Muscle pain induces task-dependent changes in cervical agonist/antagonist activity

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Falla D, Farina D, Kamstrup Dahl M, Graven-Nielsen T. Muscle pain induces task-dependent changes in cervical agonist/antagonist activity. J Appl Physiol 102: 601–609, 2007. First published October 12, 2006; doi:10.1152/japplphysiol.00602.2006.—This study examined the effect of experimental neck muscle pain on the EMG-force relationship of cervical agonist and antagonist muscles. Surface EMG signals were detected from the sternomastoid, splenius capitis, and upper trapezius muscles bilaterally from 14 healthy subjects during cervical flexion and extension contractions of linearly increasing force from 0 to 60% of the maximum voluntary contraction (MVC). Measurements were performed before and after injection of 0.5 ml hypertonic and isotonic saline into either the sternomastoid or splenius capitis in two experimental sessions. EMG average rectified value (ARV) of the sternomastoid, splenius capitis, and upper trapezius muscles and the muscle fiber conduction velocity (CV) of the sternomastoid muscle were estimated at 5% MVC force increments. During cervical flexion with injection of hypertonic saline in sternomastoid, ARV of sternomastoid was lower on the side of pain in the force range 25–60% MVC (P < 0.05) and was associated with a bilateral reduction of splenius capitis and upper trapezius ARV (P < 0.01). During cervical extension, injection of hypertonic saline in splenius capitis resulted in lower estimates of splenius capitis ARV on the painful side from 45 to 60% MVC (P < 0.05), which was associated with a bilateral increase in upper trapezius ARV estimates from 50 to 60% MVC (P < 0.001). However, no significant change was identified for estimates of sternomastoid ARV. Experimentally induced neck muscle pain resulted in task-dependent changes in cervical agonist/antagonist activity without modifications in muscle fiber CV.

Because decreased muscle activity has been observed with unchanged force (14, 21), compensatory mechanisms should occur to allow similar motor output in painful and non-painful conditions. A possible adaptation is an acute change in muscle fiber contractile properties induced by modification of the extracellular environment with injection of painful substances. However, injection of hypertonic saline is not associated with a change in muscle fiber electrophysiological membrane properties (15, 21). Thus adaptation of the coordination at the level of the muscle group acting on the joint seems a more likely compensatory mechanism.

Results of the effect of local muscle pain on muscle group coordination are less consistent. In isometric dorsiflexion and plantarflexion contractions sustained until exhaustion, EMG amplitude of both the painful and nonpainful synergistic muscles decreased with pain (9). Moreover, the relative activity of painful and nonpainful muscles changed with fatigue, which was considered a compensatory mechanism to maintain force (9). However, group III and IV afferent activity is also influenced by accumulation of metabolic products with fatigue (5, 19); therefore, it is not possible to separate the effects due to pain and fatigue on muscle coordination during sustained contractions. The effect of muscle pain on nonpainful synergists and antagonists has also been investigated in dynamic contractions (2, 21, 40); however, these results cannot be directly extrapolated to isometric conditions.

The effect of local excitation of nociceptive afferents on muscle coordination is particularly relevant in joints with multiple degrees of freedom. The cervical spine is a complex biomechanical system composed of numerous degrees of freedom of movement about each of its joints and at least 20 pairs of muscles, many of which are capable of performing similar functions (26). This system is highly redundant, and specific forces may be produced by several combinations of muscle actions (31). Decreased muscle activity due to pain may be compensated, for example, by decreased antagonist or increased synergic contribution. Given the complexity of the cervical spine musculature, the effect of pain on muscle coordination may depend on the specific direction of movement and intensity of contraction. If force maintenance is mainly due to reorganization among painful and nonpainful muscles, it is expected that there would be a differential effect on the antagonist muscles depending on the force direction and thus the ability to recruit synergic muscles. This hypothesis has never been tested. Therefore, the aim of the study was to examine the effect of experimental neck pain on the EMG-force relationship in agonist and antagonist muscles during cervical flexion and extension.

METHODS

Subjects. Fourteen volunteers (6 women) participated in the study after providing informed consent. Subjects [age, 26.3 yr (SD 3.6); height, 1.73 m (SD 0.11); weight, 71.9 kg (SD 14.6)] were free of neck pain and headache at the time of testing. In addition, subjects were excluded if they had a history of headache or orthopedic disorders affecting the cervical spine or a history of neurological disorders. The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.
disorders. Ethical approval for the study was granted by the Ethics Committee (VN 2005/38), and all procedures were conducted according to the Declaration of Helsinki.

**Procedures.** Subjects were comfortably seated in a height-adjustable chair of a cervical force-measuring device (4) with their back supported, their knees and hips in 90° of flexion, and their head positioned in a padded head support. The adjustable head support was fastened across the forehead, which stabilized the head and provided resistance during cervical isometric contractions. The electrical signals from the load cells incorporated into the device were amplified (strain gauge amplifier, Aalborg University, Aalborg, Denmark), and their output was displayed on an oscilloscope as visual feedback to the subject.

Subjects performed three maximum voluntary isometric contractions (MVCs) of 3- to 4-s duration in cervical flexion and extension. The order of the MVCs was randomized between movement directions, and an interval of 5 min was provided between each set of three contractions. Verbal encouragement was provided to induce the subject to reach a higher level in each trial. The highest value of force recorded over the three MVCs for each direction was selected as the reference MVC and used to calculate the submaximal targets.

A rest of 30 min followed the maximal contractions. The subject then performed cervical flexion and extension ramped contractions, linearly increasing the force from 0 to 60% MVC in 2 s. Visual feedback on the oscilloscope was provided to the subject. A rest of 1 min was provided between the two movement directions. Following a rest of 10 min, the subject repeated the ramped contractions after the injection of isotonic saline into either the sternomastoid or splenius capitis muscle. Following a further 10-min rest, the subject performed the final set of ramped contractions following the injection of hypertonic saline into the same muscle as injected with isotonic saline, on the opposite side. The order of flexion and extension was randomized throughout the experimental session. Injections for sternomastoid and splenius capitis muscles were conducted in two experimental sessions separated by a minimum of 7 days. The entire experimental procedure was repeated on the second day.

**Experimental muscle pain.** Experimental muscle pain was induced by injection (27-gauge cannula) of 0.5 ml of sterile hypertonic saline (5.8%) into the sternal head of the sternocleidomastoid and the splenius capitis muscle. Isotonic saline (0.5 ml, 0.9%) was used as a control injection. For all injections, subjects were positioned in comfortable sitting. For the injection into the splenius capitis, the needle was inserted in a caudal direction at the level of the third cervical spinous process approximately midway between the posterior boarder of the sternocleidomastoid and the lateral boarder of the trapezius muscle (35). For the sternomastoid, the subject’s head was positioned in ipsilateral lateral flexion with slight contralateral rotation. The needle was inserted into the muscle belly 2 cm cephalad to the midpoint between the sternum and the mastoid process (35). The bolus was injected over a 10-s period. The location and side of injection were randomized for each experimental session. The participants were blinded to each injection and were told that one or both might be painful.

**Pain measurements.** Participants were asked to verbally rate their level of perceived pain on an 11-point numerical rating scale (NRS) anchored with “no pain” and the “worst possible pain imaginable.” Pain intensity ratings were obtained immediately following the injection and every 30 s until pain was no longer reported. Peak pain intensity, time to peak pain (time point at which the maximum pain is first reached), duration of pain, and area under the NRS-time graph were extracted.

Participants documented the area of pain on a body chart. Pain drawings were subsequently digitized (ACECAD D9000+, Taipei, Taiwan), and pain areas were estimated. Area of local and referred pain were calculated. Pain areas that were isolated from the area of local pain caused by injection were selected as referred pain areas. Quality of the pain was assessed by completion of the McGill Pain Questionnaire (28). Pain rating indexes based on rank values of the word descriptors were calculated (28). In addition, words chosen by more than 25% of subjects (n = 4) were noted.

**Linear array surface EMG.** Multichannel surface EMG signals were detected from the sternomastoid muscle bilaterally using linear adhesive arrays of 8 electrodes (bar electrodes, 5-mm × 1-mm size, 5 mm apart; LISSIN-SPES Medica, Torino, Italy). The detection surface was separated from the skin by a small cavity (~1 mm deep) filled with 20 µl of conductive gel. Myoelectric signals were amplified (AISE16, 16-channel amplifier, LISSIN Centro di Bioingegneria, Politecnico di Torino, Torino, Italy; gain: 5,000), filtered (~3–4dB bandwidth, 10–450 Hz), sampled at 2,048 Hz, and converted to 12-bit digital samples.

Before electrode placement, the sternomastoid muscles were assessed during preliminary test contractions with a dry array of eight electrodes (silver bars, 10-×1-mm size, 10 mm apart) with the subject positioned in sitting. The innervation zone location of the sternomastoid muscle was identified from the surface EMG recordings, as described previously (13, 27). The subject’s skin was prepared by gentle local abrasion using abrasive paste and cleaned with water and the array electrodes were positioned between the innervation zone and the caudal tendon region of the sternomastoid muscle. A reference electrode was placed over the upper thoracic spine.

**Bipolar surface EMG.** Bipolar surface EMG was recorded with pairs of electrodes positioned 20 mm apart (Neurileone 72001-k, Medicotest, Ølstykke, Denmark) over the splenius capitis and upper trapezius muscles bilaterally following skin preparation. For the splenius capitis, electrodes were positioned over the muscle belly at the C2–C3 level between the uppermost parts of trapezius and sternocleidomastoid. For the upper division of trapezius, the electrodes were positioned 20 mm lateral to the midpoint along the line between the acromion and the seventh cervical vertebra (23). Signals were bandpass filtered (10–500 Hz), amplified (EMG amplifier, Aalborg University, Aalborg, Denmark; gain: 5,000–10,000), and sampled at 2 kHz. Skin temperature (Ellab, Copenhagen, Denmark) over the neck region was monitored before and after completion of each of the set of contractions throughout the experimental session.

**Signal analysis.** The force signal was low-pass filtered (anticausal Butterworth filter of order 4, cutoff frequency 10 Hz) and normalized with respect to maximum force. Muscle fiber conduction velocity (CV) (17) and average rectified value (ARV) (16) were estimated from the EMG signals over 250-ms windows in which the average force was 5–60% MVC (5% MVC increments). Thus, in all conditions, the analyzed average force levels corresponded to the same percentage of the maximum force measured without pain. Conduction velocity was estimated from the array channels (double differential) that showed propagation of the action potentials with minor shape changes (visual inspection). The average cross-correlation over all signal pairs after alignment with the estimated delay was larger than 0.8 in all cases, indicating reliable estimates of CV (16). Estimates of ARV were computed from the central channels of those chosen for CV estimation. All comparisons between painful and nonpainful conditions were performed on estimates selected from the same set of channels.

**Statistical analysis.** The Wilcoxon test was used to identify differences between parameters of pain intensity, duration, area, and quality of sternomastoid and splenius capitis muscle pain. A three-way ANOVA was applied to the values of ARV for the sternomastoid and splenius capitis with condition (baseline, isotonic saline, and hypertonic saline), side (ipsilateral and contralateral to the injection), and force (5% MVC intervals; 5–60% MVC) as repeated measures. In addition, a three-way ANOVA was applied to the values of CV for sternomastoid with condition (baseline, isotonic saline, and hypertonic saline), side (ipsilateral and contralateral to the injection) and force (5% MVC intervals; 5–60% MVC) as repeated measures. Significant differences revealed by ANOVA were followed by post hoc Student-Newman-Keuls pairwise comparisons. Results are re-
ported as means (SD) in the text and means and SE in the figures. Statistical significance was set at $P < 0.05$.

RESULTS

Sensory characteristics of experimental neck muscle pain. Peak pain intensity was 5.5 (SD 1.9) and 4.9 (SD 1.7) following injection of hypertonic saline into the sternomastoid and splenius capitis muscles, respectively ($P < 0.05$; Fig. 1). No significant differences were identified between the injection sites for time to peak pain [sternomastoid, 30.0 s (SD 34.3); splenius capitis, 34.3 s (SD 28.5 s)], duration of pain [sternomastoid, 379.2 s (SD 28.5 s); splenius capitis: 413.5 s (SD 26.9 s)], and area under the NRS-time graph [sternomastoid, 1,252.5 (SD 563.6); splenius capitis: 1,276.1 (SD 591.3)].

Fig. 2. Area of pain following the injection of hypertonic saline into the sternomastoid and splenius capitis muscles. Sternomastoid muscle pain caused more frequent and larger area of referred pain compared with splenius capitis pain (7 vs. 4 subjects, respectively; $P < 0.05$).
Isotonic saline injection of the sternomastoid and splenius capitis produced lower scores on all measured pain parameters compared with hypertonic saline injection of the same sites \((P < 0.05; \text{Fig. } 1)\). Total mapped pain areas were similar between the two muscles [sternomastoid, 6.09 arbitrary units (SD 5.16); splenius capitis, 4.90 arbitrary units (SD 5.25); \text{Fig. } 2]. Sternomastoid muscle pain caused more frequent (number of participants, \(n = 7\)) and larger area of referred pain [3.16 arbitrary units (SD 5.14)] compared with splenius capitis [number of participants, \(n = 4\); area, 1.69 arbitrary units (SD 5.24); \(P < 0.05\); \text{Fig. } 2]. In addition, the area of referral was more commonly in the territory of trigeminal innervation for sternomastoid muscle pain (sternomastoid, \(n = 7\); splenius capitis, \(n = 2\)). For sternomastoid muscle pain, the most commonly chosen word from the McGill Pain Questionnaire was “pressing” (\(n = 5\)). The words “sharp,” “taut,” “tight,” and “annoying” were each selected by 29% of participants (\(n = 4\)). For splenius capitis muscle pain, the most commonly chosen word was “tiring” and “tight,” both 36%, and the second most common word was “taut,” at 29%. The overall pain rating index was 10.5 (SD 5.3) for sternomastoid and 9.8 (SD 8.6) for splenius capitis pain. For the individual pain categories, the rating index for sternomastoid and splenius capitis pain were 7.6 (SD 3.9) vs. 7.0 (SD 5.5) for the sensory, 0.4 (SD 0.7) vs. 0.6 (SD 0.7) for the affective, 1.0 (SD 1.4) vs. 1.1 (SD 1.5) for the evaluative, and 1.6 (SD 1.5) vs. 1.1 (SD 1.9) for the miscellaneous category. The overall number of words chosen was 4.9 (SD 2.6) for sternomastoid pain and 4.6 (SD 3.2) for splenius capitis pain.

**Muscle activity during flexion.** Representative force and EMG data from sternomastoid and splenius capitis are presented in \text{Fig. } 3. During the cervical flexion ramped contraction, significantly lower estimates of ARV were identified for the sternomastoid muscle on the side of pain from 25 to 60% MVC following injection of hypertonic saline into the sternomastoid muscle compared with baseline values \((F_{22} = 1.9, P < 0.05; \text{Fig. } 4A)\). In addition, at force levels of 50% and above, sternomastoid ARV was lower on the side contralateral to the injection \((F_{22} = 1.9, P < 0.05; \text{Fig. } 4B)\). In association with reduced sternomastoid ARV at higher force levels, lower estimates of ARV were identified for the splenius capitis \((F_{22} = 2.0, P < 0.01; \text{Fig. } 4C, D)\) and upper trapezius \((F_{22} = 2.1, P < 0.01; \text{Fig. } 4E, F)\) muscles bilaterally.

Following the injection of hypertonic saline into the splenius capitis muscle, lower estimates of ARV were identified for the splenius capitis muscle ipsilateral to the side of the injection across all force levels during ramped cervical flexion compared with baseline values \((F_{2} = 2.9, P < 0.05; \text{Fig. } 5C)\). Moreover, lower estimates of ARV were identified for the sternomastoid muscle bilaterally from 15 to 60% MVC \((F_{22} = 3.0, P < 0.001; \text{Fig. } 5A, B)\).

The average number of channels from which conduction velocity was estimated was 5.6 (SD 1.5). Sternomastoid muscle fiber conduction velocity increased with increasing cervical flexion force [across all conditions 5% MVC: 3.96 m/s (SD 1.6), 60% MVC: 5.3 m/s (SD 1.2); \(F_{11} > 6.1, P < 0.001\); however, no significant changes in CV were identified following injection of hypertonic saline in either muscle. Skin tem-

**Fig. 3.** Representative force and EMG data from 1 subject performing a linearly increasing cervical flexion force contraction from 0 to 60% of the maximum voluntary contraction (MVC). Data are presented before (baseline) and during (pain) hypertonic saline-induced sternomastoid muscle pain. \(A\): force. \(B\): sternomastoid EMG ipsilateral to the injection. \(C\): sternomastoid EMG contralateral to the injection. \(D\): splenius capitis EMG ipsilateral to the injection. \(E\): splenius capitis contralateral to the injection.
perature did not significantly change during both experimental sessions [average, 33.5°C (SD 0.9)].

Muscle activity during extension. During the cervical extension ramped contraction, significantly lower estimates of ARV were identified for the splenius capitis muscle on the side of injection from 40 to 60% MVC following injection of hypertonic saline into the sternomastoid muscle compared with baseline values ($F_{22}/H_{11005} = 1.7$, $P_{/H_{11021}} < 0.05$; Fig. 6C).

Following injection of hypertonic saline into the splenius capitis muscle, significantly lower estimates of ARV for the splenius capitis muscle on the painful side were identified during cervical extension from 45 to 60% MVC ($F_{22} = 1.6$, $P < 0.05$; Fig. 7C). Reduced splenius capitis ARV was associated with a bilateral increase in upper trapezius ARV estimates from 50 to 60% MVC ($F_{22} = 2.5$, $P < 0.001$; Fig. 7, E and F).

Cervical flexion and extension MVC force were not significantly different between days.

**DISCUSSION**

Experimentally induced neck muscle pain changed cervical agonist/antagonist activity without modification in muscle fiber membrane properties. EMG activity was consistently reduced in the painful agonist muscle, whereas modulation of antagonist activity depended on the task.

Subjective characteristics of experimental neck muscle pain. Injection of 0.5 ml of hypertonic saline in the sternomastoid and splenius capitis resulted in muscle pain of moderate intensity, consistent with previous reports of hypertonic saline-induced muscle pain (e.g., Refs. 22, 38). Pain evoked in the sternomastoid muscle was of significantly greater intensity than in the splenius capitis muscle. The difference may reflect the smaller average cross-sectional area of sternocleidomastoid (3.72 cm$^2$) relative to the splenius muscle (4.26 cm$^2$) (25). However, pain following injection of isotonic saline in sternomastoid and splenius capitis was
comparable, indicating that changes in intramuscular pressure due to the injection were not the main reason for the difference in pain sensitivity. Compared with splenius capitis, the sternomastoid muscle has lower pressure pain thresholds (33), which may reflect a greater density of afferent fibers.

Pain areas drawn by the subjects confirmed previous experimental studies demonstrating that splenius and sternomastoid muscle pain is frequently referred in the distribution of both cervical and trigeminal innervated areas and may mimic the clinical representation of headache (3, 33). Previous work has identified a correlation between pain intensity and occurrence of referred pain (20, 24, 39). Accordingly, a greater number of subjects (7 vs. 4) reported referred pain and a greater area of referred pain following noxious stimulation of the sternomastoid muscle compared with splenius capitis. In addition, the distribution of referred pain associated with sternomastoid muscle pain frequently included the oculo-fronto-temporal region, which is consistent with the distribution of the ophthalmic division of the trigeminal nerve. Experimental studies (1, 32) and clinical observations of headache associated with the cervical spine (10) suggest that predominately the ophthalmic division of the trigeminal nerve is involved in the mechanism of cervicogenic headache attributed to the convergence of cervical afferents and the trigeminal nerve (7). Therefore, experimental sternomastoid muscle pain may be superior to splenius capitis as an experimental model of cervicogenic headache.

**Effect of pain on agonist muscle activity.** Injection of hypertonic saline into either the sternomastoid or splenius capitis muscle resulted in decreased EMG amplitude when the muscle was acting as an agonist, in agreement with previous work (21, 36). Decreased muscle activation likely reflects decreased motoneuron discharge rate (15, 36) due to reflex inhibition mediated by small-diameter muscle afferents. Inhibition of
motoneuron discharge rates is also observed as a consequence of fatigue-induced excitation of group III and IV muscle afferents (5, 19).

The decrease in EMG activity following hypertonic saline injection depended on the exerted force, which may suggest different sensitivity of motoneurons to nociceptive input depending on their size. In agreement, a differential effect of fatigue on the discharge rate of low- and high-threshold motor units has been observed previously (8, 11).

Effect of muscle pain on synergist and antagonist muscle activity. The activity of muscles other than the painful muscle was also affected, indicating a redistribution of load in the muscle group contributing to the task. Change in muscle coordination with pain has been shown in previous work, although results are often contradictory. Injection of hypertonic saline in the gastrocnemius muscle resulted in decreased gastrocnemius EMG and increased tibialis anterior EMG when it acts as an antagonist during the gait cycle (21). However, other studies have identified no effect on the antagonist (6, 34) or even decreased antagonist muscle activity during muscle pain (12). Such opposing results may reflect different methodologies or variability in the change of motor strategy depending on the task performed, as hypothesized in this study.

For cervical flexion, decreased sternomastoid EMG activity following noxious stimulation of the sternomastoid muscle was associated with a bilateral reduction of splenius capitis and trapezius motor output that may explain the maintenance of constant force. This strategy seems necessary considering that sternocleidomastoid muscle is the main producer of torque in the cervical flexor muscle group. Similarly, reduced splenius capitis EMG activity during cervical flexion corresponded to a bilateral reduction of sternomastoid EMG activity.

The strategy to maintain cervical extension force in the presence of muscle pain appeared less dependent on the coordination between agonist and antagonist muscles. Pain in splenius capitis resulted in reduced EMG activity of splenius capitis and increased trapezius EMG activity during cervical extension.
capitis and a force-dependent increase in trapezius EMG activity (synergist) in the absence of changes in sternomastoid EMG activity. Thus, for cervical extension, the observed strategy for maintaining force output in the presence of an inhibited agonist appears to be recruitment of synergistic muscles and not modification of antagonistic muscle activity. A contribution of cross-talk cannot be entirely excluded; however, it is unlikely to have a major influence on the conclusions because the results have been obtained by comparison to control conditions.

Reorganization of synergistic and antagonistic muscle activity is a response to the inhibition of the painful muscle and may be mediated by reflex mechanisms or changes in the descending drive from higher centers to the motoneuron pool. In a previous study, percutaneous stimulation of the motor cortex resulted in a bilateral response for the sternocleidomastoid but only a unilateral response for the splenius capitis muscle (18). In agreement with this observation, during cervical flexion the agonist (sternomastoid) demonstrated bilateral inhibition to muscle pain, whereas during cervical extension, the agonist (spleunius capitis) demonstrated a unilateral inhibition.

In conclusion, this study showed that local excitation of nociceptive afferents has an inhibitory effect on the painful muscle that is counteracted by a complex reorganization of the motor strategy at the level of the muscle group involved in the task. This change in muscle coordination depends on the task so that the motor output is maintained unchanged in the painful condition (22, 38).

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