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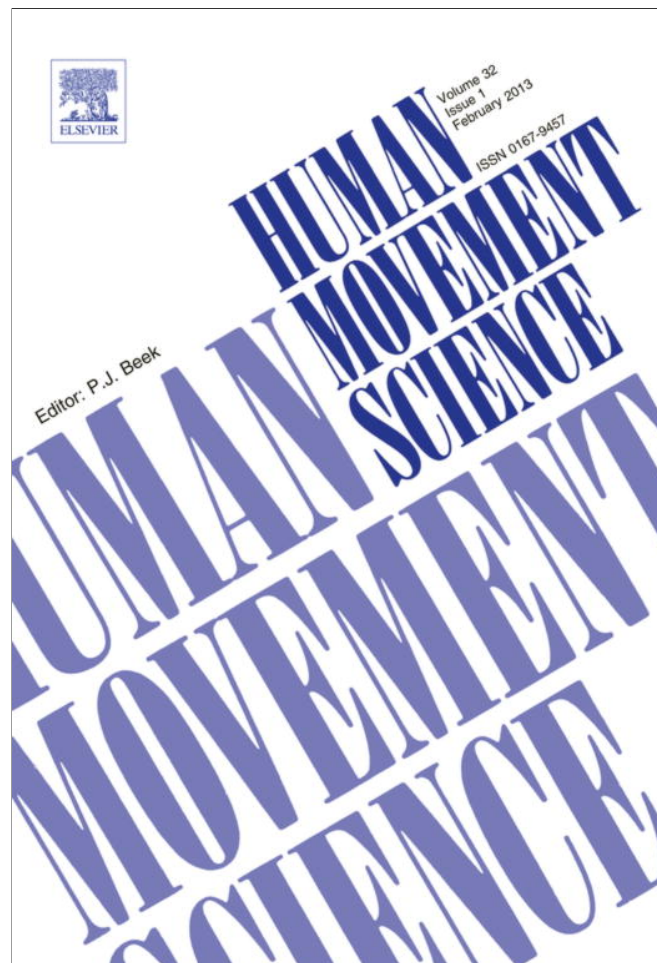
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Precision control of trunk movement in low back pain patients

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ABSTRACT

Motor control is challenged in tasks with high precision demands. In such tasks, signal-dependent neuromuscular noise causes errors and proprioceptive feedback is required for optimal performance. Pain may affect proprioception, muscle activation patterns and resulting kinematics. Therefore, we investigated precision control of trunk movement in 18 low back pain (LBP) patients and 13 healthy control subjects. The subjects performed a spiral-tracking task requiring precise trunk movements, in conditions with and without disturbance of proprioception by lumbar muscle vibration. Tracking task performance and trunk muscle electromyography were recorded. In conditions without lumbar muscle vibration, tracking errors were 27.1% larger in LBP patients compared to healthy controls. Vibration caused tracking errors to increase by 10.5% in healthy controls, but not in LBP patients. These results suggest that reduced precision in LBP patients might be explained by proprioceptive deficits. Ratios of antagonistic over agonistic muscle activation were similar between groups. Tracking errors increased trunk inclination, but no significant relation between tracking error and agonistic muscle activation was found. Tracking errors did not decrease when antagonistic muscle activation increased, so, neither healthy subjects nor LBP patients appear to counteract trunk movement errors by increasing co-contraction.

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1. Introduction

Precise motor control is hampered by neuromuscular noise. The exact origin of this noise is still unknown (Christakos, Papadimitriou, & Erimaki, 2006; De Luca, LeFever, McCue, & Xenakis, 1982; Jones, Hamilton, & Wolpert, 2002), but synaptic noise resulting in fluctuations in motor unit firing rates and firing intervals is suggested to be one of the main causes (Matthews, 1996; Selen, Beek, & van Dieën, 2005). Neuromuscular noise is signal-dependent, in that force variability increases with muscle activation level (Allum, Dietz, & Freund, 1978; Christou, Grossman, & Carlton, 2002; Jones et al., 2002; Newell & Carlton, 1988; Sherwood, Schmidt, & Walter, 1988; Slifkin, Vaillancourt, & Newell, 2000; Tseng, Scholz, Schöner, & Hotchkiss, 2003; Visser et al., 2003). The effects of neuromuscular noise become apparent in a lack of precision, for example in aiming and tracking tasks. Performance on such tasks reflects the ability to reduce kinematic variability and can be used as a measure of quality of motor control.

Precise motor control appears to be impaired by pain. Huysmans and colleagues found larger upper limb tracking errors in subjects with shoulder pain compared to pain-free controls (Huysmans, Hoozemans, van der Beek, de Looze, & van Dieën, 2010). It has been suggested that this is due to the effect of nociceptive afference on muscle spindle feedback, which would impair proprioception (Pedersen, Sjolander, Wenngren, & Johansson, 1997). In addition, chronic pain has been shown to cause reorganization in the primary somatosensory cortex (Flor, Braun, Elbert, & Birbaumer, 1997), which may modulate the processing of both noxious and nonnoxious input (Moseley & Flor, 2012). In the study on shoulder pain the reduced precision indeed coincided with a reduced proprioceptive acuity in the pain group (Huysmans et al., 2010). Proprioceptive impairments in low back pain (LBP) patients have been demonstrated using lumbar muscle vibration, which is known to perturb proprioceptive feedback from muscle spindle afferents by inducing a lengthening illusion (Burke, Hagbarth, Lofstedt, & Wallin, 1976; Roll, Vedel, & Ribot, 1989). Brumagne and colleagues found reduced trunk repositioning accuracy in LBP patients compared to healthy controls and, interestingly, paraspinal muscle vibration negatively affected trunk repositioning accuracy in healthy controls, but not in LBP patients (Brumagne, Cordo, Lysens, Verschueren, & Swinnen, 2000). Also, in a variety of postural tasks with vision occluded, the relative effects of lumbar muscle vibration and calf muscle vibration on postural sway differed between LBP patients and healthy controls (Brumagne, Cordo, & Verschueren, 2004; Brumagne, Janssens, Knapen, Claeys, & Suuden-Johanson, 2008; Claeys, Brumagne, Dankaerts, Kiers, & Janssens, 2011). LBP patients tended to use a more ankle-steered strategy, in that their response to calf muscle vibration was larger than their response to lumbar muscle vibration, which might point at a lower weighting of proprioceptive information from lumbar muscle spindles. Moreover, individuals with LBP have been shown to have increased levels of co-activation (van Dieën, Selen, & Cholewicki, 2003), which may indicate a compensatory joint stiffening strategy to deal with impaired proprioception.

Indeed, joint impedance modulation by antagonistic co-activation has been suggested as a means to counteract kinematic variability due to neuromuscular noise. Modeling work suggests that, by activating antagonistic muscle pairs around a joint, joint stiffness increases and kinematic variability decreases in spite of an increase in force variability of each of the muscles separately (Selen et al., 2005). In upper extremity tracking tasks, increased precision indeed coincided with increased joint impedance (Selen, Beek, & van Dieën, 2006; Selen, van Dieën, & Beek, 2006) and EMG activity (Huysmans et al., 2010), suggesting that the tracking error was successfully reduced by antagonistic co-activation.

Thus, whereas increased agonistic muscle activation can reduce precision, antagonistic co-activation can increase precision. In the trunk, however, no evidence for the use of such a co-activation strategy was found in static positioning tasks (Willigenburg, Kingma, & van Dieën, 2010). Instead, feedback control appeared to be used to regulate precision. Given this stronger reliance on feedback instead of co-activation to modulate precision in the trunk compared to the upper extremity and given the proprioceptive impairments associated with pain, we expect LBP to have pronounced effects on precision control of the trunk.

While tracking tasks are often called visuo-motor tasks, proprioceptive feedback is an important source of information in controlling tracking movements (Eidelberg & Davis, 1976; Nagaoka & Tanaka,

1981). In the present study, we therefore used a tracking task that required precise trunk movement with varying levels of trunk inclination, to investigate the effects of muscle activation level, lumbar muscle vibration and pain on kinematic error, as a measure of quality of trunk control. Subjects with and without LBP performed this tracking task with and without disturbance of proprioception through paraspinal muscle vibration. Muscle activity was continuously monitored by means of surface-electromyography (sEMG). We hypothesized that subjects with LBP would make larger tracking errors and that, as patients may rely less on proprioception, muscle vibration would have a larger effect in subjects without LBP. Furthermore, we hypothesized that tracking errors would increase with trunk inclination, and thus with the level of agonistic muscle activity. Ratios of antagonistic co-activation were hypothesized to be higher in the patient group.

2. Methods

2.1. Subjects

Eighteen subjects with non-specific LBP (31 ± 14 [$M \pm SD$] years old, BMI 23.4 ± 2.4 kg/m², 11 male) and 13 healthy controls (34 ± 12 years old, BMI 22.9 ± 2.4 kg/m², 9 male) with no (history of) LBP participated in the experiment. No significant differences between the LBP and control groups were found in age and BMI and all subjects had normal or corrected to normal sight. One LBP patient was not able to participate due to a very limited range of lumbar motion. The protocol was approved by the local medical ethics committee and all subjects provided written informed consent before participating. Inclusion criteria for the LBP patient group were self-reported LBP for the last 6 weeks or longer, specific diagnosis excluded by general practitioner or physical therapist, no previous surgery on the spine, score ≤ 105 on yellow flags screening questionnaire (Linton & Hallden, 1996), no other conditions (e.g., neurological or mental disorders, allergy to plaster) hindering participation or performance and age between 18 and 65. LBP patients scored 15.2 ± 4.2 at the Oswestry pain and disability index (10 = minimal disability, 60 = maximal disability) and 26.8 ± 18.9 at the visual analogue scale (0–100) for pain intensity before participation.

2.2. Experimental setup

Participants maintained a semi-seated/kneeled position with their pelvis fixed and their lower legs supported by a frame (Fig. 1). Subjects placed their hands on top of their head, in order to minimize upper limb contributions to trunk postural control. Opto-electronic markers (Optotrak 3020, Northern Digital Inc, Canada; 100 samples/s) were placed on the spinous process of T12 and on the frame (fixed point in space) at pelvis height. Trunk angle was defined as the angle of the line through the T12 marker and the marker on the frame with respect to the vertical. Real-time (delay max. 10 ms) feedback of trunk angle was presented as a black dot (cursor) on a 17 inch computer screen, which was located 120 cm in front of the subject, at chest height. Trunk angle changes in the frontal plane corresponded to movements of the cursor along the x-axis (left-right) and trunk angle changes in the sagittal plane corresponded to movements of the cursor along the y-axis (up-down). The origin of the display was defined by each individual's neutral trunk angle (self-chosen, as described in the next section). The x-axis ranged from -30° (lateral flexion to the left) to $+30^\circ$ (lateral flexion to the right). The y-axis ranged from -20° (extension) to $+40^\circ$ (flexion). Both height and width of the display were 600 pixels, so 1 pixel corresponded to 0.1 degree of trunk angle. In addition to the cursor (a circle with a diameter of 20 pixels), a target area was presented on the screen. This target area was a yellow square (30 by 30 pixels), which moved following a spiral-shaped trajectory. A yellow line preceded the square, to show subjects the to-be-tracked trajectory 10 s in advance.

Electromyography (Porti 17, TMS, Enschede, The Netherlands; 22 bits AD conversion after 20x amplification, input impedance $>10^{12} \Omega$, CMRR >90 dB) of four abdominal and four back muscles was measured, both left and right, using pairs of surface EMG electrodes (Ag/AgCl, inter-electrode distance 25 mm) that were attached to the skin after shaving and cleaning with alcohol. Activation of thoracic back muscles was recorded 4 cm lateral to T9 (thoracic part of m. longissimus (LT)) and

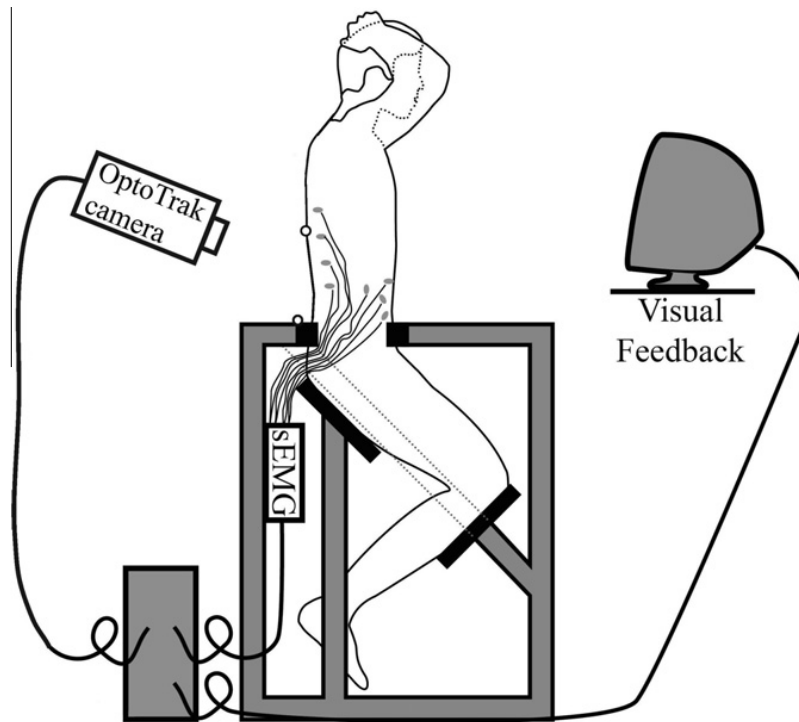


Fig. 1. Experimental setup.

6 cm lateral to T11 (thoracic part of *m. iliocostalis* (IT)) spinous processes. Electrodes to record lumbar back muscle activation were placed 6 cm lateral to L2 (lumbar part of *m. iliocostalis* (IL)) spinous process and 3 cm lateral to the midpoint between the spinous processes of L3 and L4 (lumbar part of *m. longissimus* (LL)). For abdominal muscles, electrodes were placed 3 cm lateral to the umbilicus or somewhat lower when a tendinous intersection was present there (*m. rectus abdominus* (RA)), 3 cm medial to the anterior superior iliac spine (ASIS) (*m. obliquus internus* (OI)), in the mid-axillary line between the iliac crest and the 10th rib (lateral part of *m. obliquus externus* (OEL)) and at the crossing point of a horizontal line through the umbilicus and a vertical line through the ASIS (anterior part of *m. obliquus externus* (OEA)). Muscle activation was recorded at a sample rate of 1000 samples/s and a pulse signal was used to synchronize the kinematic and EMG data.

In conditions with lumbar muscle vibration, a motor rotating an eccentric mass (Maxon Graphite Brushes S2326.946 driven by a 4-Q-DC Servo Control LSC 30/2 in a velocity-loop, frequency 90 Hz) was positioned at the lumbar longissimus EMG electrodes; 3 cm lateral of L3/L4 spinous processes, both left and right (Fig. 2). The vibration motor was fixed with neoprene elastic bands and vibration was applied continuously during the experimental task.

2.3. Experimental protocol

To determine each individual's neutral trunk angle, subjects were first asked to sit comfortably with their hands on their head. This neutral angle defined the origin of the computer screen and the inner endpoint of the spiral-shaped trajectory was in 5° flexion with respect to that neutral posture. Subjects were instructed to adjust their trunk posture in order to stay within the yellow square target area while it followed the spiral-shaped trajectory. The center of the target reflected the target angle. The target angle in the frontal plane was calculated by $x = 0.3t * \cos(t)$ and the target angle in the sagittal plane was calculated by $y = 0.45t * \sin(t) + 5$, with t ranging from 0 to $10 * \pi$, in steps of 0.005 radians. Since each rotation of 360° corresponds to $2 * \pi$ radians, this resulted in a spiral trajectory that consisted of five rotations (Fig. 3). Speed of trunk movement was normalized to a constant change in trunk angle of 1.76°/s, and the task took about 2 min. The outer boundaries of the spiral corresponded to approximately 10° extension and lateral flexion and 17° flexion. Two trials started in the

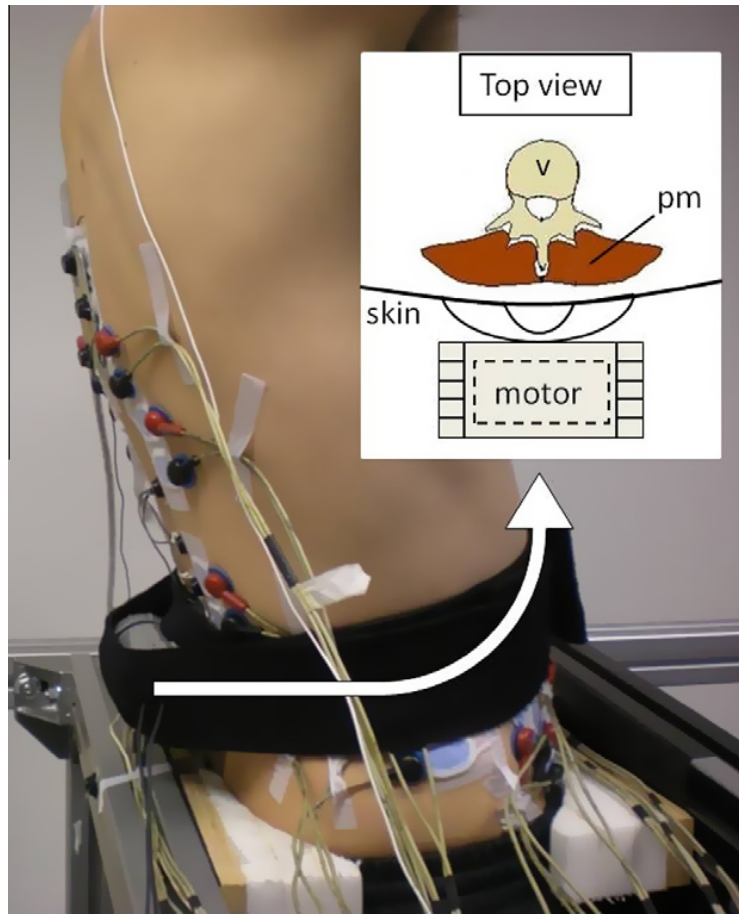


Fig. 2. Vibration device as attached over the paraspinal muscles (pm) at the level of L3/4. The vibrating motor was stored in a plastic cylinder, which in turn was fixed to a solid plastic U-shape to apply bilateral vibration to the paraspinal muscles while leaving the spinous process of the lumbar vertebra (v) free.

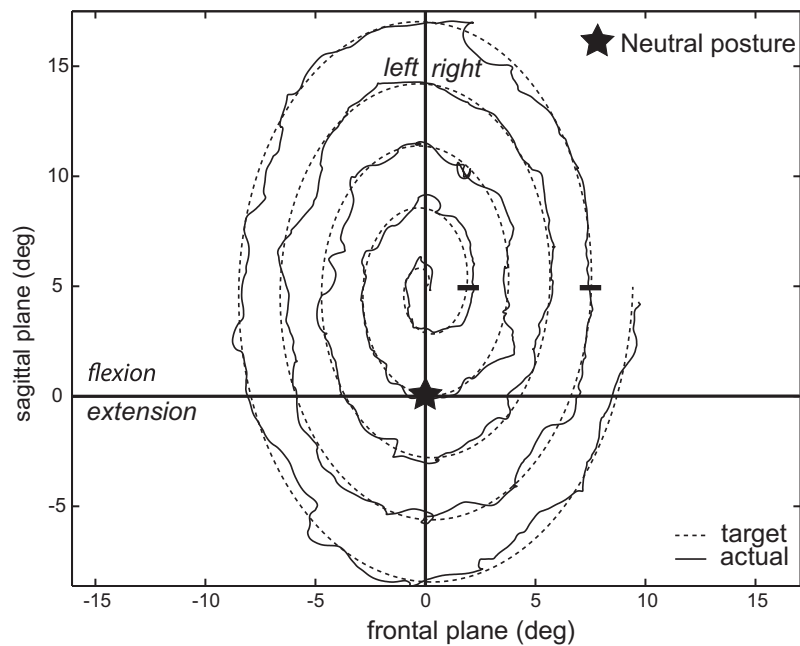


Fig. 3. Example of a single trial without vibration for one (healthy) subject. Black bars indicate the start and end of the part of the trajectory that was used for data analysis.

center of the spiral and consisted of 5 counterclockwise rotations with increasing amplitude. In two additional trials, the target started at the outer endpoint of the same spiral, at about 10° lateral flexion to the right, and thus followed 5 clockwise rotations with decreasing amplitude towards the center of the spiral. These four trials were performed with and without lumbar muscle vibration, resulting in a total of 8 trials. The trial order was counterbalanced between subjects, in that for both groups (with and without LBP), half of the subjects started with vibration and the other half started without vibration. Within these subgroups, half of the subjects started with the clockwise trajectory and the other half started with the counterclockwise trajectory. Subjects were instructed not to rotate around the longitudinal axis.

2.4. Data analysis

Matlab R2010a was used for data analysis. Tracking error was defined as the absolute difference between the trunk angle and the target angle at each instant of time. Specifically, we calculated the hypotenuse of the angular tracking error in the frontal (x -axis) and sagittal (y -axis) planes of motion:

$$\text{tracking error} = \sqrt{(x_{\text{angle,target}} - x_{\text{angle,trunk}})^2 + (y_{\text{angle,target}} - y_{\text{angle,trunk}})^2}.$$

After 22 bit AD-conversion at 1000 samples/s, EMG data were 30 Hz high-pass filtered to remove contamination from the electrocardiogram (Redfern, Hughes, & Chaffin, 1993) and 49.5–50.5 Hz band-stop filtered to remove any 50 Hz interference. Then Hilbert amplitudes were calculated and 2.5 Hz low-pass filtered (2nd order Butterworth), uni-directionally in order to obtain a linear envelope and to correct for electromechanical delay (Potvin, Norman, & McGill, 1996). Trials with lumbar muscle vibration were discarded from EMG analyses, since some EMG signals were contaminated by the vibration. Both tracking error and muscle activation time series were averaged over two repetitions for each trial.

Since subjects needed some time to catch up with the target at the start of each trial, we only analyzed the inner three rotations of the spiral-shaped trajectory (Fig. 3). In order to be able to use time series as input for our statistical procedures, a 2 Hz running average was calculated from the time series of the tracking error and EMG linear envelopes. Co-activation ratios were obtained by dividing non-normalized antagonist by agonist EMG amplitudes. For the other analyses we normalized EMG amplitudes to the highest value recorded during the four trials without vibration, in order to reduce inter-subject variance. Note that we did not normalize to maximally voluntary contractions as this may introduce a bias, because patients may not activate their back muscles maximally.

Discrimination between agonist and antagonist was based on trunk posture imposed by the target trajectory and the corresponding gravitational moment. Specifically, we discriminated four quadrants of the trajectory in which trunk flexion or extension moments in combination with lateral bending moments to the left or right were required (Fig. 3). Since trunk movements were rather slow, accelerations of the trunk were neglected and agonist and antagonist muscles were defined based on the moments that these muscles generated around the lumbar spine relative to the gravitational moment, i.e., they were considered as agonists when acting against the gravitational moment and hence contributing to the moment required, and as antagonists when acting in the direction of the gravitational moment. We averaged the linear envelopes over muscles within each quadrant, i.e., left abdominal, right abdominal, left back and right back muscles, resulting in 4 muscle groups consisting of 4 muscles each. Then EMG time series were split into periods in which these muscle groups acted either as agonist or as antagonist. So, in parts of the trajectory requiring trunk flexion and lateral bending to the left, where the upper body center of mass was located anterior and left with respect to the lumbar spine, the right back muscles were defined as primary agonists (Rback_ago) and the left abdominal muscles were defined as primary antagonists (Labd_anta). Similarly, for parts of the trajectory requiring trunk extension and lateral bending to the left, where the upper body center of mass was located posterior and left with respect to the lumbar spine, right abdominal and left back muscle activation were defined as Rabd_ago and Lback_anta, respectively. Activation levels were set to zero for periods when muscle groups did not act as primary agonist or antagonist.

2.5. Statistics

SPSS version 16.0 was used for statistical analysis. One way ANOVA was performed to compare co-activation ratios between groups after averaging over the four quadrants. In addition, we performed two regression analyses for repeated measures, using generalized estimation equations (GEEs). Linear models with exchangeable working correlation structures, robust covariance matrix estimators and Wald Chi-Square statistics were used. Tracking error was defined as dependent variable, and two different combinations of predictor parameters were defined (Fig. 4).

To detect differences in tracking errors between LBP patients and healthy controls and between conditions with and without vibration, the first GEE model included the factors 'group' and 'vibration'. Healthy controls and conditions without vibration were considered as references. To evaluate the effect of trunk inclination on tracking errors, we included trunk inclination, calculated as the square root of the sum of the squared frontal plane and sagittal plane trunk angles, as a covariate. Interactions with the factor group were included to test whether the effects of vibration condition and trunk inclination differed between groups. When not significant, interactions were removed one-by-one, starting with the highest p-value.

The second GEE model was aimed at elucidating the effects of agonist and antagonist muscle activation on tracking errors, as well as the potential interference of pain with these effects. GEE model 2 therefore included the factor 'group', which discriminated between LBP patients and healthy controls (as reference), as well as eight EMG covariates. These covariates reflected time series of the four different muscle groups, acting as primary agonist or antagonist, with zero values for periods when the muscle group did not act in that function. Recall that the trials with lumbar muscle vibration were

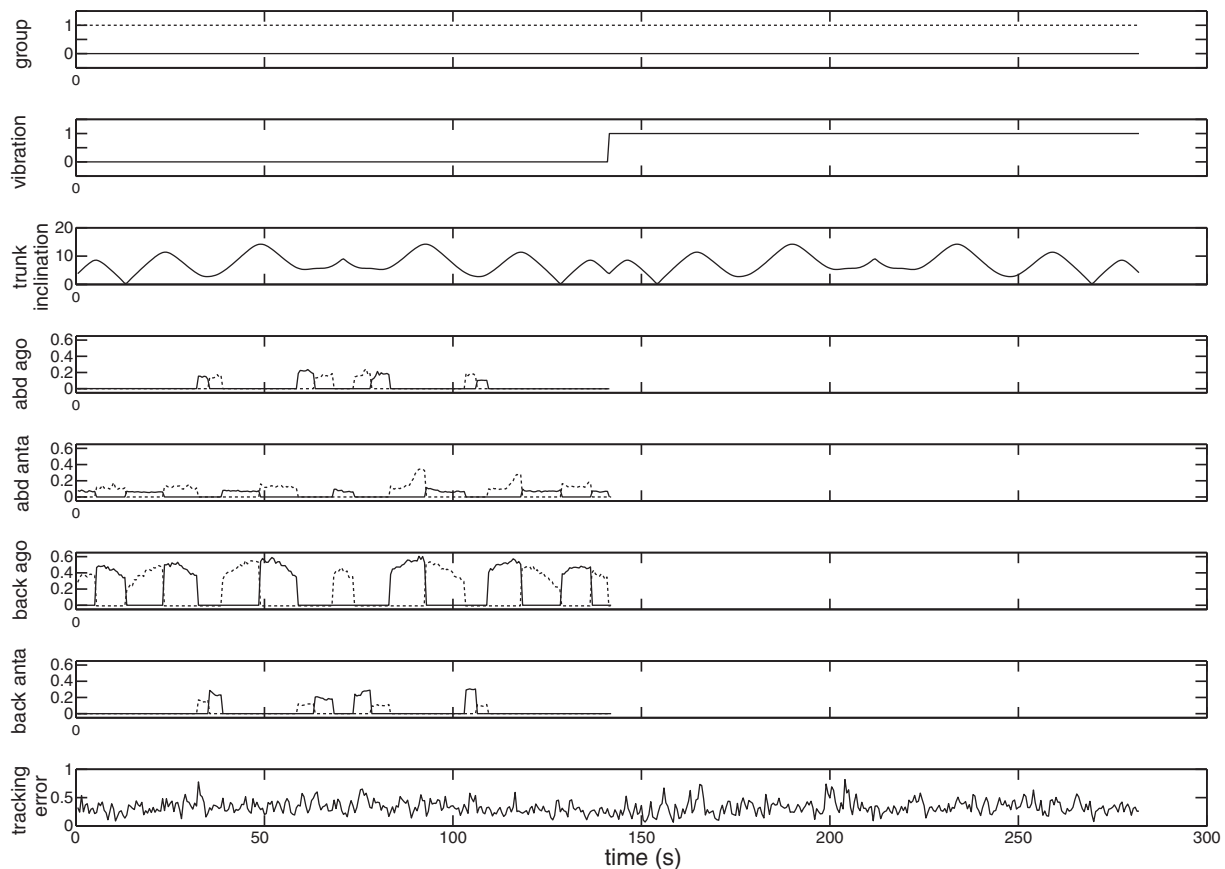
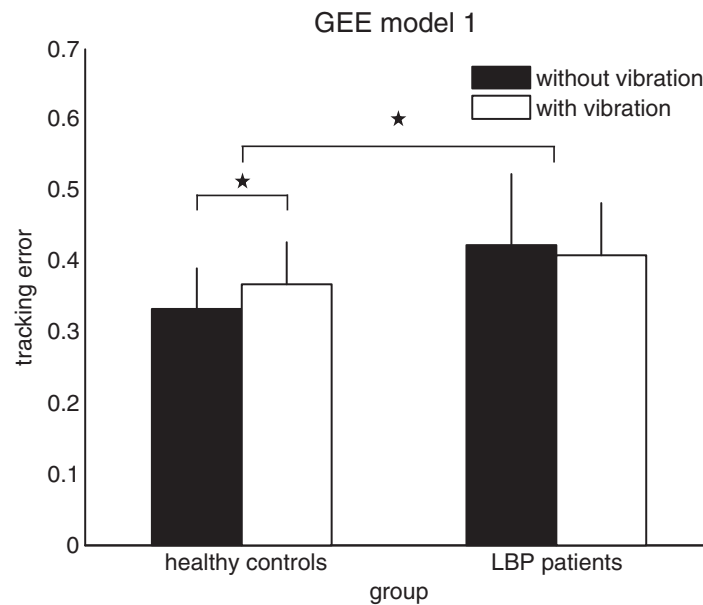


Fig. 4. Typical example of variables of interest against time to visualize the variables that were included in the GEE models. GEE model 1 included the factor group (upper panel), the factor vibration (second panel), and the covariate trunk angle amplitude (third panel). GEE model 2 included the factor group (upper panel) and eight covariates: right (solid) and left (dotted) abdominal and back muscle activation acting as agonist and as antagonist (panels 4–8). Tracking error (lower panel) was the dependent variable in both GEE models.

Table 1
GEE model 1.

	Coefficient	95% Confidence interval	P-value
Intercept	.304	.271–.337	<.001
Group (LBP = 1)	.090	.036–.144	.001
Vibration (on = 1)	.035	.006–.064	.016
Trunk inclination	.004	.002–.006	.001
Group * vibration	–.048	–.086–.011	.012

**Fig. 5.** Averages and SDs (error bars) of tracking errors for both groups and vibration conditions.**Table 2**
GEE model 2.

	Mean \pm SD	Coefficient	95% Confidence interval	P-value
Intercept		.263	.218–.307	<.001
Group (LBP = 1)		.112	.052–.173	.001
Rabd_ago	.25 \pm .10	.175	–.071–.420	.162
Labd_ago	.26 \pm .10	.171	–.010–.352	.065
Rabd_anta	.19 \pm .10	.224	.038–.409	.018
Labd_anta	.18 \pm .10	.274	.074–.474	.007
Rback_ago	.40 \pm .11	.001	–.074–.076	.980
Lback_ago	.41 \pm .10	.004	–.065–.073	.902
Rback_anta	.17 \pm .07	.263	–.041–.566	.090
Lback_anta	.17 \pm .07	.270	–.143–.683	.200

discarded from EMG analysis, so for GEE model 2 we only used the data without vibration. Again, two-way interactions with the factor 'group' were examined, but removed when not significant.

3. Results

3.1. Effects of pain, proprioception disturbance, and trunk inclination on tracking error

Results for GEE model 1 (Table 1), evaluating the effects of group (pain), vibration (proprioception disturbance), and trunk inclination revealed one significant interaction. In the absence of lumbar

muscle vibration, the tracking error was significantly larger (27.1%) in LBP patients (0.422°) compared to healthy controls (0.332° , Fig. 5). In line with our hypothesis, vibration affected the performance in the control group more than in the LBP patients. Specifically, the tracking error significantly increased by 10.5% with vibration (to 0.367°) in the control group, while the patient group showed a slight (but not significant) decrease in tracking error in conditions with lumbar muscle vibration (to 0.409°). Furthermore, as hypothesized, tracking errors significantly increased with trunk inclination.

To further evaluate the effect of low back pain on tracking error within the LBP group, we calculated the correlation between tracking error (averaged over all trials), and pain intensity (score on the visual analogue scale before the start of the experiment). Interestingly, tracking error and intensity of LBP were positively correlated ($r = 0.48$, $p = 0.042$), indicating that subjects with more severe LBP performed worse on the tracking task.

3.2. Antagonistic co-activation

Mean co-activation ratios were 1.68 ± 0.83 for healthy controls and 1.23 ± 0.72 for LBP patients and the difference between groups was not significant ($p = 0.111$). These ratios of antagonistic over agonistic muscle activation levels were rather high, since back muscle activation levels substantially exceeded abdominal muscle activation levels, even when acting as antagonist.

3.3. Effects of agonist and antagonist trunk muscle activation and pain on tracking error

GEE model 2 did not reveal any significant interaction with group (all $p \geq .184$), so no differences between LBP patients and healthy controls were found in the effects of agonist and antagonist activation on tracking errors. Therefore, interactions were not included in the final model (Table 2). The significant effect of group (again) indicated that LBP patients performed significantly worse than healthy controls. While all coefficients for muscle activation were positive, indicating an increase in tracking error with increasing activation, only two of these covariates (left and right antagonistic abdominal muscle activation) reached significance. Coefficients for Rback_ago and Lback_ago were close to zero, indicating that agonistic back muscle activation did not affect tracking errors.

4. Discussion

The current study evaluated trunk motor control during a tracking task that required precise trunk movements with varying levels of trunk inclination. This task was performed by subjects with and without LBP, in conditions with and without lumbar muscle vibration. As hypothesized, tracking errors were higher in the LBP patients compared to healthy controls and increased with trunk inclination. Patients with more severe LBP made larger errors than patients with lower pain levels. In addition, lumbar muscle vibration deteriorated performance in healthy controls, but not in LBP patients, while levels of antagonistic co-activation were similar between groups. In contrast with our hypothesis, no significant increase in tracking errors was found with increasing agonistic muscle activation.

Our results showed that the tracking performance of healthy controls decreased in the condition with lumbar muscle vibration, despite the continuous presence of visual feedback. However, LBP patients already performed worse without vibration and their performance did not deteriorate with vibration. This suggests that vibration affected the contribution of proprioceptive feedback from lumbar muscle spindles to tracking performance in healthy controls but not in LBP patients and, hence, that proprioception is impaired or not used as much in LBP patients. Previous studies have already found reduced weighting of lumbar proprioception relative to other sources of proprioceptive information in postural control (Brumagne et al., 2000, 2004, 2008; Claeys et al., 2011), but, to our knowledge, reduced weighting of lumbar proprioception relative to visual information has not been reported before. This reweighting of information sources could be due to reduced quality of proprioceptive information.

LBP patients did not show increased levels of trunk muscle co-activation to compensate for their proprioceptive impairments. Interestingly, previous studies reported trunk muscle activation patterns apparently aimed at trunk stiffening in LBP patients during other tasks (van Dieën et al., 2003). However, we did not find evidence for the use of such a strategy in the current study. Maybe our inclusion criterion of a <105 score at the questionnaire for yellow flags can explain why we did not find increased co-activation in our patient group. The yellow flags questionnaire evaluates psychosocial factors, which may contribute to the chronicity of LBP. LBP patients with high levels of stress and anxiety will score high at this questionnaire. Therefore, this subgroup of patients, who may be more likely to increase muscle activation (van der Hulst, Vollenbroek-Hutten, Schreurs, & Hermens, 2010), did not participate in our study.

The finding that tracking errors significantly increased with trunk inclination seems to support previous findings on signal dependent force variability (Allum et al., 1978; Christou et al., 2002; Jones et al., 2002; Newell & Carlton, 1988; Sherwood et al., 1988; Slifkin et al., 2000; Tseng et al., 2003; Visser et al., 2003). However, in contrast with our hypothesis, GEE model 2 did not reveal a significant relation between agonistic muscle activation and tracking errors. While the positive coefficients of agonistic abdominal muscle activation tended to approach the level of significance, the effect of agonistic back muscle activation was almost zero. This suggests that another mechanism may dominate the effect of muscle activation on tracking error. For instance, this could be related to the differences in anatomical characteristics between abdominal and back musculature. While most back muscles have several segmental insertions, the abdominal muscles span all lumbar segments with their insertions on thorax and pelvis. The segmental insertions of the back muscles may facilitate precise trunk control and could therefore explain why tracking errors did not increase with agonistic back muscle activation.

The finding that antagonistic muscle activation did not reduce tracking error is in contrast with previous findings on precision control in the upper extremity (Gribble, Mullin, Cothros, & Mattar, 2003; Huysmans et al., 2010; Selen, Beek et al., 2006; Selen, van Dieën et al., 2006), but supports a recent suggestion based on static trunk control (Willigenburg et al., 2010), that precision of the trunk is regulated by feedback corrections rather than by a co-activation strategy. This difference between precision control of the trunk and the limbs can be explained by several factors. First, trunk movements are multi-articular, whereas the investigated limb movements occurred in individual joints. Second, the trunk has a large mass and, thus, a high inertia compared to the limbs. Therefore trunk movements are relatively slow, which may facilitate feedback control. Moreover, trunk stiffening may interfere with other functions of trunk musculature, such as breathing.

A limitation of the present study is that we did not specifically measure EMG activity of deep trunk muscles such as m. multifidus and m. transversus abdominis, whereas these muscles have been attributed an important role in trunk motor control (Hodges & Richardson, 1997; Moseley, Hodges, & Gandevia, 2002). However, surface electrodes at the reported locations probably reflected some of these deeper muscles' activity (Stokes, Henry, & Single, 2003) as well. Furthermore, some variations in task execution were observed. Cursor position in our tracking task corresponded to the frontal and sagittal plane angles of the T12 marker, and did therefore not constrain relative movements between lumbar spinal segments, nor motion in the thoracic and cervical spine. Moreover, the neutral posture was self-defined by each individual and therefore did not (necessarily) reflect a similar (zero) external moment around L5/S1 for each subject. While these variations in task execution enhance the ecological validity, they caused substantial variations in trunk muscle activation.

Another limitation was the EMG normalization procedure. We did not normalize to activation levels during isometric (sub)maximally voluntary excitation, because this involves the risks of introducing a bias between patients and healthy controls (van Dieën et al., 2003). The current normalization method (to the highest value recorded during four measurements), that we used for GEE model 2, could mask potential differences in EMG amplitudes between groups. Therefore, we could not use the normalized EMG amplitudes to compare trunk muscle activation levels between healthy controls and LBP patients. Since this comparison was not required for testing our hypotheses on effects of agonist and antagonist activation on precision, this limitation did not affect our conclusions. Importantly, in order to compare trunk muscle co-activation ratios between groups, we used the non-normalized

EMG amplitudes. This involved the assumption that volume conduction did not differ between groups, which was considered plausible given the successful matching of BMI at the group level.

In conclusion, LBP patients made larger errors in a spiral tracking task requiring circular trunk movements. Lumbar muscle vibration did not affect performance in LBP patients, but caused tracking errors to increase in healthy participants. These results suggest that LBP is associated with proprioceptive impairments. Antagonistic co-activation ratios did not differ between groups and, while tracking errors significantly increased with trunk inclination, no significant relation between agonistic muscle activation and tracking errors was found.

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References

- Allum, J. H., Dietz, V., & Freund, H. J. (1978). Neuronal mechanisms underlying physiological tremor. *Journal of Neurophysiology*, 41, 557–571.
- Brumagne, S., Cordo, P., Lysens, R., Verschueren, S., & Swinnen, S. (2000). The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. *Spine*, 25, 989–994.
- Brumagne, S., Cordo, P., & Verschueren, S. (2004). Proprioceptive weighting changes in persons with low back pain and elderly persons during upright standing. *Neuroscience Letters*, 366, 63–66.
- Brumagne, S., Janssens, L., Knapen, S., Claeys, K., & Suuden-Johanson, E. (2008). Persons with recurrent low back pain exhibit a rigid postural control strategy. *European Spine Journal*, 17, 1177–1184.
- Burke, D., Hagbarth, K. E., Lofstedt, L., & Wallin, B. G. (1976). The responses of human muscle spindle endings to vibration during isometric contraction. *Journal of Physiology*, 261, 695–711.
- Christakos, C. N., Papadimitriou, N. A., & Erimaki, S. (2006). Parallel neuronal mechanisms underlying physiological force tremor in steady muscle contractions of humans. *Journal of Neurophysiology*, 95, 53–66.
- Christou, E. A., Grossman, M., & Carlton, L. G. (2002). Modeling variability of force during isometric contractions of the quadriceps femoris. *Journal of Motor Behavior*, 34, 67–81.
- Claeys, K., Brumagne, S., Dankaerts, W., Kiers, H., & Janssens, L. (2011). Decreased variability in postural control strategies in young people with non-specific low back pain is associated with altered proprioceptive reweighting. *European Journal of Applied Physiology*, 111, 115–123.
- De Luca, C. J., LeFever, R. S., McCue, M. P., & Xenakis, A. P. (1982). Control scheme governing concurrently active human motor units during voluntary contractions. *Journal of Physiology*, 329, 129–142.
- Eidelberg, E., & Davis, F. (1976). Role of proprioceptive data in performance of a complex visuomotor tracking task. *Brain Research*, 105, 588–590.
- Flor, H., Braun, C., Elbert, T., & Birbaumer, N. (1997). Extensive reorganisation of primary somatosensory cortex in chronic back pain patients. *Neuroscience Letters*, 224, 5–8.
- Gribble, P. L., Mullin, L. I., Cothros, N., & Mattar, A. (2003). Role of cocontraction in arm movement accuracy. *Journal of Neurophysiology*, 89, 2396–2405.
- Hodges, P. W., & Richardson, C. A. (1997). Feedforward contraction of transversus abdominis is not influenced by the direction of arm movement. *Experimental Brain Research*, 114, 362–370.
- Huysmans, M. A., Hoozemans, M. J., van der Beek, A. J., de Looze, M. P., & van Dieën, J. H. (2010). Position sense acuity of the upper extremity and tracking performance in subjects with non-specific neck and upper extremity pain and healthy controls. *Journal of Rehabilitation Medicine*, 42, 876–883.
- Jones, K. E., Hamilton, A. F., & Wolpert, D. M. (2002). Sources of signal-dependent noise during isometric force production. *Journal of Neurophysiology*, 88, 1533–1544.
- Linton, S. J., & Hallden, K. (1996). Risk factors and the natural course of acute and recurrent musculoskeletal pain: Developing a screening questionnaire. In: *Proceedings of the eighth world congress on pain*.
- Matthews, P. B. (1996). Relationship of firing intervals of human motor units to the trajectory of post-spike after-hyperpolarization and synaptic noise. *Journal of Physiology*, 492(Pt. 2), 597–628.
- Moseley, G. L., & Flor, H. (2012). Targeting cortical representations in the treatment of chronic pain: A review. *Neurorehabilitation and Neural Repair*, 26, 646–652.
- Moseley, G. L., Hodges, P. W., & Gandevia, S. C. (2002). Deep and superficial fibers of the lumbar multifidus muscle are differentially active during voluntary arm movements. *Spine*, 27, E29–E36.
- Nagaoka, M., & Tanaka, R. (1981). Contribution of kinesthesia on human visuomotor elbow tracking movements. *Neuroscience Letters*, 26, 245–249.
- Newell, K. M., & Carlton, L. G. (1988). Force variability in isometric responses. *Journal of Experimental Psychology: Human Perception & Performance*, 14, 37–44.
- Pedersen, J., Sjolander, P., Wenngren, B. I., & Johansson, H. (1997). Increased intramuscular concentration of bradykinin increases the static fusimotor drive to muscle spindles in neck muscles of the cat. *Pain*, 70, 83–91.
- Potvin, J., Norman, R., & McGill, S. (1996). Mechanically corrected EMG for the continuous estimation of erector spinae muscle loading during repetitive lifting. *European Journal of Applied Physiology and Occupational Physiology*, 74, 119–132.

- Redfern, M. S., Hughes, R. E., & Chaffin, D. B. (1993). High-pass filtering to remove electrocardiographic interference from torso EMG recordings. *Clinical Biomechanics*, 8, 44–48.
- Roll, J. P., Vedel, J. P., & Ribot, E. (1989). Alteration of proprioceptive messages induced by tendon vibration in man: A microneurographic study. *Experimental Brain Research*, 76, 213–222.
- Selen, L. P., Beek, P. J., & van Dieën, J. H. (2005). Can co-activation reduce kinematic variability? A simulation study. *Biological Cybernetics*, 93, 373–381.
- Selen, L. P., Beek, P. J., & van Dieën, J. H. (2006). Impedance is modulated to meet accuracy demands during goal-directed arm movements. *Experimental Brain Research*, 172, 129–138.
- Selen, L. P., van Dieën, J. H., & Beek, P. J. (2006). Impedance modulation and feedback corrections in tracking targets of variable size and frequency. *Journal of Neurophysiology*, 96, 2750–2759.
- Sherwood, D. E., Schmidt, R. A., & Walter, C. B. (1988). The force/force-variability relationship under controlled temporal conditions. *Journal of Motor Behavior*, 20, 106–116.
- Slifkin, A. B., Vaillancourt, D. E., & Newell, K. M. (2000). Intermittency in the control of continuous force production. *Journal of Neurophysiology*, 84, 1708–1718.
- Stokes, I. A., Henry, S. M., & Single, R. M. (2003). Surface EMG electrodes do not accurately record from lumbar multifidus muscles. *Clinical Biomechanics*, 18, 9–13.
- Tseng, Y. W., Scholz, J. P., Schöner, G., & Hotchkiss, L. (2003). Effect of accuracy constraint on joint coordination during pointing movements. *Experimental Brain Research*, 149, 276–288.
- van der Hulst, M., Vollenbroek-Hutten, M. M., Schreurs, K. M., & Hermens, H. J. (2010). Relationships between coping strategies and lumbar muscle activity in subjects with chronic low back pain. *European Journal of Pain*, 14, 640–647.
- van Dieën, J. H., Selen, L. P., & Cholewicki, J. (2003). Trunk muscle activation in low-back pain patients, an analysis of the literature. *Journal of Electromyography and Kinesiology*, 13, 333–351.
- Visser, B., de Looze, M. P., Veeger, D. H., Douwes, M., Groenesteijn, L., de Korte, E., et al (2003). The effects of precision demands during a low intensity pinching task on muscle activation and load sharing of the fingers. *Journal of Electromyography and Kinesiology*, 13, 149–157.
- Willigenburg, N. W., Kingma, I., & van Dieën, J. H. (2010). How is precision regulated in maintaining trunk posture? *Experimental Brain Research*, 203, 39–49.