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Background

Low back pain

With a worldwide point prevalence of 12% and a one-month prevalence of 23% [1], low back pain (LBP) constitutes a large health and socio-economic problem. In the Netherlands, the one-year prevalence was 44% in 1998, with over 85% of the respondents who indicated to have had LBP reporting recurrent or continuous symptoms [2]. Only in 10% of the cases a specific diagnosis is made, and the vast majority is thus labeled as ‘a-specific’ LBP [3]. In 2007, the total costs of LBP in the Netherlands were 3.5 billion euro, consisting of 12% direct costs (e.g. related to medication and visiting a general practitioner or physical therapist) and 88% indirect costs (e.g. associated with production loss and disability) [4].

Mechanical loading of the spine has been reported to be an important risk factor for developing LBP [5-7]. Therefore, ergonomic interventions, aimed at both primary and secondary prevention, typically address trunk postures and movements during work, with special attention for lifting objects and working with flexed or twisted trunk postures. Furthermore, several psychosocial risk factors for LBP, such as job satisfaction, lack of social support and fear of movement, have been reported [8-10]. However, evidence for a primary causal role of psychosocial factors is weak [11-12]. Still, current guidelines for management of LBP recommend early identification of psychosocial factors that could prevent rapid recovery [13-14]. Over the last years, motor behavior has gained increasing interest as potential risk factor for the development and recurrence of LBP.

Despite the growing body of knowledge on changes in motor behavior coinciding with LBP, the relationship between LBP and motor behavior is still poorly understood. Specifically, we do not know whether changes in motor behavior are causal for LBP or whether motor behavior changes as a consequence of LBP. In the latter case, affected motor behavior could reflect an adaptive strategy to reduce LBP, but it could also promote recurrence or chronicity of LBP. So, while several indications for affected motor behavior in LBP patients have been reported, we do not understand the nature of these differences between LBP patients and healthy subjects. Obviously, insight in the nature of these differences is needed for adequate prevention and treatment of LBP. Current therapies involve a large variety of exercises mainly aimed at trunk muscle strengthening and
enhancing motor control. While such therapies show some success, effect sizes are rather small [15]. Therefore, the need for a better understanding of affected motor behavior in LBP patients was the starting point of this thesis.

**Control of trunk posture**

When studying trunk control, we consider the pelvis and the thorax as rigid segments, with the pelvis consisting of the sacral and iliac bones, and the thorax consisting of the thoracic spine, ribcage and sternum. Five lumbar vertebrae connect these rigid segments, with intervertebral discs in between (Figure 1.1). This system is inherently unstable and, without additional support, would buckle under compression forces of about 90 N [16], which is less than the compression forces in upright standing and only a fraction of the compression forces during lifting [17-18]. About two decades ago, Panjabi suggested that three subsystems provide the support to prevent the human spine from buckling [19]. The passive subsystem consists of the vertebrae, discs and ligaments that surround the spine. The active subsystem consists of the abdominal and back musculature. The neural subsystem consists of the central and peripheral nervous system, which generates and transfers sensory information and motor commands. This thesis focuses mainly on the active and the neural subsystems that ‘act on’ a given passive subsystem.

![Schematic representation of the pelvis, five lumbar vertebrae with intervertebral discs and a part of the thorax.](image)

*Figure 1.1. Schematic representation of the pelvis, five lumbar vertebrae with intervertebral discs and a part of the thorax.*
With regard to the active subsystem, we are interested in how the trunk muscles (Figure 1.2) control trunk posture and movement. Therefore, trunk muscles are clustered in muscle groups with specific functions. The most obvious distinction between trunk muscles in terms of functioning is grouping the abdominal muscles, which generate a flexion moment, versus the back muscles, which generate an extension moment. Similarly, muscles on the right side of the trunk (generating lateral flexion moments to the right) can be distinguished from muscles on the left side of the trunk (generating lateral flexion moments to the left). Such muscle groups with opposite functions are called antagonists. Another distinction between trunk muscles can be made based on their relative distance to the lumbar spine. Deep muscles (e.g. m. transversus abdominis, mm. multifidi) have relatively small moment arms with respect to the spine compared to superficial muscles (e.g. m. rectus abdominis, m. latissimus dorsi), which are located closer to the skin surface. When the moment arm is small, a similar muscle force results in smaller moments, which might make deep muscles more suitable for subtle motor control. Another division can be made based on the insertions of the different muscles. Intersegmental muscles (e.g. m. longissimus) have multiple segmental origins and insertions causing the muscles to span only one or a few spinal segments. These muscles are also referred to as ‘local’ muscles and allow for subtle control of intersegmental movements. Moreover, these muscles may function as guy wires that stabilize the lumbar spine and thereby prevent buckling [20]. Multisegmental muscles (e.g. m. iliocostalis), on the other hand, span multiple spinal segments and have larger moment arms. Therefore, these ‘global’ muscles would be more suited for making larger trunk movements and less for subtle trunk control.

*Figure 1.2. Schematic representation of the trunk musculature in the transversal plane.*
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Regarding the neural subsystem, this thesis compares the role of lumbar muscle spindles in the control of trunk posture and movement between LBP patients and healthy individuals. These sensory organs, which are present in each muscle, are sensitive to changes in length and rate of lengthening of the muscle. This information on changes in muscle length plays an important role in proprioception: how we ‘feel’ our joint angles and movements. Afferent feedback from muscle spindles can be disturbed by local muscle vibration. Specifically, vibration causes proprioceptive information from muscle spindles similar to that due to muscle lengthening, thus inducing a lengthening illusion. This experimental manipulation can be used to evaluate the relative importance of proprioceptive information from lumbar spindles for motor control. In addition to proprioceptive feedback, information from the visual and vestibular systems is available. The vestibular system is sensitive to the orientation and movement of the head/trunk with respect to the gravitational field. The visual system provides information on the orientation and movements of the head/trunk in space. The neural subsystem combines the information from these three different sources to obtain an estimate of the orientation and movements of the trunk [21].

Motor control in LBP patients

An example
One of the most basic activities of daily life is upright standing. The ability to control such an upright posture can be evaluated by measuring center of pressure (CoP) trajectories during stance. A reduced precision in maintaining a static orientation of the trunk will cause increased postural sway. A recent review concluded that the majority of studies comparing CoP trajectories between LBP patients and healthy individuals report increased postural sway in LBP patients, or no effect of LBP on postural sway [22]. Interestingly, a minority of studies reports decreased sway in LBP patients, while no obvious systematic differences were found between studies with conflicting results. These inconsistent findings made the authors suggest that changes in motor control may be the result of potentially conflicting factors, such as pain, which could cause increased sway, and fear of pain, which could evoke more rigid motor behavior, thus less sway.
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**Potential mechanisms**

In an attempt to elucidate which aspect of motor control is impaired in LBP patients, Radebold and colleagues studied responses to sudden unloading of the trunk as well as seated balancing in subjects with and without LBP [23]. It was found that LBP patients demonstrated poorer balance performance than healthy individuals, coinciding with delayed muscle responses to sudden force releases. A correlation between these phenomena was suggested to reflect a causative role of the delays in the reduced control over the lumbar spine. However, with a sudden release experiment, measuring motor responses in a closed-loop system, it is impossible to determine the exact nature of the problem, as will be discussed below.

Roughly, these differences between groups could be explained by differences in initial state, by affected detection or processing of sensory information or by impaired muscle recruitment. If, for instance, trunk stiffness were higher in LBP patients (difference in initial state), a similar force perturbation would evoke a smaller response in terms of movement amplitude. Alternatively, if detection or processing of sensory information is impaired in LBP patients, information on the size and type of the perturbation would be less accurate, resulting in a less adequate response. If initial state and sensory processing are unaffected in LBP patients, a difference in response to force perturbations could be explained in terms of muscle recruitment. In that regard, several studies found indications for pain related inhibition, quantified by a reduced motor unit discharge rate [24-25] and by reduced muscle activation or muscle force [26-28].

This thesis focuses on the first two above-mentioned mechanisms behind affected trunk motor control in LBP patients, without going into detail on pain related inhibition of motor units. Specifically, we wonder (1) whether LBP patients have increased trunk stiffness and (2) whether sensory information detection/processing is affected in LBP patients. Before describing the objectives and outline of this thesis, the sections below first briefly discuss the current knowledge on increased trunk stiffness and impaired proprioception as potential mechanisms underlying motor control deficits in LBP patients.
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Trunk stiffening
The initial state of a mechanical system largely defines the response to a sudden perturbation. When trunk stiffness, and thereby the resistance to external perturbations, is increased, a similar force perturbation results in a smaller change of the initial position and thus requires a different response. Several studies reported indications for increased trunk stiffness in LBP patients. For instance, during walking, the coupling between pelvis and thorax movements is stronger in LBP patients than in healthy control subjects [29-30]. LBP patients also demonstrated reduced spinal movements preceding rapid bilateral shoulder flexion [31], which imposes a predictable perturbation to trunk equilibrium. Furthermore, LBP patients showed increased trunk stiffness in a sudden release experiment [32]. This suggests that adaptations to pain may have one common goal: protecting the affected area by stiffening the trunk [33]. If this is indeed the case, differences between LBP patients and control subjects in response characteristics to sudden perturbations, as well as differences in seated balancing performance [23, 34], could be the result of such a trunk stiffening strategy.

Stiffening of the trunk can be realized by antagonistic co-activation of trunk muscles. Simultaneous activation of abdominal and back muscles results in a corset-like structure surrounding the lumbar spine. Another way to stiffen the trunk may be preferential recruitment of deep rather than superficial muscles, or local rather than global trunk muscles [35-37]. Stiffening strategies may protect the spine by limiting intervertebral movements. However, trunk stiffening also has disadvantages. For instance, an increase in compression forces due to simultaneous abdominal and back muscle activation may hamper intervertebral disc nutrition and accelerate disc degeneration. Also, there may be loss of flexibility (i.e. the trunk becomes a rigid segment), which has to be compensated in other joints. Furthermore, physiological costs of such a strategy may be high, and muscles may be overexerted, especially when continuous co-activation is employed.

Evidence for changes in muscle recruitment that could be aimed at stiffening the trunk have been found in LBP patients. For instance, increased antagonistic co-activation and increased ratios of local over global muscle activation during slow voluntary trunk movements have been found in LBP patients [37]. Moreover, LBP patients showed increased erector spinae and rectus abdominis muscle activation levels during walking when compared to healthy controls [38].
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Joint stiffening has also been associated with the precision with which joint postures are maintained or movements are made. Precision of motor control is hampered by neuromuscular noise, which is signal-dependent [39-41]. So, kinematic variability generally increases with muscle activation levels. However, kinematic variability can be reduced by increasing joint stiffness through antagonistic co-activation, despite the increase of signal dependent noise [42]. Several studies found evidence for increased antagonistic muscle activation [43-47] or joint stiffening [48-50] to increase precision in the limbs. This thesis will evaluate whether a similar strategy is used to increase precision of trunk control, in LBP patients and healthy subjects.

Sensory information

Another potential source of differences in motor control between LBP patients and healthy controls could be an impairment of the sensory system. When the sensory system is affected, detection of positional changes due to perturbations will be hampered and, as a consequence, muscle responses will be less adequate. Indications for increased excitation of muscle spindle afferents due to intramuscular bradykinin injections (an experimental model for pain and inflammation) have been reported [51], suggesting that proprioception from lumbar muscle spindles may be affected in LBP.

In addition, several studies investigated the effect of muscle vibration on balance control [52-54]. Results indicated that the relative effect of vibrating calf muscles versus lumbar muscles differed between LBP patients and healthy individuals. Specifically, the change in posture due to calf muscle vibration relative to the change in posture due to lumbar muscle vibration was larger in LBP patients than in healthy control subjects. This finding indicates that LBP patients weight lumbar spindle information lower than information from spindles in the calf muscles, and thus supports the idea of impaired trunk proprioception. However, it remains unclear whether the quality of proprioceptive information is compromised, whether LBP patients just tend to ‘ignore’ this information, regardless of its quality, or whether a combination of reduced quality and reduced weighting occurs in LBP patients. Furthermore, the above-mentioned findings in postural sway during upright standing only provide indirect evidence as there are a number of joints in between the affected area (the low back) and the location where effects are measured (the ground reaction force).
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Objectives of this thesis

The main goal of this thesis is to elucidate motor control deficits in LBP. Therefore, precision and accuracy of trunk control are evaluated as measures of quality of trunk control in LBP patients and healthy individuals. Precision is high when the variability of trunk movement is low, whereas accuracy is high when the mean difference between the desired and actual trunk movement or posture is small. We used several experimental setups and experimental tasks requiring precise and accurate trunk control, in order to unravel the differences between LBP patients and healthy individuals. The experiments were performed under highly controlled conditions, with the upper and lower extremities restricted and without external perturbations to the trunk. Sensory information was manipulated by lumbar muscle vibration and vision occlusion. Kinematics and trunk muscle activation were recorded to gain insight in the effects of pain and proprioception disturbance on trunk control.

Outline of this thesis

Chapter 2 describes a study on precision control of the trunk in healthy participants. Subjects maintained a semi-seated position with their pelvis fixated in the experimental setup. They received real-time visual feedback of trunk angle and were instructed to stay within a target area. By manipulating the location and dimensions of the target area, we assessed the reduction of kinematic variability in different trunk postures in the sagittal and frontal planes of motion. We hypothesized that the directionally specific precision demands are met by increasing antagonistic co-activation.

In chapter 3, subjects with and without LBP performed a tracking task that required circular movements of the trunk, while their pelvis was fixated. To evaluate differences between groups in the use of lumbar proprioception, subjects performed this task with and without paraspinal muscle vibration. Given the signal-dependency of neuromuscular noise, we hypothesized that tracking errors increase with agonistic muscle activation, and thus with trunk inclination. Assuming proprioceptive impairments in LBP, we further hypothesized that LBP patients make larger tracking errors and show more antagonistic co-activation compared to healthy subjects and that the effect of proprioception disturbance is larger in healthy individuals.
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Chapter 4 compares precision control of the trunk in an upright trunk posture between LBP patients and healthy control subjects, again in a semi-seated position with pelvis fixation. As in chapter 2, precision demands were manipulated by changing the target dimensions, and, as in chapter 3, the task was performed with and without lumbar muscle vibration. Again, we hypothesized that trunk control is less precise and antagonistic co-activation is higher in LBP patients, while the effect of lumbar muscle vibration is larger in healthy individuals.

In chapters 2, 3 and 4, precise trunk control was the explicit aim of the tasks. As a step towards more realistic tasks, with control of trunk posture as a means to achieve a higher goal, chapter 5 describes differences between subjects with and without LBP during a seated balancing task. In addition to conditions with and without lumbar muscle vibration, subjects also performed this task with their eyes closed. Trajectories of the center of pressure, trunk kinematics and muscle activation strategies were compared between groups. We hypothesized poorer balance control, coinciding with higher co-activation ratios and smaller thoraco-lumbar movements in LBP patients. Moreover, we hypothesized interactions between group and conditions, with the effects of vision occlusion being larger in LBP patients and the effects of paraspinal muscle vibration being larger in healthy individuals.

To improve the interpretation of trunk muscle EMG in future studies, chapter 6 compares several methods to remove contamination from the electrocardiogram from EMG recordings and describes a promising alternative to currently used methods. Finally, chapter 7 discusses the major findings of these studies in relation to each other and provides recommendations for future studies.