MUSCLE ACTIVATION PATTERNS

IN POSTTRAUMATIC NECK PAIN

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MUSCLE ACTIVATION PATTERNS

IN POSTTRAUMATIC NECK PAIN

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CHAPTER 1

INTRODUCTION AND OUTLINE OF THE THESIS

INTRODUCTION

In Western industrialized societies, the development of chronic pain and related disability following a whiplash injury has become a significant public health problem. Annual incidence rates vary between 70 per 100,000 in Quebec, Canada[22] and 106 per 100,000 in Australia.[16] Not only does chronic neck pain affect patients' physical and psychological well being, it also puts a great burden on societal and health care costs. Indirect costs per whiplash patient in Canada are estimated to be around $2,500.[22,23]

Although it is generally accepted that the acute symptoms can be attributed to soft tissue injuries of the neck ("neck sprain"), there is no convincing evidence of a specific lesion that can be expected to cause chronic damage in the neck and as such be linked to chronic pain and disability. Moreover, considering the strong association of sociocultural factors such as expectation of pain[18,20] and type of compensation system[2] with longterm outcome, it is suggested that persistence of pain and disability after a whiplash trauma is multifactorial.[6]

Understanding the complex nature of the clinical entity associated with the whiplash injury, the 'Whiplash-associated Disorder' (WAD), the Quebec Task Force[22] proposed a classification system offering four grades roughly corresponding to the severity of injury. The four categories of WADs include:

- grade 0: no complaints and no physical signs
- grade 1: neck complaint of pain, stiffness, or tenderness only, but no physical signs
- grade 2: neck complaints and musculoskeletal signs
- grade 3: neck complaints and neurological sign(s)
- grade 4: neck complaints and fracture or dislocation

This injury severity classification system is generally considered as a “gold standard” in clinical practice and research, although none of these categories are based on the cause or even source of pain, but describe the clincical manifestations. Clinical guidelines for the management of acute WAD patients, including recommendations for the necessity and planning of additional diagnostic evaluation, are based on this classification.[22] In addition, the clinical relevance of this classification in relation to the outcome has been demonstrated (i.e. the rate of recovery appears to decrease with the severity grade).[23]

Despite the clinical usefulness of the classification system, there are some ambiguities in the construct of WAD grade 2, which is defined as the presence of musculoskeletal signs. These musculoskeletal signs are manifested in a *limited range of motion* putatively due to *muscle spasm*. Muscle spasm however, is an ambiguous clinical sign because the inter-examiner reliability of manual testing of muscle has shown to be poor. Simons and Mense proposed a clearer definition: "Muscle spasm is electromyographic (EMG) activity that is not under voluntary control and not dependent upon posture." [21]

Consistent with the WAD grade II definition, several studies report limitations in range of motion in neck trauma patients.[5,13,14,19] However, there are no studies that directly confirm the presence of increased electromyographic muscle activity in these patients. Moreover, the existing theories about the development of musculoskeletal pain, and whether there is an increased or a decreased level of muscle activity, are controversial.

In the 'pain-spasm-pain model', the assumption is that pain causes an *increase* in muscle activation to immobilize painful movements, with pain and increased muscle activity reinforcing one another, resulting in a 'vicious circle' of pain-muscle spasm-pain.[11,24] Although the effect of pain on muscle activation has been demonstrated, the reverse relation of muscle spasm causing pain is more complicated. Ergonomic studies confirm that sustained muscle activity is a risk factor in work related myalgia.[9,12,25,26] However, it is not clear how long it takes before persistence of increased muscle activity leads to secondary pain. As such, this pain-spasm-pain model has received a lot of criticism during the last decades.[15,17]

In contrast to this model, the 'pain adaptation model' proposed by Lund et al [15] suggests that an injury will lead to a *reduced* activity of muscles. Particularly muscles causing painful movements show this decrease. In addition, the model suggests that there is also a more general change in muscle coordination during movement with a concomitant increase of activity of antagonistic muscles. Similar to the pain-spasm-pain model, this muscle activation pattern is considered a useful reflex adaptation, protecting the injured area from further injury and pain.

In addition to these two neurophysiologically-oriented models, the cognitive-behavioral perspective, as described by the fear avoidance model, introduces a supraspinal (i.e., cognitive) influence of pain related fear on muscle activation.[27] This model suggests that subjects may develop fear of movement and physical activity, as these are (wrongfully) assumed to cause (re)injury. When movement is believed to be harmful, the generation of fear of movement may have different

effects on muscular activation. First, during physical exercise, requiring maximal performance level, fear of movement may result in a decreased muscle activity, aiming at avoidance of forceful, painful contractions.[3,4] Second, during specific movements, the ratio of relaxation and contraction of muscles may be changed in order to 'guard' the injured area.[28] Finally, in patients with a high fear of movement, the performance of a painful exercise may be perceived as a personal relevant stress stimulus. As a consequence, the induced hyper-arousal of the autonomic nervous system, may result in a psychophysiological response, manifested by an increased muscle reactivity.[7,8,1]

Each of the three models describing the interaction between pain and muscle activity have different consequences for therapeutic management. The increased muscle activity in the pain-spasm-pain model and the suggested role in the development of chronicity indicates a treatment approach that is aimed at lowering the level of muscle activity.[10] In contrast, the decrease of muscle activity in the pain adaptation model is suggested to be a useful adaptation of the neuromuscular control upon pain in that region. As such, it is questionable whether this type of muscle activation pattern needs to be treated at all. Finally, if the muscle activation pattern is considered a psychophysiological response, the treatment should include components focused at the (psychological) factors that modulate the interaction between pain and muscle activity.
Considering the different consequences for therapeutic strategies, a thorough understanding of the different muscle activation patterns in post traumatic neck pain is required.

The two main aims of this thesis are

- To clarify the characteristics of 'musculoskeletal signs' in acute and chronic post-traumatic neck pain patients.
- To determine its clinical relevance in relation to the management of acute and chronic post-traumatic neck pain patients

OUTLINE OF THIS THESIS

The starting point in this thesis was that, in accordance with the WAD injury severity system, WAD grade II would display a muscle activation pattern characterized by an increase in muscle activation. First, we investigated whether this assumption was correct. However, we did not know the optimal task condition that would demonstrate increased muscle activity. So, the aim of this first study

(Chapter 2) was to evaluate which tasks could be used to discriminate between patients designated as chronic WAD grade II and healthy control subjects. Surface electromyographic (sEMG) analysis of the upper trapezius muscles was performed during static, dynamic, and relaxation tasks. Patients with WAD II reacted with an *increase* in activity of the trapezius muscles *in response* the performance of a physical task.

We considered this 'muscle reactivity' to result from an increased psychophysiological arousal and pain. As such, we hypothesized, that this muscle activation pattern could be considered a nonspecific sign. To address this specificity, we replicated the sEMG study in neck pain patients with and without a history of traumatic whiplash injury (chapter 3). The results confirm that muscle reactivity is present in non-specific neck pain as well as WAD grade II patients. Thus, we conclude that acceleration-deceleration trauma is not an exclusive cause for the presence of muscle reactivity.

In accordance with the pain-spasm-pain model, the muscle reactivity is assumed to be part of a "causal," pain-eliciting psychophysiological mechanisms. Consequently, we hypothesized that the muscle reactivity, would be present in the acute stage following the motor vehicle accident. Furthermore, we suggested that this muscle activation pattern would persist in patients with chronic neck pain disability, and disappear during follow up in patients that recovered. These considerations were the basis for the design of a prospective study, in which we followed up a cohort of patients with acute post-traumatic neck pain due to a motor vehicle accident.

In contrast to what was expected, we found that the muscle activation patterns in patients with acute post-traumatic neck pain were not characterized by 'muscle reactivity' *in response* to the exercise, but rather by a *decreased* muscle activation *during* the performance of the exercise (Chapter 4). We conclude that the muscle reactivity is not a "causal," pain-eliciting psychophysiological mechanism, initiated in the acute phase of neck pain. We also conclude that the decrease in muscle activation level, is more in accordance with the (neurophysiological) 'pain adaptation model' *and/or* (cognitive behavioral) 'fear avoidance model.'

These results served as the basis for an additional analysis, evaluating the extent to which pain and fear of movement influence the activation patterns of the upper trapezius muscles in patients with posttraumatic neck pain disability (Chapter 5). The results show that an increased level of both fear of movement and pain intensity are *independently* associated with a decreased level of muscle

activation. Therefore we conclude that the results support *both* the 'pain adaptation' *and* the 'fear avoidance' models.

The final question addressed in this thesis was the extent to which the decreased muscle activation patterns and other variables of the fear avoidance model could predict future outcome. We hypothesized that apart from predicting outcome, characterization of the behavioral response to pain, confrontation or avoidance, would provide an initial indication of the target for intervention. Therefore, in addition to the severity of acute neck pain, assessed by pain intensity and related disability, we investigated the predictive value of three fear avoidance variables: the 'avoidance' of muscle activation of the trapezius muscles, the level of fear of movement, and the level of catastrophizing (Chapter 6). The results revealed that a simple rating of baseline neck pain disability in combination with 'fear of movement', is most effective in predicting future outcome. Compared to these variables, the predictive value of muscle activation level is modest.

I conclude the thesis with a general discussion of some methodological issues, the relevance of muscle activation patterns for the development of chronic pain (Chapter 7) and the usefulness of assessing muscle activity in the clinical setting.

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CHAPTER 2

CERVICAL MUSCLE DYSFUNCTION IN CHRONIC WHIPLASH ASSOCIATED DISORDER GRADE II (WAD II)

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ABSTRACT

Study design: In a cross-sectional study, surface electromyography (s-EMG) measurements of the upper trapezius muscles were obtained during different functional tasks in patients with a chronic whiplash associated disorder grade II and healthy control subjects.

Objectives: To investigate whether muscle dysfunction of the upper trapezius muscles, as assessed by surface electromyography, can be used to distinguish patients with whiplash associated disorder grade II from healthy control subjects.

Summary of Background Information: In the Whiplash Associated Disorder there is need to improve the diagnostic tools. Whiplash associated disorder grade II is characterised by the presence of "musculoskeletal signs". Surface electromyography to assess these musculoskeletal signs objectively may be a useful tool.

Methods: Normalised smoothed rectified electromyography levels of the upper trapezius muscles of patients with whiplash associated disorder grade II (n=18) and healthy control subjects (n=19) were compared during three static postures, during a unilateral dynamic manual exercise, and during relaxation after the manual exercise. Coefficients of variation were computed to identify the measurement condition that discriminated best between the two groups.

Results: The most pronounced differences between patients with whiplash associated disorder grade II patients and healthy control subjects were found particularly in situations in which the biomechanical load was low. Patients showed higher coactivation levels during physical exercise and a decreased ability to relax muscles after physical exercise.

Conclusions: Patients with whiplash associated disorder grade II can be distinguished from healthy control subjects according to the presence of cervical muscle dysfunction, as assessed by surface electromyography of the upper trapezius muscles. Particularly the decreased ability to relax the trapezius muscles seems to be a promising feature to identify patients with whiplash associated disorder grade II. Assessment of the muscle (dys)function by surface electromyography offers a refinement of the whiplash associated disorder classification and provides an indication to a suitable therapeutic approach.

INTRODUCTION

The Whiplash Associated Disorder (WAD), caused by an acceleration-deceleration injury most often due to a rear -end motor vehicle collision, is a serious clinical and social problem in medical practice. In a recent scientific monograph, the "Quebec Task Force" noted that there is a lack of methodological profound research on many aspects of the WAD.[19] One of the recommendations of the Quebec Task Force regarding diagnosis concerns qualitative acceptable research focusing on clinical evaluation and specialised diagnostic testing. Until now the diagnosis has been primarily based on the interpretation of personal information, the medical history of reports of pain, and some associated symptoms arising after an acceleration-deceleration trauma and physical examination. According to these non-specific signs, the Quebec Task Force proposed a clinical classification of the WAD into 5 grades (grade 0-grade IV). [19] WAD grade II is characterised by symptoms of neck pain and neck stiffness or tenderness. Signs at physical examination are limited to palpatory abnormalities of musculoskeletal structures, including decreased range of motion and point tenderness. An alternative description corresponding to WAD grade II and accepted by the Task Force is the clinical presentation of "neck pain with limited range of motion due to muscle spasm".

However, the use of palpation to assess either muscle point tenderness or muscle spasm is questionable because manually tested musculoskeletal signs have shown a poor inter-examiner reliability, [22] and very little is known about its diagnostic validity.

It is assumed that the palpable musculoskeletal signs are a reflection of a disturbance in the function of the muscles. However, there is no consensus about how pain, tenderness, and muscle consistency relate to muscle dysfunction,[1, 10, 14, 18] but the onset of pain obviously initiates neuromuscular (and behavioural) responses. Particularly in the acute pain situation, this response is considered as an adaptive reaction in order to prevent or reduce further pain or injury.[15] Persistence of muscle dysfunction in chronic pain patients, beyond the healing phase of soft tissue injury, however, may contribute to chronicity of the pain.[3] This unfavourable role of prolonged muscle dysfunction is underlined by some ergonomic studies that show that sustained muscle activity during stereotyped work is a risk factor in the development of work related myalgia. [4, 6, 20, 21]

Our research is directed toward an improvement of the diagnostic procedure in the WAD syndrome and toward a better understanding of the course of the syndrome. The goal of the present research is to investigate whether muscle dysfunction of the upper trapezius muscles, as assessed by surface-electromyography (s-EMG), can be used to identify patients with WAD-II. Therefore, is there any difference in neuromuscular activation of the upper trapezius muscles of patients with WAD II compared to healthy control subjects, during the execution of a task ?

Researchers of an analogous syndrome, chronic low back pain syndrome, have also investigated the use of sEMG to discriminate between patients and normal subjects. However, the results of this research are inconclusive[1, 14], with no agreement on the optimal task for discrimination. Therefore, we decided to investigate the neuromuscular activation under both static and dynamic conditions as well as during relaxation. By doing so we hoped to identify the most optimal task to distuinguish patients with WAD-II from healthy control subjects.

METHODS

SUBJECTS

A group of 18 patients with chronic WAD II and 19 healthy control subjects participated in the study. Demographic variables are shown in Table 1. Patients were recruited from the waiting list of a pain rehabilitation program in the Center of Rehabilitation “Het Roessingh” Enschede. All patients reported pain in the neck, head, or shoulder region for more than 6 months with a mean of more than 3 days a week. In all patients, pain started within the first 48 hours after a rear-end motor vehicle collision.Excluded were patients with head-contact trauma, patients in a coma, patients with retrograde or posttraumatic amnesia, patients reporting preexisting pain in the neck, head, or shoulder region for more than 12 weeks, and patients with either preexisting or trauma-related orthopedic or neurological signs.

The healthy control subjects were recruited from employees of the rehabilitation center. We chose healthy control subjects without any reports of pain in the neck, head, or shoulder region or without an experience of a whiplash injury to obtain the best contrast when determining any possible abnormal EMG-activity in WAD patients. We did not match the patients and controls, as it is not likely that any factors other than the acceleration-deceleration trauma could influence the EMG outcome. However, the two groups appeared to be rather similar in their mean values for age and gender ratio.

Approval of the medical ethical committee was attained and all participants signed an informed consent before start of the study.

Table 1. Demographic Variables

	Patients with WAD II	Healthy control subjects
Number of subjects	18	19
Mean Age (range) yr.	35.3 (20-56)	30.3 (21-49)
Male:female	3:15	4:15

MEASUREMENT SET-UP

Surface Electromyography

EMG activity of the upper trapezius muscle was bipolarly recorded, amplified using a differential amplifier and band pass filtered (3 Hz-10,000 Hz) to remove movement artefacts and prevent aliasing. The raw EMG was processed to a Smooth Rectified EMG (SRE) applying a double sided rectifier (12 bits, 1024Hz) and stored digitally.

Electrode placements

This study followed the recommendations of the EC-concerted action SENIAM (Surface EMG for Noninvasive Assessments of Muscles) project for proper sensor placement procedures. [5] After the skin was shaved and rubbed with sandpaper it was cleaned with 70 % alcohol. The subject was put in a sitting position to palpate the anatomical landmarks (C7, Acromion).

The electrodes (pre-gelled Ag/AgCl, type Meditrace; Graphic Controls Corp., Buffalo, NY) were placed 2 cm lateral to the midpoint of the leadline between the acromion and the easily palpable spinous process of vertebra C7. Positioning of the electrodes was parallel to the leadline with a center to center interelectrode distance of 20 mm. The reference electrode was placed over the processus spinosus of C7. After the electrode placement the electrodes as well as the cables were fixed to the skin with tape. The electrodes were connected to a portable data acquisition unit, which was attached to the waist with a belt.

EXPERIMENTAL DESIGN

Before starting the experiment, a normalization procedure was performed in order to be able to express the SRE level of the upper trapezius in relation to a reference value.[12] The SRE level was expressed as a percentage of a Reference Voluntary Electrical activation (%RVE), *i.e.*, percentage of the electrical activity obtained during a submaximal reference voluntary contraction (RVC). We used RVCs instead of maximal reference contractions (MVCs) to decrease inter- and intragroup variance due to possible confounding effects of pain, fear of pain,[11] and/or volitional regulation of performance.[16]

The RVE was assessed by averaging four consecutively recorded epochs of trapezius SRE (Figure 1, N1-4) while holding the arms straight and horizontally in 90° abduction in the frontal plane of the body, with the hands relaxed and the palms pointing downwards. Each epoch lasted 15 seconds followed by 1-minute rest. The average SRE-level was calculated for the middle 10 seconds of that epoch.

After the normalization procedure, the participants performed several simple tasks with a varying physical demand (figure 1). Measurements were obtained during the following situations:

Situation A: three static postures:

1) Sitting in a comfortable chair with head supported ,
2) sitting on a stool, and
3) standing

Situation B: a unilateral dynamic manual activity (previously described in an ergonomic study among healthy subjects): [23, 24]

The subject sat at a table and was then asked to continuously move the dominant arm/hand between three target areas by putting marks with a pencil in circles with a diameter of 70 mm. During this task the nondominant arm rested on the table without moving. The pace of 88 marks/min was kept constant with help of a metronome. This activity was performed for around 2½ minutes. During this activity the SRE level of the upper trapezius muscle of both the dominant “active” side and the nondominant “passive” side was measured at 10 seconds, 60 seconds, and 120 seconds.

Situation C: a condition requiring relaxation:

A second standing task was performed following the dynamic task (post exercise), and the SRE-levels of both the active side as well as the passive side were compared with those of the first standing task (pre-exercise).

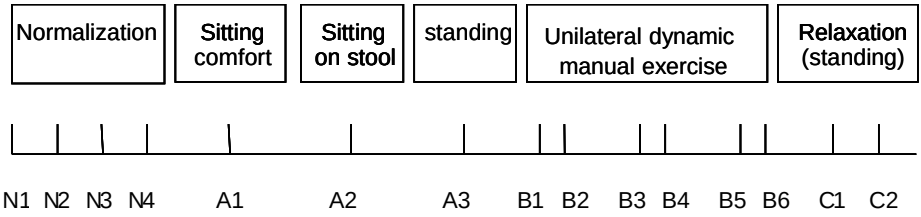

Figure 1: **Study design: Normalization procedure (N1-N4) comprising four reference contractions. Static activity (A1-A3) comprising 3 measurements during (A1) sitting in a comfortable chair with head supported, (A2) sitting on a stool, and (A3) standing. Unilateral dynamic manual exercise (B1-B6) comprising six measurements: Active and passive sides were measured at 10 seconds (B1-B2), 60 seconds (B3-B4) and 120 seconds (B5-B6). Relaxation (C1-C2) comprising two measurements: Active (C1) and passive sides (C2) were measured during a second standing task.**

OUTCOME MEASURE

During the consecutive experiments, the recorded epochs of SRE lasted 15 seconds and the SRE level was calculated for the middle 10 seconds. In both the dynamic measurement condition (situation B) and the relaxation measurement condition (situation C), the assessments produced an SRE value of the active and passive side. In case of the measurements of the three static postures (situation A), this division was not necessary, so investigators averaged both sides.

In the static and dynamic measurement conditions, differences in mean SRE levels between the two groups were computed. In the situation of relaxation, the differences in mean change from pre- to postexercise SRE level between the two groups were computed both for the active side as well as the passive side. This

yielded 11 comparisons (three during the static postures, six during the dynamic exercise, and two during relaxation).

STATISTICAL ANALYSIS

None of the variables appeared to have a skewed distribution. Mean, standard deviation and a coefficient of variation (CV) were used to describe the differences between groups. The CV is a measure of variability and is expressed as the ratio between the standard error and the difference between sample means.

The CV was used to decide which of the parameters showed best discriminative ability between the two groups. A low percentage indicates a large difference between groups and/or a small variation of the mean differences. High percentages indicate the reverse.

RESULTS

Table 2 shows the mean SRE-level (expressed as % RVE) and standard deviation of the static and dynamic measurement conditions (situations A and B), and the pre- to postexercise situations (situation C). Some data were lost because of occasional technical problems in the EMG apparatus as indicated in the N values in table 2.

SRE-levels during the two sitting conditions were lower than during standing (situation A). Furthermore, in the situation where head support was provided, as in the case of sitting in the comfortable chair, the SRE-level appeared to be lowest.

During the unilateral dynamic task (situation B) the SRE value did not change in time. There was no difference in SRE level between the beginning and the end of the task, although patients reported aggravation of pain and stiffness.
Healthy control subjects showed hardly any change in SRE level between pre-and postexercise conditions (situation C). However, patients showed considerable increase in SRE level in the active side and even more increase in the "nondominant" passive side.

In all three measurement conditions, there were large interindividual differences in both the patients and the healthy control subjects.

Figure 2 shows the mean differences and the standard error of the difference between patients with WAD II and healthy control subjects. CV was computed (table 2) to identify the measurement condition that had the best discriminative ability. In this figure, it appears that in all measurement conditions, there was a difference between the two groups, with consistently higher EMG

activity in patients than in healthy control subjects. Differences were most pronounced and CV values were lowest in the pre-to postexercise changes (active side CV= 27 %), with even more favorable values in case of the passive side (CV= 23 %). The second best measurement condition is offered by the assessment of the passive side during the dynamic task. The side that was inactive during this task showed larger differences and lower CV values than the active side.

Table 2. SRE levels of Patients and Healthy Control Subjects in THree Different Measurement Conditions

	Patients with WAD II $\bar{x}_1$ *in % RVE* (SD)	Healthy control subjects $\bar{x}_2$ *in % RVE* (SD)	Difference between WAD II and control subjects $d(\bar{x}_1 - \bar{x}_2)$ (SE_{Pooled})	CV (%) $\frac{SE_{Pooled}}{d(\bar{x}_1 - \bar{x}_2)}$
Static activity *(pat. n=18,cont n=19)*				
comfortable	2.8 (1.3)	2.0 (1.7)	0.8 (0.50)	62
stool	8.9 (6.9)	8.0 (7.3)	0.9 (2.33)	259
standing	14.1 (10.4)	9.7 (9.2)	4.4 (3.23)	74
Dynamic activity *(pat n=15, cont n=17)**				
10 seconds active side	58.6 (22.6)	54.7 (25.9)	3.9 (8.57)	220
10 seconds passive side	21.9 (15.9)	13.8 (10.8)	8.1 (4.87)	60
60 seconds active side	56.9 (22.2)	51.1 (26.4)	5.8 (8.59)	148
60 seconds passive side	21.6 (15.0)	14.0 (11.4)	7.6 (4.76)	63
120 secondsactive side	56.3 (26.0)	52.3 (26.1)	4.0 (9.23)	230
120 seconds passive side	21.3 (15.3)	13.3 (10.7)	8.0 (4.71)	59
Relaxation : pre-to post exercise change *(pat n=16, cont n=18)**				
active side	9.4 (8.2)	0.5 (5.3)	8.9 (2.40)	27
passive side	12.3 (10.0)	0.2 (5.6)	12.1 (2.83)	23

*some data were missing because of technical problems.

For the static assessments, the left and right SRE values were averaged. For the other two conditions the tasks were divided into an active and a passive side. SRE levels are expressed as a percentage of a reference voluntary electrical activation (%RVE).

SRE = smooth rectified EMG; CV = coefficients of variation; pat. = patients; cont. = control subjects

This result did not depend on the moment of assessment, as it appeared that CV values were similar in all three measurements (CV≈ 60%). The mean differences of the passive side (8.1, 7.6, and 8.0 % RVE, respectively) were not only in an absolute way much higher than the mean differences of the active side (3.9 ,5.8, and 4.0 % RVE, respectively) but even more higher when these values are considered as a proportion of the patient's activity level (approximately 40% for the passive side and approximately 10% for the active side).

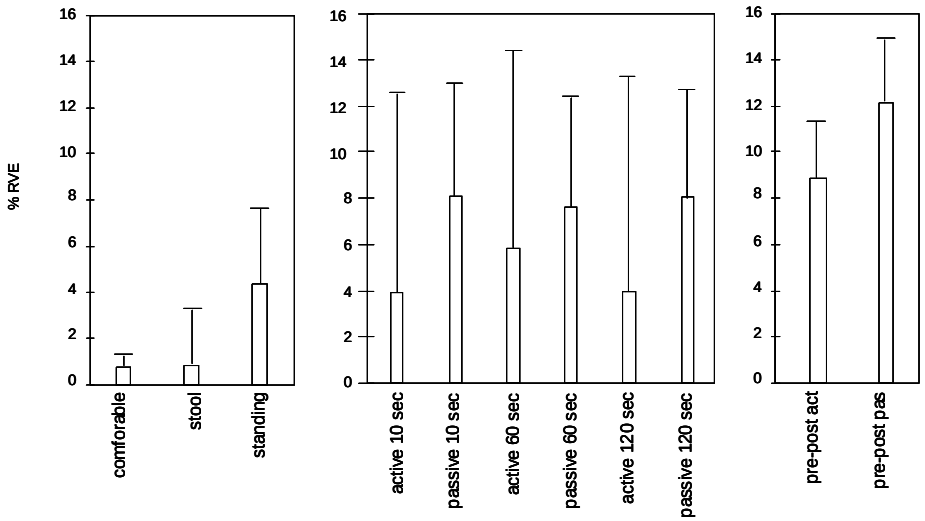

***Fig. 2*: Mean difference (+ pooled standard error) between patients with whiplash associated disorder Grade II and healthy control subjects during 11 measurement conditions. Act = active side; pas = passive side**

The mean differences between groups of the three static tasks were much lower than from those previously described. Only the condition of sitting in the comfortable chair with the head supported showed a CV value (CV= 62%) equal to those of the passive side during the dynamic task.

DISCUSSION

This study was conducted to investigate whether muscle dysfunction of the upper trapezius muscles as assessed by s-EMG, can be used to distinguish WAD-II patients from healthy control subjects.

The results of this study show that there are two particular conditions in which patients with WAD II exhibit higher EMG activity of the upper trapezius muscles than the healthy control subjects. First, during the performance of the unilateral dynamic task, the patients with WAD-II showed a substantially higher coactivation level of the upper trapezius muscles of the resting arm as compared with the healthy subjects. Second, even larger differences between the two groups appeared after having finished the dynamic task. Patients with WAD-II were not able to relax the upper trapezius muscles to baseline levels. Both findings indicate that patients with WAD II exhibit unnecessary muscle activation, particularly in situations in which there is no biomechanical demand for it. Because in the postexercise condition the inactive body side showed a similar persistence of EMG level as compared with the active side, our interpretation is that patients with WAD II have a general decreased ability to relax their trapezius muscles. Probably this can be seen as a "learned guarding response," [7] being an important aspect in the development of chronic pain.

One might question to what extend the findings of this study coincide with the description of "musculoskeletal signs" in the WAD classification of the Quebec Task Force. One of the criteria used to classify the patients with WAD II is the presence of "point tenderness" and/or "muscle spasm." Normally, muscle spasm is assessed by palpation of the compressibility (or compliance, the reciprocal of muscle stiffness) of the muscles, with the patient in a sitting or standing resting position. However, the results of this study show that during these conditions the differences between EMG levels of patients and control subjects are quite small, with a large interindividual variability. Therefore, it seems unlikely that this can explain any palpable "hardness" of muscles in the clinical diagnosis of patients with WAD II. One explanation could be that there is an effect of the muscle palpation itself on the muscle activation, suggesting an increase in EMG caused by touching the muscle. As shown in this study, the patients with WAD II show a diminished ability to relax, and an almost double-fold increase of EMG level after provocation by the physical exercise, showing a kind of muscular hyperreactivity.

It can be hypothesized that (in a clinical setting) the influence of palpation on an already aching muscle of a tensed patient has a similar provocative effect on the muscles as does the physical exercise in the experiment. This may cause an instantaneous electrogenic-induced "muscle spasm" that is felt at palpation. Another explanation might be that the palpable "musculoskeletal signs" in patients with WAD II are based on changes in viscoelastic stiffness of the muscles. In a related diagnosis, the tension type headache, it was found that 'compressibility' (as

assessed by a pressure/displacement transducer) of the trapezius muscles was found to be different from that of healthy controls.[17] However, in this study, there were no EMG assessments performed to account for a contributing effect of muscle activity during the measurements. The authors suggested that the 'hardness' is indicative of tissue edema and an increased vascular permeability caused by sustained contraction of the muscle producing hypoxia, acidosis and metabolic suppletion. Larsson et al. confirmed that in chronic pain after soft tissue injury of the cervical spine [9] and in work related trapezius myalgia [8] there is a disturbed microcirculation in painfull trapezius muscles. Therefore, the contribution of muscle activation and viscoelastic stiffness to the muscle consistency, which are in addition very likely to be interacting, remains to be clarified.

In the our opinion, muscle (dys)function, as assessed by sEMG, is a much more useful measure than compressibility to identify patients with WAD II, because it gives additional clues to a suitable therapeutic approach. In particular, sEMG measures indicate the potential benefit of sEMG feedback training.[7] Therefore, it may be helpful to refine the description of the WAD grade II by adding aspects of the muscle (dys)function of the cervical muscles.

So, sEMG as a measure of the inability to relax the upper trapezius muscles may be useful in diagnostic testing. In the literature this feature has shown to be related to cervical pain [2, 13, 23] and muscle fatigue [4, 6,20,21] and therefore supports the clinical importance of this study's findings. In two cross-sectional studies, an association was found between the inability to relax after physical exercise,[2, 13] excessive coactivation, [23] and cervical pain on the other hand . Furthermore, in research on muscle fatigue in work related neck-shoulder pain, it has been shown that sustained muscle contraction can induce signs of muscle fatigue within 5 minutes of maintaining 11% of maximal voluntary contraction[4] or by 1 hour of 5% maximal voluntary contraction[6]. In this study, the postexercise level for both the passive and active sides is approximately 26% RVE, which is comparable to approximately 5% maximal voluntary contraction. The authors did not measure the duration of the period of increased SRE level after the dynamic exercise, but if during normal activities of daily living (ADL) this reactivity as a result of physical (or psychogenic) load repeats frequently and is maintained for long enough periods, this might cause considerable discomfort and perhaps contributes to the chronicity of the WAD syndrome.

KEY POINTS

- In a cross-sectional study the goal was to investigate whether muscle dysfunction of the upper trapezius muscles, as assessed by surface-EMG, can be used to distinguish WAD-II patients from healthy control subjects.
- Particularly the decreased ability to relax the trapezius muscles appeared to be a promising possibility to identify WAD II patients.

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CHAPTER 3

CERVICAL MUSCLE DYSFUNCTION IN CHRONIC WHIPLASH ASSOCIATED DISORDER GRADE II- THE RELEVANCE OF THE TRAUMA.

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ABSTRACT

Study design: Surface electromyography measurements of the upper trapezius muscles were performed in patients with a chronic whiplash associated disorder grade II and those with nonspecific neck pain.

Objectives: To determine the etiologic relation between acceleration-deceleration trauma and the presence of cervical muscle dysfunction in the chronic stage of whiplash associated disorder.

Summary of background information: From a biopsychosocial perspective, the acceleration - deceleration trauma in patients with WAD is not regarded as a cause of chronicity of neck pain, but rather as a risk factor triggering response systems that contribute to the maintenance of neck pain. One of the contributing factors is dysfunction of the cervical muscles. Considering the limited etiologic significance of the trauma, it is hypothetized that in patients with neck pain, there are no differences in muscle activation patterns between those with and those without a history of an acceleration-deceleration trauma.

Methods: Muscle activation patterns, expressed in normalized smooth rectified electromyography levels of the upper trapezius muscles, in patients with whiplash associated disorder grade II were compared with those of patients with NSNP. The outcome parameters were the mean level of muscle activity before and after a physical exercise, the muscle reactivity in response to the exercise, and the time-dependent behavior of muscle activity after the exercise.

Results: There were no statistical significant differences in any of the outcome parameters between patients with whiplash associated disorder grade II and those with nonspecific neck pain. There was only a tendency of higher muscle reactivity in patients with whiplash associated disorder grade II.

Conclusions: It appears that the cervical muscle dysfunction in patients with chronic whiplash associated disorder grade II is *not* related to the specific trauma mechanism. Rather, cervical muscle dysfunction appears to be a general sign in diverse chronic neck pain syndromes.

INTRODUCTION

In 1995, the Quebec Task Force[14] published the first systematic review of the literature on whiplash. The authors of this report described Whiplash Associated Disorder (WAD) in the following manner: "Whiplash is an acceleration-deceleration mechanism of energy transfer to the neck. It may result from rear-end or side-impact motor vehicle collision, but can also occur during diving or other mishaps. The impact may result in bony or soft-tissue injuries (whiplash injuries), which in turn may lead to a variety of clinical manifestations." The primary symptoms of WAD include pain in the cervical region and headache. The QTF described four levels of WAD:

- grade 0: no reported problems and no physical signs
- grade 1: neck pain, stiffness, or tenderness only, but no physical signs
- grade 2: neck problems and musculoskeletal signs,
- grade 3: neck problems and neurologic signs,
- grade 4: neck problems and fracture or dislocation.

Although the general definition of WAD has been accepted broadly, it has also been challenged. [6] The breadth of the definition has profound influence on the descriptive validity, the degree to which the WAD can be distinguished from other similar disorders.[15] The descriptive validity of the WAD classification is questionable, because the two primary symptoms are nonspecific and prevalent in the general population.[1-3, 11.] This is especially true in the chronic phase (*i.e.*, symptoms persisting more than 6 months after the trauma and after healing of soft tissue injury) Thus, the extent to which the traumatic injury has etiological significance is not clear. In practice, the question is whether the diagnostic label, WAD, is clinically meaningful.

In chronic noncancer pain syndromes, the biopsychosocial model [17] provides a rationale describing how pain can become a persistent problem independent of the precise physiological etiology and extent of impairment. The biopsychosocial model proposes that three response systems (behavioral, cognitive, and psychophysiologic) each contribute to the experience of pain and chronic symptoms.[16] As such, the trauma associated with the acceleration-deceleration injury may have only limited etiologic significance in WAD. Rather the trauma triggers the three response systems comprising the biopsychosocial model.

The contribution of the psychophysiologic response system in WAD has been demonstrated previously.[13] In this study, the presence of "musculoskeletal signs",

characteristic for patients with WAD grade II (WAD II), were assessed by surface electromyography, and compared with a matched set of healthy control (HC) subjects. The muscle activity of the upper trapezius muscles was measured during three static postures, during a unilateral dynamic manual exercise, and during relaxation after a physical exercise. The results showed different neuromuscular responses in the cervical muscles between the two groups. In particular, the WAD II group displayed a statistically significant decrease in the ability to relax the cervical muscles after physical exercise. This phenomenon was defined as "cervical muscle dysfunction." The authors hypothesized that this response was provoked by psychophysiologic arousal and pain.[5, 13]

This study aimed to determine whether the muscle activation patterns observed in patients with WAD II are a manifestation of a biopsychosocial response system, similar to that detected in patients with a chronic pain syndrome. Because the crucial difference is the history of an acceleration-deceleration trauma in WAD II, the muscle activation patterns of patients with WAD II were compared with those of patients with chronic non-specific neck pain (NSNP) but no traumatic onset of symptoms. We hypothesized that in patients with neck pain, there are no differences in muscle activation patterns between between those with and those without a history of traumatic whiplash injury.

METHODS

PARTICIPANTS

The participants in this study were 19 patients with chronic WAD II, 18 patients with chronic neck pain unrelated to any traumatic event – nonspecific neck pain (NSNP), and (3) 18 healthy control (HC) subjects without any history of pain in the cervical region or chronic headaches.

Patients with Whiplash-Associated Disorder Grade II

The patients with WAD grade II consisted of consecutive referrals to a pain rehabilitation program (Het Roessingh, Enschede, The Netherlands). This part of the study involved 8 women and 11 men with a mean age of 39.1 ± 12,9 years. The mean duration of pain was 20.4 ± 15.0 months.

Whiplash associated disorder grade II is characterised by neck pain and musculoskeletal signs. The musculoskeletal signs include decreased range of motion and point tenderness. Because these particular musculoskeletal signs that discriminate WAD grade II from the other WAD grades[14] can be obtained

objectively by surface electromyography,[13] the choice was made to include a homogeneous group of patients with WAD II.

All the patients were diagnosed clinically and referred by rehabilitation physicians or physiatrists. The patients who reported pain in the neck, with possible co-existing headache or shoulder pain, averaging more than 3 days a week for more than 6 months were included in the study. On the average, patients reported that the onset of their pain began within 48 hours after a rear-end motor vehicle collision. At the time the test was performed, they were still in pain.

Patients were excluded from the study if they had head-contact trauma; coma; retrograde or posttraumatic amnesia; preexisting pain in the neck, head or shoulder region longer than 12 weeks; and either the preexisting or trauma-related orthopedic or neurologic signs.

Patients with Chronic Nonspecific Neck Pain

The patients with chronic NSNP were recruited from a neurological outpatient clinic in a general hospital. This part of the study involved 13 women and 5 men with a mean age of 47.1 ± 12.2 years. The mean duration of pain was 80.1 ± 67.7 months. The selection criteria were similar to those for patients with WAD II, but in the NSNP group there was a gradual and progressive evolution of symptoms over time and no traumatic incident or history of prior motor vehicle accidents**.**

Healthy control subjects

A group of HC subjects were included to provide normative data. The HC subjects were recruited from an unselected general population. This part of the study involved 8 women and 10 men with a mean age of 38.9 ± 2.4 years. The HC subjects had no prior experience with the rehabilitation center. They did not have any pain in the neck, head, or shoulder region and had not experienced a whiplash injury or a motor vehicle accident in the past.

The demographic variables appeared to be somewhat different among the three groups. The NSPN group reported a significant longer duration of pain than the WAD group (P=0.001, Student *t* test). Furthermore, although not statistically significant, the NSNP group was older and consisted of more women than the other two groups. The possible influence of these variables on the results was analyzed by creating subgroups with the mean values of age, gender and pain duration as cutoff points. Analysis by Mann Whitney U tests did not show any differences between young and old, male and female or longer and shorter pain duration in any of the three diagnostic categories. Before the study began, approval

of the medical ethical committee was attained, and all the participants signed an informed consent form.

EXPERIMENTAL DEVICE

Surface electromyography

Electromyographic activity of the upper trapezius muscle was recorded bipolarly, amplified using a differential amplifier and band-pass filtered (3 Hz-10,000 Hz) to remove movement artifacts and prevent aliasing. The raw electromuography was processed to a smooth rectified electromyography (SRE) applying a double-sided rectifier and stored digitally (12 bits, 1024Hz).In each part of the experiment, the electromyography was obtained in epochs of 15 seconds. This duration was chosen to obtain a good estimate of the mean level of muscle activation, without encountering large irregularities in the surface electromyography signal.[13] A period of 1 minute was chosen between the consecutive epochs.

Electrode placements

To ensure proper sensor placement procedures, we followed the recommendations of the EC-concerted action Surface Electromyography for Non-invasive Assessment of Muscles project.[8, 9] After the skin was shaved and abraded with sandpaper, it was cleaned with 70 % alcohol. The subject was seated in an upright position to allow for palpation of the anatomical landmarks (C7, acromion). The electrodes (pre-gelled Ag/AgCl, type Meditrace) were placed 2 cm lateral to the midpoint of the lead line between the acromion and the easily palpable spinous process of vertebra C7. The electrodes were positioned parallel to the lead line, with a center-to-center interelectrode distance of 20 mm. The reference electrode was placed over the processus spinosus of C7. After electrode placement, the electrodes and the cables were fixed to the skin with tape. The electrodes were connected to a portable data acquisition unit attached to the waist with a belt.

EXPERIMENTAL PROTOCOL

The experiment proceeded in 4 stages: a normalization procedure, a preexercise baseline condition, a physical exercise, and a postexercise condition. The experimental protocol is depicted in Figure 1.

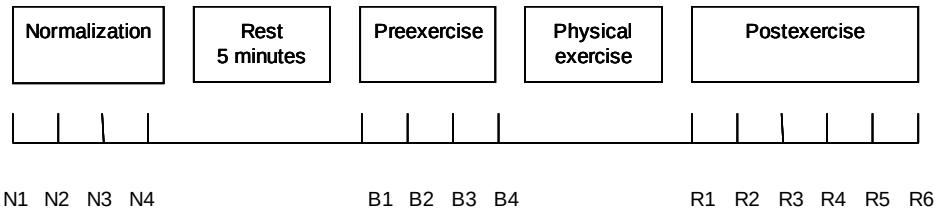

Figure 1: Study design: Normalization procedure (N1-N4) comprising four reference contractions. Preexercise condition (B1-B4) comprising four assessments with the subjects sitting in a desk chair. Postexercise condition (R1-R6) comprising six assessments with the subjects sitting in a desk chair. Between the consecutive assessments, there was a 1-minute rest.

Normalization procedure

Smooth rectified electromyography signals were normalized to decrease interindividual differences. The SRE-level was expressed as a percentage of reference voluntary electrical activation (*i.e.,* percentage of the electrical activity obtained during a submaximal reference voluntary contraction.[12] Submaximal rather than maximal reference voluntary contractions were used to decrease intergroup and intragroup variability caused by possible confounding effects of pain, fear of pain, and volitional regulation of performance.[12]

Reference voluntary electrical activation was assessed by averaging four consecutively recorded epochs of trapezius SRE (Figure 1; N1-4) while the arms were held straight and horizontally in 90° abduction in the frontal plane of the body, with the hands relaxed and the palms pointing downward.

Pre-exercise baseline condition

After five minutes of rest, the subjects assumed a sitting position in a desk chair for the measurements of baseline muscle activity. Four epochs of upper trapezius SRE (B1-B4) were obtained with the back supported, the hips and knees at 90^0, and the hands resting in the lap.[13] Four surface electromyography epochs were obtained for a good estimate of the mean muscle activation level..

Physical exercise

Immediately after the baseline measurements, a unilateral dynamic exercise was performed. During this exercise, the subject sat in a desk-chair at a table. The

subject was asked to move his or her dominant arm (active side) continuously between three target areas by marking circles with a diameter of 70 mm using a pencil. During this task, the subjects were instructed to rest their nondominant arm or hand (passive side) on the table without moving it. A metronome was used to maintain a constant pace of 88 marks per minute. After a short explanation and 20 seconds of practice, the subjects performed this activity for approximately 2 minutes. [20, 21]

Post-exercise condition

After the exercise, the level of muscle activity was obtained by another six epochs of surface electromyography (R1-R6) for use in studying the time course of muscle relaxation.

OUTCOME MEASURES

The normalized mean SRE-level was calculated for the middle 10 seconds of each recorded epoch for both the active and passive sides. Four parameters were used to study the muscle activation patterns of the upper trapezius muscles:

1. Muscle reactivity computed as the mean preexercise SRE level subtracted from the mean postexercise SRE level.
2. Time-related recovery pattern of muscle activity, analyzed by using regression on the six SRE values obtained after the exercise.
3. Mean level of preexercise muscle activity computed by averaging the four baseline SRE values.
4. Mean level of the postexercise muscle activity computed by averaging the 6 SRE values obtained after the exercise.

These outcome parameters were used to compare patients who had WAD II with those who had NSNP. To verify the ability of the experimental design to provoke muscle reactivity, differences in muscle activation patterns between the patients with WAD II and the HC subjects were also tested.

STATISTICAL ANALYSIS

Differences between the average levels of muscle reactivity as well as differences in the average preexercise and postexercise SRE levels between the patients with WAD II and the other two groups were analyzed with Mann-Whitney *U* tests. Non-parametric tests were used because the distributions of the individual SRE levels were skewed.

The time-related recovery pattern of each subjects' post-exercise muscle activity was determined by univariate linear regression analysis. In each diagnostic category, the proportion variance (R^2) explained by the factor time and the β-coefficient for the individual regressions was averaged to one value. Differences between the slopes of the averaged regression plots for the patients with WAD II and the other two groups were analyzed by analysis of variance.

RESULTS

Visual inspection of the data in Figure 2A and 2B shows a similar irregular pattern over the course of muscle activity on the active and passive sides (Figure 2A and 2B) for both the patients with WAD II and those with NSNP. The course for the HC subjects, however, appears to be less variable in the consecutive points in time, and the neuromuscular activation to be systematically lower in amplitude.

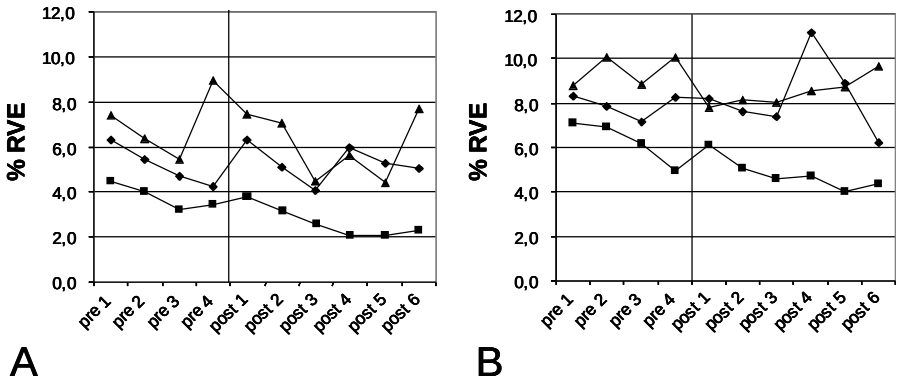

Figure 2. A, Course of pre- and postexercise muscle activation of the arm, involving a unilateral dynamic manual exercise (active side) for patients with whiplash associated disorder grade II (WAD II [¨], n=19), patients with nonspecific neck pain (NSNP [5], n=18) and healthy control subjects (HC [<], n=18). The four pre-exercise and six post-exercise smooth rectified electromyography levels are expressed as a percentage of reference voluntary electrical activation (% RVE). B, Course of pre- and postexercise muscle activation of the resting nondominant arm (passive side) for patients with chronic WAD II, patients with NSNP, and HC subjects. The four preexercise and six postexercise SRE levels are expressed as % RVE.

DIFFERENCES BETWEEN CHRONIC WHIPLASH ASSOCIATED DISORDER GRADE II AND CHRONIC NON SPECIFIC NECK PAIN

No differences were found between the patients with WAD II and patients with chronic NSNP in terms of muscle reactivity (Table 1). However, the patients in the latter group showed a tendency to relax cervical muscles after the exercise, whereas the patients with WAD II showed a slight increase in muscle activity. This difference in reactivity was most prominent in the arm that performed the exercise (active arm). In this respect, the patients with NSNP resemble the HC subjects, who also showed this tendency to relax after the exercise.

Table 1: Differences in levels of muscle reactivity and pre-and post exercise muscle activity in two groups of neck pain patients and a group of healthy control subjects

Measurement condition	Measured side	WAD II (N=19)		NSNP (N=18)		HC (N=18)		Mann-Whitney U test	
		%RVE	SD	%RVE	SD	%RVE	SD	WAD II NSNP	WAD II HC
Muscle-reactivity	Active	0.1	3.9	-0.9	8.5	-1.1	3.9	.06	.01
	Passive	0.4	2.5	-1.0	4.8	-1.5	4.5	.98	.21
Pre-exercise muscle activity	Active	5.2	7.8	7.0	9.1	3.8	4.0	.74	.85
	Passive	7.9	12.1	9.5	10.0	6.3	6.3	.49	.47
Postexercise muscle activity	Active	5.3	5.0	6.1	7.1	2.6	1.1	.86	.06
	Passive	8.3	10.8	8.5	9.6	4.8	4.7	.86	.09

Smooth Rectified electromyography levels are expressed as a percentage of a reference voluntary electrical activation (%RVE). Preexercise muscle activity was computed by averaging the four baseline measurements. Postexercise measurements were computed by averaging the six measurements during the recovery phase. Muscle reactivity was computed by subtracting the mean post-exercise from the mean pre-exercise level of muscle activity. WAD II = whiplash associated disorder grade II; NSNP = non specific neck pain; HC = healthy control subjects;SD = standard deviation

In both neck pain groups, there appeared to be no consistent pattern of muscle activity level during the post-exercise phase (figure 2A and 2B). Consequently, the regression analysis showed a rather low proportion of variance (R^2) explained by the factor time. Nevertheless, time was found to have a significant influence on the course of muscle activity, as assessed by one sample Student *t* test on the R^2 values. This was true for both the active and passive sides, which displayed *P* values less than .001 (Table 2). With the exception of the passive side in the NSNP group, the β-coefficients all were negative, indicating that the

initial increase in muscle activity, provoked by the physical exercise, was followed by a tendency to relax during the postexercise phase as time progresses. However, the rather low values of the averaged β-coefficients and the high standard deviations indicate that the time dependent change in the level of muscle activity for the group level is not impressive, and that there also are individuals who do not show this decline. Assessments with analysis of variance showed no statistically significant differences between the slopes of the three groups.

On the average, the patients with chronic NSNP showed slightly higher preexercise and postexercise muscle activity levels than the patients with WAD II. However, the intersubject variability in both groups was high. Consequently, no statistical significant differences between the two groups were found.

Table 2: Time- related recovery pattern of post exercise muscle activity:

Arm side	WAD-II		NSNP		HC		Difference in β-coefficient
	r^2	β-coefficient	r^2	β-coefficient	r^2	β-coefficient	ANOVA
Active	.28 (.24)*	-.10 (1.35)	.29 (.24)*	-.16 (3.0)	.38 (.29)*	-.32 (.44)	.941
Passive	.26 (.28)*	-.06 (.50)	.42 (.26)*	.33 (1.9)	.33 (.24)*	-.34 (.83)	.252

r^2 = proportion of variance explained by time; ß -Coefficient = slopes of the linear refression model; ANOVA= Analysis of variance; WAD II = whiplash associated disorder grade II, NSNP = non specific neck pain, HC =healthy control subjects.

* =statistically significant different from zero ($p \leq .001$, one-sample Students t-test).

Values are reported as mean (standard deviation).

DIFFERENCES BETWEEN PATIENTS WITH CHRONIC WHIPLASH ASSOCIATED DISORDER GRADE II AND HEALTHY CONTROL SUBJECTS

There was a difference in muscle reactivity between the patients with WAD II and the HC subjects (Table 1). The patients with WAD II demonstrated greater reactivity, with a small increase in muscle activity in response to the exercise (Figure 2), whereas the HC subjects reacted with a decline in muscle activity. This difference was statistical significant on the active side. The decline in muscle activity among the HC subjects, rather than the increase among the patients with WAD II accounts for most of the observed differences.

Muscle relaxation after the exercise tended to progress during the 6 post exercise measurements among HC subjects (Figures 2A and 2B). The larger

proportion variance (R^2), explained by time and the higher and negative β-coefficients of the regression plot, confirm the conclusion that as time progresses, the HC group was better able to relax the muscles after provocation with a physical exercise than the two neck pain groups.

As observed in Figures 2A and 2B and calculated from the regression equations, the influence of time in the HC group caused a decrease of almost one half from the preexercise level of muscle activity, whereas the other groups stayed at about the same high level.

There were no differences between the patients with WAD-II and the HC subjects in the comparison between the mean pre- and postexercise muscle activity levels. However, the postexercise muscle activity level in the patients with WAD-II was almost twice as high as that in the HC subjects (table 1). These differences, however, only approached statistical significance (active side $P = 0.06$, passive side $P = 0.09$).

DISCUSSION

From a biopsychosocial perspective, the acceleration-deceleration trauma in patients with WAD is not considered as a cause of chronicity of neck pain, but rather as a risk factor triggering response systems that contribute to the maintenance of neck pain. One of the contributing factors is cervical muscle dysfunction, characterized as inability to relax the cervical muscles after a physical exercise. This is thought to be provoked by psychophysiologic arousal and pain.[5, 13]

This study investigated the etiological significance of acceleration-deceleration trauma for the presence of cervical muscle dysfunction in the chronic stage of the WAD II. The results suggest that acceleration-deceleration trauma is not an exclusive cause for cervical muscle dysfunction. Therefore, cervical muscle dysfunction appears to be a general sign of chronic neck pain that is not specific for WAD II. As such, it does not contribute to the descriptive validity of WAD.

In this study, the abnormalities in muscle responses in this study can be explained by the cognitive-behavioral model of "fear of movement/(re) injury." [18] According to this model, subjects may acquire fear of movement and physical activity, because these are (wrongfully) assumed to cause (re) injury. This fear leads to guarding of the injured area and to a decreased ability to relax the muscles.

In a study of patients with chronic low back pain, the guarding response of the lumbar paraspinal muscles was demonstrated during a specific dynamic flexion movement. In contrast to pain-free control subjects, the patients with chronic low back pain demonstrated an inability to relax the paraspinal muscles when bending

from standing to a fully flexed position.[19] The methods in the current study were somewhat different. Muscle activation was assessed after rather than during exercise so ability to return to a preexercise muscle activation level could be studied. In both studies however, the differences between the patient group and the HC group was based on the inability to relax muscles in a painful condition.

The contribution of fear avoidance and muscle reactivity to the presence of pain has never been studied. However, such a study may yield clinical information on the process of becoming a patient with chronic WAD. The relevance of these factors is being investigated in additional experimental studies.

The results of the present study are consistent with those of earlier studies,[13] in which patients with WAD II also showed an increase in muscle activity in response to a physical exercise. In contrast, this increase was almost two-fold in the authors' earlier study. The most important difference between the methods used in the current study and those used in the earlier study is that the previous subjects performed a set of consecutively executed tasks, whereas only one specific task was used in the current study. It is likely that this change in task load induced less provocation and consequently smaller muscle responsiveness in patients with WAD II. This may also explain the skewed distribution of the SRE values, suggesting that only a subset of the sample demonstrated clear muscle reactivity. Detailed inspection of the individual subject data, suggests a subdivision of the patients in the two groups, with one subgroup demonstrating low muscle activity levels and weak or no reaction in response to the exercise, and the other group demonstrating irregular but high muscle activity levels and a stronger reaction to the exercise. In this experiment, it was not possible to characterize the two groups further, but it would be worthwhile to direct future studies toward the role that fear of movement/(re)injury plays in this subdivision.

The clinical significance of increased muscle activity in response to physical or mental load is underscored by ergonomic studies, indicating that this is associated with (secondary) myalgia [4, 21] and signs of muscle fatigue. [7, 1] The clear tendency of patients with WAD II to show both higher and longer muscle activation patterns in reaction to a physical load, suggests that they are involved in a vicious cycle that contributes to, and is maintained by (secondary) muscle pain. Such abnormal muscle activation behavior strongly suggests that intervention programs should focus on the relaxation of myalgic muscles, for example by means of electromyography feedback.

KEY POINTS

- It appears that the cervical muscle dysfunction in patients with chronic whiplash associated disorder grade II is not related to the specific trauma mechanism.
- Cervical muscle dyfunction appears to be a general sign in diverse chronic neck pain syndromes.

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CHAPTER 4

CHRONIC NECK PAIN DISABILITY DUE TO AN ACUTE WHIPLASH INJURY

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ABSTRACT

Several theories about musculoskeletal pain syndromes such as Whiplash Associated Disorder (WAD) suggest that pain and muscle activity interact and may contribute to the chronicity of symptoms. Studies using surface electromyography (sEMG) have demonstrated abnormal muscle activation patterns of the upper trapezius muscles in the chronic stage of WAD (grade II). There are, however, no studies that confirm that these muscle reactions are initiated in the acute stage of WAD, nor that these muscle reactions persist in the transition from acute neck pain to chronic neck pain disability.

We analysed the muscle activation patterns of the upper trapezius muscles in a cohort of 92 subjects with acute neck pain due to a motor vehicle accident (MVA). This cohort was followed up in order to evaluate differences in muscular activation patterns between subjects who have recovered and those subjects who have not recovered following an acute WAD and developed chronic neck pain. sEMG parameters were obtained at 1, 4, 8, 12, and 24 weeks after an MVA. The level of muscle reactivity (the difference in pre- and postexercise EMG levels) and the level of muscle activity during an isometric and a dynamic task were used as EMG parameters.

The results revealed no elevated muscle reactivity either in the acute stage, or during the follow up period. The results of both the isometric and dynamic task, showed statistically significant different EMG levels between four neck pain disability subgroups (analysis of variance reaching *P*-levels of 0.000), with an inverse relationship between the level of neck pain disability and EMG level. Furthermore, follow up assessments of the EMG level during these two tasks, did not show a time related change.

In conclusion, in subjects with future disability, the acute stage is characterized by a reorganization of the muscular activation of neck and shoulder muscles, possibly aimed at minimizing the use of painful muscles. This change of motor control, is in accordance with both the (neurophysiological) 'pain adaptation model' and (cognitive behavioural) 'fear avoidance model.'

INTRODUCTION

In whiplash-associated disorders (WAD), an acceleration deceleration mechanism of energy transfer to the neck may lead to a variety of clinical manifestations.[25] The primary symptoms of WADs include pain in the cervical region and headache. It is generally accepted that in the acute stage these symptoms are attributed to soft tissue injury in the cervical region. However, there is controversy as to whether chronic pain disability can be related solely to the soft tissue injury resulting from the accident.[8,27] Non-physical factors, such as expectation of pain[14,20,24] and type of compensation system[5] have also been suggested to play a role in the long-term outcome. Moreover, the primary symptoms neck pain and headache are non-specific and prevalent in the general population,[1,3,4] further emphasizing the unclear etiological significance of the traumatic injury in chronic WAD.

For a better understanding of chronification of acute symptoms following a whiplash injury it is necessary to consider biological, psychological, and social factors integrated within a biopsychosocial model.[8] Such a model provides a rationale describing how pain can become a persistent problem independent of the precise physiological etiology and extent of impairment. A biopsychosocial model proposes that three response systems, behavioral, cognitive, and psychophysiological response, each contribute to the experience of pain and chronic symptoms.[30] As such, the trauma associated with the acceleration-deceleration injury triggers a change in the three response systems comprising the biopsychosocial model.

The contribution of the psychophysiological response system in chronic WAD has been demonstrated in two previous experiments.[18,19]. One study showed different muscular responses in the upper trapezius muscles between a group of WAD patients and a matched set of healthy control subjects. In particular, the WAD group reacted with an increase in muscle activity in response to the performance of a dynamic physical exercise, indicating a decrease in the ability to relax the cervical muscles after being subject to a physical load.[18] In a second study this muscle 'hyperreactivity' also appeared to be present in a group of non-specific neck pain patients without a traumatic onset. Nederhand et al.[19] concluded that this muscle activation pattern is not related to the specific mechanism associated with a trauma. The assumption in these studies in chronic WAD is that the increase in muscle activation level is a learned behavior, initially arising during the acute phase

as a protective reaction to immobilize painful movements of the injured tissue. A further assumption is that pain and increased muscle activity reinforce one another, resulting in a 'vicious circle' of pain -muscle spasm- pain.[13,29] There are, however, no scientific studies that confirm this protective role of cervical muscles in the acute phase of WAD, nor is there any evidence of the persistence of this increased muscle activity in the transition of acute to chronic neck pain disability.

In the present, prospective, longitudinal study we analysed the neuromuscular activity of the upper trapezius muscles in a cohort of patients with acute neck pain due to a motor vehicle accident (MVA) over a period of 24 weeks, beginning 1 week after the accident. This cohort was followed in order to assess differences in course of neuromuscular activity of the upper trapezius muscles in people who have recovered and those who have not have recovered and developed chronic pain disability after a whiplash injury.

METHODS

SUBJECTS

The study population consisted of patients admitted to the emergency room of a general hospital following a MVA, between July 1999 and December 2001. Subjects were considered eligible if they were between 18 and 70 years old and reported of pain in neck or head region that started within 48 h of the accident.

Table 1: demographic and collision related variables

Mean age (range), yr	34.5 (18-63)	
male:female	31:67	
Site and estimated speed of collision*	N	km/h (sd)
front[1]	16	56.1 (28.7)
rear[2]	71	48.2 (18.9)
side[3]	11	62.7 (27.3)

N= Number of subjects; km/h= speed in kilometres per hour; sd= standard deviation; * Depending on the direction of travel and the site of impact of cars, namely whether head on, side or rear collision, the speed at the time of collision was estimated by: 1=the addition of the two speeds, 2=the difference in the speed of the two cars, 3=the speed of the car colliding from the side

Furthermore, there should have been some form of acceleration or deceleration of the motor vehicle, caused by colliding with another vehicle or a stationary object (e.g., a wall, traffic light).
Subjects with signs of a concussion, retrograde or post-traumatic amnesia, serious injuries such as fractures, traumatic internal organic pathology, or any neurologic signs were excluded. To be included, subjects had to be able to speak and read the Dutch.

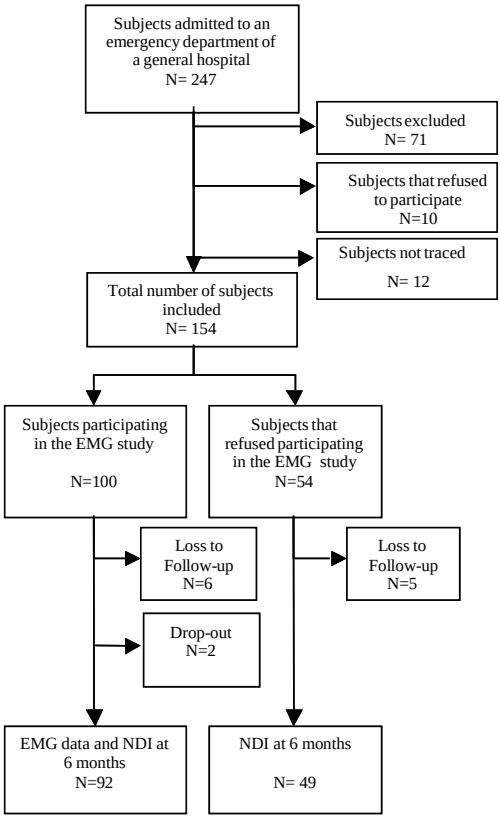


Figure 1: Patients participating in the study

During their visit to the emergency room, subjects were assessed clinically by a consultant surgeon and screened for exclusion due to serious injuries. Eligible subjects were then asked to give permission to be contacted by the investigators

for further screening on the selection criteria. Once serious injuries were ruled out in the emergency room, routine treatment consisted of analgesic medication, and advice to rest for a few days, and advice to visit a general practitioner if the symptoms persisted.

Before the study began, approval of the medical ethical committee was attained and all the participants were asked to complete an informed consent form. Of the 247 admitted patients ten refused to participate, 71 failed to meet the inclusion criteria, and 12 could not be traced because of unknown telephone number or address. One hundred of the 154 subjects eligible for the study agreed to participate in the study. The remaining 54 subjects completed questionnaires and consented to be contacted in 6 months, but refused to participate in the EMG study. At baseline demographic and collision related information were obtained (Table 1). The functional status as assessed by the Neck Disability Index (NDI)[31] was used to divide the cohort into four different outcome categories at 24 weeks: recovered 0-4, mild 5-14, moderate 15-24, and severe/complete 25-50. Smooth Rectified surface Electromyography (SRE) recordings of the upper trapezius were obtained in order to assess the muscle activity of the upper trapezius muscles in the acute phase and during the four follow-up periods.
We compared this latter group to the participants to determine if selection bias occurred (Figure 1).

STUDY DESIGN

The study was conducted as a prospective, longitudinal design. Subjects were assessed five times with baseline assessment performed within 1 week, and follow-up assessments 4, 8, 12, and 24 weeks after the MVA (Figure 2).

DESCRIPTION OF PHYSICAL DISABILITY

Neck Disability Index (NDI)

The NDI is a 10-item self-reporting instrument for the assessment of physical disability of subjects with neck pain, particularly from whiplash-type injuries.[31] Each item is scored from 0 to 5. The index was developed as a modification of the Oswestry Low Back Pain Disability Index.[7] The NDI has been shown to have a high degree of test-retest reliability, internal consistency, and acceptable level of validity being sensitive to severity levels and to changes in severity over time.[23,31] Disability categories for the NDI are: 0-4 = no disability, 5-14 = mild, 15-24 = moderate, 25-34 = severe, above 34 = complete. A 5-point change is required to be clinically meaningful.[28]

Because of small numbers the 'completely disabled group' (n=2) in this study was classed under the 'severe disabled group.'

EMG RECORDINGS AND ANALYSES

Experimental Set-up

Surface Electromyography (sEMG) of the upper trapezius muscle was recorded bipolarly, amplified using a differential amplifier and band pass filtered (3 Hz-10.000 Hz) to remove movement artifacts and prevent aliasing. The raw EMG was processed to an SRE applying a double-sided rectifier and stored digitally (12 bits, 1024Hz). After the skin was shaved and abraded with sandpaper it was cleaned with 70 % alcohol. The subject was seated in an upright position to allow for palpation of the anatomical landmarks (C7, Acromion). In order to ensure proper sensor placement procedures, we followed the recommendations of the European Community concerted action SENIAM (Surface EMG for Non-invasive Assessment of Muscles) project.[10,11]. The electrodes (pre-gelled Ag/AgCl; type Meditrace, manufactured by Graphic control corporation, Buffalo, NY, USA) were placed 2 cm laterally to the midpoint of the lead line between the acromion and the easily palpable spinous process of vertebra C7. The electrodes were positioned parallel to the lead line with a center-to-center inter-electrode distance of 20 mm. The reference electrode was placed over the processus spinosus of C7. The electrodes as well as the cables were fixed to the skin with tape and connected to a portable data acquisition unit.

In each phase of the study, the EMG was recorded in epochs of 15-s duration. Of each epoch one mean value of the SRE was calculated of the middle 10 s of the signal. This duration was chosen to obtain a good estimate of the mean level of muscle activation, without encountering large irregularities in the surface EMG signal. A period of 1 min was chosen between the consecutive epochs.

The experiment consisted of four stages: a baseline pre-exercise at rest phase, an isometric physical exercise phase, a dynamic physical exercise phase, and a post- exercise at rest phase (Fig. 2).

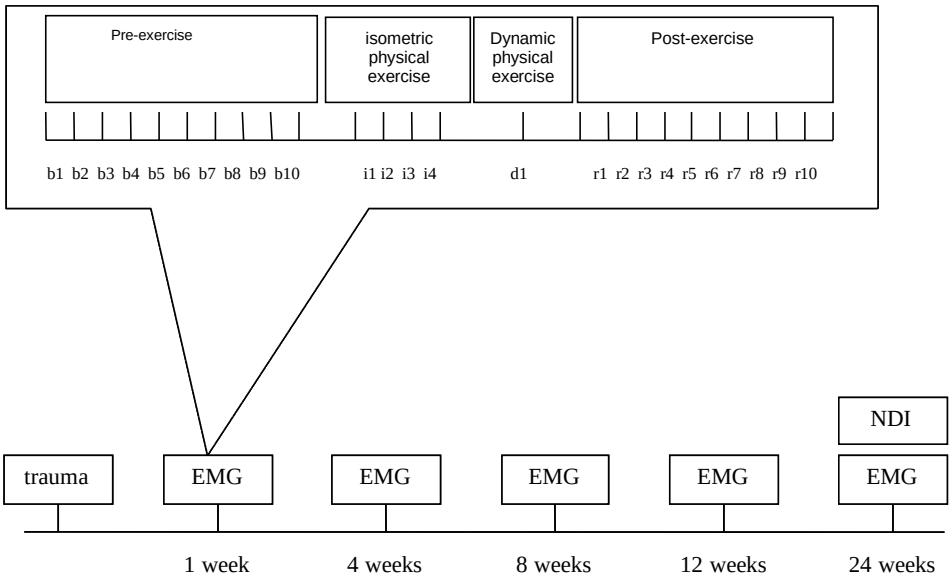


Figure 2: Study design: Electromyographic (EMG) assessments during 24 weeks follow-up after a MVA. Pre-exercise condition (b1-b10) comprising 10 EMG assesments during sitting in a desk chair. Isometric muscle activity (i1-i4) comprising 4 EMG assessments during sitting in a chair with the arms held horizontally. Dynamic muscle activity (d1) comprising 1 EMG assessment during the performance of a unilateral dynamic exercise. Post-exercise condition (r1-r10) comprising 10 EMG assesments again during sitting in a desk chair. The Neck Disability Index (NDI) is assessed at 24 weeks.

Pre-exercise baseline condition (b1-b10): The subjects maintained a sitting position in a desk-chair with the back supported, the hips and knees in 90º flexion and the hands resting in the lap. Ten epochs of upper trapezius SRE were obtained.

Isometric physical exercise (i1-i4): The subjects still assumed a sitting position in a desk-chair with the back supported, the hips and knees in 90^0 flexion, but now with the arms held straight and horizontal in 90° abduction in the frontal plane of the body, with the hands relaxed and the palms pointing downwards. Four epochs of upper trapezius SRE were obtained.

Physical exercise (d1): Immediately after the isometric exercise, a unilateral dynamic exercise was performed. During this exercise, the subject sat in a desk-chair at a table. The subject was asked to move his or her dominant arm (active side) continuously between three target areas by marking circles with a diameter of 10 mm using a pencil. During this task the subjects were instructed to rest their non-dominant arm (passive side) on the table without moving it. A metronome was used to maintain a constant pace of 88 marks per minute. After a short explanation and 20-s of practice the subjects performed this activity for approximately 2 min. After 1 minute 1 epoch of upper trapezius SRE activity was obtained.[18,33,34]

Post-exercise condition (r1-r10): After the dynamic exercise, the level of muscle activity was obtained by another ten measurements equal to the ten epochs of upper trapezius SRE obtained during the pre-exercise condition.

EMG Analysis

Three parameters were used to study the muscle activity:

1. The muscle reactivity, computed as the mean pre-exercise SRE level subtracted from the mean post-exercise SRE level. Elevated muscle reactivity was considered present if this computation resulted in a positive value, indicating an increase in muscle activity in response to the exercise. It was considered normal if a subject showed a value less than zero.[18,19]
2. The isometric muscle activity, computed as the mean muscle activity during the performance of the isometric physical task
3. The dynamic muscle activity, namely the muscle activity during the performance of the dynamic physical task.

Both left and right trapezius muscles were assessed by SRE. The active side is defined as the side (left or right), with which the subject chose to perform the dynamic task. This subdivision was consequently maintained in all of the EMG parameters.

STATISTICAL ANALYSES

Data were analyzed using the SPSS Statistical Package for Windows 10.0 (SPSS Inc. Headquarters, Chicago, Illinois). Non-parametric Mann-Whitney U test was used to determine whether the NDI score at 24 weeks follow-up was different

between the group of participants and the group of subjects that refused to participate in the EMG study.

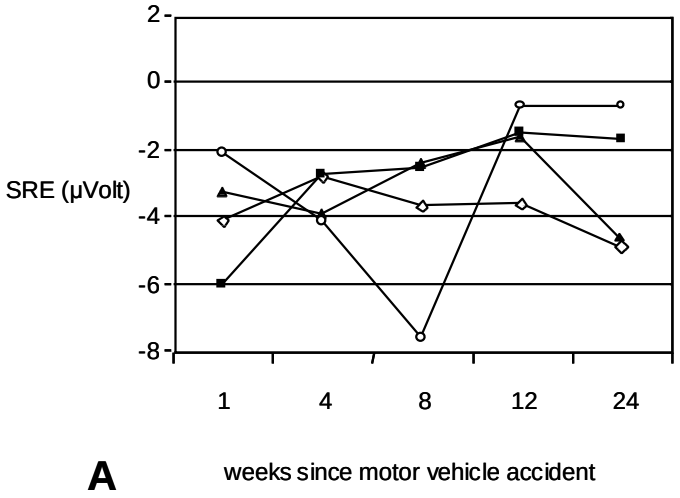
The differences in course of muscle activity patterns between the four NDI categories were analysed with two-way analysis of variance (ANOVA) for testing on main effects of NDI category and change in muscle activity level over time. P values < 0.05 was considered statistically significant. Post hoc testing of differences between each pair of means was adjusted for by a Bonferroni procedure.

RESULTS

A follow-up rate of 91.6 % was achieved for the outcome measure as assessed by the NDI. At 24 weeks the mean NDI score of the group of 92 subjects that participated in the study (NDI = 10.3, SD 10.6) did not differ significantly from the group of 49 subjects that refused (NDI = 8.0, SD 10.0) (Mann-Whitney U test: $Z = -1.213$, $p=0.225$). Thus, it appeared that selection bias did not occur. Twenty-four weeks post-injury the subjects were classified based on their scores on the NDI as: 43 recovered, 17 mildly, 21 moderately, and 11 severe/completely disabled. Six subjects were lost to follow up, without known reason, 1 subject dropped out because of a second accident and the results of 1 subject were not considered in the analysis because of suspected malingering. The EMG activity in the acute phase was obtained on average 7.9 (SD 3.5) days after the MVA.

THE COURSE OF MUSCLE REACTIVITY DURING 24-WEEKS FOLLOWING A MVA

The course of muscle reactivity of the four NDI subgroups is illustrated in Figure 3a and 3b. The results of an ANOVA showed no significant differences in level of muscle reactivity between the four NDI subgroups, nor change of muscle reactivity over time (Table 2). Moreover, in the acute phase as well as during each of the follow up periods, the levels of muscle reactivity of both the active (Figure 3a) and passive side (Figure 3b) of all four NDI subgroups are *below* zero, indicating a normal ability to relax the upper trapezius muscles after performing the physical exercise. The results of this analysis indicate that, regardless of the extent of final disability, there was no inability to relax the trapezius muscles during 24 weeks of follow-up after the MVA. Furthermore, ANOVA showed no differences between the 4 NDI subgroups in level of the pre-and post exercise muscle activity (Table 2). In addition, no changes over time were found of the pre and post exercise muscle activity levels.



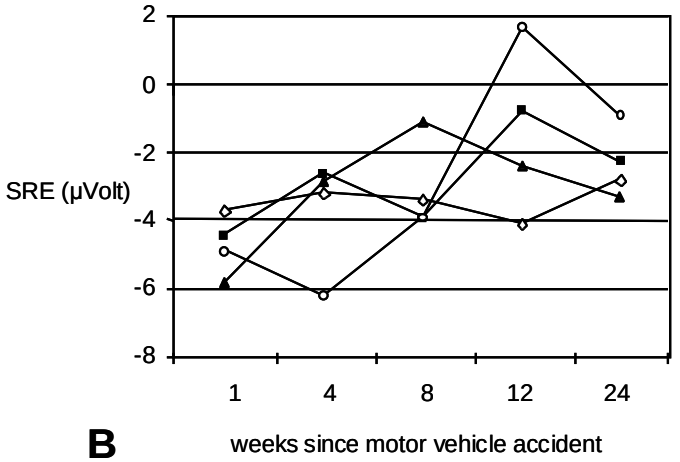


Figure 3: Course in muscle reactivity of A: active and B: passive side during 24 weeks follow-up after an MVA. The sample is divided in four categories of disability (G = recovered 0-4, n = mild 5-14, 5 = moderate 15-24, O = severe and complete 25-50) according to the NDI score at 24 weeks follow-up.

THE COURSE OF ISOMETRIC MUSCLE ACTIVITY DURING 24 WEEKS FOLLOWING A MVA

The course of isometric muscle activity of the four NDI subgroups is illustrated in Figure 4a and 4b. The results of an ANOVA (Table 2) revealed a statistical significant difference in level of isometric muscle activity of the active side between the four subgroups of subjects. Pairwise comparisons (adjusted for multiple comparison) showed statistically significant differences between the subgroups recovered and moderate disability, between the subgroups recovered and severe/complete disability, and between the subgroups mild and severe/complete disability.

As expected, the level of isometric muscle activity of the passive side showed very similar results, as the exercise is performed in a symmetrical posture. There were statistical significant differences between the four subgroups of subjects (Table 2), with both subgroups recovered and mild differing from subgroups moderate and severe/complete.

For both sides, active and passive, the results of an ANOVA revealed no statistical significant changes in isometric muscle activity over time. The level of isometric muscle activity appeared to be inversely related to the extent of pain disability. In a sense, subjects suffering from the *highest pain disability* level revealed the *greatest reduction* in recruitment of upper trapezius muscles during an isometric exercise.

THE COURSE OF DYNAMIC MUSCLE ACTIVITY DURING 24 WEEKS FOLLOWING A MVA

The course of dynamic muscle activity of the four NDI subgroups is illustrated in Figure 5a and 5b. The results of an ANOVA (Table 2) revealed a statistical significant difference in level of dynamic muscle activity of the active side between the four NDI subgroups. Pairwise comparison (adjusted for multiple comparison) showed statistically significant differences between the subgroups recovered and moderate disability and between the subgroups recovered and severe/complete disability. The level of dynamic muscle activity of the passive side showed similar differences between the four subgroups, with the subgroup recovered differing statistically significant from the subgroups moderate disability and severe/complete disability.

For both sides, active and passive, the results of an ANOVA revealed no statistical significant changes of dynamic muscle activity over time. Once again, subjects suffering from the highest pain disability level showed the lowest level of dynamic muscle activity.

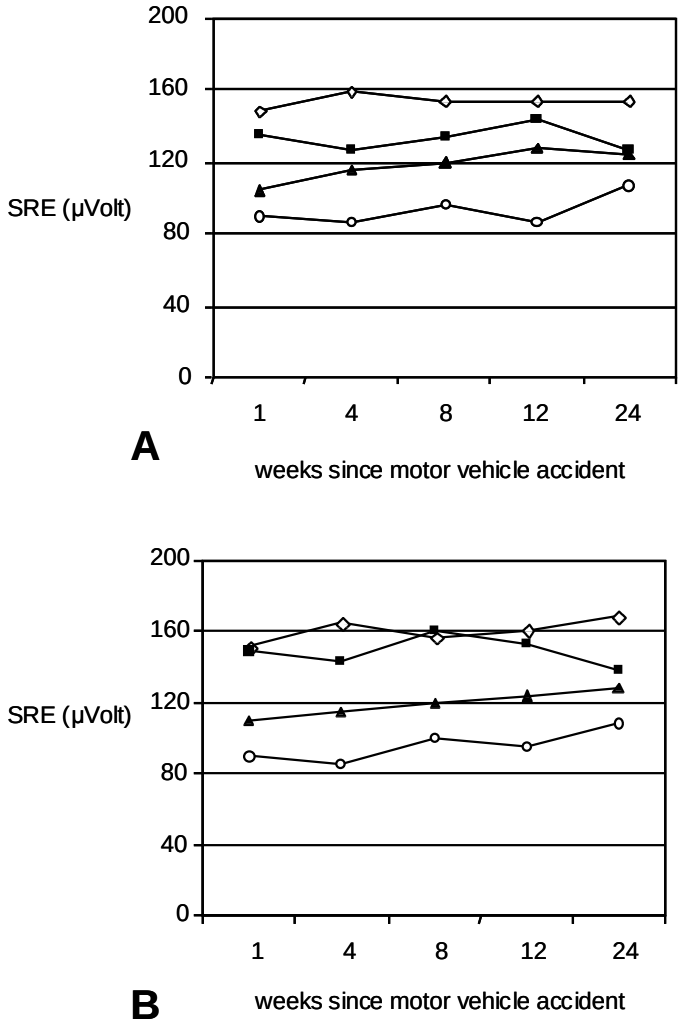


Figure 4: Course in isometric muscle activity of A: active and B: passive side during 24 weeks follow-up after an MVA. The sample is divided in four categories of disability (G = recovered 0-4, n = mild 5-14, 5 = moderate 15-24, O = severe and complete 25-50) according to the NDI score at 24 weeks follow up.

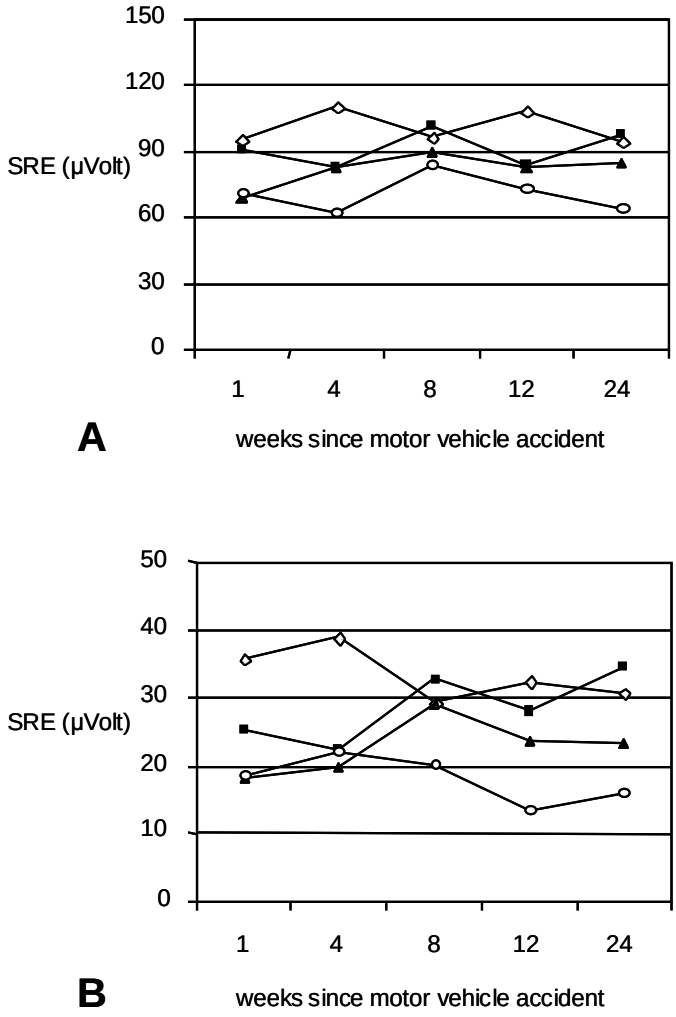


Figure 5: Course in dynamic muscle activity of A: active and B: passive side during 24 weeks follow-up after an MVA. The sample is divided in four categories of disability (G= recovered 0-4, n = mild 5-14, 5 = moderate 15-24, O = severe and complete 25-50) according to the NDI score at 24 weeks follow up.

Table 2: ANOVA (and Bonferroni corrected pairwise comparisons) for the main effects of 'NDI category' and 'change over time' on the level of the pre- and postexercise muscle activity, the muscle reactivity and the isometric and dynamic muscle activity.

condition	side	ANOVA	F	df	Sig.
Pre-exercise	active	corrected model	0.91	7	0.913
		change in level	0.16	4	0.957
		Level	0.67	3	0.574
	passive	corrected model	0.60	7	0.754
		change in level	0.48	4	0.747
		Level	0.74	3	0.527
Post exercise	active	corrected model	0.79	7	0.593
		change in level	0.31	4	0.872
		Level	1.42	3	0.237
	passive	corrected model	0.65	7	0.711
		change in level	0.41	4	0.803
		Level	0.98	3	0.401
Muscle Reactivity	active	corrected model	0.36	7	0.923
		change in level	0.43	4	0.789
		Level	0.27	3	0.847
	passive	corrected model	0.45	7	0.868
		change in level	0.67	4	0.611
		Level	0.15	3	0.932
Isometric muscle activity	active	corrected model	5.99	7	0.000
		change in level	0.41	4	0.803
		Level	13.53	3	0.000
		recovered - moderate			0.000
		recovered - severe/complete			0.000
		mild - severe/complete			0.004
	passive	corrected model	6.42	7	0.000
		change in level	0.49	4	0.744
		Level	14.52	3	0.000

- Table 2 continued -

condition	side	ANOVA	F	df	Sig.
		recovered - moderate			0.000
		recovered - severe/complete			0.000
		mild - moderate			0.049
		mild - severe/complete			0.000
Dynamic muscle activity	active	corrected model	2.53	7	0.015
		change in level	0.36	4	0.843
		Level	5.44	3	0.001
		recovered - moderate			0.029
		recovered - severe/complete			0.003
	passive	corrected model	2.97	7	0.005
		change in level	.08	4	0.988
		Level	6.81	3	0.000
		recovered - moderate			0.006
		recovered - severe/complete			0.001

F = F-statistic; df = degrees of freedom; Sig = significance level

DISCUSSION

The results of this study demonstrate that in the acute phase of WAD (grade 1 and 2), the painful traumatic injury does not initiate hyperreactivity in the upper trapezius muscles, nor is there any tendency to develop hyperreactivity during the transition from acute to chronic neck pain disability 6 months after the initial MVA. Moreover, throughout this period there appears to be *decreased* rather than increased muscle activation in the upper trapezius muscles, with an inverse relationship between the level of muscle activation during physical exercise and the extent of disability 6 months post-accident. These results suggest that the anticipated 'vicious circle' of pain and increased muscle activity reinforcing one another, does not appear during 6 months follow-up of patients with acute WAD. Furthermore, throughout the period of transition from acute to chronic neck pain disability it is the dynamic and isometric muscle activation level, rather than the

muscle reactivity, that differentiates between subjects who suffer from more serious neck pain disability and subjects who have recovered following an acute WAD.

The pain adaptation model[15,26] provides an explanation for the reduced muscle activation levels observed during isometric and dynamic exercise. This neurophysiological model suggests that nociceptive interneurons at the segmental level affect agonist and antagonist muscles in a reciprocal way. As a consequence, musculoskeletal injuries will result in a *decrease* of muscle activity of painful muscles acting as agonists or of muscles located in the surrounding of pain source(s). Concomitantly, avoidance of painful movements will result in a recoordination of muscles in the neck shoulder region, instigating an increase of activity of muscles acting as antagonists or synergists.

Results of several studies support this functional reorganization of muscle activities during experimental induced muscle pain.[4,9,16] Experimental muscle pain induced in healthy subjects by intra-muscular injection of hypertonic saline into the trapezius muscle resulted in a change in motor control during low load simulated repetitive occupational work task. The injected muscles showed a decrease in muscle activity. At the same time EMG activity of the infraspinatus muscle tended to increase and the EMG activity of the deltoid muscles decreased.[16]

There are also studies demonstrating a supraspinal modulation of the effect of pain on muscle activity by means of pain-related fear (fear of pain/physical activity/(re)injury).[6,17,32] In this regard it is worth mentioning three studies that demonstrated flexibility of the shoulder muscles during performance of a specific work task by means of a reduction of trapezius muscle activity while maintaining static posture.[12,21,22] The results of two of these studies showed a voluntary reduction of 47% - 68% of upper trapezius muscle activity if it was visually fed back to the subject while maintaining the arm in a fixed position of 90^0 glenohumeral abduction.[21,22] A significant portion of the load was transferred to the rhomboid muscles and the transverse part of the trapezius muscle.[22]

In a previous study, hyperreactivity was found in a group of chronic WAD patients.[18,19] The differences with the present study are intriguing, since there was no significant time effect up to 6 months after the accident. It is unlikely that there is a confounding factor in the calculated index of muscle reactivity, as there were no differences in pre and post exercise muscle activity levels between the four subgroups, and no changes of these parameters over time. The most likely explanation is that the differences in results are caused by a difference in selection of patients. In the previous study, the patients were recruited from a waiting list for

treatment at a pain rehabilitation program. They show considerable higher neck disability scores than the disabled subjects at 6 months follow up in the current study (i.e., NDI =26, sd 8.5 versus 19.0, sd 8.1 respectively). Moreover, the patients from the pain rehabilitation waiting list had a much longer duration of pain disability (mean 22, range 10 -53 months). This suggests that there are two different kinds of alterations in motor control in a musculoskeletal pain condition, each with a different interaction between pain and muscle activation. First, a response related to the injury, aiming at minimizing the use of painful muscles; secondly, the development of elevated muscle reactivity as a psychophysiological response to the presence of long exposure to chronic pain.

A question that remains is how a change in muscle behavior of the upper trapezius muscles observed in the acute phase of WAD contributes to the development of chronic pain disability. In the pain-spasm-pain model, the muscle hyperactivity is hypothesized to be a maintaining factor in the process of 'chronification.' However, the results of our study that indicate a decrease of muscle activity level, are more in favor of the pain adaptation model. As such, the muscle behavior is considered to be a normal protective adaptation, without a causal link to chronic pain. As such, the question concerning the relation to chronicity remains unanswered.

In conclusion, in acute WAD, changes in muscle activity of painful muscles may result from segmental and supraspinal inhibitatory effects. The methods of our study did not include EMG assessment of other neck and shoulder muscles, but according to the theories described,[5,16,17,31] the subjects with acute pain in the neck in the current study may also have developed a new synergy aiming at minimizing the use of the painful trapezius muscles during the physical exercise. Future studies should focus on changes in motor control of multiple muscles in the shoulder complex. Furthermore, studies should evaluate the relationship of pain, pain-related fear, and muscle activity of the upper trapezius muscles.

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CHAPTER 5

THE EFFECT OF FEAR OF MOVEMENT ON MUSCLE ACTIVATION IN POSTTRAUMATIC NECK PAIN DISABILITY.

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Submitted

ABSTRACT

Studies using surface electromyography (sEMG) have demonstrated a decrease in activation of the upper trapezius muscles in patients with posttraumatic neck pain disability. The neurophysiologically-oriented 'pain adaptation' model explains this inhibition as a useful adaptation to prevent further pain and injury. The cognitive-behavioral oriented 'fear avoidance' model suggests that fear of movement, in addition to the inhibitory effects of pain modulates the muscle activation level.

Ninety-two people with an acute traumatic neck injury after a motor vehicle accident (MVA) were followed for 24 weeks. Visual Analogue Scale ratings of pain intensity (painVAS), response on the Tampa Scale of Kinesiophobia (TSK) – fear of movement, and sEMG of the upper trapezius muscles during a submaximal isometric physical task were obtained at 1, 4, 8, 12, and 24 weeks following the MVA. We analyzed the extent to which pain and fear of movement influenced the activation patterns of the upper trapezius muscle in patients with posttraumatic neck pain disability. In addition, we analyzed the time related changes of this relationship during follow-up.

Multilevel analysis revealed that an increased level of both fear of movement (t-value = -2.26, p = 0.025) and pain intensity (t-value = -3.03, p = 0.003) were *independently* associated with a decreased level of muscle activation. Moreover, the results suggest that the association between the effect of fear of movement and lower muscle activity level is stronger in patients reporting high pain intensity (t-value = 2.18, p = 0.031). The contribution of pain intensity to the muscle activation level appeared to decrease over time post-trauma (t-value = 2.65, p = 0.009).

The results support both the 'pain adaptation' and the 'fear avoidance' models. It is likely that the decrease in muscle activation level is aimed at ‘avoiding’ the use of painful muscles.

INTRODUCTION

As proposed by the Quebec Task Force in 1995, a whiplash associated disorder (WAD) is defined by '....an acceleration deceleration mechanism of energy transfer to the neck which may lead to a variety of clinical manifestations...' .[22] According to the presenting signs and symptoms, the severity of the injury can be classified into one of four grades, with higher grades indicating more severe injury (i.e., grade 4 includes cervical fractures and dislocations). The characteristic feature of WAD grade 2 is the presence of 'neck pain and musculoskeletal signs.' These musculoskeletal signs are manifested in a *limited range of motion* putatively due to *muscle spasm*[22]. Muscle spasm is defined by 'an involuntary increased muscle activity that is not under voluntary control and not dependent upon posture'.[21] When observed, increase in muscle activity is postulated to be secondary to soft tissue injury.

Several studies report limitations in range of motion in neck trauma patients meeting criteria for WAD grade II and thereby provide some support for the WAD classification system.[6,12,13,19] However, there are no studies that directly confirm the presence of increased muscle activity in these patients. Moreover, recent prospective cohort study demonstrated that subjects with acute post traumatic neck pain, assessed by surface electromyography, actually show a *decrease* rather than an increase in muscle activity of the upper trapezius muscles.[18] These findings conflict with the Quebec Task Force definition of WAD grade 2.

Two models, the neurophysiological 'pain adaptation' model and the cognitive behavioural 'fear avoidance' model can explain the decreased muscle activity found in the Nederhand et al. (in press) study.[18] The pain adaptation model assumes that nociceptive interneurons induce reciprocal inhibition at segmental level.[15,23] Therefore, it would be expected that musculoskeletal injuries would result in a *decrease* in activity of agonist muscles causing a painful movement. In addition, a simultaneous increase of activity of antagonist muscles would further prevent this painful movement. These characteristic changes in muscle activity can be explained as a useful adaptation because they prevent further pain and injury. Results of several studies support this expected recoordination of muscles during experimentally-induced muscle pain.[1,8,16]

Alternatively, the cognitive-behavioral perspective, as described by the fear-avoidance model,[27] can explain the decrease in muscle activity.. This model introduces the supraspinal (i.e., cognitive) influence of pain-related fear on behavioral performance, resulting in avoidance of physical activity to prevent

anticipated exacerbations of pain and any further injury. In the long term, avoidance of movement can produce maladaptive changes in the musculoskeletal system such as physical deconditioning and impairments in muscle coordination.[32, 28] Crombez et al[3,5] confirmed the importance of pain-related fear by demonstrating a significant association between performance level and pain-related fear in a group of chronic low back pain patients. There was, however, no relationship between performance level and pain intensity.

A limitation of these studies is that either the mechanism has been investigated in acute experimentally induced pain or in crosssectional studies. As such, these studies do not address the initiation of these pain mechanisms in the acute clinical setting, and especially the time-related changes during the transition into chronic pain disability.

Our primary goal in the present study was to analyze the extent to which pain and pain-related fear determine characteristic muscle activation patterns during a submaximal isometric physical task. An additional goal was to examine the influence of the time post trauma on these relations.

METHODS

SUBJECTS

The sample of subjects included in this study has already been described in detail elsewhere.[18] Briefly, the sample consists of 92 patients admitted to the emergency room of a general hospital after an MVA, between July 1999 and December 2001. Subjects were considered eligible if they were between 18-70 years old and reported of pain in neck or head region that started within 48 hours of the accident. Furthermore, some form of acceleration or deceleration of the motor vehicle, caused by colliding with another vehicle or a stationary object (e.g. a wall, traffic light) was identified. Subjects with signs of a concussion, retrograde or post-traumatic amnesia, serious injuries such as fractures, traumatic internal organic pathology, or any neurologic signs were excluded. Thus, the subjects included met the Quebec criteria for WAD grade 1 or 2. Finally, all subjects included had to be able to speak and read the Dutch language.

STUDY DESIGN

All subjects were assessed five times with baseline assessment performed within 1 week, and follow-up assessments 4, 8, 12, and 24 weeks subsequent to the MVA (Figure 1). Functional status, was assessed by the Neck Disability Index (NDI) a 10-item self-reporting instrument for the assessment of physical disability of

subjects with neck pain, particularly from whiplash-type injuries.[26] Vernon et al[26] interpreted scores from 0-4 as no disability, scores from 5-50 represent increasing levels of disability. In accordance with this cut-off point, at 24 weeks follow-up, subjects were dichotomized into two groups, recovered (NDI<5) and disabled (NDI≥5).

Smooth Rectified surface Electromyography (SRE) recordings of the upper trapezius were obtained in order to assess the muscle activity of the upper trapezius muscles in the acute phase and during each of the four follow-up periods. Prior to each EMG assessments the subjects rated their level of pain intensity on a VAS and completed the TSK.

Before the study began, approval of the medical ethical committee was attained and all the participants were asked to complete an informed consent form.

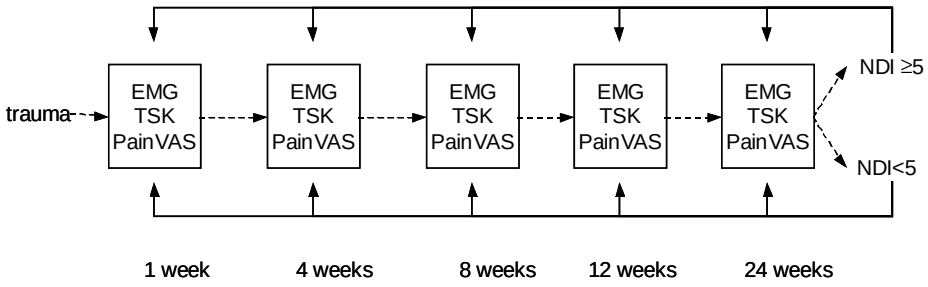

Figure 1: Study design: Assessments of Electromyography (EMG), fear of movement (TSK) and Visual Analogue Scale rating of pain intensity (PainVAS) in chronic disabled (NDI ³ 5) and recovered (NDI < 5) subjects.
(MVA=Motor Vehicle Accident; TSK= Tampa Scale of Kinesiophobia; NDI= Neck Disability Index)

EMG RECORDINGS AND ANALYSES

Experimental protocol

Surface Electromyography (sEMG) of the upper trapezius muscle was recorded bipolarly and amplified using a differential amplifier and band pass filtered (3 Hz-10.000 Hz) to remove movement artifacts and prevent aliasing. The raw EMG was processed to an SRE applying a double-sided rectifier and stored digitally (12 bits, 1024Hz). After the skin was shaved and abraded with sandpaper it was cleansed

with 70 % alcohol. Subjects were seated in an upright position to permit palpation of the anatomical landmarks (C7, Acromion).

In order to insure proper sensor placement procedures, we followed the recommendations of the European Community concerted action SENIAM (Surface EMG for Non-invasive Assessment of Muscles) project.[9,10]. The electrodes (pre-gelled Ag/AgCl; type Meditrace, manufactured by Graphic Control Corporation, Buffalo, NY, USA) were placed 2 cm laterally to the midpoint of the leadline between the acromion and the easily palpable spinous process of vertebra C7. The electrodes were positioned parallel to the leadline with a center-to-center inter-electrode distance of 20 mm. The reference electrode was placed over the processus spinosus of C7. The electrodes as well as the cables were fixed to the skin with tape and connected to a portable data acquisition unit.

EMG analysis

Subjects maintained a sitting position in a desk-chair with their backs supported and their hips and knees in 90^{O} flexion. Arms were held straight and horizontal in 90° abduction in the frontal plane of the body, with the hands relaxed and the palms pointing downwards. Four epochs of upper trapezius SRE were obtained. Each epoch lasts 15 seconds, separated by a period of one minute of rest between the consecutive epochs. The mean SRE was calculated for the middle 10 seconds of each recorded epoch. The isometric muscle activity (IMA) is computed as the mean muscle activity of the dominant arm during the performance of the physical task.

PAIN INTENSITY

Visual Analogue Scale rating of pain intensity (painVAS), was performed using two vertical marks placed 100 mm apart marked left with the words “No pain” and right with “Worst pain ever experienced”. Subjects were asked to rate the averaged pain intensity they experienced during the preceding week.

FEAR OF MOVEMENT/ (RE) INJURY

A Dutch version of the Tampa Scale for Kinesiophobia (TSK-DV) is a 17-item questionnaire that is designed to assess fear of (re) injury due to movement. Each item is scored on a 4-point Likert scale ranging from' Strongly agree' to 'Strongly disagree.' Sum scores range from 17 to 68 with higher score indicating greater fear of movement/(re)injury. Normative values obtained from a sample of 319 Dutch

and Flemish patients with chronic musculoskeletal pain (chronic low back pain and fibromyalgia) showed a median score of 39 and a inter quartile distance of 33-45.[7]

STATISTICAL ANALYSES

Multilevel analysis (MLA) was used to analyze the longitudinal data set. Compared to a repeated measures analysis, MLA has the advantage that it can deal with occasional missing data and censored data. Furthermore, it has the advantage that continuous variables do not have to be transformed into ordinal 'dummy' variables, thereby loosing information.

Since the TSK-DV can only be used in patients suffering from pain, follow-up data was only available from people until their complete recovery. This indicates that the missing data during follow up were not random, but is attributable to recovery.

Determinants included in the model were painVAS, TSK, Body Mass Index (BMI), time and the interaction terms painVAS X TSK, painVAS X time, and TSK X time. The variable 'BMI' was included in this analysis to control for differences in the thickness of subcutaneous fat layer between patients. The hypothesis that pain can influence the muscle activity directly, but also modifies the effect of fear of movement on muscle activity was assessed by examining interaction between painVAS and TSK. The variable 'time' and the interaction between time and both painVAS and fear of movement, controls for possible changes in the relationship between these factors and the level of muscle activation during the follow-up period.

There was a skew in the distribution of the data presented by 10-90 percentiles. P values < 0.05 was considered statistically significant. Data were analyzed using S-Plus 2000 for windows.

Table 1: Demographic Variables of a group of recovered (Neck Disability Index < 5) and chronic disabled (Neck Disability Index ^{3} 5) subjects.

	NDI < 5	NDI ≥ 5
Number of subjects	43	49
Mean age (sd), yr	37.4 (11.6)	31.3 (11.3)
male:female	18:25	11:38
Body Mass Index, (sd), kg^2/cm	24.2 (3.7)	25.7 (5.1)

RESULTS

Of the 92 patients included in the sample, 43 recovered and 49 still suffered from disabling pain 24 weeks after their MVA (Table 1). Baseline and follow-up assessments of both groups are shown in Table 2. The disabled group showed a low baseline level of EMG activity (mean = 106.9 μVolt, sd = 50.5) that is rather constant during follow-up. The baseline pain intensity of this group (mean VAS = 53.7 mm, sd = 21.4) can be classified as almost severe,[2] and declines gradually to a more moderate level by 24 weeks (mean pain VAS = 38.8, sd = 21.0). The level of baseline TSK (median 39.0, 10-90th percentile 30.8-47.0) corresponds to the median level of a normative group of a sample of 319 Dutch and Flemish patients with chronic musculoskeletal pain.[7] During follow-up the TSK level remained relatively constant.

Table 2: Course of Isometric Muscle Activity (IMA), pain intensity (Pain VAS) and fear of movement (TSK) in a group of recovered (Neck Disability Index < 5) and chronic disabled (Neck Disability Index ³ 5) subjects, during 24 weeks follow-up after a MVA.[1]

	Recovered at follow-up				disabled at follow-up			
wks	N*	IMA mean(μVolt) (SD)	Pain VAS mean(mm) (SD)	TSK median (10 - 90 % tile)	N**	IMA mean(μVolt) (SD)	Pain VAS mean(mm) (SD)	TSK** median (10 - 90 % tile)
1	38	147.7 (77.8)	30.6 (20.3)	34.5 (25.9-45.1)	43	106.9 (50.5)	53.7 (21.4)	39.0 (30.8 - 47.0)
4	22	152.0 (91.3)	24.8 (19.2)	34.5 (25.3-47.4)	43	115.8 (55.2)	52.1 (18.6)	41.0 (31.6 - 49.0)
8	8	180.0 (67.0)	23.9 (16.8)	30.0 (25.0-50.0)	47	120.5 (70.5)	47.6 (17.9)	38.0 (29.4 - 48.6)
12	10	180.6 (76.5)	21.4 (11.8)	30.0 (21.5-42.7)	45	123.1 (64.5)	45.0 (20.3)	38.0 (27.5 - 49.0)
24	0	Recovered	recovered	recovered	47	121.7 (65.8)	38.8 (21.0)	37.5 (28.0 - 48.2)

N= number of subjects IMA= Isometric Muscle Activity, painVAS= Visual Analogue Scale rating pain severity, TSK= Tampa Scale for Kinesiophobia

[1]The data in the recovered group are from censored cases, because the TSK can only be scored until patients have fully recovered

*Five of the 43 subjects were already recovered at first assessment and could not score the TSK. ** Incomplete data set because of occasional missing data.

Of the 43 recovered subjects, five had recovered prior to the first assessment. The level of EMG activity of the recovered group (mean = 147.7 μVolt, sd = 77.8) gradually increases during follow-up. By the 24 weeks follow up their EMG values were comparable to that in healthy subjects (unpublished data). The initial pain intensity of this group is moderate (mean pain VAS = 30.6 mm, sd = 20.3), and

gradually declines during follow-up.[2] The level of baseline TSK is low (median 34.5, 10-90th percentile 25.9-45.1) and corresponds to the 3rd decile of the normative group.[7] During follow-up the TSK further decreases to a median level of 30.0 (10-90th percentile 21.5-42.7). It appears then that during the follow-up period of 24 weeks the level of muscle activity in the disabled group is systematically lower than in the recovered group, whereas the levels of pain intensity and TSK are higher for the disabled group.

Multilevel analysis including main effects and interaction terms resulted in explained variance of approximately 54% for the model (Table 3). An increased level of both fear of movement (t-value = -2.26, p = 0.025) and pain intensity (t-value = -3.03, p = 0.003) were independently associated with a decreased level of muscle activation (Figures 2 and 3). In addition, the effect of fear of movement on muscle activity is modified by pain intensity, in a sense that the association between the effect of fear of movement and lower muscle activity level is stronger in patients reporting a high pain intensity (Figure 4) (t-value = 2.18, p = 0.031). Furthermore, the effect of a decrease in muscle activity level caused by an increased painVAS level diminishes as time post-trauma proceeds (t-value = 2.65, p = 0.009).

Table 3: Multilevel analysis of isometric muscle activity as dependent variable. The determinants included in the model were painVAS, TSK, Body Mass Index (BMI), time and the interaction terms painVAS X TSK, painVAS X time, and TSK X time

	beta	SE	t-value	p-value
BMI	-7.09	1.27	-5.58	<0.0001
Time	-1.48	0.90	-1.65	0.102
TSK	-1.45	0.64	-2.26	0.025
PainVAS	-1.35	0.45	-3.03	0.003
Time X TSK	0.015	0.03	0.61	0.545
Time X painVAS	0.024	0.01	2.65	0.009
TSK X painVAS	0.024	0.01	2.18	0.031

BMI= Body Mass Index; time= time post trauma; painVAS= Visual Analogue Scale rating pain intensity; TSK= Tampa Scale of Kinesiophobia rating fear of movement; beta= beta coefficients of the determinants, SE= standard error of the beta coefficient.

As expected, a large part of the variance was explained by the BMI (t-value = -5.58, p < 0.0001), because in subjects with higher percentage of fat the EMG

signal will be decreased. The importance of this variable in using sEMG is illustrated in Figures 2 to 4. The plotted models show 9 EMG values below 50 micoVolt. These values come from the two patients with the two most extreme BMI values of 40.0 and 43.3 respectively. The fact that these subjects have non-realistic predicted EMG values, indicate a non-linear relationship between BMI and EMG level.

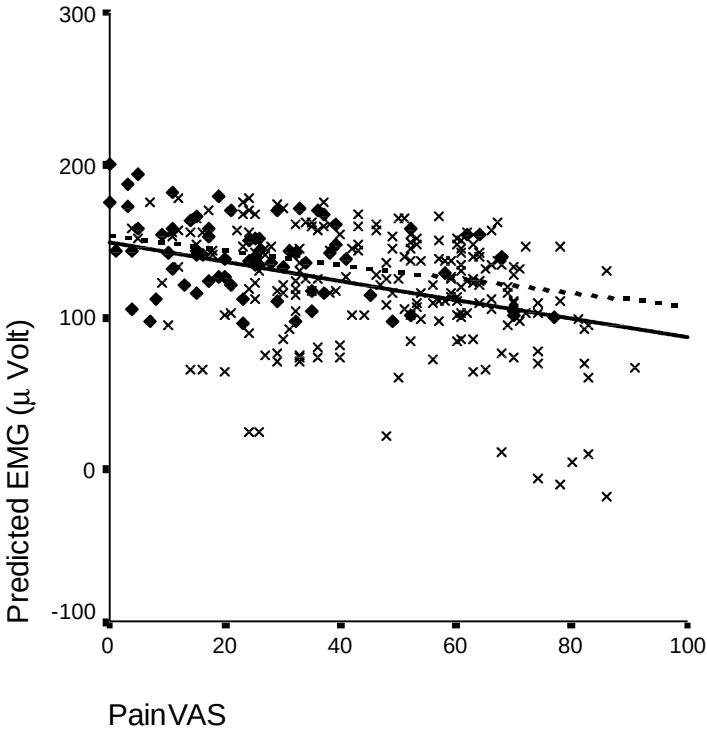

Figure 2: The level of EMG during a submaximal isometric exercise, predicted by the independent effect of pain intensity (painVAS), for both the disabled group (´, straight line) and recovered group (¨, dotted line).

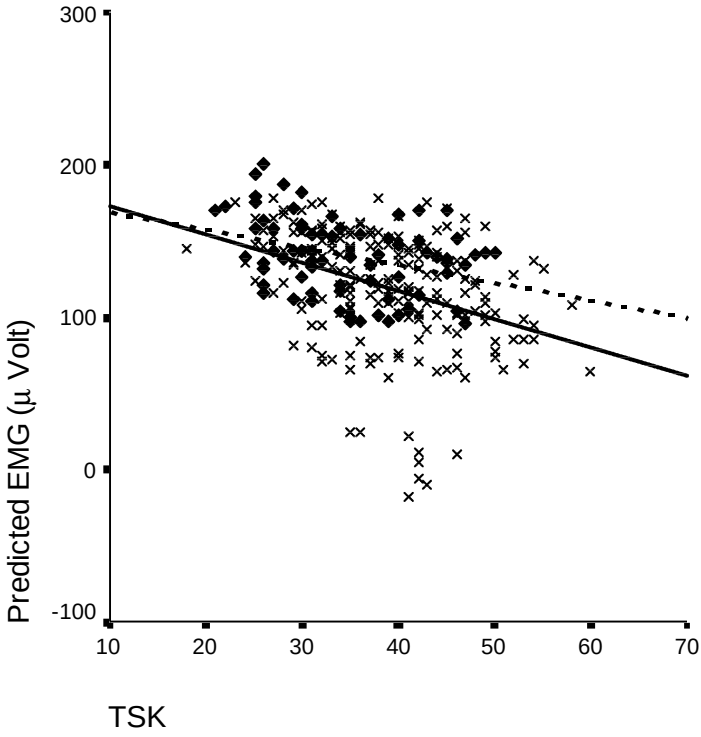

Figure 3: The level of EMG during a submaximal isometric exercise, predicted by the independent effect of fear of movement (TSK) for both the disabled group (´, straight line) and recovered group (¨, dotted line)
(TSK; Tampa Scale of Kinesiophobia)

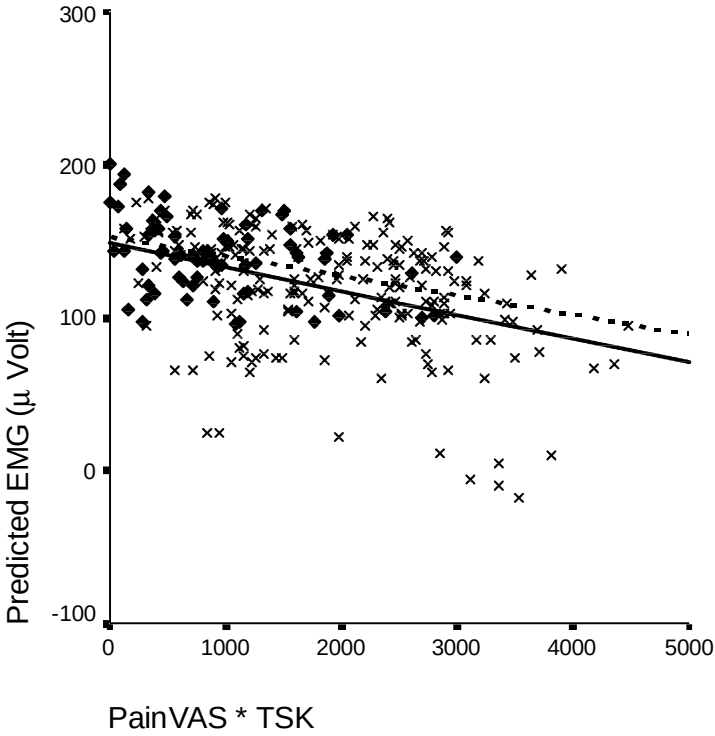

Figure 4 The level of EMG during a submaximal isometric exercise, predicted by the interaction between pain intensity and fear of movement (painVAS X TSK), for both the disabled group (´, straight line) and recovered group (¨, dotted line) (TSK; Tampa Scale of Kinesiophobia)

DISCUSSION

The goal of this study was to evaluate the role of pain and fear of movement in the muscle activation pattern of the upper trapezius muscles in post-traumatic neck pain patients. The results show that in addition to the inhibitory effects of pain, fear of movement is *independently* associated with the level of muscle activation. This means that, in patients with high pain intensity *or* fear of movement, the level of muscle activity used during the task has diminished. The results also indicate

that the association between the effect of fear of movement and lower muscle activity level is stronger in patients reporting a high pain intensity. These results are in agreement with both the pain adaptation model[15,23] and the fear avoidance model.[27]. Thus, it is likely that the decrease in muscle activation level is aimed at 'avoiding' the use of painful muscles to prevent amplification of pain and further injury.

Crombez et al[3,5] found similar results regarding the influence of pain and pain-related fear on physical performance. In these studies, pain-related fear, more than pain was associated with poor performance on a knee-extension flexion task[3], a trunk extension-flexion task, and a weight lifting task.[5] During these tests, subjects were requested to flex and extend as quickly and forcefully as possible. The results demonstrated that the most consistent predictor of the peak torque of this test was pain-related fear, and not pain.

Our study differs from that of Crombez in several ways. Using a submaximal well defined task[17] will diminish interindividual differences related to anatomical variability, and motivational aspects related to the readiness to perform maximally. Therefore, this method probably is more consistent in assessing the consequences of pain related fear on muscle activation.

Another difference is that our results demonstrate the independent contribution of pain and fear of movement on muscle activity. In contrast, Crombez et al.[5] demonstrated that pain-related fear predicted the physical performance better than pain. It is likely that this is caused by differences in subject selection. The current study included acute pain patients. Assuming that in these patients the healing of the soft tissue injury takes 6-8 weeks, during this period nociceptive stimuli can have a direct effect on muscle activity. The pain adaptation model[15,23] provides an explanation for this mechanism. However, after the healing phase of the soft tissue injury, the injury related nociception is supposed to dissipate. The results of the present study provide some evidence to support this process by the fact that the influence of pain on muscle activity diminished during follow-up.

There are several remaining questions concerning the role of decreased muscle activity in the development of chronic pain. In the pain adaptation model the inhibition of muscle activation is considered an adaptive reaction in order to avoid painful movement and (re)injury. As such, the decrease of upper trapezius muscle activity, is merely a normal protective adaptation in response to pain. Because the model does not involve a kind of a vicious circle, the decrease in muscle activation in the development of chronicity remains unclear. From the

perspective of the fear avoidance model, a persistent reduction in daily physical activity may result in a worsened physical condition in the long term, thereby contributing to physical disability.[28,31] However, research is needed to explain how the observed decrease in muscle activity in the current study can result in 'deconditioning'effects and disability. Moreover, demonstration of the generalization of these relations outside the laboratory context is needed to clarify how changes in muscle activation pattern co-vary with changes in pain and disability. Future studies may address this by using ambulatory EMG equipment and following subjects in natural environments.[11]

It is intriguing to observe that the baseline values of TSK and pain intensity of the recovered group are low compared to that of the disabled group. This suggests that other antecedent variables may be of importance to explain the different reactions to accidents. According to the pain adaptation model, this may be the extent of injury, resulting in a different level of nociception.[15,23] Alternatively, according to the fear avoidance model, this difference could be explained by differences in 'pain catastrophizing' --- an exaggerated negative orientation toward noxious stimuli --, and is considered to be a precursor to fear of movement.[27,4]

There are some limitations in this present study that need to be acknowledged. First, the results show only a moderate contribution of pain and fear of movement to the level of muscle activation. This could be explained by the fact that the pain and the TSK scores do not directly apply to the actual physical task used in this study. Future studies should therefore focus on the effects of fear in relation to that particular movement on muscle activation level.[25] A second limitation is that we only have follow-up data of patients until they were recovered, because patients who have fully recovered cannot score the TSK. Especially the group that recovers is expected to show dynamic changes in determinants and outcome, providing an opportunity to demonstrate more clear relations between these variables. A modified version of the TSK that is applicable by persons without musculoskeletal pain, recently became available, unfortunately after the initiation of the study.[20]

The clinical implication of this study is that the Quebec Task Force injury severity classification system[22] needs to be adjusted since the results of this study suggest that WAD grade II is not characterized by muscle spasm but rather by muscle *inhibition*. Moreover, because fear of movement independently explained part of the muscle inhibition, in addition to pain, it is questionable whether this musculoskeletal signs should be considered an aspect of injury severity. It is more

likely that it represents a behavior that affects the coordination of muscles in order to avoid painful movements and in an effort to prevent further injury. Psychosocial factors, in particular fear avoidance beliefs, appear to be more important than biomedical one for the development of chronic pain.[20,28]. In the current study, the differences in baseline fear of movement and muscle activity between recovered and disabled patients support these findings. Such data suggest that refinement of the WAD classification, including assessments of fear of movement and related physical measures, may be more useful for the prediction of long-term outcome and consequently for prevention of chronic disability. Prevention of disability might be fostered if psychosocial factors are routinely targeted for treatment in the earliest stages of pain.[24,29,30]

In conclusion, this study indicates that pain-related fear affects physical performance by altering the motor control during the performance of a physical activity that is perceived as a potential threat of physical integrity. Further investigation is required to determine whether fear of movement and the consequent changes in motor control in WAD are predictive for long-term disability.

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CHAPTER 6

PREDICTIVE VALUE OF FEAR AVOIDANCE IN DEVELOPING CHRONIC NECK PAIN DISABILITY - CONSEQUENCES FOR CLINICAL DECISION MAKING

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Submitted

ABSTRACT

Recent conceptions of spinal pain suggest that fear-avoidance beliefs play an important role in the transition of acute to chronic pain. In addition, fear-avoidance beliefs may seriously interfere with intervention programs that focus on promoting physical activity.

Ninety-two people with an acute traumatic neck injury after a motor vehicle accident (MVA) were assessed at baseline, 1-week post trauma, and were followed for 24 weeks. Responses on a Visual Analogue Scale rating pain intensity (painVAS), Tampa Scale of Kinesiophobia (TSK) – fear of movement, Pain Cognition List - subscale catastrophizing (PCL-catas), and Neck Pain Disability Index (NDI) were assessed. In addition, surface electromyography (sEMG) of the upper trapezius muscles during a submaximal isometric physical task was examined. We investigated whether these variables can predict chronic neck pain disability 24 weeks following an MVA. Receiver Operating Characteristic (ROC) curves were used to calculate the overall accuracy of each test variable separately and in combination with a second variable. Using pre-specified cut-off points, we determined the predictive value of these (combinations of) tests.

When tested separately, the baseline NDI demonstrate the highest overall accuracy (Area under the curve: 0.872, 95% CI: 0.794-0.950). Conditional on the results of the NDI in a first test, the TSK demonstrate the highest overall accuracy (Area under the curve: 0.770, 95% CI 0.634-0.907). Using these two tests, it appeared to be possible to predict chronic disability with a probability of 54.3% (95% CI: 35.2-72.3%) after entering the NDI (cut-off 15) as a first test, and with a probability of 83.3% (95% CI: 70.3-91.3%) after entering the TSK (cut-off 40) in a second test.

A simple rating of baseline neck pain disability within a week after the trauma, separately, or in combination with a second test 'fear of movement' can be used to predict future outcome. In addition, by evaluating the involvement of 'fear of movement', the patients at risk for showing an 'avoidant' behavior style, can be offered an intervention that focuses on reduction of this fear.

INTRODUCTION

In Western industrialized society the development of chronic pain and related disability following a whiplash injury has become a significant public health problem. Incidence rates vary between 70 per 100,000 yearly in Quebec (Canada),[20] and 106 per 100,000 in Australia.[12] Not only does chronic neck pain affect patients' physical and psychological well being, it also puts a great burden on societal and health care costs. Indirect costs per whiplash patient in Canada are estimated to be around $2,500.[20,21] Consequently, prevention of chronic disability following a whiplash injury is an important objective. The efficacy of prevention is based on two essential factors, i.e. patients at risk should be identified correctly, and preventive actions must have proven to be effective. A thorough understanding of the mechanism by which chronic disability develops is required to accomplish this aim.

With respect to identifying patients at risk, a recent study systematically reviewed the prognostic factors in Whiplash Associated Disorder (WAD).[5] The authors concluded that the initial pain intensity and related physical manifestations (e.g., neck pain on palpation and muscle pain) are important predictors of recovery. In addition, however, sociocultural factors such as expectation of pain and type of compensation system appeared to be important predictors. These results indicate that prognosis is multifactorial, integrating physical and non-physical dimensions.

In addition to the prognostic factors identified in WAD, recent conceptions in spinal pain, suggest that psychological factors play an important role in the transition of the acute to the chronic phase.[10,24] As such, these factors may have relevance in predicting future outcome in acute post-traumatic neck pain as well. In particular, the fear-avoidance model of Vlaeyen[27] offers a framework conceptualizing the process of developing chronic musculoskeletal pain. It postulates two opposing behavioral responses, ‘confrontation’ and ‘avoidance.’ In addition, the model suggests possible pathways by which injured patients become enmeshed in a downward spiral of increasing avoidance, disability, and pain. The central element in this model is whether avoidance behavior will evolve. This is especially the case in patients who interpret pain as threatening (pain catastrophizing) and exhibit 'fear of movement' (or kinesiophobia). Several prospective studies have confirmed the importance of this model, demonstrating that catastrophizing and fear avoidance beliefs are important predictors for the development of chronic low back pain.[2,8,9,18]

In addition to its relevance in low back pain, a recent study in whiplash disorders suggested that the fear avoidance model could also be generalized to post-traumatic neck pain.[13] In this study, neck pain patients with a high degree of disability, showed avoidance of contraction of painful neck muscles during exercise. An additional analysis confirmed the assumption that the decrease in activation level was associated with fear of movement.[14]

The aim of the present study is to improve clinical decision-making in patients with acute post-traumatic neck pain and to enable an early intervention in order to prevent chronic symptoms. Therefore, we investigated whether fear avoidance variables have additional value in predicting future outcome when compared to established prognostic factors in post-traumatic neck pain. Characterization of the behavioral response to pain --confrontation or avoidance -- will provide an initial indication of the target for intervention. The question addressed in this study was to what extent the fear avoidance variables 'catastrophizing', 'fear of movement', and the associated 'avoidance muscle behavior', can predict chronic neck pain disability.

METHODS

SUBJECTS

The sample of subjects included in this study was part of an inception cohort. Their characteristics have been described in detail in a previous paper.[13] Briefly, the sample consists of patients admitted to the emergency room of a general hospital after a motor vehicle accident (MVA), between July 1999 and December 2001. Patients were considered eligible to be subjects in this study if they were between 18-70 years old and reported of pain in neck or head region that started within 48 hours of the accident. Furthermore, some form of acceleration or deceleration of the motor vehicle, caused by colliding either with another vehicle or a stationary object (e.g., a wall or traffic light) was identified. Subjects with signs of a concussion, retrograde or post-traumatic amnesia, serious injuries such as fractures, traumatic internal organic pathology, or any neurologic signs were excluded. Thus, the subjects included met the Quebec criteria for WAD grade 1 or 2. To be included, subjects had to be able to speak and read the Dutch language. Approval of the medical ethical committee was attained and all the participants were asked to complete an informed consent form before the study began.

PREDICTIVE FACTORS

Neck Disability Index

The Neck Disability Index (NDI) is a 10-item self-reporting instrument for the assessment of physical disability of subjects with neck pain, particularly from whiplash-type injuries.[25] Disability categories for the NDI are: 0-4 = no disability, 5-14 = mild, 15-24 = moderate, 25-34 = severe, above 34 = complete. A change of 5-point is required to be clinically meaningful.[21]

Pain intensity

Pain intensity was performed using a Visual Analogue Scale (consisting of two vertical marks placed 100 mm apart marked left with the words “No pain” and right with “Worst pain ever experienced”-- (painVAS) Subjects were asked to rate the averaged pain intensity they experienced during the period from onset of the pain to the EMG assessments. A pain VAS score in of 30 mm or more is considered moderate pain and VAS scores above 54 mm are considered severe pain.[4]

Fear of movement/(re) injury

A Dutch version of the Tampa Scale for Kinesiophobia (TSK) is a 17-item questionnaire that is designed to assess fear of (re) injury due to movement.[7] Each item is scored on a 4-point Likert-type scale ranging from' strongly agree' to 'strongly disagree.' Sum scores range from 17 to 68 with higher score indicating more fear of movement/(re)injury.

Catastrophizing

The Pain Cognition List (PCL-e;39) is a 77-item questionnaire designed to assess distorted pain cognitions and experienced self-control. The results are presented in: pain impact, catastrophizing, outcome-efficacy, acquiescence, and reliance on health care. For the current study only the catastrophizing subscale (PCL-catas) was used. This variable is considered an exaggerated negative orientation toward noxious stimuli and is highly correlated with depressive symptoms. The mean value for a group of 188 chronic low back pain patients is 49.1 (sd 14.3).[26] Higher scores indicate more catastrophizing thoughts.

Isometric muscle activation

The methods we used to assess surface Electromyography (sEMG) of the upper trapezius muscles has been described in detail elsewhere.[13] Briefly, the isometric muscle activity (IMA) was obtained with the subjects maintaining a sitting position in a desk-chair with their backs supported and their hips and knees in 90^0 flexion. Arms were held straight and horizontal in 90° abduction in the frontal plane of the body, with the hands relaxed and the palms pointing downwards. Four epochs of upper trapezius raw EMG were obtained and processed to a Smooth Rectified EMG (SRE). Each epoch lasted 15 seconds, separated by a period of one minute of rest between the consecutive epochs. The mean SRE was calculated for the middle 10 seconds of each recorded epoch. The IMA is computed as the mean muscle activity of the dominant arm during the performance of the isometric physical task.

OUTCOME

We divided the cohort of subjects into one group who recovered from initial complaints (NDI <15) and a second consisting of subjects who developed chronic neck pain disability (NDI • 15) at 24 weeks post injury. By choosing this cut off point we tend to approach the average NDI score of a population submitted to a multidisciplinary rehabilitation program (NDI=24.4, SD 7.1, personal data). In this population the extent of disability is such severe that it is associated with a high degree of interference with daily life and a high degree of medical consumption

STATISTICAL ANALYSES

Differences in baseline characteristics between the recovered and disabled group were analyzed by Students t-test for scale measures and by chi^2 test for nominal and ordinal measures. Differences in outcome of the experimental group and the group of subjects that completed the questionnaire at 24 weeks (Figure 1) were tested with non-parametric Mann-Whitney U test. As such, we could test whether the results of the experimental group were biased due to differences in selection.

Receiver Operating Characteristic (ROC) curves were used to determine the overall accuracy of each variable separately and in combination with a second variable. We used the 90% sensitivity as the decisive criterion in determining the cut-off points for dichotomizing the sample. The actual cut-off values that were determined for the different determinants are displayed in Table 2. Sensitivity instead of specificity was used, because we did not expect serious adverse effects of treatment in patients with a false positive test result.

The variable with the most optimal predictive value was analyzed first, followed by each of the other variables, conditional on the first test. The increase in post-test probability of the second test was calculated conditional on the post-test probability of the first test. Data were analyzed using the SPSS Statistical Package for Windows 10.0 (SPSS Inc. Headquarters, Chicago, Illinois). A p-value < 0.05 was considered statistically significant.

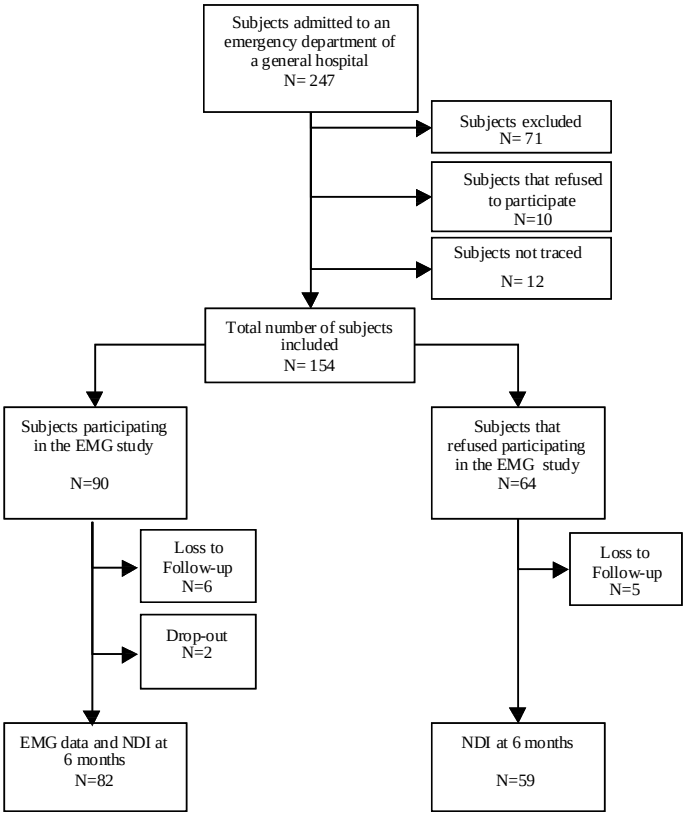

Figure 1 Patients participating in the study

RESULTS

Of the 247 admitted patients 71 failed to meet the inclusion criteria, 10 refused to participate, and 12 could not be traced because of unknown telephone number or address. Of the 154 subjects eligible for the study 90 (58%) participated in the study. The remainder 64 subjects did not participate in the study, but agreed to be contacted at follow-up for assessing the NDI. In the group of 90 subjects, 6 subjects were lost to follow up without known reason, 1 subject dropped out because of a second accident, and 1 subject was not included in the analysis because of suspected malingering (Figure 1). Thus, 91.1 % (N = 82) of the sample provided 24-week follow-up on the NDI. In the group of 64 subjects who did not participate in the study, 5 subjects were lost to follow up without known reason. At 24 weeks the NDI score of the experimental group (NDI mean = 10.2, 10-90th percentile 0-25.7) did not differ significantly from the group of subjects that only completed the NDI (mean = 8.2, 10-90th percentile = 0-24) (Mann-Whitney U test: Z = -0.953, p = 0.341). Thus, selection bias was not likely to occur.

Table 1: Baseline characteristics of post-traumatic neck pain patients.

	non-disabled (n=55)	disabled (n=27)	Difference
Age in yr mean (sd)	33.0 (11.2)	38.0 (12.7)	0.071‡
male:female	20:25	5:22	0.128#
Body Mass Index kg/m^2 mean (sd)	24.1 (4.0)	26.8 (5.7)	0.015‡
Site of collision: front/rear/side	10/37/8	3/22/2	0.399 #
estimated mean speed of collision*	50.4 (23.1)	56.2 (25.0)	0.355‡
IMA[1]	141.2 (70.3)	96 (50.1)	0.004‡
Pain VAS[2] mean (sd)	33.6 (20.5)	60.7 (19.4)	0.000‡
TSK[3] mean (10-90 percentile)	35.0 (26.0 - 45.0)	41.5 (33.8 - 50.4)	0.000†
Catas[4] mean (sd)	28.6 (22.2)	53.2 (24.4)	0.000‡
NDI[5] mean (10-90 percentile)	14.2 (4.6 - 25.4)	27.9 (15.4 - 40.0)	0.000†

Depending on the direction of travel and the site of impact of cars, the speed at the time of collision was estimated by :the addition of the two speeds in case of 'head on' collision, the difference in the speed of the two cars in case of 'rear' collision and the speed of the car colliding from the side in case of 'side' collision.

‡ = Students t-test # = Chi square test, † =Mann Whitney U test

[1]IMA= Isometric Muscle Activity; [2]VAS = Visual Analogue Scale, [3]TSK = Tampa Scale of Kinesiophobia; [4]Catas = Catastrophjzing; [5]NDI = Neck Disability Index

Patient characteristics, EMG assessments, and the questionnaires were obtained on average 8.1 (sd = 3.7) days after the MVA. Twenty-four weeks later the subjects were classified based on their NDI scores as disabled (n = 27) and recovered (n = 55). Baseline characteristics of the two groups (Table 1) reveal that the disabled patients have a higher mean Body mass Index (BMI), suffer from more intense neck pain and disability, and have higher responses on the TSK and PCL-catas, and lower IMA levels. There were no significant differences on any of the demographic or collision-related variables.

Table 2: Test performance of five predictive tests, separately and in combination.

First test								second test conditional on first test	
	AUC	95% CI	cut off	LR+	PPV	LR-	NPV	AUC	95% CI
NDI	0.872	0.794 - 0.950	15	2.4	54.3	0.11	5.6		
VAS	0.820	0.727 - 0.912	27	1.9	48.1	0.14	6.9	0.718	0.569 - 0.867
TSK	0.763	0.659 - 0.866	34	1.7	45.5	0.16	7.7	0.770	0.634 - 0.907
CATAS	0.782	0.676 - 0.888	15	1.4	41.7	0.2	9.5	0.734	0.588 - 0.881
IMA	0.707	0.588 - 0.826	165	1.3	38.5	0.26	11.8	0.683	0.523 - 0.843

NDI= Neck disability index; VAS= pain intensity Visual Analogue Scale; TSK= Tampa scale for kinesiophobia; CATAS= catastrophizing, IMA = Isometric muscle activity.
AUC= Area under Receiver Operating Curve, 95% CI= Ninety five percent Confidence Interval, LR+=Likelihood ratio for a positive test result, LR- =Likelihood ratio for a negative test result. PPV= positive predictive value, NPV=negative predictive value.
For the calculation of the PPV and NPV, the prevalence of disability was estimated from our study.

The overall accuracy of the five predictive tests, as determined by ROC curves, shows an area under the curve (AUC) that is statistically significant higher than 0.5 (Table 2). The NDI (AUC = 0.872, 95% CI = 0.794-0.950) and painVAS (AUC = 0.820, 95% CI = 0.727-0.912) demonstrate the best ability to predict outcome 24 weeks post accident. IMA levels appeared to have the least predictive ability (AUC = 0.707, 95% CI = 0.588-0.826). We chose to use the NDI as a first test as the best predictor and then to test whether further improvement in predictive value could be achieved when combining this variable with each of the other variables. The overall test accuracy showed that each combination of tests resulted in a statistical significant AUC higher than 0.5 (Table 2). The TSK score

(AUC = 0.770, 95% CI = 0.634-0.907) appeared to have the best ability to further increase the predictive value, when used as a second test.

Figure 2 visually depicts an evaluation scenario using the combination of NDI with a cut off point of 15 in a first test, and TSK with a cut-off point of 40, as a second test. This test strategy resulted in a probability of disease of 54.3% (95% CI = 35.2-72.3%) after using the NDI as a first test. Subsequently, the TSK score further increased the prediction of disease to 83.3% (95% CI = 70.3-91.3%) in 18 out of 46 patients. The remainder of the 28 patients showing a TSK score lower than 41 did not benefit from the second test. The negative predictive value determined by NDI in a first test is 5.6%, or conversely, the probability of a favorable outcome is 94.6 %, and this is valid for 36 out of 82 patients. Additional testing with the TSK does not improve this post-test probability substantially.

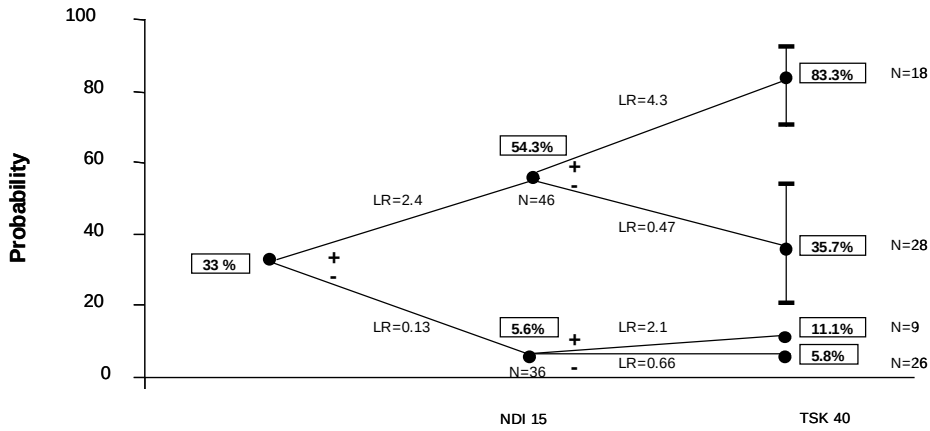

Figure 2: Probability plot combining the Neck Disability Index (NDI: cut off 15) and Tampa Scale of Kinesiophobia (TSK cut off 40). [+] indicates a positive test results, [-] indicates a negative test result. LR=Likelihood ratio, N= number of patients.

DISCUSSION

The aim of the present study was to improve clinical decision making in patients with acute post-traumatic neck pain due to a whiplash injury. The results indicate that a simple rating of baseline neck pain disability (NDI) can be used to predict which patients will be still disabled 6 months after a whiplash injury. Furthermore,

combining the baseline neck pain disability with fear of movement, will improve the prediction of outcome significantly. As such, these variables, either separately or in combination, are capable of identifying which patients require an early intervention to prevent chronic disability.

Interventions that promote physical activity have proven to be most effective in the early treatment of post-traumatic neck pain patients.[1,17,19] Consequently, patients showing 'avoidance' behavior should be encouraged to 'confront' physical activity despite pain, in order to prevent chronic symptoms.

The current study differs from previous ones investigating the prediction of chronic post-traumatic neck pain in several ways. The results of the current study provide additional information that may be useful in the management of acute post-traumatic neck pain. By evaluating the involvement of 'fear avoidance' in the acute pain situation, the patients at risk for showing an 'avoidance' behavior style, can be identified. This behavior may seriously interfere with the 'active' intervention programs, and thus slow recovery. Thus, providing patients with a high fear of movement a structured treatment program, focusing on gradual confrontation with fear eliciting activities, might increase the efficacy of 'active' treatment. The effectiveness of such a 'graded exposure in vivo' treatment program was demonstrated in chronic low back pain patients.[29,30] By gradually 'confronting' the patients with physical activity that was perceived as harmful, the level of fear of movement, fear of pain and pain catastrophizing were decreased. This was accompanied by an improvement in physical functioning.

Another difference between the present and previous prognostic studies concerns the methodology. Most often, a regression model is used that calculates the most optimal combination of tests that can maximally predict outcome. The choice of determinants in such a model is based on statistical grounds. In the current study we calculated the subsequent change in probability of the second test conditional on the first test. Such a test sequence not only enables us to calculate the additional predictive value of a second test, but also provides clinical information on 4 test results with easier interpretation for clinical purpose. As such, the increase in prediction of outcome by using the TSK as a second test, gives direction to treatment strategy. The baseline NDI cutoff point of 15 identifies patients who perceive the acute pain situation as seriously interfering with daily activities. If their situation does not change, they may need intensive multidimensional rehabilitation in future. Furthermore, the TSK cut-off point of 40 was chosen because the Vlaeyen et al. study[30] showed that particularly patients

with such a high fear of movement or higher, may benefit from graded exposure in vivo treatment, focusing on confrontation with fear eliciting activities.

In addition to the effectiveness of graded exposure in vivo in clinical experiments, the use of a booklet with information and advice on fear avoidance beliefs in primary care setting also have proven to be effective in improving clinical outcome.[3] Structured advice and information booklet may be very helpful in the emergency department of hospitals,[11] because pain originating from traumatic events may cause fear avoidance beliefs.[23]

The role of the isometric muscle activation in predicting outcome was modest. The test accuracy was low compared to the NDI and the TSK. However, the additional advantage of IMA is that it may identify patients in whom fear of movement actually is accompanied by a change in physical performance. The fear-avoidance model assumes that a persistent reduction in daily physical activity may result in a worsened physical condition in the long term, thereby contributing to physical disability.[28,31] Although the decrease in muscle activity has been shown to be associated with fear of movement,[14] whether the persistence of this decreased muscle activity will result in physical deconditioning needs to be demonstrated. So, it is not clear whether retraining of normal muscle coordination can increase the effectiveness of an active intervention program.

The results of our study need to be interpreted cautiously. Particularly in post-traumatic neck pain, symptom expectation and amplification are suggested to contribute to chronicity of the condition.[6] This is supported by the fact that in cultures with low therapeutic involvement and no litigation, symptoms tend to be short lived with little or no link to chronicity.[15,16] Because the sample of subjects in this study are assessed repeatedly during follow-up for other analyses,[13] it is likely that there was increased attention to the symptoms. The assessments took place in a research department of a rehabilitation clinic that is particularly well known in the region by rehabilitation of whiplash patients. Consequently, effects of symptom expectation and amplification cannot be ruled out.

In general, the validity of prediction of a (set of) variables requires external validation (e.g in an independent population, or alternatively, by using a statistical technique with sample splitting). In order to decrease the effects of symptoms amplification, a replication of our study with only a baseline and outcome assessment may therefore result in a more representative prevalence of disabled patients and more accurate predictive values.

With regard to the practical implications of our study, the assessments of the NDI and the TSK ask simple questions, which appear to be easily understood, and

require only a few minutes to complete. This implies that, even in emergency departments, a quick risk profile can be achieved and the decision to prescribe an active intervention program or just watchful waiting can be easily made. In addition, because hectic situation in emergency departments, booklets with advice and information on fear avoidance beliefs[2,11] may be used as a good alternative to more extensive intervention.

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CHAPTER 7

GENERAL DISCUSSION AND CONCLUSIONS

GENERAL DISCUSSION

As outlined in the introduction, the injury severity classification system of post-traumatic neck pain patients has been based on the clinical presentation of signs and symptoms. The Whiplash Associated Disorder (WAD) grade 2 is defined by the presence of musculoskeletal signs, including 'muscle spasm'. However, the straightforward concept of ‘muscle spasm caused by a painful injury’ is questionable. More complex relationships among pain and muscle control are described by the 'pain adaptation model' and the 'fear avoidance model'.

In this final chapter I present a discussion on how to interpret the different muscle activation patterns observed in acute and chronic pain, and their role in the transition of acute to chronic pain. In addition, we discuss the usefulness of assessing muscle activity in the clinical setting. We end with some methodological issues.

MUSCLE ACTIVATION PATTERNS IN ACUTE AND CHRONIC POST TRAUMATIC NECK PAIN

In the literature, the 'pain-spasm-pain' model and the 'pain adaptation' model have been posed as reflecting two opposing hypotheses, each with a different interaction between pain and muscle activation. The results of different studies, either revealing an increased *or* a decreased muscle activity, were considered as evidence which legitimates rejecting the alternative hypothesis.[8,15]

An important point of interest in our research is the finding of *both* kinds of muscle activation patterns in post-traumatic neck pain. Muscle activation patterns in acute post-traumatic pain are characterized by a decrease in activity (Chapter 4), [12] whereas in chronic neck pain disability muscles react with an increase in activity (Chapter 2 and 3).[10, 11] This suggests that the 'pain-spasm-pain' model and the 'pain adaptation' models do not contradict each other, but display different aspects of the chronification process, each with its own developmental time frame. As such, avoiding painful movements, aiming at minimizing the use of painful muscles, is likely to be a normal protective adaptation in acute musculoskeletal pain. In addition, the increase in muscle activity *in reaction to* a physical exercise is a psychophysiological stress-related response, which is a characteristic of a more general chronic pain syndrome. It is likely that a subgroup of acute neck pain patients who will develop chronic disability will also build up such an increase in muscle reactivity in the long term. Future research demonstrating such a transition

during a prolonged follow-up period (e.g. 2-3 years) will further clarify the discussion in literature concerning the two pain models.

The two distinct muscle activation patterns we found both have significance for clinical practice. Pain rehabilitation program can be refined by including a stress-management component for those chronic neck pain patients who react with stress-related hyperreactivity. We did not investigate the exact mechanism by which the patients were aroused when performing the exercises, but according to the fear avoidance model, fear of movement may play a significant role in the increased arousal. Further investigations should be focussed on this possibility, thereby giving reason to direct treatment toward dealing with this fear of movement.[19, 20]

In the acute phase of post-traumatic neck pain, the avoidance muscle behavior has the ability to predict outcome (Chapter 6).[14] However, the predictive value was much smaller than both pain and disability assessments. Furthermore, from a practical perspective, the Neck Disability Index (NDI) and the Tampa Scale for Kinesiophobia (TSK) ask simple questions, which appear to be easily understood, and require only a few minutes to complete, may be included as part of assessment. This implies that, even in emergency departments, a quick risk profile can be achieved and the decision to prescribe an active intervention program or just watchful waiting can be easily made. In contrast, the use of sEMG in emergency departments or in primary care setting is less practical because it is time consuming, requires expensive equipments and trained personal.

Although the predictive ability of the 'avoidance muscle behavior' is low, there is a possible role in the therapeutic setting. After having identified the patients at risk for chronic neck pain by the NDI and TSK scores, the additional assessments of muscle activity may identify patients in whom fear of movement actually is accompanied by a change in physical performance. Since the most effective prevention of chronicity is to stay physically active,[18] a retraining of this change in muscle coordination, might be an important component in the early intervention. However, we first need to explain whether the decrease in muscle activity contributes to the persistence of pain. Thus, it is still not clear whether a return to normal muscle coordination is effective for the prevention of chronic symptoms.

THE ROLE OF MUSCLE ACTIVATION IN THE TRANSITION OF ACUTE TO CHRONIC PAIN

The studies reported in this thesis are the first that prospectively investigate the muscle activation patterns of acute neck pain patients untill they recover or develop chronic disability. The prospective design of the studies offers an opportunity to study the evolution of adaptive and maladaptive processes leading to these outcomes. Considering the different mechanisms described in the pain-spasm-pain model and the pain adaptation model, an important remaining question is how the muscle activation patterns play a role in the development of chronic pain disability.

In the pain-spasm-pain model, it is hypothesized that pain and increased muscle activity are reciprocally linked, setting up the 'vicious circle' that maintains the condition. However, as demonstrated in our research, the vicious circle posited is not instigated in the acute phase of neck pain. Moreover, the finding that the muscle hyperreactivity appears at the moment that the chronic pain condition has been established, suggests that this muscle reaction is not initiated by acute pain but rather by secondary factors related to persistent suffering from pain.

In the 'pain adaptation' model the inhibition of muscle activation is considered an adaptive reaction in order to avoid painful movement and reinjury. As such, the decrease of upper trapezius muscle activity is simply a normal protective adaptation in response to pain. Because the model does not involve a vicious circle, its role in the development of chronic pain remains unclear.

Alternatively, from the perspective of the fear avoidance model, a persistent reduction in physical activity may result in a worsened physical condition in the long term, thereby contributing to physical disability. However, research is needed to explain how the observed decrease in muscle activity in the current study can result in ‘deconditioning’ effects and disability.

We can conclude from this research that, the muscle activation patterns are secondary manifestations of a musculoskeletal pain condition. However, we did not demonstrate that these muscle activation patterns play a significant role in the transition of acute to chronic pain.

METHODOLOGICAL ISSUES

NORMALIZATION OF UPPER TRAPEZIUS MUSCLE ACTIVITY

In studies comparing the EMG signal between groups and between days, technical and physical factors influence the EMG signals. Technical factors include the

specific electrode configuration, such as the electrode and skin impedance and the exact location and spacing of the electrodes. Physical factors include thickness of the subcutaneous fat layer of the neck region. Therefore, the EMG signal need to be normalized (i.e., expressed in terms of a signal obtained during standardized and reproducible conditions).[7,9] In our research we used a normalization procedure, expressing the EMG amplitude as a percentage of a submaximal reference contraction

In Chapter 5 we used this same reference contraction to investigate which factors contribute to the level of muscle activity. Therefore, as an alternative correction for the effects of subcutaneous fat on the EMG signal we included the body mass index (BMI) in the analysis. In agreement with what we expected, a large part of the variability of the EMG signal could be explained by the BMI. As such, these results confirm the necessity of normalizing EMG signals for interindividual comparisons. However, a substantial part of the variability of the EMG signal could be explained by pain and fear of movement. This indicates that we cannot completely rule out the effects of pain related fear on the performance of such submaximal reference contractions.

It is apparent that in investigations that wish to study inter-individual differences in EMG level should normalize the signal to decrease the contrast caused by differences in BMI. However, by normalizing there is a risk of leveling out the differences in pain and fear of movement. This indicates that particularly in studies that compare groups of pain patients with healthy control subjects, the choice not to normalize the EMG signal should be controlled for by matching the subjects for BMI.

SELECTION OF PATIENTS

A point of interest was that the group of chronic WAD II patients demonstrated muscle hyperreactivity (Chapter 2 and 3), whereas the acute neck pain patients in the prospective study did not (Chapter 4). Part of these differences can be explained by differences in selection of patients.

Because muscle hyperreactivity is a stress-related response[5,6] , a possible variable that is responsible for the differences is stress vulnerability. Burns et al[1] showed however, that the association of muscle hyperreactivity and pain is modified by feelings of depression and emotional vulnerability to stress. Therefore, we expect that acute neck pain patients differ from chronic neck pain patients particularly with respect to these variables. The chronic WAD II patients were

recruited from a waiting list of a pain rehabilitation program. In these patients, the chronic pain considerably interfered with their daily physical, social, and mental functioning. Blokhorst et al[2] demonstrated that such a group of chronic WAD patients are more vulnerable and react to everyday stressors with more distress than healthy people. This was particularly the case when these stressors were a consequence of the impaired functioning, caused by the whiplash injury. In addition, Borchgrevink et al.[3] demonstrated that chronic WAD patients showed signs of depression. However, these signs developed with a certain time lag since the initial whiplash injury. This indicates that depression is also secondary to suffering from chronic pain.

In conclusion, the chronic patients from the waiting list are not just a sample of the group of patients in the prospective cohort study who were still disabled at 6 months follow-up. Rather, they are a set of patients that react with secondary physical and psychologic symptoms. Since that patients with acute and chronic pain show essential differences in these pain related aspects, the selection of patients in pain research should be performed with great care.

VALIDITY OF PREDICTION

The results of the prospective study, investigating the predictive value of several variables, need to be interpreted cautiously. Particularly in post-traumatic neck pain, symptom expectation and amplification are suggested to contribute to chronicity of the condition[4]. This is supported by the fact that symptoms tend to be short lived with little or no link to chronicity in cultures with low therapeutic involvement and no litigation.[16,17] Because the subjects in this study were assessed repeatedly during follow-up for other analyses (Chapter 4 and 5), one could argue that there was increased attention to the symptoms. The assessments took place in a research department of a rehabilitation clinic that is particularly well known in the region by rehabilitation of whiplash patients. Consequently, effects of symptom expectation and amplification cannot be ruled out.

Because the validity of prediction of a (set of) variables requires external validation in an independent population, we recommend that a replication study should be desgned with only assessments of baseline parameters and outcome. This may result in a more representative prevalence of chronic disability.

GENERAL CONCLUSIONS

The research described in this thesis shows two distinct activation pattern of the trapezius muscles in post-traumatic neck pain. These muscle activation patterns are altered by two distinct mechanisms, each with its own developmental time frame, cause and therapeutic consequences. The decrease in muscle activity in the acute phase is a reflection of pain, but also of fear of movement. The increase in muscle activity in the chronic phase seems to be a stress-related phenomenon. As such, both muscle activation patterns are not in agreement with the concept of the injury severity classification system proposed by the Quebec Task Force, because it is not the injury severity that determines the muscle activation patterns but rather the perception of the symptoms.

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SUMMARY

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In Western industrialized societies, the development of chronic pain and related disability following a whiplash injury has become a significant public health problem. Although it is generally accepted that the acute symptoms in whiplash associated disorders are attributed to soft tissue injuries of the neck (‘neck sprain’), there is no convincing evidence of a specific lesion that could cause chronic damage in the neck and as such be linked to chronic pain and disability. Rather, the disease entity 'chronic WAD’ is assumed to be caused by multiple factors, both physical and non-physical.

In 1995, the Quebec Task Force proposed a classification system offering four grades of WAD, roughly corresponding to the severity of injury. The characteristic feature of WAD grade 2 is the presence of 'neck pain and musculoskeletal signs.' These musculoskeletal signs are manifested in a *limited range of motion* putatively due to *muscle spasm*. Muscle spasm is ‘electromyographic (EMG) activity that is not under voluntary control and not dependent upon posture’. Several studies report limitations in range of motion in neck trauma patients meeting criteria for WAD grade 2, and thereby provide some support for the WAD classification system. However, there are no studies that directly confirm the presence of increased muscle activity in these patients. Moreover, in models describing the development of musculoskeletal pain there are controversies whether the muscle activation patterns are characterized by an increased or rather by a decreased level of muscle activity. Clearly, therapeutic management will be different.

Therefore, the two main aims of this thesis were:

§ To clarify the characteristics of 'musculoskeletal signs' in acute and chronic post traumatic neck pain patients.

§ To determine its clinical relevance in relation to the management of acute and chronic post traumatic neck pain patients

The starting point in this thesis was that, in accordance with the WAD injury severity system, WAD grade II would display a muscle activation pattern characterized by an increase in muscle activation. First, we investigated whether this assumption was correct. However, we did not know the optimal task condition that would demonstrate increased muscle activity. So, the aim of this first study (Chapter 2) was to evaluate which tasks could be used to discriminate between

patients designated as chronic WAD grade II and healthy control subjects. Surface electromyographic (sEMG) analysis of the upper trapezius muscles was performed during static, dynamic, and relaxation tasks.

The results show that there are two particular conditions in which WAD II patients exhibit higher EMG activity of the upper trapezius muscles than the healthy controls. First, during the performance of a unilateral dynamic task, the WAD-II patients show a substantially higher co-activation level of the upper trapezius muscles of the contralateral (resting) arm as compared to the healthy subjects. Secondly, even larger differences between the two groups were demonstrated after having finished the dynamic task. WAD-II patients were not able to relax the upper trapezius muscles to baseline levels. Both findings indicate that WAD II patients exhibit unnecessary increased muscle activation, particularly in situations in which there is no biomechanical demand for it.

We considered this 'muscle reactivity' to result from an increased psychophysiological arousal and pain. As such, we hypothesized, that this muscle activation pattern could be considered a nonspecific sign. To test this hypothesis, we replicated the sEMG study in neck pain patients with and without a history of traumatic whiplash injury (Chapter 3). As expected, we found no differences in muscle activation patterns between the two groups. These results confirm that the acceleration-deceleration trauma is not an exclusive cause for the muscle activation pattern. Thus, the muscle reactivity can be considered as a psychofysiological response, present in diverse chronic neck pain syndromes.

In accordance with the pain-spasm-pain model, the assumption in these studies in chronic WAD was that the increase in muscle reactivity was initiated in the acute phase, as a protective reaction to prevent painful movements. Furthermore, the muscle reactivity was assumed to be part of a "causal," pain-eliciting psychophysiological mechanisms, resulting in a vicious circle of pain and increased muscle activity reinforcing one another. As such, we hypothesized that the muscle reactivity, would be present, or would soon become apparent in the acute pain condition. Furthermore, we suggested that this muscle activation pattern would persist in patients with chronic neck pain disability, whereas it would disappear in patients who recovered.

These considerations were the basis for the design of a prospective cohort study, in which we followed up a cohort of patients with acute neck pain due to a motor vehicle accident. Subjects were assessed five times with baseline assessment performed within 1 week, and follow-up assessments 4, 8, 12, and 24 weeks after the motor vehicle accident.

In contrast to what was expected, we found that the traumatic injury does not initiate hyperreactivity of the upper trapezius muscles in acute post traumatic neck pain (Chapter 4). In addition, during the transition from acute neck pain to chronic neck pain disability 6 months post injury, there is still no tendency to develop muscle hyperreactivity. Moreover, throughout this period, the upper trapezius muscles showed a *decreased* rather than increased muscle activation during the task. We conclude that the muscle reactivity is not part of a "causal," pain-eliciting psychophysiological mechanisms, initiated in the acute phase of neck pain. We also conclude that the decrease in muscle activation level, is more in accordance with the (neurophysiological) 'pain adaptation model' *and/or* (cognitive behavioral) 'fear avoidance model.'

The 'pain adaptation model' suggests that nociceptive stimuli of the injury area will result in a *decreased* activity of muscles causing painful movements. Similar to the pain spasm pain model this is considered a useful reflex adaptation, protecting the injured area from further injury and pain. Alternatively, the 'fear avoidance' model suggests that subjects may develop 'fear of movement and physical activity', as these are (wrongfully) assumed to cause (re)injury. During physical exercise, fear of movement may result in a decreased muscle activity, also aiming at avoidance of painful contractions

An intriguing finding in the prospective study, was that the level of muscle activation during physical exercise showed to be inversely related to the extent of disability. Starting from the concepts of both the pain adaptation and fear avoidance model, we assumed a kind of 'dose response' relationship between pain and/or fear of movement and the level of muscle activity. Therefore we performed an additional analysis evaluating the extent to which pain and fear of movement influence the activation patterns of the upper trapezius muscles (Chapter 5). A multilevel analysis shows that in addition to the inhibitory effects of pain, fear of movement is *independently* associated with the level of muscle activation. This means that, in patients with high pain intensity *or* fear of movement, the level of muscle activity used during the task is diminished. These results are in contrast with previous studies in chronic musculoskeletal pain, showing higher contribution of pain related fear to performance level than pain intensity. Our results suggest that in acute pain the nociceptive stimuli still have a direct effect on muscle activity. However, our study also provides some evidence that after the healing phase of the soft tissue injury, the injury related nociception dissipates.

In conclusion the additional analyses supported *both* the 'pain adaptation' *and* the 'fear avoidance' models in acute post traumatic neck pain.

The final question in this thesis was the extent to which the decreased muscle activation patterns and other variables of the fear avoidance model could predict future outcome. Characterization of the behavioral response to pain, confrontation or avoidance, would provide an initial indication of the target for intervention. Therefore, in addition to the severity of acute neck pain, assessed by pain intensity and related disability, we investigated the predictive value of three fear avoidance variables: the 'avoidance' of muscle activation pattern of the trapezius muscles, the level of fear of movement, and the level of catastrophizing (Chapter 6). Outcome of interest was chronic neck pain disability determined by the Neck Disability Index. The results revealed that a simple rating of baseline neck pain disability in combination with 'fear of movement', was most effective in predicting future outcome. In addition, compared to these variables, the test accuracy of the muscle activity pattern is modest. However, the additional advantage of assessing the muscle activity level is that it may identify patients in whom fear of movement actually is accompanied by a change in physical performance. The fear-avoidance model assumes that a persistent reduction in daily physical activity may result in a worsened physical condition in the long term, thereby contributing to physical disability. Although the decrease in muscle activity has been shown to be associated with fear of movement, it needs to be demonstrated whether persistence of this decreased muscle activity will result in physical deconditioning. As such, it is not clear whether retraining of normal muscle coordination can increase the effectiveness of an active intervention program.

In the general discussion (chapter 7), the two characteristic muscle activation patterns in acute and chronic post traumatic neck pain patients, and their relevance for clinical practice are discussed. The research indicates that the pain-spasm pain model and the pain adaptation model do not contradict each other, but display different aspects of muscle behavior, each with a specific developmental time frame, cause and therapeutic consequences.

As an important consequence of our research, we question the relevance of the criteria of the WAD injury severity classification system. We showed that the musculoskeletal signs in WAD grade II are not characterized by muscle spasm, (i.e. increase of muscle activity), but rather by a decrease in muscle activation. Moreover, by also showing a strong association of this muscle activation pattern with fear of movement, we concluded that musculoskeletal signs in post traumatic neck pain do not reflect the severity of injury but rather the perception of symptoms, influenced by psychosocial factors from the fear avoidance model.

SAMENVATTING

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In de westerse gezondheidszorg worden de gevolgen van een whiplashongeval, in Engelstalige literatuur het 'Whiplash Associated Disorder' (WAD) genoemd, als een groot medisch en maatschappelijk probleem ervaren. Het is algemeen geaccepteerd dat klachten in de acute fase na een whiplashtrauma toegeschreven kunnen worden aan traumatische afwijkingen van lichamelijke structuren of functies. Er is echter geen overtuigend bewijs dat in het geval van chronische WAD het persisteren van klachten en beperkingen hun oorzaak hebben in enige vorm van fysieke beschadiging. Het is aannemelijker dat het een multiconditioneel bepaald chronisch pijn syndroom is dat alleen begrepen kan worden vanuit zowel een somatisch als psychosociaal perspectief.

In 1995 werd door de Quebec Task Force een classificatie systeem ontwikkeld, welke de mate van ernst van het klachtenbeeld van acute WAD weergeeft. Dit systeem heeft een onderverdeling in 4 categorieën, waarbij WAD graad 2 wordt gekarakteriseerd door de aanwezigheid van pijnklachten in de nek, gepaard gaande met symptomen in het spierskelet systeem. Deze symptomen uiten zich in een verminderde beweeglijkheid van de nek, waarvan wordt verondersteld dat het wordt veroorzaakt door een verhoogde afweerspanning van de nekspieren. Enkele studies bevestigen een afname van beweeglijkheid van de nek bij WAD 2 patiënten, en onderkennen daarmee het WAD classificatie systeem. Er zijn echter geen studies die direct de verhoogde spierspanning aantonen bij deze patiëntengroep (in de zin van toegenomen activiteit gemeten met oppervlakte electromyografie - EMG). Het is zelfs zo dat de in de literatuur beschreven modellen over pijn en spiergedrag conceptueel verschillen in hoe pijn en spieractiviteit elkaar beïnvloeden. De diverse modellen veronderstellen namelijk zowel een verhoging maar ook een verlaging van spieractiviteit. Het is van belang om te bedenken dat deze tegengestelde inzichten verschillende consequenties hebben voor hoe inhoud gegeven dient te worden aan therapie.
In dit proefschrift is derhalve ten doel gesteld:

- Het inzicht krijgen in de spieractivatie patronen bij zowel acute als chronisch posttraumatische nekpijn.
- Het bepalen van de klinische relevantie van deze spieractivatie patronen in relatie tot het klinisch handelen in acute en chronische posttraumatische nekpijn.

Het uitgangspunt in dit proefschrift wordt gevormd door het WAD classificatiesysteem, waarbij WAD graad 2 wordt gekenmerkt door verhoogde spieractiviteit. Het achterliggende concept is dat een dergelijk spieractivatiepatroon wordt veroorzaakt door pijn. De veronderstelling daarbij is dat door de toename in spieractiviteit pijnlijke bewegingen van nek en hoofd worden vermeden en daarmee verdere beschadiging van het nekletsel kan worden voorkomen. Echter, overmatig of persisterend verhoogde spieractiviteit kan op zich zelf weer een nieuwe bron van pijn worden, waardoor een vicieuze cirkel van pijn -hypertonie-pijn ontstaat. Een dergelijke negatieve spiraal kan uiteindelijk, onafhankelijk van het initiële letsel, tot een chronisch pijnsyndroom leiden.

Het doel van het eerste onderzoek (hoofdstuk 2) was om in chronische WAD graad 2 de veronderstelde spierhypertonie van de nekspieren met oppervlakte EMG aan te tonen. Echter, het was niet op voorhand bekend bij welke taakcondities deze verhoogde spieractiviteit is waar te nemen. Derhalve werd tijdens een 3-tal taken de spieractiviteit van de m. trapezius descendens gemeten: een statische taak, een éénhandige dynamische taak, en een ontspannings taak. Bij 2 taken bleek de groep WAD-graad 2 patiënten een hogere mate van spieractiviteit te vertonen dan gezonde controle personen. Ten eerste is dit tijdens het uitvoeren van de éénhandige dynamische taak. WAD-graad 2 patiënten vertonen ten opzichte van gezonden, een substantieel hoger co-activatie van de (niet bij de oefening betrokken) contralaterale m.trapezius descendens. Ten tweede blijkt dat er zelfs een groter verschil tussen deze beide groepen te zien is als EMG gemeten wordt direct nadat de éénhandige dynamische taak is beëindigd. De WAD patiënten blijken dan namelijk niet in staat om de activiteit van de m.trapezius descendens te laten terugkeren tot een normaal rustniveau. Ofwel, behalve dat er een toegenomen spieractiviteit is *tijdens* de dynamische taak is er tevens een soort reactiviteit van de nekspieren *na* het uitvoeren van de dynamische taak. Opvallend daarbij is dat in beide situaties de verhoging in spieractiviteit, in biomechanische zin, onnodig is.

De veronderstelling was derhalve dat deze verhoogde activiteit van de nekspieren een vorm van psychofysiologische reactiveit is wat veroorzaakt wordt door een verhoging van het alertheidsniveau (arousal) en/of pijn. Een tweede veronderstelling was dat deze spieractivatiepatronen niet specifiek zijn voor nekpijn patiënten waarbij de pijn van traumatische oorsprong is. Om dit te testen hebben we het EMG experiment herhaald met nekpijnpatiënten mèt, en zonder

een traumatisch begin van de klachten (hoofdstuk 3). Conform onze verwachtingen was in beide groepen een vergelijkbare mate van reactiviteit van de nekspieren uit te lokken met de dynamische taak. De conclusie was derhalve dat de reactiviteit van de nekspieren als een aspecifiek psycho-fysiologische response is te beschouwen welke kenmerkend is voor een algemeen chronische pijnsyndroom.

In lijn met de theorie beschreven in het pijn-hypertonie-pijn model, werd verondersteld dat de toegenomen reactiviteit van de nekspieren direct na het trauma geïnitieerd wordt. Onze hypothese was derhalve dat deze reactiviteit van de nekspieren reeds in de acute pijnfase met oppervlakte EMG aan te tonen zou moeten zijn. Tevens zou volgens deze theorie de reactiviteit van de nekspieren moeten normaliseren in patiënten die herstellen, en persisteren in patiënten die chronische klachten ontwikkelen.
Deze overwegingen vormden aanleiding voor het ontwerpen van een prospectieve cohort studie waarbij een groep mensen met acute nekpijn na een auto-ongeval voor langere tijd worden vervolgd. Tijdens de follow-up werden de deelnemers aan deze studie vijf maal gemeten, waarbij baseline metingen na 1 week, en vervolgmetingen 4, 8, 12 en 24 weken na het ongeval werden verricht.

In tegenstelling tot wat wij hadden verwacht, werd er geen verhoogde reactiviteit van de nekspieren gevonden tijdens de fase van acute post traumatische nekpijn (hoofdstuk 4). Er was gedurende de 6 maanden follow-up ook geen trend zichtbaar waarbij de reactiviteit van de nekspieren tot ontwikkeling kwam. Sterker nog, gedurende de gehele follow-up periode bleek er zelfs een afname van activiteit van de nekspieren te zijn *tijdens* het uitvoeren van een fysieke taak. We kunnen derhalve uit dit onderzoek concluderen dat in tegenstelling tot onze bevindingen bij chronische WAD patiënten er in de acute pijn fase geen psychofysiologische reactiviteit van de nekspieren is uit te lokken. Als zodanig lijkt dit spieractivatiepatroon geen deel uit te maken van een causale keten reactie. We kunnen tevens concluderen dat de afname in niveau van spieractiviteit meer in lijn is met twee andere modellen die de relatie tussen pijn en spieractiviteit beschrijven, namelijk het pijn -adaptatie model en het pijn -vermijdingsmodel.

Het neurofysiologische georiënteerde *pijn-adaptatie-model* is gebaseerd op het idee dat nociceptieve prikkels via het ruggenmerg, reflectoir tot een afname van activiteit leidt in met name die spieren die een pijnlijke beweging kunnen bewerkstelligen. Het cognitief gedragsmatige *pijn-vermijdings-model* gaat uit van een

proces waarbij pijn via een keten van psychologische reacties tot een excessief vermijdende gedragsstijl leidt. Onderdeel in deze keten is een catastroferende denkstijl, waarbij patiënten (onterecht) veronderstellen dat belastende fysieke activiteiten tot (hernieuwd) letsel en/of blijvende pijntoename leidt. Een dergelijke denkstijl resulteert in bewegingsangst (of kinesiofobie) wat zich kan uiten in het vermijden van pijnlijke bewegingen en/of fysieke activiteiten. Als zodanig kan de verminderde spieractiviteit tijdens een motorische taak verklaard worden door bewegingsangst.
Een opmerkelijke bevinding van deze studie was dat de hoogte van de spieractiviteit tijdens een fysieke taak omgekeerd evenredig bleek te zijn met de mate van ervaren nekbeperkingen, zoals gemeten met de nek beperkingen vragenlijst. Met het pijn-adaptatie- model en het pijn-vermijdingsmodel als uitgangspunt veronderstelden we een dosis -response relatie tussen de mate van pijn en/ of bewegingsangst en de hoeveelheid spieractiviteit. Met behulp van multilevel analyse technieken werd aangetoond dat in acute posttraumatische nekpijn patiënten, *zowel* pijn *als* bewegingsangst een remmende invloed heeft op de hoeveelheid spieractiviteit (hoofdstuk 5). Deze resultaten zijn in tegenspraak met eerdere onderzoeken in chronische lage rugpijnpatiënten, waarbij bewegingsangst een groter negatief effect bleek te hebben op de prestatie tijdens een fysieke taak dan de hoeveelheid pijn. Een mogelijke verklaring is dat in de acute fase de nociceptieve prikkels van het traumatisch letsel nog van invloed zijn op spieractiviteit. Deze veronderstelling wordt ondersteund door de bevinding dat na de herstelperiode van het acute letsel het effect van pijn op spieractiviteit geleidelijk aan minder wordt. De conclusies van deze analyses zijn derhalve dat de resultaten zowel het pijn-adaptatie model alsook het pijn-vermijdingsmodel onderschrijven.

Uitgaande van de gevonden spieractivatiepatronen in de acute fase van posttraumatische nekpijn en de onderliggende neurofysiologische en psychologische mechanismen, hebben we een prognostische studie opgezet met de data van het prospectieve cohort (hoofdstuk 6). De vraagstelling was in welke mate de variabelen uit het pijn-vermijdingsmodel de toekomstige gezondheidstoestand beter kan voorspellen dan pijn en/of pijngerelateerde beperkingen. De klinische relevantie van het karakteriseren van vermijdende dan wel confronterende gedragsreacties op acute pijn, is dat daarmee tevens de inhoud van therapie beter vormgegeven kan worden. In het geval dat er bijvoorbeeld sprake is van een catastroferende denkstijl en/of bewegingsangst kan gedacht worden aan een meer specifieke behandeling gericht op het verminderen van deze angst.

Er werd een analyse verricht met als voorspellers: pijnintensiteit, ervaren beperkingen, het niveau van spieractiviteit, de mate van bewegingsangst, en de mate van catastroferende gedachten. De resultaten laten zien dat de ervaren beperkingen in combinatie met de mate van bewegingsangst, het meest voorspellend is voor de toekomstige gezondheidstoestand. Beide variabelen worden met een korte en weinig tijdsintensieve vragenlijst afgenomen en vormen daarmee een éénvoudige screening om bij de eerste opvang van patiënten met post traumatische nekpijn een snelle inschatting te maken van het risico van chroniciteit. De voorspellende waarde van het spieractivatie –niveau bleek slechts matig te zijn. Echter, het voordeel van het meten van spieractivatie –niveau in aanvulling op de twee vragenlijsten, is dat patiënten kunnen worden geïdentificeerd waarbij de bewegingsangst daadwerkelijk tot uiting komt in verminderd fysiek presteren. Het pijn-vermijdingsmodel veronderstelt dat een langdurig aanhoudende verminderde fysieke activiteit tot een verslechterde fysieke conditie kan leiden. Alhoewel in ons onderzoek de afname in spieractiviteit geassocieerd bleek te zijn met bewegingsangst, dient echter nog te worden aangetoond of het persisteren van verminderde spieractiviteit uiteindelijk tot deconditionering van deze spieren leidt. Het in de literatuur aangetoonde bewezen gunstige effect van 'fysiek actief blijven' op herstel, doet vermoeden dat het herleren van een normale spiercoördinatie gezondheidsbevorderend is.

In de algemene discussie (hoofdstuk 7) wordt de betekenis van de twee karakteristieke spieractivatiepatronen voor de ontwikkeling van chronische pijn besproken. Tevens wordt de relevantie van beide modellen voor het klinisch handelt besproken. Geconcludeerd wordt dat het pijn-hypertonie-pijn model en het pijn-adaptatie model elkaar niet tegenspreken, maar dat ieder model verschillende aspecten van posttraumatische nekpijn weergeeft. De kenmerkende spieractivatiepatronen zoals beschreven in deze modellen hebben beiden een specifieke ontstaanswijze en ontwikkeling in het verloop van de tijd en daarmee specifieke implicaties voor therapie.
Een belangrijke consequentie van ons onderzoek is dat de relevantie van het WAD classificatiesysteem in twijfel getrokken dient te worden. We hebben namelijk aangetoond dat WAD graad 2 niet door een verhoogde, maar een door een verlaagde spieractiviteit wordt gekenmerkt. Tevens blijkt deze verlaagde spieractiviteit sterk geassocieerd te zijn met bewegingsangst. Derhalve concluderen wij dat het specifieke spieractivatiepatroon in acute WAD niet zozeer een uiting is van de ernst van het letsel, maar vooral van de perceptie van de klachten. De

manier waarop de pijn beleefd wordt, wordt in sterke mate beïnvloedt door psychologische processen zoals beschreven in het pijnvermijdingsmodel.

NAWOORD

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De totstandkoming van dit proefschrift heeft zeven jaar geduurd. Op het moment dat ik dit nawoord schrijf ligt het concept bij de commissieleden ter beoordeling. Het wordt tijd om nog eens van een afstand op de afgelopen 7 jaar terug te kijken.

Een promotieonderzoek begint vaak bij een stuk verbazing en verwondering over een bepaald onderwerp. Na lang gisten en borrelen kan dat leiden tot het formuleren van onderzoeksvragen. Als het aantal vragen toeneemt en antwoorden niet direct gevonden worden kan dat leiden tot het besluit een promotieonderzoek te starten. Aldus de ideale manier waarop een nieuwe promovendus zijn aantree doet.

Hoe anders ben ik in 1995 dit promotieonderzoek ingerold. Tijdens een tweede gesprek in een sollicitatieronde naar een opleidingsplaats revalidatiegeneeskunde werd me door professor Zilvold namelijk de mogelijkheid geboden als 'Assistent Geneeskundige In Opleiding tot Klinische Onderzoeker' (AGIKO) in 'Het Roessingh' aangesteld te worden. Dit voor de revalidatie nieuwe concept bood de mogelijkheid om naast de specialistenopleiding tevens een periode onderzoek te doen. Dit zou resulteren in een promotie aan de technische universiteit Twente. Ik had nog niet eerder over een dergelijke mogelijkheid nagedacht, en met name de associatie met het 'technische' aan het geheel bracht me nog even in twijfel, aangezien ik niet eens in staat was mijn eigen band op een behoorlijke manier te plakken. En 7 jaar weg uit de Randstad is een aardig lange periode.

Ik ben nu 7 jaar verder en ik kijk met veel genoegen terug naar de periode die ik als arts en onderzoeker in het Roessingh heb mogen werken. Alle genoemde twijfels lijken nu wat onwerkelijk als ik besef hoeveel het mij inmiddels heeft opgeleverd. De specialistenopleiding is afgerond, en het eindresultaat van het promotieonderzoek ligt voor me. Mijn gezinsleven heeft zich geworteld in Enschede en mijn kinderen spreken inmiddels accentloos Twents. En, ondanks dat ik nog steeds geen banden kan plakken, is er voldoende reden een aanstelling overeen te komen in een (revalidatie-) technologische omgeving van Roessingh, Research and Development.
Er zijn velen die hebben bijgedragen aan het volslagen van dit onderzoek en het plezier dat ik daarbij heb beleefd. In het besef dat enkele mensen zich vergeten

kunnen voelen, wil ik toch met name een aantal personen nadrukkelijk bedanken in dit nawoord.

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Inmiddels is Roessingh, Research and Development zo groot gegroeid, dat ik de overige mensen niet ieder apart met name kan noemen, maar als groep wil bedanken voor hun betrokkenheid.

Het personeel van het Medisch Spectrum Twente, het Streekziekenhuis MiddenTwente, en het Twenteborghziekenhuis wil ik bedanken voor hun bereidwilligheid mee te helpen bij het rekruteren van de patiënten.

Het belangrijkste wat een promovendus tijdens een promotieonderzoek deelt met zijn gezin, zijn de sociale opofferingen. Annelies, je meent dat deze promotie nauwelijks jou verdienste is, en dat je daarom niet in het dankwoord thuishoort. Ik denk daar beslist anders over.

CURRICULUM VITAE

Marc Nederhand werd geboren op 15 juli 1966 en is opgegroeid in Heemskerk, Noord Holland. Na het volgen van de middelbare school in Beverwijk studeerde hij geneeskunde aan de Vrije Universiteit van Amsterdam. Hij rondde zijn studie af in 1992 en deed nadien diverse assistentschappen, waaronder neurologie en revalidatie. In 1995 begon hij zijn specialisatie revalidatiegeneeskunde in het revalidatiecentrum 'Het Roessingh' in Enschede. Als één van de eersten in dit vak volgde hij een opleidingstraject als assistent geneeskundige in opleiding tot klinisch onderzoeker, en werd daarmee een zogenoemde AGIKO. Deze opleidingsconstructie voorzag in een periode van 4 jaar waarin het specialisme revalidatiegeneeskunde kon worden voltooid. De overige 3 jaar gaven de mogelijkheid om wetenschappelijk onderzoek te verrichten binnen het cluster Non-Invasive Assessment of Neuromuscular Function(ing) (NINA), onder leiding van clusterleider Prof.dr.ir H.J. Hermens.

Momenteel is hij als revalidatiearts in 'Het Roessingh' aangesteld en gedetacheerd in het streekziekenhuis Koningin Beatrix in Winterswijk. Daarnaast is hij als onderzoeker verbonden aan Roessingh, Research and Development. In 'Het Roessingh' houdt hij zich bezig met de organisatie van het onderwijs aan de revalidatieartsen in opleiding. Daarnaast is hij als gast docent verbonden aan de opleiding tot orthopedisch instrumentmaker van het SVGB in Nieuwegein.