The Effect of Fear of Movement on Muscle Activation in Posttraumatic Neck Pain Disability

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Abstract: Studies using surface electromyography have demonstrated a reorganization of muscle activation patterns of the neck and shoulder muscles in patients with posttraumatic neck pain disability. The neurophysiologically oriented "pain adaptation" model explains this reorganization 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 effects of pain, modulates the muscle activation level. We analyzed the extent to which pain and fear of movement influenced the activation patterns of the upper trapezius muscle during the transition from acute to chronic posttraumatic neck pain.

Ninety-two people with an acute traumatic neck injury after a motor vehicle accident were followed up for 24 weeks. Visual analog scale ratings of pain intensity, response on the Tampa Scale of Kinesophobia—fear of movement, and surface electromyography of the upper trapezius muscles during a submaximal isometric physical task were obtained at 1, 4, 8, 12, and 24 weeks after the motor vehicle accident.

Multilevel analysis revealed that an increased level of both fear of movement (t value = -2.19, $P = 0.030$) and pain intensity (t value = -2.94, $P = 0.004$) were *independently* associated with a decreased level of muscle activation. Moreover, the results suggest that the association between fear of movement and lower muscle activity level is stronger in patients reporting high pain intensity (t value = 2.15, $P = 0.033$). The contribution of pain intensity to the muscle activation level appeared to decrease over time after the trauma (t value = 2.58, $P = 0.011$). 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.

Key Words: WAD, motor vehicle accident (MVA), muscle activity, neck pain disability, fear avoidance, electromyography, kinesophobia

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As proposed by the Quebec Task Force in 1995, whiplash-associated disorder (WAD) is defined as "…an acceleration deceleration mechanism of energy transfer to the neck which may lead to a variety of clinical manifestations…"[1] According to the presenting signs and symptoms, the severity of the injury can be classified into 1 of 4 grades, with higher grades indicating more severe injury (ie, 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 as a *limited range of motion* putatively due to *muscle spasm*[1] that is not under voluntary control and not dependent on posture[2] when observed, increase in muscle activity is postulated to be secondary to soft-tissue injury. However, results of several studies demonstrating a decrease rather than an increase in muscle activity during experimentally induced muscle pain[3–5] conflict with the presence of muscle spasm in injury-related acute pain. Also, a clinical study assessing the surface electromyographic (sEMG) activity of the upper trapezius muscles in patients with acute WAD showed a *decrease* in muscle activity.[6]

Two models, the neurophysiological "pain adaptation" model and the cognitive-behavioral "fear avoidance" model, explain the reorganization of muscle activation patterns in musculoskeletal pain syndromes. In the pain adaptation model, the assumption is that nociceptive interneurons induce reciprocal inhibition at the segmental level.[7,8] Therefore, it would be expected that musculoskeletal injuries would result in a *decrease* in the activity of agonist muscles, causing a painful movement. In addition, a simultaneous *increase* in the activity of antagonist muscles would further prevent this painful movement. These characteristic changes in motor function can be explained as a useful adaptation because they prevent further pain and injury. Such different effects of pain on muscle activation in acute WAD became apparent when in parallel with the Nederhand et al study[6] a similar clinical study in patients with acute WAD showed an increased activity of the sternocleidomastoid muscles.[9]

Alternatively, the cognitive-behavioral perspective, as described by the fear avoidance model,[10] introduces the influence of pain-related fear on behavioral and physical performance. This model explains how fear of movement and/or reinjury can result 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.[11,12] Crombez et al[13,14] confirmed the importance of pain-related fear by demonstrating a significant association between performance level and pain-related fear in a group of patients with chronic low back pain.

A limitation of the studies supporting both models is that the mechanism has been investigated either in acute experimentally-induced pain or in cross-sectional studies of chronic pain. As such, these studies do not address pain mechanisms during the transition from the acute clinical setting to chronic pain disability or to full recovery.

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 on these associations after the trauma.

METHODS

Participants

The sample of participants included in this study has already been described in detail elsewhere.[6] Briefly, the sample consisted of 92 patients admitted to the emergency room of a general hospital after a motor vehicle accident (MVA), between July 1999 and December 2001. Participants were considered eligible if they were aged between 18 and 70 years and reported of pain in the 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 (eg, a wall or traffic light), was identified. Participants with signs of a concussion, retrograde or posttraumatic amnesia, serious injuries such as fractures, traumatic internal organic pathology, or any neurological signs were excluded. Thus, the participants included met the Quebec criteria for WAD grade 1 or 2. Finally, all the participants included had to be able to speak and read the Dutch language.

Study Design

The study was conducted as a prospective, longitudinal design. Participants were assessed 5 times, with baseline assessment performed within 1 week and follow-up assessments 4, 8, 12, and 24 weeks after the MVA. At each follow-up visit, the muscle activity was assessed by electromyographic (EMG) recording of the upper trapezius muscles. Before each EMG assessment, the subjects completed the Neck Disability Index (NDI) and the Tampa Scale of Kinesophobia (TSK) questionnaire and rated the level of pain on a visual analog scale rating of pain intensity (painVAS). During the follow-up period, only those patients who were still suffering from neck pain disability (NDI > 5) were still monitored. The moment these patients recovered (NDI $\leq$ 5), they were dropped from the study.

The functional status was scored by the NDI, a 10-item self-reporting instrument for the assessment of physical disability in participants with neck pain, particularly from whiplash-type injuries.[15] The NDI has been shown to have a high degree of test–retest reliability and internal consistency and an acceptable level of validity, being sensitive to severity levels and to changes in severity over time.[15,16] Vernon et al interpreted scores from 0 to 4 as no disability; scores from 5 to 50 represent increasing levels of disability. Before the study began, approval from the medical ethical committee was attained and all the participants were asked to complete an informed consent form.

EMG Recordings and Analyses

Experimental Protocol

The sEMG of the upper trapezius muscle was recorded bipolarly and amplified using a differential amplifier and band-pass filtered (3 to 10,000 Hz) to remove movement artifacts and prevent aliasing. The raw EMG was processed to a smooth rectified EMG (SRE) by applying a double-sided rectifier and stored digitally (12 bits, 1024 Hz). After the participants' skin was shaved and abraded with sandpaper, it was cleansed with 70% alcohol. The participants were seated in an upright position to permit palpation of the anatomical landmarks (C7, acromion). We used a KL-100 EMG monitoring system (K-Lab, Haarlem, The Netherlands).

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.[17,18] The electrodes (pregelled Ag/AgCl, type Meditrace, manufactured by Graphic Control Corporation, Buffalo, NY, USA) were placed 2 cm lateral to the midpoint of the lead line between the acromion and the easily palpable spinous process of 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. The electrodes and the cables were fixed to the skin with tape and connected to a portable data acquisition unit.

EMG Analysis

Participants were seated on a desk chair with their backs supported and their hips and knees in 90-degree flexion. Their arms were held straight and horizontal in 90-degree abduction in the frontal plane of the body, with the hands relaxed and the palms pointing downwards. To control the head positioning, the participants were instructed to keep their head upright and not to move, by fixing their eyes on an object on the wall. Four epochs of upper trapezius SRE were obtained. Each epoch lasted 15 seconds, separated by a period of 1 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 was computed as the mean muscle activity of the dominant arm during the performance of physical task.

Pain Intensity

PainVAS was performed using 2 vertical marks placed 100 mm apart, with the words "No pain" marked in the left and "Worst pain ever experienced" marked in the right. The participants were asked to rate the averaged pain intensity they experienced during the preceding week.

Fear of Movement/(re)injury

A Dutch version of the TSK is a 17-item questionnaire that is designed to assess fear of (re)injury due to movement, specifically in patients suffering from musculoskeletal pain. 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 scores 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 interquartile distance of 33 to 45.[19]

Statistical Analysis

Multilevel analysis (MLA) was used to analyze this longitudinal data set. Similar to repeated measures of analysis of variance, this method can deal with intercorrelated repeated measures. However, MLA has the advantage that it can deal with occasional missing data and censored data. In this study, data were censored because the moment patients recovered (ie when follow-up NDI score remained ≤ 5) their dataset was incomplete because the Dutch version of the TSK questionnaire was not applicable to pain-free participants.

The determinants included in the model were painVAS, TSK, body mass index (BMI), WEEK (number of weeks after accident), DISAB (recovered = 0 and chronic disabled = 1), and the interaction terms painVAS × TSK, painVAS × WEEK, and TSK × WEEK.

The variable BMI was included in this analysis to control for differences in the thickness of subcutaneous fat layer between patients. The interaction between painVAS and TSK was included in the analysis because in the fear avoidance model of Vlaeyen et al,[10] pain can influence the muscle activity directly but can also modify the effect of fear of movement on muscle activity. The variable WEEK and the interactions between WEEK and both painVAS and fear of movement were included to evaluate possible changes over time in the relationship between these factors and the level of muscle activation.

The variable GROUP was included because after recovery (NDI ≤ 5) the missing follow-up data were not at random but were attributable to recovery. To avoid biased EMG data toward patients with disability who contributed to all 5 assessments, the variable GROUP could evaluate whether the relations between EMG and the other variables were similar in participants who showed early recovery and in those who were disabled even at the 24-week follow-up. P values < 0.05 were considered statistically significant. Data were analyzed using S-Plus 2000 for Windows.

RESULTS

Of the 92 patients included in the sample, 43 recovered during follow-up 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 μV, SD = 50.5) that was rather constant during follow-up. The baseline pain intensity of this group (mean painVAS = 53.7 mm, SD = 21.4) was classified as almost severe[20] and gradually declined to a more moderate level by 24 weeks (mean painVAS = 38.8 mm, SD = 21.0). The level of baseline TSK (median 39.0, 10th to 90th percentile 30.8 to 47.0) corresponded to the median level of a normative group of a sample of 319 Dutch and Flemish patients with chronic musculoskeletal pain.[19] During follow-up, the TSK level remained relatively constant.

Of the 43 recovered participants, 5 had recovered before the first assessment, 28 had recovered during the following 12 weeks, and the remaining 10 participants had recovered between 12 and 24 weeks (Table 2). In the recovered group, the level of muscle activity was systematically higher than that in the chronic disabled group, whereas the levels of pain intensity and TSK were lower. Also, when patients dropped out, the remaining patients who were monitored until their recovery showed higher mean EMG levels and lower levels of pain intensity and TSK.

MLA including the main effects and interaction terms resulted in a model (Table 3) that could predict the level of muscle activation by the levels of both the fear of movement (t value = -2.19, $P = 0.030$) and pain intensity (t value = -2.94, $P = 0.004$) independently. Figures 1 and 2 show that the predicted EMG level is inversely related to both pain and kinesophobia, indicating that higher pain and kinesophobia levels are associated with a lower EMG level. In addition, the effect of fear of movement on muscle activity was modified by pain intensity such that in patients with a

TABLE 1. Demographic Variables of a Group of Recovered (Neck Disability Index <5) and Chronic Disabled (Neck Disability Index ≥ 5) Participants

	NDI < 5	NDI ≥ 5
Number of participants	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)

SD, standard deviation.

TABLE 2. Course of Isometric Muscle Activity, Pain Intensity (PainVAS), and Fear of Movement (TSK) in Recovered (Neck Disability Index <5) and Chronic Disabled (Neck Disability Index ≥ 5) Participants during 24-week Follow-up After a MVA*

	N	IMA	PainVAS	TSK	N‡	IMA	PainVAS	TSK‡
	Recovered at Follow-up				Disabled at Follow-up			
Weeks Since MVA		Mean (μV) (SD)	Mean (mm) (SD)	Median (10th to 90th Percentile)		Mean (μV) (SD)	Mean (mm) (SD)	Median (10th to 90th Percentile)
MVA	43				49			
1	38†	147.7 (77.8)	30.6 (20.3)	34.5 (25.9 to 45.1)	43‡	106.9 (50.5)	53.7 (21.4)	39.0 (30.6 to 47.0)
4	22	152.0 (91.3)	24.8 (19.2)	34.5 (25.3 to 47.4)	43‡	115.8 (55.2)	52.1 (18.6)	41.0 (31.6 to 49.0)
8	8‡	180.0 (67.0)	23.9 (16.8)	30.0 (25.0 to 50.0)	47‡	120.5 (70.5)	47.6 (17.9)	38.0 (29.4 to 48.6)
12	10	180.6 (76.5)	21.4 (11.8)	30.0 (21.5 to 42.7)	45‡	123.1 (64.5)	45.0 (20.3)	38.0 (27.5 to 49.0)
24	0	Recovered	Recovered	Recovered	47‡	121.7 (65.8)	38.8 (21.0)	37.5 (28.0 to 48.2)

In the group of recovered patients, the reported values are for those who have not recovered at that point of time.
N, number of participants; IMA, isometric muscle activity.
*The data in the recovered group are from censored cases because the TSK can be scored only until patients have fully recovered.
†Five of the 43 subjects had already recovered at first assessment and could not score the TSK.
‡Incomplete data set because of occasional missing data.

high level of pain intensity the level of fear of movement predicted higher EMG levels compared to those with low pain intensity (Fig. 3) (t value = 2.15, $P = 0.033$). Furthermore, the effect of a decrease in muscle activity level caused by an increased painVAS level diminished as time passed after the trauma (t value = 2.58, $P = 0.011$). There was no effect of grouping variables (t value = 0.89, $P = 0.376$), so biasing of data toward the disabled group is unlikely.

As expected, a large part of the variance was explained by BMI (t value = -5.42, $P < 0.0001$) because in participants with higher percentage of fat the EMG signal was decreased. The importance of this variable in using sEMG is illustrated in Figures 1–3. The plotted models show 9 EMG values < 50 mV. These values come from the 2 patients with the 2 most extreme BMI values of 40.0 and 43.3. The fact that these subjects have nonrealistic predicted EMG values indicate a nonlinear relationship between BMI and EMG levels.

As an example, this model can predict that the EMG level is approximately 153 mV in the first week after the MVA of a participant with a BMI of 22, a painVAS of 25, and a TSK of 32, who will not recover within the 24 weeks follow-up. The model can predict that another person, who has a higher painVAS of 64 and a higher TSK of 42, will show an EMG level of 131 mV.

TABLE 3. Multilevel Analysis of Isometric Muscle Activity as a Dependent Variable. The Determinants Included in the Model Were PainVAS, TSK, BMI, WEEK and the Interaction Terms PainVAS × TSK, PainVAS × WEEK, and TSK × WEEK

	β	SE	t Value	P
PainVAS	–1.32	0.45	–2.94	0.004
TSK	–1.41	0.64	–2.19	0.030
BMI	–6.93	1.28	–5.42	< 0.0001
WEEK	–1.42	0.91	–1.57	0.118
DISAB	–10.47	11.77	–0.89	0.376
TSK × painVAS	0.024	0.01	2.15	0.033
WEEK × TSK	0.015	0.02	0.59	0.553
WEEK × painVAS	0.024	0.01	2.58	0.011

SE, standard error of the β coefficient.

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 during the transition of acute to chronic posttraumatic neck pain. 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 during the task is diminished. The results also indicate that higher levels of pain intensity result in a stronger effect of fear of movement on decreased muscle

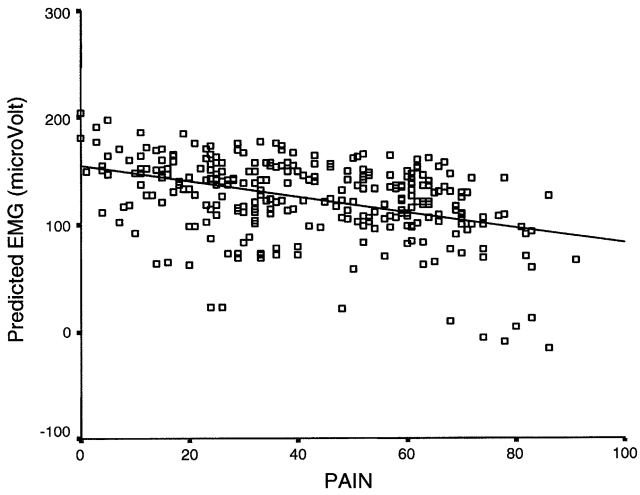

FIGURE 1. The level of EMG during a submaximal isometric exercise, predicted by the independent effect of pain intensity (painVAS).

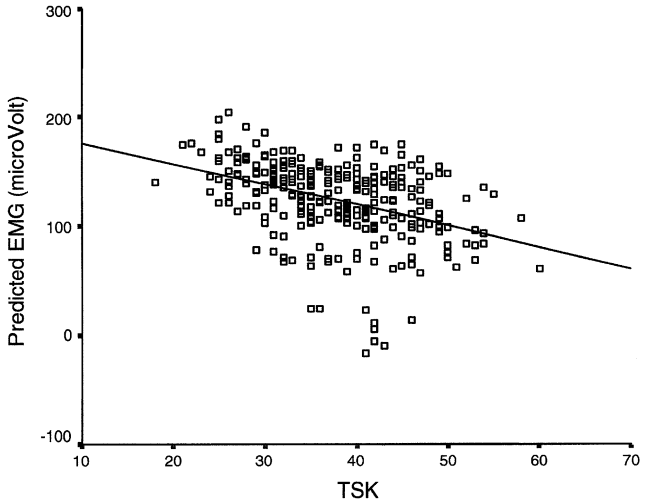

FIGURE 2. The level of EMG during a submaximal isometric exercise, predicted by the independent effect of fear of movement (TSK).

activity level. These results are in agreement with both the pain adaptation model[7,8] and the fear avoidance model.[10] According to both these models, the decrease in muscle activation level can be explained by "avoiding" the use of painful muscles to prevent the amplification of pain and further injury. In addition, because the effect of pain intensity on muscle activation level decreases during follow-up, there is an indication that the contribution of both mechanisms in this avoiding muscle behavior change during the transition from acute to chronic pain.

Crombez et al,[13,14] 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 in a knee extension–flexion task,[13] a trunk extension–flexion task, and a weight lifting task.[14] During these tests, the participants 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.

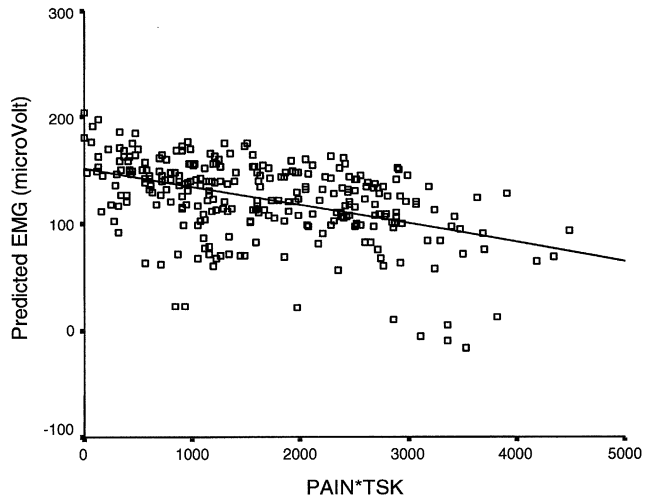

FIGURE 3. The level of EMG during a submaximal isometric exercise, predicted by the interaction between pain intensity and fear of movement (painVAS × TSK).

Our study differs from that of Crombez et al in several ways. Using a submaximal well-defined task[21] will diminish the interindividual differences related to anatomical variability and motivational aspects related to the readiness to perform maximally. Therefore, this method is probably 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 demonstrated that pain-related fear predicted physical performance better than pain. It is likely that this is caused by differences in participant selection. The current study included patients with acute pain. Assuming that in these patients the healing of soft-tissue injury takes 6 to 8 weeks, during this period, nociceptive stimuli can have a direct effect on muscle activity. The pain adaptation model[7,8] 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 fact that the influence of pain on muscle activity decreases over time but the effects of TSK on EMG do not change over time is consistent with this interpretation.

There are several questions concerning the role of decreased muscle activity in the development of chronic pain that remain unanswered. In the pain adaptation model, the inhibition of muscle activation is considered an adaptive reaction to avoid painful movement and (re)injury. As such, the decrease in the upper trapezius muscle activity is merely a normal protective adaptation in response to pain. Because the model does not involve any kind of a vicious circle, the role of 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.[11,22] However, research is needed to explain how the observed decrease in muscle activity in the current study can result in "deconditioning" effects and disability.

It is intriguing to observe that the baseline values of TSK and pain intensity of the recovered group are low compared to those of the disabled group. This suggests that any influence of pain and kinesophobia on the outcome either is premorbid or develops within 1 week of injury. According to the pain adaptation model, this may be the extent of injury, resulting in a different level of nociception.[7,8] Alternatively, according to the fear avoidance model, this difference could be explained by differences in the preinjury disposition toward pain-related fear of movement. As such, further research toward the role of "pain catastrophizing"—an exaggerated negative orientation toward noxious stimuli, which is considered to be a precursor to fear of movement[10,23]—is

needed. Moreover, the interaction between acute pain and TSK levels within the first week of injury may result in higher levels of fear of movement, specifically in those patients with high pain levels, ultimately leading to more negative outcomes.

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 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 this particular movement on the muscle activation level.[24] 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. The group that recovers is expected to show dynamic changes in determinants and outcome, thereby providing an opportunity to demonstrate more clear relations between these variables. A modified version of the TSK that is applicable to persons without musculoskeletal pain became available recently, unfortunately after the initiation of the study.

The clinical implication of this study is that the Quebec Task Force injury severity classification system[1] needs to be adjusted because the results of this study suggest that WAD grade 2 is not characterized by muscle spasm but rather by muscle *recoordination*. Moreover, because fear of movement independently explained part of the muscle inhibition, in addition to pain, it is questionable whether these 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 to avoid painful movements and in an effort to prevent further injury. Psychosocial factors, in particular, fear avoidance beliefs, seem to be more important than the biomedical factors for the development of chronic pain.[25,26] This is supported by the additional analysis of the data in the current study, indicating the predictive value of fear avoidance beliefs in patients with acute WAD for the development of chronic disability.[27] Such data suggest that the 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 the prevention of chronic disability.[28] Furthermore, prevention of disability might be fostered if psychosocial factors are routinely targeted for treatment in the earliest stages of pain.[29–31]

In conclusion, this study indicates that, in addition to injury-related pain, pain-related fear affects physical performance by altering motor control during the performance of a physical activity that is perceived as a potential threat to physical integrity.

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