**Summary**

**Trunk control in low back pain**

Low back pain (LBP) is a large health and socio-economic problem and current interventions for prevention and treatment of LBP have limited success. Indications for changes in motor control in LBP patients have been reported, but the nature of the relationship between LBP and altered motor behavior is poorly understood. For instance, it has remained unclear whether changes in motor behavior between LBP patients and healthy individuals are a cause or a consequence of LBP. In addition, the mechanisms behind these changes have not been revealed.

This thesis aims at a better understanding of motor control deficits in LBP patients. Therefore, we evaluated the quality of trunk control during a variety of tasks and experimental conditions in subjects with and without LBP. In a part of the trials, we disturbed lumbar proprioceptive feedback by bilateral paraspinal muscle vibration. We measured precision and accuracy of trunk control and we measured trunk muscle activation by surface electromyography, to evaluate recruitment strategies.

In chapter 2, we assessed how healthy subjects regulate precision of trunk control. To this end, they received visual feedback of trunk angle in the frontal and sagittal planes of motion and they were instructed to stay within a predefined target area. By changing the location of the target, we imposed different trunk postures and by changing the dimensions of the target, we manipulated the precision demands in the frontal and sagittal planes of motion. During the measurements, visual feedback was only present when subjects left the target and five different targets (horizontal and vertical rectangle and small, medium and large square) were used at five different locations (self-chosen neutral posture, 20° flexion, 10° extension and 10° lateral flexion to the left and right). Results from the rectangular targets showed that subjects controlled their trunk directionally specific, limiting kinematic variability in the plane where the precision demand was high, while using the available space in the other direction. No effects of target dimensions on EMG amplitudes were found, which implied that precision was not increased by increasing trunk muscle co-activation. Rather, it appeared that subjects waited until they left the target and then adjusted their trunk posture in order to get back in target again. Unpublished data revealed that LBP patients also show this directionally specific trunk control without increasing antagonistic co-activation to limit kinematic variability.
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In chapter 3, we evaluated precision and accuracy of trunk control during a tracking task that required circular trunk movements. Subjects with and without LBP performed this task in conditions with and without lumbar muscle vibration. We found that tracking errors were larger in LBP patients compared to healthy controls, and that lumbar muscle vibration caused tracking errors to increase 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. Furthermore, tracking errors increased with trunk inclination and did not decrease when antagonistic muscle activation increased. So again we concluded that, neither healthy subjects, nor LBP patients counteract trunk movement errors by increasing antagonistic co-contraction.

In chapter 4, we assessed control of an upright trunk posture in conditions with high and low precision demands, again with and without lumbar muscle vibration in subjects with and without LBP. Similar to chapter 2, a cursor on a computer screen reflected the trunk angle in the frontal and sagittal planes and subjects were instructed to stay within a target area. Again, visual feedback was only provided when they left the target. The precision demand was manipulated by changing target size (small or large square) and the location of the target was always the center of the screen, reflecting a self-chosen neutral posture. With the small target, patients controlled their trunk as accurately and precisely as healthy controls. For the large target, subjects mostly stayed within the target, but patients showed a larger postural drift compared to healthy controls. Lumbar muscle vibration deteriorated control over trunk posture in both groups and ratios of antagonistic co-activation did not differ between groups or conditions. These results indicate that, in contrast to the task described in Chapter 3, the weighting of proprioceptive feedback from lumbar muscle spindles did not differ between groups. However, low back pain patients were less able to detect low frequency drift in posture.

In chapters 2, 3 and 4, precise trunk control was the explicit aim of the tasks. In chapter 5, subjects with and without LBP performed a seated balancing task, which implicitly requires adequate trunk control to achieve a higher goal: to limit movement of the chair. We recorded excursions of the center of pressure, lumbar kinematics and trunk muscle electromyography, in conditions with and without disturbance of lumbar proprioception and occlusion of vision. We found that the effects of proprioception
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disturbance and vision occlusion were similar between groups. Interestingly, low-back pain patients grabbed the safety rail more often, while differences between groups in sway measures were rather subtle. This suggests that low-back pain patients were more cautious. Furthermore, LBP patients showed a more upright lumbar posture than healthy individuals. In contrast to our hypotheses, LBP patients made larger thoraco-lumbar movements in the sagittal plane and activation of the intersegmental longissimus relative to the iliocostalis muscle, which spans all lumbar segments, was lower in low-back pain patients compared to healthy individuals. This difference in muscle activation may be a cause or a consequence of the larger thoraco-lumbar movements and could be related to the difference in trunk posture.

To improve interpretation of trunk muscle EMG in future studies, we proposed and evaluated a method to remove ECG from EMG recordings based on independent component analysis (ICA) in chapter 6. To mimic realistic contamination while having uncontaminated reference signals, we employed EMG recordings from peripheral muscles with different activation patterns and superimposed distinct ECG signals that were recorded during rest at conventional locations for trunk muscle EMG. ICA decomposition was performed with and without a separately collected ECG signal as part of the data set and contaminated ICA modes representing ECG were identified automatically. We found that ICA-based filtering largely preserved the EMG’s spectral content. Performance on amplitude measures was especially successful when a separate ECG recording was included. Our test protocol revealed important differences between procedures for ECG removal, which resulted in recommendations for choosing the optimal technique given a specific data set and outcome measure of interest.

In chapter 7, we discussed the main findings of the preceding chapters in relation to each other. The results of these studies contributed to a better understanding of motor control deficits in LBP patients. Differences in quality of trunk control between subjects with and without LBP are quite subtle and seem to relate to reduced weighting and/or quality of proprioceptive information from lumbar muscle spindles. No indications for increased trunk stiffening were found in LBP patients. Moreover, no stiffening strategy was used to increase precision of trunk control. ICA-based ECG removal can improve interpretation of trunk muscle EMG in future studies aimed at elucidating the complex relation between motor behavior and LBP.