The use of technology in measuring low back function and morphology in low back pain patients
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Chapter 1

General Introduction
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General introduction

We start this thesis with the description of a clinical scenario of a 39-year-old woman who experienced low back pain (LBP).

Clinical scenario

A 39-year-old woman has been experiencing low back pain (LBP) for 4 weeks. She has suffered from short episodes of low back pain before; however, her pain now does not seem to dissipate. This time, her pain radiates from the low back to the dorsal side of the upper leg above her right knee. The pain increases during the day, especially with loading (bending, lifting, starting to move after sitting), and the pain in the low back and dorsal side of the right upper leg is predictable (biomechanical pattern). However, she also experiences unexpected shooting pain in her low back a couple of times per day.

She presented to her general practitioner (GP), who initially prescribed pain medication (non-steroidal anti-inflammatory drugs). Unfortunately, this did not reduce the pain. Next, her GP referred her for X-rays, which indicated some degree of osteoarthritis with disk space narrowing at levels L4-L5 and L5-S1. The patient was worried about the diagnosis and went to the physiotherapist (Anne) to ask: “The X-ray of my low back showed some degeneration and wear of my vertebras. Can I continue my work as a home care nurse?” Because she had become anxious and had doubts about her recovery and thought that her work caused the ongoing low back and leg pain, she reduced her working hours and the intensity of activities of daily living (ADL) at home. She also asked another physiotherapist (Tukker) for advice.

Physiotherapist Anne: I would choose stabilization training (lumbar multifidus training) in this case, based on the instability lumbar spine score from the Hicks Clinical Prediction Rule.¹ The patient has pain on flexion and de-flexion, and the Prone Instability test was positive. Her low back muscle mass and coordination should be trained before she does exercises for her upper and lower body. She should start with easy-to-perform low dose exercise with the focus on improving motor control and improving her trust in her body/low back. I would monitor the low back muscle mass by measuring the lumbar multifidus with an ultrasound.

Physiotherapist Tukker: Based on the ongoing pain accompanied by dysfunctional perceptions and anxiety, I would choose cognitive functional training.²,³ Scores on questionnaires like the Illness Perception Questionnaire (IPQ), Self-efficacy (Low Back Activity Confidence Scale), and Fear-Avoidance Belief Questionnaire (FABQ) motivate that decision. Emphasis would first lie on education to improve the patient’s insight into her pain neurophysiology and to decrease anxiousness, and would later switch to graded activity to improve physical fitness and self-confidence.⁴ The physical training
would be focused on the optimal performance of functional tasks (ADL and work) with the use of external stimuli to improve motor learning. I would monitor the low back muscle activity during function training exercises by using surface electromyography.

This case focuses on one of the many patients confronted with LBP daily. LBP is a very common symptom experienced by people in all countries, independent of income level or age group. Worldwide, low back pain has the highest ranking in the years lived with disability (YLD) index, a score that increased by 54% from 1990 to 2015. In Europe, LBP has a reported lifetime prevalence up to 84%. Low back pain results in significant levels of disability and restrictions in daily activities, including the inability to work.

Approximately 90% of LBP is classified as multifactorial or “non-specific low back pain” (nLBP). The term nLBP is used when the cause is unknown, for example when there is no pathological or anatomical cause, when no specific nociceptive source can be determined, or when the anomalies cannot be related to the patient’s symptomatology. nLBP can be related to an accident, sprains, strains, physical impairments in morphology, such as a decrease in low back muscle thickness, or function of the low back, such as decreased trunk range of motion, as well as to psychosocial aspects, such as increased fear of movement. All these factors demonstrate the complex nature of nLBP.

Like the patient in our clinical scenario, most patients with nLBP are first seen in primary care. For patients in an acute phase, recovery from nLBP can be fast by following the physiotherapist’s suggested interventions or even without any intervention. However, 30-40% of patients do not completely recover in three to six weeks (with or without intervention), and their pain is persistent (chronic or recurrent). In most cases, the physiotherapist does not know which of the suggested interventions has contributed the most to the nLBP reduction in that individual patient. It is expected that the number of patients with recurrent episodes of nLBP will increase. Therefore, the need for physiotherapy treatments will likely increase linearly. For these reasons, there is a great need to improve evidence of treatment rationale for the clinical approach(es) to nLBP treatment at the individual patient level in physiotherapy research.

As described in the clinical scenario, two physiotherapists may choose a different treatment modality for a similar patient, with substantial reasoning and evidence for both approaches. For a physiotherapist, the first step in the diagnostic evaluation is to rule out specific underlying pathology for the development of the pain and low back dysfunction. This step is often rather straightforward. However, the next step is more complicated. The physiotherapist or primary care clinician has to observe and evaluate clinically relevant factors that could play a role in the development and persistence of nLBP, and which of these factors can be addressed by a certain treatment modality.
Chapter 1

However, there is limited knowledge about which risk factors or mechanisms play an essential role in patients with ongoing nLBP and how these variables can be validly identified in primary care. Nevertheless, many tests, including clinical ones, can provide information about different factors that may contribute to the nLBP of a patient. There are no clusters of tests that provide the solution for patients’ most critical limiting factors, leading to ambiguity on which intervention and which intensity or frequency to choose. Physiotherapist Anne, from the clinical scenario, based her choice for stabilization training on the clinical test results indicating a limited range of motion in trunk flexion with aberrant movements, a positive Prone Instability Test, and a straight leg raise test (> 90 degrees). On the other hand, physiotherapist Tukker based his choice for functional training on a high score on the FABQ, a score of 4 on the STarT Back Screening Tool, and a low self-efficacy score (Low Back Activity Confidence Scale). In addition, no single intervention will be sufficient to address all the different aspects. Therefore, an intervention such as exercise therapy has more than one rationale and can address different treatment objectives at the same time.

In the presented clinical scenario, the treatments of physiotherapists Anne and Tukker could both be helpful for this patient. There is evidence for the efficacy of both exercise therapy treatments, stabilization training such as lumbar multifidus (LM) training, and function-directed training (general exercise therapy). A reason for the choice of physiotherapist Anne is that the limited range of motion of trunk flexion may be caused by atrophy of the muscles that are important for stabilizing the lumbar spine. This LM training aims to increase the volume and coordination of the lumbar spine muscles, which can lead to better stabilization of the lumbar spine. The explanation of physiotherapist Tukker’s approach is that the patient has a dysfunctional/unrealistic view of the possible onset of her nLBP. The approach he uses is based on the operant conditioning theory, in which movement behavior is gradually increased with positive reward after each step. This behavioral model is prominent in the scientific literature and recommended in guidelines. Still, there may be little room for biological factors. However, a uniform theoretical background for the treatments proposed by both Anne and Tukker is lacking. Given that the LM is regarded as the major stabilizing muscle of the spine, the anatomy and topography of this muscle might offer at least some explanation for the opposing effects of stabilizing therapy.

It is assumed that the most substantial (about 70 percent) influence of spine stabilization is created by the LM. It is widely accepted in the literature that the LM contributes to the stabilization of the lumbar spine. However, in the literature, there are differences in descriptions of the morphology and function of LM. Several anatomical descriptions have appeared throughout the years. Already in 1986, LM was depicted as a deep and superficial muscle by Macintosh and colleagues. The LM is presented as a superficial muscle in the lumbar spine, according to Deng et al. 2015. On the other hand, the LM was described as a deep muscle by Fortin et al. 2015. These differences in descriptions
of the morphology and function of LM also have consequences for understanding the contribution of the LM in nLBP. Moreover, reaching a consensus about measurement tool(s) to measure the function and anatomy of the LM is a challenge. Tools such as electromyography, ultrasound, MRI, and X-rays are used to quantify LM morphology and function aspects. So, how should physiotherapists Anne and Tukker know which of the measurement tools they should use to measure the contribution of LM function in nLBP patients? What is the function of the LM in healthy subjects, and how does the LM contribute to pain and disability in patients with nLBP? What is the role of the LM in the underlying mechanisms resulting in nLBP and how does the LM relate to appropriate therapy for nLBP patients?

This thesis aims to increase our understanding of the role of the LM in nLBP patients. Due to the variety in the descriptions of the topography and morphology of the LM, Chapter 2 contains an overview of the topography and morphology of the LM depicted in anatomy atlases and literature. In Chapter 3, our description of the LM sonoanatomy, obtained from comparing high-resolution 3D digital spine reconstructions with standard LM ultrasonography, is depicted. After gaining more insight into the anatomy of the LM, the next step was to measure LM muscle activity. To this end, we studied intramuscular electromyography and surface electromyography of the LM and erector spinae, which is described in Chapter 4. This goal of this study was to gain insight into how well LM muscle activity can be measured with surface electromyography without crosstalk or co-contraction.

After these three studies describing and measuring LM functioning, we were interested in measuring specific variables in nLBP patients, i.e., LM function and morphology variables. In Chapter 5, a clinical study is described assessing differences between subacute nLBP patients, chronic nLBP patients, and healthy controls with regard to LM thickness, trunk range of motion, and LM muscle activity in primary care nLBP patients. Chapter 6 investigated and described low back morphology and function in nLBP patients to analyze the relationships with patient-reported measurement instruments. Finally, the results are summarized and discussed in Chapter 7.


General introduction


