Cervical Muscle Dysfunction in Chronic Whiplash-Associated Disorder Grade 2
The Relevance of the Trauma

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FROM ABSTRACT

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.

THESE AUTHORS ALSO NOTE:

The primary symptoms of whiplash-associated disorder (WAD) include neck pain and headache.

The Quebec Task Force 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

The definition for “chronic phase whiplash” used by these authors is “symptoms persisting more than 6 months after the trauma and after healing of soft tissue injury."

These authors note that in the biopsychosocial model of chronic pain, that pain can become a persistent problem independent of the precise physiologic etiology and extent of impairment.

The biopsychosocial model proposes that three response systems contribute to the experience of pain and chronic symptoms
(1) behavioral
(2) cognitive
(3) psychophysiologic

They propose that in whiplash, the trauma triggers the three response systems noted above comprising the biopsychosocial model.
In this study, the “musculoskeletal signs” in these patients with WAD Grade 2 were assessed by surface electromyography and compared with a matched set of healthy control (HC) subjects.
From my perspective, this approach is problematic for three reasons:
(1) Studies continue to affirm the primary injury in whiplash is articular (facet and disc), and not muscular.
(2) Studies also continue to affirm that chronic spine pain is articular, and not muscular.
(3) To assess “musculoskeletal signs” by examining only a superficial muscle (trapezius) and not deeper muscles or articulations appears shortsighted.

“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 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.”

I believe that there is a higher probability that this hypothesis is more wrong than correct.

I believe that there is a higher probability that the “cervical muscle dysfunction” is secondary to reflexes emanating from damaged articular structures.

If, as hypothesized by the authors, “pain” provoked the “cervical muscle dysfunction,” pain is not one of the three responses in their biopsychosocial model, other studies have shown that chronic organic pain does cause abnormal psychological profile.

This study compared the muscle activation patterns of three groups:
(1) Chronic patients with WAD grade 2 (n = 19).
(2) Patients with chronic nonspecific neck pain (NSNP) and no traumatic onset of symptoms, but a gradual and progressive evolution of symptoms over time (n = 18).
(3) Healthy controls (HC) without any history of neck pain or headaches (n = 18).

Whiplash-associated disorder Grade 2 patients had neck pain and musculoskeletal signs including decreased range of motion and point tenderness.

The authors note that surface electromyelography can discriminate WAD 2 from the other WAD grades. [IMPORTANT].

[However, they support this statement using one reference, an article the same authors wrote in 2000: Nederhand MJ, Ijzerman MJ, Hermens HJ, et al. Cervical}

The surface electromyelographic activity of the upper trapezius muscle was recorded bipolarily, amplified using a differential amplifier, and band-pass filtered (8–500 Hz) to remove movement artifacts and prevent aliasing.

“To ensure proper sensor placement procedures, the recommendations of the EC-concerted-action Surface Electromyography for Noninvasive Assessment of Muscles project were followed. 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.”

[These references were cited for the above protocols:

This experiment had these surface EMG stages on the trapezius:

(1) A preexercise baseline surface EMG
(2) A unilateral surface EMG while doing physical exercise of the upper extremity
(3) A postexercise surface EMG

Muscle reactivity and time-related recovery patterns were evaluated and compared between the two groups.

RESULTS

“No differences were found between the patients with WAD 2 and patients with chronic NSNP in terms of muscle reactivity.”

“However, the patients in the latter group [chronic NSNP] showed a tendency to relax cervical muscles 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).” [IMPORTANT]
"In this respect, the patients with NSNP resemble the HC [healthy controls] subjects, who also showed this tendency to relax after the exercise." [IMPORTANT]

In response to exercise, the patients with WAD 2 demonstrated greater reactivity, whereas the HC subjects showed a decline in muscle activity, and this “difference was statistically significant.” [IMPORTANT]

“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.”

“The postexercise muscle activity level in the patients with WAD 2 was almost twice as high as that in the HC subjects.” [IMPORTANT]

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 psychophysiological arousal and pain.”

“Cervical muscle dysfunction appears to be a general sign of chronic neck pain that is not specific for WAD 2.”

“In this study, the abnormalities in muscle responses can be explained by the cognitive–behavioral model of ‘fear of movement/(re)injury’. 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.”

“The contribution of fear avoidance and muscle reactivity to the presence of pain has never been studied.”

“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.”

KEY POINTS FROM THE AUTHORS:

(1) It appears that the cervical muscle dysfunction in patients with chronic whiplash-associated disorder Grade 2 is not related to the specific trauma mechanism.
(2) Cervical muscle dysfunction appears to be a general sign in diverse chronic neck pain syndromes.

KEY POINTS AND COMMENTS FROM DAN MURPHY

(1) This is yet another study that supports surface EMG as a valid research tool.

(2) This article notes that surface electromyelography can discriminate WAD 2 from the other WAD grades.

(3) Numerous studies have shown that the primary injury in whiplash is to the facet capsule and to the annulus of the disc (rim lesion).

(4) Numerous studies have shown that the primary tissue source initiating chronic neck pain is the facet capsule and the annulus of the disc, not the muscles.

(5) Animal studies have shown that irritations to the facet capsules and disc cause a reflex contraction of the small, segmental multifidus muscle, not the large mover trapezius.

(6) The authors note that the primary symptoms of WAD are neck pain and headache. Studies have shown that post-whiplash headache are also articular (C2-C3 facet), and not muscular.

(7) To assess “musculoskeletal signs” by examining only a superficial muscle (trapezius) and not deeper muscles or articulations appears shortsighted and problematic.

(8) To note that in “the biopsychosocial model of chronic pain, that pain can become a persistent problem independent of the precise physiologic etiology” ignores the physiologic etiologies of receptive field enlargement, synaptogenesis / neuroplasticity, wind-up, and altered threshold, etc.

(9) To hypothesize that the trapezius muscle response and chronic symptoms are the consequence of “psychophysiologic arousal and pain” is problematic because:

(A) Studies have shown that chronic whiplash pain will cause an abnormal psychological profile, and that this abnormal psychological profile can only be resolved by successful treatment of the chronic somatic pain.

(10) This study only assessed Quebec Grade 2 patients (neck problems and musculoskeletal signs), but not Grade 3 patients (neck problems and neurologic signs).