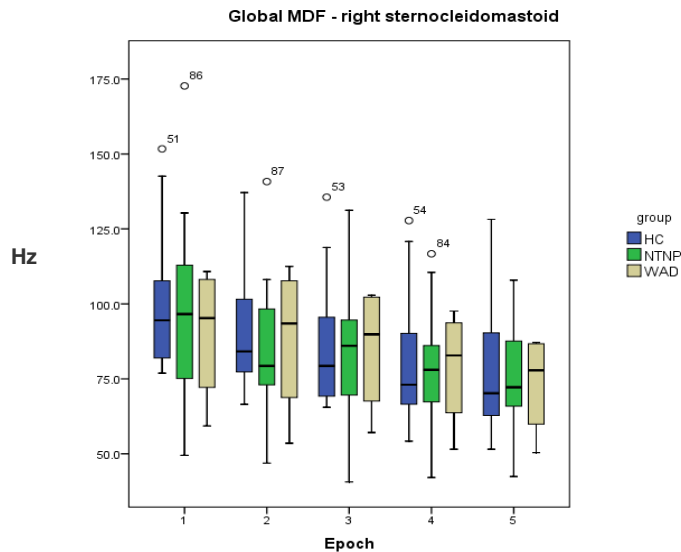
Figure 7.20 Distribution of GMDF values for the right sternocleidomastoid.

There were no statistically significant differences in GMDF between the groups over the 5 epochs (Table 7.34). Figure 7.20 shows a trend of higher median values for the WAD group in epochs 2 to 4.

Table 7.34 Repeated measures ANOVA between groups (with BMI as a covariate) for the right sternocleidomastoid

Repeated measures ANOVA - RSCM	
	Test of between subjects effects (p value)
GRMS	0.437
GMDF	0.952

The test of between subjects' effects with BMI as a covariate showed no statistically significant difference in GRMS and GMDF when comparing the 3 groups as shown in Table 7.34.

7.3.4   Summary of right sternocleidomastoid

There were no significant differences in the activity of the right sternocleidomastoid muscle between the groups (Table 7.34). All groups showed a trend for an increase in activity over the 5 epochs. The WAD group showed slightly lower GRMS values that the HC and NTNP groups. All groups showed similar slopes of fatigue.

7.3.5   Left Sternocleidomastoid

The Kruskal-Wallis test was applied to β regression coefficients of GRMS and GMDF for the left sternocleidomastoid. No statistically significant differences were found between the groups.

Table 7.35 Mean β regression coefficients of GRMS (µV per epoch) for the left sternocleidomastoid.

	Mean β regression coefficients of GRMS (std)
HC (N=17)	10.02 (8.41)
NTNP (N=13)	16.51 (17.03)
WAD (N=3)	11.33 (7.50)

It can be seen in Table 7.35 that although not significantly different, the NTNP group show the largest regression coefficient of the 3 groups.
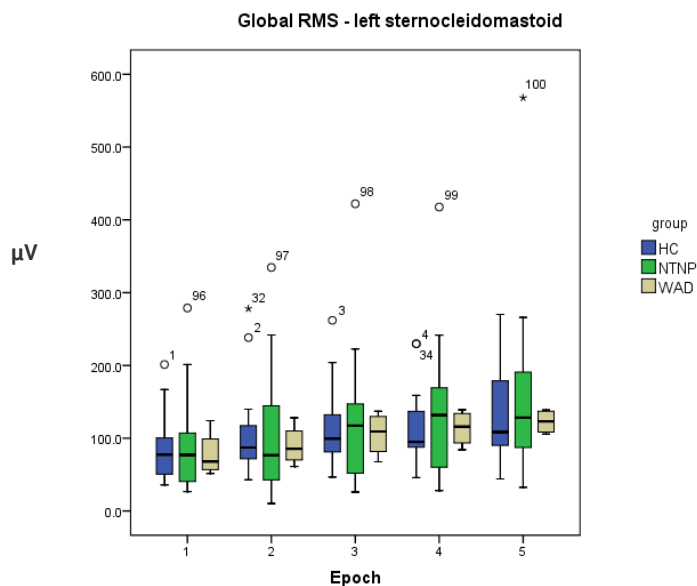
Figure 7.21 Distribution of GRMS values for the left sternocleidomastoid.

There are no statistically significant differences in GRMS between the groups (Table 7.36). Figure 7.21 shows that over the 5 epochs the WAD group has the smallest distribution of data and the NTNP group has the largest.

Table 7.36 Mean β regression coefficients of GMDF (Hz per epoch) for the left sternocleidomastoid

	Mean β regression coefficients of GMDF (std)
HC (N=17)	-4.14 (2.37)
NTNP (N=13)	-5.04 (4.80)
WAD (N=3)	-2.95 (2.93)

It can be seen in Table 7.36 that the WAD group show the least negative β regression coefficient of the 3 groups.
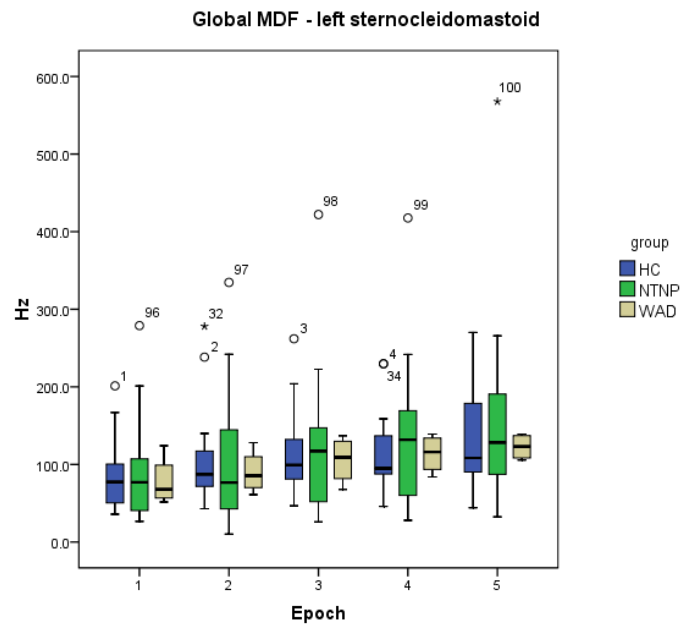
Figure 7.22 Distribution of GMDF values for the left sternocleidomastoid.

There are no statistically significant differences in GMDF between the groups (Table 7.37). The NTNP group shows the greatest distribution of data and the WAD group shows the smallest distribution of data over the 5 epochs, as seen in Figure 7.22.

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Table 7.37 Repeated measures ANOVA between groups (with BMI as a covariate) for the left sternocleidomastoid

Repeated measures ANOVA - LSCM	
	Test of between subjects effects (p value)
GRMS	0.506
GMDF	0.977

In Table 7.37 The test of between subjects' effects with BMI as a covariate showed no statistically significant difference in GRMS and GMDF when comparing the 3 groups over the 5 epochs.

7.3.6   Summary of left sternocleidomastoid

The activity of the left sternocleidomastoid showed no significant differences between the groups (Table 7.37). The NTNP group had the greatest distribution of data for both parameters, with a more positive slope for GRMS and a more negative slope for GMDF than the HC and WAD groups.

7.3.7   Summary table of muscle synergy in the head raise activity

Table 7.38 A summary of muscle activation in the arm raise activity. The changes are based on the differences between epochs 1 and 5. Cross-referencing to the relevant results and discussion sections are indicated.

Muscle	Parameter	HC	NTNP	WAD	Results reference	discussion reference
RUTRP	GRMS	↑	↑	↔	7.19	8.1.4
LUTRP	GRMS	↑	↑	↔	7.20	8.1.4
RSCM	GRMS	↑↑	↑↑	↑↑	7.21	8.1.5
	GMDF	↓	↓	↓	7.22	8.1.5
LSCM	GRMS	↑↑	↑↑	↑↑	7.23	8.1.5
	GMDF	↓	↓	↓	7.24	8.1.5

Key to muscle activity over the 5 epochs: ↑↑large Increase, ↑ increase, ↓↓ large decrease ↓ decrease, ↔ No / very little change.

7.3.8   Side to side differences

Side to side differences were explored for all sEMG parameters in the upper trapezii and sternocleidomastoid muscles during the head raise activity. No trends or significant differences were seen.

7.3.9   Correlations between subjective measures of pain and fatigue with objective measures of muscle activity

During the head raise activity subjective scores of pain and fatigue were given every 10 seconds. Individual scores were correlated with corresponding GRMS and GMDF values. The number of individual significant correlations are expressed as both numbers and percentages within each group. Groups that show more than 50% of significant correlations are highlighted

Table 7.39 Number of subjects and percentage per group of significant positive (p<0.05) Spearman's correlations of individual pain and fatigue scores with GRMS values.

		RUTRP GRMS number of correlations (%)	LUTRP GRMS number of correlations (%)	RSCM GRMS number of correlations (%)	LSCM GRMS number of correlations (%)
Pain	HC (N=16)	6 (38)	5 (31)	9 (56)	6 (38)
	NTNP (N=13)	6 (46)	8 (62)	12 (92)	10 (77)
	WAD (N=4)	0 (0)	1 (25)	3 (75)	3 (75)
Fatigue	HC (N=16)	6 (38)	6 (38)	9 (56)	9 (56)
	NTNP (N=13)	7 (54)	7 (54)	12 (92)	9 (69)
	WAD (N=4)	1 (25)	0 (0)	2 (50)	3 (75)

An increase in pain and fatigue scores with an increase in GRMS values result in positive correlations. As seen in table 7.39. the NTNP group showed the highest number of significant positive correlations for pain and fatigue in all muscles with the exception the LSCM and fatigue.

Table 7.40 Number of subjects and percentage per group of significant positive (p<0.05) Spearman's correlations of individual pain and fatigue scores with GMDF values.

		RSCM GMDF number of correlations (%)	LSCM GMDF number of correlations (%)
Pain	HC (N=16)	7 (44)	6 (38)
	NTNP (N=13)	7 (58)	9 (69)
	WAD (N=4)	2 (50)	2 (50)
Fatigue	HC (N=16)	9 (56)	6 (38)
	NTNP (N=13)	8 (66)	10 (83)
	WAD (N=4)	2 (50)	2 (50)

Increasing pain and fatigue scores with decreasing GMDF values result in negative correlations. As shown in Table 7.40 the NTNP group showed the highest number of significant negative correlations and the HC group showed the lowest number of significant correlations for all parameters.

Table 7.41 Number of subjects and percentage per group of significant positive (p<0.05) Spearman's correlations of individual pain and fatigue scores

	HC (N=16) number of correlations (%)	NTNP (N=13) number of correlations (%)	WAD (N=4) number of correlations (%)
Pain v Fatigue	15 (94)	13 (100)	3 (75)

Table 7.41 shows that all subjects in the NTNP group showed significant positive correlations of pain and fatigue scores. The WAD group showed the least percentage of significant correlations.

There were no identifiable patterns or trends in participants with side specific pain.

Correlations were undertaken for each muscle of pre-test and NDI scores with mean β regression coefficients for GRMS and GMDF. There were no trends seen for the NTNP and WAD groups for any parameter.

Chapter 8 - Discussion

This study has examined the simultaneous activity of the upper trapezius and sternocleidomastoid muscles during non-forced fatiguing activities. Amplitude, as measured by global root mean square (GRMS) and motor unit action potential root mean square (MRMS) fatigue as measured by global median frequency (GMDF) and motor unit action potential median frequency (MMDF), motor unit action potential rate (MR) and synergies between flexor and extensor muscle activity have been compared in three groups: in chronic neck pain, of a non traumatic onset (NTNP) and from a whiplash injury (WAD) and a sample of healthy controls (HC).

This study has also compared the relationship between subjective pain, fatigue and sEMG parameters during non-forced fatiguing activities across the same groups.

8.1   Summary of main findings

In the arm raise activity the HC and NTNP group showed a similar increase in upper trapezius activity (GRMS and MRMS) with a similar increase in MR (see results sections 7.2.1 & 7.2.3). The WAD group demonstrated trends for lower values and a reduced increase in GRMS activity. Significant differences in MRMS activity with slightly higher values and a greater increase in MR for the WAD group were seen in the right upper trapezius. Similar, but non-significant, trends were seen in the left upper trapezius. GMDF and MMDF results were similar for all groups.

The NTNP group showed slightly higher mean values in the sternocleidomastoid muscles although these values were quite similar to the HC group (see results sections 7.2.5 & 7.2.7). The WAD group showed significantly lower values and a lower increase in GRMS and MRMS values in the right sternocleidomastoid muscle. Similar but non-significant trends were seen in the left sternocleidomastoid muscle. The MR values for all groups were similar for the right sternocleidomastoid muscle, the HC group showed a greater increase in the left sternocleidomastoid muscle as compared to the NTNP and WAD groups.

There were no significant differences in upper trapezius activity between the groups in the results of the head raise activity (see results sections 7.3.1 & 7.3.2). Identified trends showed the WAD group produced lower values with a lower increase in GRMS values in the right upper trapezius. A reduced increase in GRMS values was also seen in the WAD group for the left upper trapezius, although the values were quite similar for all groups. The NTNP group showed the greatest increase in GRMS values in both sternocleidomastoid muscles (see results sections 7.3.3 & 7.3.5). The GMDF values were similar for all groups in the sternocleidomastoid muscles.

In the arm raise activity, the NTNP group showed the greatest number of individual significant correlations between subjective pain and fatigue ratings with GRMS, the WAD group showed the least number (see results section 7.2.11). No group achieved more than 50% individual significant correlations between pain and fatigue ratings with GMDF (see results section 7.2.11). All groups achieved 100% individual significant correlations between subjective pain and fatigue ratings (see results section 7.2.11).

In the head raise activity the NTNP group showed the highest number of individual significant correlations with only the right upper trapezius and GRMS not achieving more than 50% significant individual correlations (see results section 7.3.9).

8.2   Discussion of sEMG results

Myoelectric manifestations of fatigue have been established as a decrease in GMDF and an increase in GRMS (Merletti et al., 1990). This has been reported in the upper trapezius muscles and the sternocleidomastoid muscles (Falla et al., 2002a; Madeleine et al., 2002) (for further detail on fatigue see sections 2.2.5 & 2.9.9). Differences in muscle fatigue between individuals with chronic neck pain and healthy controls have been reported in the upper trapezii and the sternocleidomastoid muscles (Falla et al., 2004a; Falla et al., 2004c; Falla et al., 2003b; Johnston et al.,2008b; Jull et al., 2004; Kallenberg et al., 2007; Nederhand et al., 2002; Nederhand et al., 2000; Nederhand et al., 2003).

A technique that observes the activity of specific motor units has recently been developed. This is a novel area of research that observes both motor control and the effects of fatigue on selected motor units. There are few published studies in this specific area of research, the most pertinent have been reported by Kallenberg and Hermens, 2006; Kallenberg et al., 2007, and Kallenberg and Hermens, 2008. As such the results from the present study using this technique are mainly compared with these papers.

This section discusses the sEMG findings of the arm raise activity followed by the head raise activity. A discussion of muscle activation, including detail of each recorded parameter for both the upper trapezius and sternocleidomastoid muscles is presented. This is followed by an overview of muscle synergy for each activity.

8.2.1   Arm raise activity – upper trapezius muscles

Table 7.24 provides a summary of the results that are discussed in the following sections.

GRMS increased during the exercise for all groups in both upper trapezius muscles (see results sections 7.2.1 & 7.2.3). This could reflect increased firing or recruitment of MUs. The HC and NTNP groups showed a trend for higher GRMS values than the WAD group. These results could imply that the WAD group have a different proportion of fibre types in the muscles with more motor units consisting of type I fibres than the HC and NTNP group. Alternatively, the WAD group may have been less reliant on the upper trapezius muscles to maintain the arm raise position. There are many muscles involved in lateral arm raises including the deltoid muscles; the recording method used did not measure the involvement of other muscles. It could be that due to pain adaptation or fear avoidance (for details on models of pain see section 2.6.4) that the trapezii were used less and other muscles such as the deltoids were relied upon more to maintain the arm raise.

Different responses in muscle activation between chronic neck pain and control groups have been reported (Kallenberg et al., 2007). They found a reduced increase of GRMS during contractions in chronic neck pain subjects during a low-level

sustained contraction. Although low-level contractions were used, this was a forced activity (40 N for 15 minutes), as compared to the non-forced activity used in the present study. The different type of muscle activation may affect muscle fibre recruitment. The participants in the study by Kallenberg et al. were suffering from work-related pain whereas the present study did not define the cause of pain other than it was non-traumatic in origin or from a whiplash injury. Participants that are not part of a treatment group or recruited through hospitals or pain clinics may respond to pain differently when compared to those that are. An individual who is seeking treatment may not have as efficient pain coping strategies as those who do not seek treatment or support. They may suffer from greater pain intensity, or either be more affected by the pain. An individual's perception and response to pain may affect the way they employ their muscles, and therefore affect how those muscles fatigue. The different aspects of coping with chronic pain are reflected in the avoidance-endurance model (Hasenbring, 2000) (for the avoidance endurance model of pain see section 2.6.4.iv).

In agreement to the present study, Nederhand et al. (2003) reported decreased sEMG amplitude in WAD participants during isometric arm raises compared to the control group. The reduction in amplitude was reported to be inversely related to the level of pain. The trend in the present study (see sections 7.2.1 & 7.2.3) alongside the reported decreased sEMG amplitude in WAD patients by Nederhand et al. implies that either these muscles are recruited differently by this chronic pain group or that the morphology of the muscles has changed as a result of altered use (for details on muscle morphology see section 2.2.2, for motor control strategies see section 2.8).

During a functional upper limb task a trend for increased GRMS in the left upper trapezius muscle was seen in individuals with chronic pain (from a whiplash injury and of an insidious onset) compared to the control group (Falla et al., 2004a). The whiplash group showed a trend for higher values than the insidious onset group. These findings are contrary to the findings of the present study. Falla et al. also showed that the reverse was seen in the right upper trapezius, which are in agreement with the findings of the present study. The functional upper limb task was not fatiguing as the activity in the present study, and this is likely to affect the

recruitment of MUs. However, it suggests that differences occur at different activation levels and during different tasks or activities between individuals with chronic neck pain and healthy controls

Significant differences were seen between the groups over the 5 epochs for MRMS in the right upper trapezius, with the WAD group having lower values than the HC and the NTNP groups (see sections 7.2.1 & 7.2.3). A trend for lower MRMS values in the left upper trapezius was also seen in the WAD group. The lower MRMS values and the less positive β regression coefficients seen in the WAD group are in partial agreement with Kallenberg et al. (2007) who reported a reduced increase in MRMS for chronic pain cases during low-level, forced fatiguing contractions. The lower values seen in the present study for the WAD group were not seen in the NTNP group, which implies that it is not simply chronic pain that causes altered muscle activation. Methodology is a possible reason for the different outcomes of these studies. MUs are recruited to perform specific tasks and the MRMS values are taken from MUs situated directly under the electrodes. Therefore these results are only reflective of the superficial portion of the muscle, under the array electrodes as opposed to the whole muscle. Different values may reflect the different proportions of motor units and therefore fibre types recruited within the muscle. Smaller MRMS values suggest smaller motor units indicating more MUs under the electrodes may contain type I fibres. Equally MU recruitment is task dependent, therefore the myoelectric signals may differ with the type of task performed. The ways in which an individual uses their muscles may also differ, some may avoid the use of painful muscles or endure the pain as proposed by the avoidance-endurance model (Hasenbring, 2000).

In agreement with the findings of the present study, a reduction in MRMS as compared to GRMS in the upper trapezius has been reported in both healthy controls and chronic neck pain participants (Kallenberg & Hermens, 2006). GRMS has a larger inter-electrode distance and records more MUAPs than MRMS. In addition with field potentials and cross-talk removed, MRMS values should show reduced activity compared to GRMS. The larger difference between these values observed in the WAD group in the present study could be due the MUs under the

electrodes being smaller or less active, being further away from the electrodes (higher BMI), having fewer rest gaps or a combination of these factors.

Conversely, a trend for higher MRMS values was identified in chronic pain subjects as compared to healthy controls while performing computer related tasks (Kallenberg & Hermens, 2006). However, the participants had work-related pain and the task was unilateral and of a less sustained and less active nature, which makes comparisons with the present study difficult. The higher MRMS values could be due to the recruitment of MUs required to perform more fine and controlled movements used in Kallenberg and Hermen's study as opposed to the task in the present study, which required a greater and more sustained use of the upper trapezius muscles.

The present study reported a significant difference in the left upper trapezius for higher MR values in the WAD group as compared with the HC and NTNP groups. A similar trend was seen in the right upper trapezius (see section 7.2.3). MUAP rate, which is the mathematical product of the number of MUs and the rate of firing, has been shown to increase, along with RMS, with increasing force during a step contraction in the biceps brachii (Kallenberg and Hermens, 2008). However, it was proposed that the MR is a more sensitive measure than the GRMS. This is because MR is a reflection of CNS output properties and GRMS is reflective of MU size. Therefore, the MR has been proposed as a more suitable as a measure of low-level contractions as opposed to high force contractions.

Chronic neck pain participants have demonstrated higher MR in the upper trapezii as compared to controls (Kallenberg & Hermens, 2006), which is in partial agreement with the findings of the present study. In the present study, the higher MR was only seen in the WAD group and not the NTNP group (see sections 7.2.1 & 7.2.3). This conflict in findings could be due to the work-related activity used by Kallenberg and Hermens (2006), rather than to the point of perceived fatigue. This may influence initial MU recruitment and recruitment over time. It could also be a result of different proportions of motor unit types in the muscles of the groups.

The use of linear array electrodes enabled the detection of more parameters than is possible using traditional bipolar electrodes (see sections 2.9.3.ii & 2.9.9.i for detail on linear array electrodes). This system allowed the identification of innervation

zones and the orientation of muscle fibres, to optimise the quality of the sEMG signals.

The alteration of motor control strategies have been shown by increased upper trapezius activity during elbow flexion, as a response to experimentally induced pain in the biceps brachii (Schulte et al., 2004). The present study did not analyse the origin of pain so it was not possible to ascertain whether muscle activity was directly related to areas of muscle pain or to avoid the use of painful muscles (see section 2.8 for further detail on motor control strategies). Experimental pain is a reflection of acute rather than chronic pain, although mechanisms in certain aspects of acute pain may be seen as an indicator of chronic pain such as fear avoidance. However, a benefit of using experimental pain is that the location of the pain is known.

The effects of pain on the sEMG of upper trapezii have been observed using experimental pain in the form of hypertonic saline injections (Falla et al., 2007). Decreased activity in painful muscles and compensatory reorganisation of motor output in the upper and lower trapezius was reported. In a comparison of chronic neck pain and acute neck pain (experimentally induced) similar alterations in muscle activity were seen in the performance of work related tasks (Madeleine et al., 1999). However, experimental pain cannot reflect the changes brought about by modulation of the nervous system over time. Therefore direct comparison between responses to experimental pain and chronic pain participants in the present study is not possible.

The difference in muscle activity seen between groups in the present study could be due to habituated reorganisation of the subdivisions of the upper trapezius in the WAD group as a result of fear avoidance (for subdivisions of the upper trapezius see section 2.1). The results of the NTNP group could reflect endurance in the use of painful muscles (see section 2.6.4 for further information on models of pain). During a sustained contraction of the upper trapezius it was found that MR was related to the muscles' subdivisions within the UTRP (Falla & Farina, 2008). Increased recruitment occurred in the upper, cranial region over time while a decrease in MR was observed in the lower, caudal region. It is possible that change

to the motor neurone pool can occur as a result of pain and this may impact of the MR of the UTRPs of individuals with chronic pain.

A possible conclusion to the RMS and MR results reported in the present study is that the WAD group have altered the use of the upper trapezius muscles to adopt a more postural role to avoid the use of painful deeper structures. Fibre type transformations resulting in more MUs containing type I fibres, therefore making the muscle more fatigue resistant, would be reflective of such an increase in use. This would also explain the increased MR, as an increased firing rate of MUs containing type I fibres would be required to maintain the position. Larger MUs containing type II fibres would be recruited in the HC and NTNP groups for this purpose, causing the trend for a greater increase in RMS parameters seen in these groups.

The fatiguing nature of the arm raise activity is reflected by the decreasing GMDF results. GMDF values and β regression coefficients were similar for all groups in both trapezius muscles, with the WAD group showing a slightly reduced slope of fatigue compared to the HC and NTNP groups (see sections 7.2.1 & 7.2.3). However, when looking at the results of the study presented by Kallenberg et al. (2007) individuals with chronic neck pain showed constant or slightly increased GMDF values during low-level forced contractions as compared to healthy controls. This difference in findings could be due to the fatiguing contractions of the present study design being non-forced and being maintained until the participants could no longer hold the required position. This may well explain the slightly greater level of fatigue seen using the present study design.

The β regression coefficients of MMDF in the present study were negative (see sections 7.2.1 & 7.2.3). This indicates increasing muscle fatigue, with the exception of a slight positive MMDF value for the WAD group in the left upper trapezius muscle. The results of the present study show that the data collected from the right upper trapezius corresponds to those seen in chronic neck pain participants during low level forced contractions (Kallenberg et al., 2007). However this was not the case for the left upper trapezius. Although there were no statistically significant side to side differences, it is possible that side dominance did have some effect.

It is possible that median values may not discriminate between muscles of differing fibre type proportions as accurately as mean values due to the large amount of outlying data points seen in some muscles. This may account for the similar responses seen in all groups. With the natural variation seen between individuals performing the activities in the present study it is difficult to determine if the outlying data points were due to recording problems such as electrical noise or were due to a large variation of normal responses. The software used allowed significant noise interference to be identified during analysis. Therefore it is more likely that the outlying data was the result of normal muscle activity and that the mean values would have been more appropriate.

MMDF values were higher than the GMDF values in the present study (see sections 7.2.1 & 7.2.3). The same relationship between global and MUAP parameters has been shown for MDF (Kallenberg and Hermens, 2008). The frequency content of the signals from the MUs directly under the electrodes would be expected to be higher than those further away, because the tissues that separate the electrodes and the muscle fibres have a low-pass filtering effect (Merletti & Parker 2004). Signals emanating from sources at a larger distance from the electrodes, such as cross talk and field potentials, are included in the global parameters, whereas the MUAP parameters are mostly related to signals originating from superficial MUs.

8.2.2   Arm raise activity – sternocleidomastoid muscles

The sternocleidomastoid muscles were not directly involved in lateral arm raises but showed an increase in recruitment over the 5 epochs for all groups. This could be due to their recruitment to aid maintenance of the arm raise position.

The present study reported significant differences of GRMS values over the 5 epochs in the right sternocleidomastoid muscle between the groups (see sections 7.2.5 & 7.2.7). The highest values were seen in the NTNP and the lowest values were seen in the WAD group. The WAD group also showed the least positive mean β regression coefficient. No significant differences were seen in the left sternocleidomastoid, but the WAD group showed a slight trend for lower GRMS values. However, the WAD group also showed the most positive mean β regression

coefficient of the 3 groups in the left sternocleidomastoid, reflecting the greater increase in values over the 5 epochs of the 3 groups. Despite the different results for GRMS values in the left and right muscles, there were no side to side differences in the mean β regression coefficients. The β regression coefficient is a summary measure. By summarising data, differences in the way that muscle activation can change during the course of an activity may be lost.

High numbers of outlying data points, especially in the right sternocleidomastoid, indicate there was a lot of individual variation in the way muscles were recruited. This is an important factor when considering the outcomes of the present study. Although there may be differences between groups, there are also differences between individuals within a particular group. This is an important clinical consideration; individuals may show motor control strategies that are different from the norm, yet they are normal for that individual. This may prove problematic when trying to establish whether an individual's motor control strategy has been affected by pain.

Studies have observed direct activation of the sternocleidomastoid muscles in cervical flexion and have observed increased activity in chronic pain participants compared to healthy controls (Falla et al., 2004a; Falla et al., 2003b). However, few have looked at their synergy with upper trapezius activity. One study observed superficial muscle synergy during functional and stressful tasks. Females with work-related chronic neck pain showed higher sternocleidomastoid synergy than the workers without chronic pain and a non-worker control group during a unilateral keyboard task (Johnston et al., 2008b). A similar study which compared the same participant groups during standard typing tasks and typing tasks with mental stressors also reported higher sternocleidomastoid activation in workers, with and without pain, compared with controls (Johnston et al., 2008a). This is in agreement with the findings of the present study for the NTNP group but not the WAD group. It is possible that an increased reliance on upper trapezius muscles in protraction of the neck may result in a reduction in use of the sternocleidomastoid muscles, as seen in the WAD group.

The results of the studies by Johnston et al. (2008a, 2008b) could imply that the type of work undertaken caused muscle alteration, which could lead to the development of pain. However, some workers in this study did not have chronic pain, which could suggest that it is not simply the work that causes pain. It is possible that posture may be involved in the differences seen in pain development. How an individual responds to the discomfort of work may also be a factor. The designs of these studies were quite different to the present study and did not involve isometric or fatiguing contractions, which may be why the results differ.

In the present study significant differences were also seen between the groups for MRMS values in the right sternocleidomastoid (see sections 7.2.5 & 7.2.7).The WAD group had a slightly less positive mean β regression coefficient and less outlying data points that the HC and NTNP groups. There were no significant differences or trends seen in the left sternocleidomastoid between the groups (see sections 7.2.5 & 7.2.7). Contrary to the upper trapezii, the GRMS values were lower than the MRMS in the NTNP group. This could indicate that there were more rest gaps and larger MUAPs in the GRMS signal. Larger MUAPs could be the result of a higher number or increased recruitment of type II fibres in the muscles of this group, possibly due to fatigued type I fibres.

The difference between the right and left sternocleidomastoid muscles may be due to side dominance, the participants were either right handed or stated that they were only left hand dominant for writing. The increased use of the right side might have increased the fatigue resistant nature of the muscles on that side. Also increased use of superficial muscles may be seen in individuals with chronic pain in pain avoidance (see section 2.6.4 for further detail on models of pain).

MR increased in all three groups over the 5 epochs for both muscles which suggests increased MU recruitment and/or firing. In the right sternocleidomastoid MR values were highest for the NTNP group and lowest for the WAD group. In the left sternocleidomastoid the MR values were similar for the HC and NTNP group and lower for the WAD group which reflects the GRMS activity seen in these muscles.

Increasing sternocleidomastoid GRMS and MRMS over time could indicate that these muscles, possibly along with others, were recruited to provide extra support when the upper trapezius muscles began to fatigue. The increasing GRMS and MR could suggest that all the groups have similar muscle morphology. However, it was not possible to ascertain a direct association between MR and GRMS in the present study.

Increased reliance on extensor muscles may reduce reliance on flexor muscles. However, without recording from all muscles involved in the arm raise activity it is difficult to draw firm conclusions regarding adaptations of motor control strategies and synergy seen in chronic pain.

8.2.3   Summary of muscle synergy during the arm raise activity

The results of the present study show some statistically significant differences between the groups in activation and synergy of superficial neck flexor and extensor muscles during the arm raise activity. The WAD group showed significantly more CNS input, as measured by MR, in the upper trapezius muscles and less in the sternocleidomastoid muscles than the HC and NTNP groups. The WAD group also showed a trend for decreased GRMS and MRMS in the upper trapezius and left sternocleidomastoid muscles, alongside a statistically significant decrease in both RMS values for the right sternocleidomastoid muscle. The HC and NTNP groups showed quite similar responses in both pairs of muscles. This could suggest that the WAD group may increase the postural use of the upper trapezii to minimise the use of deeper more painful muscles and tissues, which in turn could lead to altered morphology of these muscles. This response could have been a result of the trauma or from the resulting pain. The increased reliance on the upper trapezius muscles could also result in protraction of the neck and explain the decreased use of sternocleidomastoid muscles seen in the WAD group.

It is also feasible that the NTNP group had developed better coping strategies, which could have been due to the lack of trauma involved. The larger increase in activity of the sternocleidomastoid muscles seen in this group could indicate dynamic reorganisation of superficial muscles to minimise pain or fatigue (see section 8.6 for future studies).

8.2.4   Head raise activity – upper trapezius muscles

Table 7.38 provides a summary of the results that are discussed in the following sections.

The upper trapezii were not primarily involved in lifting the head from a supine position and there is sparse literature regarding their synergy with flexor muscles during neck flexion (Falla et al., 2004a; Johnston et al., 2008a; Johnston et al., 2008b). In the present study, the WAD group showed a trend for lower GRMS values in both upper trapezii compared to the HC and NTNP groups (see sections 7.3.1 & 7.3.2). This could indicate that, unlike the HC and NTNP groups, the WAD group did not activate the upper trapezius muscles as much to stabilise the head raise position during the exercise. It is possible that if these muscles are painful, motor control strategies may be altered to avoid their use. It is also possible that these muscles have a higher proportion of smaller MUs, containing type I fibres compared to the other groups as suggested in the discussion of the arm raise results. This could also result in reduced amplitude in the sEMG signals. The HC group showed the most positive β regression coefficient of the upper trapezii.

Differences were observed in GRMS of the upper trapezii between whiplash, insidious neck pain and control participants while performing a timed task (where dots were drawn, using a pencil, in 3 circles in an anticlockwise direction) (Falla et al., 2004). A pattern of increased GRMS was reported by Falla et al. where the greatest amplitude was seen in the left upper trapezius muscle for the whiplash group, with the lowest values recorded for the control group. A reverse effect was apparent for the right upper trapezius muscle. Due to the different nature of the tasks it is not possible to draw firm comparisons with the present study, but similar decreased activity for the WAD group in the right upper trapezius in both studies were observed.

During the head raise activity the signal to noise ratio was poor, due to the low levels of activity in these muscles. This resulted in the inability to detect MUAP variables. Without recording MR it is not possible to ascertain the fibre types contained in the muscle.

The increasing GRMS values in the upper trapezius muscles over time seen in the HC and NTNP groups suggest these muscles were recruited when the flexor muscles became increasingly fatigued, possibly to stabilize the neck and help maintain the head raise position. Although not significantly different to the HC and NTNP groups, the WAD group did not show this pattern of increasing amplitude over time. The reduced increase in GRMS values seen in the WAD group could be due to different fibre type proportions in these muscles. As previously discussed, the results from the arm raise activity showed that in the WAD group the upper trapezius muscles had reduced amplitude and slightly reduced CNS input, indicating a change in fibre type proportions. This could also apply to the results of the head raise activity. However, without MR values the results could also indicate decreased recruitment of the muscle as a whole in the WAD group, suggesting that upper trapezius muscles may be more involved in maintaining the head raise position in the HC and NTNP groups.

The response of the upper trapezii synergy in the head raise activity was similar to that seen in the sternocleidomastoid muscles during the arm raise activity, where the synergistic muscles displayed trends for higher GRMS values in the HC and NTNP groups than in the WAD group.

8.2.5   Sternocleidomastoid muscles – head raise activity

The present study reported that GRMS values increased in both sternocleidomastoid muscles with no statistically significant differences seen over time between the groups (see sections 7.3.3 & 7.3.5). The NTNP group had a trend for the highest GRMS values and the WAD group showed a trend for the lowest values. Contrary to these findings insidious onset neck pain and WAD participants showed increased GRMS compared to controls during a cranio-cervical flexion test (Jull et al., 2004). However, the exercise employed consisted of forced, short duration contractions and could have resulted in a different MU recruitment.

The use of linear array electrodes for measuring sEMG in the present study was problematic. The flexion movement caused the skin to move over the muscle and

the ideal electrode position was subsequently lost. A different activity with reduced levels of movement could be developed to enable optimal use of the array electrodes. A task with less cervical movement and therefore improved electrode positioning, would enable better quality data to be collected. This may provide a clearer picture of the muscle activity and fibre content.

GMDF decreased similarly in all groups in the right and left sternocleidomastoid, indicating that this activity was fatiguing (see sections 7.3.3 & 7.3.5). Fatigue in the sternocleidomastoid muscles has been shown to be greater in neck pain participants as compared to controls (Falla et al., 2003b). Also greater fatigability has been shown in the painful side of neck pain patients (Falla et al., 2004).The use of short-duration, forced contractions in these studies may account for the difference with the results of the present study. In addition, the studies by Falla et al. used mean frequency values whereas the present study used median frequency, which may also provide an explanation for the differences between these results and those of the present study.

8.2.6   Summary of muscle synergy during the head raise activity

All groups showed similar GRMS and GMDF values for both sternocleidomastoid muscles. The NTNP group showed the highest GRMS values and the WAD group showed the lowest values. However, there were no significant differences seen. The upper trapezii showed a trend for increased GRMS activity in the HC and NTNP groups, possibly to help stabilise the position as other muscles begin to fatigue. The WAD group clearly showed lower upper trapezii values, which could be due to altered proportions of fibre types.

The pattern of muscle activation in the head raise activity was similar to that seen in the arm raise activity, where the synergistic muscles were more active in the HC and NTNP group than in the WAD group. It must be considered when interpreting these data that, as seen in the arm raise exercise, all groups showed a large range of individual variation in the use of these muscles.

8.2.7 Summary of muscle synergy during the activities

Reorganisation of dynamic muscle activation in response to pain could explain the slight increase in use of synergistic muscles in the NTNP group. The upper trapezii in the arm raise activity and the sternocleidomastoid muscles in the head raise activity responded similarly in the HC and NTNP groups. However, the NTNP group showed a trend for increased use of the synergistic muscles as compared to the HC group. This could be an endurance response whereby the group use muscles despite pain and increase the dynamic use of the synergistic muscles as pain increases. The WAD showed the least activity in the non-dominant muscles.

8.3 Subjective scores

The following sections will discuss the relationship between subjective pain and fatigue scores with GRMS and GMDF. In addition, the relationship between subjective pain and fatigue for both activities will be assessed. For each activity, individual subject correlations were performed for pain and fatigue with GRMS and GMDF. Groups that displayed more that 50% significant correlations for any parameter were highlighted in the results section (see sections 7.2.11 & 7.3.9).

8.3.1 Pain scores with GRMS and GMDF

The upper trapezii were directly involved with the arm raise activity and showed greater numbers of significant correlations between pain and GRMS than the sternocleidomastoid muscles. The HC and NTNP showed more than 50% significant correlations for pain with GRMS in the upper trapezius muscles (see section 7.2.11). It is possible that the pain felt by the NTNP participants originated more from upper trapezii activity than in the other groups during the arm raise, hence the higher number of significant correlations. The pain felt by the WAD and HC group may have originated more from other areas, for example the HC group may have felt more generalised pain from a wider range of muscles and the WAD group may have felt pain from deeper structures that had been damaged or atrophied as a result of the injury (see section 2.2.3 on innervation of skeletal muscle).

During the arm raise activity the NTNP and WAD groups showed more than 50% individual significant correlations for pain with GRMS in the left sternocleidomastoid muscle. There were no significant correlations for pain with GRMS in the right sternocleidomastoid muscle. The HC group showed less than 50% individual significant correlations for both sternocleidomastoid muscles. These muscles were not directly involved in the arm raises and showed much less activity during the exercises than the trapezii muscles. The low number of significant correlations for the HC group suggests that the recruitment and activity of the sternocleidomastoid muscles was not directly involved in the participants' perception of pain.

The NTNP and WAD groups showed more than 50% significant correlations in the left sternocleidomastoid muscle but not in the right, which may be due to side dominance. The right sternocleidomastoid muscle may be activated more at low levels than the left due to the increased use of the right side, e.g. for writing. This could result in the right side musculature being more fatigue resistant and less likely to be a source of pain.

There were no significant correlations for pain with GMDF in the upper trapezii during the arm raise activity (see section 7.2.11). This implies that change in median frequency of the MUs during fatigue was not related to the pain felt by the participants during the exercise.

A tendency for individuals with higher self reported pain to exhibit higher sEMG activity in the upper trapezius, as compared to control subjects, has been reported (Bansevicius et al., 1997). However, this study recorded activity during stress tests as opposed to isometric contractions in pain free participants. Self reported pain has also been shown to correlate with reduced range of motion in individuals with chronic pain as compared to controls (Johnston et al., 2008b). Although different methods are employed in these studies, pain does appear to be related to muscle activity and this is reflected by the results of the present study.

Although similarities in pain and sEMG responses were shown separately in fibromyalgia and chronic shoulder/neck pain patients, a lack of correlation between these measures during a stress-related computer task was reported (Nilsen et al.,

2006). A similar method of recording pain to the present study was employed, whereby measures were taken at regular intervals during the activity. The difference in results could be due to the different activity, upper trapezius activation would be far less in a computer task than during fatiguing isometric arm raises.

In the head raise activity the sternocleidomastoid muscles were directly recruited to maintain the position. The NTNP and WAD groups showed more than 50% significant correlations in both sternocleidomastoid muscles (see section 7.3.9) with the NTNP group showing the highest number of significant individual correlations. The HC group showed more than 50% significant correlations in the right sternocleidomastoid muscle but not the left. The participants expressed that this activity was more difficult and more uncomfortable to perform than the arm raise activity. With the exception of the left sternocleidomastoid muscle in the HC group the increasing activity of the sternocleidomastoid muscles correlates with the majority of participants increasing perception of pain.

The NTNP group was the only group to show more than 50% individual significant correlations with pain and GRMS in the upper trapezii during the head raise activity, this was in the left muscle (see section 7.3.9). The lack of significant correlations for the trapezii and pain during the head raise exercise indicates the activity of these muscles was not directly related to the pain felt by the participants.

The NTNP group showed more than 50% individual significant correlations with GMDF and pain in both sternocleidomastoid muscles during the head raise activity. The HC and WAD group showed less than 50% for pain and GMDF for both sternocleidomastoid muscles (see section 7.3.9). Although more than 50%, the number of individual significant correlations for pain and MDF were not as high as seen for other parameters. For both the arm and head raise exercise the MDF values did not correlate well with perceived pain scores. This shows that changes in median frequency in upper trapezii and sternocleidomastoid muscles did not directly relate to the pain experienced during these fatiguing exercises.

The results of the present study indicate that GRMS was a more reflective measure of an individuals' perception of pain than GMDF. The muscles directly involved in

the activities, e.g. upper trapezius in the arm raise, also demonstrate more consistent correlations than the muscles not directly involved. The NTNP group showed the most consistently high correlations with pain and RMS in the upper trapezii and sternocleidomastoid during the arm raise and head raise activities respectively.   This suggests that the pain experienced by these participants originates more from the activated muscles than in the HC and WAD groups.

8.3.2   Fatigue scores with GRMS and GMDF

In the arm raise activity the HC and NTNP groups showed more than 50% significant individual correlations for fatigue with GRMS in the upper trapezius muscles (see section 7.2.11).   The WAD group had 50% significant individual correlations in the left upper trapezius but not in the right. As seen in the results of the pain scores with GRMS, the upper trapezii showed a greater numbers of significant correlations with fatigue scores than the sternocleidomastoid muscles in the arm raise activity. The NTNP group showed the highest number of significant individual correlations for fatigue and GRMS in both upper trapezii. It is possible that, as with the results of the pain scores, the fatigue felt by the NTNP participants originated more from upper trapezii activity than in the other groups during the arm raise, hence the higher number of significant correlations. The fatigue felt by the WAD and HC group may have had more generalised origins, from a wider range of muscles, the fatigue may also be related to the pain felt by the participants. The WAD participants had higher initial pain scores and this may have had an impact on the reported fatigue.

All groups showed more than 50% significant individual correlations for fatigue with RMS in the left sternocleidomastoid muscle during the arm raise activity (see section 7.2.11). There were no significant correlations for fatigue with RMS in the right sternocleidomastoid muscle. These results mirror the correlations of pain and activity in the sternocleidomastoid muscles during the arm raise activity. As previously discussed, it is possible that the right sternocleidomastoid muscle may be activated more at low levels than the left sternocleidomastoid due to the increased use of the right side, e.g. for writing and typing. This could result in the right side musculature being more fatigue resistant, hence the lack of correlation with the subjective scores.

In the head raise activity the HC and NTNP groups showed more than 50% significant individual correlations for fatigue and GRMS in both sternocleidomastoid muscles (see section 7.3.9). The WAD group showed more than 50% significant correlations in the left sternocleidomastoid muscle but not the right. However, the small numbers in the WAD group mat have affected these results. The WAD group achieved 50% significant individual correlations in the right sternocleidomastoid muscle. The results show that self-reported fatigue correlated well with increasing activity in the sternocleidomastoid muscles.

Similarly to the results of the pain scores, the only group with more than 50% significant correlations for fatigue and GRMS in the upper trapezii were the NTNP group during the head raise activity (see section 7.3.9). The lack of significant correlations for the GRMS and fatigue for the HC and WAD groups during the head raise exercise indicates the activity of these muscles was not directly related to the fatigue reported by these participants. It is likely that the low levels of activity in these muscles meant that their activation did not contribute greatly to the participants' perception of fatigue.

There were no significant individual correlations for fatigue with GMDF in the upper trapezii during the arm raise activity (see section 7.2.11). Whereas the NTNP group showed more than 50% significant correlations with GMDF and fatigue in both sternocleidomastoid muscles during the head raise activity (see section 7.3.9). The HC group showed more than 50% significant correlations with GMDF and fatigue in the right sternocleidomastoid. The WAD group showed less than 50% for fatigue and GMDF for both sternocleidomastoid muscles. These results were similar for those seen with GMDF and self reported pain. The change in GMDF values correlated well with the fatigue scores in NTNP group indicating that the fatigue in these muscles was involved in these participants' perception of fatigue.

A strong correlation was found between perceived fatigue and force production measured by a CR10 Borg scale, and the slope of muscle activation of lumbar muscles has been reported (Dedering et al., 2002). However, a low correlation between the slope of perceived fatigue was reported from the same study. The

different strength of correlations between this and the present study could be due to the different methods of analysis. Using a slope rather than distinct values could mask subtle changes in EMG activity. A slope may not reflect different values, e.g. initial values, only the relationship between the values over time.

Significant correlations have been reported between MDF slopes and subjective fatigue at specific Borg ratings (Dedering et al., 1999). The study by Dedering et al. observed lumbar muscles during isometric, fatiguing contractions in pain-free participants. The endurance task employed was performed until fatigue prevented maintenance of the position, which corresponds to the type of non-forced fatiguing isometric contraction used in the present study. These results and the results of the present study show that self perception of fatigue may relate to fatigue in both cervical and lumbar muscles.

A significant relationship between perceived exertion and mean power frequency of the upper trapezius during shoulder elevation has been reported (Hummel et al., 2005). This study used the Borg scale to measure perceived exertion, as opposed to fatigue, with the slope of the mean power frequency. Although this agrees in principle with the present study, the contractions were forced and of a fixed duration which are likely to produce different results than from a non-forced fatiguing activity.

Similarly to the results of the pain scores with GRMS and GMDF, these results indicate that GRMS from the muscles directly involved in the activities was the more reflective measure of an individuals' perception of fatigue than GMDF.

8.3.3   Pain and fatigue scores

For both activities all groups showed more than 50% significant individual correlations between subjective scores of pain and fatigue. All groups had 100% correlations in the arm raise activity (see section 7.2.11) whereas only the NTNP group had 100% correlations in the head raise activity (see section 7.3.9). The WAD group showed the least number of significant correlations with 75%.

A significant correlation between pain and fatigue has been shown in headache patients and healthy controls during a one-hour reaction time test (Bansevicius et al., 1999). This is in agreement with the present study, where correlation of pain and fatigue scores showed the strongest significance in the NTNP and HC groups. The weaker correlations seen in the WAD group could be the result of pain originating from deeper structures as opposed to the superficial muscles. Higher pre-test pain scores were seen in the WAD group, which could imply that this group does not cope with pain as efficiently or that they have increased pain sensitisation as compared to the NTNP group. Good coping strategies could explain why the NTNP group showed similar results to the HC group.

It has been shown that subjective measures of pain can be affected by pain catastrophising, which has been related to increased pain perception but not to increased physiological reflexes to painful stimuli (France et al., 2002). Methods to develop coping strategies, and therefore minimise catastrophising, have been seen to be effective in reducing subjective measures of pain and disability. Cognitive behavioural intervention was shown to be effective for individuals with persistent neck and back pain in preventing future disability as reported by sick leave (Linton & Ryberg 2001). Ergonomic counselling and muscle feedback training have also been seen to be effective in reducing pain and disability in individuals with neck and shoulder pain (Voerman et al., 2007). Coping strategies empower an individual and make them feel less helpless regarding their pain; this can affect how that individual perceives their pain.

The NTNP and WAD participants responded voluntarily to poster advertisements and information sheets which stated that the activities may cause minor pain and discomfort. These two groups were therefore not representative of the chronic neck pain population but rather a distinct, self-selected, subset of the population. This is an important consideration when comparing the results of other chronic pain studies. The attitudes of patients in pain clinics may be very different to those who volunteer themselves from a workplace environment. It is feasible that they were prepared to undertake the activities because they had developed efficient coping strategies and were not unduly concerned about the possibility of minor pain or discomfort in their muscles, which may have impacted on the results.

8.3.4   Summary of subjective scores

The measure that demonstrated the most consistent number of significant correlations was the GRMS values (from the muscles directly involved in both activities), with subjective pain and fatigue scores. The NTNP group showed the most consistent, high number of individual significant correlations for both activities. One explanation for the differences in subjective scores between the NTNP group and the WAD group could be that the NTNP group have developed better coping strategies. The duration of pain was longer in the NTNP group than in the WAD group and therefore may have provided more time for participants to develop coping strategies and develop more effective descending pain inhibition. Also, the trauma of the whiplash injury may have resulted in greater tissue damage and initial pain leading to increased pain, and possibly catastrophising, in the WAD group.

However, evidence for effective coping strategies was not reflected by the similar duration of activities shown by all groups. It may have been expected that the HC group would hold positions for longer than the other groups as they were not experiencing any pre-existing pain. However, the NTNP and WAD groups could be better equipped to cope with the pain resulting from the activities than the HC group, therefore being able to maintain the positions for similar lengths of time. This does not explain the different relationships between pain and fatigue shown by the HC and NTNP groups. The HC group, not having any pre-existing pain, may perceive their pain and fatigue from a wider range of muscles involved in the activities, resulting in less direct associations between the subjective scores and the sEMG parameters.

8.4   Limitations of the study

A major limitation of this study was the small number of WAD participants. It proved difficult to recruit sufficient WAD participants from the staff and students of The University of Southampton, which was not ideal for statistical comparison. The sample size, informed by the pilot study, was 20 per group (based on the results of three muscles). This was not achieved and the WAD group size was especially low.

The consequence of the small group size was that there was an insufficient weight of data to confirm the differences and trends seen (refer to section 8.6 for future studies). It was also possible that the differences seen in WAD group were due to the small size of the group.

A factor that may affect the reproducibility of the present study was the order that the two non-forced fatiguing activities were performed. The order was specific, with the arm raise activity being undertaken first. This may impact on future research if the protocol for the head raise activity was undertaken without the preceding arm raise activity. It is possible that the fatiguing nature of the arm raise activity may impact on the ability of participants to perform the head raise activity.

Another factor that should be considered is that this study was not a blind trial, the researcher knew which participants had pain and which did not, this may have influenced how the participants were approached. It is possible that the researcher may have unknowingly not encouraged the participants with pain as much as the pain-free participants in order to minimise aggravation of the participants' pain.

The skill of researcher in using the technique may have affected the results in this study as well as the ability to replicate this method in future studies. Although training was undertaken by the researcher, there was a large amount of operator judgement in using the decomposition software. The threshold value for MUAP detection is dependent upon operator judgement; at high activity levels there is a trade off between losing smaller MUAPs and preventing superimposition of MUAPs. This could result in an underestimation of MUAPs and their subsequent parameters.

The methodology for main study was not tested for reliability in the pilot study, due to the linear array electrodes and recording system becoming available after completion of the pilot study. The reliability for the linear array electrodes was based on another study (Kallenberg et al., 2009). However, the researcher training in the use of the software was provided by Dr Kallenberg, the author of the reliability study, to provide a level of consistency.

Another aspect of the methodology that may limit the reliability of this study was the application of electrodes. There was movement of electrodes and skin over the

sternocleidomastoid muscles, which impaired the accuracy of the electrode position. This affected the ability to detect motor unit action potentials in many participants. Although the method employed allowed an accurate measure of head height during the head raise, the recordings were compromised due to the movement required to assume the position. The electrodes could not be placed on the neck in the raised position as this would be impractical and uncomfortable for the participants and would have required muscle effort to hold the position. A study using only isometric muscle movement, similar to the cranio-cervical flexion test (Falla et al., 2003c) would limit the problem of electrode movement.

In the less active muscles, e.g. the upper trapezius muscles during the head raise activity) the small signal that was produced by some participants resulted in a poor signal to noise ratio. This prevented the signals from being able to be processed accurately.

The use of the linear array electrodes did, in many instances, allow a more detailed analysis of the motor units directly under the electrodes, but as with traditional bipolar electrodes, the recorded muscle response was limited. The recordings cannot be assumed to represent the whole muscle and only those motor units detected by the electrodes placed directly above them. This is especially relevant for the MUAP variables where field potentials and cross talk are removed from the signal. Results of sEMG studies, including the present study can only describe what is happening in a relatively small area of the muscle, this does not necessarily reflect what is happening in the whole muscle.

Although the area of pain felt by the participants was recorded, e.g. posterior, right shoulder, this may not be where damage had occurred or be the source of the pain. Muscle pain is often referred and can also be the result of nerve damage or inflammation around a nerve so a comparison of the location of pain with the results of activity in a particular muscle may not provide an accurate reflection of muscle/pain relationships. The majority of participants had bilateral pain and this made side specific analysis difficult.

Another limitation of this study was a lack of data on the pain participants coping strategies. The NTNP group showed a higher mean duration in the arm raise activity. It is possible that due to their pain, and the non-traumatic nature of the onset, that they had developed good coping strategies and therefore were better able to cope with the pain resulting from the activity. It would have been interesting to see if an individual's results were related to their coping strategies.

8.5   A proposed model for the development of chronic neck pain with clinical implications

Understanding the mechanisms of the development of chronic neck pain, traumatic or non-traumatic, could help approaches to prevention, treatment and rehabilitation of this condition. A model proposing how chronic neck pain develops from traumatic and non-traumatic origins is shown in Figure 8.1 (developed from the model in section 2.8, figure 2.7). This model proposes that, irrespective of the cause of trauma, initial pain can result in hypertonicity of the muscles involved. This can lead to decreased blood flow in the muscles and ischaemia, which can exacerbate the pain.

Peripheral and central sensitisation can then occur, which is a normal physiological response at this point. However, increased pain sensations can then lead to altered motor control strategies to minimise the use of the painful muscles. Inappropriate or over reliance of alternative muscles, employed to avoid using those muscles that elicit a painful response, can also result in hypertonicity, decreased blood flow, and ischaemia, which can compound the problem.

The manner of the reorganisation appeared to differ between the groups in this study and this has implications for the long term outcomes. The NTNP group appear to recruit alternative superficial muscles to assist in movement (dynamic reorganisation); this was seen in this study with the slight increase in use of the

Figure 8.1 A model for the development of chronic pain from different onsets

Cause	Initial response	Secondary response and outcome

Repetitive non-impact injury e.g. Poor posture, daily keyboard use → Superficial tissue damage **Initial pain** → Hypertonicity, decreased blood flow, ischaemia / Peripheral and central sensitisation → Alteration of superficial dynamic muscle activation (pain adaptation) → Reorganisation of motor control strategies. → **Chronic pain**

Single high-impact trauma e.g. whiplash injury → Deep tissue damage e.g. Muscle, tendon and ligament. **Initial pain** → Reliance on superficial muscles for posture (fear avoidance) → Changes in muscle fibre type / Atrophy of deeper muscles → **Chronic pain**

Non-habituation of response → **Recovery**

Main areas for the effects of contributory factors (dashed line)
Negative response: Contributing factors include pain-catastrophising, a lack of a sense of control over pain, unsatisfactory employment, unsatisfactory employment status, lack of self-awareness, fear of pain. **Positive response**: Contributing factors include no pain-catastrophising, a sense of control over pain, satisfactory employment, satisfactory employment status, self-awareness, lack of fear of pain.

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sternocleidomastoid muscles in the arm raise activity. The WAD group appeared to show a different alteration in motor control strategies with an increased longer term reliance on superficial muscles for posture. This was reflected by the different activation and fatiguing properties of the upper trapezius during the arm raise activity. These results correspond with the avoidance/endurance model Hasenbring (see section 2.6.4.iv) with the NTNP group appearing to demonstrate endurance and the WAD group showing avoidance in activities due to pain.

The fundamental difference between the two groups appears to be in the extent of the reliance upon the superficial muscles and the consequence of the alteration of muscle use. The NTNP group showed quite similar responses to the HC group in the activation of the upper trapezius in the arm raise activity whereas the WAD group results indicated that their muscles had more, smaller motor units possibly as a result of a transition to more fatigue resistant type I fibres. This change in morphology of the motor units and the fibres they contain may reflect the increased use of these muscles for posture. A result of this alteration in use could be disuse atrophy of the deeper muscles (see section 2.2.3), which could be a result of the initial trauma in the WAD group. It is more likely that the WAD group had damage to the deeper muscles due to the nature of a whiplash injury, and these are the muscles that they avoid using. This could be due to fear of the damage caused to the neck, leading to fear avoidance (see section 2.6.4.iii). This pattern of fear avoidance could lead to the development of a vicious cycle perpetuating chronic pain.

The pain from initial damage, poor posture and inappropriate use of muscles can all cause repetitive firing of the nociceptive neurons, this can lead to increased sensitisation. Often the response to the pain can also cause pain, resulting in increased reorganisation of the nervous system. These factors can all contribute to the chances of an individual developing chronic neck pain.

A lack of knowledge or control of the pain could cause a negative response to the initial injury, leaving individuals fearful of using the painful area. This fear may not be so apparent in the NTNP group due to the lack of trauma involved, possibly with more of an understanding of their pain. The difference in the pain was reflected in

the initial scores. Efficient education, diagnosis and rehabilitation could prevent the reorganisation of motor control strategies, reducing the incidence of chronic pain. Both groups demonstrated alterations in response to pain, however, both groups continued to have pain. It is postulated that avoiding overuse and underuse may increase the chance of recovery and avoid the development of chronic neck pain.

8.6   Future studies

One of the major limitations of the present study was the small participant number in the WAD group. Therefore, further research could be undertaken with comparable group sizes of increased numbers. This may show whether the differences seen between the groups were due to the nature of their chronic pain or due to the small numbers in the group. Larger participant numbers would also enable investigation into gender differences to determine if there are differences in alteration of motor control and muscle activation between males and females.

It would also be interesting to compare chronic neck pain participants from a working environment and from a healthcare setting to observe if the treatment or lack of treatment may impact on the results. It may be that those seeking medical attention place more emphasis on their pain rather than those individuals who do not seek treatment. Participants that are not part of a treatment group or recruited through healthcare settings may respond to pain differently when compared to those that are. They may not suffer from the same pain intensity or they may cope with pain more effectively.

Inclusion of questionnaires regarding efficacy of coping strategies could provide an interesting insight into subjective responses. It could also be possible to identify individuals employing different motor control strategies and to explore these specific subcategories in larger groups. Specific chronic pain groups and extended outcome measures could then be used to gain a better insight into muscle activation and further explore the avoidance-endurance model of pain.

The present study investigated non-forced fatiguing activities, which is not a particularly common method of assessing muscle function. This made comparison

with many other studies difficult. It would be interesting to study individuals with chronic neck pain performing a variety of tasks, e.g. functional and fatiguing, forced and non-forced, to observe the effects on characteristics of muscle activation. This would allow a greater comparison of results with other studies undertaken in this area.

Further studies could simultaneously investigate a wider range muscles involved in the arm raise activity. This could provide more detail regarding altered muscle recruitment during a fatiguing activity. It may also show different shifts in muscle use that occur in order to maintain a particular position for a long time. The simultaneous recording of the sub divisions of the trapezius may also provide further insight into reorganisation of muscle activity.

A reliability study involving the subjective responses as compared to the mean and median frequency of the sEMG may determine the most appropriate method. The median is often used to prevent data being skewed as a result of outlying data points. However, sEMG responses are very variable and therefore participant groups may often have many outlying data point. Therefore the mean may be more appropriate. A comparison of analysis methods may clarify this.

In light of the problems with the electrode placement for the sternocleidomastoid muscles in the head raise activity a different isometric activity, which does not require initial movement to assume the recording position, could be developed. This would allow a more detailed analysis of these muscles under the non-forced fatiguing conditions.

Chapter 9 – Conclusions

The use of non-forced fatiguing activities has provided a novel insight into the effects of chronic neck pain on muscle activation and synergy. Statistically significant differences between the groups were seen in activation and synergy of the superficial neck muscles during bilateral arm raises, trends were also seen in the head raise activity. The WAD group showed an increased CNS input as measured by MR and decreased RMS in the upper trapezius muscles in the arm raise activity as compared to the HC and NTNP groups. Trends for a slight increase in the activity and MR of the synergistic muscles were seen in the NTNP group during the arm raise exercise.

The pattern of muscle activation in the head raise activity was similar to that seen in the arm raise activity, where the synergistic muscles showed increased activity in the HC and NTNP group as compared to the WAD group.

Increased amplitude of the sEMG signal from the muscles directly involved in the arm and head raise activities showed high numbers of statistically significant correlations with self reported pain and fatigue. The NTNP group showed the highest and most consistent numbers of individual significant correlations. This indicates that the activity of these muscles was more involved in the perceived pain and fatigue in the NTNP group.

A larger sample size is required to establish whether the results of this study can be inferred to a wider population. It is possible that differences in the responses were representative, but the insufficient weight of data did not allow this to be proved statistically.

The results of this study will help to determine the efficacy of sEMG as an assessment tool for chronic pain and resulting muscle fatigue. Possible alterations in muscle synergy that could lead to the development of chronic neck pain are demonstrated in the chronic neck pain model. This could be a valuable resource in clinical rehabilitation.

Appendix A – Numerical rating scale

Numerical Rating Scale

Please indicate along the scale the intensity of your pain.
Please circle your answer


```
0    1    2    3    4    5    6    7    8    9    10
|    |    |    |    |    |    |    |    |    |    |
```
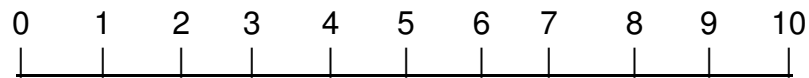
  0 = No pain   10 = The worst
                                                           pain imaginable

The Neck Disability Index

The Neck Disability Index This questionnaire has been designed to give the researcher information as to how your neck pain has affected your ability to manage everyday life. Please answer every section and mark in each section only the ONE box that applies to you. We realise that you may consider that two of the statements in any one section may relate to you, but please just mark the box that most closely describes your problem.

Neck Disability Index

Section 1 – Pain intensity		Section 4 - Reading	
	I have no pain at the moment		I can read as much as I want with no pain in my neck
	The pain is very mild at the moment		I can read as much as I want with slight pain in my neck
	The pain is moderate at the moment		I can read as much as I want with moderate pain in my neck
	The pain is fairly severe at the moment		I can't read as much as I want because of moderate pain in my neck
	The pain is very severe at the moment		I can hardly read at all because of the severe pain in my neck
	The pain is the worst imaginable at the moment		I cannot read at all
Section 2 – Personal care		Section 5 – Headaches	
	I can look after myself normally without causing extra pain		I have no headaches at all
	I can look after myself normally but it causes extra pain		I have slight headaches which come infrequently
	It is painful to look after myself and I am slow and careful		I have moderate headaches which come infrequently
	I need some help but I manage most of my personal care		I have moderate headaches which come frequently
	I need help every day in most aspects of self care		I have severe headaches which come frequently
	I do not get dressed, I wash with difficulty and stay in bed		I have headaches almost all of the time
Section 3 - Lifting		Section 6 - Concentration	
	I can lift heavy weights without extra pain		I can concentrate fully when I want to with no difficulty
	I can lift heavy weights but it gives me extra pain		I can concentrate fully when I want to with slight difficulty
	Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, e.g. on a table		I have a fair degree of difficulty in concentrating when I want to
	Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned		I have a lot degree of difficulty in concentrating when I want to
	I can lift very light weights		I have a great deal of difficulty in concentrating when I want to
	I cannot lift or carry anything at all		I cannot concentrate at all

Section 7 – Work		Section 9 - Sleeping	
	I can do as much work as I want to		I have no trouble sleeping
	I can only do my usual work, but no more		My sleep is slightly disturbed (less than 1 hour sleeplessness)
	I can do most of my usual work, but no more		My sleep is mildly disturbed (1-2 hours sleeplessness)
	I cannot do my usual work		My sleep is moderately disturbed (2-3 hours sleeplessness)
	I can hardly do any work at all		My sleep is greatly disturbed (3-5 hours sleeplessness)
	I cannot do any work at all		My sleep is completely disturbed (5-7 hours sleeplessness)
Section 8 - Driving		Section 10 – Recreation	
	I can drive my car without any neck pain		I am able to engage in all my recreation activities with no neck pain at all
	I can drive my car as long as I want with slight pain in my neck		I am able to engage in all my recreation activities with some pain in my neck pain
	I can drive my car as long as I want with moderate pain in my neck		I am able to engage in most, but not all my usual recreation activities because of pain in my neck
	I can't drive my car as long as I want because of moderate pain in my neck		I am able to engage in a few, of my usual recreation activities because of pain in my neck
	I can hardly drive at all because of severe pain in my neck		I can hardly do any recreation activities because of pain in my neck
	I can't drive my car at all		I cannot do any recreation activities at all

Appendix C – Ethics approval letter, pilot study

NHS

**SOUTHAMPTON & SOUTH WEST HAMPSHIRE
LOCAL RESEARCH ETHICS COMMITTEES**

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Ref: CW/hph
28 July 2004

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Dear Miss Curtis,

***Full title of study: Are altered cervical muscle activation patterns related to chronic neck pain
in whiplash patients (pilot study to refine methodology)
REC reference number: 04/Q1702/34
Protocol number: 1***

Thank you for your letter of 22 July 2004, responding to the Committee's request for further
information on the above research.

The further information has been considered on behalf of the Committee by the Chairman, Dr David
Briggs.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above
research on the basis described in the application form, protocol and supporting documentation.

**Please note: There is a typographical error on the Patient Information Sheet page 34 Answer
to question 'What happens if something goes wrong?' last sentence. Change "University os
Southampton" to University of Southampton.**

The favourable opinion applies to the following research site:

Site: University of Southampton
Principal Investigator: Miss Sally Curtis, Lecturer

Conditions of approval

The favourable opinion is given provided that you comply with the conditions set out in the attached
document. You are advised to study the conditions carefully.

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document Type: Application
Version:
Dated: 17/05/2004
Date Received: 17/05/2004

Appendix C – Ethics approval letter, pilot study

Management approval

The study may not commence until final management approval has been confirmed by the organisation hosting the research.

All researchers and research collaborators who will be participating in the research must obtain management approval from the relevant host organisation before commencing any research procedures. Where a substantive contract is not held with the host organisation, it may be necessary for an honorary contract to be issued before approval for the research can be given.

Notification of other bodies

We shall notify the research sponsor, University of Southampton that the study has a favourable ethical opinion.

Statement of compliance *(from 1 May 2004)*

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

REC reference number: 04/Q1702/34 **Please quote this number on all correspondence**

Yours sincerely,

[signature]

Mrs Clair Wright
LREC Manager

Enclosures

Standard approval conditions [SL-AC1 or SL-AC2]

An advisory committee to Hampshire and Isle of Wight Strategic Health Authority

Appendix D – Participant information sheet (pilot study)

Ethics Number : 04/Q1702/34

Research title: Muscle activity in pain

This is a pilot study designed to investigate the application of the recording technique. The information gained from this pilot study will be used in a subsequent full-scale research project.

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take the time to read the following information carefully and discuss it with others if you wish. Please contact Sally Curtis (Tel: 023 8059 5069 or email: sac3@soton.ac.uk) if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part in this study.

Thank you for reading this.

The purpose of this study:
Whiplash injury results from an impact that causes the body to accelerate at a rate greater than that of the head. This can cause the neck to form an "S" shape and cause damage to the soft and bony structures of the neck. Chronic pain (pain that lasts longer than 4 weeks) occurs in a proportion of individuals who have suffered a whiplash injury, the mechanisms leading to this pain is unclear. It is possible that injury leads to a change in the way that muscles are used leading to long-term changes in the muscle structure that may result in muscles that become fatigued or tired more easily. These changes may be a contributory factor in developing chronic pain. Identifying such changes may help therapists in the future to devise treatment with the aim of preventing the development of long-term pain.

How long will it take?
This study will require one visit of one hour.

How many participants?
This study will involve a total of 15 participants.

Why have I been asked?
You have been asked to take part in this study following your response to my request for volunteers. You fall into either of the following two categories:

    a.   Participants who have non-specific neck pain that has lasted for more than 4 weeks within the last 12 months

    b.   Participants that do not have neck pain.

Participation in this study is on an entirely voluntary basis
It is entirely up to you whether you decide to take part in this study. If you do decide to take part you will be given this information sheet to keep and asked to sign a consent form. It is important that you know that if you do decide to take part in this study you are free to withdraw at any time without giving a reason.

What does being a part of this study involve?
If you give your consent to be a part of this study you will be contacted by telephone or email and asked some questions. Participants will then be asked to attend one session lasting 1 hour. This session will take place in a research laboratory at the University of Southampton. All travelling and out of pocket expenses will be reimbursed. At each session you will be asked to fill in a questionnaire regarding the pain you experience and two recordings will be taken. The first will be from your neck muscles during arm raises. You will be asked to hold your arms outstretched until your arms get tired. Secondly you will be asked to lie on the floor and raise your head until your neck muscles become tired.

The recordings are called electromyograms or EMG's and they are a recording of the electrical activity that occurs within muscles.

This is a totally non-invasive technique; electrodes will be placed on the skin to obtain the recording and simply peeled off when the procedure is finished.

This study does not require any activity outside of the research laboratory.

What are the risks or disadvantages of taking part in this study?
There is a possibility that you may experience some discomfort during the exercise and some muscle soreness afterwards. There is also a possibility that you may have

an allergic reaction to the electrodes. Hypoallergenic electrodes can be used if you have sensitive skin.

What are the benefits of taking part in this study?
There are no direct benefits to you in taking part in this study. However the information gathered from this study may aid future treatment methods for long-term neck pain.

What happens when the research stops?
When the research has been completed you will be sent a copy of the results to tell you what information has been found.

What happens if something goes wrong?
If you suffer any harm as a result of taking part in this research project, there are no special compensation arrangements. If you are harmed due to someone's negligence, then you may have grounds for a legal action but you may have to pay for it. Regardless of this, if you wish to complain, or have any concerns about any aspect of the way you have been approached or treated during the course of this study, the normal University of Southampton complaints mechanisms should be available to you.

Will my taking part in this study be kept confidential?
All information that is collected about you during the course of this study will be kept strictly confidential. Any information that leaves the University will have your name and address removed so that you cannot be recognised from it.

What happens to the results of this study?
It is hoped that the results from this study will be published within 18 months of its completion in a relevant healthcare or scientific journal. You will not be identified in any such publication.

Who is organising and funding this research?
This research is funded by a studentship awarded by the School of Healthcare Professions and Rehabilitation Sciences, University of Southampton. The researcher receives no payment, other than the costs of the research, for undertaking this study.

Who has reviewed this study?
This study has been reviewed and approved by the Southampton and South West Hampshire Local Research Ethics Committee.

Contact for further information
If you have any questions or require any further information please contact:

Sally Curtis
C/o School of Medicine
New College Campus
University of Southampton
The Avenue
Southampton
SO17 1BG

Tel: 023 8059 7422
Email: sac3@soton.ac.uk

Thank you very much for taking the time to read this information sheet. If you decide to take part in this study you will be given a copy of this information sheet and a signed consent form to keep.

Date:

Appendix E – Consent form (Pilot study)

University of Southampton

School of Health Professions & Rehabilitation

Professor Maureen J Simmonds, Head of School

University of Southampton	tel +44 (0)23 8059 2142
Highfield	Admissions +44(0)23 8059 5260
Southampton	Fax +4(0)23 8059 5301
SO17 1BJ	Email sotpadm@soton.ac.uk
United Kingdom	Web www.sohp.soton.ac.uk/sohp

Centre number

Study number

Patient identification number

Title of project: Muscle activity in pain

Name of Researcher: Sally Curtis

Please
initial
box

1. I confirm that I have read and understand the information sheet dated……………. (version…..) for the above study and have had the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason. ☐

3. I agree to take part in the above study. ☐

_____   _____   _____

Name of patient   Date   Signature

_____   _____   _____

Name of person
Taking consent
(if different from
researcher)   Date   Signature

_____   _____   _____

Name of researcher   Date   Signature

Appendix F – Case record sheet

<u>Case Record Sheet</u>

Participant No. ……………………….

Male/Female ………………………

Height ……………

Weight ……………

Age ………….

Handedness ……………

Duration of pain ………………

Any previous neck pain or neck injury
………………………………………………………………………………………………

………………………………………………………………………………………………

………………………………………………………………………………………………

Upper trapezius fatigue ratings

………………………………………………………………………………………………

Upper trapezius pain ratings

………………………………………………………………………………………………

Sternocleidomastoid fatigue ratings

………………………………………………………………………………………………

Sternocleidomastoid pain ratings

………………………………………………………………………………………………

Appendix G – Ethics approval letter, main study

UNIVERSITY OF
Southampton
School of Health Professions
and Rehabilitation Sciences

28 June 2007

Sally Curtis
School of Health Professions and Rehabilitation Sciences
University of Southampton

Dear Sally

Submission No: PO7/04-01

Title: Are altered cervical muscle activation patterns related to chronic neck pain in whiplash patients?

The School of Health Professions and Rehabilitation Sciences Ethics Committee has considered your application for the above study at its recent meeting and I am pleased to inform you that **Full Approval** was granted.

You are required to complete a University Research Governance Form (enclosed) in order to receive insurance clearance before you begin data collection. You need to submit the following documentation in a plastic wallet to Dr Martina Dorward in the Research Support Office (RSO, University of Southampton, Highfield Campus, Bldg. 37, Southampton SO17 1BJ):

- Completed Research Governance form (**signed by both student and supervisor**)
- Copy of your research protocol (final and approved version)
- Copy of participant information sheet
- Copy of SoHPRS Risk Assessment form, **signed by yourself and supervisor** (original should be with Zena Galbraith)
- Copy of your information sheet and consent form
- Copy of this SoHPRS Ethical approval letter

Your project will be registered at the RSO, and then automatically transferred to the Finance Department for insurance cover. **You can not commence data collection until you have received a letter stating that you have received insurance clearance.**

Please note that you have ethics approval only for the project described in your submission. If you want to change any aspect of your project (e.g., recruitment or data collection) you must discuss this with your supervisor and you may need to request permission from the Ethics Committee.

Yours sincerely

Dr Emma Stack
Chair, SHPRS Ethics Committee

Enc. SoHPRS RG Form and Guidance notes

Appendix H – Participant information sheet NTNP group (Main Study)

School of Health Professions & Rehabilitation Sciences

Professor Roger Briggs, Head of School

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United Kingdom	Web www.sohp.soton.ac.uk/sohp/

PARTICIPANT INFORMATION SHEET - (Ethics No. PO7/04-01)

Research title:

Are altered cervical (neck) muscle activation patterns related to chronic neck pain in whiplash patients?

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being doneand what it will involve. Please take the time to read the following information carefully and discuss it with others if you wish. Please contact Sally Curtis (Tel: 023 8059 5609 or email:sac3@soton.ac.uk) if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part in this study; you may like to discuss it with your family or your GP.

Thank you for reading this.

The purpose of this study:

Whiplash injury results from an impact that causes the body to accelerate at a rate greater than that of the head. This can cause the neck to form an "S" shape and cause damage to the soft and bony structures of the neck. Chronic pain (pain that lasts longer than 2 months) occurs in a proportion of individuals who have suffered a whiplash injury; the mechanisms leading to this pain are unclear. It is possible that injury leads to a change in the way that muscles are used leading to long-term changes in the muscle structure. These changes may mean that the muscles become fatigued or tired more easily and contribute to the development chronic pain. Identification of muscle changes soon after the injury may, in the future, help therapists to treat whiplash injury so that the risk of long-term pain can be reduced.

How long will it take?
This study will require one visit of one and a half hours.

How many participants?
This study will involve a total of 60 participants.

Why have I been asked?
You have been asked to take part in this study following your response to my request for participants who have experienced neck pain for longer than 2 months in the last 2 years with no history of neck injury or trauma.

Participation in this study is on an entirely voluntary basis
It is entirely up to you whether you decide to take part in this study. If you do decide to take part you will be given this information sheet to keep and asked to sign a consent form. It is important that you know that if you do decide to take part in this study you are free to withdraw at any time without giving a reason and that this will not affect the standard of care you will receive.

What does being a part of this study involve?
If you give your consent to be a part of this study you will be contacted by telephone or email and asked some questions. Following this you will need to attend a one and a half hour session. This session will take place in a health research laboratory at the School of Health Professions, University of Southampton. All travelling and out of pocket expenses will be reimbursed. At the session you will be asked to fill in a questionnaire regarding the pain you experience. You will be asked to undertake two activities during the first a recording will be taken from your neck muscles during arm raises. You will be asked to hold your arms outstretched for a set time or until your arms get tired, whichever happens first. During the second activity a recording will be taken from your neck muscles during head raises. You will be given the opportunity to practice the activity before the recordings. The recordings are called electromyograms or EMG's and they are a recording of the electrical activity that occurs within muscles. This is a non-invasive technique; electrodes will be placed on the skin to obtain the recording and simply peeled off when the procedure is finished.

This study does not require any activity outside of the University of Southampton.

What are the risks or disadvantages of taking part in this study?
There is a possibility that you may experience some discomfort during the exercise and some muscle soreness afterwards. There is also a possibility that you may have

an allergic reaction to the electrodes. Hypoallergenic electrodes can be used if you have sensitive skin.

What are the benefits of taking part in this study?
There are no direct benefits to you in taking part in this study. However the information gathered from this study may aid future treatment methods for long-term neck pain.

What happens when the research stops?
When the research has been completed you will be sent a copy of the results to tell you what information has been found.

What happens if something goes wrong?
If you suffer any harm as a result of taking part in this research project, there are no special compensation arrangements. If you are harmed due to someone's negligence, then you may have grounds for a legal action but you may have to pay for it. Regardless of this, if you wish to complain, or have any concerns about any aspect of the way you have been approached or treated during the course of this study, the University of Southampton's complaints mechanisms should be available to you.

Will my taking part in this study be kept confidential?
All information that is collected about you during the course of this study will be kept strictly confidential. Any information that leaves the University will have your name and address removed so that you cannot be recognised from it.

What happens to the results of this study?
It is hoped that the results from this study will be published within 18 months of its completion in a relevant healthcare or scientific journal. You will not be identified in any such publication.

Who is organising and funding this research?
This research is funded by a studentship awarded by the School of Health Professions and Rehabilitation Sciences, University of Southampton. Equipment has been donated by the Gerald Kerkut Trust. The researcher receives no payment, other than the costs of the research, for undertaking this study.

Who has reviewed this study?
This study has been review by the Ethics Committee, School of Health Professions and Rehabilitation Sciences, University of Southampton.

Contact for further information

If you have any questions or require any further information please contact:

Sally Curtis
C/o School of Medicine
Boldrewood Campus
University of Southampton
Bassett Crescent East
Southampton
SO16 7PX

Tel: 023 8059 5609
Email: sac3@soton.ac.uk

Thank you very much for taking the time to read this information sheet. If you decide to take part in this study you will be given a copy of this information sheet and a signed consent form to keep.

Date:

School of Health Professions & Rehabilitation Sciences

Professor Roger Briggs, Head of School

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United Kingdom	Web www.sohp.soton.ac.uk/sohp/

Reply Slip

I give permission for Sally Curtis to contact me on telephone number…………………………………. or email ……………………… with regards to participation in this study.

Are you currently taking part in any other research studies? Yes / No

Signed …………………………………….

Print Name…………………………………………

Date ………………………

Thank-you

Appendix I – Consent form (main study)

University of Southampton | **School of Health Professions & Rehabilitation Sciences**

Professor Roger Briggs, Head of School

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CONSENT FORM

Participant identification number

Title of project: Muscle activity in pain

Name of Researcher: Sally Curtis

Ethics number: PO7/04-01

Please initial box

4. I confirm that I have read and understand the information sheet dated……………for the above study and that I have had the opportunity to ask questions.

5. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason.

6. I agree to take part in the above study.

_____   _____   _____
Name of participant   Date   Signature

_____   _____   _____
Name of researcher   Date   Signature

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References

Aguggia, M. 2003, Neurophysiology of pain., Neurological science, vol. 24, p. S57-S60.

Anderson, D. M. 2002, Mosby's medical, nursing and allied health dictionary, 6th edn.

Atherton, K., Wiles, N. J., Lecky, F. E., Hawes, S. J., Silman, A. J., Macfarlane, G. J., & Jones, G. T. 2006, Predictors of persistent neck pain after whiplash injury., Emergency Medicine Journal, vol. 23, no. 3, pp. 195-201.

Banic, B., Petersen-Felix, S., Andersen, O. K., Radanov, B. P., Villiger, P. M., Arendt-Nielsen, L., & Curatolo, M. 2004, Evidence for spinal cord hypersensitivity in chronic pain after whiplash injury and in fibromyalgia., Pain, vol. 107, no. (1-2), pp. 7-15.

Bansevicius, D., Westgaard, R. H., & Jensen, C. 1997, Mental stress of long duration: EMG activity, perceived tension, fatigue and pain development in pain free subjects., Headache, vol. 37, pp. 499-510.

Bansevicius, D., Westgaard, R. H., & Sjaastad, O. M. 1999, Tension-type headache: pain, fatigue, tension, and EMG responses to mental activation., Headache, vol. 39, no. 6, pp. 417-425.

Barbe, M. F. & Barr, A. E. 2006, Inflammation and pathophysiology of work-related musculoskeletal disorders., Brain, Behaviour and Immunity, vol. 20, pp. 423-429.

Barnsley, L., Lord, S., & Wallis, B. J. 1995, The prevalence of chronic cervical zygopophyseal joint pain after whiplash., Spine, vol. 20, pp. 20-26.

Barnsley, L., Lord, S., & Bogduk, N. 1994, Whiplash injury., Pain, vol. 58, pp. 283-307.

Baron, R. 2006, Mechanisms of disease: neuropathic pain - a clinical perspective., Nature Clinical Practice Neurology, vol. 2, no. 2, pp. 95-106.

Bendsten, L. 2000, Central sensitization in tension-type headache - possible pathophysiological mechanisms., Cephalagia, vol. 20, pp. 486-508.

Bernard, B. P. 1997, Musculoskeletal disorders and workplace factors. A critical review of epidemiologic evidence for work related musculoskeletal disorders of the neck, upper extremity, and low back. US Department of Health and Human Services (DHSS). National Institute of Occupational Health, Cincinnatti, 97-141.

Bogduk, N. & Yoganandan, N. 2001, Biomechanics of the cervical spine Part 3: Minor injuries., Clinical Biomechanics, vol. 16, no. 4, pp. 267-275.

Boonstra, A. M., Schiphorst-Preuper, H. R., Reneman, M. F., Posthumous, J. B., & Stewart, R. E. 2008, Reliability and validity of the visual analogue scale for disability in

patients with chronic musculoskeletal pain., International Journal of Rehabilitation Research, vol. 31, no. 2, pp. 165-169.

Booth, F. W. (1982). Effect of limb immobilization on skeletal muscle. Journal of Applied Physiology. 52, 1113-1118.

Brault, J. R., Seigmund, G. P., & Wheeler, J. B. 2000a, Cervical muscle response during whiplash: evidence of a lengthening muscle contraction., Clinical Biomechanics, vol. 25, no. 6, pp. 426-435.

Cailliet, R. 1991, Neck and Arm Pain., 3rd edn, F.A. Davies Company, Philadelphia.

Campbell, C. M. & Edwards, R. R. 2009, Mind-body interactions in pain: the neurophysiology of anxious and catastrophic pain-related thoughts., Translational Research, vol. 153, no. 3, pp. 97-101.

Campbell, M. J. & Machin, D. 2005, Correlation and linear regression., in Medical statistics: a commonsense approach, 3rd edn, M. J. Campbell & D. Machin, eds., John Wiley and Sons, Chichester, pp. 94-114.

Carlson, B. M. & Faulkner, J. A. 1983, The regeneration of skeletal muscle fibres following injury: a review., Med Sci Sport Exercise, vol. 15, no. 3, pp. 187-198.

Chein, A., Eliav, E., & Sterling, M. 2008b, Hypoesthesia occurs in acute whiplash irrespective of pain and disability levels and the presence of sensory hypersensitivity., Clinical Journal of Pain, vol. 24, no. 9, pp. 759-766.

Chein, A., Eliav, E., & Sterling, M. 2008a, Whiplash (grade II) and cervical radiculopathy share a similar sensory presentation: an investigation using quantitative sensory testing., Clinical Journal of Pain, vol. 24, no. 7, pp. 595-603.

Constanzo, L. S. 2006, Neurophysiology., in Physiology, 3rd edn, L. Constanzo, ed., Saunders, Philadelphia, pp. 65-110.
Cook, A. J., Brawer, P. A., & Vowles K.E. 2006, The fear-avoidance model of chronic pain: Validation and age analysis using structural equation modeling., Pain, vol. 121, pp. 195-206.

Cote, P., Cassidy, J. D., & Carroll, L. J. 1998, The Saskatchewan health and back pain survey. The prevalence of neck pain and related disability in Saskatchewan adults., Spine, vol. 23, pp. 1689-1698.

Cram, J. R., Kasman, G. S., & Holtz, J. 1998, Introduction., in Introduction to surface electromyography, J. R. Cram, ed., Aspen Publishers, Maryland, pp. 3-7.

Croft, P., Lewis, M., & Hannanford, P. 2003, Is all chronic pain the same? A 25-year follow-up study., Pain, vol. 105, pp. 309-317.

De Luca, C. J., 1984, Myoelectric manifestations of localised muscle fatigue in humans., Crit. Rev. Biomed. Eng. vol. 11, pp. 251-279.

De Luca, C. J. Encyclopedia of Medical Devices and Instrumentation. Webster, J. G. http://www.delsys.com/Attachments_pdf/Electromyography%20Medical%20Encyclopedia. pdf . 2006. John Wiley and Sons.

De Luca, C. J. 1997. Surface electromyography: Detection and recording. http://www.delsys.com/Attachments_pdf/WP_SEMGintro.pdf .

Dedering, A., Nemeth, G., & Harms-Ringdahl, K. 1999, Correlation between electromyographic spectral changes and subjective lumbar muscle fatigue in subjects without pain from the lower back., Clinical Biomechanics, vol. 14, pp. 103-111.

Dedering, A., Oddsson, L., Harms-Ringdahl, K., & Nemeth, G. 2002, Electromyography and ratings of lumbar muscle fatigue using a four-level staircase protocol., Clinical Biomechanics, vol. 17, pp. 171-176.

Dimitrova, N. A. & Dimitrov, G. V. 2002, Interpretation of EMG changes with fatigue: facts, pitfalls and fallacies., Journal of Electromyography and Kinesiology, vol. 13, no. 1, pp. 319-332.

Disslehorst-Klug, C., Bahm, J., Ramaekers, V., Trachterna, A., Rau, G. 2000, Non-invasive approach of motor unit recording during muscle contraction n humans., European Journal of Applied Physiology, vol83, pp 144-150.

Dommerholt, J. 2003, Persistent myalgia following whiplash., Curr Pain Headache Rep, vol. 9, no. 5, pp. 326-333.

Duchateau, J., Semmler, J. G., & Enoka, R. M. 2006, Training adaptations in the behaviour of motor units., Journal of Applied Physiology, vol. 101, no. 6, pp. 1766-1775.

Dumas, J. P., Arsenault, A. B., Boudreau, G., Magnoux, E., Lepage, Y., Bellavance, A., & Loisel, P. 2001, Physical impairments in cervicogenic headache: traumatic vs. non-traumatic onset., Cephalagia, vol. 21, no. 9, pp. 884-893.

Eck, J. C., Hodges, S. D., & Humphries, S. C. 2001, Whiplash: A review of a commonly misunderstood injury., The American Journal of Medicine, vol. 110, no. 8, pp. 651-656.

Enoka, R. M. & Duchateau, J. 2008, Muscle Fatigue., Journal of Physiology, vol. 586, no. 1, pp. 11-23.

Falla, D., Arendt-Nielsen, L., & Farina, D. 2008, Gender-specific adaptations of upper trapezius muscle activity to acute nociceptive stimulation., Pain, vol. 138, pp. 217-225.

Falla, D., Bilenkij, G., & Jull, G. 2004a, Patients with chronic neck pain demonstrate altered patterns of muscle activation during performance of a functional upper limb task. Spine, vol. 29, no. 13, pp. 1436-1440.

Falla, D., Dall'Alba, P., Rainoldi, A., Merletti, R., & Jull, G. 2002b, Location of innervation zones of sternocleidomastoid and scalene muscles - a basis for clinical and research electromyography applications., Clinical Neurophysiology, vol. 113, pp. 57-63.

Falla, D., Dall'Alba, P., Rainoldi, A., Merletti, R., & Jull, G. 2002a, Repeatability of surface EMG variables in the sternocleidomastoid and anterior scalene muscles., European Journal of Applied Physiology, vol. 87, no. 6, pp. 542-549.

Falla, D. & Farina, D. 2008, Non-uniform adaptation of motor unit discharge rates during sustained static contraction of the upper trapezius muscle., Experimental Brain Research, vol. 191, pp. 363-370.

Falla, D., Farina, D., & Graven-Nielsen, T. 2007, Experimental muscle pain results in reorganisation of coordination among trapezius muscle subdivisions during repetitive shoulder flexion., Experimental Brain Research, vol. 178, pp. 385-393.

Falla, D., Jull, G., Dall'Alba, P., Rainoldi, A., & Merletti, R. 2003a, An electromyographic analysis of the deep cervical flexor muscles in performance of craniocervical flexion., Physical Therapy, vol. 83, no. 10, pp. 899-906.

Falla, D., Jull, G., & Hodges, P. W. 2004b, Patients with neck pain demonstrate reduced electromyographic activity of the deep cervical flexor muscles during performance of the craniocervical flexion test., Spine, vol. 29, no. 19, pp. 2108-2114.

Falla, D., Jull, G., Rainoldi, A., & Merletti, R. 2004c, Neck flexor muscle fatigue is side specific in patients with unilateral neck pain., European Journal of Pain, vol. 8, pp. 71-77.

Falla, D., Rainoldi, A., Jull, G., stavrou, G., Tsao, H. 2004d, Lack of correlation between sternocleidomastoid and scalene fatigability and duration of symptoms in chronic neck pain patients., Clinical Neurophysiology, vol. 34, pp. 159-165.

Falla, D., Rainoldi, A., Merletti, R., & Jull, G. 2003b, Myoelectric manifestations of sternocleidomastoid and anterior scalene muscle fatigue in chronic neck pain patients., Clinical Neurophysiology, vol. 114, no. 3, pp. 488-495.

Farasyn, A. 2007, Referred muscle pain is primarily peripheral in origin: The "barrier-dam" theory., Medical Hypotheses, vol. 68, no. 1, pp. 144-150.

Farina, D., Kallenberg, L. A. C., Merletti, R., & Hermens, H. J. 2003a, Effect of side dominance on myoelectric manifestations of muscle fatigue in the human upper trapezius muscle., European Journal of Applied Physiology, vol. 90, pp. 480-488.

Farina, D., Merletti, R., & Enoka, R. M. 2003b, The extraction of neural strategies from the surface EMG., Journal of Applied Physiology, vol. 96, pp. 1486-1495.

Farina, D., Merletti, R., & Stegeman, D. F. 2004, Biophysics of the generation of EMG signals., in Electromyography. Physiology, engineering and non-invasive applications, 1st edn, R. Merletti & P. A. Parker, eds., John Wiley & Sons, New Jersey, pp. 81-106.

Farmer, C. M., Wells, J. K., & Lund, A. K. 2003, Effects of head restraint and seat redesign on neck injury risk in rear-end crashes., Traffic Injury Prevention, vol. 4, pp. 83-90.

Fernández-de-las-Peñas, C., Albert-Sanchís, J. C., Buil, M., Benitez, J. C., Alburquerque-Sendín F. 2008, Cross-sectional Area of Cervical Multifidus Muscle in Females with

Chronic Bilateral Neck Pain Compared to Controls., Journal of Orthopaedic and Sports Physical Therapy, vol 38, no 4, pp 175-180.

Ferrari, R. 2002, Prevention of chronic pain after whiplash., Emergency Medicine Journal, vol. 19, pp. 526-530.

Ferrari, R. & Russell, A. S. 2003, Neck Pain., Best Practice and Clinical Rheumatology, vol. 17, no. 1, pp. 57-70.

Ferrari, R. & Schrader, H. 2001, The late whiplash syndrome: a biopsychosocial approach., J Neurol Neurosurg Psychiatry, vol. 70, pp. 722-726.

Feyer, R., Kyvik, K. O., & Hartvigson, K. O. 2006, The prevalence of neck pain in the world population: a systematic critical review of the literature., European Spine Journal, vol. 15, pp. 834-848.

Fleiss, J. 1986, The design and analysis of clinical experiments., John Wiley and Sons, New York.

Flor, H. & Turk, D. C. 2006, Cognitive and Learning Aspects., in Wall and Melzack's Textbook of Pain, 5th edn, S. B. McMahon & M. Koltzenburg, eds., Elsevier Churchill Livingstone, China, pp. 241-253.

Foreman, S. M. & Croft, A. C. 1995, Whiplash Injuries: The cervical acceleration/deceleration syndrome., 2nd edn, Williams and Wilkins, Maryland.

Franco, G. & Fusetti, L. 2003, Bernardino Ramazzini's early observations of the link between musculoskeletal disorders and ergonomic factors., Applied Ergonomics, vol. 35, no. 1, pp. 67-70.

Garson, G. Reliability Analysis. http://www2.chass.ncsu.edu/garson/pa765/reliab.htm . 2007. 12-6-0007.

Ge, H. Y., Arendt-Nielsen, L., Farina, D., & Madelaine, P. 2005, Gender specific differences in electromyographic changes and perceived pain induced by experimental muscle pain during sustained contractions of the upper trapezius muscle., Muscle and Nerve, vol. 32, pp. 726-733.

Gerdle, B., Karlsson, S., Crenshaw, A. G., Elert, j., & Friden, J. 2000, The influences of muscle fibre proportions and areas upon EMG during maximal dynamic knee extensions., European Journal of Applied Physiology, vol. 81, pp. 2-10.

Gifford, L., Thacker, M., & Jones, M. A. 2006, Physiotherapy and pain., in Wall and Melzaks textbook of Pain, 5th edn, S. B. McMahon & M. Koltzenburg, eds., Elsevier Churchill Livingstone, China, pp. 603-620.

Guez, M., Brannstrom, R., Nyberg, L., Toolanen, G., & Hildingsson, C. 2005, Neurophysiological functioning and MMPI-2 profiles in chronic neck pain: A comparison of whiplash and non-traumatic groups, Journal of Clinical and Experimental Neuropsychology, vol. 27, pp. 151-163.

Guez, M., Hildingsson, C., Nascic, S., & Toolanen, G. 2006, Chronic low back pain in individuals with chronic neck pain of traumatic and non-traumatic origin., Acta Orthapaedica, vol. 77, no. 1, pp. 132-137.

Guez, M., Hildingsson, C., Stegmay, B., & Toolanen, G. 2003, Chronic neck pain of traumatic and non-traumatic origin. A population based study., Acta Orthop Scand, vol. 74, no. 5, pp. 576-579.

Guyton, A. C. 1991a, Membrane potentials and action potentials., in Textbook of Medical Physiology, 8th edn, A. C. Guyton, ed., W.B. Saunders Company, Philadelphia, pp. 37-66.

Guyton, A. C. 1991d, Somatic sensations II. Pain, headache, and thermal sensations., in Textbook of medical physiology, 8th edn, A. C. Guyton, ed., W.B. Saunders Company, Philadelphia, pp. 520-531.

Guyton, A. C. 1991b, Motor functions of the spinal cord; the cord reflexes., in Textbook of medical physiology, 8th edn, A. C. Guyton, ed., W.B. Saunders, Philadelphia, pp. 590-602.

Guyton, A. C. 1991c, Muscle blood flow and cardiac output during exercise; the coronary circulation; and ischaemic heart disease., in Medical Physiology, 8th edn, W.B. Saunders, Philadelphia, pp. 234-244.

Hägg, G. M. 1991, Comparison of different estimators of electromyographic spectral shifts during work when applied on short test contractions., Medical and Biological Computing and Engineering, vol. 29, no. 5, pp. 511-516.

Hansen, J., Sander M., Hald, C.F., Victor R.G., Thomas, G.D. 2000, Metabolic modulation of sympathetic vasoconstriction in human skeletal muscle: Role of tissue hypoxia., Journal of Physiology, vol. 527, no. 2, pp. 387-396.

Hasenbring, M. I. 2000, Attentional control of pain and the process of chronification., Progress in Pain Research, vol. 129, pp. 525-534.

Hasenbring, M. I., Hallner, D., & Rusu, A. C. 2009, Fear-avoidance and endurance related responses to pain: Development and validation of the Avoidance Endurance Questionnaire., European Journal of Pain, vol. 13, pp. 620-628.

Henneman, E., Somjen, G., & Carpenter, D. O. 1965, Functional significance of cell size in spinal motoneurons., Journal of Neurophysiology, vol. 28, pp. 560-580.

Herbert, R.D., de Noronha, M. 2007, Stretching to prevent muscle soreness after exercise., Cochrane Database Systematic Review, vol 17, no. 4: CD004577.

Hermens, H. J., & Freriks, B. 1999, European recommendations for surface electromyography. Results of the SENIAM (Surface EMG for Non-Invasive Assessment of Muscles,. 1st edn.

Herrero, J. F., Laird, J. M. A., & Lopez-Garcia, J. A. 2000, Wind up of spinal cord neurones and pain sensation: much ado about something?, Progress in Biology, vol. 61, pp. 169-203.

Hodges, P. W., Moseley, G. L., Gabrielsson, A., & Gandevia, S. C. 2003, Experimental muscle pain changes feedforward postural responses of the trunk muscles., Experimental Brain Research, vol. 151, pp. 262-271.

Hodges, P. W. & Richardson, C. A. 1999, Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds., Arch Phys Med Rehabil, vol. 80, pp. 1005-1012.

Hollen, P. J., Gralla, R. J., Kris, M. G., McCoy, S., Donaldson, G. W., & Moinpour, C. M. 2005, A comparison of visual analogue and numerical rating scale formats for the Lung Cancer Symptom Scale (LCSS): does format affect patient ratings of symptoms and quality of life?, Quality of Life Research, vol. 14, pp. 837-847.

Holm, L. W., Carroll, L. J., Cassidy, J. D., Hogg-Johnson, S., Cote, P., Guzman, J., Peloso, P., Nordin, M., Hurwitz, E., van der Velde, G., Carragee, E., & Haldeman, S. 2009, The burden and determinants of neck pain in whiplash-associated disorders after traffic collisions: results of the Bone and Joint Decade 2000-2010 Task Force on Neck Pain and Its Associated Disorders., Journal of Manipulative and Physiological Therapeutics, vol. 32, no. 2(supp), pp. S61-69.

Houtman, C. J., Stegeman, D. F., Van Dijk, J. P., & Zwarts, M. J. 2003, Changes in muscle fibre conduction velocity indicate recruitment of distinct motor unit populations., Journal of Applied Physiology, vol. 95, pp. 1045-1054.

Huether, S. & Leo, J. 2002, Pain, temperature regulation, sleep, and sensory function., in Pathophysiology: The biologic basis for disease in adults and children, 4th edn, K. L. McChance & S. E. Huether, eds., Mosby, St.Louis, pp. 401-431.

Huijing, P. 1997, Muscle as a collagen fibre reinforced composite: A review of force transmission in muscle and whole limb., Journal of Biomechanics, vol. 32, pp. 329-345.

Hummel, A., Laubli, T., Pozzo, M., Schenk, P., Spillmann, S., & Klipstein, A. 2005, Relationship between perceived exertion and mean power frequency of the EMG signal from the upper trapezius muscle during isometric shoulder elevation., European Journal of Applied Physiology, vol. 95, no. 4, pp. 321-326.

Isacsson, A., Hansson, B. S., Ranstam, J., Ranstam, L., & Isacsson, S. O. 1995, Social network, social support and the prevalence of neck and low back pain after retirement: a population study of men born in 1914 in Malmo, Sweden., Scan J Soc Med, vol. 23, pp. 17-22.

Janda, V. 1988, Muscle and cervicogenic pain syndromes., in Clinics in Physical Therapy V, Physical therapy of the cervical and thoracic spine, Churchill Livingstone.

Jackman, R.W., Kandarian S.C. 2004, The molecular basis of skeletal muscle atrophy., Invited Review. Am J Physiol Cell Physiol vol. 287: C834–C843.

Johansen, M. K., Graven-Nielsen, T., Olesen, A. s., & Arendt-Nielsen, L. 1999, Generalised muscle hyperalgesia in chronic whiplash syndrome., Pain, vol. 83, no. 2, pp. 229-334.

Johansson, H. & Sojka, P. 1991, Pathophysiological mechanisms involved in genesis and spread of muscular tension in occupational muscle pain and in chronic musculoskeletal syndromes. A hypothesis., Medical Hypotheses, vol. 35, pp. 196-203.

Johnston, V., Jull, G., Darnell, R., Jimmieson, N. L., & Souvlis, T. 2008a, Alterations in cervical muscle activity in functional and stressful tasks in female office workers with neck pain., European Journal of Applied Physiology, vol. 103, pp. 253-264.

Johnston, V., Jull, G., Souvlis, T., & Jimmieson, N. L. 2008b, Neck movement and muscle activity characteristics in female office workers with neck pain., Spine, vol. 33, no. 5, pp. 555-563.

Jones, D., Round, J., & de Haan, A. 2004, Introduction to fatigue., in Skeletal muscle from molecules to movement, 4th edn, D. Jones, J. Round, & A. de Haan, eds., Churchill Livingstone, Philadelphia, pp. 91-104.
Jonsson, H., Bring, G., & Rauschning, W. 1994, Hidden cervical spine injuries in traffic accident victims with skull fractures., Journal of Spinal Disorders, vol. 4, pp. 251-263.

Jull, G., Kristjansson, E., & Dall'Alba, P. 2004, Impairment in the cervical flexors: a comparison of whiplash and insidious onset neck pain patients., Manual Therapy, vol. 9, pp. 89-94.

Jull, G. & Sterling, M. 2009, Bring back the biopsychosocial model for neck pain disorders., Manual Therapy, vol. 14, pp. 117-118.

Kallenberg, L. A. C. 2007, Multichannel array EMG in chronic neck-shoulder pain., Roessingh Research and Development, University of Twente.

Kallenberg, L. A. C. & Hermens, H. J. 2006, Motor unit action potential rate and motor unit action potential shape properties in subjects with work-related chronic pain., European Journal of Applied Physiology, vol. 96, no. 2, pp. 203-208.

Kallenberg, L.A.C. & Hermens, H.J. 2008, Behaviour of a surface EMG based measure for motor control: Motor unit action potential rate in relation to force and muscle fatigue. Journal of Electromyography and Kinesiology, vol. 18 no. 5. pp. 780-788. I

Kallenberg, L.A.C., Preece, S., Nester, C., & Hermens, H.J. 2009, Reproducibility of MUAP properties in array surface EMG recordings of the upper trapezius and sternocleidomastoid muscle. Journal of Electromyography and Kinesiology, vol. 19 no. 6, pp 536-542.

Kallenberg, L. A. C., Schulte, E., Disselhorst-Klug, C., & Hermens, H. J. 2007, Myoelectric manifestations of fatigue at low contraction levels in subjects with and without chronic pain., Journal of Electromyography and Kinesiology, vol. 96, pp. 203-208.

Kasch, H., Qerama, E., Kongstead, A., Bach, F. W., Bendix, T., & Jensen, T. S. 2008, Deep muscle pain; Tender points and recovery in acute whiplash patients. A 1-year follow-up study., Pain, vol. 140, pp. 65-73.

Kerns, R. D., Thorn, B. E., & Dixon, K. E. 2006, Psychological treatments of persistent pain: An introduction., Journal of Clinical Psychology, vol. In Press.

Klein, G. N., Mannion, A. F., Panjabi, M. M., & Dvorak, J. 2001, Trapped in the neutral zone: another symptom of whiplash-associated disorder?, European Spine Journal, vol. 10, pp. 141-148.

Kongstead, A., Bendix, T., Qerama, E., Kasch, H., Bach, F. W., Korsholm, L., & Jensen, T. S. 2008, Acute stress response and recovery after whiplash injuries. A one-year prospective study., European Journal of Pain, vol. 12, no. 4, pp. 455-463.

Kristjansson, E., Dall'Alba, P., & Jull, G. 2003a, A study of five cervicocephalic relocation tests in three different subject groups, Clinical Rehabilitation, vol. 17, pp. 768-774.

Kristjansson, E. & Jonsson, E. 2002, Is the sagittal configuration of the cervical spine changed in women with chronic whiplash syndrome? A computer-assisted radiographic assessment., Journal of Manipulative and Physiological Therapeutics, vol. 25, no. 9, pp. 550-555.

Kristjansson, E., Leivseth, G., Brinckmann, P., & Frobin, W. 2003b, Increased sagittal plane segmental motion in the lower cervical spine in women with chronic whiplash associated disorders, grades I-II., Spine, vol. 28, no. 19, pp. 2215-2221.

Kupa, E. J., Roy, S. H., Kandarian, S. C., & De Luca, C. J. 1995, Effects of muscle fibre type and size on EMG median frequency and conduction velocity., Journal of Applied Physiology, vol. 79, no. 1, pp. 23-32.

Lieber, R. L., Thornell, L. E., & Friden, J. 1996, Muscle cytoskeletal disruption occurs within the first 15 minutes of cyclic eccentric contraction., Journal of Applied Physiology, vol. 80, no. 1, pp. 278-284.

Linton, S. J. & Ryberg, M. 2001, A cognitive-behavioral group intervention as prevention for persistent neck and back pain in a non-patient population: a randomized controlled trial., Pain, vol. 90, no. 1-2, pp. 83-90.

Lord, S., Barnsley, L., Wallis, B. J., & Bogduk, N. 1996, Chronic cervical zygapophesial joint pain after whiplash; A placebo-controlled prevalence study., Spine, vol. 21, no. 15, pp. 1737-1744.

Lund, J. P., Donga, R., Widmer, C. G., & Stohler, C. S. 1991, The pain adaptation model: A discussion of the relationship between chronic musculoskeletal pain and motor activity., Can J Physiol Pharmacol, vol. 69, no. 5, pp. 683-694.

Lynn, B. 2006, Repetitive strain injury., in Wall and Melzaks Textbook of Pain, 5th edn, P. D. Wall & R. Melzack, eds., Elseiver Churchill Livingstone, China, pp. 709-718.

Madeleine, P., Farina, D., Merletti, R., & Arendt-Nielsen, L. 2002, Upper trapezius muscle mechanomyographic and electromyographic activity in humans during low force and non-fatiguing contractions., European Journal of Applied Physiology, vol. 87, pp. 327-336.

Madeleine, P., Lundager, B., Voigt, M., & Arendt-Nielsen, L. 1998, Sensory manifestations in experimental and work-related chronic neck-shoulder pain., European Journal of Pain, vol. 2, pp. 251-260.

Madeleine, P., Lundager, B., Voigt, M., & Arendt-Nielsen, L. 1999, Shoulder muscle co-ordination during chronic and acute experimental neck-shoulder pain. An occupational pain study. European Journal of Applied Physiology, vol. 79, pp. 127-140.

Magnusson, M. L., Pope, M. H., Hasselquist, L., Bolte, K. M., Ross, M., Goel, V. K., Lee, J. S., Spratt, K., Clark, C. R., & Wilder, D. G. 1999, Cervical electromyography activity during low speed rear impact., European Spine Journal, vol. 8, pp. 118-125.

Malanga, G. A. & Nadler, S. F. 2002, Whiplash. Hanley and Belfus, Philadelphia.

Marshall, P. D., O'Connor, M., & Hodgkinson, J. P. 1995, The perceived relationship between neck symptoms and precedent injury., Injury, vol. 26, pp. 17-19.

McLean, S. & Clauw, D. J. 2005, Biomedical models of fibromyalgia., Disability and Rehabilitation, vol. 27, no. 12, pp. 659-665.

McLean, S., Williams, D. A., & Clauw, D. J. 2005, Fibromyalgia after motor vehicle collision: Evidence and implications., Traffic Injury Prevention, vol. 6, pp. 97-104.

McMahon, S. B. & Jones, N. G. 2004, Plasticity of pain signalling: Role of neurotrophic factors exemplified by acid induced pain., Journal of Neurobiology, vol. 61, no. 1, pp. 72-84.

Melzack, R. & Katz, J. 2006, Pain assessment in adult patients., in Wall and Melzack's Textbook of Pain, 5th edn, S. B. McMahon & M. Koltzenburg, eds., Elsevier Churchill Livingstone, China, pp. 291-304.

Merletti, R. 2005, Surface EMG generation and detection., International Summer School on non invasive electromyography. Moncalieri (Torino, Italy).

Merletti, R., Farina, D., & Gazzoni, M. 2003, The linear electrode array: A useful tool with many applications., Journal of Electromyography and Kinesiology, vol. 13, no. 1, pp. 37-47.

Merletti, R., Knaflitz, M., & De Luca, C. J. 1990, Myoelectric manifestations of fatigue in voluntary and electrically elicited contractions., Journal of Applied Physiology, vol. 69, pp. 1810-1820.

Merletti, R., & Parker, P. A. 2004, Electromyography: Physiology, Engineering and Non-Invasive applications. IEEE Press, John Wiley and Sons, USA.

Merskey, H. & Bogduk, N. 1994, Classification of chronic pain. Descriptions of chronic pain syndromes and definitions of pain terms., IASP Press, Seattle.

Meyer, R. A., Ringkamp, M., Campbell, J. N., & Raja, S. N. 2006, Peripheral mechanisms of cutaneous nociception., in Wall and Melzak's Textbook of Pain, 5th edn, S. B. McMahon & M. Koltzenburg, eds., Elsevier, China, pp. 3-34.

Michael-Titus, A., Revest, P., & Shortland, P. 2007, Motor systems 1: Descending pathways and cerebellum., in The Nervous System Basic Science and Clinical Conditions, 1st edn, A. Michael-titus, P. Revest, & P. Shortland, eds., Churchill Livingstone, Europe, pp. 171-194.

Miles, T. S. 1994, The control of human motor units., Clinical and Experimental Pharmacology and Physiology, vol. 21, pp. 511-520.

Moore, A., Jackson, A., Jordan, J., Hammersley, S., Mercer, C., Smith, C., Thompson, J., Woby, S., & Hudson, A. 2005, Clinical guidelines for the physiotherapy management of whiplash associated disorder., Chartered Society of Physiotherapy, London.

Morgan, J. E. & Partridge, T. A. 2003, Muscle satellite cells., The International Journal of Biochemistry and Cell Biology, vol. 35, pp. 1151-1156.

Musacchia, X. J., Steffen, J. M. and Fell, R. D. (1988). Disuse atrophy of skeletal muscle: animal models. *Exercise and Sport Science. Review.* 16, 61-87.

Nachemson, A. L. & Jonsson, E. 2000, Neck and Back Pain: The scientific evidence of causes, diagnosis and treatment., Lippincott, Williams and Wilkins, Philadelphia.

Nederhand, M. J., Hermens, H. J., Ijzerman, M. J., Groothuis, K. G. M., & Turk, D. C. 2006, The effect of fear of movement on muscle activation in posttraumatic neck pain disability., Clinical Journal of Pain, vol. 22, pp. 519-525.

Nederhand, M. J., Hermens, H. J., Ijzerman, M. J., Turk, D. C., & Zivold, G. 2002, Cervical muscle dysfunction in the chronic whiplash-associated disorder grade 2, Spine, vol. 10, pp. 1056-1061.
Nederhand, M. J., Hermens, H. J., Ijzerman, M. J., Turk, D. C., & Zivold, G. 2000, Cervical muscle dysfunction in the chronic whiplash-associated disorder grade II (WAD II), Spine, vol. 25, pp. 1938-1943.

Nederhand, M. J., Hermens, H. J., Ijzerman, M. J., Turk, D. C., & Zivold, G. 2003, Chronic neck pain disability due to an acute neck pain injury., Pain no. 102, pp. 63-71.

Nederhand, M. J., Ijzerman, M. J., Hermens, H. J., Turk, D. C., & Zivold, G. 2004, Predictive value of fear avoidance in developing chronic neck pain disability: Consequences for clinical decision making., Arch Phys Med Rehabil, vol. 85, pp. 496-501.

Newell, R. L. M. 2008, Spinal cord and spinal nerves., in Gray's Anatomy, 40th edn, S. Standring, ed., Churchill Livingstone, Elsevier, Spain, pp. 749-763.

Nilsen, K. B., Westgaard, R. H., Stovner, L. J., Helde, G., Ro, M., & Sand, T. H. 2006, Pain induced by low-grade stress in patients with fibromyalgia and chronic neck/shoulder pain, relation to surface electromyography., European Journal of Pain, vol. 10, pp. 615-627.

Nordander, C., Hansson, G., Rylander, L., Asterland, P., Bystrom, J. U., Ohlsson, K., Balogh, I., & Skerfving, S. 2000, Muscular rest and gap frequency as EMG measures of

physical exposure: the impact of work tasks and individual related factors., Ergonomics, vol. 43, no. 11, pp. 1904-1919.

Obelieniene, D., Schrader, H., Bovim, G., Miseviciene, I., & Sand, T. 1999, Pain after whiplash: A prospective controlled inception cohort study., J Neurol Neurosurg Psychiatry, vol. 66, pp. 279-283.

Partheni, M., Miliaris, G., & Constantayannis, C. 1999, Whiplash Injury [Letter]., Journal of Rheumatology, vol. 26, pp. 1206-1207.

Peebles, J. E., McWilliams, L. A., & MacLennan, R. 2001, A comparison of symptom Checklist 90 - Revised profiles from patients with chronic pain from whiplash and patients with other musculoskeletal injuries., Spine, vol. 7, pp. 766-770.

Proske, U. & Morgan, D. L. 2001, Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical application., Journal of Physiology, vol. 537, no. 2, pp. 333-345.

Research, D. S. S. Researchers toolkit.
http://www.dssresearch.com/toolkit/sscalc/size_a2.asp. Date accessed: June 2006.

Riedel, W. & Neeck, G. 2001, Nociception, pain and antinociception: current concepts., Zeitschrift fur Rheumatologie, vol. 60, no. 404, p. 415.
Rosenquist, S. & Rook, J. L. 2003, Whiplash injuries., Butterworth-Heinemann, Philadelphia.

Salmons, S. 1995, Muscle., in Gray' Anatomy, 38th edn, L. H. Bannister et al., eds., Churchill Livingstone, New York, pp. 737-900.
Sandsjo, L., Melin, B., Rissen, D., Dohns, I., & lundberg, P. 2000, Trapezius muscle activity, neck and shoulder pain, and subjective experiences during monotonous work in women., European Journal of Applied Physiology, vol. 83, pp. 235-238.

Scholton-Peeters, G. G. M., Verhagen, A. P., Bekkering, G. E., van der Windt, D. A. W. M., Barnsley, L., Oostendorp, R. A. B., & Hendriks, E. J. M. 2003, Prognostic factors of whiplash-associated disorders: a systemic review of prospective cohort studies., Pain, vol. 104, no. (1-2), pp. 303-332.

Schrader, H., Obelieniene, D., Bovim, G., Surkiene, D., & Sand, T. 1996, Natural evolution of late whiplash syndrome outside the mediological context., Lancet, vol. 347, pp. 1207-1211.

Schulte, E., Ciubotariu, A., Arendt-Nielsen, L., Disselhorst-Klug, C., Rau, G., & Graven-Nielsen, T. 2004, Experimental muscle pain increases trapezius muscle activity during sustained isometric contraction of arm muscles., Clinical Neurophysiology, vol. 115, pp. 1767-1778.

Schwartz, C., Davidson, G., Seaton, A., & Tebbit, V. 1988, Chambers English Dictionary., 7th edn, W & R Chambers, Edinburgh.

Scott, D., Jull, G., & Sterling, M. 2005, Widespread sensory hypersensitivity is a feature of chronic whiplash-associated disorder but not chronic idiopathic neck pain., Clinical Journal of Pain, vol. 21, no. 2, pp. 175-181.

Shah, J., Phillips, T. M., Danoff, J. V., & Gerber, L. H. 2005, An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle., Journal of Applied Physiology, vol. 99, no. 5, pp. 1977-1984.

Simoneau, J. A., Bouchard, C. 1989, Human variation in skeletal muscle fiber-type proportion and enzyme activities. American Journal of Physiology, Endocrinology and Metabolism, 257: E567-E572

Sjolander, P., Michaelson, P., Jaric, S., & Djupsjobacka, M. 2008, Sensorimotor disturbances in chronic neck pain - Range of motion, peak velocity, smoothness of movement and repositioning accuracy. Manual Therapy, vol. 13, no. 2, pp. 122-131.

Soderland, A. & Lundberg, P. 2003, Whiplash-associated disorders-predicting disability from a process-orientated perspective of coping., Clinical Rehabilitation, vol. 17, no. 1, pp. 101-107.

Soderland, A., Olerud, C., & Lindberg, P. 2000, Acute whiplash-associated disorders (WAD): the effects of early mobilization and prognostic factors in long-term symptomatology., Clinical Rehabilitation, vol. 14, pp. 457-467.

Sommerich, C. M., Joines, S. M. B., Hermans, V., & Moon, S. D. 2000, Use of surface electromyography to estimate neck muscle activity., Journal of Electromyography and Kinesiology, vol. 10, no. 6, pp. 377-398.

Spitzer, W. O., Skorvron, M. L., Salmi, L. R., Cassidy, J. D., Duranceau, J., Suissa, S., & Zeiss, E. 1995, Scientific monograph of the Quebec Task Force on whiplash-associated disorders: redefining "whiplash" and its management, Spine, vol. 20, pp. S1-73.

Standring, S. 2008, Head and Neck., in Gray's Anatomy: The anatomical basis of clinical practice, S. Standring, ed., Churchill Livingstone, Elsevier, Spain, pp. 395-705.

Stapley, P. J., Beretta, M. V., Toffola, E. D., & Scheippati, M. 2006, Neck muscle fatigue and postural control in patients with whiplash injury., Clinical Neurophysiology, vol. 117, no. 3, pp. 610-622.

Staron, R. S., Hagerman, F.C., Hikida, R.S., Murray, T.F., Hostler, D.P., Crill, M.T., Ragg, K.E., Toma, K. 2000, The Fiber Type Composition of the Vastus Lateralis Muscle of Young Men and Women ., Journal of Histochemistry and Cytochemistry, Vol. 48, 623-630.

Staud, R., Robinson, M. E., & Price, D. D. 2005, Isometric exercise has opposite effects on central pain mechanisms in fibromyalgia patients compared to normal controls., Pain, vol. 118, pp. 176-184.

Stegeman, D. F., Blok, J. H., Hermens, H. J., & Roeleveld, K. 2000, Surface EMG models: properties and applications., Journal of Electromyography and Kinesiology, vol. 10, pp. 313-326.

Sterling, M., Jull, G., & Kenardy, J. 2006, Physical and psychological factors maintain long-term predictive capacity post-whiplash injury., Pain, vol. 114, pp. 141-148.

Sterling, M., Jull, G., Vincenzino, B., & Kenardy, J. 2003a, Sensory hypersensitivity occurs soon after whiplash injury and is associated with poor recovery., Pain, vol. 104, no. 3, pp. 409-517.

Sterling, M., Jull, G., Vincenzino, B., Kennardy, J., & Darnell, R. 2003b, Development of motor system dysfunction following whiplash injury., Pain, vol. 103, pp. 65-73.

Sterling, M., Kenardy, J., Jull, G., & Vicenzino, B. 2003c, The development of psychological changes following whiplash injury., Pain, vol. 106, no. 3, p. 481.

Sterling, M., Vicenzino, B., Kenardy, J., & Darnell, R. 2005, Physical and psychological factors predict outcome following whiplash injury., Pain, vol. 114, no. 1-2, pp. 141-148.

Suissa, S., Harder, S., & Veilleux, M. 2001, The relationship between initial symptoms and signs and the prognosis of whiplash., European Spine Journal, vol. 10, no. 44, p. 49.

Taylor, J. L., Butler, J. E., & Gandevia, S. C. 2000, Changes in muscle afferents, motoneurones and motor drive during muscle fatigue., European Journal of Applied Physiology, vol. 83, pp. 106-115.

Taylor, J. L. & Taylor, M. 1996, Cervical spine injuries: An autopsy study of 109 blunt injuries., Pain, vol. 4, pp. 61-79.

Thompson, D. T. Electromyography (EMG). http://moon.ouhsc.edu/dthompso/pk/emg/emg.htm#processing. 2-3-1998. 29-6-2007.

Tominaga, Y., Ndu, A. B., Coe, M. P., Valenson, A. J., Ivancic, P. C., Ito, S., Rubin, W., & Panjabi, M. M. 2006, Neck ligament strength is decreased following whiplash trauma., BMC Musculoskeletal Disorders, vol. 7, pp. 103-112.

Travell, J. G. & Simons, D. G. 1983, Background and principles., in Myofascial Pain and Dysfunction. The trigger point manual. The upper extremities., 1st edn, J. G. Travell & D. G. Simons, eds., Williams and Wilkins, Baltimore, pp. 5-45.

Uhlig, Y., Weber, B. R., Grob, D., & Muntener, M. 1995, Fibre compositions and fibre transformations in neck muscles of patients with dysfunction of the cervical spine., Journal of Orthopaedic Research, vol. 13, pp. 240-249.

Ustun, T. B. Towards a Common Language for Functioning Disability and Health. ICF . 2007.

Van Dieen, J. H., Selen, L. P. J., & Cholewicki, J. 2003, Trunk muscle activation in low-back pain patients, an analysis of the literature., Journal of Electromyography and Kinesiology, vol. 13, no. 4, pp. 331-351.

Vasavada, A. N., Brault, J. R., & Siegmund, G. P. 2007, Musculotendon and fasicle strains in anterior and posterior neck muscles during whiplash injury., Spine, vol. 32, no. 7, pp. 756-765.

Verhaaka, P. F. M., Kerssensa, J. J., Dekkera, J., Sorbib, M. J., & Besinga, J. M. 1998, Prevalence of chronic benign pain disorder among adults: a review of the literature., Pain, vol. 77, pp. 231-239.

Vernon, H. & Mior, S. 1999, The neck disability index: A study of reliability and validity., Journal of Manipulative and Physiological Therapeutics, vol. 14, no. 7, pp. 101-107.

Visser, B. & van Dieen, J. H. 2006, Pathophysiology of upper extremity muscle disorders., Journal of Electromyography and Kinesiology, vol. 16, pp. 1-16.

Vitti, M., Fujiwara, M., Basmajian, J. V., & Iida, M. 1973, The integrated roles of the Longus Colli and Sternocleidomastoid muscles: An electromyographic study., Anatomical Records, vol. 117, no. 4, pp. 471-484.

Vlaeyen, J. W. S. & Linton, S. J. 2006, Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art., Pain, vol. 85, pp. 317-332.

Voerman, G., Sandsjo, L., Vollenbroek-Hutten, M., Larsman, P., Kadefors, R., & Hermens, H. J. 2007, Effects of ambulant myofeedback training and ergonomic counselling in female computer workers with work-related neck-shoulder complaints: A randomized controlled trial., Journal of Occupational Rehabilitation, vol. 17, pp. 137-152.

Voerman, G., Vollenbroek-Hutten, M., & Hermens, H. J. 2006, Changes in pain, disability, and muscle activation patterns in chronic whiplash patients after ambulant myofeedback training., Clinical Journal of Pain, vol. 22, no. 7, pp. 656-663.

Vollenbroek-Hutten, M., Hermens, H. J., Voerman, G., Sandsjo, L., & Kadefors, R. 2006, Are changes in pain induced by myofeedback training related to changes in muscle activation patterns in patients with work-related myalgia?, European Journal of Applied Physiology, vol. 96, pp. 209-215.

Walker-Bone, K. & Cooper, C. 2005, Hard work never hurt anyone: or did it? A review of occupational associations with soft tissue musculoskeletal disorders of the neck and upper limb., Annals of Rheumatic Disorders, vol. 64, pp. 1391-1396.

Walker-Bone, K., Reading, I., Coggon, D., Cooper, C., & Palmer, K. T. 2004, The anatomical pattern and determinants of pain in the neck and upper limbs: An epidemiological study., Pain, vol. 109, pp. 45-51.

Wall, P. D. & Melzack, R. 1994, Textbook of Pain, 3rd edn, Churchill Livingstone, London.

Weber, B. R., Uhlig, Y., Grob, D., Dvorak, J., & Muntener, M. 1993, Duration of pain and muscular adaptations in patients with dysfunction of the cervical spine., Journal of Orthopaedic Research, vol. 11, pp. 805-810.

WHO 1985, Identification and control of work related disease, WHO, Geneva, 714.

Wigley, C. 2008, Cells, tissues and systems., in Gray's Anatomy: The anatomical basis of clinical practice, 40 edn, S. Standring, ed., Churchill Livingstone, Elsevier, Spain, pp. 1-145.

Willis, W. D. 1999, Dorsal root potentials and dorsal root reflexes: a double-edged sword., Experimental Brain Research, vol. 124, pp. 395-421.

Winter, D. A., Fuglevand, A. J., Archer S. E., 1994, Crosstalk in surface electromyography: Theoretical and practical estimates., Journal of Electromyography and Kinesiology, vol. 4, pp. 15-26.

Woodhouse, A. & Vasseljen, O. 2008, Altered motor control patterns in whiplash and chronic neck pain., BMC Musculoskeletal Disorders, vol. 20, no. 9, pp. 90-99.

Wyke, B. D. 1987, The lumbar spine and back pain Churchill Livingstone, Edinburgh.

Yassi, A. 1997, Repetitive Strain Injuries., Occupational Medicine, vol. 349, pp. 943-947.

Yoganandan, N. & Pintar, F. A. 2003, Biomechanics of human head-neck in rear impacts., International Journal of Vehicle Design, vol. 32, no. 1/2, pp. 53-67.

Zedka, M., Prochazka, A., Knight, B., Gillard, D., & Gauthier, M. 1999, Voluntary and reflex control of human back muscles during induced pain., Journal of Physiology, vol. 520.2, pp. 591-604.

Zwarts, M. J., Bleijenberg, G., & van Englen, B. G. M. 2008, Clinical neurophysiology of fatigue., Clinical Neurophysiology, vol. 119, pp. 2-10.

Zwarts, M. J. & Stegeman, D. F. 2003, Multichannel surface EMG: basic aspects and clinical utility., Muscle and Nerve, vol. 28, no. 1, pp. 1-17.