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Effects of experimentally induced pain and fear of pain on trunk coordination and back muscle activity during walking

Claudine J.C. Lamoth [a,b,*], Andreas Daffertshofer [a], Onno G. Meijer [a], G. Lorimer Moseley [c], Paul I.J.M. Wuisman [b], Peter J. Beek [a]

[a] Faculty of Human Movement Sciences, Vrije Universiteit, Van der Boechorststraat 9, 1081 BT Amsterdam, The Netherlands
[b] Department of Orthopedic Surgery, Medical Center Vrije Universiteit, Amsterdam, The Netherlands
[c] Department of Physiotherapy, Royal Brisbane Hospital and The University of Queensland, Brisbane, Australia

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Abstract

Objective. To examine the effects of experimentally induced pain and fear of pain on trunk coordination and erector spinae EMG activity during gait.

Design. In 12 healthy subjects, hypertonic saline (acute pain) and isotonic saline (fear of pain) were injected into erector spinae muscle, and unpredictable electric shocks (fear of impending pain) were presented during treadmill walking at different velocities, while trunk kinematics and EMG were recorded.

Background. Chronic low back pain patients often have disturbed trunk coordination and enhanced erector spinae EMG while walking, which may either be due to the pain itself or to fear of pain, as is suggested by studies on both low back pain patients and healthy subjects.

Methods. The effects of the aforementioned pain-related manipulations on trunk coordination and EMG were examined.

Results. Trunk kinematics was not affected by the manipulations. Induced pain led to an increase in EMG variability and induced fear of pain to a decrease in mean EMG amplitude during double stance.

Conclusions. Induced pain and fear of pain have subtle effects on erector spinae EMG activity during walking while leaving the global pattern of EMG activity and trunk kinematics unaffected. This suggests that the altered gait observed in low back pain patients is probably a complex evolved consequence of a lasting pain, rather than a simple immediate effect.

Relevance

Variability of EMG data and kinematics may explain pain-dependent alterations of motor control, which in turn might contribute to a further understanding of the development of movement impairments in low back pain.
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Keywords: Low back pain; Principal component analysis; Variability; EMG; Trunk coordination, gait; Fear of pain

1. Introduction

Low back pain (LBP) is often accompanied by changes in gait, such as a decrease in comfortable walking speed, step length and swing time (Keefe and Hill, 1985, e.g., Khodadadeh et al., 1988), which may all be related to changes in trunk coordination (Lamoth et al., 2002b; Selles et al., 2001). In healthy subjects, transverse pelvis–thorax coordination gradually evolves from more in-phase (synchronous pelvis and thorax rotations in the same direction) at lower walking velocities toward more antiphase (synchronous opposite rotations) at higher velocities. Persons with chronic LBP may encounter problems in adjusting thorax–pelvis coordination with increasing walking velocity, while at low walking velocities a rather rigid locking between thoracic and pelvic rotations may be observed. In contrast, the amplitude of segment oscillations appears not to be affected by LBP (Lamoth et al., 2002b). It is unknown whether these changes in coordination are accompanied by changes in lumbar erector spinae (ES) activity.

* Corresponding author. Address: Faculty of Human Movement Sciences, Vrije Universiteit, Van der Boechorststraat 9, 1081 BT Amsterdam, The Netherlands.
E-mail address: c_lamoth@fbw.vu.nl (C.J.C. Lamoth).

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Several studies have investigated the effect of nociception on motor control, and both increases and decreases in muscle activity have been reported (Van Dieën et al., 2003). Patients with chronic LBP may alter the neuromuscular control of gross motor activities such as locomotion, by way of "protective guarding" or "splinting" (e.g., Ahern et al., 1990; Marras and Wongsam, 1986). The pain-adaptation model (Lund et al., 1991) posits that activity of the agonist is inhibited and that of the antagonist augmented to minimize movement of the painful segment. This model received support from several studies in which acute pain was induced by intra-muscular injection of hypertonic saline. For instance, experimentally induced LBP caused increased activity of the lumbar ES muscle during the swing phase and decreased activity during double stance when walking, conform to the patterns of muscle activity observed in LBP patients (Arendt-Nielsen et al., 1996). Furthermore, experimentally induced LBP caused a reduction in trunk acceleration (Moe-Nilssen et al., 1999).

Lamoth et al. (2002b) and Selles et al. (2001) hypothesized that changes in trunk coordination during gait in chronic LBP patients may be aimed at splinting the lumbar spine in order to avoid or reduce pain during walking. However, when this strategy persists beyond the acute phase, it may sustain pain or lead to new complaints. Current models of chronic pain behaviour propose that fear of pain or (re)injury is sometimes more important than pain intensity itself and may contribute to the emergence or preservation of motor impairments in LBP (Vlaeyen et al., 1995; Vlaeyen and Linton, 2000). On this perspective, fear-avoidance beliefs are assumed to prevent regain of normal function, promote the development of guarded movements (Main and Watson, 1996), and lead to disability (Vlaeyen and Linton, 2000).

At present, the relationship between changes in coordination patterns and muscle activity observed in LBP patients is not well understood: Do changes in trunk coordination occur directly at the onset of pain and do changes in muscle activity occur simultaneously, or are these changes mediated by the consequences of pain, such as fear? The present study was performed to help resolve these issues by addressing the following questions: (1) Does acute pain elicit changes in trunk coordination that are similar to those observed in chronic LBP patients when walking at different velocities? (2) Does experimentally induced fear of pain in the absence of actual pain bring about changes in trunk coordination?, and (3) Does pain and fear of pain have an impact on ES activity during gait?

To date, most studies of walking in LBP patients have focused on global changes in trunk coordination and electromyographic (EMG) amplitudes, averaged over strides. In the study of motor control, however, it is well recognized that human movement is intrinsically variable, and that studying the variability properties of coordination patterns may provide insight into underlying control structures (e.g., Collins and De Luca, 1994; Hausdorff et al., 1995; Scholz and Schöner, 1999). Similarly, in the study of movement disorders the analysis of within-subject variability has received considerable attention in recent years, both for diagnostic purposes and with the aim to gain fundamental insights into pathologic motor behaviour (Donker and Beek, 2002; Hamill et al., 1999; Hausdorff et al., 1997; Vogt et al., 2001). The implication of this development for the present study is that it is deemed important to study invariant (global) as well as variable properties of movement patterns and ES activity, which, to anticipate, will be achieved by identifying time resolved changes of the overall pattern over repetitions and to quantify its corresponding variability.

2. Methods

2.1. Subjects

The study was approved by the Ethics Committee of the Medical Center of the Vrije Universiteit. Data were collected from 12 healthy university students with no history of LBP or any other musculoskeletal disorder (4 women, 8 men; mean age 21 years, range 18–25 years). Subjects were paid €45 for their (voluntary) participation. After having been familiarized with the protocol, all subjects signed an informed consent form before participation.

2.2. Experimental design

Prior to the walking experiment subjects completed a modified version of the Tampa Scale for Kinesiophobia (TSK) (Vlaeyen, unpublished report) and the pain catastrophizing scale (PCS) (Sullivan et al., 1995). The original TSK (Vlaeyen et al., 1995) was developed for patients with musculoskeletal pain and included statements such as, "It's really not safe for a person with a condition like mine to be physically active". The modification included a slight change in wording, rendering it applicable to persons without musculoskeletal pain.

The experiment was performed on a treadmill. Before the recording began, subjects walked for a few minutes on the treadmill at different velocities to become familiar with the treadmill and the experimental setup. The experiment consisted of four conditions, which were administered in fixed order, but with the second and third conditions randomised (see below). During each condition, all subjects walked for 4 min at four successive velocities: 2.2, 3.8, 4.6 and 5.4 km/h, 1 min at each velocity level, from which the last 30 s were recorded.

The first condition was normal walking (control condition). Thereafter, in the second and third condition subjects walked after intra-muscular (i.m.) injection of hypertonic saline in the lumbar ES (pain and fear) and after i.m. injection of isotonic saline (fear, no pain).

In human research i.m. injection of hypertonic saline is a standard method for producing acute experimental pain. It has minimal risks and closely simulates clinical musculoskeletal pain. The effect profile is typically one of a rapid onset of a dull, deep pain, which reaches a maximum within 2 min after injection, and slowly fades to baseline over a period of 3–10 min (e.g., Graven-Nielsen et al., 1997b). Isotonic saline is thought not to activate nociceptors and is often used as a placebo (Graven-Nielsen et al., 1997a).

While the subject was standing, a physician injected 0.5 ml of 5% hypertonic or 0.9% isotonic saline using standard sterile procedures. The bolus was delivered over ~10 s, into the right longissimus muscle, ~65 mm lateral to the L3 spinous process, at a depth of ~30 mm. The sequence of i.m. hypertonic and isotonic saline injections was random. Subjects were blinded to the order of injections and told that each injection would produce an immediate and severe pain, pain after some time, or no pain at all. When hypertonic saline was injected, walking trials commenced once the pain was stable for about 30 s. Pain intensity was verbally rated at each walking velocity at 10 s intervals using an 11-point numerical ratio scale (NRS) anchored with ''no pain at all'' and ''very severe pain''. Prior to each injection subjects were asked to rate on two visual analogue scales (VAS, scale 0–100 mm) if they feared the pain induced by the injections (anchored with ''not at all fearful'' and ''extremely fearful'') and how painful they expected the injection to be (anchored with ''not at all'' and ''extremely painful''). After each injection condition, when the data recording of the four velocities was completed, subjects rested until they reported to be completely pain free (between 5 and 10 min). Before the next condition commenced, subjects walked for 5 min on the treadmill at 4.6 km/h to ensure that they were also pain free during walking.

Finally, in the last condition, subjects walked while expecting electric shocks on the skin in the low back area above tolerance (fear of impending pain, no pain). There is some evidence that the motor response associated with fear of pain is influenced by the predictability of its onset (Rhudy and Meagher, 2000). To examine the effect of this confounder, we elicited fear of impending pain by applying unpredictable electric shocks to the skin. Electric cutaneous stimulation produces a sharp, brief and sting-like pain (Crombez et al., 1996; Noteboom, 2000). A pair of surface EMG electrodes was placed on the posterior spina iliaca superior (PSIS) (inter-electrode distance 20 mm). The stimulus (100 ms train, 1 ms pulse duration, 60 Hz) was delivered with increasing intensity and the subject was instructed to indicate when the stimulus became very unpleasant. Subjects were informed that they would receive, at random and without warning, painful shocks that were between 50% and 120% of the intensity previously rated as very unpleasant. Actually, only one stimulus was delivered at each walking velocity and this stimulus was always below 50% of the original intensity. Subjects were invited afterwards to rate how fearful they were of the impending pain using a VAS anchored with ''not at all fearful'' and ''extremely fearful''.

2.3. Recording

Angular rotations of the pelvis and trunk segments were recorded using a 3D active marker movement registration system (Optotrak 3020, Northern Digital™, Waterloo, Ontario, Canada). Clusters of three markers were fixed on a light plate mounted on rigid fixtures and attached to the trunk at the level of Th3, L2 (spinous processi) and the sacrum between PSIS using neoprene bands. These markers defined thoracic, lumbar, and pelvic segments. The fixtures at the level of Th3 and L2 were designed to span the ES muscle and the processus spinae. To detect characteristic moments of the gait cycle, infrared-light emitting markers were placed on the heels and the fifth metatarso-phalangeal joint.

In the area of electrode placement, the skin was shaved and cleaned with alcohol. EMG's were recorded from the left and right ES, at the level of spinous processi Th12, L2 and L4, using pairs of bipolar surface EMG electrodes (AG/AgCl discs, 1 cm diameter, 2 cm inter-electrode distance; Blue sensor N-00-S, Medicotest, Ølstykke, Denmark). Electrodes were placed at a distance of 3 cm lateral to the vertebral column (Vink et al., 1989). At each walking velocity, EMG data were sampled at 1 KHz (Porti5, TMS-international™, Enschede, The Netherlands) and the kinematic data at 100 Hz, both for 30 s. Recording of kinematic and EMG data was synchronized by means of a shared trigger pulse. All raw data were exported for analysis using Matlab 6.12 (Mathworks, Natic, MA, USA). Before each condition, a reference measurement was taken in quiet upright standing position (anatomical position).

2.4. Data-processing

Kinematic data were analysed in a global reference frame in the form of fixed *xyz*-Euclidian coordinates with the *x*-axis corresponding to the line of progression, the *y*-axis perpendicular to the *x*-axis and parallel to the ground and the *z*-axis pointing vertically upward. The three non-collinear cluster markers represented the motion of the trunk segments. From each marker cluster a segment reference frame was defined and transformed to a new coordinate system with the same orientation as

the global coordinate system. Using the reference measurement, segment axes were aligned with the global frame of reference. Angular rotations of trunk segments were obtained from segment angles with respect to the axial in the transverse and frontal plane of motion and calculated as (four quadrant) arctangent, specified by the *xyz*-coordinates of the segment coordinate system. Heel strikes were estimated to have occurred at the minimum in the vertical velocity of the toe marker, and toe-off at the maximum in the vertical velocity of the heel marker (Pijnappels et al., 2001). A stride cycle was defined as the distance between subsequent heel strikes of the same leg.

EMG data were first rectified using the Hilbert transform, which is a common demodulation technique to eliminate irrelevant phase information (Gabor, 1946). Kinematic and EMG data were low pass filtered using a 4th order bi-directional Butterworth filter with a cut-off frequency of 10 and 20 Hz, respectively. For the PCA analysis and to calculate the mean amplitude of ES EMG, using the stride intervals, kinematic and EMG data were split into subsequent time series each containing a single stride cycle. Subsequently, each of these time series was time normalized and re-sampled using a cubic spline interpolation to 0–100% of the stride cycle. For each original kinematic or EMG recording, we generated at least $S \geqslant 15$ "individual" time series of equal length, each containing one stride cycle.

2.5. Coordination between trunk segments

To calculate the relative phase between trunk segments in the transverse plane, we computed the Fourier phase for each time series. To cope with time-dependent changes of the phases, we estimated the Fourier transform within a finite frame of size ε, which was shifted in time. In detail, we computed for a time series $x(t)$

$$x(t) \mapsto x_\varepsilon(\omega,\tau) = \int_{-\infty}^{\infty} x(t) W_\varepsilon(t-\tau) \mathrm{e}^{-\mathrm{i}\omega(t-\tau)}\,\mathrm{d}t$$
$$\text{with } W_\varepsilon(t) = \begin{cases} 1 & \text{for } 0 \leqslant t < \varepsilon \\ 0 & \text{otherwise} \end{cases} \quad (1)$$

and defined the phase as

$$\varphi_\varepsilon(\omega,\tau) = \arctan\left(\Im\{x_\varepsilon(\omega,\tau)\}/\Re\{x_\varepsilon(\omega,\tau)\}\right) \quad (2)$$

We focused on phases at the fundamental movement frequency ω_0 of the proximal segment (Lamoth et al., 2002a) by which the frame size was fixed at twice the corresponding period length $\varepsilon = 2$, $T_0 = 4\pi/\omega_0$. Hence, the relative phase (RP) between two signals $x(t)$ and $y(t)$ was defined as

$$\Delta\varphi_{\omega_0}^{(x,y)}(\tau) = \varphi_{2T_0}^{(x)}(\omega_0,\tau) - \varphi_{2T_0}^{(y)}(\omega_0,\tau) \quad (3)$$

with x, y representing the thoracic and pelvic, thoracic and lumbar or lumbar and pelvic segmental oscillations in the transverse plane. In general, a phase difference of 180° indicates antiphase coordination and 0° in-phase coordination. To evaluate the degree of coupling between thoracic–pelvic, thoracic–lumbar and lumbar–pelvic segmental oscillations in the transverse plane, the corresponding power spectra were correlated using weighted coherence statistics. Weighted coherence summarizes the proportion of shared variances over a specified band of frequencies (Porges et al., 1980). First, power spectra were estimated using Welch's periodigram method. The length of the Hanning window was based on a 0.95% confidence interval with error bounds not exceeding 25% of the estimated spectral power. The weighted coherence was calculated at the frequency band of the fundamental frequency ±0.2 Hz, indicating the strength of coupling normalized to values between 0 and 1.

2.6. Mean EMG activity

Double stance and swing phase ipsi- and contralateral to the right and left heel strikes were detected using the foot contact data. First, for each condition and velocity the rectified and time normalized EMG time series were averaged over strides per EMG recording. Second, the mean EMG amplitude was calculated for different periods in the gait cycle.

2.7. Principal component analyses

Principal component analysis (PCA) was applied to detect similarities and eventual deviations between experimental conditions in trunk coordination and EMG activity patterns and to examine the effect of condition on the variability in the data. In general, PCA is the most generic and efficient method of reducing multidimensional data sets (e.g., Chau, 2001). Used as a data-driven filter, this method allows for a separation of coherent (invariant) patterns of activity (e.g., kinematic and EMG) and more variant aspects within a data set. For an introduction to this specific form of PCA, we refer to Daffertshofer et al. (submitted).

First, stride cycle time series were rescaled to unit variance to eliminate mean amplitude effects. Subsequently, successive stride cycle time series of lumbar ES EMG and segment rotations at all walking velocities and conditions were combined to vectors $\vec{q}(t)$, each representing a new data set. PCA was applied separately to the kinematic data of segmental rotations in the transverse and in the frontal plane as well as to the EMG data of the lumbar ES. For these data sets, the covariance matrices were calculated. Diagonalization of each of these matrices resulted in pairs of eigenvectors and eigenvalues, with the latter representing a measure for the variance of the data along its corresponding

eigenvector (mode). The amount of variance explained by each mode decreases with each subsequent eigenvector. The time evolution along each mode was defined by projection of the original data set onto the mode in question. The number of principal modes that represented the coherent signal structures in the time series was determined by visual inspection of the eigenvalue spectra, using discontinuities in the eigenvalue spectra as cut-off criterion, and by examining its corresponding time series. Thereafter, these global signal forms $\vec{q}^{(\text{global})}(t)$ were subtracted from the data, which was realized by projecting the data onto all the remaining modes. Thus, we obtained the residual pattern $\vec{q}^{(\text{residual})}(t)$, constituting the residual variance. Stated formally, we rewrote the data as $\vec{q}(t) = \vec{q}^{(\text{global})}(t) + \vec{q}^{(\text{residual})}(t)$ and analysed $\vec{q}^{(\text{global})}(t)$ and $\vec{q}^{(\text{residual})}(t)$ in terms of their variance. For further details, we refer to Appendix A. In fact, the eigenvectors were defined by the entire data set, that is, for all conditions, velocities and segment rotations or EMG recordings, which allowed for an examination of the variability of the global and residual patterns separately for each condition. First, to identify eventual differential effects of condition on individual ES EMG (m) or segment rotations (r), the variance of $q^{(\text{global})}_{c,v,m \text{ or } r,s}(t)$ and $q^{(\text{residual})}_{c,v,m \text{ or } r,s}(t)$ was examined, that is, along time series of each condition ($c = 1, \ldots, 4$) and velocity ($v = 1, \ldots, 4$) of individual EMG ($m = 1, \ldots, 4$) or segment rotations ($r = 1, 2, 3$) during single strides cycles ($s = 1, \ldots, S$). To relate the variance of pain and fear conditions to the control condition, we computed for each experimental condition the ratio between the mean SD and that of the control condition (Appendix, Eq. (A.10)). This resulted in the indices $R^{(\text{global})}_{v,m \text{ or } r}$ and $R^{(\text{residual})}_{v,m \text{ or } r}$ for the pain and fear of pain conditions. Second, for each pattern the variance over time series of all segment rotations and the EMG of all lumbar ES recordings was calculated for each velocity and condition, again with $R^{(\text{global})}_{v}$ and $R^{(\text{residual})}_{v}$ being defined as the mean SD of the pain and fear condition divided by that of the control condition (Appendix, Eq. (A.11)).

2.8. Statistics

For each continuous relative Fourier phase (RP), per condition, subject and velocity, the mean RP and its intra-individual variance was calculated using circular statistics. The circular variance was transformed into a linear 'standard deviation' (SD) that could be subjected to statistical tests based on standard normal theory such as ANOVAS (Mardia, 1971).

Dependent variables were means and SDs of the relative phases, the coupling between thoracic and pelvic, thoracic and lumbar, and lumbar and pelvic transverse oscillations and the mean EMG amplitude of the ES recordings around double stance and during ipsi- and contralateral swing phases. Separate ANOVA with repeated measures were performed on those dependent measures using a factorial design involving the factors condition (4 levels) and velocity (4 levels). The differences between the control and experimental conditions were evaluated using a post hoc ANOVA with the factors condition (2 levels) and velocity (4 levels). If no significant effect of velocity was found, the velocity trials were combined. To examine differences between conditions with respect to ratios of global and residual patterns, ANOVAS were performed with the within-factor condition. To differentiate between conditions, post hoc pair-wise analyses were applied with Bonferroni corrections for multiple comparisons.

The relationships between pain intensity scores and relative phases, coupling values, mean EMG amplitude and ratios were examined using Spearman correlations. For all statistical analyses P-values < 0.05 were considered to be significant.

3. Results

The average TSK and PCS scores were 38 (SD, 5.6) and 18 (SD, 9.0), respectively, which are within the normal range. The mean maximum pain reported, after i.m. injection of hypertonic saline, was 6.1 (SD, 1.9). In all subjects, initial pain was at least 3. In 10 subjects, pain remained above this level throughout all velocity trials, whereas in 2 subjects it decreased to 2 at the highest walking velocity. Pain spread out from the injection point toward the entire right lumbar ES without radiation. Thoracic ES was never described as painful. Within 5–10 min after hypertonic saline injection, all subjects were pain free. No subject experienced pain levels greater than 2 after the injection of isotonic saline.

Fear of pain was induced during both injection conditions, indicated by a mean VAS of 54 mm (SD, 18) prior to hypertonic saline injection and of 50 mm (SD, 17) prior to isotonic saline injection. The injection sequence did not affect the VAS scores. Subjects reported less fear for the electric shock condition than for the injection conditions (mean VAS score of 38 mm; SD, 13). All subjects expected both injections to be moderately painful and reported on average a VAS score of expected pain of 63 mm (SD, 15) and 60 mm (SD, 17), before isotonic and hypertonic saline injection, respectively.

3.1. Relative phase and coupling

In all conditions, thoracic–pelvic, thoracic–lumbar, and lumbar–pelvic mean RP increased with increasing walking velocity. The effect of walking velocity was

Table 1
The effects of velocity and experimental condition on the relative phases (repeated measures ANOVA)

Relative phase	Velocity		Condition		Interaction	
	$F_{(3,33)}$	P	$F_{(3,33)}$	P	$F_{(9,99)}$	P
Thorax–pelvis						
Mean	28.14	<0.01	0.14	ns	3.07	ns
Intra-individual SD	1.04	ns	1.78	ns	0.92	ns
Thorax–Lumbar						
Mean	15.82	<0.01	0.41	ns	0.31	ns
Intra-individual SD	18.00	<0.01	0.82	ns	0.99	ns
Lumbar–pelvis						
Mean	5.69	<0.01	0.47	ns	0.74	ns
Intra-individual SD	0.86	ns	1.96	ns	0.54	ns

ns = not significant.

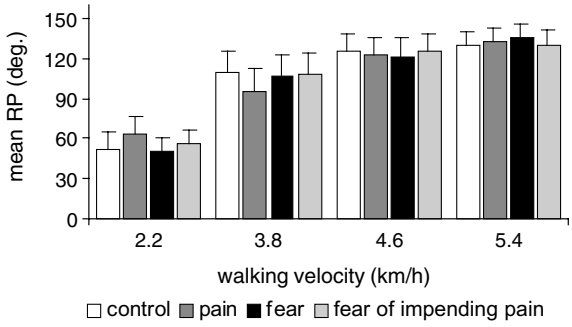

Fig. 1. Group average of the mean relative phase (mean RP) between thoracic and pelvic transverse rotations for the control, pain and fear conditions. Error bars indicate SE.

significant, while that of condition and their interaction were not (Table 1). With increasing velocity mean thoracic–pelvic RP increased from more in-phase (mean, 57°; SD, 38°) to more antiphase (mean, 133°; SD, 35°) (Fig. 1), whereas mean thoracic–lumbar RP increased from 8° (SD, 5°) to 50° (SD, 37°) and mean lumbar–pelvic RP from 45° (SD, 34°) to 63° (SD, 40°) and thus remained more or less in-phase. Only the intra-individual standard deviation of the thoracic–lumbar RP increased significantly from 2° (SD, 3°) to 12° (SD, 13°) with increasing velocity. There were no main significant effects of condition on the intra-individual standard deviations of the RP's, nor significant velocity by condition interactions.

No significant velocity effect was found on the intersegmental couplings. Moreover, pain or fear of pain had no significant effects on the coupling between segments, nor was there a significant velocity by condition interaction.

3.2. Kinematic global patterns and variability

The eigenvalue spectra of the kinematic angular data revealed that averaged across subjects the first three principal modes covered almost the entire variance of the kinematic data set, that is, 95% (66% + 24% + 5%) and 94% (57% + 31% + 6%) for the segment rotations in the transverse and frontal plane, respectively. Analysis of the coefficients of the eigenvectors revealed an almost equal contribution of each experimental condition to the modes, implying that the experimental conditions had no effect on the global kinematic patterns in both planes (see Fig. 2 for an example from one subject).

The variability of the time resolved patterns of the individual segment rotations and averaged over segments were at all walking velocities almost similar in the

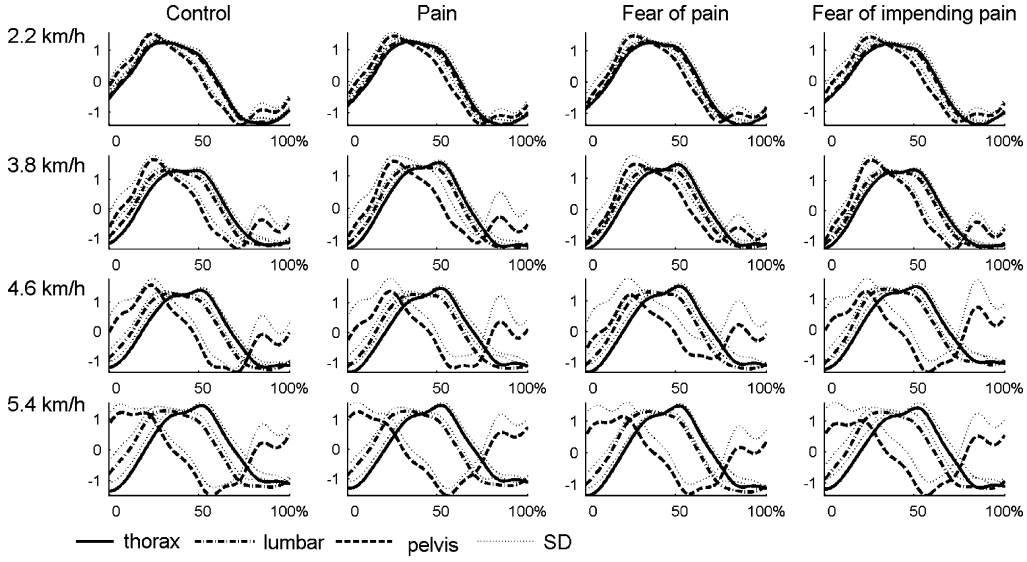

Fig. 2. Typical example of one subject of the global pattern (averaged over strides) of thoracic, lumbar and pelvic angular rotations in the transverse plane for each walking velocity for control, pain and fear of pain conditions. The x-axis represents the time normalized stride cycle, and the y-axis the amplitude normalized angular rotations in degrees.

pain, fear, and control conditions, as was evidenced by $R_{v,r}^{(\text{global})}$ and $R_{v}^{(\text{global})}$ values of almost 1. The residual variance constituted only 5% of the data, again with $R_{v,r}^{(\text{residual})}$ and $R_{v}^{(\text{residual})}$ approaching 1, at all walking velocities (range 0.99–1.1; SD, 0.09–0.16) for both transverse and frontal segment rotations. Hence, no significant effect of condition was present.

3.3. Effect of velocity and experimental condition on mean EMG amplitude

In all conditions, the mean amplitude of the lumbar ES EMG activities decreased significantly with increasing walking velocity in the ipsi- and contralateral swing phase, whereas velocity had no significant effect on mean amplitude of the ES EMG around double stance. A significant effect of experimental condition on the mean amplitude was found for all lumbar ES EMG in the ipsilateral swing phase, and in the contralateral swing phase for right L2 ES EMG activity (Table 2). Velocity by condition interaction was significant for right L2 ES EMG due to an increase in activity in the pain condition from 2.2 to 3.8 km/h. During double stance, a significant effect of condition was found for left L2 and right L4 ES EMG. No significant effect of experimental condition was found on the thoracic part of the ES during the different phases of the stride cycle.

Post hoc pair-wise analysis revealed that when pain was present, the mean EMG amplitude was significantly higher during the swing phases than in the control condition, for the right ES recordings during ipsilateral and contralateral swing phases, and for the left ES recordings during the ipsilateral swing phase. In contrast, EMG amplitude in both fear conditions did not differ significantly from that in the control condition (Table 2).

Around double stance, post hoc pair-wise comparisons between the control and experimental conditions revealed a significant decrease in amplitude for both L2 ES EMG during fear elicited by injection of isotonic saline, and for the left L2 and the right L4 during fear elicited by electric shock. Pain did not lead to a significant decrease in the mean amplitude compared to the control condition. Since the mean thoracic activity was

Table 2
The effects of walking velocity and experimental condition on mean EMG amplitudes during swing phases and double stance, and significant effects of the post hoc analysis

Mean ES EMG amplitude	Repeated measures ANOVA				Post hoc analyses		
	Velocity		Condition				
	$F_{(3,33)}$	P	$F_{(3,33)}$	P	Condition	$F_{(1,11)}$	P
Ipsilateral swing phase							
Left							
Th12	9.2	<0.001	0.3	ns			
L2	45.2	<0.001	3.6	0.02	Pain	6.5	0.03
L4	31.6	<0.001	3.4	0.03	Pain	9.6	0.01
Right							
Th12	15.9	<0.001	0.5	ns			
L2	37.9	<0.001	4.2	<0.01	Pain	7.5	0.02
L4	23.4	<0.001	3.3	0.03	Pain	19.4	<0.01
Contralateral swing phase							
Left							
Th12	2.1	ns	2.0	ns			
L2	8.5	<0.001	2.2	ns			
L4	4.8	<0.01	1.0	ns			
Right							
Th12	7.0	<0.001	4.9	ns			
L2	3.7	<0.02	5.3	<0.01	Pain	4.7	0.04
L4	12.1	<0.001	1.0	ns	Pain	7.5	0.02
Double stance							
Left							
Th12	0.7	ns	1.4	ns			
L2	1.0	ns	4.0	0.02	$Fear^{+}$	4.0	0.02
					$Fear^{++}$	20.1	<0.01
L4	1.8	ns	1.5	ns			
Right							
Th12	2.8	ns	0.3	ns			
L2	0.7	ns	0.7	ns	$Fear^{+}$	4.8	0.04
L4	0.7	ns	3.4	0.03	$Fear^{++}$	7.2	0.02

ns = not significant.
$Fear^{+}$ = isotonic saline injection (fear of pain); $Fear^{++}$ = electric shocks (fear of impending pain).

not significantly affected by condition, these signals were not included in the subsequent PCA.

3.4. Global EMG patterns and variability

Visual inspection of the time series revealed a global pattern over stride cycles, which was remarkably consistent over conditions: two main bursts of activity per stride cycle, occurring at, or immediately following heel contacts, and reduced activity between the two bursts. However, particularly during pain, the lumbar ES EMG activities were also more variable in terms of irregular phase shifts and the presence of additional frequencies (Fig. 3). PCA was applied to separate these coherent patterns of activity from the more variant aspects and to quantify both.

Combining all conditions averaged over subjects, the first three principal modes covered about 54% (SD, 15%) of the variance (34% + 13% + 7%), and comprised the global coherent pattern. When examining individual lumbar ES EMG recordings, the variability of the global patterns was across all walking velocities about the same in pain, fear and control conditions as was evidenced by $R_{v,m}^{(\text{global})}$ values approximating 1. Since velocity did not have a significant effect on the variability of the global and residual patterns, EMG data were collapsed over velocities. Only for right L2 ES EMG activity a significant effect of condition was found ($F_{2,94} = 6.97$, $P < 0.01$). Post hoc pair-wise comparisons revealed that this effect was due to a higher variability of the global pattern of right L2 in the control condition than in the pain condition. The variability of the residual pattern ranged from 1 to 1.22. A significant effect of condition was found for $R_{v,m}^{(\text{filtered})}$ of right and left L2 ES EMG activities ($F_{2,94} = 8.4$, $P < 0.01$; $F_{2,94} = 4.1$, $P = 0.02$) and right L4 ES EMG activity ($F_{2,94} = 3.7$, $P = 0.03$). Post hoc analysis showed that pain resulted in a significant increase in variability in the lumbar ES EMG activities compared to the control condition, whereas fear of pain did not.

For all lumbar ES EMG activities combined, and at all velocities, $R_v^{(\text{global})}$ was < 1.01. Comparisons between the ratios revealed a significant effect of condition ($F_{2,94} = 4$, 13, $P = 0.02$), due to the higher variability of right L2 in the control condition. The variability of the residual pattern was affected by condition ($F_{2,94} = 6.32$, $P < 0.01$). Post hoc comparisons revealed that the variability was larger in the pain condition showing a significant difference between $R_v^{(\text{residual})}$ of the pain–control condition and $R_v^{(\text{residual})}$ of the fear–control conditions

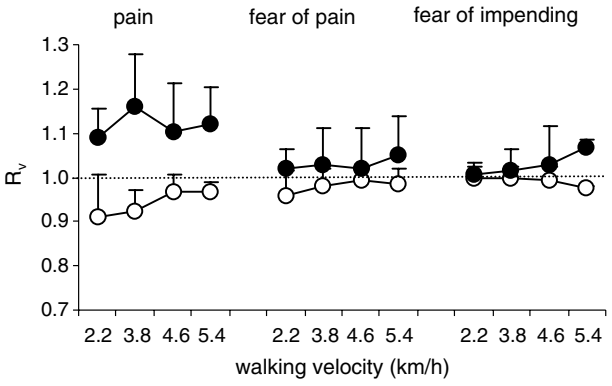

Fig. 4. The effect of pain and fear of (impending) pain on the variability of the residual and global patterns of ES EMG activities of all lumbar recordings. The variability, averaged over subjects, was calculated as the SD of the residual or global pattern of each experimental condition divided by that of the control condition (R_v). Open markers represent the variability of the global pattern, closed markers that of the residual pattern of EMG activity. Error bars indicate SD.

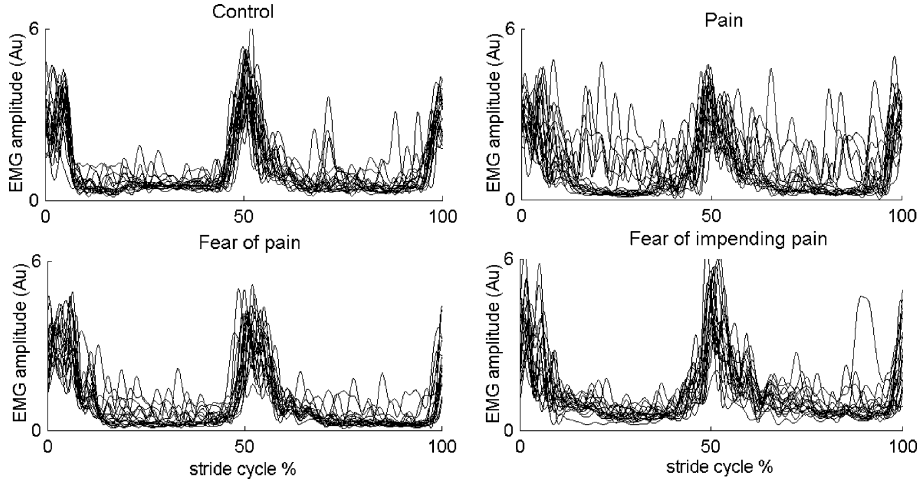

Fig. 3. Left lumbar ES EMG activity of individual stride cycles recorded at a walking velocity of 3.8 km/h of one subject for the control condition (upper left panel), the pain condition (upper right panel), the isotonic induced fear condition (lower left panel) and the electric shock fear condition (lower right panel). The *y*-axis values are given in arbitrary units (Au), because in this figure the EMG profiles are normalized with regard to amplitude.

($P < 0.02$ for both). In contrast, the variability of both fear of pain conditions was almost equal to that of the control condition (range mean ratio 1–1.04) (see Fig. 4).

3.5. Pain and fear of pain

For the pain condition a significant correlation was found between higher self-reported pain intensities and increased mean EMG amplitude in the ipsilateral swing phase of left and right L2 recordings, however, the correlation was relatively low (Spearman's rho = 0.4; $P < 0.05$). The correlation between pain intensity and residual variance approached significance (Spearman's rho 0.32; $P = 0.054$).

4. Discussion

To gain insight into the development of motor impairments in chronic LBP, we examined the impact of induced pain and fear of pain on trunk coordination and ES EMG during walking at different velocities. We focused on the effect of pain and fear of pain on (i) the global coordination between trunk and pelvis rotations in the transverse plane and the average amplitude of ES activity during different phases of the stride cycle, and on (ii) invariant and variant properties of trunk kinematics and ES EMG activity.

The results of the kinematic analyses did not indicate any significant effect of induced pain or fear of pain on the relative timing between segment rotations. Across all pain-related conditions, increases in walking velocity induced larger phase differences between thoracic–lumbar and lumbar–pelvic rotations and a gradual change from in- to antiphase thoracic–pelvic coordination. Over strides, about 95% of the variance was explained by the first three principal components, indicating the presence of consistent global patterns in the transverse and frontal plane across conditions. The global pattern largely corresponded to sinusoidal displacement patterns of the trunk and pelvis. Thus, trunk coordination during walking appeared to involve rather steady coordination modes that were not affected by induced pain or fear of pain. As a consequence, overall variability of global and residual patterns did not differ significantly between conditions. Moe-Nilssen et al. (1999) found a small effect of induced pain, but they only examined trunk accelerations at customary walking velocities rather than inter-segmental coordination.

In contrast to the kinematics, significant changes in lumbar ES EMG activity were observed during pain. During acute pain, a reduced modulation depth was found and, especially during the swing phase, EMG amplitudes were elevated. In general, these results are consistent with those of previous studies (e.g., Arendt-Nielsen et al., 1996), except that we found no significant decrease in amplitude during double stance. Unlike pain, fear of pain did not significantly affect the mean EMG amplitude during the swing phase, but rather resulted in a decrease in EMG amplitude during double stance.

PCA revealed that in all conditions, and at all velocities, coordinated activity of lumbar ES was remarkably consistent over strides, with peaks of activity of the lumbar ES around foot strike. After subtracting these common invariant features (global pattern) from the EMG time series, it was found that the remaining residual variability of the lumbar ES was greater during pain than during control and fear conditions at all velocities. In fact, the observed increase in mean lumbar ES activity during the swing phase may have resulted from these modifications, rather than being a simple amplitude effect. The increased variability due to pain was caused by additional irregular timing deficits as well as the presence of additional frequency components. In contrast, fear of pain did not lead to a noticeable increase in the variability of the residual pattern.

Using PCA as a data-driven filter, we identified invariant patterns of lumbar ES activity as well as small but significant modifications therein, which would have remained undetected when the analysis would have focused solely on the global pattern of coordination of ES activity, as in standard applications of PCA. In so doing, we established that the various fear conditions mainly led to a more "careful" style of walking. We found no pronounced correlations between pain intensity and ES EMG changes, which may have been due to large inter-individual differences in rating (see also Arendt-Nielsen et al., 1996; Moe-Nilssen et al., 1999).

The results do not support our initial hypothesis that during acute pain subjects adapt trunk coordination in terms of "splinting" behaviour to avoid pain provocation by walking. In addition, we found no support for the suggestion that fear of pain leads to guarded movements during walking and elicits similar changes in motor control as reported for clinical low back pain (Lamoth et al., 2002b).

The latter finding suggests that in patients modifications in trunk coordination during walking do not arise immediately in the acute stage but may be preceded by increased EMG variability. Accordingly, our results do not support the proposal that pain merely results in either an increase or a decrease of amplitude by way of a purely protective response (Arendt-Nielsen et al., 1996; Lund et al., 1991). Kinematics remained unaffected in the present study, whereas for the EMG the PCA showed that besides amplitude effects, changes in the frequency content and timing deficits over the stride cycle occurred. This was caused by an erratic activation pattern of the residual pattern of lumbar ES EMG during pain (see Fig. 3). Along similar lines, Zedka et al.

(1999) examined voluntary extension-flexion movements of the trunk after pain induced by hypertonic saline and found reduced lumbar ES EMG modulation depth associated with slower and reduced range of movement. Notably, these EMG changes were preserved when movements were corrected for speed and amplitude. Both that data and the present results suggest that modifications in EMG reflect more than just a strategy to minimise movement (and reduce pain). Consistent with these findings, a number of studies on the effect of low back pain have showed that changes in muscle activity may serve another goal than limiting the movements of the spine, without having a direct relation to pain intensity. In a recent review by Van Dieën et al. (2003), it was concluded that changes in motor control in low back pain depend on the prevailing task and are thus highly task specific.

In a similar vein, a possible explanation for the unaltered trunk coordination during pain might be that task constraints during walking are quite severe (Donker et al., 2002). For instance, in normal gait, within one gait cycle the pelvis rotates around the stance leg with the side of the swing leg moving forward, contributing to lengthening of the step, which occurs at walking velocities higher than about 3.0 km/h (e.g., Nottrodt et al., 1982). To keep the time-averaged total rotational impulse of the body zero, pelvic rotations must be counterbalanced, which requires coordination between pelvic and thoracic rotations. Accordingly, an important contribution of the ES during walking is the maintenance of dynamic equilibrium (e.g., Thorstensson et al., 1982; Waters and Morris, 1972). Thus, the observed increase in variability in lumbar ES EMG activation may be interpreted as a neuromuscular strategy to maintain trunk coordination during walking under changing circumstances.

In the present study, changes in neuromuscular control due to acute induced pain were found to be limited to an increase in variability of the residual pattern, leaving the global patterns of lumbar ES EMG activity and trunk kinematics intact. This suggests that the coordinated activity of ES, the on- and off-set with regard to specific phases of the stride cycle, is crucial to inter-segmental (thoracic, lumbar and pelvic) coordination in the transverse plane. Unlike induced pain, low back pain may result in poorly coordinated activity of the ES. Perhaps for low back pain to affect trunk coordination, it must first have become chronic and brought about long-term changes in neuromuscular functioning. Thus, the results of the present study suggest that the altered gait observed in low back pain patients is a complex evolved consequence of pain and not a simple, immediate effect, while fear of pain is probably not a major factor in gait adaptation.

For a proper appreciation of the latter conclusion, some limitations of the present study should be discussed. First, a limitation of induced pain by injection of hypertonic saline is that pain levels do not stay constant, but slowly fade away. However, pain remained stable within one recorded velocity level and a significant effect on ES EMG activity was found at all velocity levels.

Second, the participants of this study were younger than most patients with low back pain (mean age of 21 years, range 18–25 years). Arendt-Nielsen et al. (1996), however, found similar effects of induced pain in a group of healthy subjects with a mean age of 25 years as in a group of individuals with chronic low back pain with a mean age of 40 years.

Third, the experimental paradigm used has certain limitations in generalizing the results to clinical low back pain. One limitation is that we did not elicit genuine fear of injury as addressed in the fear-avoidance theory. This type of problem presents a paradox to all experimental pain research as ethical and practical constraints on eliciting pain and fear of pain are such that subjects know beforehand that pain will end soon, will not get worse in the long term and will not lead to damage. These limitations may have influenced the effect of fear of pain in our experiment. However, the key component of the fear-avoidance model is fear related to bodily threat and our experimental protocol was effective in eliciting this, at least in that the subjects showed fear on the VAS scores. Using a similar experimental paradigm to elicit fear of pain during voluntary limb movements, Moseley et al. (2002) found that fear of impending pain caused disruption of postural activation of the deep trunk muscles. Moreover, the effect was different to that induced by experimental pain.

Finally, it has been proposed that uncertain painful events induce anxiety, whereas certain painful events induce fear (Rhudy and Meagher, 2000). Therefore, our paradigm may have elicited anxiety even though subjects reported being fearful. This may represent a shortcoming of the experimental protocol, or, alternatively, it may reflect a difficulty in distinguishing between fear and anxiety. Either way, although this finding implies doubts about unpredictable and predictable pain, the main results of the study were not affected. To examine if fear of pain was influenced by the predictability of its onset, two fear conditions were applied and no major differences were found between them.

5. Conclusions

The timing between trunk segments and global patterns of angular rotations of the trunk were not affected by induced pain or fear of pain, whereas pain caused changes in lumbar ES EMG activity. These changes in muscular control were limited to an increase in variability of the residual pattern, leaving the coordinative structure of lumbar ES activity and thus the trunk

kinematics intact. Like induced pain, induced fear of pain led to subtle rather than salient changes in coordination in that fear of pain appeared to induce a more careful style of walking. This suggests that the altered gait observed in low back pain patients is probably a complex evolved consequence of a lasting pain, rather than a simple immediate effect.

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Appendix A

Each data set was rewritten as an N-dimensional vector

$$\vec{q}(t) = [q_1(t), q_2(t), \ldots, q_N(t)]^{\mathrm{T}} = \sum_{k=1}^{N} q_k(t)\vec{e}^{(k)} \tag{A.1}$$

where N is the number of stride cycle time series and $\vec{e}^{(k)}$ denotes the kth unit vector. In general, PCA removes redundancies in multidimensional data by recasting them into a set of $M < N$ vectors or modes $\vec{v}^{(k)}$ (see Daffertshofer et al. (submitted) for a detailed explanation). The criteria for determining these modes is given via the least square error, which can be reflected by the covariance matrix $\{\mathrm{Cov}_{ij}\}$ whose coefficients read

$$\mathrm{Cov}_{ij} = \left\langle [q_i(t) - \langle q_i(t)\rangle_T][q_j(t) - \langle q_j(t)\rangle_T] \right\rangle_T \tag{A.2}$$

Diagonalization of this covariance matrix results in pairs of eigenvalues λ_k and eigenvectors $\vec{v}^{(k)}$. Indeed, because the covariance matrix is real and symmetric, we find for the eigenvalues λ_k

$$\lambda_1 \geqslant \lambda_2 \geqslant \cdots \geqslant \lambda_N \geqslant 0, \quad \sum_{k=1}^{N} \lambda_k = 1 \tag{A.3}$$

Due to the orthogonality of the eigenvectors $\vec{v}^{(k)}$, the evolution along each mode is calculated by means of the projection

$$\xi_k(t) = \vec{v}^{(k)} \cdot \vec{q}(t) \tag{A.4}$$

The global pattern $q^{(\mathrm{global})}(t)$ is defined as

$$\vec{q}^{(\mathrm{global})}(t) = \sum_{k=1}^{M} \xi_k(t)\vec{v}^{(k)} \tag{A.5}$$

in which M denotes the number of modes that represent the coherent signal structures. The residual pattern $q^{(\mathrm{residual})}(t)$ is defined as

$$\vec{q}^{(\mathrm{residual})}(t) = \sum_{k=M+1}^{N} \xi_k(t)\vec{v}^{(k)} = \sum_{k=M+1}^{N} [\vec{v}^{(k)}]^{\mathrm{T}}\vec{q}(t)\vec{v}^{(k)} \tag{A.6}$$

For the present analysis the eigenvectors were defined based on the entire data set, that is, for all lumbar EMG recordings (m) or segments (r) for all velocities (v) for all conditions (c), thus $\vec{q}(t)$ contains N time series, i.e. for EMG data

$$\vec{q} = \begin{pmatrix} \vec{q}_{\mathrm{condition}_1} \\ \vec{q}_{\mathrm{condition}_2} \\ \vec{q}_{\mathrm{condition}_3} \\ \vec{q}_{\mathrm{condition}_4} \end{pmatrix},$$

$$\vec{q}_{\mathrm{condition_c}} = \vec{q}_c = \begin{pmatrix} \vec{q}_{c,\mathrm{velocity}_1} \\ \vec{q}_{c,\mathrm{velocity}_2} \\ \vec{q}_{c,\mathrm{velocity}_3} \\ \vec{q}_{c,\mathrm{velocity}_4} \end{pmatrix}, \tag{A.7}$$

$$\vec{q}_{c,\mathrm{velocity}_v} = \vec{q}_{c,v} = \begin{pmatrix} \vec{q}_{c,v,\mathrm{emg}_1} \\ \vdots \\ \vec{q}_{c,v,\mathrm{emg}_4} \end{pmatrix}$$

where substituting the last expression with

$$\vec{q}_{c,v,\mathrm{emg}_m} = \vec{q}_{c,v,m} = \begin{pmatrix} q_{c,v,m,\mathrm{stride}_1} \\ \vdots \\ q_{c,v,m,\mathrm{stride}_S} \end{pmatrix} \tag{A.8}$$

$q_{c,v,m,\mathrm{stride}_n} = q_{c,v,m,s}(t)$ represents the EMG time series per condition $c = 1, \ldots, 4$ ($1 = \mathrm{control}, 2, \ldots, 4 = \mathrm{experimental}$ conditions) and one velocity $v = 1, \ldots, 4$ of one lumbar EMG recording $m = 1, \ldots, 4$, during single stride cycle $s = 1, \ldots, S$. For kinematic data EMG (m) reads as segments $r = 1, 2, 3$.

To distinguish between the variance of the global and the residual pattern, $\vec{q}(t)$ is replaced by $\vec{q}^{(\mathrm{global})}$ or $\vec{q}^{(\mathrm{residual})}$. For $q^{(\mathrm{global})}_{c,v,m,s}(t)$ and $q^{(\mathrm{residual})}_{c,v,m,s}(t)$ separately, the variance along strides for each time series of condition c at velocity v and EMG m was calculated as

$$\begin{aligned} \sigma^2_{c,v,m,s} &= \sigma^2_t[q_{c,v,m,s}(t)] \\ &= \frac{1}{T}\int_0^T \left(q_{c,v,m,s}(t) - \frac{1}{T}\int_0^T q_{c,v,m,s}(t')\,\mathrm{d}t' \right)^2 \mathrm{d}t \end{aligned} \tag{A.9}$$

with $q_{c,v,m,s}(t)$ representing $q^{(\mathrm{global})}_{c,v,m,s}(t)$ or $q^{(\mathrm{residual})}_{c,v,m,s}(t)$.

For $q^{(\mathrm{global})}_{c,v,m,s}(t)$ and $q^{(\mathrm{residual})}_{c,v,m,s}(t)$, the ratio between the SD, $\sigma_{c,v,m,s} = \sqrt{\sigma^2_{c,v,m,s}}$, of each of the experimental conditions separately ($c = 2, \ldots, 4$) and that in the control condition ($c = 1$) was computed as

$$R_{v,m} = \sum_{s=1}^{S} \sigma_{c_{2,\ldots,4},v,m,s} \Big/ \sum_{s=1}^{S} \sigma_{c1,v,m,s} \quad \text{with}$$
$$R_v = R_{v,m}^{(\text{global})} \text{ or } R_{v,m}^{(\text{residual})} \tag{A.10}$$

$R_{v,m}^{(\text{global})}$ and $R_{v,m}^{(\text{residual})}$ serve as indices of variability for $\vec{q}^{(\text{global})}$ and for $\vec{q}^{(\text{residual})}$, respectively.

Equivalently, the ratio of the mean SD over strides and EMG recordings for each velocity and condition was calculated as

$$R_v = \sum_{m=1}^{4} \sum_{s=1}^{S} \sigma_{c_{2,\ldots,4},v,m,s} \Big/ \sum_{m=1}^{4} \sum_{s=1}^{S} \sigma_{c_1,v,m,s} \quad \text{with}$$
$$R_v = R_v^{(\text{global})} \text{ or } R_v^{(\text{residual})} \tag{A.11}$$

Note that for all the ratios $R_{v,m}$ and R_v the fundamental value of variance along a stride was computed, that is, the variability over time (cf. also Eq. (A.9)).

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