# The Effect of Chronic Neck Pain and Training on Directional Specificity of Neck Muscle Activation

# The Effect of Chronic Neck Pain and Training on Directional Specificity of Neck Muscle Activation

**PhD Thesis by**

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# Indhold

## SHORT ABSTRACT

This thesis examines the neural control of selected neck muscles in patients with chronic neck pain, and further examines if a specific training intervention can improve the neural control of neck muscle activation. The thesis includes four extracts from four studies: 1) the directional specificity of the sternocleidomastoid and splenius capitis muscles activity in patients with chronic neck pain and healthy controls, 2) the neural drive to the sternocleidomastoid by evaluating the behavior of single motor units during isometric contractions in the horizontal plane in patients with neck pain and healthy controls, 3) the influence of reduced sternocleidomastoid directional specificity on neck strength, and 4) the effect of a specific training intervention on the directional specificity of neck muscle activity in patients with chronic neck pain. The studies revealed that patients with neck pain display reduced directional specificity of neck muscle activity and that the directional specificity of neck muscle activity can be enhanced by specific training. Taken together these findings contribute to the evidence on impaired neuromuscular control of the cervical spine in patients with neck pain and further demonstrate a method for management of impaired motor function in patients with neck pain.

## DANSK ABSTRACT

This thesis examines neural control of neck muscles in patients with chronic neck pain by assessing the directional specificity of neck muscle activity via tuning curves of the electromyographic (EMG) signal on polar plots. EMG tuning curves, which depict muscle activity over a range of force or moment directions, have been used to study activation strategies of the neck muscles in healthy individuals. When tuning curves are consistent among subjects, analyzing the orientation and focus (mean direction and spread of EMG activity, respectively) of EMG tuning curves in relation to musculoskeletal mechanics provide insight into central nervous system control. For example, EMG tuning curves of neck muscles can be recorded by having a subject perform contractions at a predefined force (e.g. 15N of force) with continuous change in force direction in the range 0-360º in the horizontal plane. The EMG amplitude as a function of the angle of force direction can be referred to as directional activation curves. The directional activation curves represent the modulation in intensity of muscle activity with the direction of force exertion and represent a closed area when expressed in polar coordinates. The line connecting the origin with the central point of this area defines a directional vector, whose length is expressed as a percent of the mean EMG amplitude during the entire circular task. This normalized vector length represents the specificity of muscle activation (Directional Specificity): it is equal to zero if the muscle is active in the same way in all directions and, conversely, it corresponds to 100% if the muscle is active in exclusively one direction. In healthy subjects, neck muscles show consistent and well-defined preferred directions of activation, which are in accordance with their anatomical position relative to the spine.

This thesis examines the directional specificity of selected neck muscles in patients with chronic neck pain, and further examines the effect of specific training on the directional specificity of neck muscle activity. Four Studies were conducted which assessed 1) the directional specificity of the sternocleidomastoid and splenius capitis muscles activity in patients with chronic neck pain and healthy controls, 2) the neural drive to the sternocleidomastoid by evaluating the behavior of single motor units during isometric contractions in the horizontal plane in patients with neck pain and healthy controls, 3) the influence of reduced sternocleidomastoid directional specificity on neck strength, and 4) the effect of a specific training intervention on the directional specificity of neck muscle activity in patients with chronic neck pain.

The results showed that patients with idiopathic neck pain have reduced specificity of sternocleidomastoid and splenius capitis muscle activity with respect to asymptomatic individuals, and that the reduced specificity of neck muscle activity in patients with neck pain may be linked to a reduced modulation in discharge rate of motor units with force direction indicating a potential change in motor neuron excitability. No correlation was found in the third Study between maximum voluntary contraction and directional specificity; however the average neck strength in patients was moderately and inversely correlated to the pain experienced by the patient during maximal contraction, to fear of movement and to

some aspects of neck disability. Finally in the fourth Study, it was shown that specific training of the neck muscles can enhance directional specificity of neck muscle activity in patients with chronic neck pain. This was the first study to show that training can improve such fine or skilled control of the neck muscles which underlies deficits in movement function in the horizontal plane.

Overall, this thesis has elucidated mechanism which may contribute to impaired neuromuscular control in patients with neck pain. Furthermore the results contribute to the evidence base supporting the efficacy of specific exercise in patients with neck pain.

**ABSTRAKT**

Denne afhandling undersøger nakkemusklernes neurale kontrol hos patienter med kroniske nakkesmerter ved at vurdere retningsspecificiteten af elektromyografi (EMG) signaler fra nakkens muskler. EMG tuningskurver kan vise cirkulær muskel aktivitet, og med disse kurver er aktiveringsstrategier af nakkens muskler hos raske personer blevet undersøgt. Tunings kurver fra raske personer viser ensartet orientering og fokus (gennemsnitlig retning og spredning af EMG aktivitet). Desuden giver tunings kurverne indsigt i centralnervesystemets muskulære kontrol. For eksempel kan EMG tunings kurver fra nakkens muskler optages ved at udføre muskel kontraktioner med en forud defineret kraft og retning f.eks. 15N isometrisk kontraktion med kontinuerlig ændring af kraftretningen fra 0 til 360 ° i det horisontale plan. EMG amplitude kan beregnes som funktion af vinklen for kraftretningen og betegnes som den retningsbestemte muskel aktiverings kurve. Den retningsbestemte aktiverings kurve repræsenterer den retnings og krafts afhængige modulation af muskel aktiviteten og repræsenterer et lukket område, når den afbilledes i et cirkulært koordinatsystem. En linje gennem centrum og periferien i koordinatsystemet defineres som en retningsbestemt vektor, hvis længde er funktionen i procent af den gennemsnitlige EMG amplitude fra den cirkulære muskelaktivitet. Denne normaliserede vektors længde repræsenterer den retningsspecifikke aktivitet i den undersøgte nakke muskel (retnings specificitet), og vektoren er lig med nul, hvis musklen er aktiv på samme måde i alle retninger, og omvendt lig med 100 %, hvis musklen udelukkende er aktiv i én retning. Hos raske personer, viser nakkens muskler en konsistent og veldefineret muskelaktivering, som tilsvarer musklernes funktionelle anatomi.

Denne afhandling undersøgerretnings specificiteten af udvalgte nakkemuskler hos patienter med kroniske nakkesmerter. Desuden undersøger afhandlingen effekten af specifik træning af nakkens muskler på musklernes retnings specificitet. Fire Studier blev udført som vurderede 1) en sammenligning af retnings specificitet fra sternocleidomastoideus og splenius capitis musklerne, optaget hos patienter med kroniske nakkesmerter og raske kontrolpersoner, 2) den neurale kontrol af sternocleidomastoideus gennem undersøgelse af frekvensen af nerve signaler ved isometriske kontraktioner foretaget i det horisontale plan hos patienter med nakkesmerter og raske kontrolpersoner, 3) indflydelsen af nedsat sternocleidomastoideus retningsbestemt specificitet på maksimal frivillig kontraktion af nakkens muskler, og 4) virkningen af et 8 ugers specifikt træningsprogram på retningsspecificitet optaget fra nakkens muskler hos patienter med kroniske nakkesmerter.

Resultaterne viste, at patienter med nakkesmerter har reduceret retningsbestemt specificitet af sternocleidomastoideus og splenius capitis i forhold til raske kontrolpersoner, samt at den reducerede retnings specificitet kan være forbundet med en nedsat modulation i frekvensen af nervesignaler til musklernes motoriske enheder. Dette indikerer at en potentiel ændring i motor neuronernes excitabilitet kan foreligge hos patienter med kroniske nakkesmerter. I det tredje studie blev der ingen sammenhæng fundet mellem maksimal frivillig kontraktion

af nakkens muskler og retnings specificitet; men for patienter med nakkesmerter var gennemsnitlig muskel styrke moderat og omvendt korreleret med den smerte som patienten oplever under maksimal frivillig kontraktion, med frygten for smerte ved bevægelse og med nogle aspekter af nedsat funktion i nakken.

Endelig i den fjerde undersøgelse blev det påvist, at specifik træning af nakkens muskler kan forbedre den retningsbestemte specificitet af musklernes aktivering hos patienter med kroniske nakkesmerter. Dette er den første undersøgelse der påviser, at træning kan forbedre den motoriske kontrol af nakkens muskelfunktion i det horisontale plan.

Samlet set bidrager denne afhandlings resultater til evidensen for nedsat neuromuskulær kontrol af halshvirvelsøjlen hos patienter med nakkesmerter. Endvidere bidrager resultaterne til evidensgrundlaget for effekten af specifik træning på patienter med kroniske nakkesmerter.

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# 1. Introduction

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## 1.1 PREVALENCE AND IMPACT OF NECK PAIN

Neck pain is a common disorder (1). The one year prevalence of neck pain ranges from 16.7% to 75.1% with an average of 37.2% (2). The incidence rate increases with longer observation periods and the prevalence decreases with neck pain lasting longer than 3 months. The incidence rate of neck pain has been reported to be higher in Scandinavian countries compared to the rest of Europe and Asia (2) and women consistently report higher incidence rates than men (2).

Neck pain is an economic burden. The neck pain expenditures in the Netherlands alone were estimated to be 1% of the total healthcare costs and 0.1% of the Gross National Product (3), and similarly the total annual cost of neck and back pain in Sweden estimated to be equivalent to 1% of the gross national product (4).

## 1.2 MOTOR CONTROL OF THE CERVICAL SPINE AND THE INFLUENCE OF NECK PAIN

Motor control of the cervical spine is complex since over 20 pairs of muscles act on seven vertebrae, as well as the head and thorax (5, 6). The stability of the cervical spine is dependent on passive structures, together with coordination of the agonist and antagonist muscles (7). Co-activation of neck muscles has been investigated in the last three centuries (8); however the current evidence is still partial and studies are often limited to the evaluation of neck muscle function and contraction in a single force direction.

The static and dynamic motor control of the cervical spine is dependent on feedforward and feedback mechanisms, proprioception, and integrated exteroception from sensory organs (9, 10). Cervical stability is reported to be compromised in healthy controls during fast movements as the antagonist activity

does not increase relative to the agonist activity (11); however co-contraction is also reported to be increased by other factors not related directly to biomechanical stability such as motor learning (12) and pain (13, 14).

Both experimentally-induced neck pain and clinical neck pain result in altered cervical motor control strategies (10). However it remains unclear to which extent motor control is altered by pain alone or is influenced by underlying pathologies. Often the etiology behind neck pain is illusive and may be multifactorial (15). Demographics, lack of threshold values and the lack of gold standards cause much conjecture with regard to the cause and effect of neck pain (16). However, there is accumulating evidence for an association between chronic neck pain and dysfunction of the muscles of the cervical spine (17, 18). Neck pain has been associated with neuromuscular impairment including changes in muscle structure such as cross sectional area, fatty infiltration (19) and altered fiber type (20). Likewise is cervical motor control altered with chronic neck pain, these alterations include impaired coordination between the deep and superficial neck muscles (18) and delayed activity of neck muscles in response to perturbations (21, 22).

## 1.3 NEUROMUSCULAR DYSFUNCTION

### 1.3.1 Impaired motor output

Impaired submaximal and maximal (23-26) motor output have been reported in patients with neck pain. Maximum voluntary contraction (MVC) is reported reduced from 13.3% (27) to 90% (28). Demographics or subjects' daily activity may influence these observations of reduced motor output, as a large study of tall active young adult male patients with neck pain found no difference in maximal torque (29).

Reduced MVC may be due to reduced voluntary drive to the muscle and this type of impaired motor output can be demonstrated by superimposed electrical stimulation (30). The reduced output may imply that the neck muscles cannot generate, maintain and sustain the required muscular performance for acuity and smoothness of movements; however it is difficult to substantiate the exact etiology for impaired motor output across multifactorial neurological and structural origins (10, 20, 23, 25, 31-33). Large variation across healthy subjects and a lack of gold standard for measuring reduced neck strength (23, 25, 26, 34) degrade the clinical value of MVC and clinically relevant strength loss may best be established by persistent very low neck strength.

Impairment in submaximal motor output has also been demonstrated by observations that chronic neck pain patients display reduced endurance, decreased force steadiness (35) and decreased neuromuscular efficiency (35, 36). Large variability of flexion endurance times have been demonstrated between healthy controls (14.5 -95.7 s) and patients with neck pain (16.6 – 24.1s) (37, 38).

### 1.3.2 Pattern of neck muscle activity

Evidence from experimental and clinical neck pain studies indicate that neck pain may alter patterns of agonist and antagonist neck muscle activity (10, 17). Thus pain does not only change the activity of the painful muscle but rather changes the load sharing between neck muscles (10, 22). Reduced activation of the deep cervical flexors, longus colli and longus capitis has been demonstrated in patients with neck pain compared to healthy controls during performance of an isometric cranio-cervical flexion task (39-41). Decreased focus of muscle activity and decreased electromyography (EMG) amplitude of the deep cervical extensor, semispinalis cervicis, has also been observed in patients with neck pain during isometric contractions (42).

The activity of the superficial flexors such as anterior scalene and sternocleidomastoid (SCM) is reported to be higher in patients with neck pain compared to controls (39, 43, 44). Patients with neck pain also show higher activation of the superficial extensor muscles during a unilateral upper limb task, compared to healthy controls (45).

Neck pain also alters the temporal pattern of neck muscle activity (21). Patients with chronic neck pain have delayed activation of the deep and superficial cervical flexor muscles when performing rapid arm movements (21). Deep and superficial muscles were activated within ~100 ms of the deltoid onset during rapid flexion movements and ~200 ms for rapid arm extension in patients with neck pain, whereas a faster activation was seen in control subjects where the activation occurred within 50 ms (43).

The relation between altered motor control and neck pain cannot be characterized by a linear correlation as it is complex and multifactorial (46); however weak linear correlations have been reported between different aspects of neck pain and motor control. For example, the EMG amplitude of the superficial flexors, SCM and anterior scalenes, showed a weak positive correlation with pain intensity during a craniocervical flexion test (47). Furthermore has neck pain experienced during the past week been correlated with reduced activity of the deep flexor muscles (22).

The delay in onset of the cervical flexors during rapid shoulder flexion has been correlated to measures of neck pain (22) and further SCM muscle activation during repetitive arm movements has been correlated to the level of neck pain and neck disability (43).

### 1.3.3 Changes in motor unit behaviour in response to pain

Single motor unit recordings provide a direct estimate of neural drive to the motor units and muscular force production. Motor unit recordings can be viewed as a product of motor unit recruitment, motor unit firing rate and the intramuscular environment (48). Experimental pain was found to reduce the discharge rate of motor units while maintaining the pre-pain recruitment thresholds (48). Reduced motor unit discharge rates may reduce maximal performance of agonist muscles;

while the submaximal performance may be maintained by recruiting more motor units or shifting the activity to motor units in other muscles (48). Motor units discharge rates increases in pain free and under experimental pain conditions with increased force production; however the interspike interval (ISI) is more irregular at low discharge frequencies (48). The discharge rate of motor units also decreases less during a sustained contraction in painful conditions compared to non-painful conditions (49).

Two well-known and discussed theories have been proposed to help explain the interaction between pain and motor control. One of these theories is the vicious cycle theory and the other is the pain adaptation model. The vicious cycle theory proposes an increased muscle activity as a consequence of the activation of group III and IV muscle afferents (50). The pain-adaptation model predicts an inhibitory effect of pain on motor neurons during agonist activity and an excitatory effect during antagonist action (51). These theories have not given satisfactory explanations for the observed interactions between pain and motor control and a new and more comprehensive theory has been proposed (52) consisting of 5 elements 1) redistribution of activity within and between muscles; 2) changes in the biomechanical behavior such as modified movement and stiffness; 3) changes related to protection from further pain or injury, or from threatened pain or injury; 4) the pain and motor control interactions cannot be explained by simple changes in excitability but involves changes at multiple levels of the motor system, and these changes may be complementary, additive, or competitive; and 5) positive short-term benefit may have potential negative long-term consequences due to factors such as increased load, decreased movement, and decreased variability (52). This new theory appears better suited to explain the adaptations in motor control caused by pain; however the theory does not encompass motor control adaptations caused by etiology, learning and posture.

### 1.3.4 Structural changes occurring in the cervical region

The cervical region is continuously undergoing structural changes driven by growth, repair, regeneration, degeneration and physical activity. Large structural changes in patients with neck pain can be observed with imaging technologies, these structural changes may be the result of continuous cellular remodeling over time, trauma, degeneration and tissue damage. Structural changes in neck muscle include alterations in cross sectional areas (CSA), fat infiltration and muscle fiber transformation. Semispinalis cervicis and multifidus showed decreased CSA in patients with neck pain following a whiplash injury compared to healthy controls (53).

The CSA of the multifidus muscle was also reported to be reduced in patients with chronic non-traumatic neck pain compared to healthy controls (54). On the contrary, fighter pilots with chronic neck pain demonstrated greater CSA of semispinalis cervicis and multifidus compared to asymptomatic fighter pilots (55). Healthy women with low levels of physical activity in their daily life showed larger CSA of the cervical extensors compared to women with higher levels of

physical activity (56). Changes of CSA in the cervical muscles appear to be variable and the significance of these changes in CSA is inconclusive for neck pain.

Increased fatty infiltration of the neck muscles has been associated with whiplash (31) but not with idiopathic neck pain (19). In patients with insidious onset of neck pain fat infiltration has not been identified consistently (19).

Biochemical alterations, such as increased levels of glutamate and serotonin, have been found in the upper trapezius muscles of patients with neck pain. Levels of glutamate and serotonin were positively correlated to pain intensity (57). Muscle fiber transformation from "slow oxidative" to "fast glycolytic" has also been reported for neck patients who underwent spondylodesis (20). Fibre transformation from type I to type II (i.e. from slow to fast twitch fibres) were found in neck muscles of patients with neck pain including suboccipital, splenius capitis (SPL) and trapezius muscles, and these findings were independent of diagnosis (20). The fibre transformation were found to be on-going, as 2-3 years after onset of neck pain a higher amount of transitional type-IIC fibres could be found (20).

## 1.4 NECK EXERCISE AND THE EVIDENCE FOR EFFICACY

Neck interventions have been the focus of over 30 reviews, three of these reviews have specifically addressed exercise therapy; however there is no consensus on how to diagnose, quantify and select patients which may benefit for treatment of neck pain (58, 59). Exercise therapy incorporates a large variety of methods such as mobilizing exercises, stretching, isometric/static or dynamic strengthening, endurance training and proprioceptive exercises (60). The clinician is faced with shortage of evidence which can predict the patient response to an exercise intervention (58, 59). The missing evidence impedes the matching of individual patients to the present evidence, in order to evaluate if exercises can be a relevant part of a forthcoming treatment plan. The matching is difficult as too many distinctions or assumptions are made in current research without matching gold standards (58-61), thus trial and error evaluations become an important part of clinical practice and modern day treatment of neck pain. Variations in demographic, interventions, outcomes, psychosocial effects and preferences across treatments (59), justify the available evidence to be integrated with a large amount of clinical experience, as there is no evidence indicating that a particular resistance type of exercise is superior to the other (58-61).

Exercise is frequently used as a treatment for neck pain. There is moderate evidence for the efficacy of exercise when the exercise is performed alone, however, there is stronger evidence of efficacy when exercise is combined with mobilization or manipulation for subacute and chronic mechanical neck disorders both in the short and long term (61). There is moderate evidence that supports exercise interventions focused on the neck and shoulder region (61). It is unclear if exercise is more effective than other types of treatment (61). The multimodal

approach which incorporates exercise and other interventions is favored by the evidence (59, 61).

The understanding of what triggers remission of symptoms is not clear; however a better understanding is much desired as illustrated by the results from a randomized controlled trial investigating cervicogenic headache with 4 groups: control, manipulative therapy, therapeutic exercise and a combination of the two interventions. The results demonstrated 100% symptom relief for approximately 40% of the combined group, approximately 30% of the non-combined groups and 4% of the control group. The combined therapies gave 100% relief for 25% more headache victims compared to the non-combined therapies, indicating for 75% of patients were the choice of therapies of no consequence for their relief; however also demonstrating that specific indication for treatment may be relevant for 25% of possible 100% responders (62).

Cost benefit analysis may indicate new treatments approaches, which favors easy to apply exercise. A recent study demonstrated that two minutes a day of an exercise intervention was sufficient to make clinically relevant reductions in pain and tenderness after 10 weeks of training (63).

Exercises are not of a universal nature with an equal outcome on all bodily structures, the outcome of an exercise is perceived to have a local effect on the exercised structures (64). However this perception is challenged by clinical practice, where patients often experience benefits of exercises in muscles, structures or areas with no direct relations to the muscles improved by the exercises. This reflects that the physiological basis for understanding adaptations to pain and exercises remains limited (46).

An example of the complexity and multiple mechanisms underlying exercise could be that cervical strength training typically involves near-maximal contractions, thus more likely provides a physical stress for high-threshold motor unit that are infrequently used in daily postural neck activities (64). In contrast, low load exercises may involve the activation of lower threshold motor units involved in postural activities. Fig. 1 depicts examples of three diverse types of neck training.

**Fig 1:** Cervical extension strength training with emphasis on provision of load (Left). Cranio-cervical flexion training with emphasis on activation of the deep cervical flexors (Center), the

patient is given feedback on muscle activation from an air pressure device placed below the neck, and the instructor is, by palpation, controlling for minimal activity of the superficial flexor muscles. Cervical flexion training without provision of additional load and with activity in the superficial flexor muscles (Right).

# 2.Background

## 2.1 NEURAL CONTROL OF THE NECK

### 2.1.1 Neural control of muscle

The muscles are composed of parallel muscle fibers which are bundled into fascicles and the fascicles are further bundled to form the muscle (65). The ends of the muscle fiber are specialized to transmit forces to bony structures through tendons (65). The force production within the muscles fibers is dependent on the contractile proteins actin and myosin (66). The force production and the rate of force production are controlled by the nervous system via the frequency of action potentials sent through motoneurons to the motor units. A motor unit consists of a motor neuron and the muscle fibers innervated by that motor neuron (66). The muscle fibers within a motor unit range from fewer than 10 to more than1000 in large muscles (66). The muscle fibers of a motor unit are dispersed throughout the muscle and most muscles are composed of hundreds of motor units (65).

Muscle fibers are classified by their contraction time into fast twitch or slow twitch fibers. The slow twitch fibers are involved in maintaining low level contractions, while fast twitch fibers are activated when more force is needed. Slower motor units are described to innervate fewer muscle fibers and to contain motoneurons with smaller bodies, thinner axons and to have slower conduction velocities (66). Motor units are recruited in a fixed order during muscle contraction, with the smallest fibers first (Henneman size principle) (66).

The action potential is the basis for transport of information in the nervous system. The action potential materializes due to an ion influx across membranes in the nerve fiber. The action potential is a propagating occurrence along the entire length of the nerve fiber and is a repeatable all or none occurrence (66).

The action potential encodes significant amounts of information by generating sequences of action potentials (66). The topographical organization of the nervous system is so rigid that higher sensory centers can identify the origin of a nerve signal via the termination of the pathway that relays the nerve signal to the higher center.

Sensory and motor information are transmitted to and from central nervous system in continuous pathways. The communication between the periphery and

cortex is transmitted through relay nuclei. Relay nuclei are capable of transmitting and processing incoming signals (66). However the topographical organization of the spinal cord allows little convergence, divergence or processing of signals compared to the higher centers (66).

Delays in nerve signal transmission can have a profound influence on the timing of motor control events (66). The structure of the nervous system reflects the need for transmission speed with high speed signals transmitted in myelinated fibers and slow signals transmitted in unmyelinated fibers. There are several centers for motor control reflexes in the spinal cord; spinal signal processing allows for faster responses because of the shorter distance the nerve signal travels.

While signal transmission and signal speed is well understood, the processing of signals in the spinal cord and higher centers is unclear. However it is well understood, that higher center activation of neck muscles is dependent on muscle synergies, which generates patterns of muscle activation (5, 6, 9). Muscle activity of agonist and antagonist are controlled in pre-programmed patterns, which can be modified through central activity, reflexes, feedback from the locomotive system and nociceptive signals such as pain (66). There are many possible neck muscle activation patterns, as many neck muscles have similar lines of action (5). The origin of these pre-programmed muscle control patterns is not clear; they are presumably learned in infancy (66) and then modified as a result of growth, external or internal factors.

***Electromyography***

The best direct tool for analysis of muscle activation is EMG, which is the recording of action potentials from muscle fibres firing individually or in groups within the recording electrodes' acquisition areas (67). The electrode can be inserted into the muscle via a needle (intramuscular EMG) or placed on the skin (surface EMG).

The resting muscle normally shows no action potentials, however when a sufficiently high level of excitatory synaptic input is received by the motoneuron, it generates an action potential and subsequent contraction. Higher synaptic input to one motoneuron results in an increase in the rate of action potentials, and the EMG signal represents a summation of all signals acquired by the electrodes and the interference pattern of EMG describes the superposition of action potentials from different motor units. The acquired EMG signal is amplified, processed and quantified (67).

The amplitude of interference EMG does not reflect the strength of the recorded muscle fibres as several factors influence the EMG signal. The variable space between the active neuron and the receptive electrode allows several factors to influence the recorded electrical signal like the distance and conductibility of the tissue between the muscle fibre and the electrode, the distribution of the motor unit territories, the recruitment of new motor units over time as a consequence of fatigue, and the size of the muscle fibre (67).

### 2.1.2 Directional specificity of neck muscle activity

An approach to monitor the amplitude of neck muscle activity with EMG is to measure the change in EMG amplitude with EMG tuning curves on polar plots. EMG tuning curves can be constructed to depict the level of muscle activation over a range of directions. Tuning curves have been used to study activation strategies of neck muscle activation in the horizontal plane (5, 6, 68). The result show consistent muscle activation across subjects, when analyzing the orientation and focus (mean direction and spread of EMG activity, respectively; defined below). EMG tuning curves in relation to musculoskeletal mechanics has provided further insight into central nervous system control (5).

EMG tuning curves of the neck muscles can be constructed by recording EMG activity from the neck muscles while the subject performs isometric contractions at predefined force intensity (e.g. 15N of force) with continuous change in force direction in the range 0-360º in the horizontal plane (6).

For the circular contractions, the amplitude of the surface EMG can be estimated. The average rectified value (ARV) of the recorded EMG activity can be expressed as a function of the angle of force direction and is referred to as directional activation curves. The directional activation curves represent the modulation in intensity of muscle activity with the direction of force exertion and represent a closed area when expressed in polar coordinates. The line connecting the origin with the central point of this area defined a directional vector, whose length was expressed as a percent of the mean ARV during the entire task (Fig 2). This normalized vector length represents the directional specificity of muscle activation (Fig 2) and the vector is equal to zero when the EMG amplitude is the same in all directions and, conversely, it corresponds to 100%, when the muscle is active in exclusively one direction (Fig 3) (68).

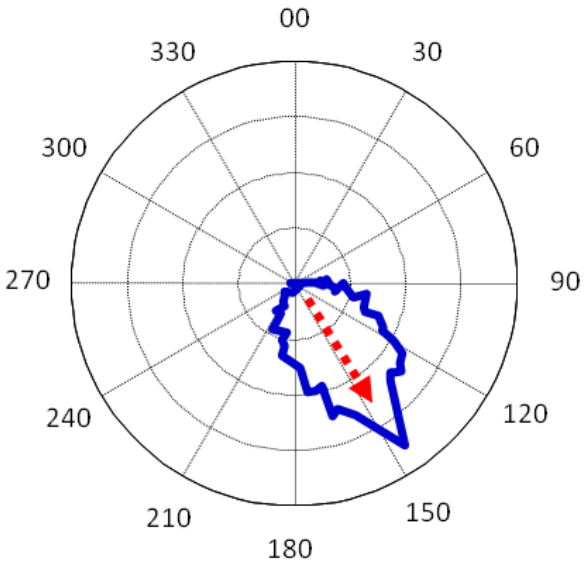


**Fig 2:** Polar plot with EMG tuning curve acquired from left SPL (the blue trace) and the directional specificity vector associated with the EMG tuning curve (orange dotted line).

EMG tuning curves have been constructed for circular isometric contractions of the trapezius, levator scapula, SCM, SPL, and multifidus muscles in healthy subjects. EMG tuning curves show that activation of the neck muscles is consistent and well defined. Muscle activation increases with contraction force and shows a more focused activation pattern (6). This indicates that muscle activation patterns are consistent for similar tasks, however modified by force (6).

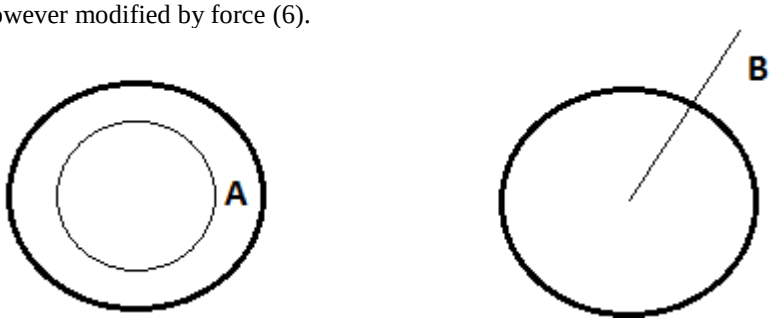


**Fig 3:** Example of 0% (**A**) and 100% (**B**) directional specificity of muscle activity (0% is a round circle and 100% is a straight line from circle center through the periphery). The thick line represents the reference circle for polar plot.

### 2.1.3 Anatomy of the Neck Muscles

The primary function of the neck is to support the head while providing multiple degrees of freedom for head movements with respect to the torso. Neck muscles are generally fatigue resistant and their function is postural in addition to orientating sensory functions located in the head.

The cervical spine has seven vertebrae which are specialized to support the head and protect the spinal cord. The cervical vertebrae carry less weight and are smaller than their lumbar counterparts. The center of gravity of the head is placed superior and anterior to atlanto-occipital joint causing the head without muscle support to go into flexion (69).

The SCM muscles are located on either side of the neck. The origin of the SCM muscle arises from the sternal head which is attached to the anterior surface of the manubrium sterni and the wider clavicular head which arises from the upper surface of the medial third of the clavicle (70). The insertion of SCM is lateral and adjacent to the insertion of SPL, and the two heads of the SCM merge as the muscle passes upwards, laterally and posterior to insert onto the lateral surface of the mastoid process of the temporal bone and the adjacent part of the superior nuchal line (70). The spinal part of the accessory nerve supplies the SCM on its way to the trapezius muscle.

The SCM receives arterial supply from branches of the superior thyroid, occipital, posterior auricular and suprascapular arteries (70). The action of the SCM varies according to whether one or both SCM muscles are activated. When one muscle acts, the head is ipsilateral lateral flexed and rotated to face the contralateral side. When the muscles act bilateral, the head moves forwards and is flexed (70).

The origin of SPL is attached proximally to the lower half of the ligamentum nuchae, spinous processes of C7 to T4 and intervening supraspinous ligaments. The muscle passes supero-laterally to attach to the mastoid process and occipital bone below the lateral third of the superior nuchal line deep to SCM (70). SPL receives its nerve supply from the lateral branches of the dorsal rami of C3 to C5 (70). The SPL receives arterial supply from muscular branches of the occipital artery originating from the external carotid artery. When the SPL is working individually it extends the head and neck, accompanied by lateral flexion of the neck and rotation of the face to the same side. Pure extension is achieved when SPL is activated bilaterally.

The stability of the cervical spine is dependent on the integrated function of bone, joints, ligaments and muscles. The location and small moment arms of muscles deep in the neck support the concept of their anti-gravitational function, while the larger moment arms of the superficial muscles are more suited for gross movement (5, 6). The apparent functional division of the deep and superficial neck muscles may also be reflected in the muscle activation patterns. The muscle activation patterns are reported to be altered by pain in the neck region, where decreased activation of the deep muscles and increased activation of the superficial muscles have been observed (17, 18).

## 2.2 RATIONALE FOR THE THESIS

The neural control of the neck muscles have before these studies only been investigated using EMG tuning curves in the horizontal plane during circular isometric contractions in healthy individuals. In this thesis, it was hypothesized that the tuning curves of neck muscles in patients with chronic neck would be less focused, as neck pain is recognized to generate short and long term alterations of cervical motor control including increased antagonist activity (17, 39).

Two cross sectional studies investigated activity and directional specificity of neck muscle activity by 1) comparison of the directional specificity of neck muscle activity between patients with neck pain and healthy controls and 2) assessment of mechanisms underlying altered directional specificity of neck muscle activity. The functional consequences of reduced directional specificity of neck muscle activity were assessed in the third study by assessing the effect of reduced specificity on maximal voluntary neck strength. Finally, the effect of specific training on directional specificity of neck muscle activity was evaluated in the fourth Study, which was a randomized controlled trial in patients with chronic neck pain.

Four aims were formulated for the four studies and relevant data were extracted for the thesis.

## 2.3 AIMS OF THE THESIS

1) To observe differences in directional specificity of the SCM and SPL muscles between patients with chronic neck pain and healthy controls.
2) To determine if the neural drive to the SCM, as reflected by motor unit behavior, during isometric contractions in the horizontal plane differs between patients with neck pain and healthy controls.
3) To investigate the effects of reduced SCM directional specificity on neck strength in patients with chronic neck pain.
4) To conduct a randomized controlled trial to investigate whether directional specificity of neck muscle activity can be enhanced via training.

## 2.4 OUTLINE OF THESIS

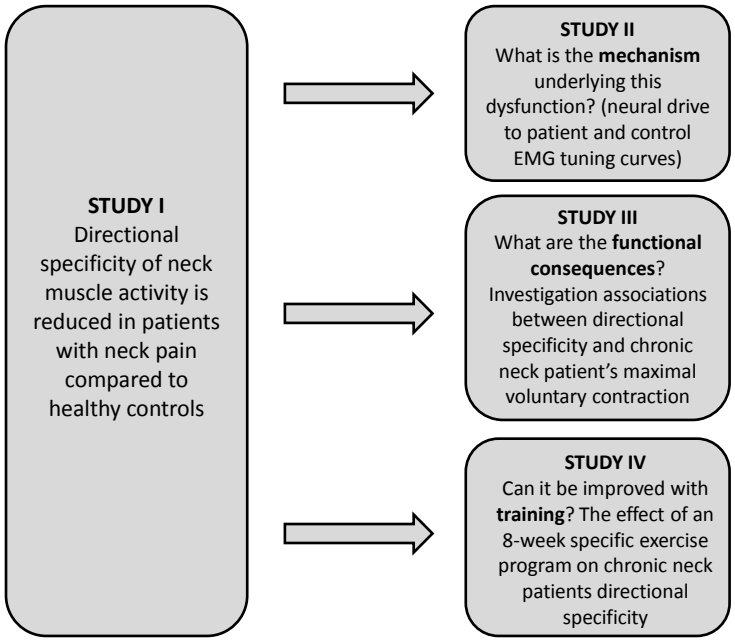


**Fig 4:** This PhD thesis is derived from four papers investigating directional specificity of neck muscle activity assessed from EMG tuning curves. Study 1 assesses differences in directional specificity between patients and controls for the SCM and SPL muscle in a cross sectional study. Study 2 assesses differences in the neural drive to the SCM by evaluating the behavior of single motor units during isometric contractions in the horizontal plane between patients with neck pain and healthy controls. Study 3 assesses the effect of reduced SCM directional specificity on neck strength and finally Study 4 investigates, via a randomized controlled trial, whether directional specificity of neck muscle activity can be enhanced via training.

# 3. Methods

## 3.1 DESCRIPTION OF STUDIES

**Study 1** "Association between neck muscle co-activation, pain, and strength in women with neck pain" investigated the directional specificity of neck muscle activity in a cross sectional study of patients with chronic neck pain and healthy controls. Measures of neck pain and MVC were collected in addition to surface EMG recordings from SCM and SPL during isometric circular contractions and ramped isometric contractions. The two populations were compared for differences in outcomes and further, directional specificity of muscle activity, pain, disability and MVC were investigated for correlations.

**Study 2** "Effect of pain on the modulation in discharge rate of Sternocleidomastoid motor units with direction of force" was a cross sectional study. The primary outcomes were the discharge rate of SCM motor units during submaximal isometric contractions in the horizontal plane in patients with chronic neck pain and in healthy controls. Secondary outcomes were correlations between MVC, directional specificity of SCM activity and clinical data from questionnaires.

**Study 3** "Current pain and fear of pain influence neck strength in patients with chronic neck pain" examined the strength of association between cervical MVC and factors that might explain the reduced MVC identified in patients with chronic neck pain. MVC were assessed in chronic neck patients and healthy controls and patients were further investigated with questionnaires, ultrasound, pressure algometry and surface EMG (SCM and SPL) during submaximal multidirectional isometric circular contractions. The results of each factor was averaged and entered into a multiple regression analysis with average neck strength as the dependent variable.

**Study 4** "Effectiveness of an 8-week exercise program on pain and specificity of neck muscle activity in patients with chronic neck pain – a randomized controlled study" investigated the effect of an 8-week specific training program on neck disability and directional specificity of neck muscle activity. The Neck Disability Index (NDI) score was the primary outcome measure.

An overview of study design and methodology for each study is presented at the end of Methods section.

## 3.2 SUBJECTS

The four Studies investigated women with chronic non-specific neck pain with symptoms greater than 3 or 12 months. Patients with chronic neck pain and healthy controls were matched in Studies 1, 2 and 3 and there were no significant differences ($P < 0.05$) between groups for age, height and weight. Study 4 was a Randomized Controlled Trial. The number of patients and controls included in each study are shown in Table 1.

**Table 1:** The number of patients and healthy controls included in each study of the thesis. Study 1, 2 and 3 were cross sectional studies and Study 4 a randomized controlled trial. * = number of baseline patients included in the in intervention group, # = number of baseline patients included in the non-intervention group and number in () is patients completing the trial.

| Study | Patients | Controls |
|---|---|---|
| Study 1 | 13 | 10 |
| Study 2 | 9 | 9 |
| Study 3 | 34 | 14 |
| Study 4 | * 23 (21) | # 23(21) |

## 3.3 FORCE AND EMG MEASURES

### 3.3.1 Maximum voluntary contraction

Isometric maximum voluntary contractions of the neck muscles were assessed in flexion, extension, right lateral flexion, and left lateral flexion. The MVC was assessed in N in a custom build force measuring device (Fig 5). Participants were comfortably seated in a height-adjustable chair of the device for the measurement of neck force (Aalborg University, Denmark) (68) with head, shoulders and trunk fixed to the rigid frame of the device. Knees and hips were placed in 90° of flexion with participants' feet on the ground and their hands resting comfortably in their lap. The device was equipped with eight adjustable contacts which are fastened around the head to stabilize the head and provide resistance during isometric contraction of the neck. The force device was constructed with force transducers (strain gauges) to measure force in sagittal and coronal planes (Fig 5). The strain gauge signals were amplified with two Miso II amplifiers (OT Bioelettronica, Torino, Italy), and low-pass filtered (anti-causal Butterworth filter order 4, cut-off frequency 10 Hz), sampled at 2048 Hz, and converted to digital form by a 12-bit analogue-to-digital converter and stored on a computer.

Subjects trained with a few low force contractions before recording the MVC's. Two MVC's were performed in each direction in a random order. The

MVC's were of 3-4 s duration and separated by 1 min rest. Verbal encouragement was provided to the subjects.

The maximum MVC for each direction was selected for further analysis in Study 1, 2 and 4. In Study 3 the maximum force for each direction of flexion, extension, left and right lateral flexion were averaged (Average MVC) and entered in a regression analysis as the dependent variable. In Study 3, the Average MVC was selected as a representative measure of the subject's global neck strength, as previous reports of averaged and non-averaged MVC indicate that averaging improves sensitivity to measures of neck pain (34, 71, 72).

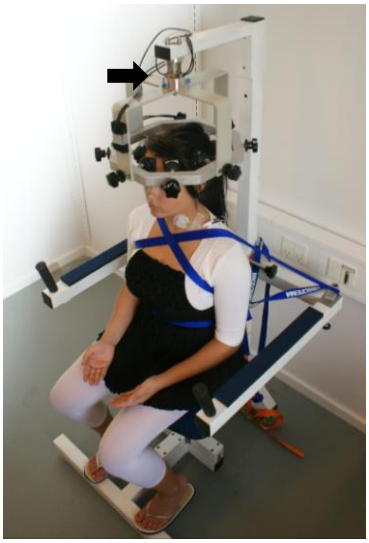

**Fig 5:** Device used to measure multidirectional isometric neck force. Participants are seated with their head rigidly fixed by 8 contacts in the headpiece. The subject's back and torso are firmly strapped to the seat back. Surface EMG was acquired from SCM and SPL muscles bilateral (SPL electrodes not visible on picture). Arrow point to force transducers based on strain gauges measuring force in coronal and sagittal planes.

### 3.3.2 Submaximal voluntary contractions

Submaximal and maximal force was assessed in the same device and with similar methodology as maximal force (Fig 5). Absolute levels of submaximal target force (15 or 30 N) were elected in all Studies excluding the ramped contractions in Study 1. Two minutes of rest periods were provided between submaximal contractions.

3.3.2.1 Multidirectional isometric circular contractions

Seated in the force measuring device, subjects performed multidirectional isometric contractions in clockwise and counter clockwise directions (Fig 5). Patients performed 15 N or 30 N of force in the horizontal plane with change in force direction in the range of 0-360° (Table 2) (68). Subjects were guided to the magnitude and direction of force by real time visual feedback on an oscilloscope with a two dimensional plot of sagittal and coronal forces (68). The oscilloscope was placed at eye height 60 cm in front of subjects (Fig 6).

**Table 2:** Displays the force, direction of the multidirectional circular isometric contractions, muscle and averaging of multidirectional outcomes included in each study. Columns from the left: Study number, 15N clockwise and anticlockwise contractions, 30N clockwise and anticlockwise contractions, SCM, SPL and averaged across muscles or directions (clockwise and anticlockwise). * = right and left SCM, X* = right and left SCM and SP.

| Study | Clockwise and Anticlockwise contractions at 15 N | Clockwise and Anticlockwise contractions at 30 N | SCM | SPL | Averaged across Muscle and Direction | |
|---|---|---|---|---|---|---|
| Study 1 | X | X | X | X | X | |
| Study 2 | X | X | X | | X | * |
| Study 3 | X | | X | X | X | X* |
| Study 4 | X | | X | X | X | X* |

**Fig 6:** Experimental set up with real time visual feedback of force during isometric circular contractions.

Multidirectional force signals Acquired from the amplifiers were and stored on a computer. Subjects had a ~10 min practice period before the tasks. A complete

circular contraction was performed over 12 s. The subjects followed a pre-recorded voice instruction during each contraction. Surface EMG was recorded from the SCM and SPL muscles bilaterally during multidirectional contractions.

#### 3.3.2.2 Isometric unidirectional contractions

Ten second 15 N unidirectional isometric contractions were performed in Study 2 for recordings of single motor unit behavior. Preliminary tests showed that decomposition of the intramuscular EMG signal was not sufficiently reliable from isometric circular contraction with sweeping force; therefore the experimental procedure consisted of eight unidirectional isometric contractions (45º intervals in range 0-360º) in a horizontal circle as a substitute for the isometric circular contractions. Intramuscular and surface EMG was acquired from SCM during these unidirectional isometric contractions.

#### 3.3.2.3 Ramped contractions

Ramped contractions were applied in Study 1. Subjects performed linear increasing ramped contractions from 0% to 50% MVC over 3 s in cervical flexion and extension. Real time visual feedback of exerted force was provided during the ramped contractions. Surface EMG was acquired from the SCM and SPL during ramped contractions.

### 3.3.3 Electromyography acquisitions and analysis

Muscle activity during the multidirectional isometric contractions was assessed with surface EMG. Surface EMG and intramuscular EMG were acquired from the SCM during the isometric contractions in Study 1. Surface EMG of SCM and SPL was further assessed during the ramped contractions in Study 1. Electrodes, electrode positioning and instrumentation were similar for all surface EMG acquisitions. Outcomes after signal processing were the ARV in μV analyzed in ms time intervals.

#### 3.3.3.1 Surface EMG acquisition

Bipolar surface electrodes (AMBU A/S Ballerup, Denmark) were placed on the sternal head of the SCM and the SPL muscles bilaterally with pairs of electrodes positioned 20 mm apart following gentle local abrasion. Electrodes were positioned over the distal 1/3 of the SCM muscle (73). Electrodes were placed on the cranial region of the SPL muscle (14). A reference electrode was place around the right wrist. The EMG signals were amplified (128-channel surface EMG amplifier, LISIN-OT Bioelecttronica, Torino Italy; -3dB bandwidth 10-500 Hz) by a factor of 500 – 5000, sampled at 2048 Hz, and converted to digital form by a 12-bit analogue-to-digital converter.

#### 3.3.3.2 Intramuscular acquisitions

Intramuscular EMG was acquired bilaterally from the SCM muscle during the isometric unidirectional contractions in Study 2. Subjects performed isometric contractions of 10-s duration (constant force direction) exerting a force of 15 N in eight directions (45 intervals) in the range 0–360º (0º: flexion, 90º: right lateral flexion, 180º: extension, 270º: left lateral flexion). An absolute level of force was selected as the target to eliminate variation due to differences in strength between the two groups. Real-time visual feedback of force direction and magnitude was provided on an oscilloscope positioned in front of the subject, which displayed a template with force targets for each direction. The direction of the contractions was randomized and each contraction was followed by rest periods of 2 min. Intramuscular EMG was not acquired from the isometric circular contractions as decomposition of signals was unreliable.

Single motor units action potentials were recorded with a pair of Teflon coated stainless steel wires (diameter: 0.1 mm; A-M system carlsborg, WA) inserted into the SCM ~2-cm cephalad to the midpoint between the sternum and the mastoid process via a 25-gauge hypodermic needle. The wires provided a bipolar signal and were cut only to expose the cross section. The signal was amplified (Counterpoint EMG, DANTEC Medical, Skovlunde, Denmark), band-pass filtered (500Hz-5 kHz), sampled at 10,000 Hz, and stored after 12-bit A/D conversion. A common reference electrode was placed around the wrist.

Single motor units action potentials were identified and extracted from the intramuscular EMG with a decomposition algorithm (74). The discharge rate of the identified motor units was obtained across the 10-s constant force contraction. The variability in ISI was computed as the ratio (%) between SD and mean ISI. Discharge rate and ISI were computed from the entire contraction.

#### 3.3.3.3 Multidirectional isometric contractions

The amplitude of muscle activity was assessed during clockwise and counterclockwise multidirectional isometric contractions with bipolar surface EMG. The ARV in μV was estimated in intervals of 250 ms and analyzed as a function of the angle of force direction (directional activation curve) for each muscle. The directional activation curves represent the modulation in intensity of muscle activity with the direction of force exertion and represent a closed area when expressed in polar coordinates (68). The line connecting the origin with the central point of this area defined a directional vector, whose length was expressed as a percent of the mean ARV during the entire task. This normalized vector length represents the directional specificity of muscle activation. This vector is equal to zero, when the EMG amplitude was the same in all directions and corresponds to 100%, when the EMG amplitude was exclusively in one direction (the muscle was only active in one direction) (68). In addition, the ARV was averaged across the entire circular contraction, to provide the Mean EMG which is an indicator of the average amplitude of muscle activity.

EMG data extracted with from the clockwise and counter-clockwise contractions showed no significant differences therefore the data were combined to obtain an average.

#### 3.3.3.4 Ramped contractions

Ramped contractions were performed over 3 seconds from 0% to 50% MVC. The ARV was estimated from the EMG signal over 5 intervals of 250ms with10% MVC force increase per interval.

## 3.4 PAIN, DISABILITY AND FEAR OF MOVEMENT

### 3.4.1 Visual analogue pain

A visual analogue scale (VAS) was used to assess pain. The pain was assessed on a 0 - 10 cm VAS with anchors “No pain” and “Worst pain imaginable” (75). Patients were asked in Study 1 and 2 for their perception of average pain during the active day in an unspecified time frame. A time frame of 4 weeks was imposed in Study 3 and 4 for average pain during the active day. Two additional VAS pain measures were collected in Study 3, 1) pain immediately before the MVC and 2) maximum pain experienced during MVC contractions. The maximum pain experienced during MVC was further averaged for analysis across flexion, extension, right lateral flexion and left lateral flexion to construct the measure of “Contraction pain”. The three measures of pain were entered in a regression analysis in Study 3.

### 3.4.2 Neck Disability Index

Neck disability was assessed with a Danish version of the NDI, which assess disability on a Likert scale (score: 0-50), the NDI consists of 10 questions with a maximum score of 5 points per question. The questionnaire assesses pain, limitation in physical or cognitive performance during leisure or work (76). The NDI was applied in all four Studies.

### 3.4.3 Patient-Specific Function Scale

Perceived disability was assessed in Study 3 and 4 with a Danish version of the Patient-Specific Function Scale (PSFS). The PSFS assesses three patient activities of daily life. The patients were instructed to select activities which affected their neck pain. The PSFS is scored on a 0 – 10 cm VAS anchored with “cannot perform activity” and “no problem at all” (77).

### 3.4.4 Fear avoidance belief questionnaire

A Danish version of the Fear Avoidance Belief Questionnaire (FABQ) (78) was administered in Study 2. The FABQ assesses the patients' fear of movement and fear of movement related pain. The FABQ is scored in two parts; one with 7 questions relating to work activities: maximum score: (42) and one with 4 questions relating to physical activities: maximum score: (24), the total score (0-66) was included in the analysis. The instruction to the patients was to focus on their neck when answering the questions. Unemployed patients were instructed to answer the questionnaire based on their last previous work experience.

An overview of study design and methodology across Studies are presented in Table 3.

## 3.5 OVERVIEW OF STUDY DESIGN AND METHODOLOGY

**Table 3:** Summary of study design and methodology across Studies.

| | **Study 1** | **Study 2** | **Study 3** | **Study 4** |
|---|---|---|---|---|
| **Title** | Association between neck muscle co-activation, pain, and strength in women with neck pain | Effect of pain on the modulation in discharge rate of SCM motor units with direction of force | Current pain and fear of pain influence neck strength in patients with chronic neck pain | Effectiveness of an 8-week exercise program on pain and specificity of neck muscle activity in patients with chronic neck pain - a randomized controlled study |
| **Research question** | Is directional specificity different between patients with pain and healthy controls? | Are discharge frequencies in the horizontal plane different between patients with chronic neck pain and healthy controls? | Is directional specificity associated with the reduction of maximum voluntary contractions observed in patients with chronic neck pain? | Can directional specificity be altered by training? |
| **Study design** | Cross sectional design with matched(age, height and weight) | Cross sectional design with matched (age, height and weight) | Cross sectional design with matched (age, height and weight) patient (n = 34) and | Examiner blinded randomized controlled trial with intervention (n = 23) and control (n = 23) |

| | | | | |
|---|---|---|---|---|
| | patient (n = 13) and control (n = 9) groups | patient (n = 9) and control (n = 9) groups | control (n = 14) groups | group |
| **Patient inclusion criteria** | Women with chronic non traumatic neck pain for at least one year between the age of 18 and 50 | Women with chronic non traumatic neck pain for at least 3 month between the age of 18 and 50 | Women with persistent chronic non traumatic neck pain for at least one year between the age of 18 and 50 limiting their daily physical activity | Women with persistent chronic non traumatic neck pain for at least one year between the age of 18 and 50 limiting their daily physical activity |
| **Patient exclusion criteria** | Cervical spine surgery, neurological signs in the upper limps or had a participated in neck exercises within past 12 month | Cervical spine surgery, neurological signs in the upper limps or had a history of torticollis | Neck pain induced by trauma, systemic disease influencing their neck pain, moderate to severe degeneration of the cervical spine, spinal surgery, pain radiating to the upper extremity, neurological signs or pregnant. | Trauma-induced neck pain, neck pain from inflammatory or infectious condition, neurological signs, cervical spine surgery, exercise therapy within 3 months prior to entry into the Study, current treatment for neck pain from health care providers or pregnant. |
| **Control subject** | Free of neck and shoulder pain, no past history of orthopedic disorders affecting neck or shoulder region and no history of neurologic disorders | Free of neck and shoulder pain, no past history of orthopedic disorders affecting neck or shoulder region and no history of neurologic disorders | Free of neck pain, neurologic signs, or any neck and shoulder disorder, which may affect their neck function. | Patients with chronic neck pain |
| **Question-naire and pain measure** | NDI and VAS pain score | NDI and VAS pain score | Three VAS pain scores, NDI, Danish SF-36, Patient- Specific Functional | VAS pain score, NDI, Danish SF-36, Patient-Specific Functional Scale |

| | | | | |
|---|---|---|---|---|
| | | | Scale and Fear-Avoidance Beliefs Questionnaire | and Fear-Avoidance Beliefs Questionnaire |
| **Acquisition** | Surface EMG bilaterally from SCM and SPL during ramped contractions from 0 to 50% MVC followed by 15N and 30N clockwise and counter clockwise contractions | Surface EMG bilaterally from SCM with 15N and 30N clockwise and counter clockwise contractions Intramuscular EMG bilateral from SCM with 15N at 45° intervals | Surface EMG bilaterally from SCM and SPL with 15N clockwise and counter clockwise contractions | Surface EMG bilaterally from SCM and SPL with 15N and 30N clockwise and counter clockwise contractions |
| **Force measure** | Flexion, extension, right lateral flexion and left lateral flexion MVC | Flexion, extension, right lateral flexion and left lateral flexion MVC | Flexion, extension, right lateral flexion and left lateral flexion MVC | Flexion, extension, right lateral flexion and left lateral flexion MVC |
| **Primary outcome** | Directional specificity. and Mean EMG from circular contractions, EMG from ramped contractions, MVC, NDI and VAS pain | Motor unit discharge rate, directional specificity. and Mean EMG from circular contractions and MVC | Directional specificity. and Mean EMG from circular contractions, MVC, VAS pain, NDI and FABQ | Directional specificity. and Mean EMG from circular contractions, MVC, VAS pain and NDI |

### 3.6 TRAINING INTERVENTION

In Study 4, the randomized controlled trial tested the effect of act as usual against an 8-week progressive exercise program for the neck flexors and extensor muscles (79). Patients received personal instruction and supervision by a physiotherapist for ~30 min once per week. The exercise program consisted of two-stages. The principal exercise during the first 6 weeks was incremental cranio-cervical flexion in a relaxed, supine lying position. The exercise targets the deep flexors of the upper cervical region, the longus capitis and colli (40), rather than the superficial flexors, SCM and anterior scalene muscles. The patients were instructed to perform

and hold progressively inner range positions of cranio-cervical flexion. In addition the patients performed cranio-cervical extension, flexion and rotation in a prone on elbows position while maintaining the cervical spine in a neutral position.

The last two 2 weeks involved higher load exercise with head weight as the load. During this stage, the patients performed up to 15 repetitions of a head lift for flexors and extensor muscles. For the head lift, the patients were instructed to perform cranio-cervical flexion followed by cervical flexion to just lift the head from the bed. For the neck extension exercise the patients were instructed to keep their cranio-cervical region in a mid-position while they extended the cervical region. The higher load repetitions were performed over a 3 s period with no rest between repetitions. Patients were requested to practice their respective regime twice per day without any provocation of neck pain, to ensure pain free training the level and number of repetitions of each exercise was individually tailored to each patient. Patients were asked to refrain from seeking new intervention for their neck during the training period.

## 3.7 PATIENT PROTECTION AND ADVERSE EFFECTS

Ethical approval for the Studies was granted by the Regional Ethics committee and procedures were according to the declaration of Helsinki. Patients and control subject were under constant supervision by the investigator pain and discomfort was monitored throughout the laboratory sessions. The investigator was an experienced practitioner with 20 years' experience of managing patients with neck pain.

There were a few minor increases in neck pain due to the experimental procedures. The increased pain was of varying duration and lasted up to a maximum of 24 hours. The increased neck pain was recognized by the patients as normal for strenuous activities similar to the laboratory procedures; however no patients expressed concerns of being overexerted. The increased pain experienced by patients appeared to be random across all experimental procedures and no procedure could be recognized as harmful.

# 4. Results

## 4.1 ASSOCIATION BETWEEN NECK MUSCLE COACTIVATION, PAIN, AND STRENGTH IN WOMEN WITH NECK PAIN (STUDY 1)

### 4.1.1 Pain, Disability and Maximum voluntary contraction

Thirteen patients with chronic neck pain and 10 controls were matched with no statistical difference between age, height or weight (Table 4). The patients' average score for the NDI (0-50) was 21.6 ± 8.4 and their average pain intensity rated on a VAS (0-10) was 5.1 ± 1.8.

**Table 4:** Subject demographics

| | Age | Height | Weight |
|---|---|---|---|
| Controls (n = 13) | 37.7 ± 7.8yrs | 168.8 ± 4.0cm | 77.2 ± 18.5Kg |
| Patients (n = 10) | 33.1 ± 9.3yrs | 165.9 ± 8.2cm | 66.8 ± 13.0Kg |

*Motor output*

The maximum voluntary neck strength was dependent on force direction ($F = 46.7$, $P < 0.00001$); extension and bilateral lateral flexion showed higher values of force compared to flexion (Student-Newman-Keuls (SNK): all $P < 0.001$). Furthermore, extension force was greater than left and right lateral flexion force (SNK: both $P < 0.05$). However, the patient group exerted lower force across all directions compared to the control subjects ($F = 6.8$, $P < 0.05$; Table 16 and17, Appendix).

### 4.1.2 Directional specificity of muscle activity

Representative directional activation curves during a circular contraction performed at 15 N are illustrated in Fig 7 for a control subject and a patient. In this example, the control subject presents with defined activation of the SCM and SPL muscles with the highest amplitude of activity towards ipsilateral anterolateral flexion and ipsilateral posterolateral extension respectively. Note that both the

SCM and SPL are minimally active during the antagonist phase. Conversely, the directional activation curves for the representative patient show activation of the SCM during extension and SPL during flexion. Accordingly, overall the patient group displayed reduced values of directional specificity in the surface EMG for both the SCM and SPL muscles bilaterally for both the 15 N and 30 N circular contractions (main effect for group: $F = 6.0$; $P < 0.05$).

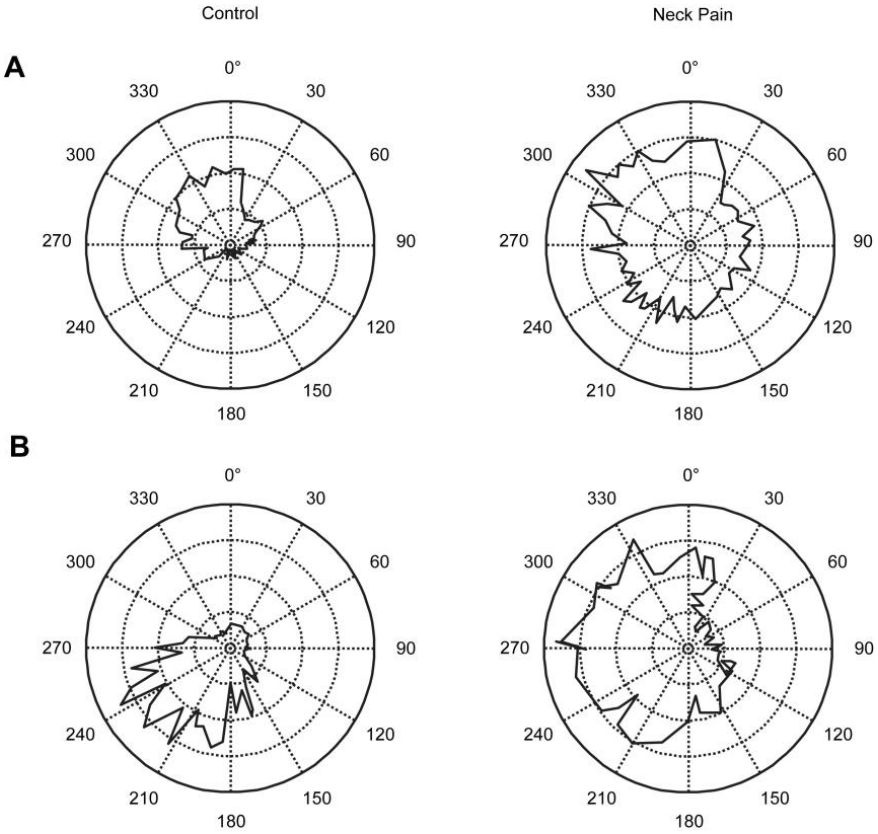


**Fig 7:** Representative directional activation curves for the left SCM (**A**) and left SPL (**B**) of a control subject and a patient performing a circular contraction in the horizontal plane at 15 N with change in force direction in the range 0-360º.

*Ramp contractions*

Both SCM and SPL ARV increased with increasing cervical flexion force ($F=110.7$, $P< 0.0001$). The ARV of SCM (agonist) did not differ between patients and controls during cervical flexion, however higher values of ARV were observed for the right SPL (antagonist) at all force levels in the patient group (SNK: all P

<0.05). Higher values of left SPL ARV were also observed for the patient group during cervical flexion at force levels 20-50% (all SNK: $P < 0.05$).

Both SPL (agonist) and SCM (antagonist) ARV increased with increasing cervical extension force ($F= 23.1$, $P <0.0001$). However, SPL and SCM ARV were greater for the patients across all force levels compared to the control subjects ($F= 4.4$, $P< 0.05$).

### 4.1.3 Associations between pain, strength and antagonist activity

The ARV acquired when SPL was acting as antagonist during ramped cervical flexion (averaged across sides) was positively correlated with patients' reported pain ($R^2 = 0.35$, $P < 0.05$) and perceived disability ($R^2 = 0.53$, $P < 0.01$).

Furthermore, the total neck strength (sum across all directions of force), showed inverse correlation with the amount of SPL activation during cervical flexion ($R^2 = 0.54$, $P < 0.01$). The ARV of SPL (averaged across sides) during cervical flexion showed a tendency to be inversely correlated with patients maximum cervical flexion force ($R^2 = 0.26$, $P = 0.07$).

In contrast to SPL during flexion, no correlations were observed between the amount of activation of the SCM muscle during cervical extension and extension strength ($R^2 = 0.00$; $P = 0.97$), total neck strength ($R^2 = 0.01$; $P = 0.73$) or perceived pain and disability (pain: $R^2 = 0.08$; $P = 0.24$; NDI: $R^2 = 0.12$; $P = 0.34$).

### 4.1.4 Discussion

The hypothesis that directional specificity would be decreased in patients with chronic neck compared to healthy controls was confirmed. The results of the study show that directional specificity of the SCM muscle was significantly reduced and less defined compared to healthy controls (5, 6). Likewise the SPL showed less defined directional activation curves compared to healthy controls.

The reduced specificity of muscle activity results mainly from increased co-activation of the muscles when acting as antagonist. This finding suggests that increased antagonist activity is a common characteristic of neck pain and may reflect an altered motor control strategy to enhance cervical stability (80) in an attempt to make the spine more rigid (11). Increased co-activation of the SPL muscle was associated with lower neck strength and higher levels of pain and associated disability. Although increased coactivation of the neck muscles may be beneficial in the presence of pain to increase cervical stability, as observed in this Study, it is associated with functional consequences, i.e. reduced neck strength. Furthermore, increased neck muscle coactivation may contribute to recurrent pain by altering the load distribution on the spine and subsequently aggravating the patients' condition.

Coactivation of agonist/antagonist muscles significantly increases spinal stiffness (81) and spinal compression which is considered sufficient to induce lumbar spine injuries and consequently low back pain (82). Unique to Study 1, we showed that the degree of coactivation of the SPL muscle is positively correlated

with the patients reported pain and perceived disability which supports this premise. Surprisingly, a similar relation was not observed for the SCM muscle despite reduced specificity of SCM activity and increased activation of the SCM muscle during ramped cervical extension contraction in the patient group. This finding may be attributed to the greater reduction in neck flexion strength for the patient group (31.7% less than controls) compared to the extension (22.6% less than controls).

Presented in the Appendix in Tables 17 and 18, is the relative standard deviation in % (%RSD) from the MVC of flexion, extension and lateral flexion. The relative standard deviations indicate that the extensor MVC in healthy controls is less varied compared to the %RSD for the flexor MVC and this finding may partly explain why the ARV from SPL showed significant correlations as opposed to ARV from the flexor SCM.

Although the ramped and circular contractions were both isometric contraction; the circular contractions is multidirectional and more complex than the unidirectional ramped contractions which may explain why no significant correlations were found with directional specificity.

## 4.2 EFFECT OF PAIN ON THE MODULATION IN DISCHARGE RATE OF STERNOCLEIDOMASTOID MOTOR UNITS WITH DIRECTION OF FORCE (STUDY 2)

### 4.2.1 Pain, Disability and Maximum voluntary contraction

Nine patients with chronic neck pain and nine controls were matched with no statistical difference for age height and weight (Table 5). The patients' average score for the NDI (0-50) was 16.5 ± 8.8 and their average pain intensity rated on a VAS (0-10) was 4.3 ± 1.5. The maximum voluntary neck strength was dependent on force direction ($F = 31.49$, $P < 0.00001$) with extension showing highest values of force compared to the other directions (SNK: all $P < 0.001$). In addition, the maximum force produced in cervical flexion was lower than in the other directions (SNK: all $P < 0.05$). The patient group exerted lower force across all directions compared to the control subjects ($F = 4.7$, $P = 0.045$; Table 16 and 17 in the Appendix).

**Table 5:** Subject demographics

| | Age | Height | Weight |
|---|---|---|---|
| Control (n = 9) | 35.4 ± 7.5yrs | 164.8 ± 7.7cm | 65.0 ± 12.3Kg |
| Patient (n = 9) | 40.4 ± 3.5yrs | 170.8 ± 5.5cm | 73.7 ± 10.1Kg |

### 4.2.2 Modulation of discharge rate

Representative data for motor units recorded from the left SCM muscle during the 10-s contractions in constant force directions are presented in (Fig 8). For the

control subject a clear modulation in the discharge rate is observed: the discharge rate was 12 pulses per second (pps) during ipsilateral lateral flexion, 16 pps in ipsilateral anterolateral flexion, 14 pps in flexion, and 8 pps in contralateral lateral flexion. In the other force directions, motor units were not active. On the contrary, a single motor unit recorded from the left SCM from a patient with neck pain which was tracked over several force directions showed a consistent discharge rate of 12 pps except in the directions of contralateral posterolateral extension and extension where it was de/recruited.

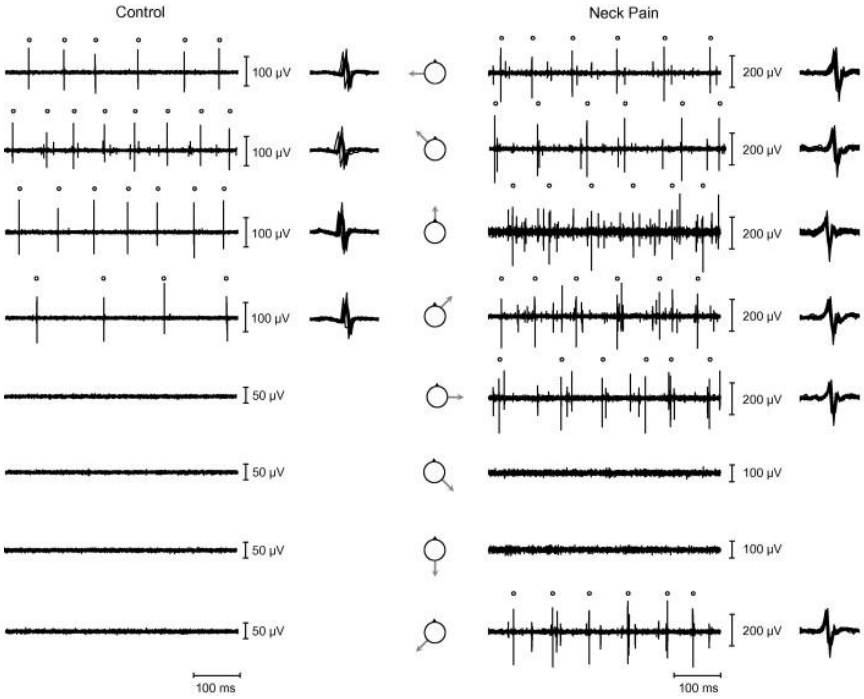


**Fig 8:** Representative single motor unit recordings from the left SCM muscle of one control subject and one patient with neck pain during the 10-s contractions in constant force directions. The symbols in the center of the image illustrate the directions of force. Segments (500 ms) of intramuscular EMG signals are presented with the identified motor unit discharge represented by circles above each trace. The corresponding template of the identified motor unit is presented to confirm that the same motor unit has been tracked in the different force directions. Note the modulation in the discharge rate for the control subject but consistent discharge rate for the patient.

The group data confirmed the characteristics in motor unit behavior shown in the representative example. The discharge rates of the left SCM motor units were dependent on the interaction between group and force direction ($F = 2.0$, $P = 0.045$). For the control group, motor unit discharge rate for the left SCM was

higher in the anterior position (0º), 45º, and 315º compared to 90º, 135º and 180º (all $P < 0.05$). The discharge rate of motor units in the right SCM was also dependent on the interaction between group and force direction ($F = 2.9$, $P = 0.004$). For the control group, motor unit discharge rate for the right SCM was higher in the 0º, 45º, 270º and 315º directions compared to 180º (all $P < 0.05$). On the contrary there were no differences in left or right SCM discharge rate between directions for the patient group.

### 4.2.3 Directional specificity, Mean EMG and chronic neck patients

The patients demonstrated higher values of ARV (averaged across the circular contractions) for the SCM bilaterally during the circular contractions performed at 15 N ($P = 0.044$) but not 30 N. For the 15 N and 30 N circular contractions, the ARV (average across left and right SCM) for the patient group were 43.5 ± 54.2 µV and 57.6 ± 51.9 µV and for the control group 16.9 ± 14.9 µV and 47.6 ± 16.9 µV, respectively. In accordance, the patient group showed greater values of SCM ARV for the 10-s contractions in constant force directions (main effect for group; $P = 0.039$; Fig 9).

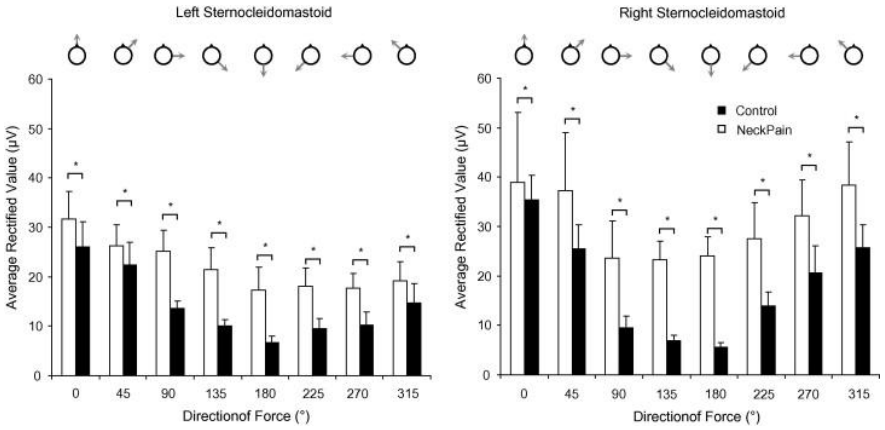


**Fig 9:** Mean ± SD of left and right SCM EMG average rectified value (µV) obtained for the control (n = 9) and patient groups (n = 9) across the 10-s contractions performed at 15 N of force in eight directions (45º intervals from 0º to 360º) in the horizontal plane. The symbols at the top of the image illustrate the directions of force. The patient group showed higher values of SCM average rectified value in all force directions (main effect for group; *$P < 0.05$).

The patient group displayed reduced values of directional specificity in the surface EMG of the SCM muscle bilaterally for both the 15 N and 30 N circular contractions ($F = 4.45$; $P = 0.041$) (Table 6).

**Table 6:** Mean ± SD of the directional specificity in the surface EMG of the right and left SCM obtained during the circular contractions at both 15 and 30 N of force for the patients with neck pain (n = 9) and control subjects (n = 9).

| | Neck pain | | Controls | |
|---|---|---|---|---|
| | **Right SCM** | **Left SCM** | **Right SCM** | **Left SCM** |
| 15N Circular contraction | 22.1 ± 9.7 | 21.0 ± 9.6 | 33.3 ± 18.4 | 32.1 ± 21.7 |
| 30N Circular contraction | 27.0 ± 9.8 | 28.7 ± 9.2 | 37.3 ± 11.5 | 35.3 ± 14.7 |

### 4.2.4 Discussion

In the presence of chronic neck pain the modulation in discharge rate of individual motor units in the SCM muscle differed with respect to control subjects. The neural drive to the muscle was tuned selectively with direction of force production in the control subjects whereas in patients with neck pain it was similar for a large range of directions.

The patient data showed reduced specificity of SCM muscle activation with respect to the controls, resulting in increased activation of the muscle when acting as an antagonist. This finding is consistent with the results of Study I and supports the finding of augmented activity of the SCM muscle in patients with neck pain, regardless of the task examined, e.g., cervical flexion (83), cranio-cervical flexion (39, 84) and movements of the upper limb (43, 45).

The main contribution of this Study is the analysis of the behavior of individual motor units for multiple force directions in women with neck pain and controls. The discharge rates of the active motor units during a task depend on the net excitatory input to the motor neuron pool and the intrinsic excitability of the motor neurons. It was expected that the excitatory input changed with direction of force with a consistent modulation that reflects the biomechanical efficiency in force production. The results obtained in this Study for the control subjects confirmed this hypothesis by revealing a consistent modulation in discharge of motor units across the subject sample. Possible explanations for these findings include the direct effects of nociception on motor neuron output, effects of pain on sympathetic activity, and changes in motor planning. Among these mechanisms, an effect of nociceptive input from the SCM muscle is unlikely since patients reported pain in the posterior aspect of their neck and not anteriorly. Furthermore, experimental pain studies have shown that local excitation of SPL nociceptive afferents or SCM afferents following injection of hypertonic saline, results in a consistent reduction in SCM muscle activity (14) and not augmented activity. The reduced modulation in discharge rate of individual motor units in the SCM muscle and enhanced SCM muscle activity for the patient group may be due to an increased sympatho-adrenal outflow as a consequence of pain.

Theories have been proposed to explain the links between pain and motor control, the theories usually encompasses a few factors and has to be discussed with the perspective of limited application (52). The pain-adaptation model predicts an inhibitory effect of pain on motor neurons during agonist activity and an excitatory effect during antagonist action (51). Is not fully supported by this

Study, as increased agonist activity of the SCM muscle was observed for the patients in the circular plane together with increased antagonist activity was observed. Contrary to the pain-adaptation model, the vicious cycle theory suggests an increased muscle activity as a consequence of activation of group III and IV muscle afferents (50). In this Study, altered activity of the SCM muscle was observed for the patient group regardless of the direction of force which may be in agreement with the vicious cycle theory. However the model may agree with the Study results but the model appears too simplified to explain the link between neck pain and motor control, as the model does not explain the altered motor control which takes place between muscles (10, 17). The model proposed by Hodges and Tucker appears to better encompass the relation between pain and motor control (52).

## 4.3 CURRENT PAIN AND FEAR OF PAIN INFLUENCE NECK STRENGTH IN PATIENTS WITH CHRONIC NECK PAIN (STUDY 3)

### 4.3.1 Baseline variables

Thirty-four patients with chronic neck and fourteen controls were matched with no statistical difference for age height and weight (Table 7). The patients' average score for the NDI (0-50) was 17.5 ± 6.5 and their average pain intensity during the last 4 weeks rated on a VAS (0-10) was 4.9 ± 1.8. The average pain experienced before and during MVC were 3.8 ± 2.2 and 4.2 ± 2.1, respectively (Table 19 in the Appendix). The average MVC ± SD was significantly lower for the patients (130.0 ± 6.0N) compared with the controls (166.9 ± 11.7N; $P<0.01$). MVC results for both patients and controls are reported in the Appendix in Tables 17 and 18.

**Table 7:** Patient demographics

| | **Age** | **Height** | **Weight** |
|---|---|---|---|
| Patients (n = 34) | 40.5 ± 7.9 yrs | 169.3 ± 6.4 cm | 70.0 ± 15.0 Kg |
| Controls (n = 14) | 37.2 ± 7.6 yrs | 168.1 ± 5.9 cm | 67.7 ± 13.2 Kg |

### 4.3.2 Regression analysis

Baseline values of directional specificity averaged across muscles (SCM, SPL) and directions (clockwise, counterclockwise) (mean ± SD) (18.7 ± 4.7%), and Mean EMG averaged across muscles and directions (21.7 ± 16.3μV) are presented in Table 8 together with other baseline measures for patients and average MVC for patients and control subjects.

**Table 8:** Mean ± SD variables measured in the patient group, which were entered in the regression analysis. Mean EMG, mean EMG acquired from circular contraction; Ave. Dir. Spec.

%, average directional specificity in % across circular contraction; Ave. pain 4 week, averaged pain exercised last 4 weeks; Contraction pain, average of pain experienced performing MVC.

| Measure | Patient | |
|---|---|---|
| | **Mean** | **SD** |
| Mean EMG (µV) | 21.7 | 16.3 |
| Ave. Dir. Spec. (%) | 18.7 | 4.7 |
| NDI (0-50) | 17.5 | 6.5 |
| FABQ (0-66) | 27.2 | 13.6 |
| PSFS (0-10) | 4.3 | 2.0 |
| Ave. pain 4 week (VAS, 0-10) | 4.9 | 1.8 |
| Contraction pain (VAS, 0-10) | 4.2 | 2.1 |
| Pain before MVC (VAS, 0-10) | 3.8 | 2.2 |

Directional specificity and Mean EMG were together with other averaged outcomes (see Methods Section, Chapter 3.3) entered in univariate regression analysis with Average MVC (average across Flexion, Extension, Right Lateral Flexion and Left Lateral Flexion) as the dependent variable. The results are reported in Table 9 and Table 10. Directional specificity and Mean EMG were not significant in the regression analysis (Table 9).

**Table 9:** Results of the univariate regression analyses with Average MVC as the dependent variable. Mean EMG, mean EMG acquired from circular contraction; Ave. Dir. Spec. %, average directional specificity in % across circular contraction.

| Independent Variable | $R^2$ | *P-value* | Constant | B | SE |
|---|---|---|---|---|---|
| Mean EMG | 0.083 | 0.115 | 143.2N | -0.656N | 0.404 |
| Ave. Dir. Spec. | 0.015 | 0.518 | 112.1N | 0.897N | 1.369 |

### 4.3.3 Regression with other factors

The Average MVC was significantly correlated with FABQ ($R^2$ = 0.134), NDI ($R^2$ = 0.138) and Contraction pain (Average of VAS pain experienced during Flexion, Extension, Right Lateral Flexion and Left Lateral Flexion MVC) ($R^2$ = 0.211) in univariate regression analyses (Table 10). In contrast, pain before MVC, PSFS and Average pain over the past 4 weeks were not significantly associated with Average MVC in univariate regression analyses (Table 10).

**Table 10**: Results of the univariate regression analyses with Average MVC as the dependent variable.

| Independent Variable | $R^2$ | *P-value* | Constant | B | SE |
|---|---|---|---|---|---|
| Contraction pain | 0.211 | 0.006 | 162.6N | -7.801N | 2.667 |
| NDI | 0.138 | 0.030 | 164.9N | -1.995N | 0.880 |
| FABQ | 0.134 | 0.033 | 155.6N | -0.940N | 0.422 |
| Pain before MVC | 0.100 | 0.068 | 149.4N | -5.094N | 2.701 |
| PSFS | 0.001 | 0.845 | 127.4N | 0.621N | 3.154 |
| Ave. pain 4 week | 0.000 | 0.948 | 128.9N | 0.220N | 3.347 |

FABQ, NDI and Contraction pain were included in a multivariate regression analysis. FABQ and NDI combined ($R^2$ = 0.187) explained less variation compared to contraction pain alone ($R^2$ = 0.211). The combination of Contraction pain and FABQ ($R^2$ = 0.266) explained 5.5% more than Contraction pain alone. This is marginally better than Contraction pain and NDI ($R^2$ = 0.253), where NDI explains an additional 4.2% (Table 11).

The three factors FABQ, NDI and Contraction pain combined explained ($R^2$ = 0.282) of the variation in Average MVC (Table 12). $R^2$ was only increased by 0.016 when NDI was entered into the regression with FABQ and Contraction pain.

**Table 11:** Multiple linear hierarchical regressions with the average MVC as the dependent variable and the combination of 2 out of the 3 significant variables identified from the univariate regression analyses (FABQ, NDI, and contraction pain). The univariate variables are combined with the variable with the lowest $R^2$ entered first (Variable 1). The $R^2$ and P values presented for Variable 1 is the result of the univariate analysis, while the $R^2$ and P values of Variable 2 shows the combined or multivariate result.

| Variable | Independent | $R^2$ | *P-value* |
|---|---|---|---|
| 1 | FABQ | 0.134 | 0.033 |
| 2 | NDI | 0.187 | 0.040 |
| 1 | NDI | 0.138 | 0.030 |
| 2 | Contraction pain | 0.253 | 0.011 |
| 1 | FABQ | 0.134 | 0.033 |
| 2 | Contraction pain | 0.266 | 0.008 |

**Table 12:** Multiple Linear Hierarchical Regression with the average MVC as the dependent variable and the combination of all 3 significant variables identified from the univariate regression analyses (FABQ, NDI and contraction pain). The $R^2$ and P values presented for Variable 1 is the univariate result, while the $R^2$ and P values of Variable 2 and 3 show the combined or multivariate result.

| Variable | Independent | $R^2$ | *P-value* |
|---|---|---|---|
| 1 | FABQ | 0.134 | 0.033 |
| 2 | NDI | 0.187 | 0.040 |
| 3 | Contraction pain | 0.282 | 0.018 |

### 4.3.4 Discussion

In this Study we hypothesized that maximum force production and the specificity of neck muscle activity had a common origin or some other common mechanism, which would associate average directional specificity or average Mean EMG to average MVC. However there were no correlations between average MVC and average directional specificity or average Mean EMG. This may be attributed to the large variability of the EMG measures (6); however it is more likely that the factors underlying changed directional specificity and Mean EMG have limited linear common origin with the factors underlying maximal force production and Average MVC.

The main results from this Study showed a correlation between average MVC and current pain, fear of pain (78) and the NDI (76). These results provide a method to relate current pain to MVC. The Study further showed that the association between pain reported from patients with chronic neck pain and MVC only was present for pain reported at the moment of force production. The pain that patients experienced during the maximal contractions explained 21% of the variation in average neck MVC. Previously significant inverse relations between MVC and experimental pain intensities in healthy subjects have been reported (13, 85, 86) illustrating that pain per se is capable of reducing the MVC. The level of pain intensity reported before the MVC was marginally associated with MVC and pain averaged over the past 4 weeks showed no relation.

Fear of pain explained 13.4% of the variability of the patients Average MVC. Fear of movements and consequently inactivity is thought to lead to deconditioning (87) which may explain the association between MVC and FABQ. However it is not clear whether reduced MVC due to fear-avoidance is transient or long-lasting. The FABQ score explained an additional 5.5% of the variability of Average MVC when it was combined with the measure of pain experienced by the patient during the MVC. The combined regression result showed that pain alone explained more than half of the variability in the FABQ score. The remaining variability may be explained by muscle inhibition caused by fear-avoidance or long-lasting deconditioning. Fear avoidance may result in a submaximal performance from patients when asked to perform the MVC thereby contributing to a reduced MVC. For example, previous work using superimposed electrical stimulation of the quadriceps during a MVC of the knee extensors showed that patients with low back pain performed submaximally compared healthy controls when asked to perform their maximal effort (30).

The NDI score explained 13.8% of the variation in Average MVC which is in agreement with previous studies (71, 72); however it is not clear which aspects of perceived disability are correlated to MVC since the questionnaire assesses pain and limitations in physical and cognitive performance during leisure or work (76). When NDI and Contraction pain were combined in the multivariate regression, NDI explained an additional 4.2% which suggests that the patient's current pain is one of the main factors contributing to the regressions results although not the only one.

As said, there were no correlations between Average MVC and Average directional specificity or Mean EMG. This may be attributed to the large variability of the EMG measures (6). In addition there was no association between muscle CSA and Average MVC. Thus, the best multivariate result leaves 71.8 % of the variability in Average MVC unexplained. A large part of the unexplained variability may be attributed to the substantial variation in healthy MVC (23, 88) and to other factors not measured in the Study such as fatigue or structural changes in the neck muscles such as muscle fiber transformations (20), or the presence of fatty tissue infiltration within the neck muscles in some patients with neck pain (19).

The averaging conducted in this Study may have faded significant correlations between unidirectional MVC and unidirectional directional specificity or unidirectional Mean EMG. Some studies indicate that side to side differences in muscle activity exist in patients with unilateral neck pain (89) suggesting that it would have been relevant to assess the outcome measures ipsilateral to the side of greatest pain. However previous studies also suggest that averaging of outcome measures results in stronger correlations between physical impairments, pain and disability compared to correlations of non-averaged measures (34, 71, 72). Since the vast majority of patients reported bilateral pain, we selected to average the data in this Study.

The results from Study 1 may indicate that extensor EMG is more strongly correlated to strength and pain compared to flexor EMG. Tables 17 and 18 in the Appendix present the %RSD from unidirectional MVC assessed in Studies 1, 2 and 3. The %RSD from the neck extension MVCs were smaller compared to the neck flexion MVCs which may partially explain why no significant correlations were found in this Study when the flexor and extensor EMG was averaged.

## 4.4 EFFECTIVENESS OF AN 8-WEEK EXERCISE PROGRAM ON PAIN AND SPECIFICITY OF NECK MUSCLE ACTIVITY IN PATIENTS WITH CHRONIC NECK PAIN – A RANDOMIZED CONTROLLED STUDY (STUDY 4)

The study was a randomized controlled trial to test the effectiveness of specific exercise for subjects with chronic neck pain. The trial was a single blind trial design with blinded outcome assessment.

### 4.4.1 Pain and Disability

Twenty tree patients were randomized in 2 groups. There were no significant differences in baseline variables between the groups (all $P > 0.05$) except for the SF-36 which, despite the randomization of patients to groups, was lower (poorer general health status) in the intervention group ($F = 7.9$; $P < 0.01$). Baseline characteristics of the intervention and control group are presented in Table 13.

**Table 13:** Baseline characteristics of the intervention and control groups. Values are presented as mean ± (SD).

| Characteristic | Intervention | Control |
|---|---|---|
| Age (yrs) | 39.1 (8.7 ) | 38.6 (9.0) |
| Height (cm) | 171.0 (5.6) | 168.9 (7.6) |
| Weight (kg) | 72.0(15.1) | 67.0(12.9) |
| Duration of Pain (yrs) | 10.0 (7.4) | 8.4 (5.1) |
| NDI | 18.2 (7.4) | 17.5(6.3) |
| PSFS | 4.4 (2.1) | 3.9 (1.8) |
| Average Days per Month with Neck Pain | 22.6 (8.7) | 18.9(10.4) |
| Employed (%) | 86 | 90 |
| SF-36 (Total) | 53.3(17.8) | 68.6(17.0) |
| Physical | 46.8(16.5) | 63.7(18.5) |
| Mental | 55.7(20.6) | 70.3(15.5) |

The mean changes in primary outcomes are presented in Table 14. A significant interaction was observed between group and time for the NDI score ($F = 4.4$, $P < 0.05$). A significant reduction in reported NDI was observed for the intervention group post treatment (pre: 18.2 ± 7.4; post: 14.1 ± 6.6; SNK: $P < 0.01$) but not for the control group (pre: 17.5 ± 6.3; post: 16.6 ± 7.4). The effect size of this primary outcome was 0.65. Likewise a significant interaction was observed between group and time for the PSFS score ($F = 12.9$, $P < 0.001$) and VAS ($F = 4.4$, $P < 0.05$). An increase in PSFS scores were observed for the intervention group post treatment (pre: 4.4 ± 2.1; post: 5.6 ± 2.2; SNK: $P < 0.001$) but not for the control group (pre: 3.9 ± 1.8; post: 3.9 ± 1.7). Similarly, the average intensity of neck pain over the last 4 weeks was lower for the patients in the training group (pre: 5.3 ± 2.8; post: 3.6 ± 2.4; SNK: $P < 0.001$) but did not change for the control group (pre: 5.1 ± 2.0; post: 4.9 ± 2.3).

**Table14:** Mean ± SD of the change in the primary and secondary outcome measures for the intervention group and control group

| Outcome | Intervention | Control |
|---|---|---|
| NDI | -4.1 (4.8) | -1.0 (4.4) |

| | | |
|---|---|---|
| PSFS | 1.1 (1.1) | 0.08 (0.7) |
| VAS | -1.7 (2.2) | -0.3 (2.1) |

### 4.4.2 Directional specificity of muscle activity

Representative directional activation curves during a clockwise circular contraction performed at 15 N are illustrated in Fig 10 for a patient allocated to the training group and to the control group both pre and post intervention. At baseline for both patients the directional activation curves show quite even activation levels of the left and right SCM and SPL muscles for all directions. The lack of specificity of muscle activity remains for the patient assigned to the control group for the post measurement. On the contrary, the patient assigned to the training intervention displays more defined activation of their neck muscles post training.

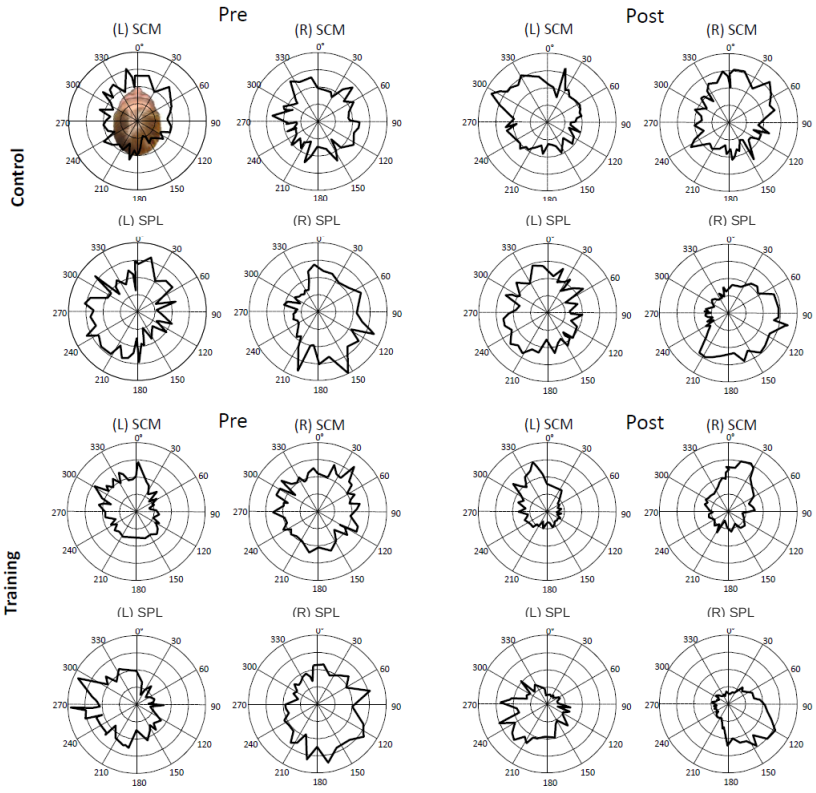


**Fig 10:** Representative directional activation curves for a patient from the intervention group and a patient from the control group performing an isometric contraction of their neck muscles in the horizontal plane at 15 N in a clockwise direction with change in force direction in the range 0-360 at baseline and at week 9. Note that at baseline both patients show undefined tuning curves of their neck muscles largely due to coactivation of the neck muscles when acting as an antagonist,

that is, activation of the SCM muscle during the extension phase of the contraction and activation of the SPL during the flexion phase of the contraction. However after the training the patient from the intervention group displays more defined tuning curves which more appropriately reflect the anatomical action of the muscle.

The group results showed that the training group had higher specificity of neck muscle activity post intervention (average across SCM and SPL: pre: 18.6 ± 9.8 %, post: 24.7 ± 14.3 %; $P < 0.05$) whereas no change was observed for the control group (pre: 19.4 ± 11.9 %, post: 18.2 ± 10.1 %; Fig 11). Similarly, the mean EMG amplitude for both the SCM and SPL during the circular contraction was reduced for the patients in the training group post intervention (average across SCM and SPL: pre: 27.4 ± 18.0 µV, post: 18.2 ± 10.2 µV; $P < 0.05$) but remained unchanged for the control group (pre: 27.7 ± 17.5 µV, post: 26.3 ± 17.3 µV).

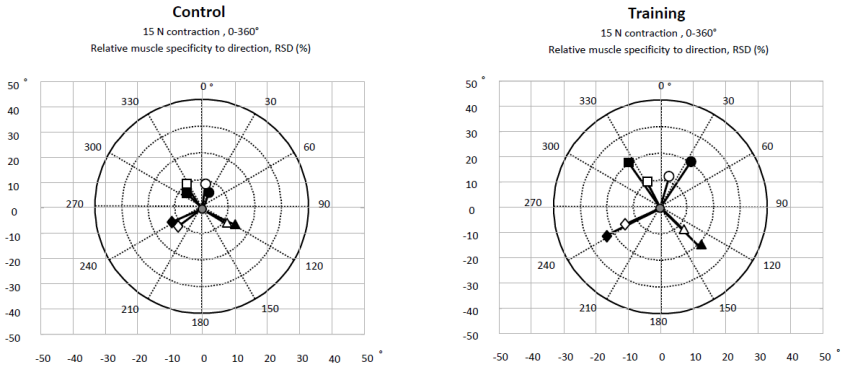


**Fig 11:** Intervention (training) and control group data for the directional vector describing the specificity of SCM and SPL muscle activity during the circular contractions performed at 15 N. Baseline data are presented in white and the follow up measurement data are in black. Note the increased values of directional specificity in the surface EMG of the SCM and SPL muscles bilaterally for the training group. Squares represent left SCM, circles right SCM, diamonds the left SPL and triangles the right SPL.

## *Maximum voluntary strength*

An interaction between group, time and direction was observed ($F = 2.7$, $P < 0.05$; Table 15). A small (9.7 ± 20.7 %) yet significant (SNK: $P < 0.05$) increase in neck flexion strength was observed for the intervention group post training (Table 15), whereas no change were observed for extension, right lateral flexion and left lateral flexion MVC's.

**Table 15:** Mean ± SD of force measured during maximum voluntary contractions of neck flexion, extension, and bilateral lateral flexion pre and post treatment. * indicates significant difference pre to post; $P < 0.05$.

| | **Intervention** | **Control** |
|---|---|---|

| | Pre | Post | Pre | Post |
|---|---|---|---|---|
| Flexion MVC (N) | 94.7 ± 24.3 | *104.6 ± 34.2 | 93.8 ± 34.0 | 88.6 ± 34.4 |

### 4.4.3 Discussion

In this study we aimed to assess whether an exercise program could enhance the directional specificity of neck muscle activity. Although several randomized controlled trials have shown the benefit of exercise for improving motor output such as strength (63, 90-92), endurance (90), range of motion (90, 92, 93) and proprioception (94), no studies have investigated whether exercise can improve such fine or skilled control of the neck muscles which underlies deficits in movement function. The Study results confirm that directional specificity can be altered by training with the post-training values similar to values of directional specificity demonstrated in healthy subjects (5, 6, 42, 95).

At the baseline measurement, patients showed undefined tuning curves of their neck muscles, indicating reduced specificity of muscle activation, and these results are consistent with those observed previously in patients with chronic neck pain of both idiopathic (Studies 1 and 2) and traumatic (42) origins. Such reduced specificity of muscle activation is largely due to increased activation of the muscle when acting as an antagonist and is associated with reduced maximal force output in patients with neck pain (Study 1).

As discussed in Study 1 and 2, reduced specificity of neck muscle activity in patients with neck pain may be the result of multiple mechanisms including direct effects of nociception on motor neuron output, direct effects of pain on sympathetic activity, reflexes originating in tissue protection and changes in motor planning.

The eight-week specific and progressive exercise training program also resulted in an immediate reduction in pain in patients with chronic neck pain. The reduction in pain reported as a result of the exercise training confirms previous findings that exercise is effective at providing pain relief for patients with neck pain both in the short (62, 90, 91, 94, 96-98) and long term (62, 90, 96, 97) . On average the training group reduced their NDI score by ~4 points. A reduction of 5 points on the NDI has previously been determined to be a clinically relevant change for uncomplicated neck pain (99). However the recommended minimal detectable change on the NDI varies between reports ranging from 10.2 points for a patient population with cervical radiculopathy (100) to 1.6 points for sample which included stable patients with recurrent neck pain (101). In this Study the average NDI score at the commencement of training was ~18 indicating moderate to severe neck pain which falls in between the average NDI score reported in the two above mentioned studies. For comparison, in previous trials examining the efficacy of exercise for chronic neck pain, significant reductions in the NDI score were associated with a change on the NDI by 3.5-5 points (98), 2.8-3.5 points (91) and 5 points (90) on average. The training group also improved on the PSFS indicating an improvement in function after the intervention. The effect size for the

NDI score was moderate to large (102) confirming that the addition of specific exercise is advantageous in the management of patients with chronic neck pain.

Although both an increase in directional specificity and a reduction in pain and disability were observed in the intervention group no correlations were found between motor control measures and clinical data (Unpublished data from Study 4).

# 5. General Discussion

This thesis advances our current understanding of altered motor control of the cervical spine in patients with neck pain. The Studies of this thesis demonstrate that patients with neck pain display reduced directional specificity of neck muscle activity and that the directional specificity of neck muscle activity can be enhanced by specific training.

Four hypotheses were tested in the thesis. 1) Directional specificity of neck muscle activity would be altered in patients with chronic neck compared to healthy controls. 2) The modulation of discharge rate of SCM motor units during multi-directional isometric contractions in the horizontal plane would be different in patients with chronic neck compared to healthy controls. 3) Reduced directional specificity of neck muscle activity would be correlated to maximum neck strength. 4) Directional specificity of neck muscle activity could be enhanced by an 8-week program of specific neck exercises.

Hypothesis 1 was confirmed as the results showed a significant difference in the directional specificity of SCM and SPL muscle activity between patients with chronic neck pain and healthy controls. This was observed as the participants performed isometric contractions with sweeping force in the horizontal plane at absolute forces of 15N and 30N. Hypothesis 4 was also confirmed as a randomized controlled trial demonstrated significantly improved directional specificity of SCM and SPL muscle activity in patients with chronic neck pain following an 8-week specific exercise program. Decomposition of the intramuscular signals recorded during isometric circular contractions with sweeping force could not be conducted reliability thus in Study 2, multiple unidirectional isometric contractions were performed separated by 45° in the horizontal plane. The results of this Study demonstrated reduced modulation of the neural drive to the SCM muscle in patients with chronic neck pain confirming hypothesis 2. However, hypothesis 3 was not confirmed since directional specificity of neck muscle activity was not correlated to average neck strength in patients with neck pain.

Cervical pain is recognized to induce neuromuscular adaptations. For example, patients with neck pain show different activation of their superficial neck flexor muscles with respect to healthy controls in standardized isometric contractions of cervical and cranio-cervical flexion (39, 83, 84). Furthermore, the activation of the deep cervical flexors is reduced in patients with neck pain and also delayed with rapid arm movements (39, 43). Co-contraction of the neck muscles is greater in patients with headache (80) and patients with neck pain

further demonstrate reduced ability to relax their neck muscles following contraction (45).

Neck pain has also been associated with alterations of neck muscle properties, including impaired micro circulation within the trapezius muscle (103), fibre transformations with an increase in the proportion of type IIC fibres (20) and fat infiltration of the neck muscles (104).

EMG tuning curves acquired from neck muscles in healthy volunteers have shown consistent focus of muscle activation with the muscle activity modulated by force (5, 6). Studies 1 and 2 were the first to investigate EMG tuning curves of the neck muscles in patients with neck pain and the results of these Studies confirm altered muscle activation with reduced directional focus of muscle activity. This finding contributes to the knowledge of impaired neuromuscular control of the cervical spine in patients with neck pain as reviewed in the introductory section of this thesis. As seen from the tuning curves in Studies 1 and 2, the patients with neck pain also show higher levels of co-activation of the SCM and SPL muscles compared to control subjects. Study 1 further demonstrated that increased antagonist activity of the SPL muscle was associated with lower neck strength and higher levels of pain and associated disability. Such disturbances in neck muscle activation indicates an altered load sharing between neck muscles which may be accompanied with increased stiffness of the spine in patients with pain (52, 105-107).

Reduced neck strength is a consistent observation in patients with neck pain (24, 25, 108, 109); however, the extent of impaired strength is highly variable across patients (26). An association between neck pain and maximum voluntary contractions has previously been reported (34); but a comprehensive study of associations between multiple factors and MVC had not previously been performed. Study 3 shows that average MVC in patients with neck pain is moderately and inversely correlated to the pain experienced by the patient during maximal contraction, to fear of movement and to some aspects of neck disability, but not to the directional specificity of muscle activity, neck muscle CSA or pain assessed at other time instances than during MVC.

A significant effect may have been "washed out" by computing the mean values in Study 3, as the averaging in Study 3 was performed across flexor and extensor muscles and only the extensors in Study 1 showed significant correlations with directional specificity. Demographics homogeneity of the small sample size in Study 1 may influence results as this group did not contain patients with minor complaints. The patients recruited in Study 1 may therefore display more linear adaptation of directional specificity to pain compared to patients in Study 3.

The regression equation between current pain and MVC may provide an explanation for the clinical observation of very rapid increases in MVC with reduced cervical pain. However this needs to be substantiated in future research.

Effective treatment of neck pain is essential; however in most cases can the structural source of pain not be identified (110). Consequently a reasonable pharmacological or surgical treatment can rarely be offered (111). Moderate evidence for the efficacy of exercise exists when exercises are performed alone and

strong evidence exists when exercise is combined with mobilization or manipulation for subacute and chronic mechanical neck disorders (61). Exercise is effective at providing pain relief for patients with neck pain both in the short (62, 90, 91, 94, 97, 98) and long term (62, 90, 96, 97). No studies have investigated whether specific neck training can alter motor control aspects such as defined activation of the neck muscles during multidirectional isometric contractions. The results of Study 4 show that an 8-week specific exercise program is efficacious for improving the directional specificity of neck muscle activity and reducing pain in the immediate term. The strength of the results may not be sufficient to impact current clinical practice (112); however the results highlights that more evidence on how neck pain, motor control and training interact should be integrated in future clinical research.

Different theories have been proposed to link pain and altered motor control. The pain adaptation theory (51) and the vicious cycle theory (50) are the most frequently discussed theories. The pain-adaptation model predicts an inhibitory effect of pain on motor neurons during agonist activity and an excitatory effect during antagonist activity (51). The pain adaptation model is partly in conflict with the results of reduced specificity of neck muscle activity in patients, since inhibition of neck muscle activity was not observed in the agonist phase of movement. The vicious cycle theory suggests increased muscle activity as a consequence of activation of group III and IV muscle afferents (50). This theory can be supported by the result of augmented neck muscle activity for patients with neck pain regardless of the direction of force. Furthermore increased activity of the superficial flexors has been commonly observed in patients with neck pain in other studies during isometric and dynamic tasks (39, 43). However reduced activity of deeper neck muscles is also reported in patients with neck (39, 42) which contradicts this theory. Rather, the interaction between pain and motor control appears task and subject specific and influenced by multiple factors as suggested by the recent theory proposed by Hodges (52). This theory suggest a complex interaction between pain and motor control and consists of 5 elements 1) redistribution of activity within and between muscles; 2) changes the mechanical behavior such as modified movement and stiffness; 3) protection from further pain or injury, or from threatened pain or injury; 4) the pain and motor control interactions is not explained by simple changes in excitability but involves changes at multiple levels of the motor system, and these changes may be complementary, additive, or competitive; and 5) has short-term benefit but has potential long-term consequences due to factors such as increased load, decreased movement, and decreased variability (52). Thus the interaction between pain and directional specificity of neck muscle activity is likely a multifactorial process, and the theory proposed by Hodges (52), appears better suited explain the findings of impaired specificity of neck muscle activity observed in this thesis; however this theory may also have shortcomings, since it does not encompass aetiology, leaning, diverse anatomy or posture.

## *Methodological Considerations*

There are several sources of variation that could have influenced directional specificity of neck muscle activity between subjects and studies. The acquired muscle activity is dependent on factors including force production, body restraint and mechanical leverage, these factors influence force production across experimental methods and may result in differences in absolute force across experimental equipment (26, 113). Normalized force may also be different between studies, as differences in leverage within the experimental equipment may affect the relation between flexor and extensor muscle activity across experimental models (26).

Variation may also influence results with repeated measures, as directional specificity and Mean EMG may vary according to subjects' ability to perform equal change in degrees per second during the circular contraction. Variability in chronic neck patients' motor control may be influenced by several non-muscular factors such as current pain according to Study 3, learning may influence agonist and antagonist muscle activity (12) and fear of pain (78). Fear of pain is demonstrated to limit MVC in Study 3.

Variations in the demographic of recruitment areas, variation in clinical criteria for neck pain diagnosis (114), variation due to aetiology and variation in healthy physiology such as MVC (23, 25, 88, 115) may affect results across studies.

EMG acquisitions are subject to a wide range of factors influencing the acquired signal such as subject preparation, electrode positioning, subcutaneous tissue, fibre orientation, number of active motor units, cross talk and signal processing methods (67).

### 5.1 Future perspectives

The understanding of the physiological basis of movement in pain remains limited (52) and extensive basic research needs to be performed to understand dynamic motion firstly in healthy subjects. As research methodology develops and standards of movement in healthy subjects become established, comparisons can be made with findings from patients with chronic neck pain. Subsequently key questions of which motor control alterations are causal, transient, pre-existing and reversible has to be resolved.

The neurophysiologic changes observed in chronic neck patients has led to numerous available treatment approaches and improved practice strategies for assessment and management of chronic neck pain has commenced; however it does not appear that a consensus will be reached in the near future (58), and at present there is no evidence to suggest that one type of training intervention is superior to the other (59, 61, 116). Future clinical research should therefore focus on how to match interventions to the needs of the patient and to identify exercise

interventions which are cost effective and easy to apply (114). Future clinical research should focus on clinical markers or thresholds for when to integrate cost effective exercises in treatment interventions (59, 114).

## *General Conclusion*

Patients with chronic neck pain demonstrate reduced focus of directional muscle activity, the results further show that substantial increased antagonist muscle activity contribute to the altered motor control for directional specificity. Specific exercises can enhance focus muscle activity, patients with chronic neck pain demonstrate after 8 weeks specific training altered muscle activity towards the healthy patterns of muscle activity. The Studies contribute to the knowledge of impaired neuromuscular control of the cervical spine in patients with neck pain and demonstrate that decreased focus of muscle activity in these patients can be enhanced by specific training.

# 6. Appendix

## 6.1 Patient and healthy maximum voluntary contraction

Patients with chronic neck pain have reduced MVC as shown in Study 1 and 2 when multiple directions of force are compared between patient and control groups (ANOVA, $P<0.05$; Table 16). Study 3 compares each direction of force between patient and control groups and the t-test results show $P<0.05$ for Extension and $P<0.005$ for Right Lateral Flexion and Left Lateral Flexion, whilst the t-test for Flexion only shoved a trend (P=0.06; Table 16).

**Table 16:** Summary of statistic test and statistics result from MVC comparisons between patient with chronic neck pain and control groups. Columns from the left: Study number, Measures compared, Statistic test applied and F, T and P, Statistic test results. L=Left and R=Right.

| Study | Measures compared | Statistic | F, T AND P |
|---|---|---|---|
| Study 1 | Flexion, Extension, L. and R. Lateral Flexion | ANOVA | F= 6.8, P<0.050 |
| Study 2 | Flexion, Extension, L. and R. Lateral Flexion | ANOVA | F= 4.7, P=0.045 |
| Study 3 | Average of Flexion, Extension, L. and R. Lateral Flexion | T-test | T=-3.092, P=0.003 |
| Study 3 | Flexion | T-test | T=-1.925, P=0.060 |
| Study 3 | Extension | T-test | T=-2.585, P=0.013 |
| Study 3 | Right Lateral Flexion | T-test | T=-3.348, P=0.002 |
| Study 3 | Left Lateral Flexion | T-test | T=-3.055, P=0.004 |

Table 17 shows the averages of patient group and control group MVC from Study 1, 2 and 3 (Flexion, Extension, Right Lateral Flexion and Left Lateral Flexion). Patients with chronic neck pain have a lower average MVC across all directions of force. Likewise the %RSD is higher across all directions in the patient group compared to the control group, excluding Study 3 Right Lateral Flexion were %RSD is 0.1% higher in the control group.

**Table 17:** Maximum MVC (N) from Study 1, 2, and 3 as mean ± SD and %RSD. From the left column: Study number, Group (Chronic neck patients and Control), Flexion, Extension, R. Lat. Flexion = Right Lateral Flexion and L. Lat. Flexion= Left Lateral Flexion.

| Study | Group | Flexion | Extension | R. Lat. Flexion | L. Lat. Flexion |
|---|---|---|---|---|---|
| Study 1 | Patient | 97.7 ± 40.4 (41.4) | 182.5 ± 77.7 (42.6) | 114.0 ± 47.6 (41.8) | 119.8 ± 49.2 (41.1) |
| | Control | 143.0 ± 41.4 (29.0) | 235.7 ± 54.6 (23.2) | 170.7 ± 55.5 (32.5) | 176.7 ± 46.0 (26.0) |
| Study 2 | Patient | 102.3 ± 39.7 (38.8) | 193.7 ± 77.2 (39.9) | 129.6 ± 47.1 (36.3) | 125.2 ± 46.5 (37.1) |
| | Control | 151.8 ± 37.6 (24.8) | 243.4 ± 56.6 (23.3) | 168.8 ± 58.5 (34.7) | 175.1 ± 48.5 (27.7) |
| Study 3 | Patient | 97.9 ± 31.9 (32.6) | 179.1 ± 48.6 (27.1) | 119.3 ± 39.0 (32.7) | 123.7 ± 36.4 (29.4) |
| | Control | 118.3 ± 36.9 (31.2) | 218.5±46.3 (21.2) | 166.0±54.5 (32.8) | 164.9±55.8 (33.8) |

Table 18 shows %RSD MVC displayed in Table 19 averaged across directions of force (Flexion, Extension, Left Lateral Flexion and Right Lateral flexion) for patients and controls. The %RSD is higher for the patient group across Flexion, Extension, Left Lateral Flexion and Right Lateral flexion (Table 18). These results indicate that the strength loss in not uniform across the patient group and that the effect of neck pain on MVC varies across patients.

**Table 18:** Averaged across %RSD for Study 1, 2 and 3 of for each direction of Flexion, Extension, Right Lateral Flexion (R. Lat. Flex) and Left Lateral Flexion (L. Lat. Flex). Rows from top: Patients with chronic neck pain, Control and Difference (Patient %RSD – Control % RSD).

| | Flexion | Extension | R. Lat. Flex | L. Lat. Flex |
|---|---|---|---|---|
| Patient | 37.6 | 36.5 | 36.9 | 35.9 |
| Control | 28.3 | 22.6 | 33.3 | 29.2 |
| Difference | 9.3 | 14.0 | 3.6 | 6.7 |

## 6.2 Pain and disability

Four baseline VAS pain measures were assessed in the thesis, two assessed VAS pain related to MVC. Perceived disability was assessed with a Danish version of the NDI (Table 19).

**Table 19**: VAS pain and disability (mean ± SD) reported in all four Studies. The columns from the left show: Study number, average pain during the active day (unspecified time frame), Average pain during the active day (past 4 weeks), Pain before MVC, Pain during MVC, NDI, Int. = Intervention group, Ctrl. = control group.

| Study | Ave. pain VAS during the day | Ave. pain VAS past 4 weeks | VAS pain before MVC | VAS pain during MVC | NDI |
|---|---|---|---|---|---|
| Study 1 | 5.1 ± 1.8 | | | | 21.6 ± 8.4 |
| Study 2 | 4.3 ± 1.5 | | | | 16.5 ± 8.8 |
| Study 3 | | 4.9 ± 1.8 | 3.8 ± 2.2 | 4.2 ± 2.1 | 17.5 ± 6.5 |
| Study 4 | | 5.3 ± 2.8 Int. | | | 18.2 ± 7.4 Int. |
| | | 5.1 ± 2.0 Ctrl. | | | 17.5 ± 6.3 Ctrl. |

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