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Cervical Muscle Dysfunction in Chronic Whiplash-Associated Disorder Grade 2

The Relevance of the Trauma

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Study Design. Surface electromyography measurements of the upper trapezius muscles were performed in patients with a chronic whiplash-associated disorder Grade 2 and those with nonspecific neck pain.

Objective. 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 whiplash-associated disorder 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 hypothesized 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 2 were compared with those of patients with nonspecific neck pain. 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 2 and those with nonspecific neck pain. There was only a tendency of higher muscle reactivity in patients with whiplash-associated disorder Grade 2.

Conclusions. It appears that the cervical muscle dysfunction in patients with chronic whiplash-associated disorder Grade 2 is not related to the specific trauma mechanism. Rather, cervical muscle dysfunction appears to be a general sign in diverse chronic neck pain syndromes.
[Key words: biopsychosocial, muscle dysfunction, nonspecific neck pain, upper trapezius muscles, whiplash-associated disorder] **Spine 2002;27:1056–1061**

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 Quebec Task Force[14] 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 also has been challenged.[6] The breadth of the definition has a 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 for 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 etiologic 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 physiologic 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 the current study, the "musculoskeletal signs" characteristic of patients with WAD Grade 2 (WAD 2) were assessed by surface electromyography and compared with a matched set of healthy control (HC) subjects. The mus-

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cle 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 2 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 2 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 2, the muscle activation patterns of patients with WAD 2 were compared with those of patients with chronic nonspecific neck pain (NSNP) but no traumatic onset of symptoms. The authors hypothesized that in patients with neck pain, there are no differences in muscle activation patterns 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 2, 18 patients with chronic neck pain unrelated to any traumatic event (NSNP), and 18 HC subjects without any history of pain in the cervical region or chronic headaches.

Patients With Whiplash-Associated Disorder Grade 2. The patients with WAD 2 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 months.

Whiplash-associated disorder Grade 2 is characterized 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 2 from the other WAD grades[14] can be obtained objectively by surface electromyelography,[13] the choice was made to include a homogeneous group of patients with WAD 2.

All the patients were diagnosed clinically and referred by rehabilitation physicians or physiatrists. The patients who reported pain in the neck, with possible coexisting 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 preexisting or trauma-related orthopedic or neurologic signs.

Patients With Chronic Nonspecific Neck Pain. The patients with chronic NSNP were recruited from a neurologic 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 2, 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 ± 12.4 years. The HC subjects had no prior experience with the rehabilitation center. They did not have any pain in the neck, head, or shoulder regions and had not experienced a whiplash injury or motor vehicle accident in the past.

The demographic variables appeared to be somewhat different among the three groups. The NSNP group reported a significantly longer duration reported 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. Electromyelographic activity of the upper trapezius muscle was recorded bipolarly, amplified using a differential amplifier, and band-pass filtered (8–500 Hz) to remove movement artifacts and prevent aliasing. The raw electromyelography was processed to a smooth rectified electromyelography (SRE) applying a double-sided rectifier and stored digitally (12 bits; 1024 Hz).

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 muscle activation level without encountering large irregularities in the surface electromyography signal.[13] A period of 1 minute between the consecutive epochs was chosen.

Electrode Placements. To ensure proper sensor placement procedures, the recommendations of the EC-concerted-action Surface Electromyography for Noninvasive Assessment of Muscles project were followed.[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 anatomic landmarks (C7 acromion). The electrodes (pregelled Ag/AgCl; type, Meditrace) 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 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.

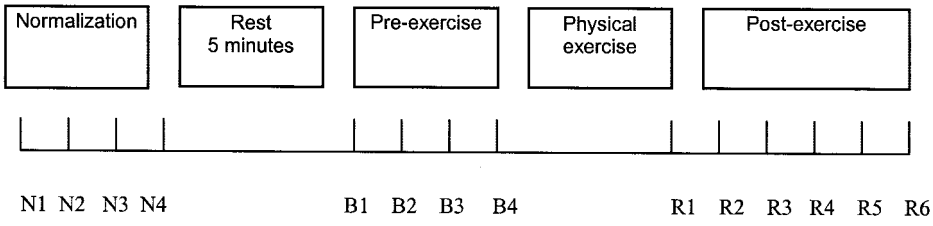

Figure 1. Study design. Normalization procedure (N1–N4) comprising four reference contractions. Preexercise condition (B1–B4) comprising four assessments with the subject sitting in a desk chair. Postexercise condition (R1–R4) comprising six assessments with the subject sitting in a desk chair. Between the consecutive assessments, there was a 1-minute rest.

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

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.

Preexercise Baseline Condition. After 5 minutes of rest, the subject assumed a sitting position in a desk chair for the measurement of baseline muscle activity. Four epochs of upper trapezius SRE (B1–B4) were obtained with the back supported, the hips and knees at 90°, 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]

Postexercise 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 six SRE values obtained after the exercise.**

These outcome parameters were used to compare patients who had WAD 2 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 2 and the HC subjects also were 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 2 and the other two groups were analyzed with Mann–Whitney *U* tests. Nonparametric tests were used because the distribution of the individual SRE levels was skewed.

The time-related recovery pattern of each subject's postexercise 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 of the equation for individual regressions was averaged to one value. Differences between the slopes of the averaged regression plots for the patients with WAD 2 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 2 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.

Differences Between Chronic Whiplash-Associated Disorder Grade 2 and Chronic Nonspecific Neck Pain

No differences were found between the patients with WAD 2 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

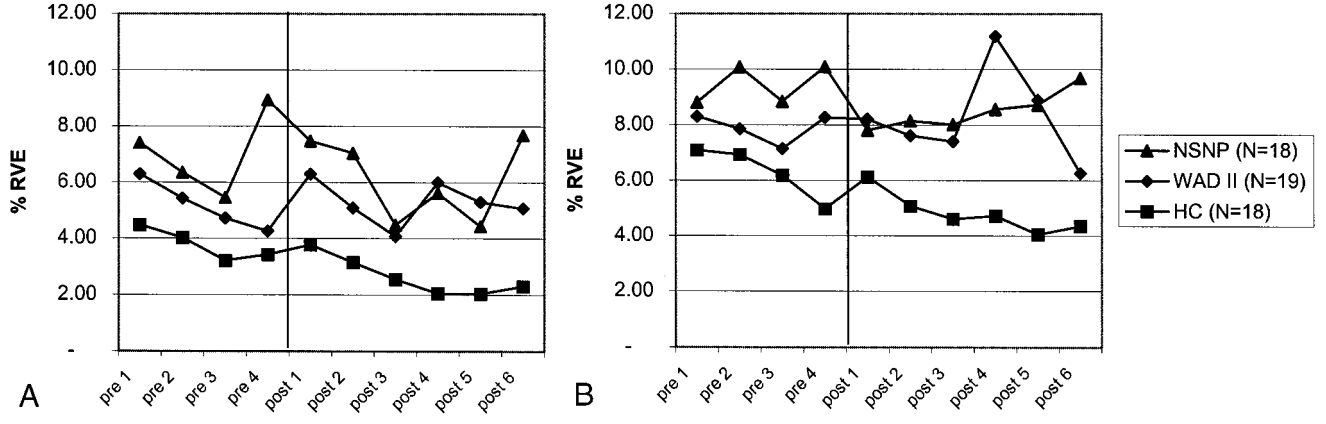

Figure 2. **A,** Course of pre- and postexercise muscle activation of the arm, involving a unilateral dynamic manual exercise (active side), for patients with whiplash-associated disorder Grade 2 (WAD 2 [◇], n = 19), patients with nonspecific neck pain (NSNP [▲], n = 18), and healthy control (■, n = 18) subjects. The four preexercise and six postexercise smooth rectified electromyography (SRE) 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 2, patients with NSNP patients, and healthy control subjects. The four preexercise and six postexercise SRE levels are expressed as %RVE.

after the exercise, whereas the patients with WAD 2 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.

In both neck pain groups, there appeared to be no consistent pattern of muscle activity level during the postexercise 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 0.001 (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 is not impressive, and that there also are individuals who do not show this decline. Assessment 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 2. However, the intersubject variability in both groups was high. Consequently, no statistically significant differences between the two groups were found.

Differences Between Patients With Chronic Whiplash-Associated Disorder Grade 2 and Healthy Control Subjects

There was a difference in muscle reactivity between the patients with WAD 2 and the HC subjects (Table 1). The patients with WAD 2 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 statisti-

Table 1. Differences in Levels of Muscle Reactivity and Pre- and Postexercise Muscle Activity in Two Groups of Neck Pain Patients and a Group of Healthy Control Subjects

Measurement Condition	Measured Side	WAD 2 (n = 19)		NSNP (n = 18)		HC (n = 18)		Mann–Whitney *U* Test	
		%RVE	SD	%RVE	SD	%RVE	SD	WAD 2–NSNP	WAD 2–HC
Muscle-reactivity	Active	0.1	3.9	−0.9	8.5	−1.1	3.9	0.06	0.01
	Passive	0.4	2.5	−1.0	4.8	−1.5	4.5	0.98	0.21
Preexercise muscle activity	Active	5.2	7.8	7.0	9.1	3.8	4.0	0.74	0.85
	Passive	7.9	12.1	9.5	10.0	6.3	6.3	0.49	0.47
Postexercise muscle activity	Active	5.3	5.0	6.1	7.1	2.6	1.1	0.86	0.06
	Passive	8.3	10.8	8.5	9.6	4.8	4.7	0.86	0.09

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 postexercise from the mean preexercise level of muscle activity. WAD 2 = whiplash associated disorder; NSNP = nonspecific neck pain; HC = healthy control subjects; SD = standard deviation.

Table 2. Time-Related Recovery Pattern of Postexercise Muscle Activity

	WAD-II		NSNP		HC		Difference in β-Coefficient
Arm Side	r^2	β-coefficient	r^2	β-coefficient	r^2	β-coefficient	ANOVA
Active	0.28 (0.24)*	−0.10 (1.35)	0.29 (0.24)*	−0.16 (3.0)	0.38 (0.29)*	−0.32 (0.44)	0.941
Passive	0.26 (0.28)*	−0.06 (0.50)	0.42 (0.26)*	0.33 (1.9)	0.33 (0.24)*	−0.34 (0.83)	0.252

r^2 = proportion of variance explained by time; β-Coefficient = slopes of the linear refression model; ANOVA = analysis of variance; WAD 2 = whiplash associated disorder, NSNP = nonspecific neck pain, HC = healthy control subjects.
* Statistically significant difference from zero ($p \leq 0.001$, one-sample Student t test.
Values are reported as mean (standard deviation).

cally significant on the active side. The decline in muscle activity among the HC subjects rather than the increase among the patients with WAD 2 accounts for most of the observed differences.

Muscle relaxation after the exercise tended to progress during the six postexercise measurements among HC subjects (Figure 2A and 2B). The larger proportion variance (R^2), explained by time and the higher, 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 Figure 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 2 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 2 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 for 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 etiologic significance of acceleration–deceleration trauma for the presence of cervical muscle dysfunction in the chronic stage of the WAD 2. 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 2. As such, it does not contribute to the descriptive validity of WAD.

In this study, the abnormalities in muscle responses 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 difference 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 current study are consistent with those of earlier studies,[13] in which patients with WAD 2 also showed an increase in muscle activity in response to a physical exercise. In contrast, this increase was almost twofold 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 2. 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 re-

sponse to the exercise, and the other subgroup 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.[7,10] The clear tendency of patients with WAD 2 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 2 is not related to the specific trauma mechanism.
- Cervical muscle dysfunction appears to be a general sign in diverse chronic neck pain syndromes.

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