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Reduced force steadiness in women with neck pain and the effect of short term vibration

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ABSTRACT

This study compares neck force steadiness in women with neck pain and controls and the way this is influenced by short term vibration of the neck. In the first experiment, 9 women with chronic neck pain and 9 controls performed 10-s isometric cervical flexion at 15 N. Intramuscular EMG was recorded from the sternocleidomastoid muscle. In the second experiment, 10 women with neck pain and 10 controls performed 10-s isometric cervical flexion at 25% of their maximal force before and after vibration to the neck (bursts of 50 Hz with duration 20, 40, 60 and 120 s). Surface EMG was acquired from the sternocleidomastoid and splenius capitis. In both experiments, force steadiness was characterized by the coefficient of variation (CoV) and the relative power in three frequency subbands (low: 0–3 Hz; middle: 4–6 Hz; high: 8–12 Hz) of the force signal. Women with neck pain exhibited decreased force steadiness (Exp 1: patients 3.9 ± 1.3%, controls 2.7 ± 0.9%, $P < 0.05$; Exp 2: patients 3.4 ± 1.2%, controls 1.7 ± 0.6%, $P < 0.01$) which was associated with higher power in the low-frequency band (patients 71.2 ± 9.6%, controls 56.7 ± 9.2%, $P < 0.01$). Following vibration, CoV (2.6 ± 1.1%, $P < 0.05$) and the power in the low-frequency band of the force signal decreased (63.1 ± 13.9%, $P < 0.05$) in the patient group. These effects were not present in controls. Motor unit behavior and surface EMG amplitude were similar between groups. In conclusion, women with neck pain have reduced force steadiness, likely due to alterations in Ia afferent input. Vibration, which modulates Ia afferent input, increases force steadiness in patients with neck pain.

1. Introduction

Recent studies have shown that pain is associated with reduced force steadiness, defined as the ability to maintain a steady force output during submaximal contractions (Tracy and Enoka, 2002). Patients with knee osteoarthritis (Hortobagyi et al., 2004) and patients with subacromial impingement syndrome (Bandholm et al., 2006) display reduced force steadiness accompanied by a deficit in proprioception (Hortobagyi et al., 2004; Bandholm et al., 2006). Patients with neck pain show reduced proprioception of the neck and disturbances in postural stability (Jull et al., 2008), which have been attributed to alterations in afferent information from the neck. Disturbances in the sensorimotor control of the neck may result from either a decrease or increase in cervical somatosensory afferent activity (Jull et al., 2008), which can be due to direct trauma or the influence of pain and sympathetic activation on muscle spindle sensitivity (Passatore and Roatta, 2006). It may be expected that patients with neck pain would also show reduced force steadiness as a consequence of a disturbance in afferent input (O'Leary et al., 2007).

The oscillations of force around a target value can be characterized with the power of the force signal in frequency subbands (Allum et al., 1978; Marsden, 1978). The low-frequency range (0–3 Hz) of the power spectrum of the force signal is influenced mainly by the net output of the motor neural pool, i.e. by the number of active motor units and discharge rate of these motor units (Allum et al., 1978). Moreover, high variability in the discharge rate of motor units has been associated with reduced force steadiness (Laidlaw et al., 2000; Moritz et al., 2005) and increased power in the low-frequency band (Allum et al., 1978). Further, input from Ia afferents also contributes to force fluctuations at low frequencies (⩽4 Hz) during non-fatiguing contractions (Yoshitake et al., 2004). In contrast, the short and long latency stretch reflexes contribute to

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oscillations in the subbands 8–12 and 4–6 Hz, respectively (Marsden, 1978).

This paper presents the results of two experiments that investigate force steadiness and the mechanisms underlying impaired force steadiness in patients with chronic neck pain. The first experiment compares force steadiness in people with neck pain and asymptomatic controls. The force signal was investigated with frequency analysis and single motor unit behavior was extracted from intramuscular EMG recordings during brief cervical flexion contractions. We hypothesized that neck pain patients would display poorer force steadiness compared to controls, consistent with previous data (O'Leary et al., 2007), and that this would be associated with increased power at low frequencies of the force spectrum, likely due to abnormal afferent information. The second experiment examined the immediate effects of vibration of the neck on force steadiness in neck pain patients and controls. Mechanical vibration can result in improved force steadiness through modulation of Ia afferents (Yoshitake et al., 2004), although this has only been examined in individuals free of pain. In this experiment, we hypothesized that force steadiness would improve in patients with neck pain following local vibration to the neck.

2. Methods

In the first experiment (Exp 1), motor unit discharge behavior and force steadiness were measured during brief, isometric cervical flexion contractions in women with chronic neck pain and asymptomatic controls. The second experiment (Exp 2) examined the effects of short-term vibration on force steadiness in neck pain patients and controls. The description of methods refers to both experimental tests unless otherwise specified.

2.1. Subjects

Exp 1 included nine women (age, mean ± SD: 40.4 ± 3.5 years; height, 171.1 ± 10.6 cm; body mass, 73.4 ± 10.6 kg) and Exp 2 included 10 women (age, mean ± SD: 35.3 ± 7.5 years; height, 169.7 ± 7.4 cm; body mass, 72.2 ± 8.5 kg) with chronic neck pain. Participants between the ages of 18–60 years were included if they reported a history of neck pain of greater than 6 months duration, scored 5 points or greater out of a possible 50 points on the Neck Disability Index (NDI) (Vernon and Mior, 1991), and demonstrated positive findings on a physical examination of the cervical spine (altered joint motion and painful reactivity to palpation). Patients were excluded if they previously had cervical spine surgery or presented with neurological signs in the upper limb.

Nine healthy women were recruited for Exp 1 (age, mean ± SD: 38.9 ± 10.5 years; height, 165.4 ± 8.2 cm; body mass, 63.6 ± 10.7 kg) and 10 healthy women were recruited for Exp 2 (age, mean ± SD: 35.4 ± 8.9 years; height, 168.1 ± 5.1 cm; body mass, 66.5 ± 11.8 kg) as controls. Control subjects were free of shoulder and neck pain, had no past history of orthopedic disorders affecting the shoulder or neck region, and no history of neurological disorders. The participants were recruited through local advertisement. The neck pain patients and control groups were different in the two experiments. Ethical approval for the study was granted by the local Ethics Committee (No. 20070045) and the procedures were conducted according to the Declaration of Helsinki.

2.2. Procedure

Participants were seated with their head rigidly fixed in a device to measure neck force (Aalborg University, Denmark) with their back supported, knees and hips in 90° of flexion, and their torso firmly strapped to the seat back. The device is equipped with eight adjustable contacts, which were fastened around the head to stabilize the head and provide resistance during isometric contractions of the neck. The force device includes a force transducer (strain gauge) to measure force in the sagittal plane. The force signals were amplified (LISiN – OT Bioelettronica, Torino, Italy) and their output displayed on an oscilloscope to provide visual feedback.

Following a period of familiarization with the device and practice contractions, subjects performed two maximum voluntary contractions (MVC) of 3–4 s duration in cervical flexion. These contractions were separated by 1 min of rest. The subjects were verbally encouraged to achieve a higher force in the second trial. The highest value of force recorded over the two maximum contractions was selected as the reference MVC.

Following the maximal contractions, subjects performed isometric cervical flexion either at 15 N (Exp 1) or 25% MVC (Exp 2) for 10 s. The target force output was displayed on an oscilloscope located 80 cm in front of the subject with the gain adjusted to view ± 10% of the target force value on a 15-cm high display. The subjects were instructed to match the force output as closely as possible to the target force for the full duration of the contraction. Force variability is associated with strength (Sosnoff and Newell, 2006), thus in Exp 1, an absolute level of force was selected as the target to eliminate variations due to differences in strength between the controls and neck pain groups (Ylinen et al., 2004; Prushansky et al., 2005). In contrast, a relative level of force was used in Exp 2, which allowed us to test whether an eventual difference in force steadiness between patients and controls is only attributable to a reduction in muscular strength in the patient group. Moreover it is known that vibration may alter strength performance (Cardinale and Bosco, 2003). The duration of the contraction was minimized to avoid fatigue because patients with neck pain show greater fatigability than controls of the sternocleidomastoid during sustained cervical flexion contractions (Falla et al., 2003).

2.3. EMG recordings

In Exp 1, action potentials of single motor units were detected with a pair of Teflon-coated stainless steel wires (diameter: 0.1 mm; A-M Systems, Carlsborg, WA) inserted into the sternocleidomastoid muscle bilaterally, ~2-cm cephalad to the midpoint between the sternum and the mastoid process via a 25-gauge hypodermic needle. The wires were cut to expose only the cross section, and provided one bipolar signal which was amplified (Counterpoint EMG, DANTEC Medical, Skovlunde, Denmark), band-pass filtered (500 Hz–5 kHz), sampled at 10,000 Hz, and stored after 12-bit A/D conversion.

In Exp 2, surface EMG signals were acquired from the sternocleidomastoid and splenius capitis muscles bilaterally using bipolar surface electrodes (Ambu® Neuroline 720 01-K/12, Ambu A/S, Ballerup, Denmark) with an inter-electrode distance of 22 mm. Electrodes were positioned over the sternocleidomastoid muscle in the distal one third of the muscle (Falla et al., 2002a,b) and over the splenius capitis at the level of C2–C3 between the uppermost parts of sternocleidomastoid and upper trapezius muscle (Falla et al., 2007). The skin was prepared using abrasive paste and cleansed with water prior to electrode placement. A reference electrode was placed around the subjects' wrist. Surface EMG signals were amplified with a gain of 2000 (EMG-USB, LISiN, Politecnico di Torino and OT-Bioelettronica, Torino, Italy), filtered (−3 dB bandwidth, 10–400 Hz), sampled at 2048 Hz, and converted to 12-bit digital samples. Contrary to Exp 1, it was not possible to investigate individual motor unit behavior using intramuscular EMG signals in Exp 2. This was due to the vibration intervention that would have resulted in relative shifts of the intramuscular wires within the muscle, so that it would have been not possible to identify the same motor units pre and post vibration.

2.4. Vibration intervention – Exp 2

Following baseline measures, the subject was positioned in a supine posture with the knees bent and the arms crossed over the chest. The head/neck and shoulders were supported in slings suspended from the ceiling. While maintaining their head in a mid-position, subjects were asked to gently press their head down (neck extension) into the sling so their shoulders were ~2 cm off the plinth (closed chain neck extension; (Kirkesola, 2009)). Vibration was then applied to the head and neck by a mechanical vibrator apparatus (Redcord Stimula, Redcord A/S, Norway) connected to the sling that supported the subject's head and neck. The protocol for the vibration included a warm-up phase during which the subject received four vibration interventions at 15, 25, 35 and 50 Hz for 10 s each followed by a 30 s rest interval. Vibration was applied at 50 Hz for four further consecutive periods of 20, 40, 60 and 120 s with 60 s rest intervals.

Pain at rest was self reported by the subjects before and immediately after the vibration intervention on a visual analogue scale (0–10 cm). Additionally, pressure pain thresholds (PPT) were assessed unilaterally over the C2/3 zygapophyseal joint (on the most symptomatic side based on manual examination of the cervical spine) at baseline and following the intervention using an electronic digital algometer with a probe size of 1 cm^2 and application rate of 30 kPa/s (Somedic AB, Farsta, Sweden). Subjects were instructed to press a handheld switch at the first perception of pain, at which point the application of pressure ceased and the pressure was recorded. The mean of three trials was used for further analysis. A 15-s pause was provided between measures. Following the intervention, the subjects repeated the baseline measures in the following order: VAS, PPT, MVCs and submaximal contractions. Submaximal contractions started ~10 min after the completion of the vibration protocol.

2.5. Signal analysis

The coefficient of variation (CoV) of force (SD divided by mean, %) was obtained from the 10-s contractions. Given that the power of the force signal is distributed in a frequency bandwidth of ~12 Hz for isometric contractions at low forces , frequency analysis of the force signal was performed in three subbands: 0–3 Hz (low-frequency band), 4–6 Hz (middle-frequency band) and 8–12 Hz (high-frequency band) (Kouzaki et al., 2004). After removal of the mean value, the power spectrum was estimated from the force signal with the periodogram estimator; the relative power in the three bands was computed as the ratio (%) between the integral of the power spectrum in each band and the integral of the power spectrum between 0 and 12 Hz.

For Exp 1, single motor unit action potentials were identified from the intramuscular EMG with a decomposition algorithm (McGill et al., 2005). The discharge rate of each identified motor unit was obtained across the 10-s cervical flexion contraction. The interspike interval (ISI) variability was computed as the ratio (%) between SD and mean ISI. Discharge rate and ISI variability were computed from the entire duration of the contraction.

For Exp 2, the average rectified value (ARV) was estimated from the surface EMG signals during the MVC, from a 250-ms portion data centered at the point corresponding to the maximal exerted force. The ARV was also calculated during the isometric contractions over the 10 s period and normalized with respect to the ARV obtained during the MVC.

2.6. Statistical analysis

In Exp1, a one-way analysis of variance (ANOVA) was used to evaluate differences between patients and controls for maximal flexion force and CoV of force with group (patient, control) as the between subjects factor. A two-way mixed model ANOVA with group (patient, control) as the between subjects variable and frequency (low, mid and high) as within subjects variable was applied to compare the relative power in each of the three frequency bands between patients and controls. In Exp 2, the time (pre and post intervention) was included in the analysis as a within subjects variable to evaluate differences in maximal flexion force and CoV of force (two-way mixed model ANOVA) and the relative power of the force signal in each of the three frequency bands following the vibration intervention (three-way mixed model ANOVA).

In Exp 1, the motor unit data from the left and right sternocleidomastoid were combined to obtain an average since no side differences were observed for sternocleidomastoid discharge rate or ISI variability. The discharge rate and ISI variability of sternocleidomastoid motor units during the isometric cervical flexion contractions were analyzed with a one-way ANOVA with group (patient, control) as the between subjects variable.

In Exp 2, the surface EMG ARV data from the right and left sternocleidomastoid and splenius capitis were averaged since there were no significant side differences. A two-way mixed model ANOVA evaluated pre to post intervention differences in ARV in both groups (patients and controls). Separate ANOVAs were performed for the two muscles (sternocleidomastoid and splenius capitis).

Significant differences revealed by ANOVA were followed by post hoc Student Newman-Keuls (SNK) pair-wise comparisons. Pearson correlation coefficient was used to identify linear relations between the vibration-induced changes in the investigated variables. Results are reported as mean and SD. Statistical significance was set at $P < 0.05$.

3. Results

In each experiment, the patients and controls did not differ in age, body mass or height ($P > 0.05$). Table 1 presents the patients Neck Disability Index score (0–50), average pain intensity rated on a visual analogue scale (VAS), cause and the duration of symptoms for both experiments.

3.1. Maximal force

In both experiments, the patient group exerted lower maximal cervical flexion force compared to the controls (Exp 1: patients 46.3 ± 37.0 N, controls 109.5 ± 31.4 N, $P < 0.05$; Exp 2: patients 55.5 ± 23.2 N, controls 99.7 ± 31.5 N (average across pre and post vibration), $P < 0.01$). Vibration did not affect maximal cervical strength (patients: $P = 0.83$; pre 55.1 ± 20.4 N, post 55.9 ± 26.5 N; controls: $P = 0.20$; pre 102.1 ± 28.4 N, post 97.2 ± 35.7 N).

3.2. Force steadiness

In Exp 1, the CoV of flexion force was dependent on group ($P < 0.05$), with greater values observed for the patients (3.9 ± 1.3%) compared to controls (2.7 ± 0.9%). Across all subjects, the relative power differed among the three subbands ($P < 0.0001$), with greater power identified in the lower frequency band compared to the mid- and high-frequency bands ($P < 0.05$). In addition, the

Table 1
Characteristics of the patients with neck pain. NDI = Neck Disability Index.

	Exp 1 ($n = 9$)	Exp 2 ($n = 10$)
Onset (idiopathic, trauma) % trauma	33.3	80.0
Length of history (years)	13.6 ± 10.2	7.6 ± 5.3
NDI (0–50)	14.8 ± 8.6	24.8 ± 7.2
Average pain (VAS, 0–10)	4.4 ± 1.7	6.0 ± 1.4

power in the mid-frequency band was greater than in the high-frequency band ($P < 0.001$).

Fig. 1 shows the power spectrum of the force signal after removal of the mean value from representative subjects in the two groups. In this example, the patient shows greater power in the low-frequency band (74%) and lower power in the mid- (12%) and high-frequency bands (2%) compared to the control subject (45%, 21% and 9%, respectively). From the group data analysis, the percent of power in the low-frequency band was higher for the patients ($61.2 \pm 9.6\%$) compared to controls ($53.7 \pm 11.1\%$; $P < 0.05$). No significant difference was observed between the mid- and high-frequency bands (mid: patients 12.4 ± 2.5, controls $17.4 \pm 5.8\%$, $P = 0.18$; high: patients $2.3 \pm 0.7\%$, controls $6.3 \pm 6.2\%$, $P = 0.29$).

In Exp 2, the CoV of force depended on the interaction between group and time ($P < 0.05$; Fig. 2). The results confirmed that patients with chronic neck pain display reduced force steadiness during submaximal sustained contractions compared to the control subjects. However, vibration influenced force steadiness in the patients. The CoV of force was reduced for the patient group following the vibration intervention ($P < 0.05$; pre $3.4 \pm 1.2\%$, post $2.6 \pm 1.1\%$), indicating improved steadiness of contraction. However, vibration did not influence force steadiness in the control group ($P = 0.46$; pre $1.7 \pm 0.6\%$, post $1.9 \pm 0.7\%$).

As in Exp 1, the results for Exp 2 showed that the relative power of the force signal across all subjects differed among the three subbands ($P < 0.001$), with greater power identified in the lower frequency band compared to the mid- and high-frequency bands ($P < 0.001$) and in the mid-frequency band compared to the high-frequency band ($P < 0.05$). Moreover, the relative power in the three subbands depended on the interaction between group, frequency band and time ($P < 0.05$, Table 2). In agreement with Exp 1, at baseline, the low frequency content of the force signal was higher for the patients than for controls ($P < 0.01$), with no significant differences observed for the mid- and high-frequency bands. Following vibration, only the patients showed decreased power in the low-frequency band ($P < 0.05$), resulting in no difference of the power in the low-frequency band between the patients and control group post intervention. No differences were observed in the mid- and high-frequency bands for the patients and controls.

In the representative example of Fig. 3, the relative power in the low-frequency band is higher for the patient (68%, Fig. 3C) than for the control (65%, Fig. 3A). In this example, following vibration, the power in the low-frequency band decreased for the patient (63%, Fig. 3D), while the control subject showed no change (66%, Fig. 3B).

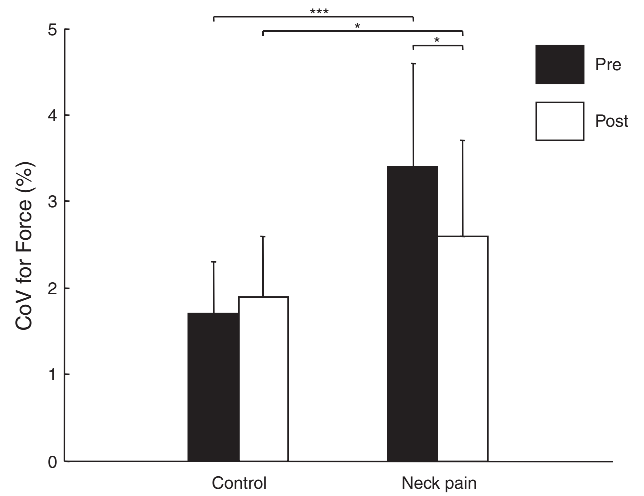

Fig. 2. Mean and standard deviation of the CoV of force obtained for the control and patient groups from the 10-s cervical flexion performed at 25% MVC at baseline and following vibration. Only the patient group showed a decrease of the CoV value following vibration. ($^{*}P < 0.05$; $^{***}P < 0.001$).

A positive correlation was identified between the relative power in the low frequency band and the force CoV (controls: $R = 0.46$, $P < 0.05$; patients: $R = 0.57$, $P < 0.01$) and between the decrease in the relative power in the low frequency band and the force CoV in patients following vibration ($R = 0.80$, $P < 0.01$).

3.3. Motor unit behavior and muscle activity

In Exp 1, a total of 62 and 48 motor units were identified from the sternocleidomastoid muscle for the controls and patients, respectively. The mean motor unit discharge rate was similar for the patients (14.7 ± 3.2 pps) and controls (15.5 ± 2.4 pps) ($P = 0.68$). Furthermore, the ISI variability did not differ between groups (patients: $18.8 \pm 4.3\%$, controls: $19.0 \pm 2.5\%$, $P = 0.74$). In Exp 2, the ARV of sternocleidomastoid and splenius capitis EMG was not statistically different between groups, and did not change following vibration in either group (Table 3).

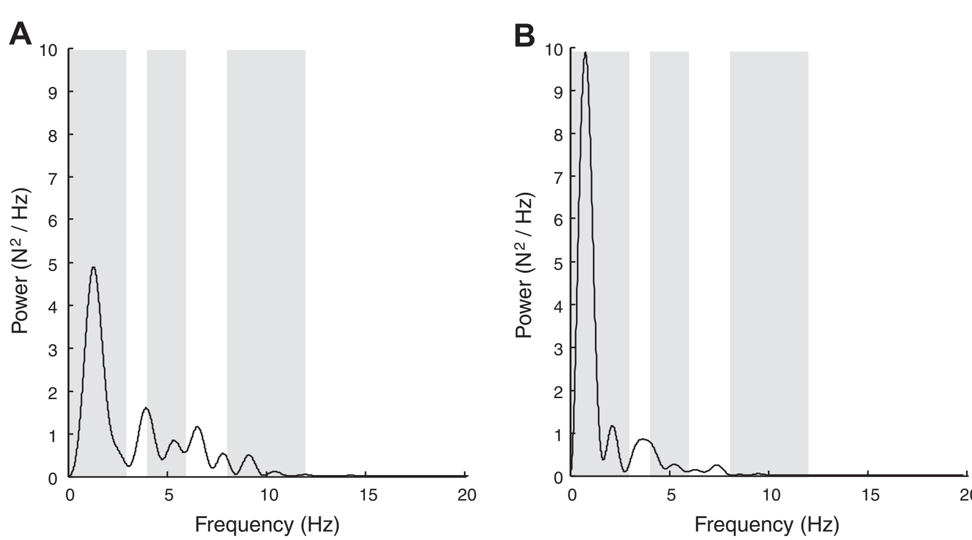

Fig. 1. Power spectrum of the force signal during a 10-s isometric cervical flexion at 25% of maximal force for a representative control subject (A) and a neck pain patient (B). The panels present the three frequency bands of interest (0–3, 4–6 and 8–12 Hz).

Table 2
Frequency analysis of force fluctuations. Relative power (mean ± SD) in the low-, middle- and high-frequency bands for controls and patients at baseline (Pre) and following the vibration intervention (Post). NS = non significant.

	Low-frequency band			Middle-frequency band			High-frequency band		
	Pre	Post	*P*-level	Pre	Post	*P*-level	Pre	Post	*P*-level
Controls	56.7 ± 9.2%	62.5 ± 12.4%	NS	17.3 ± 8.2%	12.3 ± 7.2%	NS	6.5 ± 4.9%	9.9 ± 8.8%	NS
Patients	71.2 ± 9.6%	63.1 ± 13.9%	<0.05	10.3 ± 2.7%	11.1 ± 3.4%	NS	3.1 ± 2.8%	6.2 ± 8.3%	NS
P-level	<0.01	NS		NS	NS		NS	NS	

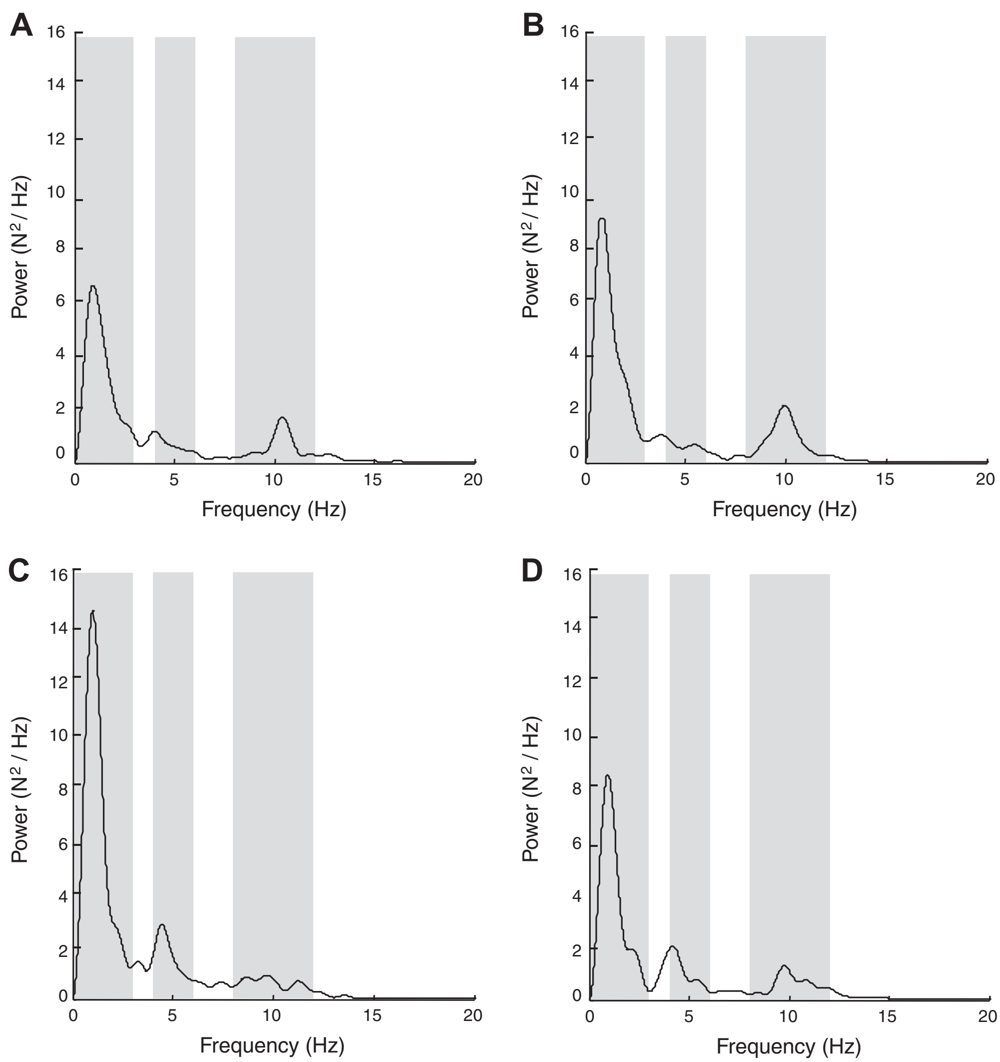

Fig. 3. Power spectrum of the force signal during a 10-s isometric cervical flexion at 25% of maximal force for a representative control subject at baseline (A) and following the vibration (B) and for a neck pain patient before (C) and after the vibration intervention (D). The panels present the three frequency bands of interest (0–3, 4–6 and 8–12 Hz).

Table 3
Average rectified value (ARV) of the surface EMG for the sternocleidomastoid and splenius capitis muscles during isometric cervical flexion (25% maximum) normalized to the ARV from the maximum voluntary contraction, left and right values averaged.

	Sternocleidomastoid		Splenius capitis	
	Pre	Post	Pre	Post
Controls	22.1 ± 7.8%	22.3 ± 7.4%	15.6 ± 8.8%	15.3 ± 8.9%
Patients	24.2 ± 10.7%	20.9 ± 11.9%	22.8 ± 17.0%	18.8 ± 14.4%

3.3.1. Pain assessment – Exp 2

PPT was significantly different between the two groups ($P < 0.05$), with lower pain thresholds observed for the patient group (134.1 ± 64.6 kPa) compared to control subjects (217.3 ± 97.1 kPa, average across pre and post vibration). Short-term vibration to the neck did not affect VAS scores ($P = 0.33$; pre 5.6 ± 1.9, post 5.1 ± 2.1) or PPT (patients: $P = 0.24$; pre 141.9 ± 74.8 kPa, post 126.3 ± 60.3 kPa; controls: $P = 0.32$; pre 223.8 ± 109.6 kPa, post 210.8 ± 87.2 kPa).

4. Discussion

Women with neck pain displayed an impaired ability to maintain a steady neck flexion force around a target value and this corresponded to a higher power of force fluctuations at low frequencies. Despite differences in force steadiness, the motor unit behavior was similar in the two groups. Local vibration of the neck improved force steadiness in the patient group but had no influence in the asymptomatic subjects. The significant decrease in force fluctuations in the patient group was associated with reduced power of the force signal at low-frequencies (0–3 Hz).

4.1. Force steadiness

Patients with neck pain exhibited reduced neck flexion strength and poorer accuracy in maintaining a steady low-load contraction. In our experiments, we tested whether this decrease in accuracy occurs in presence of neck pain at both absolute and relative levels of force. The CoV of force varies with the level of contraction, with higher values at low force levels, at least for limb muscles (Christou et al., 2002; Moritz et al., 2005). In Exp 2, the target force was expressed as a percentage of MVC, thus the relative load was the same for all subjects. However, since patients with neck pain have less neck strength compared to controls, the same relative load corresponds to lower absolute levels of force and to maintain a steady force output is a more demanding task at low force magnitude (Christou et al., 2002; Taylor et al., 2003). Moreover, fear of pain may have prevented the patients from exerting their maximal force during the MVC assessment. Thus, even if expressed in relation to the recorded MVC, the target values may not correspond to the same relative load across patients and controls, considering the actual capability of patients to produce force. However, the findings from the two experiments showed impaired force steadiness for the patient group irrespective of whether the target force was absolute or relative, indicating that the decrement in steadiness was probably not due to the reduction in flexion strength per se. We have investigated the mechanisms for this impairment through spectral analysis of the force signal and single motor unit data.

The power of the force signal in frequency subbands corresponds to different mechanisms involved in force fluctuations. Low frequencies are associated with changes in the net activity of the motorneuron pool (discharge rate and number of active motor units), including Ia afferent input (Yoshitake et al., 2004). The mid-frequency band (4–6 Hz) is associated to the long latency stretch reflex and the higher frequencies (8–12 Hz) correspond to the short latency stretch reflex (Marsden, 1978; McAuley and Marsden, 2000). During non-fatiguing, force matching tasks, most of the power of the force signal is at low frequencies (Taylor et al., 2003; Vaillancourt and Newell, 2003), as also shown in this study.

The patient group displayed greater power in the low-frequency band of the force signal compared to asymptomatic subjects. Research has demonstrated that this may be due to differences in the net activity of the motorneuron pool and alterations in afferent input from the Ia circuit. Motor unit discharge rate variability has been shown to influence force fluctuations, with maximal steadiness occurring for regular but non-synchronized discharges of motor units (Taylor, 1962). For example, reduced steadiness of contraction in older adults is associated with higher variability of single motor unit discharge rates (Laidlaw et al., 2000). This relationship, however, is not always consistent as most of the force variability is due to the low-frequency fluctuations in motor unit discharge rates (Laidlaw et al., 2000). For example, Galganski et al. (1993) found no difference in motor unit discharge behavior between young and elderly subjects during isometric abduction of the index finger at 5–50% MVC, despite greater force fluctuations in the elderly group. Similarly, a difference in force steadiness between the patients and controls in this study occurred despite an absence of a difference in the mean discharge rate or ISI variability of discharge rate of sternocleidomastoid motor units (Exp 1). This result is in agreement with the poor association between ISI variability and force variability (Negro et al., 2009). In addition, the reduced steadiness of contraction in the patient group could not be attributed to differences in the activity of the agonist or antagonist muscles since it did not differ between the patients and controls (Exp 2). Similarly, reduced steadiness of force was observed for the first dorsal interosseous muscle of older individuals in the absence of differences in agonist and antagonist muscle activity (Burnett et al., 2000).

The greater force fluctuations and power in the low-frequency band of the force signal in the patient group likely occur from altered cervical afferent input from damaged or functionally impaired neck joint and muscle receptors. There are several possible causes of disturbed cervical afferent input in neck pain, including direct damage to mechanoreceptors (Lu et al., 2005), sensitization of mechanoreceptors from pain (Seaman, 1999) and decreased central inhibition (Curatolo et al., 2001) and sympathetic effects on muscle spindle sensitivity (Hellstrom et al., 2005). These phenomena may result in either a decrease or increase in cervical somatosensory afferent activity. For example, sympathetic activation can cause a reduction (Hellstrom et al., 2005) or an overactivity (Hinoki, 1984) of spindle afferents. Information from the muscle spindles is of primary importance for cervical proprioceptive acuity and a changed sensitivity of the muscle spindles may give rise to a mismatch of sensory input, since this information can conflict with input from the vestibular and visual systems (Jull et al., 2008). Impaired proprioception is indeed a well-documented finding in patients with neck pain (Treleaven et al., 2003; Jull et al., 2008).

4.2. Effect of vibration on force steadiness

Short term vibration to the neck in women with neck pain decreased force fluctuations and reduced the power in the low-frequency band of the force signal. In contrast, vibration did not affect strength performance or muscle activity, both in people with and without neck pain.

Some studies reported enhanced maximal force production following vibration (Bosco et al., 1999, 2000), although this finding is not always consistent (de Ruiter et al., 2003), and may depend on the frequency and duration of the vibration. The primary endings of muscle spindles increase their activity during vibration (Hasan and Houk, 1975). Thus, brief vibrations increase the excitatory input to the α-motor neuron pool via Ia afferents, which in some studies has been associated with increased EMG activity and maximal force during the vibration (Cardinale and Bosco, 2003). These effects attenuate with a prolonged vibration of approximately 30 min (Hayward et al., 1986; Jackson and Turner, 2003), which is longer than in our study. Vibration is not selective to Ia afferents and IIa and Ib afferents are also stimulated. However, the primary endings of the muscle spindles are more sensitive to vibration than the secondary endings and the Golgi tendon organs (Cardinale and Bosco, 2003).

The observed reduction in force fluctuations and in spectral power of the force signal at low frequencies supports a modulation of afferent input. The finding that force steadiness only improved for the women with neck pain, may indicate that neck pain patients have abnormal cervical afferent input, as shown in previous reports (Treleaven et al., 2003). Vibration was not delivered locally over a muscle but rather was applied to the neck via a supporting sling. Thus the vibration may have also influenced the activity of other muscles including the deep neck muscles as these muscles have an unusually high spindle density (Liu et al., 2003) and may be more sensitive to vibration. Increased activation of the deeper cervical muscles could contribute to increased precision of maintaining a steady force.

4.3. Effect of vibration on pain

Short-term vibration of the neck did not influence the pressure pain thresholds or pain intensity in women with neck pain.

Previous studies suggest that vibration elicits an analgesic effect (Pertovaara, 1979; Lundeberg, 1984), albeit the mechanisms for this effect are not fully understood. Several studies demonstrated that high frequency vibrations (⩾100 Hz) more effectively alleviate pain than lower frequencies. For example, Pertovaara (1979) reported that local vibration at 240 Hz induced a significant elevation of the pain threshold with no effect observed when vibration was applied at 20 Hz. Likewise, Lundeberg (1984) reported that vibratory stimulation at 100 and 200 Hz is superior in suppressing pain than a 20 Hz vibratory stimulation. The proposed explanation is based on Melzack and Wall's gate control theory of pain (Melzack and Wall, 1965). According to this theory, fibers which transmit information from cutaneous vibration receptors could inhibit the nociceptive pathways. Pacinian corpuscles are sensitive to frequencies higher than 90 Hz (Loewenstein and Skalak, 1966), and may play a role in vibratory analgesia. In this study, the frequency may have not been high enough to activate Pacinian pathways which may explain the absence of an analgesic affect. We selected 50 Hz in this study because previous research showed that the frequency range 30–50 Hz is effective at enhancing motor output (Luo et al., 2005). The short duration of the vibration exposure could also explain the observed lack of analgesic response. Lundeberg et al. (1984) suggested that vibratory stimulation should be applied for 25–45 min to achieve a pain relieving effect. Our protocol included only 6 min of vibration intervention including the 2 min warm up phase.

4.4. Clinical considerations

Since the participants underwent only one training session, the observed improvement may only be transient. Further studies are needed to establish if a training program could potentiate the beneficial effects obtained. Moreover, frequency and amplitude of the vibration were kept constant during the application (except for the warm up phase). The influence of these parameters should be further assessed. In particular it would be relevant to examine whether higher frequency vibration provides pain relief while still retaining or further improving force performance.

5. Conclusion

Women with chronic neck pain exhibit increased force fluctuations during brief submaximal isometric cervical flexion. The results on force spectral components and motor unit behavior indicate that abnormal afferent input from neck receptors may explain these observations. Subsequently, vibration decreased the relative power in the low-frequency band of the force signal, resulting in improved force steadiness in neck pain patients.

Declaration of interests

The third author of the paper, Gitle Kirkesola, is an employee of the company Redcord A/S. Some of the equipment used in the study was supplied by this company as indicated in the text of the manuscript.

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