Chapter 3

Control of trunk movement: precision, pain and proprioception

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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 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. Performance on the tracking task 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 and agonistic muscle activation were similar between groups. Tracking errors increased with trunk inclination, but no significant relation with 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-activation.
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**Introduction**

Precise motor control is hampered by neuromuscular noise. The exact origin of this noise is still unknown [39, 41, 80], but synaptic noise resulting in fluctuations in motor unit firing rates and firing intervals is suggested to be one of the main causes [42, 81]. Neuromuscular noise is signal-dependent, in that force variability increases with muscle activation level [40-41, 82-87]. 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 [46]. It has been suggested that this is due to the effect of nociceptive afferents on muscle spindle feedback, which would impair proprioception [51]. In the study on shoulder pain the reduced precision indeed coincided with a reduced proprioceptive acuity in the pain group [46]. 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 [88-89]. 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 [90]. 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 [52-54]. 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 [33], 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 [42]. In upper extremity tracking tasks, increased precision indeed coincided with increased joint impedance [48-49] and EMG activity [46], 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 [91]. 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 [92-93]. 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 these tracking tasks 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. Antagonistic muscle activation was hypothesized to be higher in the patient group.

Methods

Subjects

Eighteen subjects with a-specific LBP (31 ± 14 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.
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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 [94], no other conditions (e.g. neurological or mental disorders, allergy to plaster) hindering participation or performance, 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.

Experimental setup

Subjects maintained a semi-seated/kneeded position with their pelvis fixed and their lower legs supported by a frame (Figure 3.1). 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. 10ms) 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° (lateral flexion to the right). The Y-axis ranged from -20° (extension) to +40° (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 object was presented on the screen. This target 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 seconds in advance.
Electromyography (Porti 17, TMS, Enschede, The Netherlands; 22 bits AD conversion after 20x amplification, input impedance >$10^{12}$ Ω, 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 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.

Figure 3.1. Experimental setup, by Paul van Drunen.
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 (Figure 3.2). The vibration motor was fixed with neoprene elastic bands and vibration was applied continuously during the experimental task.

Figure 3.2. 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.

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 spiral-shaped trajectory had its centre 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 object 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=0:0.005:10π. Speed of trunk movement was normalized to a constant change in trunk angle of 1.76°/s, and task took about 2 minutes. The outer boundaries of the spiral corresponded to approximately 10 degrees extension and lateral flexion and 17 degrees flexion. Two trials started in the center of the spiral and consisted of
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5 counterclockwise rotations with increasing amplitude. In two additional trials, the target started in maximum lateral flexion to the right and 5 clockwise rotations with decreasing amplitude were made. 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 and subjects were instructed not to rotate around the longitudinal axis.

Data analysis

Matlab R2010a was used for data analysis. Tracking error was defined as the absolute difference between trunk angle and target center at each instant of time. Specifically, we calculated the hypotenuse of the tracking error in the frontal and sagittal planes of motion. Trunk inclination was defined as the amplitude of the required trunk angle (target center) with respect to the neutral posture (again, the hypotenuse of the frontal and sagittal planes angles). EMG data were 30 Hz high-pass filtered to remove contamination from the electrocardiogram [95] 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 (2\textsuperscript{nd} order Butterworth), uni-directionally in order to correct for electromechanical delay [65]. Trials with lumbar muscle vibration were discarded from EMG analyses, since some EMG signals were contaminated by the vibration. Both tracking errors 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 (Figure 3). A running average was calculated to reduce the sample rate of both tracking error and EMG linear envelopes to 2 samples/s. 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.

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 (Figure 3.3). 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 of 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 agonists (Rback_ago) and the left abdominal muscles were defined as 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 agonist or antagonist.

Figure 3.3. 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.
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 (Figure 3.4).

To detect differences in tracking error 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 this 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 agonist or as antagonist, with zero values for periods when the muscle group did not act in that function. Again, two-way interactions with the factor ‘group’ were examined, but removed when not significant.
Results

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

Results for GEE model 1 (Table 3.1), 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 27.1% and significantly larger in LBP patients compared to healthy controls. 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 in the control group, while the patient group showed a slight (but not significant) decrease in tracking error in conditions with lumbar muscle vibration (Figure 3.5). Furthermore, as hypothesized, tracking errors significantly increased with trunk inclination.

Table 3.1. Results of GEE model 1.

<table>
<thead>
<tr>
<th></th>
<th>Coefficient</th>
<th>95% confidence interval</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>0.304</td>
<td>0.302 – 0.362</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Group (LBP=1)</td>
<td>0.090</td>
<td>0.036 – 0.144</td>
<td>0.001</td>
</tr>
<tr>
<td>Vibration (on=1)</td>
<td>0.035</td>
<td>0.006 – 0.064</td>
<td>0.016</td>
</tr>
<tr>
<td>Trunk inclination</td>
<td>0.004</td>
<td>0.002 – 0.006</td>
<td>0.001</td>
</tr>
<tr>
<td>Group X vibration</td>
<td>-0.048</td>
<td>-0.086 – -0.011</td>
<td>0.012</td>
</tr>
</tbody>
</table>

Figure 3.5. Averages and SDs (error bars) of tracking errors for both groups and conditions.

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.

**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 \(p\geq0.184\)), so no differences between LBP patients and healthy controls were found in the effects of agonist and antagonist activation on precision. Therefore, interactions were not included in the final model (Table 3.2). 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.

*Table 3.2. Results of GEE model 2, with the average (±SD) values for each EMG covariate included in the second column.*

<table>
<thead>
<tr>
<th>Covariate</th>
<th>Mean ± SD</th>
<th>Coefficient</th>
<th>95% confidence interval</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>0.263</td>
<td></td>
<td>0.218 – 0.307</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Group (LBP=1)</td>
<td>0.112</td>
<td></td>
<td>0.052 – 0.173</td>
<td>0.001</td>
</tr>
<tr>
<td>Rabd_ago</td>
<td>0.25 ± 0.10</td>
<td>0.175</td>
<td>-0.071 – 0.420</td>
<td>0.162</td>
</tr>
<tr>
<td>Labd_ago</td>
<td>0.26 ± 0.10</td>
<td>0.171</td>
<td>-0.010 – 0.352</td>
<td>0.065</td>
</tr>
<tr>
<td>Rabd_anta</td>
<td>0.19 ± 0.10</td>
<td>0.224</td>
<td>0.038 – 0.409</td>
<td>0.018</td>
</tr>
<tr>
<td>Labd_anta</td>
<td>0.18 ± 0.10</td>
<td>0.274</td>
<td>0.074 – 0.474</td>
<td>0.007</td>
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<tr>
<td>Rback_ago</td>
<td>0.40 ± 0.11</td>
<td>0.001</td>
<td>-0.074 – 0.076</td>
<td>0.980</td>
</tr>
<tr>
<td>Lback_ago</td>
<td>0.41 ± 0.10</td>
<td>0.004</td>
<td>-0.065 – 0.073</td>
<td>0.902</td>
</tr>
<tr>
<td>Rback_anta</td>
<td>0.17 ± 0.07</td>
<td>0.263</td>
<td>-0.041 – 0.566</td>
<td>0.090</td>
</tr>
<tr>
<td>Lback_anta</td>
<td>0.17 ± 0.07</td>
<td>0.270</td>
<td>-0.143 – 0.683</td>
<td>0.200</td>
</tr>
</tbody>
</table>

**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. 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 was not affected by 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 [52-54, 90], 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. Although ratios of co-activation were calculated with non-normalized EMG amplitudes, the successful matching between subject groups with respect to BMI (and age) allowed for comparisons between groups. Previous studies reported trunk muscle activation patterns apparently aimed at trunk stiffening in LBP patients during other tasks [33]. However, we did not find evidence for the use of such a strategy in the current study.

The finding that tracking errors significantly increased with trunk inclination seems to support previous findings on signal dependent force variability [40-41, 82-87]. 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
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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 trunk muscle activation did not reduce tracking error is in contrast with previous findings on precision control in the upper extremity [44, 46, 48-49], but supports a recent suggestion based on static trunk control [91], that precision of the trunk is regulated by feedback corrections rather than by a feedforward (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 [70-71]. However, surface electrodes at the reported locations probably reflected some of these deeper muscles’ activity [75] 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 might be the EMG normalization procedure. We did not normalize to (sub)maximal activation during isometric voluntary contractions, because this involves the risk of introducing a bias between patients and healthy controls [33]. The ratio of co-activation levels was therefore calculated with non-normalized data, separately for periods of trunk flexion and extension. 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. The current normalization method (to the highest value recorded during
the eight measurements) used in GEE model 2 did not allow for direct comparisons between healthy controls and patients in terms of trunk muscle activation levels. These comparisons were not required for testing our hypotheses on effects of agonist and antagonist activation on precision, so this limitation did not affect current results.

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 subjects. These results suggest that LBP is associated with proprioceptive impairments. Antagonistic co-activation 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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