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RESEARCH ARTICLE

Changes in the degree of motor variability associated with experimental and chronic neck–shoulder pain during a standardised repetitive arm movement

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Abstract The aim of the present study was to investigate the effect of experimental and chronic neck–shoulder pain on the magnitude of cycle-to-cycle variability of task timing, kinematics and muscle activation during repetitive arm movement performed for 3 or 5 min. In an experimental part, acute muscle pain was induced in healthy subjects by intramuscular injection of hypertonic saline in trapezius ($n = 10$) and infraspinatus ($n = 10$) muscles. In a clinical part, workers with ($n = 12$) and without ($n = 6$) chronic neck–shoulder pain were compared. Cycle-to-cycle standard deviations of task duration, arm and trunk movement in 3D and surface electromyographic (EMG) root mean square activity were computed to assess the degree of variability. The variability in task timing increased in presence of both experimental and chronic pain ($P < 0.05$) compared with non-painful conditions. Experimental pain increased the variability of the starting position of the arm ($P < 0.05$), the arm range of motion ($P < 0.01$), the arm and trunk movement area ($P < 0.01$) and the acceleration of the arm ($P < 0.01$). In the chronic pain condition, the variability of arm and trunk acceleration ($P < 0.01$) and EMG activity ($P < 0.05$) was decreased compared with healthy controls. These results indicate that pain alters the magnitude of motor variability, and that the transition from acute to chronic pain is accompanied by changes in motor patterns. Experimental pain likely resulted in a quest for a motor solution reducing nociceptive influx, while chronic pain was characterised by a diminished motor flexibility.

Keywords Variation · Pain status · Muscle pain · Motor pattern · Shoulder region

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Introduction

Discomfort and pain in the neck–shoulder region is particularly prevalent among workers engaged in repetitive work where a short work cycle is repeated over and over again for long periods of time (Bernard 1997; Punnett and Wegman 2004). Muscle pain influences motor control strategies via central mechanisms (Le Pera et al. 2001; Thunberg et al. 2002), and the effects of muscle pain on motor patterns have been widely studied both during isometric and dynamic contractions (Arendt-Nielsen et al. 1996; Birch et al. 2000). During dynamic contractions, neck–shoulder pain status, i.e., acute, sub-chronic or chronic, has been shown to be associated with different motor adaptation mechanisms that can explain the transitions of pain status (Madeleine et al. 2003b). Acute experimental pain induced by hypertonic saline injection was found in another part of the same study to induce a slower working rhythm, a decreased electromyographic (EMG) activity of the painful muscle and an increased amplitude of arm movements (Madeleine et al. 1999). Sub-chronic pain (pain developed after 6 months of work) resulted in lower force level, higher EMG activity, decreased arm movement amplitude and, increased trunk movement amplitude (Madeleine et al. 2003a), while

chronic pain led to a slower working rhythm, an increased background EMG activity of the trapezius muscle and an increased amplitude of arm movements (Madeleine et al. 1999).

In studies of motor adaptation to pain, one aspect of motor control has often been neglected: motor variability. The size and structure or components of motor variability in a standardised task reveal attributes of the motor control strategies, e.g., the extent that degrees of freedom are used (Latash et al. 2002; Sosnoff et al. 2006). Temporal and spatial variation in postures and muscle activity has been suggested to be a decisive determinant of risk for the development of work-related musculoskeletal disorders (Mathiassen 2006), and it has been hypothesised that individuals who perform a particular work task in a more stereotyped fashion than others, and thus do not fully benefit from the redundancy of the motor system, are more at risk (Mathiassen et al. 2003). This theory may be particularly relevant to neck–shoulder disorders since the shoulder region comprises a very complex construct of muscles and thus a large potential for achieving the same external result through different synergies. The ability and readiness to take on different muscle synergies in the neck–shoulder region has been demonstrated using biofeedback techniques (Palmerud et al. 1995), and also as a response to neck–shoulder pain (Madeleine et al. 1999). Recent studies have suggested that even a reorganisation of EMG activity among subdivisions of the trapezius muscle occurs in the presence of acute experimental pain, and that this may well explain the spreading of pain observed in clinical conditions (Falla et al. 2006; Madeleine et al. 2006).

Acute experimental muscle pain affects feed-forward control of muscles in a complex way beyond a general inhibition (Ervilha et al. 2004, 2005; Hodges et al. 2003). At chronic pain stages, motor variability is changed too as compared to a pain-free state, but both increases and decreases in motor variability have been reported (Lamoth et al. 2006). Individuals with a larger motor variability show a higher probability of returning to normal postural strategies after experimental pain than subjects with less flexibility (Moseley and Hodges 2006). Thus, recent studies have demonstrated that motor variability can be modulated by muscle pain and that the size of the motor variability may have important clinical implications.

We hypothesised that the magnitude of cycle-to-cycle motor variability in a stereotyped repetitive task decreases during the course of pain transitions; from an increased degree of variability during acute experimental pain to a decrease in chronic pain conditions. For this purpose, secondary analysis was performed on data set collected for a previous study (Madeleine et al. 1999).

Subjects and methods

Subjects

In total, 38 volunteers took part in this study which is composed of two parts. The first, experimental part involved 20 healthy participants with no pain or history of injuries in the neck–shoulder region [mean age 26.1 years (SD 2.6), weight 76 kg (SD 6.6), height 181.6 cm (SD 10.3)]. In the second, clinical part, 12 butchers suffering from chronic neck–shoulder pain participated [mean age 48.6 years (SD 7.2), weight 78.9 kg (SD 12.3), height 173.9 cm (SD 8.3)] together with 6 controls [mean age 43.8 years (SD 6.7), weight 83.0 kg (SD 8.5), height 181.2 cm (SD 7.5)]. The local Ethical Committee approved the studies (VN 94/184 and 95/3352), and informed consent was obtained from all participants. The studies were conducted in accordance with the Declaration of Helsinki.

Protocol

Motor recordings were performed during a standardised motor task in a laboratory setting. The subjects were instructed that they were to perform a task representing a common work task in the meat industry (Fig. 1). The task was time-paced using a tone generator triggered every second, and it consisted of five events (Madeleine et al. 1999): (1) pressing a sensor with the left hand; (2) micropause; (3) applying a 30 N force in the diagonal direction with the right hand; (4) applying a 30 N force in parallel to the frontal plane with the right hand; force feedback was given to the experimenter via a multimeter (Fluke 37, Tilburg, The Netherlands) and fed back verbally to the subject; and (5) pressing a sensor with the left hand. The task was thus standardised with respect to the movement amplitude and the load level. The subjects practiced until they were able to perform the task correctly and with ease. Following this practice period, the subjects performed the experimental task according to the following protocol, during which motor patterns were recorded (see "Motor recordings and analyses"):

Experimental part

First, the subjects performed the task for 3 min in a pain-free state. After 5 min rest, experimental muscle pain was induced by means of intramuscular injection of 0.75 ml of sterile hypertonic saline (6%) injected over 15 s. A 27Gx1-1/2″ cannula was inserted into the muscle belly (1.5–2 cm) of the right trapezius muscle (2 cm laterally from the mid-distance between cervical vertebra C7 and acromion) or of

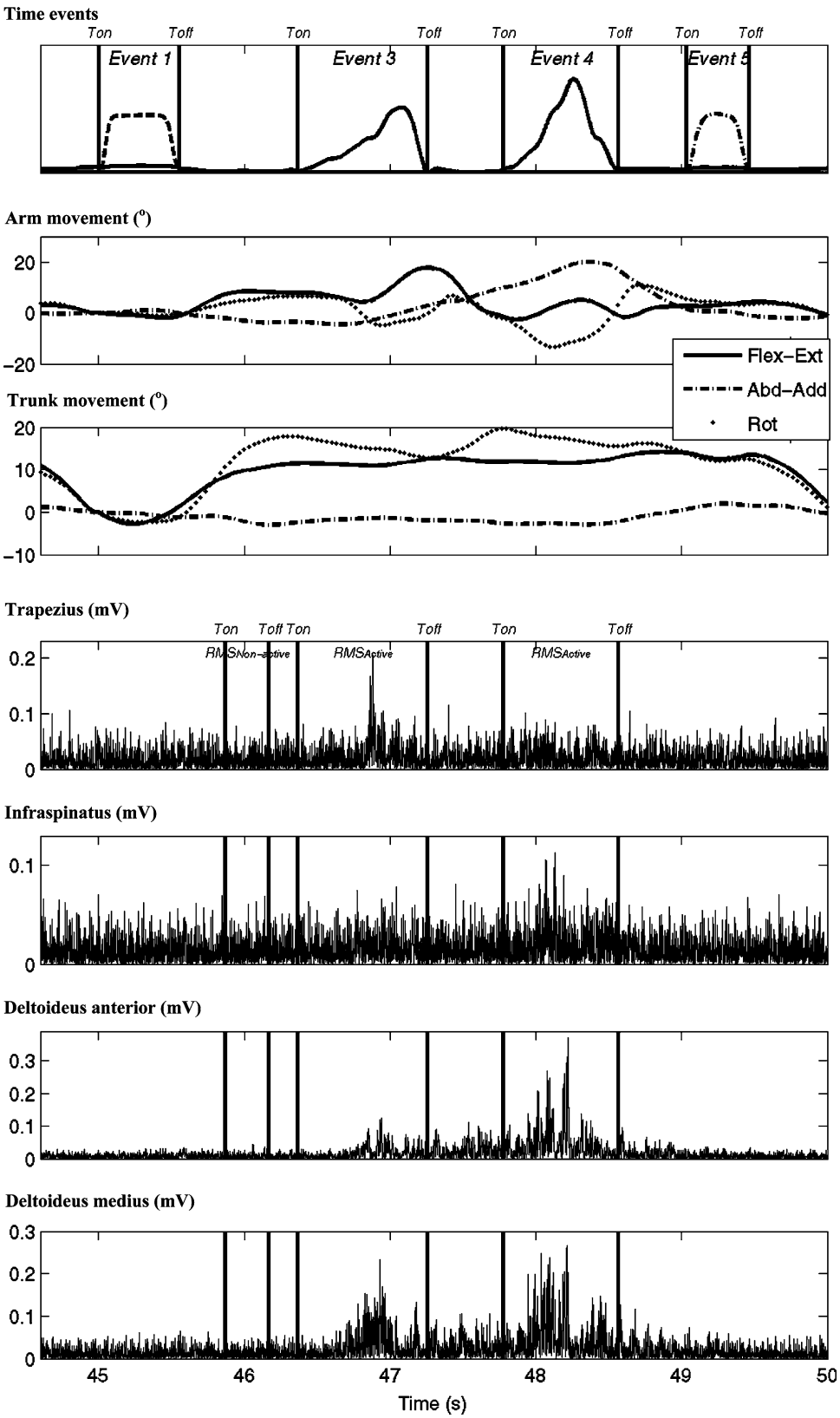

Fig. 1 Example of a task cycle for subject #10 in the experimental part; pain free state. From the *top*, task events [event (1) *dashed line*, events (3)–(4) *solid line* and event (5) *dashed-dotted line*) with their onset and offset (T_{on} and T_{off}); arm and trunk movement (°) in the flexion–extension (*Flex–Ext*), abduction–adduction (*Abd–Add*) and rotation (*Rot*) directions, corrected for offset in initial position; rectified electromyographic activity (mV) from the right trapezius, infraspinatus, deltoideus anterior and deltoideus medius muscles with the onsets and offsets used to calculate root mean square values ($RMS_{non\text{-}active}$ and RMS_{active})

the right infraspinatus muscle (two finger breadths below the medial portion of spine of scapula). Immediately after the bolus injection, the subject started performing the task and continued for 5 min. The mean pain intensities for the right trapezius and infraspinatus muscles measured across these 5 min were 4.9 (SD 2.0) and 4.4 (SD 1.0),

respectively, on a 0–10 point visual analogue scale (Madeleine et al. 1998). Data from the two injection sites were pooled to show the overall effect of neck–shoulder pain.

Clinical part

Both groups performed the task for three times 3 min with 5 min pause in between. The mean pain intensity referred to the right trapezius was measured during the motor task on a 0–10 point visual analogue scale. The butchers with chronic neck–shoulder pain and the healthy controls reported intensities of 4.8 (SD 1.8) and 0 (SD 0), respectively (Madeleine et al. 1998).

Motor recordings and analyses

Task timings from the left hand were sampled at 500 Hz by means of force sensing resistor® device (Toptronic, Echternach, Luxembourg). The durations of the task events (1) through (5) [except event (2)] as well as the total duration were computed for each cycle. T_{on} and T_{off} of each event were obtained using a level detection technique. Strain gauges mounted on a knife were used to measure the applied cutting force during events (3) and (4) (sampling frequency 500 Hz). The degree of variability of the task events and the whole cycle was measured by computing the standard deviation of their durations.

The movements of the upper right arm and trunk were recorded using a 3D motion analysis system at a sampling rate of 30 Hz (McReflex, Qualysis A/S, Partille, Sweden). Three cameras were used and acted as infrared illuminator and detector of passive reflective markers. Two complexes of three markers were attached on the back and around the upper right arm. Each marker was tracked in 3D by direct linear transformation. Reference recordings with the subjects in an upright anatomical position in the laboratory coordinate system were performed prior to work simulation recordings. After the tracking procedure, the markers' 3D coordinates were low-pass filtered using a Butterworth filter (order 4, cut-off frequency 3 Hz). Relative motion between the right arm and the trunk were computed and expressed for the upper arm as anatomical flexion–extension, abduction–adduction, and inward–outward rotation, and for the trunk as flexion–extension, right–left lateral flexion, and rotation (Madeleine et al. 1999). For each cycle, four kinematic parameters were determined in 3D for both the right arm and the trunk: (1) the starting position (°), (2) the integrated acceleration through the cycle (° s^{-2}), as obtained by double differentiation, (3) the range of motion (°, ROM) and, (4) the total area under the movement curve versus time (°s). The cycle-to-cycle standard deviations of these four parameters were used to express aspects of kinematic variability. Thus, the degree of variability was assessed at a whole-cycle level, but not with respect to sub-events within the cycle (Chau et al. 2005).

Surface EMG recordings were collected from four muscle of the right shoulder girdle. Bipolar EMG surface electrodes (Medicotest® N-10–E, Ølstykke, Denmark) were aligned (inter-electrode distance 2 cm) on abraded ethanol-cleaned skin along the direction of the muscle fibres. The four electrode centres were placed, (1) halfway between the lateral 1/3 of clavicula and the insertion of deltoideus (deltoideus anterior), (2) halfway between acromion and the insertion of deltoideus medius (deltoideus medius), (3) infraspinous fossa, two finger breadths below medial portion of the spine of scapula (infraspinatus) and, (4) 2 cm laterally from the mid-point between cervical vertebra C7 and acromion (trapezius). EMG signals were pre-amplified 100 times and amplified 2,000 times in total, band-pass filtered 10–400 Hz, and sampled at 1,000 Hz. Surface EMG activity was monitored by measuring the root mean square (RMS) values. Background RMS EMG normalised values $RMS_{non\text{-}active}$ (%) were computed for each muscle during a non-active part of the task cycle (300 ms epoch) and normalised to a reference value measured with the right arm resting on the workbench. RMS normalised values RMS_{active} (%) were also computed for each muscle and for each cycle during active phases [events (3) and (4)] and were normalised to the reference force in the same arm position. The $RMS_{active}/RMS_{non\text{-}active}$ absolute ratio (RMS_{ratio}) was also computed for each cycle. The standard deviations of $RMS_{non\text{-}active}$, RMS_{active} and RMS_{ratio} were then computed as measures of the amplitude of cycle-to-cycle variability in surface EMG activation during the task.

Statistics

Mann–Whitney test (test of the two groups) and two-way analyses of variance (ANOVA) [factors: (1) time event or muscle or direction of movement, (2) pain status] with Student–Newman–Keuls (SNK) method for multiple comparisons were applied. $P < 0.05$ was considered significant.

Results

Variability magnitude during acute experimental pain

The standard deviation of task event durations increased during experimental pain from 0.08 s (SE 0.006) to 0.09 s

(SE 0.007) ($F_{3,19} = 90.9$, $P < 0.001$, $\eta^2 = 0.97$). There was a significant interaction between pain status and time event ($F_{3,57} = 10$, $P < 0.001$, $\eta^2 = 0.19$); the standard deviation of event (3) was greater during experimental pain than before pain [0.16 s (SE 0.01) vs. 0.14 s (SE 0.01), respectively, $P < 0.001$, SNK].

Table 1 presents the results of the two-way ANOVA of the standard deviations of arm and trunk starting position, range of motion, total area and, movement acceleration. For the arm starting position, range of motion, total area (Fig. 2) and, acceleration (Fig. 3), values were greater during experimental pain than before pain [6.4° (SE 1.0) vs. 3.0° (SE 0.2), 6.4° (SE 0.7) vs. 3.1° (SE 0.2), 14.4°s (SE 1.6) vs. 6.2°s (SEM 0.4), and 5.6°s^{-2} (0.5) vs. 3.9°s^{-2} (SE 0.3), respectively, $P < 0.05$]. For the arm, there was a significant interaction between pain status and direction of movement, the standard deviation of the starting position was greater during experimental pain compared with before pain for the flexion and rotation directions ($P < 0.05$, SNK).

For the trunk, the standard deviation of the total area of movement was greater during experimental pain compared with before pain [7.9° (SE 1.0) vs. 4.6° (SE 0.4), respectively, $P < 0.05$]. There was significant interaction between pain status and direction of movement, the standard deviation of the total area of movement was greater during experimental pain compared with before pain for the flexion direction (Fig. 2, $P < 0.001$, SNK).

Table 2 presents the results of the two-way ANOVA for $RMS_{non\text{-}active}$, RMS_{active}, and RMS_{ratio} standard deviations. There was significant interaction between pain status and muscle; the standard deviation of the $RMS_{non\text{-}active}$ was smaller during experimental pain compared with before pain for the deltoideus anterior (Fig. 4, $P < 0.001$, SNK).

Variability magnitude among workers with chronic neck–shoulder pain

The standard deviation of the task cycle duration was greater among patients with chronic neck–shoulder pain than among healthy controls [0.32 s (SE 0.02) vs. 0.26 s (SE 0.04), respectively, $P = 0.042$].

The standard deviation of the arm acceleration was smaller for the patients than for the controls [4.2°s^{-2} (SE 0.2) vs. 6.2°s^{-2} (SEM 0.4), respectively, $P < 0.001$]. There was significant interaction between pain status and direction of movement, the standard deviation of acceleration was smaller for the patients than for the controls for the flexion and rotation directions (Fig. 5, $P < 0.05$, SNK).

The standard deviation of the trunk acceleration was smaller for the patients than for the controls [2.5°s^{-2} (SE 0.1) vs. 4.1°s^{-2} (SEM 0.3), respectively, $P < 0.001$]. As for the arm, there was significant interaction between pain status and direction of movement, the standard deviation of acceleration was smaller for the patients than for the controls for the flexion, abduction and rotation directions (Fig. 5, $P < 0.05$, SNK).

Table 1 Statistical results for the experimental and clinical study parts on the standard deviation (SD) of the arm and trunk starting position (start position), range of motion (ROM), total area of movement and movement acceleration ($F_{a,b}$: variance ratio with degrees of freedom, P: level of significance, η^2: partial eta squared)

Experimental part	Arm									Trunk								
	Direction			Pain			Direction × pain			Direction			Pain			Direction × pain		
	$F_{2,19}$	P	η^2	$F_{1,19}$	P	η^2	$F_{2,38}$	P	η^2	$F_{2,19}$	P	η^2	$F_{1,19}$	P	η^2	$F_{2,38}$	P	η^2
SDStart pos.	10.3	**<0.001**	0.52	6	**0.024**	0.62	6.2	**0.005**	0.25	19.1	**<0.001**	0.54	1.1	0.301	0.14	0.9	0.405	0.05
SDROM	17.6	**<0.001**	0.43	9.8	**0.005**	0.64	2.9	0.069	0.13	14.3	**<0.001**	0.48	2.9	0.105	0.27	13	**<0.001**	0.41
SDTotal area	13.3	**0.004**	0.28	6.5	**0.002**	0.69	2.4	0.106	0.11	9.2	**<0.001**	0.37	7.3	**0.014**	0.39	3.8	**0.031**	0.17
SDAcceleration	46.7	**<0.001**	0.76	8.7	**0.008**	0.65	3.1	0.055	0.14	25.9	**<0.001**	0.65	3.2	0.092	0.32	1.5	0.237	0.07

Clinical part	Arm									Trunk								
	Direction			Pain			Direction × pain			Direction			Pain			Direction × pain		
	$F_{2,16}$	P	η^2	$F_{1,16}$	P	η^2	$F_{2,32}$	P	η^2	$F_{2,16}$	P	η^2	$F_{1,16}$	P	η^2	$F_{2,32}$	P	η^2
SDStart pos.	6.4	**0.002**	0.05	0.1	0.747	<0.01	0.7	0.521	0.01	3.4	**0.037**	0.04	1.1	0.303	<0.01	1.6	0.205	0.02
SDROM	8.9	**<0.001**	0.01	0.1	0.967	<0.01	0.4	0.705	<0.01	6.8	**0.002**	0.08	0.9	0.334	0.01	2.8	0.129	0.03
SDTotal area	1.2	0.293	0.02	0.1	0.909	<0.01	0.1	0.949	<0.01	0.8	0.889	0.01	0.1	0.463	<0.01	0.4	0.655	0.01
SDAcceleration	56.4	**<0.001**	0.42	42.8	**<0.001**	0.22	4.7	**0.01**	0.06	50.2	**<0.001**	0.39	44	**<0.001**	0.22	5	**0.008**	0.06

See Figs. 2 and 5 for the results of pairwise comparisons using SNK post hoc tests

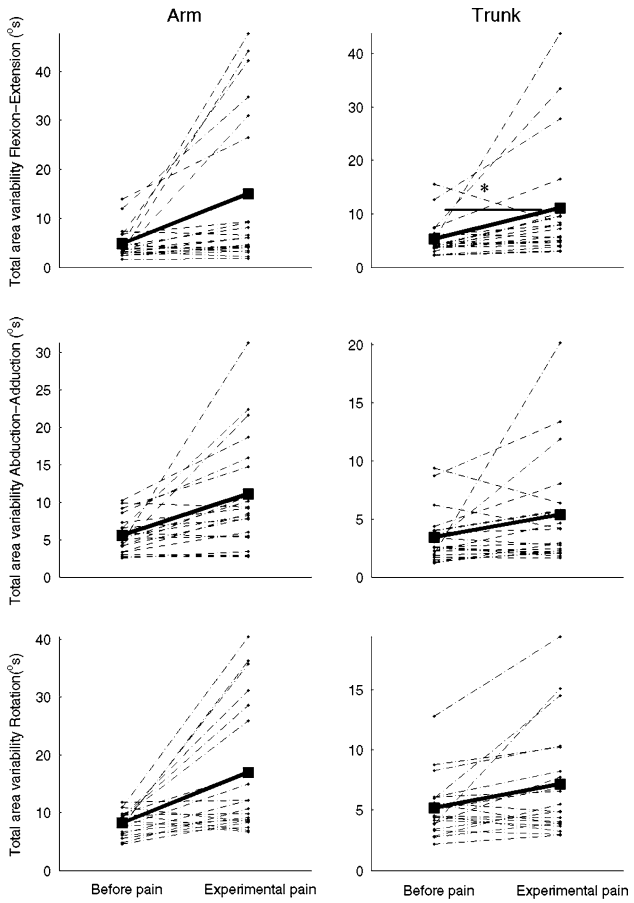

Fig. 2 Variability magnitude (SD) of the total movement area (°s) in the flexion–extension, abduction–adduction and rotation directions before and during acute experimental neck–shoulder pain ($N = 20$). Each *filled diamond* represents the movement variability amplitude of a single subject, while each *filled square* illustrates the group mean. * Indicates a significant difference between pain states ($P < 0.001$, SNK)

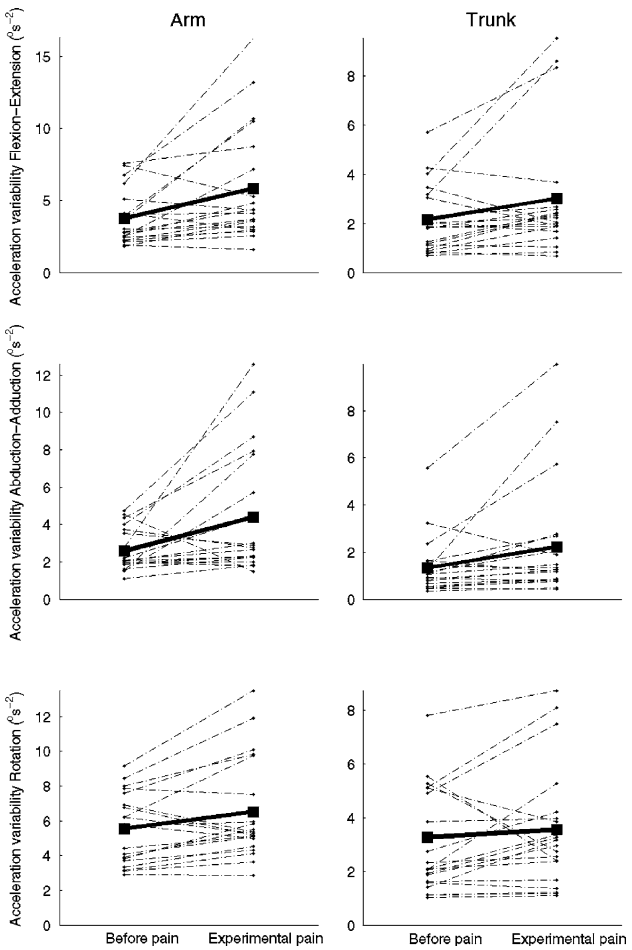

Fig. 3 Variability magnitude (SD) of the movement acceleration (°s^{-2}) in the flexion–extension, abduction–adduction and rotation directions before and during acute experimental neck–shoulder pain ($N = 20$). Each *filled diamond* represents the movement variability amplitude of a single subject, while each *filled square* illustrates the group mean

The standard deviation of the $RMS_{non\text{-}active}$ level was smaller for the patients than for the controls [14.8% (SE 0.9) and 19.3% (SE 1.4), respectively, $P < 0.01$, Table 2]. There was significant interaction between pain status and muscle, the standard deviation of the $RMS_{non\text{-}active}$ was smaller for the patients than for the controls for the infraspinatus muscle (Fig. 4, $P < 0.001$, SNK). The standard deviation of the RMS_{ratio} was smaller for the patients than for the controls [1.01 (SE 0.07) and 1.25 (SE 0.09), respectively, $P < 0.05$].

Discussion

This study investigated the magnitude of cycle-to-cycle variability in task timing, kinematics and muscle activation during repetitive standardised arm movement performed with or without pain in the neck–shoulder region. Acute experimental pain led to an increase in the variability amplitude of the task timing and of several arm and trunk movement parameters. In contrast, the cycle-to-cycle variability amplitude of arm and trunk acceleration, baseline EMG activity and the ratio between EMG during active and non-active task periods were smaller among patients with chronic neck–shoulder pain compared with healthy controls, while the task cycle duration variability was increased.

Methodological considerations

The motor task performed by the subjects was not a carbon copy of a real occupational task, but it simulated generic attributes of tasks found in the meat industry such as

Table 2 Statistical results for the experimental and clinical study parts on the standard deviation (SD) of $RMS_{non\text{-}active}$, RMS_{active}, and RMS_{ratio} ($F_{a,b}$: variance ratio with degrees of freedom, P: level of significance, η^2: partial eta squared)

Experimental part	Muscle			Pain			Muscle × pain		
	$F_{3,19}$	P	η^2	$F_{1,19}$	P	η^2	$F_{3,57}$	P	η^2
$SDRMS_{non\text{-}active}$	7.9	**<0.001**	0.33	3.3	0.085	0.08	3.3	**0.027**	0.14
$SDRMS_{active}$	2.6	0.057	0.27	0.7	0.400	0.10	1	0.389	0.05
$SDRMS_{ratio}$	9.5	**<0.001**	0.89	0.1	0.885	0.01	1.7	0.163	0.09
Clinical part	Muscle			Pain			Muscle × pain		
	$F_{3,16}$	P	η^2	$F_{1,16}$	P	η^2	$F_{3,48}$	P	η^2
$SDRMS_{non\text{-}active}$	1.5	0.227	0.02	7.1	**0.008**	0.04	3.2	**0.024**	0.04
$SDRMS_{active}$	3.8	**0.011**	0.05	2.6	0.111	0.01	1.9	0.125	0.03
$SDRMS_{ratio}$	61.2	**<0.001**	0.47	4.5	**0.035**	0.02	0.4	0.729	<0.01

See text or Fig. 4 for the results of the pairwise comparison with SNK post hoc test

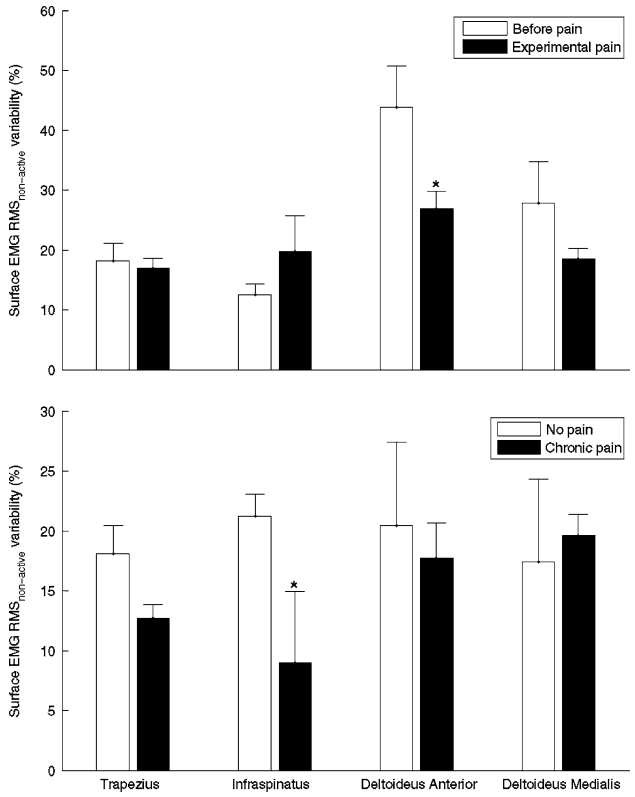

Fig. 4 Variability magnitude (SD) of the normalised surface electromyographic (EMG) root mean square (RMS) values (%) during the non-active part of the task before and during acute experimental neck–shoulder pain (*upper panel*, $N = 20$), as well as for patients with chronic neck–shoulder pain ($N = 12$) and controls ($N = 6$) in the clinical part of the study (*lower panel*). * Indicates a significant difference between conditions or groups ($P < 0.05$, SNK)

short-cycle repetitive movements at a rather constant load. The issue of whether the observed changes in motor patterns during the experimental task would also occur in real meat-cutting is therefore of concern.

The size of the control group in the clinical study was small, but we believe that these controls were representative of healthy butchers. The fact that we found significant differences in the magnitude of motor variability between patients and controls even in this limited material suggests a quite coherent change of motor strategy in response to chronic pain. While the chronic pain and control groups differed slightly in age and work experience, both of which are known to influence motor strategies (Enoka et al. 2003; Madeleine et al. 2003a; Sosnoff and Newell 2006), we judge these differences to be too small to fully account for the motor difference between the groups. In the experimental study, subjects acted as their own controls, and we suggest the response to experimental pain to represent a general change in motor strategy.

We assessed the size of motor variability by the most common measure of statistical dispersion, i.e., standard deviation. Standard deviation or variance of kinematic or kinetic summary measures have been widely used for assessing motor variability due to their simplicity (Chau et al. 2005; Christ et al. 1999; Mathiassen et al. 2003; Moseley and Hodges 2006). However, more advanced measures of the structure of motor variability and its possible change are required for analyzing motor control strategies in depth (Chau et al. 2005; Sosnoff et al. 2006). These methods include, e.g., uncontrolled manifold analyses, approximate or sample entropy, bootstrap prediction bands of register curves, and drift-diffusion techniques (Frank et al. 2006; Latash et al. 2002; Sosnoff et al. 2006; Sosnoff and Newell 2006; van Mourik et al. 2006). The primary purpose of the present study was to present descriptive data to guide further research on pain-related changes in motor variability, and thus we refrained from using these methods at this stage.

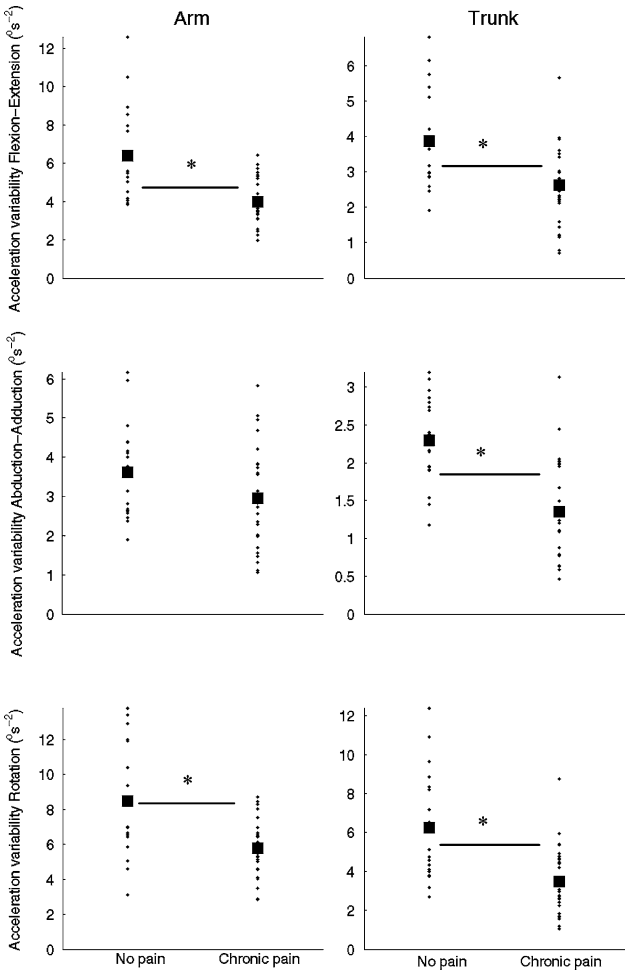

Fig. 5 Variability magnitude (SD) of the movement acceleration ($°s^{-2}$) in the flexion–extension, abduction–adduction and rotation directions for patients with chronic neck–shoulder pain ($N = 12$) and controls ($N = 6$). Each *filled diamond* represents the movement variability amplitude of a single subject (three recordings for each subject), while each *filled square* illustrates the group mean. * Indicates a significant difference between groups ($P < 0.05$, SNK)

Effects of neck–shoulder pain on the size of motor variability

Chronic and acute experimental neck–shoulder pain has been shown to decrease the rhythm of the present experimental task, which suggests a protective response (Madeleine et al. 1999, 2003b). In the present study, increases in the variability amplitude of the durations of task events and the entire cycle were found in presence of acute experimental and chronic neck–shoulder pain, respectively. The latter result, which contradicts our hypothesis of decreased variability in chronic pain, is likely to be explained by the concomitant decrease in rhythm observed in both acute and chronic pain conditions. Unilateral experimental pain in the neck–shoulder region caused bilateral changes in the realisation of the motor task since the durations of both left and right hand movements were affected. This is in line with previous results indicating that supraspinal and/or spinal mechanisms may mediate a spread of motor effects to the contralateral pain-free body side through, e.g., reflex mediated pathways (Madeleine et al. 1999; Thunberg et al. 2002).

The starting position and movement amplitude of the arm and trunk have been found to increase during acute experimental pain, suggesting a positive feedback loop that could explain the transition to a chronic stage (Madeleine et al. 1999). For the arm, chronic neck–shoulder pain has lead to increased movement amplitude, while a decrease has been found among workers with sub-chronic pain (Madeleine et al. 1999, 2003b). For the trunk, the opposite pattern was observed. According to the present results, the magnitude of arm and trunk motor variability increased in the experimental pain condition while a decrease was found with chronic pain. A decreased arm and trunk movement variability amplitude is in line with recent results obtained at an early sub-chronic stage (Madeleine et al. 2007). Thus, our results support that regional motor behaviour differs with pain at different stages and/or different origins.

Muscle pain has also been shown to influence muscle synergies in the neck–shoulder region in previous analyses of the present and other materials (Falla et al. 2006; Madeleine et al. 1999). Experimental neck–shoulder pain appears to cause a decreased level of activity during both active and non-active parts of the task, which can be interpreted as a protective adaptation (Lund et al. 1991; Madeleine et al. 1999). At sub-chronic or chronic neck–shoulder pain stages, the level of EMG activity has, however, been reported to be higher than if pain is not present (Madeleine et al. 1999, 2003b; Veiersted et al. 1990). In the present study, the variability amplitudes of the non-active EMG level and the EMG ratio were smaller in chronic pain than without pain, which is in line with results from sub-chronic pain (Madeleine et al. 2007). Thus, the pain adaptation model (Lund et al. 1991) is more consistent with our results during experimental pain, while theories predicting muscular hyperactivity (Johansson and Sojka 1991; Schmidt et al. 1981; Travell et al. 1942) are better supported at chronic pain stages. The present effects of neck–shoulder pain on the degree of EMG variability indicate a dynamic reorganisation of muscle activity under painful conditions, and that new modulated synergies develop as pain changes from acute to chronic stages.

Motor variability amplitude and the risk of developing musculoskeletal disorders

In the present study, the magnitude of motor variability increased during acute experimental pain. This is consistent

with the notion that the pain presented thoroughly new conditions for performing the task. In this case, motor patterns are adjusted by the central nervous system on the basis of integration and adaptation of sensory information so as to improve performance with respect to the goals set by the task. This is obtained by engaging and testing motor programs involving new muscle synergies (Kargo and Nitz 2003). During the acute experimental pain period, the central nervous system is presumably still in an experimenting state, and has not yet settled on a tuned strategy taking into account the influx of nociceptive input. Motor skill learning has been suggested to follow "the principle of abundance" in which redundant degrees of freedom are not decreased to a minimum but used by the central nervous system to ensure flexible and stable motor performance (Latash and Anson 2006). Thus, while motor variability would be particularly high in the learning phase, some will remain even in a fully known context. Some of this variability can be seen as "bad" in the sense that it affected performance to the worse (Latash and Anson 2006). On the other hand, it may also contain "good" components in the sense that it facilitates the return to a normal motor strategy after experimental pain, as suggested in a clinical study of postural strategies in the back (Moseley and Hodges 2006).

At a chronic pain stage, the magnitude of variability decreased compared with healthy controls. Whether this is a long-term adaptation to the pain state per se, or a sign that workers with less variable motor patterns are more at risk for developing pain than those with a larger variability cannot be inferred from cross-sectional study like this one. However, the latter "prognostic" hypothesis is supported by the finding that experienced, pain-free butchers show a larger motor variability than inexperienced controls (Madeleine et al. 2007). It is also supported by a few studies suggesting positive effects of a larger motor variability on the development of fatigue (Farina et al. 2006; Madeleine and Farina 2007; van Dieen et al. 1993) and neck–shoulder disorders (Kilbom and Persson 1987).

Conclusion

The amplitude of motor variability during a standardised repetitive motor task increased during acute experimental pain compared with before pain while it decreased among patients with chronic neck–shoulder pain compared with healthy controls. The present results support that variability is an important element in motor control and provide a possible general transition path from acute towards chronic pain stages: acute pain would lead the central nervous system to search for the least painful biomechanical solution, while at chronic stage the chosen solutions would be characterised by a reduced flexibility of the motor system.

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