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Aim of the thesis

The aim of this thesis is to determine what mechanisms can determine axial thorax-pelvis coordination during gait and to assess which of these mechanisms are adopted by patients with low back pain.

Thesis outline

We started this thesis by searching the literature for studies that assessed responses to mechanical trunk perturbations in low back pain as these studies could provide evidence for splinting in low back pain. We were primarily interested in axial trunk stiffness during gait, however, at the time of the search, no studies were published that perturbed low back pain patients during gait. Studies that described the effect of trunk perturbations during other activities, did not perturb the trunk around the longitudinal axis. Since splinting might be used by chronic low back pain patients over a wide range of activities and since splinting could reduce the effect of external perturbations in multiple planes, we did not restrict our search to the anatomical plane in which perturbations were imposed or the activity the subjects had to perform during the perturbation. The results of this systematic literature review are described in **Chapter 2**.

Since no published data on axial trunk perturbations during gait in low back pain patients were available, we performed an experiment in which we perturbed the trunk of fifteen chronic low back pain patients and fifteen healthy controls indirectly using axial perturbations of the walking surface. This experiment is described in **Chapter 3**.

To evaluate if increased axial trunk stiffness would cause more in-phase thorax-pelvis timing we constructed a forward dynamic model of the trunk. Using this model, we could estimate apparent axial trunk stiffness from experimental data and study the isolated effect of increased axial trunk stiffness on thorax-pelvis timing. The results of this experiment are described in **Chapter 4**.

In the first experimental chapter of this thesis (i.e. Chapter 3), we found an association between axial pelvis range of motion and thorax-pelvis relative phase, which was previously also reported in a different cohort. To evaluate if increased pelvis range of motion could cause more in-phase thorax-pelvis timing we performed the experiment described in **Chapter 5**, using the model introduced in the previous chapter.

In **Chapter 6** we evaluated what mechanisms, among which splinting, reduce stride-to-stride variability of axial trunk rotations in low back pain patients during gait.

Finally, in **Chapter 7**, the results are synthesized, and recommendations for future research and clinical practice are given.

Thesis Summary

In **Chapter 2,** a review of low back pain perturbation studies, a consistently longer delay in trunk EMG onset in response to perturbations was found in low back pain, but no other signs of increased apparent trunk stiffness. As a result of methodological shortcomings in many of the included studies, it is difficult to draw firm conclusions from this review. The observed delay in EMG onset could be the result of differences in baseline EMG, used to calculate these onsets. Hopefully, our suggestions to improve future study designs and data analysis techniques are adopted by future studies, so that more definitive conclusions about reactive trunk motor control in chronic low back pain can be drawn.

We found no signs of increased apparent axial trunk stiffness in chronic low back pain in response to platform perturbations during gait in **Chapter 3**. More in-phase thorax-pelvis timing was not associated with a differential effect of external platform perturbations on trunk movements compared to more out-of-phase thorax-pelvis timing, which would suggest that more in-phase axial thorax-pelvis coordination is not the result of increased apparent axial trunk stiffness.

In **Chapter 4** we demonstrated that an isolated increase in apparent axial trunk stiffness would result in more in-phase thorax-pelvis timing during gait, but apparent axial trunk stiffness could not be predicted from experimentally observed thorax-pelvis relative phase. This can be partially explained by the large effect of arm swing on thorax-pelvis coordination. We found no significantly higher values of apparent axial trunk stiffness in low back pain patients compared to healthy controls, and previous studies reported no significantly altered arm swing amplitude during gait in low back pain. Therefore, more in-phase thorax-pelvis timing during gait in low back pain appears to be caused by a different mechanism.

In **Chapter 5,** we demonstrated that increased pelvis range of motion causes more in-phase thorax-pelvis timing, regardless of apparent axial trunk stiffness. This is in line with our findings from Chapter 3, where we observed that subjects with chronic low back pain with relatively in-phase thorax-pelvis timing demonstrated significantly larger pelvis range of

motion than healthy subjects with relatively out-of-phase thorax-pelvis timing. Increased pelvis range of motion has been observed in combination with more in-phase thorax-pelvis timing in other studies as well.

In **Chapter 6** we demonstrated that, in contrast to axial thorax-pelvis relative phase, the reduced stride-to-stride variability of axial trunk rotations during gait in low back pain *does* appear to be partially caused by splinting. The (non-significantly) increased apparent axial trunk stiffness and damping in patients resulted in a larger resistance to high-frequency external pelvis perturbations, reducing the amplitude of residual trunk rotations. Low back pain patients appear to refrain from volitional low-frequency trunk movements, which causes the lower stride-to-stride variability of axial trunk rotations at low frequencies.