HEALTH SCIENCES

Hannu Luomajoki

Movement Control Impairment as a Sub-group of Non-specific Low Back Pain

Evaluation of Movement Control Test Battery as a Practical Tool in the Diagnosis of Movement Control Impairment and Treatment of this Dysfunction

Publications of the University of Eastern Finland
Dissertations in Health Sciences

UNIVERSITY OF EASTERN FINLAND

HANNU LUOMAJOKI

Movement Control Impairment as a Sub-group of Non-specific Low Back Pain

Evaluation of Movement Control Test Battery as a Practical Tool in the Diagnosis of Movement Control Impairment and Treatment of this Dysfunction

To be presented by permission of the Faculty of Health Sciences, University of Eastern Finland for public examination in Auditorium of Orton Hospital, Helsinki on Friday 22.10.2010 at 12 noon

Publications of the University of Eastern Finland

Dissertations in Health Sciences

24

Institute of Clinical Medicine, School of Medicine, Faculty of Health Sciences

Department of Physiology / Sportmedicin

University of Eastern Finland

Kuopio

2010

Kopijyvä Oy
Kuopio, 2010

Distribution
Eastern Finland University Library/Sales of Publications
P.O. Box 1627, FI-70211 Kuopio, Finland
http://www.uef.fi/kirjasto

ISBN: 978-952-61-0191-0 (print)
ISBN: 978-952-61-0192-7 (pdf)
ISSN: 1798-5706 (print)
ISSN: 1798-5714 (pdf)
ISSNL: 1798-5706

Author's address: Zürich University of Applied Sciences (ZHAW)
Department of Health, Institute for Physiotherapy
Technikumstr. 71, 8401 Winterthur, Switzerland
luom@zhaw.ch

Supervisors: Docent Olavi Airaksinen, M.D., Ph.D.
Department of Physical and Rehabilitation Medicine
Kuopio University Hospital, Institute of Clinical Medicine
School of Medicine, Faculty of Health Sciences
University of Eastern Finland
Kuopio, Finland

Dr. Jan Kool, Ph.D.
Zürich University of Applied Sciences (ZHAW)
Department of Health, Institute for Physiotherapy
Winterthur, Switzerland

PD Dr. Eling D. de Bruin, Ph.D.
Institute of Human Movement Sciences and Sport
ETH Zurich
Zürich, Switzerland

Reviewers: Professor Jaro Karppinen, M.D., Ph.D.
Institute of Clinical Sciences
Department of Physical and Rehabilitation Medicine
University of Oulu
Oulu, Finland

Professor Arja Häkkinen
Department of Biology of Physical Activity
University of Jyväskylä
Jyväskylä, Finland

Opponent: Docent Markku Kankaanpää, M.D., Ph.D.
Department of Physical and Rehabilitation Medicine
Tampere University Hospital, Tampere, Finland

Luomajoki, Hannu. Movement Control Impairment as a Sub-group of Non-specific Low Back Pain: Evaluation of Movement Control Test Battery as a Practical Tool in the Diagnosis of Movement Control Impairment and Treatment of this Dysfunction. Publications of the University of Eastern Finland. Dissertations in Health Sciences 24. 2010. 70 p.

ABSTRACT

Low Back Pain (LBP) is one of the major current concerns in health care. In the majority of patients LBP is non-specific (NSLBP). The identification of different sub-groups of patients with NSLBP has high priority in improving assessment and developing tailored, more efficient treatments. One promising sub-group of LBP is Movement Control Impairment (MCI), which is defined as impaired active movement control of the lumbar spine during functional activities. A movement control test battery was developed to evaluate movement control ability. This doctoral thesis evaluates the movement control test battery in the diagnosis of MCI and in the treatment of this dysfunction. The thesis consists of five different studies. In the first study, the reliability of the developed test battery of ten tests was evaluated and six of them were confirmed. The second study demonstrated the consistency of two movement control tests performed on two different days. In the third study, the test battery of six most reliable tests was shown to be able to differentiate between patients with LBP and healthy controls. The fourth study revealed decreased two-point discrimination, as a measure of change in body schema in the brain of patients with decreased movement control. In the final study, patients with motor control impairment were treated specifically using motor control strategies in an uncontrolled design. The results indicate that specific exercises may decrease pain and disability among patients with MCI. The developed test set of six tests for movement control of the low back provides physiotherapists and medical doctors with an easy and reliable tool for examining whether a patient has normal or disturbed movement control. Movement control deficits can be treated with specific, targeted individual exercises. Further studies on the validation of the test set in different patient populations and especially randomised controlled studies are necessary.

National Library of Medicine Classification: WE 755

Medical Subject Headings: Low Back Pain, Reliability, Motor Skills, Physiotherapy

Luomajoki, Hannu. Liikekontrollin häiriö alaselkäkivun alaryhmänä: Liikekontrollin testipatteriston arviointi käytännöllisenä työvälineenä tämän häiriön diagnostisoinnissa sekä hoidossa. Itä-Suomen yliopiston julkaisuja. Terveystieteiden tiedekunnan väitöskirjat 24. 2010. 70 s.

TIIVISTELMÄ

Alaselkäkipu on yksi terveydenhuollon suurimmista ongelmista. Suurin osa näistä potilaista kärsii epäspesifisestä alaselkäkivusta. Alaryhmien tunnistaminen tässä potilasryhmässä on tärkeä tarkemman diagnostisoinnin sekä hoidon kannalta. Yksi mahdollinen alaryhmä on liikkeen kontrollin häiriö, joka tarkoittaa että potilaan aktiivisten liikkeiden kontrolli on heikentynyt. Tämän alaryhmän tunnistukseen kehitettiin testipatteristo, jonka luotettavuutta diagnostisointivälineenä sekä hoitokäytössä evaluoitiin tässä väitöskirjassa. Ensimmäinen osatutkimus totesi kuuden testin kymmenestä olevan luotettavan. Toisessa tutkimuksessa todettiin kahden liikekontrollitestin pysyvyys ajassa kahtena eri päivänä. Kolmannessa tutkimuksessa todettiin kuuden luotettavimman testin kykenevän merkitsevästi erottelemaan selkäkipuiset terveistä. Neljännessä tutkimuksessa todettiin, että liikkeen kontrollin häiriöisillä myös kahden pisteen erottelukyky on alentunut, mikä osoittaa aivojen kehonkuvan häiriintyneen. Viimeisessä tutkimuksessa liikekontrollin häiriöisiä alaselkäkipupotilaita hoidettiin spesifisti tämän ongelman parantamiseksi. Tämä ilman vertailuryhmää tehty tutkimus osoitti, että tähän alaryhmään kuuluvien alaselkäkipuisten kivut sekä toiminnallinen haitta saattavat olla hoidettavissa. Kehitetty testipatteristo on helppokäyttöinen ja luotettava työkalu lääkäreiden sekä fysioterapeuttien käyttöön tämän alaryhmän tunnistamisessa. Liikekontrollin häiriöitä voidaan hoitaa ja parantaa. Lisätutkimukset ovat tarpeen testipatteriston validisoimiseksi sekä hoidon vaikutusta on tutkittava vertailuryhmiä käyttäen.

Luokitus: WE 755

Yleinen suomalainen asiasanasto (YSA): Selkäsairaudet, motoriset taidot, luotettavuus, fysioterapia

Dedicated to my kids Liina and Tristan

Acknowledgements

I had never thought of becoming a scientist. Having worked for 13 years as a practising physiotherapist and teaching in a physiotherapy school, I went to Australia and finished 1999 my master studies in manipulative physiotherapy. The theme of my thesis was a systematic review of exercises in relation to Low Back Pain treatment. At that time, based on 62 RCTs on the subject, it was accepted that exercise is a good treatment for back pain. However, it seemed that it did not matter what kind of exercise was recommended. How could this be the case, I wondered, when our clinical experience was showing the contrary? The type of exercise, or therapy, undertaken is a very important issue. But, scientifically, it was just a hypothesis. It took me another five years to realize that physiotherapists – such as myself - should undertake research to prove or disprove this hypothesis. After five more years, and having undertaken the studies in this thesis, I can see that it might be possible to throw some light on this enigma of low back pain. And, during this process, I have become a scientist.

Many people helped me on this road to science. My greatest thanks go to associate Professor of University of East-Finland and clinical director of rehab services in the Kuopio University Hospital, Olavi Airaksinen, M.D., PhD, who was my principal supervisor. He believed in my project from the very first moment and motivated me along the way. He was truly a "Doktor-Vater" (doctor father), as the Germans say. My other supervisors in Switzerland were two Dutch PhD physiotherapists, Dr. Jan Kool and Dr. Eling de Bruin. Jan helped me practically on a daily basis. I thank you for your strong nerves and your red pen! I learned an incredible amount from you - and we had lots of fun in the sauna meetings discussing research, methodology, papers and projects. Eling was the first person, in about 2002, who suggested to me that I should do a PhD. Thank you, Eling, for your excellent and exact methodological advice.

I want to express my gratitude to, and admiration of, the two people who reviewed this thesis, Professor Arja Häkkinen, PhD and Professor Jaro Karppinen, M.D., PhD, for their very clear comments and constructive criticism. Through their advice this thesis improved hugely.

Many thanks to my English proof readers, Ariane Knüsel and Daryl Snell, as well as Karen Linwood. I am also grateful to the staff of the Neuro-Orthopaedic Institute (NOI) in Adelaide, who helped me along the way with various tasks. The chief of NOI, David Butler has been one of my greatest

teachers and, in my opinion, one the greatest “brains” in the physiotherapy world.

I am honoured to have come to know so many distinguished contemporary physiotherapy researchers, all of whom I learned a lot along the way: Dr. Lorimer Moseley, PhD from Sydney University. Professor Peter O’Sullivan, PhD from Perth University, Australia, Professor Shirley Sahrmann, PhD from Washington University in St. Louis, USA.

Many thanks to my current employer, the Zurich University of Applied Sciences (ZHAW), and to Professor Astrid Schämann, Head of the Institute for Physiotherapy for their open mindedness towards research and their understanding for my curiosity. I want to thank all my colleagues in the ZHAW. Most of this study was carried out in my private practice in Reinach, Aargau, Switzerland and, partly, in other practices in the east of Switzerland. It is incredible how tolerant my colleagues in the practice were when I was “just” doing research and hardly participating in the “real work”. My biggest thanks go to Sandro Haller, the co-owner of the practice and a very loyal colleague for over 14 years. Thanks also to Rosario, Marianne, Regina, Zdenka, Manuela and others for trusting me and letting me do my research. You must have been thinking, “He’s a weirdo”! I thank all my OMT students, especially those between 2004 and 2010, many of whom participated in patient examinations and treatments in this study. Special thank to Yolanda and Gerold Mohr, Isabel Gloor, Angelika Mannig and Olav Lindner for their especially large contributions to the studies. Thanks to my friends and colleagues in the OMT education program in Switzerland for all the fruitful clinical discussions, Fritz Zahnd, Hugo Stam and Elly Hengeveld.

Thanks to my constant loyal friends: Harri, Reijo, Sakke, Simppa, Fille, Nelli and Kalle for simply being around. Thanks to my mother, Ritva, from whom I inherited a relaxed manner. And I am grateful to you, Tarja, for taking care of me as life got tough.

I dedicate this work to my kids Liina and Tristan. I am sorry that I spent the time away from you.

The study was financed mainly from my own pocket. However, I did get a small grant from the government-based EVO of Kuopio University Hospital in Finland, for which I am thankful.

In Zürich, 21.9.2010

List of original Publications

The present thesis is based on the following original publications*, which are referred to by their Roman numerals:

I. Luomajoki H, Kool J, de Bruin ED, Airaksinen O: Reliability of movement control tests in the lumbar spine. BMC Musculoskeletal Disorders 2007; 8:90

II. Luomajoki H, Kool J, de Bruin ED, Airaksinen O: The test retest reproducibility of active movement control tests of lumbar spine. 2010. Submitted for publication

III. Luomajoki H, Kool J, de Bruin ED, Airaksinen O: Movement control tests of the low back; evaluation of the difference between patients with Low Back Pain and healthy controls. BMC Musculoskeletal Disorders 2008; 9:170

IV. Luomajoki H, and Moseley GL: Tactile acuity and lumbopelvic motor control in patients with back pain and healthy controls. 2009. . Br J Sports Med, published online 23 Jun

V. Luomajoki H, Kool J, De Bruin ED, Airaksinen O. Improvement in low back movement control, decreased pain and disability, resulting from specific exercise intervention. 2010. Sports Med Arthrosc Rehabil Ther Technol 23;2(1):11

*Some unpublished data are also presented.

Contents

Abbreviations:

95%CI	95% Confidence Interval
Cm	Centimetre
CMD	Cumulative Musculoskeletal Disorder
CTD	Cumulative Trauma Disorder
CNS	Central Nervous System
CNSLBP	Chronic Non-Specific Low Back Pain
ES	Effect size
fMRI	functional Magnetic Resonance Imaging
GDP	Gross domestic product
ICC	Intraclass correlation coefficient
K	Kappa statistic
kg	Kilogram
LBP	Low Back Pain
MC	Movement control
MCI	Movement Control Impairment
MCT	Motor control test battery
mm	Millimeter
NSLBP	Non-Specific Low Back Pain
p	Probability, significance level
PKB	Prone knee bend test
PSFS	Patient-specific functional scale
RCT	Randomised controlled trial
RMQ	Roland Morris disability questionnaire
S1	1st sacral vertebra
s1	Primary sensory cortex
SD	Standard deviation
SDD	Smallest detectable difference
SEM	Standard error of measurement
SEP	Standard error of prediction
SKE	Sitting knee extension test
T12	12th thoracic vertebra
TA	m. Transversus abdominis
TPD	Two Point Discrimination
TNF	Tumour Necrosis Factor

1. Introduction

Low Back Pain (LBP) is defined as a pain arising from lower part of the spine, between Thoracal vertebra 12 (Th12) and first sacral vertebra (S1), which can be local but can also radiate to lower extremity (Waddell, 2004). It has been suggested that for a minimal definition it should be stated to be "bad enough to limit your usual activities or change your daily routine for more than one day" (Dionne et al., 2008). In the majority of patients LBP is non-specific (NSLBP). Schmidhauser suggests that less than 10% of LBP individuals can be assigned to a specific LBP category, such as nerve root compression, vertebral fracture, tumour, infection, inflammatory diseases, spondylolisthesis, spinal stenosis or definite instability (Schmidhauser, 2008). The treatment of NSLBP represents one of the biggest challenges of modern health care since it is one of the most costly diagnoses in the western world. A wide variety of patients suffer from NSLBP. To improve assessment and treatment regimes the identification of different sub-groups of patients with NSLBP and the development of tailored, more efficient treatments has high priority. One possible sub-group consists of patients with movement control impairment (MCI). Recent research (Dankaerts et al., 2006c, Van Dillen et al., 1998, Van Dillen et al., 2003b, Van Dillen et al., 2005, Murphy et al., 2006) has developed clinical tests to identify a sub-group of patients with NSLBP who have MCI, which is defined as impaired active movement control of the lumbar spine during functional activities (O'Sullivan, 2005). Synonyms used for movement impairment syndromes are movement control dysfunctions (Comerford and Mottram, 2001a, Comerford and Mottram, 2001b, Sahrmann, 2002) and motor control (MC) impairment (O'Sullivan, 2005).

2. Literature Review

2.1 Low Back Pain

2.1.1 Epidemiology of LBP

LBP is one of the major current concerns in health care (Airaksinen et al., 2006, van Tulder et al., 2006, Dionne et al., 2008, Chou et al., 2009, Dagenais et al., 2010). There is a high prevalence of LBP in all western industrial countries (Waddell, 2004) as well as in Africa (Louw 2007). LBP places a massive burden on the person concerned and their families, and a huge financial cost to countries' social security institutions.

In Switzerland musculoskeletal problems represent the third largest illness group, with 9.4 million consultations annually (Buri et al., 2007). Of these consultations, approximately 30% are related to LBP. The highest prevalence of the LBP medical condition is found in the working age population, with 8% of women and 13% of men affected over a 4-week period (Calmonte, 2005). Total costs of LBP in Switzerland are estimated annually at 7.4 billion Euros. Direct medical costs amount to 3.4 billion Euros (corresponding to 6.7% of total Swiss health care expenditures) while indirect costs are estimated at 4.0 billion Euros (Interpharma, 2007). The numbers are similar in other countries. In Australia annual back pain financial costs were reported in 1990 to be $10 billion, some 0.22% of its gross domestic product (GDP) and 1.65% of its entire health care expenditure. This compares with £60 billion in the UK, 0.19% of its GDP and 2.78% of its health expenditure, and $243 billion in the USA, 0.42% of its GDP and 3.22 % of its health expenditure (Kent 2005).

Chronic LBP is one of the most frequent reasons for persistent disability and inability to work. Between 1990 and 2005 the expenses of the Swiss Disability Insurance for LBP increased by 215%. In 2005, approximately 20% of disability pensions were due to LBP (Interpharma, 2007).

The point prevalence of LBP differs from 6.8% in USA to 33% in Belgium (Kent and Keating, 2005). In Australia, the point prevalence has been stated as 25.5%, six-month prevalence as 64.6% and life time prevalence 79.2% (Walker et al., 2004). One-year prevalence varies between countries from 27% to 65%. In the UK, point prevalence was 14% and one-year prevalence 36%.

Back pain can already occur in childhood. The prevalence of LBP in children is low but rises rapidly in adolescence. Taimela (1997) found within 1171 children and adolescents

that the prevalence of back pain increased with age, with a low prevalence (1%) among 7-10 year old schoolchildren, rising to 18% among 14-16 year old adolescents. No gender difference was found. Recurrent or chronic pain was reported by 26% of the boys and 33% of the girls who reported LBP, and the proportion of recurrent and chronic pain increased with age. Another Finnish study revealed that in young men attending military service (mean age 19 years; N= 7333), 12.7% had already visited a doctor due to back pain (Mattila et al., 2008). The incidence of moderate and major NSLBP was 13.2% and 7.8%, respectively, in a sample of 24- to 39-old Finns (Shiri et al., 2010c).

It is, therefore, clear that a significant health problem exists and that efficient classification and management of LBP should have high priority

2.1.2 Classification of LBP

The most frequently used subclassification of LBP is simple. Most guidelines propose three different categories of LBP diagnoses (Airaksinen et al., 2006, Waddell, 2004, Dagenais et al., 2010, Dionne et al., 2008). Serious LBP is the smallest group with around 1% of all LBP cases. It includes patients with pathologies such as fractures, anomalies or tumours. The second group, making up about 5% of all LBP instances, comprises those with radicular pain due to nerve root irritation. These patients may have in the lower extremity motor, sensory and reflex changes corresponding to the affected nerve root. The remainder (90-95%) are classified as NSLBP.

NSLBP can be divided in three stages: acute is 0-6 weeks, subacute 6-12 weeks, and chronic 12 weeks and longer (Dionne et al., 2008, Waddell, 2004, Waddell, 2005). However, recurrences are frequent, happening within a year in up to 70% of cases (Hestbaek et al., 2003, Pengel et al., 2003). This categorisation has been criticised, as there is actually a large group of patients who can be classified as subchronic recurrent, having recurrent episodes of LBP with only a few months in between without any back pain (Dionne et al., 2008). One fifth of patients still report substantial limitations in their activities after one year of an acute episode (Chou et al., 2009).

There exists no clear consensus on the factors that cause recurrent LBP. A systematic review (Chou and Shekelle 2010) found out that most helpful components for predicting persistent disabling low back pain were maladaptive pain coping behaviours, nonorganic signs, functional impairment, general health status, and presence of psychiatric comorbidities. It is immensely important to carry out studies on predictors of bad outcome and recurrences of LBP, which can be defined and examined. MCI could be one functional impairment causing recurring LBP episodes. However, an easy and reliable tool for clinical diagnosis is still lacking.

2.1.3 Current evidence and guidelines on LBP

In the last 15 years, numerous reviews, like through Cochrane collaboration, and guidelines (Dagenais et al., 2010, Airaksinen et al., 2006, van Tulder et al., 2006, Chou et al., 2009) have been published on LBP.

Currently, there is strong evidence that individual patient education is more effective on short- and long-term return to work than no intervention during the subacute phase of LBP (Engers et al., 2008). In this Cochrane review, 24 studies were included and it was concluded that 2.5 hours of individual education, consisting of encouragement to stay active, coping with and understanding back pain and not worrying about it, is effective treatment of LBP. Exercise therapy is effective for the chronic and subacute phase of LBP (Hayden et al., 2005 a,b), but is not better during the acute phase than other therapies or no therapy at all. However, there is still no consensus as to what kind of exercises should be used (Airaksinen et al., 2006, Oesch et al., 2010). There is moderate evidence that it prevents recurrences of LBP, but again, it is unclear, which exercises are the best (Choi et al., 2010). Behavioural treatment alone is not better than other therapies but it might be useful in conjunction with exercises (Ostelo et al., 2005, van Tulder et al., 2000b). Exercises and physiotherapy are effective on LBP during pregnancy (Pennick and Young, 2007). Pregnancy related back pain is thought to be a typical example of back pain caused through instability (Stuge et al 2004) However, the effect size (ES) of exercises, like most other non-surgical treatments also, is small (Keller et al 2007).

Non-steroidal anti-inflammatory drugs (NSAID) are more effective on acute and chronic pain than other treatments in reducing pain (Roelofs et al., 2008). However, the ES are small and the majority of the 65 trials included are of low methodological quality.

Massage might be beneficial in subacute and chronic LBP, especially when combined with exercises (Furlan et al., 2008). Evidence on manipulative therapy is conflicting some guidelines recommending manipulation (Chou et al., 2009), while other reviews neither refute nor recommend them (Walker et al., 2010).

Surgical treatment on chronic non-specific low back pain (CNSLBP) is not recommended (Ibrahim et al., 2008). Surgical discectomy for carefully selected patients with sciatica provides faster relief from acute attack than conservative treatment, although any positive or negative effects on the lifetime natural history of the underlying disc disease are still unclear (Gibson and Waddell, 2007). After surgical operations, exercise programs starting 4 to 6 weeks post operation seem to lead to a faster decrease in pain and disability, and high intensity programs seem better than low intensity programs (Ostelo et al., 2002).

Evidence is insufficient or limited for the usage of acupuncture (Furlan et al., 2005), radiofrequency (Niemisto et al., 2003), injection therapy (Staal et al., 2008) or for low-level laser therapy (Yousefi-Nooraie et al., 2008). Bed rest is less effective than advice to stay active, and can even be harmful (Hagen et al., 2004).

In an occupational setting, there is limited to moderate evidence that manual material handling advice and assistive devices do not prevent LBP or reduce sick leave due to LBP or back pain related disability (Martimo et al., 2007). Although the effectiveness of physical conditioning programs in reducing LBP in workers is unclear, there may be a positive effect on sick leaves in both acute (Schaafsma et al., 2010, Kool et al., 2004a) and chronic LBP (Kool et al., 2004a, Oesch et al., 2010). Multidisciplinary biopsychosocial rehabilitation has moderate evidence of positive effects in subacute LBP among working age adults (Karjalainen et al., 2003). Finally, there is moderate evidence suggesting that back schools, in an occupational setting, reduce pain and disability in the short- and long-term more than other treatments (Heymans et al., 2004).

As a conclusion, the effectiveness of treatments and management of LBP is sparse and the ES are mostly small. This might be due to the fact that LBP is a multidimensional problem. As a broad statement, there is hardly any evidence for acute LBP that a certain treatment is superior to others. In chronic LBP, psychosocial issues are given the most important value. However, behavioural programs alone are ineffective unless they are used in conjunction with exercises. Yet, there exists no consensus on what kind of exercises should be given. The problem, again, might also be that without sub-grouping the large majority of NSLBP, exercises are not specific enough. Thus, tests for sub-grouping enabling a more specific treatment are needed.

2.2 Sub-grouping LBP

2.2.1 Sub-grouping systems used

The heterogeneity of the largest group of patients with NSLBP is a huge problem. Sub-grouping these patients was already declared 15 years ago to be one of the main focuses for future research (Borkan et al., 2002, Bouter et al., 1998). In fact, it has been called “the holy grail” of LBP research (Waddell, 2005).

In a systematic review, Billis et al (2007) found 39 studies from 9 different countries on the topic of sub-grouping NSLBP. The majority of the studies used a biomedical model and tried to classify patients within a series of patho-anatomical disorders. Only a few studies utilised a psychosocial or biopsychosocial grouping. Different countries use different approaches, resulting in a lack of consensus on subclassification. Biomedical diagnostic sub-groups would consist of anomalies, disc herniations, nerve root affections, degenerative changes or spinal stenosis (Spitzer, 1987). Treatment-based

categories would be directed more by the reaction of patients to certain tests rather than to anatomical findings. Procedures are described methods, such as the McKenzie (Donelson et al., 1997) or Maitland systems (Maitland, 2006). Clinical prediction rule studies (Hicks et al., 2005, Flynn et al., 2002, Childs et al., 2003, Fritz et al., 2006) are beginning to show which patients may benefit from stabilisation or manipulative approaches. In a systematic review, Vibe Fersum (2009) found, from 767 published RCT's on LBP, 68 studies with manual therapy and exercise, but found only six studies that had sub-grouped participants before randomisation. A meta-analysis of those studies revealed that classification-based interventions statistically were significantly better in pain and disability in both short- and long-term follow up. A movement system balance approach has been proposed by the American physiotherapist Sahrmann (2002) and the reliability (Van Dillen et al., 1998, Harris-Hayes and Van Dillen, 2009) and validity (Van Dillen et al., 2003b, Scholtes et al., 2009) of the tests used within that concept have been shown to be acceptable. One of the most promising sub-grouping systems, a biopsychosocial-based system which is used in this thesis, has been suggested by Australian physiotherapist Peter O'Sullivan (2005).

Sub-grouping has also been criticised. Wand & O'Connell (2008) state that the disappointing results of clinical studies can be commonly explained by the failure of researchers to adequately attend to sub-grouping of the CNSLBP population. They suggest that the main reason for CNSLBP lies within the representational areas of the brain and that current approaches may be ineffective. They further suggest that clinicians and researchers may need to radically rethink the nature of the problem and the ways it should be managed. However, they speak only about the most severely affected of the chronic population; those who are incapable of work for months or years on account of their back pain. Again, this is only a small portion, maybe 10% of the whole LBP population. It is important to specify which kind of chronic LBP population is meant, because whether the patient is still working or not makes a huge difference to the severity of the problem.

2.2.2 Movement control impairment as a sub-group of LBP

O'Sullivan (2000 and 2005) proposes a new classification system of LBP (see Figure 1). According to his work, only about 5-10% of patients have specific LBP, including both serious illnesses and nerve root compromise in accordance to the current guidelines. The remaining patients suffer from NSLBP, which O'Sullivan classifies as either centrally or peripherally evoked LBP. The centrally evoked pain is associated with psychosocial factors, such as fear avoidance, catastrophising or depressive mood (approximately 30% of patients with LBP suffer from this kind of NSLBP). The peripherally evoked LBP is mechanically caused and is further divided into movement impairment and MCI (each approximately 30%). Patients with movement impairment have a painful restriction of movement. Patients with MCI have complaints in certain positions like sitting, standing or in twisted positions. MCI is direction-specific,

provoked either by flexion, extension, rotation or multidirectional movements. Up to one third of patients with LBP are estimated to have MCI (O'Sullivan, 2005). Inter-tester reliability of this classification has been shown to be very high; with expert raters k=0.85 and with less experienced raters k=0.6 (Dankaerts, 2006).

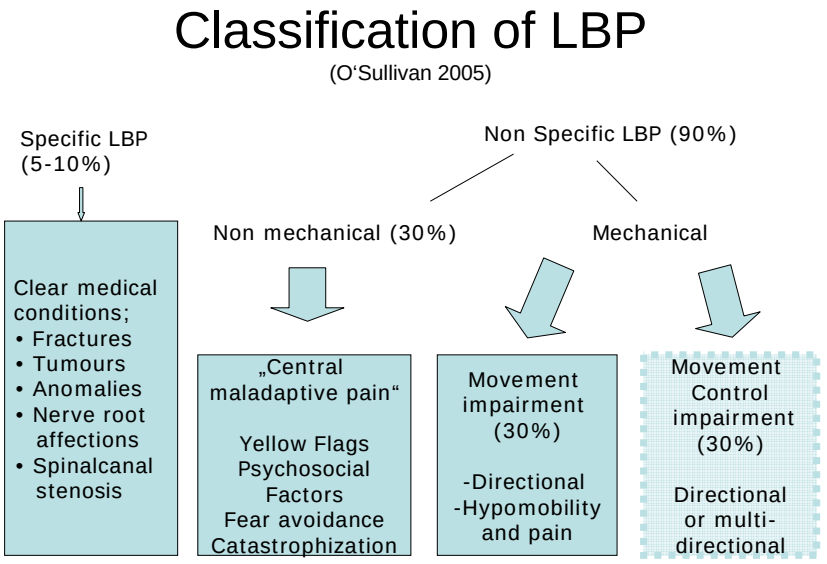


Figure 1. *Classification of Low Back Pain according to O'Sullivan (2005).*

2.2.3 Synonyms for movement control impairment

MCI is defined as impaired active movement control of the lumbar spine during functional activities (O'Sullivan, 2005). A typical clinical finding is that the patients suffer from back pain by sustained postures but movement direction is not restricted (O'Sullivan, 2005). Synonyms used for movement impairment syndromes are movement control dysfunctions (Comerford and Mottram, 2001a, Comerford and Mottram, 2001b, Sahrmann, 2002), movement system impairment (Harris-Hayes and Van Dillen, 2009), and sometimes also motor control impairment (O'Sullivan, 2005). Clinical instability (Panjabi, 2003), and even segmental instability (O'Sullivan, 2000) can be used as a synonym for MCI, as it is defined as a back problem behaving like instability but without findings in radiography. McKenzie describes these patients as having a postural syndrome (Clare et al., 2004, Kilpikoski et al., 2002).

Instability is a widely used term to explain LBP. Structural, radiological examinable instability is rare (Waddell, 2004, McGill, 2007). Thus, when instability is referred to, clinical instability is actually meant usually. That is, clinical behaviour that is like

instability but the condition is not verifiable through radiography. The term clinical instability was introduced by Panjabi (1992) and for 20 years extensive research efforts have been undertaken to prove this condition (Hides et al., 1995, Hodges et al., 1996, Jull and Richardson, 2000, Richardson, 1999, Richardson and Jull, 1995, Richardson, 2004, McGill, 2007).

As in MCI typically suffering and pain is demonstrated in sustained postures, MCI has to do with occupational postural back pain. Possible mechanisms for these problems, also called cumulative trauma disorder (CTD) (Solomonow, 2006, King et al., 2009), are explained in the next chapters. Pathokinesiology, meaning abnormal movement pathways, is a key competence of physiotherapists. The relative flexibility theory (Sahrmann, 2002) describes that movement occurs through the pathway of least resistance, e.g. if hip muscles are relatively strong compared with trunk muscles then movement is more likely to occur in the back, possibly leading to a back pain problem related to the direction of that particular movement. The movement is not as important as the way different body parts are moving in relation to each other (Klein-Vogelbach, 2001). O'Sullivan (2005) describes back pain patients with reduced movement control and excessive movement as pain provocateurs, as opposed to patients with fear of movement, which are described as pain avoiders. Patients with movement control deficits are a substantial sub-group that may benefit from specific exercises (Van Dillen et al., 2003b, Van Dillen et al., 2003a, Van Dillen et al., 2005, O'Sullivan, 2005, McGill, 2007).

2.3 Anatomical and biomechanical considerations

The individual vertebrae can move in two ways, namely to translate or to rotate. According to Panjabi, the segmental movements are controlled through three systems: passive, active and neural systems (Panjabi, 1992, Panjabi, 2003, Panjabi et al., 1994) (Figure 2). The disc, the joint surfaces and the ligaments passively restrict the movement. The myofascial system causes the active movements and the neural system controls and co-ordinates the movements.

2.3.1 Basic anatomy of lumbar spine

The lumbar spine consists of five vertebrae. The main landmarks of it are the vertebral body, inferior and superior articular processes, spinous process and transverse process. The vertebral body carries the greatest load and is connected firmly with the intervertebral disc. The articular processes build the zygapophyseal (also called facet) joints. The joint surfaces are sagittally oriented enabling a good flexion (around 50 degrees) and extension (around 15 degrees) mobility of the lumbar spine, but rotation (approx. 5 degrees) and lateral flexion (up to 10 degrees) are possible to a lesser extent in this part of the spine (Bogduk, 2008). The main feature of the transverse and spinous

processes are their insertions for ligaments and muscles.

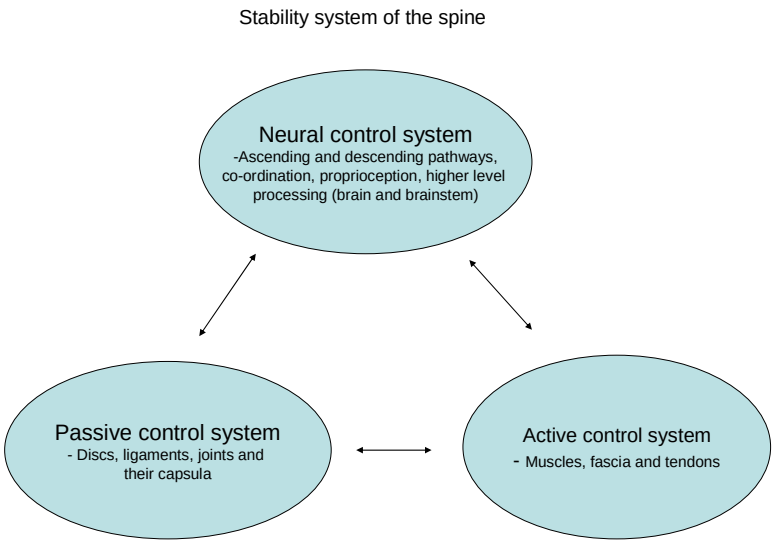


***Figure 2**. The stability system of the spine (adapted after Panjabi 1992).*

Important structures regarding possible pain sources are the intervertebral discs, apophyseal joints, the ligaments and the muscles. As a principle of nociception, all structures that are innervated can also cause pain (Bogduk, 2008)(Figure3).

The intervertebral disc consists of three basic components. A central nucleus pulposus, which is surrounded by anulus fibrosus. A third component is the vertebral end-plate, which covers the bottom and top end of the disc with layers of cartilage. The nucleus pulposus consists mainly of water, proteoglycans and hyaluronic acid. As a fluid, its volume can not be compressed. The peripheral part of the disc, anulus fibrosus, consists of collagen fibres arranged in between 10 and 20 sheets, called laminae. These are arranged in concentric rings that surround the nucleus pulposus. The orientation of fibres alternates in successive laminae, enabling resistance in rotational forces. The lamellae consist of collagen, glycosaminoglycans and proteoclycans. As the disc is responsible for weight bearing, it is a very strong structure. However, a trauma or degeneration can cause the individual lamellae to break, and when all the lamellae are torn, the nucleus pulposus can seep out causing a protrusion or herniation. The outer part of the anulus fibrosus is innervated by sinuvertebral nerve (Figure 3), which is a mixed nerve containing sympathetic efferent and somatic sensory afferent fibres (Bogduk, 2008).

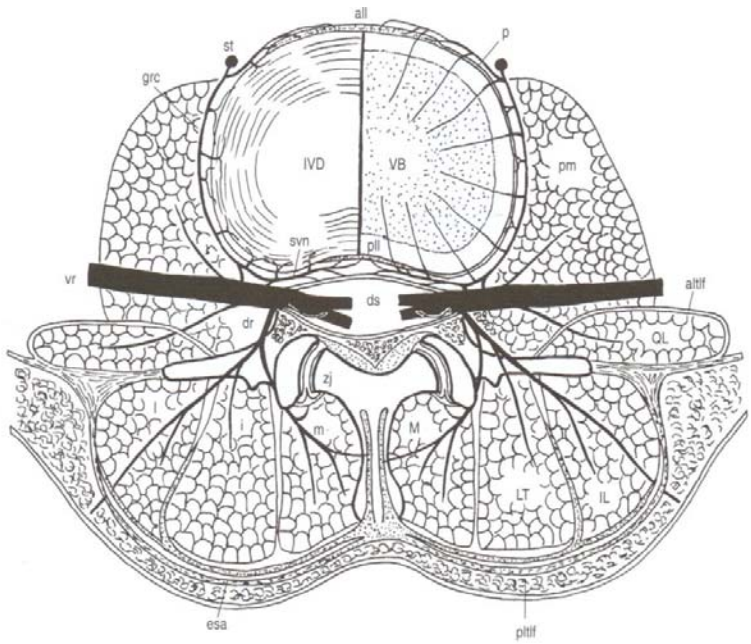


Figure 3. *Innervation of the spine structures. Nerves: Ventral ramus (vr), dorsal ramus (dr) and its medial brach (m), sinuvertebral nerve (svn). Muscles: M. psoas major (pm), m. quadratus lumborum (QL), m. multifidus (M), m. longissimus thoracis (LT), m. Iliocostalis lumborum (IL), erector spinae aponeurosis (esa). Thoracolumbar fascia; anterior layer (altlf), posterior layer (pltlf). Intervertebral disc (IVD). Vertebral body (VB). Zygapophyseal joint (zj). This figure was published in "Clinical anatomy of the lumbar spine and sacrum", N. Bogduk, 2008, Copyright Elsevier. With kind permission.*

Can LBP be explained through anatomical structures?

2.3.2 Intervertebral disc and its role on LBP

Disc lesions are hypothesised to be a major structure causing back pain and suffering (Videman and Nurminen, 2004). As the outer layer of anulus fibrosus is innervated through the sinuvertebral nerve, intradiscal changes and ruptures of the anulus could cause pain (Bogduk, 2008). The diagnoses of changes in the intervertebral disc are mostly done by magnetic resonance imaging (MRI). However, the MRI findings have to be regarded with caution, as it has also been shown that up to 50% of asymptomatic subjects have pathological findings, such as signal hypointensity, anular tears, disc protrusions and endplate changes (Kjaer et al., 2005). A Finnish cross-sectional study found in a sample of 558 twenty-one year-old subjects about a 50% prevalence of disc degeneration (Takatalo et al., 2009). Earlier studies in the 1990's (Boos et al., 1995, Jensen et al., 1994) have already raised the question of how useful MRI is in the diagnostics of LBP as even asymptomatic subjects have frequently abnormal imaging

findings. A large study on 1043 Chinese subjects also found a high prevalence of disc degeneration, 40% by 30 years and up to 90% by 55 years (Cheung et al., 2009). However, they found a significant association between symptoms and MRI findings. Degenerative changes (OR 2.2) and herniation (OR 2.0) were twice as common in patients with severe back pain compared to subjects without back pain, but anular tears (OR 1.0) or Schmorl's nodes (OR 1.3) were not associated with LBP. Videman and Nurminen (2004) found in a study of 157 male cadavers that findings of radial tears correlated more clearly with frequent lifelong LBP than usually thought.

Anular ruptures may be a reason for sudden, nonspecific LBP. However, when nucleus pulposus leaks onto the spinal cord or nerve root(s), inflammation of the neural structures can be induced (Videman and Nurminen, 2004). This would explain sciatica, pain radiating from back to lower extremity, causing possibly sensory, motor and reflex abnormalities. Inflammatory nature of sciatica, with involvement of tumour necrosis factor (TNF)-α, has been shown nicely in animal studies (Igarashi et al. 2000).

Intervertebral disc is certainly a major tissue involved in nociceptive pain when it is torn. It can be hypothesised that uncontrolled movements and postures can lead to microtrauma in the discs (Sahrmann, 2002). However, it has been shown that minor trauma do not lead to chronic LBP (Carragee et al., 2006a) or to abnormal findings on MRI (Carragee et al., 2006b). However, intervertebral discs are only one peripheral tissue source of nociceptive pain. On the other hand, LBP cannot be explained with peripheral anatomy solely. Instead, central neural mechanisms controlling movement, as well as psycho-social and cognitive issues, are of great importance.

2.3.3 Ligaments and LBP

Ligaments connect the individual vertebrae and stabilise the vertebral column. The anterior and posterior longitudinal ligaments run from the individual vertebral bodies stabilising the spine in extension and flexion directions. Ligamentum flavum connects the laminae between vertebrae and closes the spinal cord space posteriorly. In contrast to other ligaments, which consist mostly of collagenous fibres and are hardly stretchable, ligamentum flavum is built up to 80% of elastin, making it elastic and, therefore, a unique ligament in the body. With degeneration ligamentum flavum dries and can build “bumps” and cause narrowing of the spinal cord, which is a typical feature of spinal canal stenosis (Bogduk, 2008). Interspinous and supraspinous ligaments connect the spinous processes and restrict hyperflexion movement. Ligamenti intertransversii connect the transverse processes and their function is more of a fascial kind, separating different layers of muscles from each other. All the ligaments are possible pain sources, especially due to continuous strain or longstanding repetitive movements (Solomonow et al., 2003a, Solomonow et al., 2001). The posterior longitudinal ligament is innervated by the sinuvertebral nerve, which arises directly from the nerve root before it goes through the intervertebral foramen (Bogduk,

2008)(see figure 3). The other posterior ligaments are innervated through the small medial branches of dorsal ramus arising from nerve root after it has left the intervertebral foramen. Finally, iliolumbar ligaments connect the transverse processes of L4 and L5 to Iliac crest and so anchor the lower lumbar spine to pelvis. However, both the diagnosis of pain arising from ligaments as well as its treatment are unclear. However, ligaments might be the cause of pain behind troubles from prolonged postures, hypermobility and occupational pain syndromes.

2.3.4 Prolonged postures and LBP

In a longstanding position the passive and active systems are under stress. If this stress is continued and the collagenous fibres elongated, it is called strain (Solomonow, 2009). If the strain is sustained, for instance through wrong posture, shear forces are applied to discs (Panjabi et al., 1994). A twisted position causes torsion. These forces strain the anulus fibrous layers of the disc (Bogduk, 2008). If a constant force or a repetitive longstanding strain is applied to collagenous tissue, the tissue starts slowly to adapt and lengthen through this force. This is called creep. The exact biomedical basis of the creep is not known, but it appears that the collagen fibres rearrange themselves (Bogduk, 2008). After a creep, normally after hours of load, the recovery of the fibres is not immediate and the structures remain elongated even in relaxation. This is called hysteresis (Bogduk, 2008, King et al., 2009, LaBry et al., 2004, Sbriccoli et al., 2004, Solomonow, 2009). In animal models, it has been shown that 7 hours of creep causes a hysteresis for about the same period of time in relaxation (King et al., 2009, D'Ambrosia et al., 2010). No matter whether the loading of the ligaments is cyclic or static, it causes an increased expression of pro-inflammatory cytokines (D'Ambrosia et al., 2010, King et al., 2009, Solomonow, 2006). This would have consequences for example in ergonomics of workers and this is thought to be the explanation of cumulative trauma disorder (CTD) (Solomonow, 2006, King et al., 2009). With time, the structures get used to this new position and the collagenous fibres remain longer (hysteresis).

Strain could cause nociceptive pain. In a normal situation, the person would change position automatically when it hurts. However, it can be hypothesised that some people habituate to this, and do not notice it anymore and actually become accommodated to these postures. Thus, the person does not notice anymore that the position is not normal. However, this change might have more to do with habituation of the nervous system.

2.3.5 Joints, LBP and manual therapy

The apophyseal joints are important for the direction of movement (Bogduk, 2008). In the course of intervertebral disc degeneration, the intervertebral disc narrows, and the joint surfaces become compressed causing changes like osteophytes, thickening of the

joint capsule, movement restriction and possibly pain (Bogduk, 2008, Panjabi, 2003, Panjabi et al., 1994). Therefore, the degenerative changes in the apophyseal joints are regarded secondary to disc degeneration (Videman and Nurminen, 2004). The joint capsule is innervated through medial branch of dorsal ramus and is rich on proprioceptive nerve endings (Bogduk, 2008). The joint capsule plays an important role in control of position and posture (Solomonow, 2006).

For a long time it was thought that the facet joints are an important cause of spinal pain. Many treatment options such as manual therapy have been developed in the past hundred years and many physicians, chiropractors and physiotherapists have been treating the facet joints. However, anomalies of the facet joints are common (Bogduk, 2008). Although guidelines do recommend manipulative therapy for spinal problems (Dagenais et al., 2010), the reliability of the clinical examination has been shown to be poor (van Trijffel et al., 2005, Schneider et al., 2008, Fritz et al., 2005, Hicks et al., 2003).

Manual therapy does not only treat facet joints or other passive structures but it may work through segmental neurological modulation and neural hysteresis in addition to biomechanical effects (Schmid et al. 2008). However, an increasing number of studies hypothesise that an activation of the central nervous system (CNS) results in a non-segmental hypoalgesic effect with concurrent activation of other neural pathways as a potential mechanism of action. Schmid et al (2008) found in a systematic review of 15 high-quality studies consistency for concurrent hypoalgesia, sympathetic nervous system excitation and changes in motor function. Having pooled the data, it was found out that joint mobilisation improved outcomes by approximately 20% relative to controls. The authors concluded that descending pathways might play a key role in manual therapy -induced hypoalgesia and their review supports the existence of an alternative neurophysiological model, in which passive joint mobilisation stimulates areas within the CNS. Therefore, it can be hypothetised that the effects of manual therapy are more on the neurophysiological level (Schmid et al., 2008).

2.3.6 Role of the muscles in LBP

The muscles of the low back can be divided into layers, namely deeper, short and monosegmental muscles and superficial, longer and strong multisegmental muscles (Bergmark, 1989). Abdominal muscles also play an important role in the motion and stabilisation of the back (Hodges et al., 1996, Hodges and Richardson, 1997) (Figure 4). The deep layer of back muscles consists of mm. interspinales, between spinous processes; mm. intertransversarii mediales, between transverse processes and as an important segmental muscle; m. multifidus. Multifidus muscle has been suggested to be one of the most important stabilising muscles (Richardson, 1999, Richardson, 2004, Hides et al., 1996, Hides et al., 1994). Biomechanically, this makes sense, as the muscle is running from the base of spinous process directly over the apophyseal joint and inserts to mamillary process (Figure 5a). It has few layers, the deepest passing over one

segment only, and other fibres over 2-4 segments. This muscle is also rich in proprioceptors, meaning that it plays an important role in the posture and stabilisation of the spine (Le et al., 2009, Solomonow et al., 2008, Solomonow et al., 2003b, McGill, 2007). M. transversus abdominis (TA) is the deepest muscle of the abdominals and attaches over the fascia to each transverse process and spinous process (Bogduk, 2008, Hodges, 1999) (Figure 5b). Between these membrane layers lies the multifidus muscle. Through the connection by thoracolumbal fascia, tension of the TA also indirectly tensions the multifidus (Figures 4 and 5b). The more superficial muscles together form m. erector spinae, consisting of m. longissimus thoracis and m. iliocostalis lumborum. M. longissimus originates at the iliac crest and sacrum, and inserts to transverse processes, typically running over few segments. M. iliocostalis lies laterally to m. longissimus but runs similarly, with some fibres running over the whole lumbar spine up to the ribs. These muscles cannot participate much in the local segmental stabilisation because they are too long and too lateral (Bergmark, 1989). Thoracolumbal fascia connects the aponeurosis of these long muscles, the m. latissimus dorsi and also the TA. Through a co-contraction, thoracolumbal fascia is able to stabilise the lumbar spine (Richardson, 1999, Richardson and Jull, 1995, Richardson et al., 1995). However, if it is too tight, it causes movement restriction. In the front, m. oblique abdominis internus and m. oblique abdominis externus also connect on the thoracolumbal fascia and can contribute to stability of the spine. In contrast to this, the m. rectus abdominis cannot, as it has insertions only on the thorax and on symphysis. The deep abdominal and lateral muscles, namely m. iliopsoas and m. quadratus lumborum (Figure 3), are strong and large muscles contributing to force applied by stronger movements.

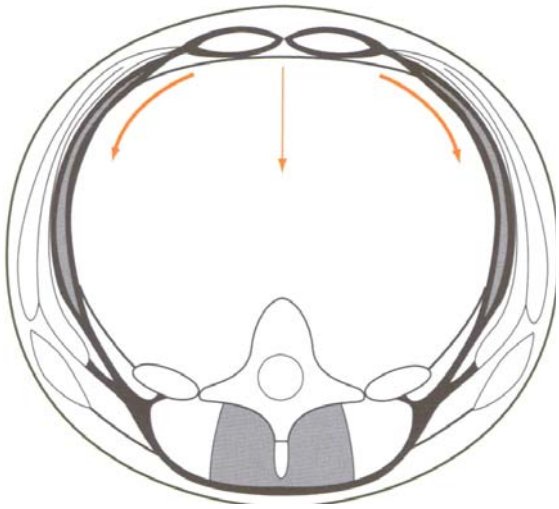


Figure 4. *Cross-sectional illustration of the thoracolumbar fascia. Posterior attachment on each Transverse and spinous processes of the vertebrae encloses the deeper muscles of the back. Anterior attachment encloses the abdominal muscles. This construction enables a segmental stabilization through the tension of the m. transverses abdominis muscle. This figure was published in "Therapeutic exercise for lumbopelvic stabilization", 2. ed, 2004: Richardson et al, Copyright Elsevier. With kind permission.*

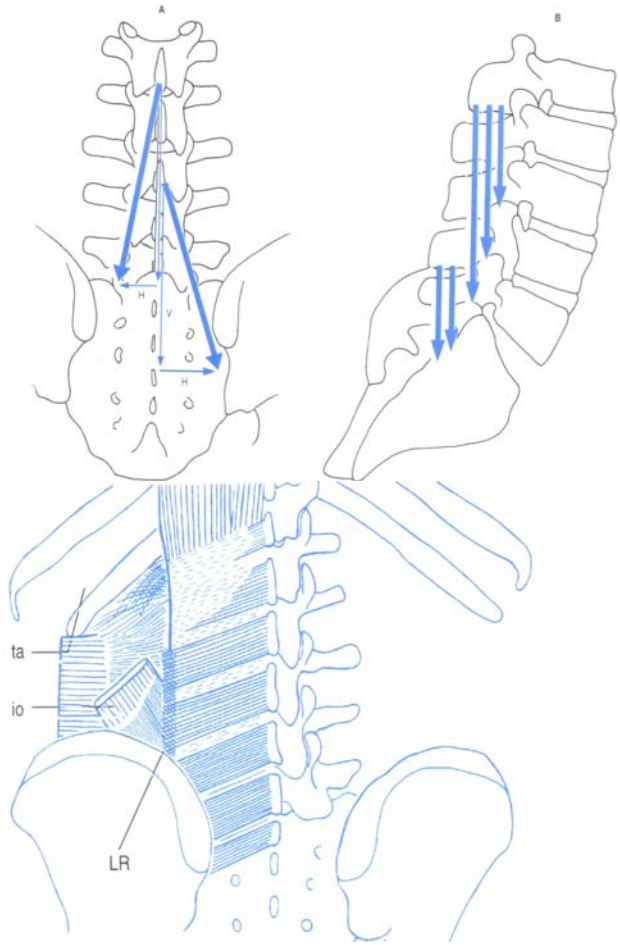


Figure 5a. *The segmental muscle, m. multifidus, connects adjacent vertebrae running directly over the zygapophyseal joints.*

Figure 5b. *The deep lamina of the posterior layer of thoracolumbar fascia. Collagenous fibres anchor individual vertebrae to iliac crest (LR lateral raphe) and also laterally through m. transverses abdominis (ta) and through m. Internal obliques abdominis (io). These figure were published in "Clinical anatomy of the lumbar spine and sacrum", H. Bogduk, 2008, Copyright Elsevier. With kind permission.*

Prolonged flexion of the lumbar spine results in tension-relaxation and laxity of its viscoelastic structures (Williams et al., 2000). Multifidus muscle reacts at first with spasms measured on EMG activity, but after 2-3 hours of loading this activity decreases and exposes the spine to a risk of instability (Youssef et al., 2008, Le et al., 2009). A static loading in flexion for 20 minutes, caused a sharp decrease in activity of feline multifidus muscle, which was not recovered in the next 7 hours (Jackson et al., 2001). As with the ligaments, longstanding static and cyclic (6 times 10 minutes for 40 N by

cats) loading in flexion causes the same loss of viscoelastic tissue compliance (Arabadzhiev et al., 2008, Olson et al., 2009, Ben-Masaud et al., 2009). The risk of cumulative musculoskeletal disorder (CMD) increases along the number of repetitions, (Sbriccoli et al., 2007, Sbriccoli et al., 2004). CMD would be a typical diagnosis of over-use syndromes, where the pain originates from the myofascial system.

The segmental muscles, m. multifidus and TA, seem to posses a feedforward activation, meaning that the muscles are recruited about 50 ms earlier than the movement starts (Hodges, 1997, Richardson, 1999). Pain impairs the muscular feedforward mechanism (Leinonen et al., 2001, Leinonen et al., 2002a, Leinonen et al., 2003). This has been shown for both disc herniation (Leinonen et al., 2001) and NSLBP (Hodges and Richardson, 1997). Multifidus muscle is suggested to atrophy after first acute back pain episode (Hides et al., 1994) and it may not recover without specific training (Hides et al., 1996). Improvement can be reached through exercises, but while this does not correlate with improved disability or reduced sick leave on short term, recurrences seem to decrease in the long run (Hides et al., 2001, Hides et al., 1996). However, isometric trunk muscle strength does not seem to differ between patients with LBP and healthy young adults (Paalanne et al., 2008). It is concluded that strength per se does not correlate with LBP (McGill, 2007)

As a conclusion on anatomical structures, the passive system can explain only a part of LBP (Carragee et al., 2006a, Carragee et al., 2006b, Carragee et al., 2006c, Kjaer et al., 2005). In recent years, considerable effort has been undertaken to explore the role of muscles in LBP (Hodges, 1999, Hodges et al., 2003, Moseley et al., 2003, Moseley et al., 2002, Richardson, 2004, McGill, 2007). However, efficacy studies on specific local muscle training show no effect (Cairns et al., 2006) or only minor effects (Costa et al., 2009), and a recent review states that specific stabilisation is not better than other exercises (Ferreira et al., 2006). Because biomechanical models are of limited value for the treatment of LBP, recent research has increasingly focused on the role of the CNS and neural control (Moseley, 2008b, Moseley, 2008a, Moseley, 2005b, Moseley, 2003, Hodges and Moseley, 2003).

2.4 Neuroanatomy, neural pathways and movement control

2.4.1 Postural stability and movement control

Trunk muscle recruitment patterns are different in patients with LBP compared to healthy controls (Cholewicki et al., 2003, van Dieen et al., 2003a, van Dieen et al., 2003b). Sensation of body position is poorer in patients with LBP compared to healthy controls and worsens through fatigue (Taimela et al., 1999). Moreover, postural balance reactions have also been demonstrated to be poorer in patients with LBP (Radebold et al., 2001). Furthermore, movement perception and postural stability are impaired in

spinal stenosis (Leinonen et al., 2002b) and sciatica (Leinonen et al., 2003).

Pain also changes motor reaction speed in other parts of the body outside the pain location. Patients with chronic LBP have decreased reaction time on sudden body perturbation in their biceps brachii muscle (Leinonen et al., 2007). One-footed and externally disturbed two-footed postural control is significantly worse in patients with LBP compared to healthy controls (Luoto et al., 1998b). Women with LBP seem to have poorer postural control than healthy controls (Luoto et al., 1996b). In contrast, the psychomotor speed of the dominant hand is slower among men (Luoto et al., 1996b). Luoto et al (1999) hypothesised that the decreased speed of information processing could be explained through impaired short term memory. Consequently, it seems that the problems LBP is causing are more on a coordinative level and, thus, the neural control mechanisms are of greater importance in understanding the MCI and postural deficits.

2.4.2 Neural control of movement and posture

The neural control is an interaction between perception as well as cognitive and action processes (Shumway-Cook, 2007). The ascending pathways bring the information of the muscles, fascias, joints and ligaments through different specific receptors, through spinal pathways to thalamus. The receptors are golgi tendon organs in tendons, pacini corpuscles in joints and merkel's disks, meissners corpuscles and ruffini endings in the skin (Lundy Ekman, 2007). They play a major role in segmental reflexes caused by sudden injury (Solomonow, 2006). However, for the postural and movement control in normal life, the ascending pathways are of more importance. These are the dorsal column-medial lemniscal system and the anterolateral system. The former conveys information from touch and pressure, while the latter contains information about pain, temperature, crude touch and pressure. The pathways overlap (Apkarian et al., 2009). Through thalamus the ascending pathways convey to somatosensory cortex (s1). From here, associated areas are recruited, meaning that the information is processed. The brain has to decide whether an action is needed or not (Apkarian et al., 2009, Baliki et al., 2008). The visual system plays an important role in the processing of the ascending information (Shumway-Cook, 2007). Through the information from the eyes, the somatosensory cortex reflects whether the information is correct, if the posture is correct or if the movements are running incorrectly. Also, the vestibular system informs the somatosensory cortex about the position of the head (Shumway-Cook, 2007). Thus, this cerebral processing checks all incoming information and then decides whether an action is needed. If this is the case, the neighbouring area of the sensory cortex, the premotor and motor cortices are activated. Outputs from primary motor cortex contribute to corticospinal tract (earlier also called pyramidal tract) (Lundy Ekman, 2007). Through polysynaptic connections through the descending pathway, the information finally lands on the ventral horn of the spinal cord of that segment where an action is needed. Peripheral afferent motoneurons fire the involved muscles, which

again are registered through the receptors and a feedback loop is activated to correct or stop or to continue the performed action.

Nervous system activity is based on learning which consists of conditioning and habituation. Impulses and pathways that are used a lot strengthen the used pathway and it gets easier. If a task is performed repeatedly, it will become automatised through the nervous system (Lundy Ekman, 2007). Actions that are not used or needed get weaker. "Use it or lose it" is a principle of neurophysiology (Moseley, 2008b). So, why would some people choose to use postures and movement habits that might cause troubles? Because they are habituated to it; they do not notice or are not aware of what is happening in their body (Moseley, 2008a).

2.4.3 Higher level motor control

The cerebellum receives information through the descending pathways from the motor cortex, and basal ganglia are also involved in coordination between different muscles and muscle groups (Shumway-Cook, 2007). The function of the cerebellum is the fine tuning and timing of coordination between different muscles working together in a certain function. Basal ganglia consist of a set of nuclei at the base of the cerebral cortex, including putamen, caudate nucleus, globus pallidus, subthalamic nucleus and substantia nigra. Basal ganglia also play a role in the coordination of the movements and muscles, this time in accuracy, reciprocal movements and coordination of the different extremities. Problems like tremors in Parkinson's disease are caused by a loss of neurons and networks in basal ganglia area (Lundy Ekman, 2007).

2.4.4 Sensory cortex and body awareness

The sensory cortex registers from which part of the body the afferent stimuli are coming (Lundy Ekman, 2007). In the homunculus (Figure 6), different body parts are differently represented. Areas where a very exact differentiation is needed, like the mouth or fingertips, invade more space in the sensory cortex than areas with less differentiation, like the lumbar spine. The homunculus is plastic (Flor, 2003b). Thus, special needs, habituation or training can change the representational area of a body part. There is evidence that these cortical maps are altered in the presence of pain in general (Moseley, 2008b, Moseley, 2008a, Moseley, 2005a), CRPS (Pleger et al., 2005), phantom limb pain (Flor and Birbaumer, 2000) and LBP (Flor et al., 1997). Currently there is a debate whether the whole chronic LBP could be a cortical disturbance (Wand et al., 2010). The cortical representation can be measured with functional magnetic resonance imaging (fMRI) (Flor, 2003b, Flor, 2003a, Flor, 2000). Two-point discrimination (TPD) is an easy and cheap clinical way to measure the tactile acuity of the skin which correlates to a cortical map of the body part (Flor and Diers, 2009).

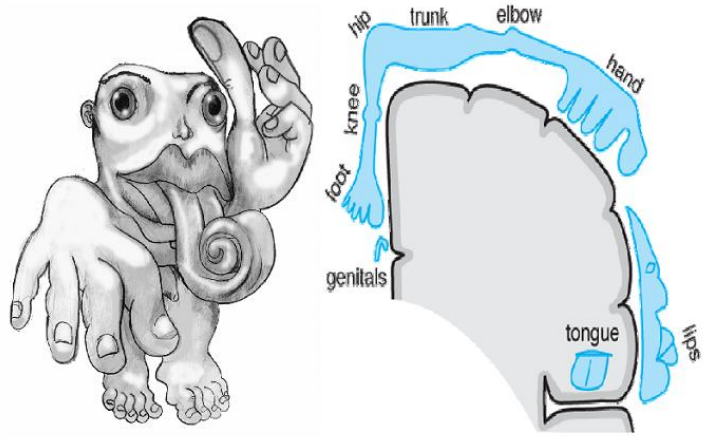


***Figure 6**. "Homunculus man"; the representational areas of different body parts in the somatosensory cortex of the brain. Butler & Moseley2003. With permission.*

The human cortex continuously reorganises itself as it lives in chronic pain (Apkarian et al., 2009, Flor et al., 1997). Multiple cortical and subcortical structures are involved (Apkarian, 2008). At least primary and secondary somatosensory cortex, inferior parietal lobule and cerebellum have been shown to be changed in a similar pattern by LBP and by fibromyalgia (Giesecke et al., 2004). It has been suggested that long-term pain alters the functional connectivity of cortical regions known to be active at rest, the so called "default mode network" (Baliki et al., 2008), which have been shown to display a reduced deactivation in chronic back pain. These findings demonstrate that chronic pain has widespread impact on overall brain function and this may underlie the cognitive and behavioural impairments accompanying chronic pain. But brain areas can be changed not only functionally but even structurally; Apkarian et al (2004) showed that prefrontal and thalamic grey matter is decreased by chronic back pain. Perception of pain is always accompanied with negative emotions (Apkarian, 2008), and chronic pain patients suffer from more than just pain, they can also suffer from depression and anxiety, sleep disturbances and decision-making abnormalities (Baliki et al., 2008). Therefore, it is important to consider the psychosocial and cognitive factors when dealing with patients with LBP.

2.5 Other factors influencing LBP and movement control

2.5.1 Psycho-social and cognitive factors

Current guidelines and evidence conclude that the factors predicting a poor outcome and chronicity by LBP are more related to cognitive and psychosocial issues than to anatomical structures (Airaksinen et al., 2006, van Tulder et al., 2006, Linton, 2000, Linton 2005). When screening these so-called "yellow flags" (Kendall, 1997), which are the psychosocial risk factors for poor outcome in LBP, it is important that the normal healing time of tissues has passed (around 4-6 weeks after the onset of acute back pain). In the following, yellow flags and some examples from each category are listed (Kendall 1997, Linton 2005):

Attitudes and beliefs about back pain

1 Belief that pain is harmful or disabling, resulting in fear-avoidance behaviour, e.g. the development of guarding and fear of movement

Behaviours

1 Use of extended rest, disproportionate 'downtime'
2 Reduced activity level with significant withdrawal from activities of daily living
3 Irregular participation or poor compliance with physical exercise, tendency for activities to be in a 'boom-bust' cycle (doing too much – long rests afterwards because of pain)
4 Avoidance of normal activity and progressive substitution of lifestyle away from productive activity

Compensation issues

1 Lack of financial incentive to return to work
2 Delay in accessing income support and treatment cost, disputes over eligibility
3 History of claim(s) due to other injuries or pain problems

Diagnosis and treatment

1 Health professional sanctioning disability, not providing interventions that will improve function
2 Experience of conflicting diagnoses or explanations for back pain resulting in confusion
3 Diagnostic language leading to catastrophising and fear (e.g. fear of ending up in a wheelchair)

Family

1 Overprotective partner/spouse, emphasising fear of harm or encouraging catastrophising (usually well-intentioned)

2 Solicitous behaviours from spouse (e.g. taking over tasks)
3 Socially punitive responses from spouse

Emotions
1 Fear of increased pain with activity or work
2 Depression, loss of sense of enjoyment
3 Mood is more irritable than usual
4 Anxiety about heightened awareness of body sensations (includes sympathetic nervous system arousal)

Work
1 History of manual work
- fishing, forestry and farming workers
- construction workers, including carpenters and builders
- nurses, labourers and truck drivers
2 Work history, including patterns of frequent job changes, experiencing stress at work, job dissatisfaction, poor relationships with peers or supervisors, lack of vocational direction
3 Belief that work is harmful; that it will do damage or be dangerous
4 Unsupportive or unhappy current work environment
5 Low educational background, low socioeconomic status

Acknowledging psychosocial factors is important as mood and emotions are strong pain modulators. It has been shown on neuroimaging that pain is rated as more unpleasant after sad mood induction, when compared with a neutral mood induction (Berna et al 2010). If patients' problem is more depended on psychosocial factors than physical factors such as movement control impairment, therapists can be treating the problem on a wrong site and, besides not getting patient improved, also getting frustrated themselves.

2.5.2 General health behaviours and LBP

Subjects with other health-related problems have more LBP compared to healthy controls. In a meta-analysis by Shiri et al. (2010b) it was found that smokers have up to 1.5-fold increased likelihood of LBP than non-smokers. Also, obesity is a risk factor of chronic LBP with similar odds than smoking (Shiri et al., 2010a). Sleep disturbance predicts LBP among adolescents (Auvinen et al., 2010) and adults (Kaila-Kangas et al., 2006). Patients with LBP also show a significantly lower level of aerobic fitness, also when corrected with other typical risk factors like disability, pain, fear or level of leisure and work activities (Smeets et al., 2006). However, back extensor muscle oxygenation and fatigability are not different to healthy controls (Kankaanpää et al., 2005) and thus the changes in endurance do not seem to be morphological.

One important role of a physiotherapist, declared by roles and competencies portfolio of world confederation of physiotherapy (WCPT), is the role of "health advocate" (www.wcpt.com). This means that physiotherapists (and medical doctors as well) are dedicated to take care of the whole patient. Many chronic pain patients have a package of problems like over-weight, smoking, sleeping disturbances etc. Therefore, although a patient would have a movement control impairment, also these other factors need to be addressed.

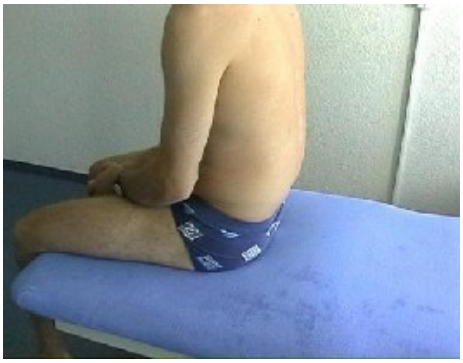
2.5.3 Summary of factors involved in LBP

LBP is a multidimensional and complex symptom, but not actually a disease. It is the "20th century medical disaster and the legacy reverberates into the new millenium" (Waddell, 2004). Structures and peripheral mechanisms are an explanation for the cause of LBP, but the role of CNS, neural controlling systems and cognitive-behavioural aspects all need to be addressed. Since structural- findings, like on MRI (Boos et al., 1995), do not explain the cause of LBP and many clinical examination findings are not reliable (Binkley et al., 1995, Essendrop et al., 2002, Fritz et al., 2005, van Trijffel et al., 2005), there is need for simple, reliable tests for clinicians to sub-group patients with LBP. There is growing evidence that postural, movement control and proprioceptive deficits may be caused through changed central sensitivity (Luoto et al., 1998, Luoto et al., 1996, Taimela and Luoto, 1999). Therefore, treatment regimes have to be directed in this way as well, which means that patients need to be treated in a cognitive-functional way (O'Sullivan, 2005, O'Sullivan, 2000). This again means that the patient has to understand what the actual problem is and has to actively change his habits. Treatment could use operand conditioning techniques (Linton 2005); i.e. the patient has to notice that when he changes his movement habits, it changes his back pain.

2.6 Assessment and treatment of movement control of the low back

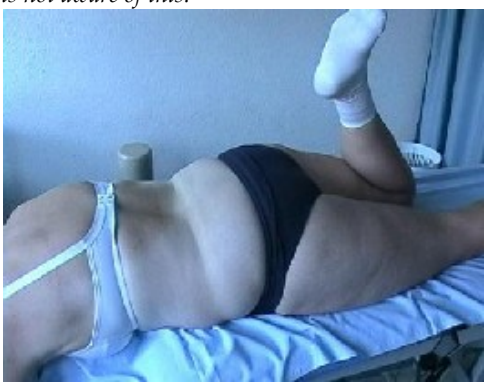
2.6.1 Examples of MCIs caused by/occurring with LBP

The pain behaviour in the sub-groups of patients suffering from MCI is consistent: the pain comes in certain sustained positions (O'Sullivan, 2005, O'Sullivan, 2000). This is contrary to those who have movement impairment; they are restricted and have pain in the movement. Figure 7 shows some typical postural habits which patients with MCI are accustomed to.



a.***Flexion pattern.*** *The patient sits predominantly with the low back in flexion.*

b. Flexion pattern. *When bending forward, the patient moves predominantly in the low back instead of hips. Typically the patient is not aware of this.*



c. Extension pattern. *In the prone knee bend test the patient moves automatically in the low back while bending the knee.*

Figure 7. *Typical movement control impairments (MCIs).*

2.6.2 Reliability and validity of the assessment of movement control in the low back

2.6.2.1 Reliability

Physiotherapists consider the assessment of movement control of the lower back important in patients with LBP (Cook et al., 2006, Sahrmann, 2002, Van Dillen et al., 1998, Comerford and Mottram, 2001a, Comerford and Mottram, 2001b, O'Sullivan, 2000, O'Sullivan et al., 2003). In a Delphi study of American Physical Therapists who were Orthopaedic Clinical Specialists or Fellows of the American Academy of Orthopaedic Manual Physical Therapists (N=168) (Cook et al., 2006), 88% of the specialised therapists considered abnormal movement patterns as the main finding in clinical instability of the low back. Maybe we should then also name the problem by its name, MCI, as this is something we can examine. How good are we at it so far?

White & Thomas (2002) investigated the reliability of 16 different tests developed by Sahrmann. They found a satisfactory inter-tester reliability. However, the difference between movement patterns in patients with LBP and individuals without LBP received little attention. Murphy et al. (2006) (N = 42) investigated the prone hip extension test. Inter-tester reliability was substantial (kappa = 0.7).

Van Dillen et al. (1998) used a whole package of physical examination items (28 different movement and postural tests) in order to categorise the patients in an impairment dysfunction sub-group. They found a very high agreement for the assessment of symptoms among the examiners (k > 0.89 and percentage agreement > 98%). Furthermore, they examined the reliability of observation of spinal alignment and movement. In general, the interpretation of the spinal alignment was slightly lower (k = 0.27–0.58) than for the observation of active movements (k = 0.26–1.00). Moreover, Harris-Hayes & Van Dillen (2009) examined the inter-tester reliability on 30 chronic patients with LBP. They categorised patients in five different groups; extension, flexion, rotation, extension-rotation and flexion-rotation. They found an overall agreement of 83% and k= 0.75 (95% CI 0.51-0.99).

The classification system of O'Sullivan has been tested in its inter-tester reliability (Dankaerts et al., 2006c). In the first phase, two experts who were blinded to each other examined 35 patients with interview and different physical tests. They almost perfectly agreed in their classification of patients (k = 0.96 and percentage agreement 97%). However, when less experienced clinicians classified the same patients after viewing them on videos, they had a lower inter-tester reliability, but still k>0.6, which means substantial reliability. Thus, experience increases the reliability. However, in this study, the tests used were not declared. Vibe Fersum (2009) in turn used the whole

examination set in order to categorise the patients in an impairment dysfunction sub-group. They found a very high agreement for the assessment of symptoms among the examiners (k = 0.89 and % agreement > 98%). The reliability was calculated stepwise; after the first interview, after physical tests and after the examination of the psychosocial factors. At the final decision making, the overall agreement was 87% (range 85%-92%) and kappa = 0.65 (range 0.57-0.74). This research setting makes sense, because in a clinical situation, clinicians also do not rely on individual tests only but on the whole "picture", including pain behaviour, anamnesis, provocative movements and postures as well as physical exams and tests.

2.6.2.2 Validity

There exists no gold standard for movement control so far and thus sensitivity and specificity cannot be tested. Such a gold standard could be kinematic analysis or functional, open magnetic resonance imaging (MRI), where real time segmental movements could be observed and compared with observation.

Studies so far have examined the discrimination validity of movement control between patients with low back pain and healthy controls. In the prone knee bend test with and without rotation of the hip, there is a significant difference in timing and amplitude of hip and lumbopelvic movements between patients with LBP and healthy controls. Scholtes et al. (2009) compared these items in 41 people without and 50 people with LBP who played rotation-related sports. During knee flexion and hip lateral rotation, people with LBP demonstrated a greater maximal lumbopelvic rotation angle and earlier lumbopelvic rotation compared to people without LBP ($p<0.05$). The interpretation of this is that patients with LBP have a poorer control of their lumbopelvic movements and, because of this, might be moving in their everyday activities and sports more on their lumbar spine which may cause pain.

The importance of subclassification has been highlighted in several studies. When sitting postures are compared between pain free subjects and patients with LBP, there are no significant differences (Astfalck et al., 2010, Dankaerts et al., 2006a, Dankaerts et al., 2006b). However, when the patients with LBP are at first subclassified in flexion or active extension control impairment, then differences are clear and significant. Patients with flexion control impairment sit more flexed in their usual sitting position than healthy controls. In contrast, patients with active extension impairment sit in more extension than the healthy control (Dankaerts et al., 2006b). The same finding correlates with muscle activity findings; the flexion group has less, the extension group more activity in their back muscles (Dankaerts et al., 2006a). This is the so called "wash out effect", which means that the findings of one sub-group wash out the effect of the other sub-group. This clearly highlights the importance of subclassification (Dankaerts et al., 2009). Another study used the same setting by adolescents. The kinematic findings were similar but the muscle activation pattern was not as clear as it was in adults

(Astfalck et al., 2010).

So far, there is no gold standard for movement control tests. In the previously mentioned studies, the judgement of quality of movement relies on inspection. We, therefore, wanted to study the reliability of this ability separated from all other information gained from subjective or objective assessments. Consequently, in this doctoral project a test battery for MCI of the low back was created, based on the descriptions of O'Sullivan (2005 and 2000), Dankaerts (2009), Sahrmann (2002) and (Van Dillen et al.,1998). From the large amount of existing tests, a set of ten tests was selected according to own clinical experience and so that all movement directions would be considered.

2.6.3 Treatment and exercises of movement control in the low back

One of the most promising treatment possibilities of NSLBP is exercise (Airaksinen et al., 2006, Hayden et al., 2005b, van Tulder et al., 2000a). However, which kind of exercises should be used, is unclear. Activity and general exercise therapy improve pain and disability, and reduce the number of sick days in patients with NSLBP (Airaksinen et al., 2006, Hayden et al., 2005b, Kool et al., 2004b). The evidence for the usage of exercise in specific sub-groups is still lacking. However, recent research has developed clinical tests to identify a sub-group among patients with NSLBP who also suffer from MCI (Van Dillen et al., 1998, Van Dillen et al., 2003b). These tests could be used to recognise the sub-group and then exercises could be adapted to the sub-group.

Only studies comparing movement control exercise with treatments not including exercise, such as manipulative therapy (Rasmussen-Barr et al., 2003) or physician consultation and instruction alone, reported significant benefits of motor control exercises. These studies do not answer the question of whether movement control exercise is superior to other types of exercise. Reviews of spinal motor control exercise conclude (Rackwitz et al., 2006, Ferreira et al., 2006) that the outcome of specific stabilisation and motor control exercises are not more effective than general exercise programs. Previous studies paid little attention to the selection of patients to receive specific movement control exercise or general exercise (Luomajoki, 2002). This may explain the inconclusive results.

Positive studies involved defined clinical sub-groups. Benefits of movement control exercises were demonstrated in other sub-groups of patients with LBP. Specific movement control exercise is more effective than general exercise in post partum women with pelvic instability (Stuge et al., 2004) and patients with spondylolisthesis (O'Sullivan et al., 1997). Therefore, research evaluating movement control exercises in the large and important sub-group of patients with NSLBP and impaired movement control would appear to be worthwhile.

Adapted treatment is more effective. There is some evidence that the outcome is better if patients receive treatment adapted to their clinical presentation (Brennan et al., 2006). Treatment options in this study were manipulation, motor control exercises or specific exercises during a four week intervention. Therapies matched to the patients' clinical problem were more effective in the short- and long-term. However, it remains unclear whether movement control exercises are more effective than general exercises in patients with MCI. Such studies can be performed when tests to reveal this sub-group are developed. Figure 8 displays some typical movement control exercises.

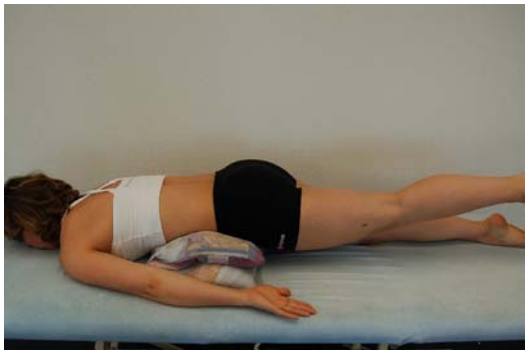


Figure 8*. Examples of exercises for the movement control coordination.*
a. For flexion control, the patient has to keep her low back in neutral while bending forwards with the hips. A tape can be used a feedback.
b. For extension control, the patient has to keep her lumbar spine in neutral while extending the hip. Pillows under waist can be used in the beginning.
More examples of possible movement control exercises are presented in Appendix 2.

3. Aims of the study

3.1 What are the unknowns?

According to a large Cochrane Collaboration review (64 RCT's) there is no evidence to support the effectiveness of one type of exercise over the other in patients with chronic NSLBP (Hayden et al., 2005a). Movement and motor control training has become an increasingly applied treatment for patients with LBP. However, there is no evidence as to whether all subjects, or rather a defined sub-group, respond to such a program. According to the European guidelines for the management of chronic NSLBP (Airaksinen et al., 2006), high quality RCT's are needed to determine the effectiveness of specific interventions aimed at specific target groups. This includes the evaluation of spinal stabilisation and motor control exercises in patients with LBP and MCI.

This thesis will evaluate the reliability (inter- and intra-tester and test-retest reliability) of tests for the sub-group of patients with LBP and MCI. Following on from this, differences between patients with LBP and healthy controls will be compared, connection between body image and movement control will be examined, and finally, a feasibility study based on a case series design will be conducted on the effect of specific movement control exercise on patients with NSLBP and a movement control deficit.

3.2 Research questions

1 What is the inter-tester and intra-tester reliability of the active movement control tests of the low back?

2 Is movement control a consistent phenomenon (in day to day comparison)?

3 Is there a difference in the movement control between healthy persons compared with LBP patients?

4 Does movement control dysfunction have a correlation with distorted body image?

5 Is improvement in movement control, through exercises, associated with a decrease in LBP and improvement of functional disability due to back pain?

4. Methods

4.1 Overview of the study designs

Study I	Inter- and intra-tester reliability study	40 subjects (23 with NSLBP and 17 healthy controls) were videoed performing 10 movement control tests for the low back. Four physiotherapists, blinded to each other and to the subjects, rated each test either positive or negative. Kappa values for the reliability were calculated.
Study II	Test-retest reproducibility study	41 subjects (28 with NSLBP and 13 without back pain) performed two movement control tests on two different days. The amount of movement taking place in the lumbar spine was measured and the consistency of the movement control between days was estimated.
Study III	Cross-sectional study	210 subjects (108 with NSLBP and 102 without back pain) were tested with a test battery consisting of six tests for the movement control ability of the low back. Means, standard deviations, significance for group differences and ES were calculated.
Study IV	Case control study	90 subjects (44 with NSLBP and 46 without back pain) were measured in their movement control ability and in two-point discrimination ability in the low back. Group means, differences, significance and ES for between group differences were calculated.
Study V	Case series study	38 patients with NSLBP and MCI were treated approximately 9 times. Back-related disability, patient-specific subjective functional suffering and movement control ability were measured before and after treatment series. Size of change in percentage and significance were calculated.

4.2 Methods and subjects

The methods used in this thesis were two reliability studies (one intra-tester and inter-tester setting, and one test-retest setting), a cross sectional and a case controls study that compared patients with LBP and healthy controls in their movement control ability as well as in their two-point discrimination performance. The treatment study was a case series one.

In the first study, the aim was to determine the intra- and inter-tester reliability of physiotherapists to categorise MCI via visual inspection. We videoed 27 patients with NSLBP and 13 patients with other diagnoses but without back pain performing a standardised test battery, consisting of 10 active movement tests for motor control. The ten tests were chosen from earlier descriptions (Sahrmann, 2002; Van Dillen, 1998, White and Thomas 2002). It was important that all movement directions would be considered and they should be as easy as possible to observe. The chosen 10 movement tests were also based on own clinical experience in the field. Description of the subjects is displayed in Table 1.

Table 1. *Subject characteristics on the videos (study no. I)*

	Patients without back pain	**Patients with back pain**	**Total**
N =	13	27	40
Female / Male	8 / 5	18 / 9	26 / 14
Mean age (SD)	55.1 (5.1)	50.8 (6.2)	52.1 (5.5)
RMQ (SD)		8.5 (5.5)	

RMQ : Roland Morris Disability questionnaire, max. 24

Methods: Four physiotherapists independently rated test performances as correct or incorrect per observation, blinded to all other patient information and to each other.

Appendix 1 shows the 10 tests used in the study I.

In the second study the consistency over time of performing the sitting knee extension (SKE) test and prone knee bend (PKB) test (Sahrmann, 2002) was assessed. In both tests the subject is instructed to prevent the movement of the back whilst bending or extending the knee (Figure 8). The assumption is that if the movement control ability is intact the person can hold the position of the back while moving the leg. Movements of the lumbar spine were measured with a computer aided tool, the SpinalMouse®. This device measures the segmental movements of the spine when the device is moved over the spinous processes on two small wheels. To assess spinal mobility between T12-S1

with the SpinalMouse, markings on the skin were drawn firmly with a waterproof pen so that the same spots could be used for the second measurement. Flexion / extension changes between T12 and S1 in degrees were recorded. These two tests were chosen as the measurement is possible with SpinalMouse. To measure the reproducibility of the other tests, kinematic analysis in a movement laboratory would have been needed and at that time this was not available.

The data was transferred wirelessly from the handheld mouse to a computer. Position One (without movement, spine in neutral position) and position Two (after the movement had happened) were recorded. The change in position One and Two was compared to the similarly measured change in day two. The difference of this change between the two days was compared and the consistency of the test result in time was estimated.

Table 2. *Study sample in test retest study (no. II)*

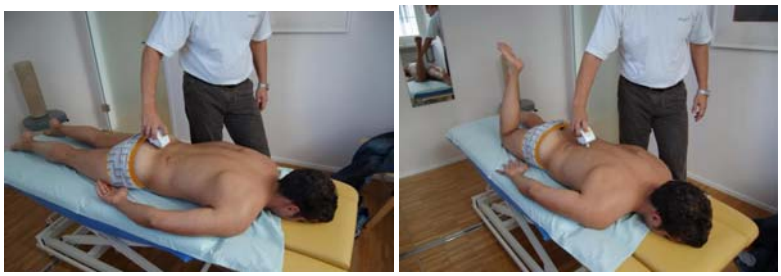
Group means	**All (N=41)**	**LBP (N=23)**	**No LBP (N=18)**	***p***
Age (SD)	47 (14)	47 (13.1)	47 (16.1)	0.88*
Height (SD)	169 (7.2)	167 (7.6)	170 (6.6)	0.26'
Weight (SD)	75 (12.7)	76 (12.5)	74 (13.2)	0.68'
Male	20 (49%)	12 (52%)	8 (44%)	0.63``
Radiating pain N		13 (57%)		
RMQ (SD)		9.2 (4.8)		

RMQ : Roland Morris Disability Questionnaire, max. 24

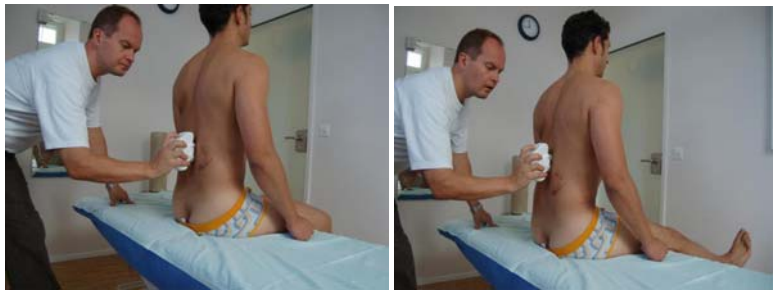
* Mann-Whitney U test for non-parametric distribution

' T-test for parametric distribution

`` Chi square test for nominal data



Prone Knee Bend test (PKB). Whilst bending the knee, the lumbar spine should stay neutral. The test is positive if the lumbar spine moves during the leg movement to extension. In this study, the change between the positions was measured and compared on two days.



Sitting Knee Extension test (SKE). Whilst extending the knee, the lumbar spine should stay neutral. The test is positive if the lumbar spine moves during the leg movement to flexion. In this study, the change between the positions was measured and compared on two days.
Figure 8. *Description of prone knee bend test (PKB) and sitting knee extension test (SKE) which were used in the test-retest study*

In the third study, we used the same test battery as in the first study. From the 10 tests used in that study, a test battery of the 6 most reliable tests was selected. A cross-sectional study was carried out in 5 outpatient physiotherapy practices in the German-speaking region of Switzerland. Using a set of 6 tests, 12 physiotherapists tested the ability of 210 subjects (108 patients with NSLBP and 102 control subjects without back pain) to control their movements in the lumbar spine. Table 3 presents the characteristics of the participants. We observed the number of positive tests out of 6 (mean, standard deviation and 95% confidence interval of the mean).

Table 3. *Background characteristics of the subjects in study III.*

Group means	**Patients with LBP N=108**	**Healthy controls N=102**	**Sig.**
Age (SD)	41 (15)	37 (12)	0.08*
Height cm (SD)	169 (9)	171 (9)	0.65'
Weight kg (SD)	67 (11)	67 (12)	0.21'
Male	36 (33%)	44 (43%)	0.81``
Working	71 (65%)	51 (50%)	0.16``
Retired	15 (14%)	15 (15%)	0.86``
Student	12 (12%)	33 (27%)	0.02``
Disability allowance	10 (9%)	0	<0.01``
Sport >2/week	45 (42%)	52 (51%)	0.58``
Other musculoskeletal problems	38 (34%)	37 (36%)	0.87``

* Mann-Whitney U test for non-parametric distribution

' t-test for parametric distribution

`` Chi square test for nominal data

In the fourth study, a convenience sample of 44 patients with NSLBP and 46 healthy controls (Table 4) participated. TPD threshold was measured according to an established protocol (Moberg, 1990) using a plastic calliper rule in the area between the first lumbar vertebra and iliac crest left and right, both horizontally and vertically. TPD threshold was defined as the shortest distance between the calliper points at which the participant could clearly detect two points instead of one, and was calculated as the average of a descending run (5 mm increments from 10 cm) and an ascending run (5 mm increments from 1 cm). Traps were included to make sure the participant was not guessing.

Table 4. *Study population study no. IV*

Group means	**LBP n= 44**	**Healthy controls N=46**
Age (SD)	41 (10)	43 (15)
Male / Female	17 / 27	18 /28
Height (SD)	172 (7)	170 (8)
Weight (SD)	69 (12)	74 (15)
Working	28 (65%)	32 (70%)
Retired	8 (15%)	5 (10%)
Student	5 (12%)	9 (20%)
Disability allowance	3 (8%)	0
Radiating pain	30 (66%)	
RMQ (SD)	9.1 (5.1)	
Catastrophizing, max. 36 (SD)	13.1 (7.5)	

RMQ : Roland Morris Questionnaire, max. 24

In the fifth study, we conducted a case series study of patients with a movement control deficit. 38 patients who suffered from NSLBP and had at least 2/6 positive movement control tests and at least 5 points in the Roland and Morris disability questionnaire (RMQ)(Roland and Morris, 1983) were treated with movement control exercises according to their MCI. Outcome measures were disability (RMQ), movement control (number of tests positive) and complaints (patient-specific functional scale (PSFS) (Stradford et al., 1995).

Selection of the subjects was conducted by physiotherapists in two clinics. The criterion for inclusion of patients was that they suffered from NSLBP with or without radiating pain, but without neurological findings (muscle weakness, loss of sensibility or reflexes). The exclusion criteria were serious pathologies, such as unhealed fractures, tumours, acute trauma or serious illnesses. A measurement criterion required that patients have at least 3 out of 6 MC tests positive. Further assessment measures included the PSFS (Stradford, 1995) and RMQ (Table 5). To avoid ceiling effects, they needed to have at least 3 out of 10 as a mean value on the PSFS and 5 out of 24 points on RMQ. The patients also had to be able to understand instructions in German. Following

application of the above criteria, suitable patients were selected, explained the aims of the study and asked to participate.

The treatment consisted of coordination and re-learning of the correct movement patterns. Figure 9 shows a typical exercise for Flexion control impairment. Appendix 2 shows a documentation of possible exercises used and progression of the treatment for different, direction-specific impairments.

Table 5. *Subjects in the study no. V*

Mean (SD)	**All**	**Men**	**Women**
N	38	17	21
Age	45 (13)	44 (14)	46 (12)
Height	170 (8)	176 (6)	166 (5)
Weight	73 (15)	82 (14)	65 (11)
RMQ	8.9 (4.7)	9.5 (5)	8.5 (4.5)
PSFS	5.7 (1.5)	6.0 (1.2)	5.6 (1.8)
MCT Score	3.2 (1.2)	3.2 (1.1)	3.2 (1.2)

All patients had CNSLBP since at least 3 months.

RMQ= Roland Morris Disability questionnaire; PSFS= Patient Specific Functional scale, MCT=Movement Control Test battery

Figure 9. *Typical exercises used in the case series study:*
For flexion control; on all fours the patient learns to control the neutral spine during a movement of the pelvis backwards.
For extension control; supine lying the patient learns how to tilt pelvis backwards.

4.3 Measurements

Most materials used in the studies were low-tech which allows a direct transfer of the methods to daily practice. Also, as the budget was low and at own cost, laboratory testing was renounced. All the studies were performed in a normally equipped private physiotherapy practice(s) with an individual examination, treatment room and plinth. Patients were asked to be examined without clothes but wearing underwear.

In the first study, a digital video camera was used to film the subjects in their performance of the movement control tests. In most of the tests, apart from a plinth, hardly any other materials were needed, except in the one leg stance test, where the starting position needed to be standardised. A practical way of standardisation is suggested by Klein-Vogelbach (2001). She claims the normal stance is a third of the width of the trochanter. For this, a large calliper rule was used to measure this width. Then, pieces of wood were prepared to fit the according stance width between the feet (see Figure 10). The sidewards sway of the belly button was measured with a lineal that was fixed on a stative (Figure 11).

Figure 10. *Materials used in the studies I and III for the one leg stance test.*

A calliper rule was used to measure the width of trochanter.
One third of trochanter width was used as stance width between feet.

Figure 11. *The sidewards sway of the belly button was measured with a lineal which was fixed on a tripod.*

In the second study, a computer aided tool, SpinalMouse was used (Figure 12). This device measures the segmental movements of the spine when it is moved over the spinous processes on two small wheels. The all over day-to-day test-retest measurement accuracy of the device has been reported to be around 2 degrees standard error of measurement (SEM) for lumbar spine and has intraclass correlation coefficients (ICC) between 0.81-0.86 (Mannion et al., 2004). The correlation between SpinalMouse® and functional X-rays is 0.97 (Pearson's r) (Bistritschan , 2003, Schulz, 1999).

Figure 12. *SpinalMouse*

In the fourth study, a plastic calliper rule (see Figure 13) was used to measure the two-point discrimination. Plastic is an appropriate material because it is neither warm nor cold. The pikes are not too sharp and, therefore, do not hurt.

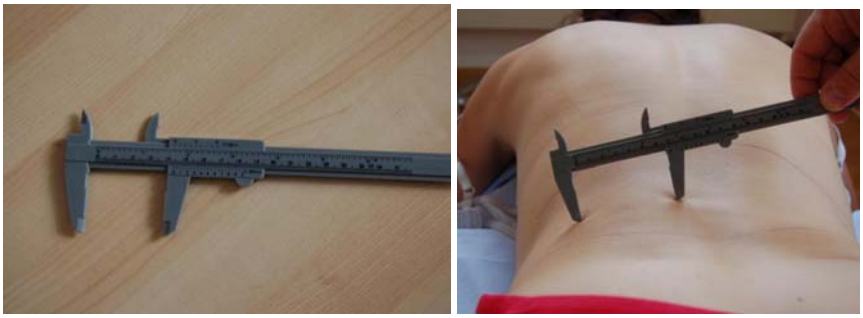


Figure 13*.Two-point discrimination on the low back. A plastic calliper rule was used to define the smallest distance which the person can acknowledge being two different points (study no. IV).*

In the fifth study, only materials that belong to a normal physiotherapy practice (plinth, gymnastic mats, elastic bands, barbells and some weights) were used. The exercises used to train the movement control are presented in the Appendix 2. The therapeutic intervention consisted of specific individual exercises to improve the movement control of the back and the treatment series had on average nine sessions. The measurements were performed before the first treatment and directly after the last treatment. No follow up later on was carried out.

4.4 Statistical analysis used in the performed studies

For the intra- and inter-tester reliability study (Study I), kappa values were calculated according to Cohen (Landis and Koch, 1977a, Landis and Koch, 1977b). The Kappa statistic takes into account the possibility of chance in the agreement. The data was calculated for the two pairs separately in order to identify whether there was a difference between experienced and less experienced therapists. A kappa coefficient of 1.0 indicates full agreement beyond chance. Values greater than 0.80 are generally considered excellent, values between 0.60 – 0.80 substantial, 0.4 – 0.6 moderate, 0.4 – 0.2 fair, and < 0.20 poor (Portney and Watkins, 2000).

The second study, the test-retest reproducibility study (II) used an interclass confidence coefficient (ICC) calculation, which is used for continuous data. Besides the ICC, the Smallest Detectable Difference (SDD) was used, since the ICC does not tell how large the differences between the days were in degrees (Portney and Watkins, 2000). The difference between day one and day two in the change of lumbar flexion or extension from position one (neutral) to position two (with knee extension or flexion) was compared. Test-retest reproducibility on the group level was analysed by calculating the ICC with a two-way random effect model (ICC 2.1) and values greater than 0.70 were considered acceptable (Portney and Watkins, 2000). On an individual level, limits of agreement between days were calculated (mean ± 1.96 x SD) and Bland

Altman Plots were drawn to reflect the stability of the tests over time and the differences between days. When there is no systematic error in the measurement, the mean difference is near zero and the confidence interval of the mean difference includes zero (Bland and Altman, 1986). We also analysed the standard error of prediction (SEP), which was SD $\sqrt{1 - ICC^2}$ as suggested by Weir (2005) and the SDD is SEP x 1.96 (Weir, 2005). The SDD reflects that component of a measure that is statistically attributable to error from the measurement process itself. The percentage of patients falling within this range was defined to display the stability of the test.

In the studies III and IV the means and differences between LBP patients and healthy controls were calculated. Since the data was interval, for the significance in the difference between the groups Student's T tests for parametric distribution, and Mann Whitney U test for non-parametric distribution were used (Portney and Watkins, 2000). Effect size for the between group differences was calculated too. In the fourth study the correlation between MCT and TPD was also calculated to see whether they seem to correlate in the first place.

In the case series study (V), the measurements before and after intervention series were compared. Within group changes for significance were calculated with Student's T test for parametric and Mann Whitney U test for non-parametric distributions. Effect sizes for the change within group before – after the intervention series (average 9 treatments) were calculated for the outcomes on MCT, RMQ and PSFS.

5. Results

5.1 Reliability

5.1.1 Inter- and intra-tester reliability

In the first study (I), the kappa values for inter-tester reliability ranged between 0.24 - 0.71. 6 tests out of 10 showed substantial reliability [k>0.6]. Intra-tester reliability was between 0.51 - 0.96, with all tests but 1 showing substantial reliability [k>0.6] and 5 tests showing almost a perfect reliability (k>0.8). We concluded that physiotherapists were able to reliably rate most of the tests in this series of motor control by viewing films of patients with and without back pain performing the tasks.

Appendix 1 shows the results of the test reliability between the raters and within raters. Kappa values are for the average rating between two tester pairs and between two different persons for intra-tester ratings.

5.1.2 Consistency of the measurement over time

Results of the second study revealed that there were no significant differences in the results between patients and healthy controls on sitting knee extension (SKE) p=0.29; nor on prone knee bend test (PKB) p=0.19. On group level, both tests reached ICC (2,1) with values over 0.7, indicating acceptable reliability: 0.78 (95% confidence interval (CI) 0.63 – 0.88) for SKE and 0.71 (95% CI: 0.51 -0.83) for PKB. For concrete measures, SEP were 2.6° for SKE and 2.3° for PKB. The SDDs were 7.1° for the SKE and 6.5° for the PKB test. Within these boundaries, 93% of the subjects performed the same way on both days in SKE and 90% in PKB. As the mean difference between days was 0.3° (95%CI: -0.1° to 1.6°) for SKE and 0.5° (95%CI: -0.5° to 1.6°) for PKB, there was non-systematic error in the measurements. The clinical signs and symptoms of patients did not change between the measurements.

As a part of this study the visual findings were also compared with measures of SpinalMouse. During the measurement on the first day, the subjects were at first asked to perform the test – PKB and SKE – before the measurement was done with SpinalMouse. The examiner (HL) rated per inspection whether the test was correct or not and noted this. The measures from SpinalMouse were then compared with the inspection's findings. In SKE, 24 subjects (total N=41), performed the test correctly, meaning that in a clinical situation the test would have been rated negative. 17 subjects were rated positive. In PKB, 29 subjects were negative and 12 positive. On the basis of SpinalMouse, it seems that the eye can acknowledge the movement when it is more

than 3-4° but not when the movement is smaller (Table 6). Furthermore, there is a difference of approximately 10° between those who performed the test correctly and those who did not. The positive and negative tests could not discriminate between healthy subjects and those with LBP, meaning that more tests (a whole test battery) is needed to discriminate patients with LBP from healthy controls.

Table 6. *Measures with Spinalmouse compared to dichotomic test per inspection. All subjects N=41.*

	Test negative Mean grade (CI95%)	**Test positive Mean grade (CI95%)**
SKE (Flexion movement in the spine)	3.5° (2.9-4.2)	12.8° (10.1-14.5)
PKB (Extension movement in the spine)	3° (2.2-3.8)	9.9° (8.6-11.1)

SKE= Sitting Knee Extension, PKB= Prone Knee Bend

5.2 Difference in movement control between LBP patients and healthy controls

On average, the number of positive tests (out of a possible total of 6) was 2.21 (95%CI: 1.94-2.48) in patients with LBP and 0.75 (95%CI: 0.55-0.95) in healthy controls. The mode, the typical number of positive tests was 2 by patients with LBP but 0 for the healthy controls. The ES (d) for the difference between the groups was 1.18 (95%CI: 1.02-1.34). The statistical test showed that this was a significant difference ($p<0.001$). Figure 14a presents graphically the difference between the groups.

We performed a sub-group analysis of the number of positive tests depending on pain duration. There was a significant difference between the groups; between acute and chronic ($p<0.01$), as well as between subacute and chronic ($p<0.03$) but not between acute or subacute ($p>0.7$) patients (Figure14b).

In the sample of 102 subjects without LBP, there was a sub-group of 51 subjects who did not have LBP but were in physiotherapeutic treatment because of some other problem like elbow, shoulder or neck pain. A sub-group analysis revealed that these subjects had significantly ($p<0.01$) more positive tests (mean 1.45 tests positive) than healthy controls.

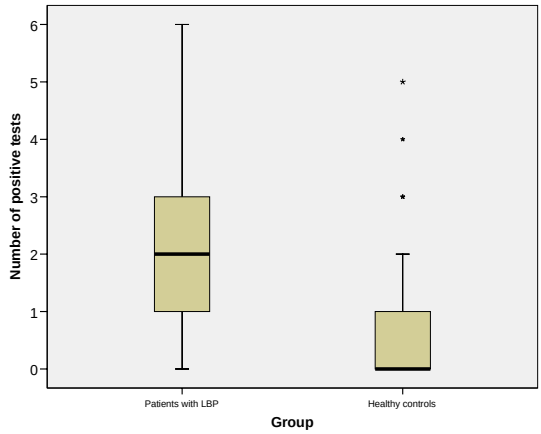
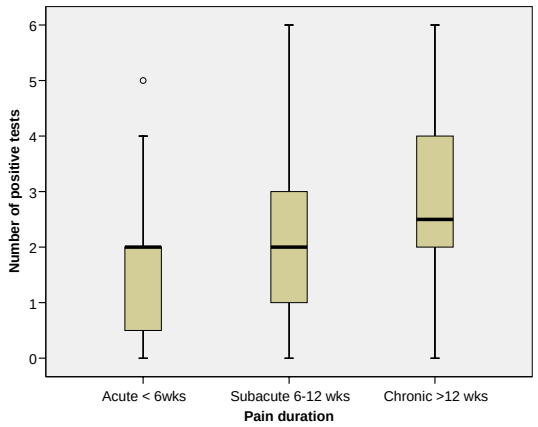


Figure 14. a. *Box Plot showing group differences between patients with LBP and healthy controls (Study III).*



14 b. *Differences within patients with LBP according to the duration of pain.*

Also, a sensitivity-specificity analysis was performed for cut-off points. For the cut-off point, which was 2 positive tests out of 6, the specificity was 0.88.

5.3. Movement control and body schema representation of the brain

TPD threshold was greater in patients (mean (95% CI)) = 60 (57 – 64 mm) than in controls = 44 (40 – 48 mm), (main effect of group $F(1,88) = 40.5$, $p<0.001$) (Figure 15). The difference in TPD threshold between patients and controls was greater in the horizontal direction than it was in the vertical direction (group x test interaction ($F(1,88) = 15.9$, $p <0.001$). The relationship between TPD threshold and lumbopelvic movement control was that TPD threshold was negatively related to lumbopelvic control. That is, the higher the TPD threshold of the back, the more positive lumbopelvic movement control tests were observed (Pearson's $r = 0.49$, $p <0.001$). Lumbopelvic control: Healthy controls had better lumbopelvic movement control than back pain patients: mean ± SD score on the lumbopelvic movement control test for patients = 3 ± 1.1 and for healthy controls = 1 ± 1.3 ($t(88) = 7.9$, $p <0.001$) (Figure 15).

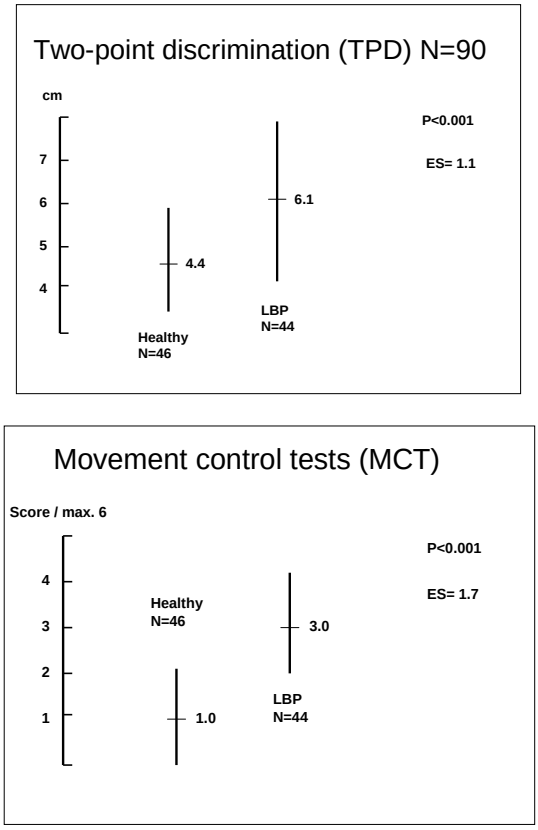
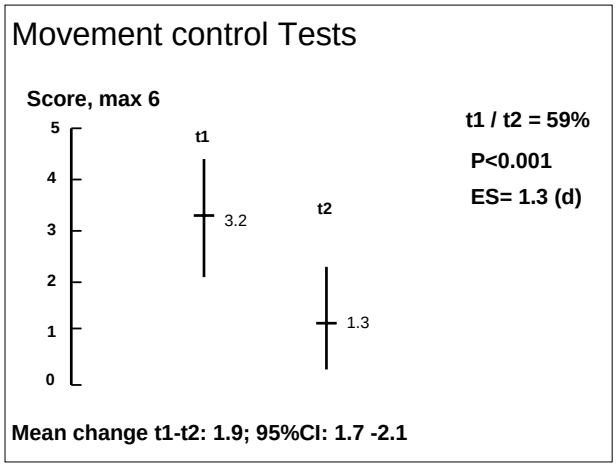


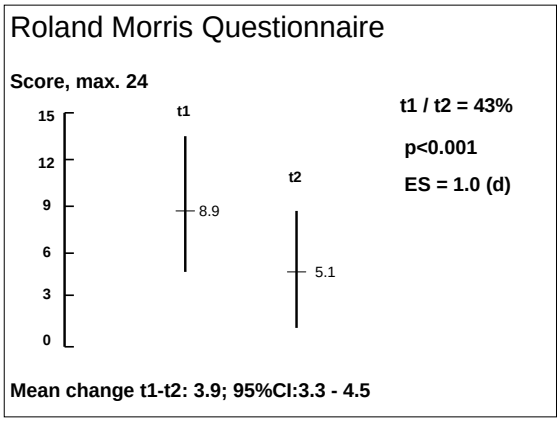
Figure 15. *Results of TPD and MCT in study no. IV*

5.4 Treatment effect of specific movement control exercises

MCT showed a 59% improvement from 3.2 (max 6) to 1.3 positive tests (d= 1.3, p<0.001), complaints (PSFS) decreased 40% from 5.9 points (max 10) to 3.5 (d= 1.3, p<0.001), and disability (RMQ) decreased 43% from 8.9 to 5.1 points (d= 1.0, p<0.001) (Figure 16).

Based on our results, we performed sample size calculations for a planned randomized controlled trial (RCT). We used a power of 0.9 and alpha was set at 0.05. In order to be able to detect a PSFS 1.3 (SD of 1.8) point difference in complaints between an experimental and control group, 40 patients in each group would be needed to detect a significant intergroup difference in the number of positive movement control tests. Detecting a benefit of 1.9 with a SD of 1.5, 48 patients per group would be needed. If disability, assessed with the RMQ, was chosen as the main outcome, 81 subjects in each group would be required to show a significant intergroup difference of 2 points with a SD of 3.9.





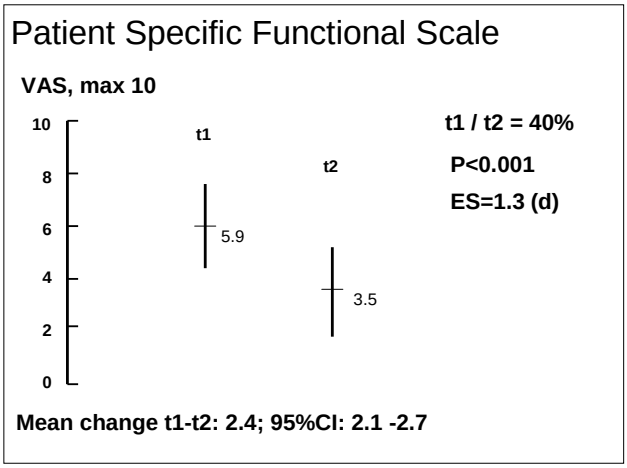


Figure 16. *The results t1 (before the intervention) and t2 (after the intervention) on MC, RMQ and PSFS in case series study where patients were treated with individually approximately 9 times individually to improve the MCI.*

6 Discussion

6.1 Main findings

The aim of this doctoral thesis was to create a concrete and reliable tool for assessment of a specific sub-group of NSLBP. Based on the results from the 5 different studies it can be claimed that the developed test battery with six tests for the measurement of movement control of the low back is reliable and usable in daily practice. It was also shown that the movement control impairment can be improved with specific, individual exercises.

Inter- and intra-tester reliability
It was found that physiotherapists could rate the performance of the patients in the videos in 6 out of 10 tests. As the inter-tester reliability was at least k>0.6 for the 6 tests and intra-tester reliability was even k>0.8 for 5 tests, this test battery can be recommended for clinical use to estimate patients' movement control performance of the low back.

Consistency of the measurement over time
The two tests seem to have an acceptable level of stability and can, therefore, be recommended for clinical use to test the movement control ability in the low back.

Difference in movement control between LBP patients and healthy controls
The only difference in the selection criteria between the groups was whether subjects had LBP or not. This study demonstrated that there is a difference between subjects with and without LBP, which is a first step in the validation process of developing diagnostic tests. In clinical practice, identifiable sub-groups of patients with LBP have been proposed, e.g. flexion, extension, rotational pattern or combinations of these, that are distinguishable from one another based on MC problems.

Movement control and body schema representation of the brain
It was found out that TPD threshold of the back is greater in patients with LBP than in healthy controls and TPD threshold of the back is negatively related to voluntary lumbopelvic control. These findings have implications for back pain rehabilitation in particular and for movement retraining in general.

Treatment effect of specific movement control exercises
Improvement of movement control ability through exercises was associated with decreasing disability and improvement on the person specific functional scale. Since there was no control group, conclusions regarding efficacy are not allowed. In addition,

it remains unclear whether specific exercises are more effective than general exercises. A RCT would address this question. It is important that this study demonstrates the feasibility of such a RCT and enables the power calculation for such a study.

6.2. What does this research add to the existing body of knowledge?

Other reliability studies have been conducted earlier. They have tested individual tests (Murphy et al., 2006) or packages of tests (Van Dillen et al., 1998) or even a comprehensive examination (Dankaerts et al., 2006c). However, we created a clear test set designed for all directions of movement control of the low back. It is easy to use and a clear cut-off point can be given (less than 2/6 positive tests is normal). For the validity, we were the first to show a clear difference in the performance of the set battery compared to healthy controls. Our test-retest study is the first study to have shown the stability of the tests over a relatively short time.

Two-point discrimination has been shown to be changed (increased) in patients with CRPS (Moseley et al., 2008c) and in amputees with phantom pain (Flor and Birbaumer, 2000). However, this is the first time that these changes have been demonstrated in back pain patients.

Finally, although some randomised studies on specific stabilisation exercises have been conducted, in the area of specific movement control only case studies have been published so far. Thus, our case series strengthens the need also for RCT's in this area of research.

6.3 Methodological considerations

6.3.1 Statistical analysis

For the intra-tester and inter-tester study, kappa values were calculated. We calculated the data for the two pairs separately in order to find out whether there was a difference between experienced and less experienced therapists. Since this was not the case, all four therapists could be compared directly with each other.

The second reliability study, in a test-retest setting, used ICC and SDD was used. Another possibility for calculating the test-retest reliability would have been to use Cohen's kappa again. However, we wanted to measure the stability of movement control ability in degrees, which is continuous data, and so ICC and SDD were used.

In the third and fourth studies the means and differences between patients with LBP and healthy controls were calculated. Although in both studies, the group differences

were large and significant, it cannot be stated whether they associate since no intervention was included. Effect sizes were calculated as it enables a comparison of results between different studies and measurements. If the group difference is nearly d=1.2, it means that the groups differentiate 1.2 standard deviations from each other which is a very large distinction between the groups and can nicely be seen in the Figure 14a.

In the case series study the measurements at the beginning were compared with those at the end of the intervention series. Although the results were significant and ES large, the results have to be regarded with caution since there was no control group included.

6.3.2 Reliability

Overall, the inter- and intra-tester reliability of the active movement control tests was considered good to excellent. In the intra-observer comparison, one of the two persons tested, could highly reliably (k=0.69 – 1.0) rate all the tests. The second person tested, rated 8 out of 10 tests (k=0.60-1.0) highly reliably, one test fair (k=0.59; and one test poorly (k=0.22). In the inter-tester reliability, six test showed substantial (k>0.6) values. This was the also the reason to carry on in the cross sectional study with these six tests.
It is worth commenting that all four participants mentioned that better protocol training could have been carried out beforehand. There were two pairs of observers. The more experienced pair demonstrated a better inter-rater reliability than the less experienced pair, which is comparable to the findings by Dankaerts (2006c) and proposed by van Dillen (1998).

6.3.3 Difference between patients with LBP and healthy controls in movement control

According to Sackett (Sackett et al., 1996, Sackett and Haynes, 2002), the first phase of diagnostic research compares test results in patients and control individuals. Ideally, healthy persons should test negative and affected persons test positive. Because LBP is a multidimensional problem, not all patients need to also have problems with MC. On the other hand, if both healthy controls and LBP patients have impaired movement control, the clinical relevance of impaired MC is limited and research on diagnosing MC would not be worthwhile. According to Sackett (Sackett and Haynes, 2002) this first phase of evaluation of a diagnostic test "*can not be translated into diagnostic action but adds to our biological insight into mechanisms of disease and may serve later research into treatment as well as diagnosis*".

Interestingly there was also a difference between patients having pain or troubles somewhere other than in the back and healthy controls. A sub-group analysis indicated that 51 subjects without LBP but pain elsewhere, also had significantly (p<0.01) more

positive tests (mean 1.45 tests compared to 0.75) than healthy controls. This in line with other studies that have found disturbances from back pain in proprioception (Luoto et al., 1998), fine coordination or psychomotor speed (Luoto et al., 1996), and reaction speed of non-back-related muscles (Leinonen et al., 2007). It seems that pain also disturbs motor control in body parts which are not painful, as hypothesised by other authors (Taimela and Luoto, 1999, Moseley, 2005b, Moseley, 2004b, Moseley, 2003, Moseley and Hodges, 2005).

Furthermore, a sensitivity specificity analysis was performed for cut-off points. An interesting finding is that for a cut-off point of 2 positive tests out of 6, the specificity was 0.88. However, this cannot be used as a diagnostic study, as LBP cannot be seen as a gold standard to compare to. We do not need the movement control tests to find out whether a patient has back pain or not because we can ask whether they do or not. This, however, means that 88% of healthy controls do not have two or more positive tests, giving a nice overall picture of what is normal. As the main data showed, the mode of the healthy controls was zero. Therefore, we can conclude that it is normal for a healthy subject to be unable to perform 1 out of 6 tests correctly, but 2 or more positive tests seem not to be normal.

6.3.4 Two-point discrimination and movement control

Both TPD and MC were changed by patients with LBP and they correlate (r=0.49). Taken together, the findings and the current results suggest that tactile discrimination training might help normalise lumbopelvic proprioception and motor control in patients with back pain– perhaps tactile discrimination training should be included in management. Studies that have examined TPD of chronic pain patients include phantom limb pain patients following an amputation (Flor, 2002, Flor and Diers, 2009, Flor et al., 1995) and CRPS patients (Acerra and Moseley, 2005, Moseley, 2005a, Moseley and Wiech, 2009, Moseley et al., 2008c). The representation of a body part in the somatosensory cortex seems to change along the course of chronic pain (Moseley, 2008a, Moseley, 2004c, Flor, 2003a, Flor et al., 1997). This opens new horizons for the rehabilitation of chronic pain patients who show a distorted body image and impaired movement control. Exercises could include motor imagery (Moseley, 2006, Moseley, 2004a, Moseley and Barnett, 2009, Moseley and Gandevia, 2005), mirror or recognition exercises with visual feedback (Claus et al., 2009, Moseley, 2007, Moseley et al., 2008a, Moseley et al., 2006, Moseley et al., 2008b) or TPD training (Moseley et al., 2008c). However, the dosage of these kind of exercises is still unclear and some patients may even get worse (Moseley et al., 2008d).

6.3.5 Improvement of movement control by NSLBP through specific exercises

This was the first study to evaluate a series of cases on movement control ability

following physiotherapy treatment. So far, only three case studies on the efficacy of exercises based on specific movement control findings in the low back have been published (Maluf et al., 2000, Van Dillen et al., 2005, Dankaerts et al., 2007). They all presented good results, also in long-term. These independent single-case studies formed the justification for our study to evaluate movement control exercises in a sub-group of patients with non-specific LBP and MCI.

6.3.6 Limitations

There are several limitations in the studies performed. In the reliability study, it can be argued that physical tests alone are not sufficient but that one should also take into account the anamnesis, history, pain behaviour and further subjective factors, as Dankaerts et al (2006) did. However, since the physical tests are based on inspection and many physiotherapeutic examinations show a poor reliability, it was decided to concentrate on this. The test-retest study can be criticised for using a measurement, SpinalMouse, which is actually not used in a clinical setting, where the MC tests are just rated positive or negative. Thus, a dichotomic rating on both days could have been more relevant. However, we wanted to avoid the inter-tester and intra-tester reliability bias (which we knew from the earlier study to be imperfect) and rely on a more objective measurement device.

In the cross-sectional study, investigating the difference in movement control between patients with LBP and healthy controls, we did not have a gold standard to enable specificity and sensitivity calculations, which are very important in diagnostic studies. Indeed, such a gold standard does not exist yet. In future studies movement laboratory analysis could be used as a gold standard. A further limitation of this study was that the assessors were not blinded and thus they knew who was patient and who not. The blinding could be attained through videos or that assessors would observe the subjects through a noise isolated window to eliminate the bias caused through verbal expressions.

In the case controls study, comparing and correlating TPD and MCT, the hypothesis is that TPD represents the body schema of the sensory cortex. This is so far only an assumption based on studies of patients with phantom limb pain or CRPS. However, TPD measurement is far cheaper than fMRI. Still, future studies with fMRI can be used for assessing the real body representational areas in the brain.

Finally, in the case series study, showing significant and large effects after individual physiotherapy, a direct association cannot be claimed, as there was no control group included. Although the results of well-designed observational studies (with either a cohort or a case–control design) do not necessarily systematically overestimate the magnitude of the effects of treatment as compared with those in RCT's on the same topic (Concato et al., 2000), we consider the lack of a control group as a limitation of our

study. Therefore, we cannot at present draw conclusions as to how patients would have improved without treatment. In addition, no follow-up examinations were conducted. Results were assessed directly only after intervention of average 9 treatment sessions. It is a possibility that improvements could have vanished relatively quickly following the treatment series. However, in the independent single-case studies the patients showed further improvement in the follow-up period. Our patients were mostly subacute with 73% of them still working. This means that our population might not be typical of patients being at great risk of chronicity.

There is strong evidence that psychosocial issues (Kendall, 1997), such as fear avoidance (Linton and Andersson, 2000, Vlaeyen and Linton, 2000, Waddell, 2004) or catastrophising, are the most pertinent factors leading to chronicity, yet we did not measure any of these properties. Furthermore, we did not subclassify the patients beyond that they should have at least 3/6 tests positive in the MCT battery and were not allowed to show ceiling effects for the other outcome measures. It is clear that patients' clinical behaviour should also match that of their movement impairment group. This is also a possible drawback of our study since, although the patients did have movement control deficits, we cannot say whether this was the cause of their back problems. However, with a clearer classification, as suggested by Dankaerts (2006) and Vibe Fersum (2008) and O'Sullivan (2005), even better results could be expected.

6.4 Future directions

As always in research, this project raises a number of new research questions. First, validation of the test battery has to be continued. Studies in a movement laboratory using computerised video analysis and EMG should follow. For a further validation, but also to study efficacy or effectiveness of the therapy, RCTs with sufficient power should follow. Since patients often show spontaneous recovery or improve just by participating in a study (Hawthorn Effect), it is eminent to include control groups with therapy according to current evidence. With back pain this would be advice, patient education and exercise, which could be combined with manual therapy. Because we do not know, whether impaired movement control is a reason or a cause of LBP, longitudinal cohort studies by people without back pain could be examined and followed over longer period of time to see whether they subjects with MCI will have more back pain over time than those with intact movement control. Also, in a RCT, results of movement control tests could be compared with other functional measurements and questionnaires. A further area of interest arising from the two-point discrimination study, is whether we can change the body mapping and awareness in the sensory cortex through TPD training or visual feedback, as has been shown to be effective in patients with phantom limb pain or CRPS.

6.5 Clinical considerations

LBP is an enigma and sub-grouping is a major research challenge. Many sub-grouping systems have been developed but consensus is not in sight. In the clinical guidelines suggested categorisation is too superficial and helps only in the first decision; whether the patient needs – medically seen - specialised care. If not, the pain is only "non-specific". However, exactly this non-specific pain, when chronic, is the biggest problem. Therefore clear, easy and from all professional groups accepted tests to clear out in which sub-group the patient belongs, are needed. Only a clear diagnosis enables a targeted treatment.

Physiotherapists do not make diagnoses. However, in order to be able to assess a patient and tailor an individual therapy program, it is crucial to have reliable and valid tests for correct assessment. Many assessments used in physiotherapy and musculoskeletal medicine have not been validated and reliability studies are missing. It is important that the researchers conducting studies within these research areas are themselves physiotherapists. Otherwise, the developed tests may not be practical for busy everyday practice. Tests and assessments have to be easy to learn and to use, as are the movement control test battery developed and studied in this thesis. Physiotherapists are encouraged to do clinically relevant, pragmatic research. Although time consuming, the studies performed in this thesis were easy and cheap to conduct.

There is a gap between research and clinical practice. On one hand, in many cases, clinicians do not understand importance of research results or cannot interpret the results correctly. On the other hand, researchers are not aware or able to think pragmatically how tests, examinations or treatments work in daily practice. It is senseless to use tests which have been shown to be unreliable or carry on applying techniques which have been shown to have no effect. Then again, there may be many good, easy and reliable tools but no one ever proved them scientifically. Therefore, research has to be a part of also clinical education programs and researchers have to, once in a while, come down to earth in order to understand how clinicians work.

Reliability and treatment results improve with experience. Also in our movement control test battery, which is easy to learn and use, the more experienced clinicians had a higher inter-tester agreement. In the case series study, the therapists had on average 7 years work experience and most of them had a post graduate musculoskeletal degree. This may partly explain the good results. Of course, the belief is that specific problems need specific therapy and this again needs experienced and well educated therapists. However, we need to work further in proving this belief.

7. Conclusions

MCI is a sub-group of NSLBP. An easy test battery was developed to assess the movement control ability of the low back. Future studies on validation of the test set and randomised outcome studies with control studies are warranted.

The research questions can be answered as following:

1. According to the results of this thesis, the inter-tester and intra-tester reliability of the active movement control tests of the low back is 'substantial'.

2. 90 percent of the tested persons performed similarly in two tests on two different days and, therefore, the reproducibility of these tests in day to day comparison is good.

3. There is a clear and significant difference between patients with LBP compared to healthy controls in their movement control.

4. Persons that have an increased TPD also have an impaired movement control of the low back. Distortion of the body scheme might explain why patients cannot control active movement of their back.

5. Improvement of movement control through exercises leads to a decrease of LBP and improves functional disability due to back pain. However, as no control groups were included, no direct conclusions on the efficacy can be drawn.

8. References

ACERRA, N. E. & MOSELEY, G. L. 2005. Dysynchiria: watching the mirror image of the unaffected limb elicits pain on the affected side. *Neurology,* 65**,** 751-3.

AIRAKSINEN, O., BROX, J., CEDRASCHI, C., HILDEBRANDT, J., KLABER-MOFFETT, J., KOVACS, F., MANNION, A., REIS, S., STAAL, J., URSIN, H. & ZANOLI, G. 2006. Chapter 4. European guidelines for the management of chronic nonspecific low back pain. *Eur Spine J,* 15 Suppl 2**,** S192-300.

APKARIAN, A. V. 2008. Pain perception in relation to emotional learning. *Curr Opin Neurobiol,* 18**,** 464-8.

APKARIAN, A. V., BALIKI, M. N. & GEHA, P. Y. 2009. Towards a theory of chronic pain. *Prog Neurobiol,* 87**,** 81-97.

APKARIAN, A. V., SOSA, Y., SONTY, S., LEVY, R. M., HARDEN, R. N., PARRISH, T. B. & GITELMAN, D. R. 2004. Chronic back pain is associated with decreased prefrontal and thalamic gray matter density. *J Neurosci,* 24**,** 10410-5.

ARABADZHIEV, T., SOLOMONOW, M., ZHOU, B. H., DIMITROVA, N. & DIMITROV, G. 2008. Power spectra characteristics associated with static reflexive activation of the multifidus muscle in feline models. *Eur J Appl Physiol,* 104**,** 873-83.

ASTFALCK, R. G., O'SULLIVAN P, B., STRAKER, L. M., SMITH, A. J., BURNETT, A., CANEIRO, J. P. & DANKAERTS, W. 2010. Sitting Postures and Trunk Muscle Activity in Adolescents With and Without Nonspecific Chronic Low Back Pain: An Analysis Based on Subclassification. *Spine (Phila Pa 1976).*

AUVINEN, J. P., TAMMELIN, T. H., TAIMELA, S. P., ZITTING, P. J., JARVELIN, M. R., TAANILA, A. M. & KARPPINEN, J. I. 2010. Is insufficient quantity and quality of sleep a risk factor for neck, shoulder and low back pain? A longitudinal study among adolescents. *Eur Spine J,* 19**,** 641-9.

BALIKI, M. N., GEHA, P. Y., APKARIAN, A. V. & CHIALVO, D. R. 2008. Beyond feeling: chronic pain hurts the brain, disrupting the default-mode network dynamics. *J Neurosci,* 28**,** 1398-403.

BEN-MASAUD, A., SOLOMONOW, D., DAVIDSON, B., ZHOU, B. H., LU, Y., PATEL, V. & SOLOMONOW, M. 2009. Motor control of lumbar instability following exposure to various cyclic load magnitudes. *Eur Spine J,* 18**,** 1022-34.

BERGMARK, A. 1989. Stability of the lumbar spine. A study in mechanical engineering. *Acta Orthop Scand Suppl,* 230**,** 1-54.

BERNA, C., LEKNES, S., HOLMES, E. A., EDWARDS, R. R., GOODWIN, G. M. & TRACEY, I. 2010. Induction of depressed mood disrupts emotion regulation neurocircuitry and enhances pain unpleasantness. *Biol Psychiatry,* 67**,** 1083-90.

BILLIS, E., MCCARTHY, C. & OLDHAM, J. 2007. Subclassification of low back pain: a cross-country comparison. *Eur Spine J,* 16**,** 865-79.

BINKLEY, J., STRATFORD, P. W. & GILL, C. 1995. Interrater reliability of lumbar accessory motion mobility testing. *Physical Therapy,* 75**,** 786-92; discussion 793-5.

BISTRITSCHAN E, D. S. W. G. E. P. 2003. Oberflächenmessverfahren (medimouse) versus Röntgenfunktionsaufnahmen zur Beurteilung der lumbalen Wirbelsäulenbeweglichkeit. *Zeitschrift für Orthopädie,* 141.

BLAND, J. M. & ALTMAN, D. G. 1986. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet,* 1**,** 307-310.

BOGDUK, N. T. L. T. 2008. *Clinical Anatomy of the Lumbar Spine and Sacrum,* Melbourne, Churchill Livingstone.

BOOS, N., RIEDER, R., SCHADE, V., SPRATT, K. F., SEMMER, N. & AEBI, M. 1995. 1995 Volvo Award in clinical sciences. The diagnostic accuracy of magnetic resonance imaging, work perception, and psychosocial factors in identifying symptomatic disc herniations. *Spine (Phila Pa 1976),* 20**,** 2613-25.

BORKAN, J., VAN TULDER, M., REIS, S., SCHOENE, M., CROFT, P. & HERMONI, D. 2002. Advances in the field of low back pain in primary care: a report from the fourth international forum. *Spine,* 27**,** E128-32.

BOUTER, L., VAN TULDER, M. & KOES, B. 1998. Methodologic issues in low back pain research in primary care. *Spine (Phila Pa 1976),* 23**,** 2014-20.

BRENNAN, G. P., FRITZ, J. M., HUNTER, S. J., THACKERAY, A., DELITTO, A. & ERHARD, R. E. 2006. Identifying sub-groups of patients with acute/subacute "nonspecific" low back pain: results of a randomized clinical trial. *Spine,* 31**,** 623-631.

BURI, M., HÄRTER, A. & SOTTAS, G. 2007. Statistiken zur Sozialen Sicherheit. IV Statistik 2007.

CAIRNS, M. C., FOSTER, N. E. & WRIGHT, C. 2006. Randomized controlled trial of specific spinal stabilization exercises and conventional physiotherapy for recurrent low back pain. *Spine (Phila Pa 1976),* 31**,** E670-81.

CALMONTE, R. E. A. 2005. Gesundheit und Gesundheitsverhalten in der Schweiz 1992-2002. Schweizerische Gesundheitsbefragung. Statistik der Schweiz.

CARRAGEE, E., ALAMIN, T., CHENG, I., FRANKLIN, T. & HURWITZ, E. 2006a. Does minor trauma cause serious low back illness? *Spine (Phila Pa 1976),* 31**,** 2942-9.

CARRAGEE, E., ALAMIN, T., CHENG, I., FRANKLIN, T., VAN DEN HAAK, E. & HURWITZ, E. 2006b. Are first-time episodes of serious LBP associated with new MRI findings? *Spine J,* 6**,** 624-35.

CARRAGEE, E. J., LINCOLN, T., PARMAR, V. S. & ALAMIN, T. 2006c. A gold standard evaluation of the "discogenic pain" diagnosis as determined by provocative discography. *Spine (Phila Pa 1976),* 31**,** 2115-23.

CHEUNG, K. M., KARPPINEN, J., CHAN, D., HO, D. W., SONG, Y. Q., SHAM, P., CHEAH, K. S., LEONG, J. C. & LUK, K. D. 2009. Prevalence and pattern of lumbar magnetic resonance imaging changes in a population study of one thousand forty-three individuals. *Spine (Phila Pa 1976),* 34**,** 934-40.

CHILDS, J. D., FRITZ, J. M., PIVA, S. R. & ERHARD, R. E. 2003. Clinical decision making in the identification of patients likely to benefit from spinal manipulation: a traditional versus an evidence-based approach. *The Journal of orthopaedic and sports physical therapy,* 33**,** 259-272.

CHOI, B., K. L., VERBEEK JOS, H., TAM WILSON, W.-S. & JIANG JOHNNY, Y. 2010. Exercises for prevention of recurrences of low-back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD006555/frame.html.

CHOLEWICKI, J., VAN DIEEN, J. H. & ARSENAULT, A. B. 2003. Muscle function and dysfunction in the spine. *J Electromyogr Kinesiol,* 13**,** 303-4.

CHOU, R. & SHEKELLE, P. 2010. Will this patient develop persistent disabling low back pain? *JAMA,* 303**,** 1295-302.

CHOU, R., LOESER, J. D., OWENS, D. K., ROSENQUIST, R. W., ATLAS, S. J., BAISDEN, J., CARRAGEE, E. J., GRABOIS, M., MURPHY, D. R., RESNICK, D. K., STANOS, S. P., SHAFFER, W. O. & WALL, E. M. 2009. Interventional therapies, surgery, and interdisciplinary rehabilitation for low back pain: an evidence-based clinical practice guideline from the American Pain Society. *Spine (Phila Pa 1976),* 34**,** 1066-77.

CLARE, H. A., ADAMS, R. & MAHER, C. G. 2004. A systematic review of efficacy of McKenzie therapy for spinal pain. *The Australian journal of physiotherapy,* 50**,** 209-216.

CLAUS, A. P., HIDES, J. A., MOSELEY, G. L. & HODGES, P. W. 2009. Different ways to balance the spine: subtle changes in sagittal spinal curves affect regional muscle activity. *Spine (Phila Pa 1976),* 34**,** E208-14.

COMERFORD, M. J. & MOTTRAM, S. L. 2001a. Functional stability re-training: principles and strategies for managing mechanical dysfunction. *Manual Therapy,* 6**,** 3-14.

COMERFORD, M. J. & MOTTRAM, S. L. 2001b. Movement and stability dysfunction - contemporary developments. *Manual Therapy,* 6**,** 15-26.

CONCATO, J., SHAH, N. & HORWITZ, R. 2000. Randomized, controlled trials, observational studies, and the hierarchy of research designs. *N Engl J Med,* 342**,** 1887-92.

COOK, C., BRISMEE, J. M. & SIZER, P. S., JR. 2006. Subjective and objective descriptors of clinical

lumbar spine instability: a Delphi study. *Manual therapy,* 11**,** 11-21.

COSTA, L., MAHER, C., LATIMER, J., HODGES, P., HERBERT, R., REFSHAUGE, K., MCAULEY, J. & JENNINGS, M. 2009. Motor control exercise for chronic low back pain: a randomized placebo-controlled trial. *Phys Ther,* 89**,** 1275-86.

D'AMBROSIA, P., KING, K., DAVIDSON, B., ZHOU, B. H., LU, Y. & SOLOMONOW, M. 2010. Pro-inflammatory cytokines expression increases following low- and high-magnitude cyclic loading of lumbar ligaments. *Eur Spine J.*

DAGENAIS, S., TRICCO, A. C. & HALDEMAN, S. 2010. Synthesis of recommendations for the assessment and management of low back pain from recent clinical practice guidelines. *Spine J,* 10**,** 514-29.

DANKAERTS, W., O'SULLIVAN, P., BURNETT, A. & STRAKER, L. 2006a. Altered patterns of superficial trunk muscle activation during sitting in nonspecific chronic low back pain patients: importance of subclassification. *Spine (Phila Pa 1976),* 31**,** 2017-23.

DANKAERTS, W., O'SULLIVAN, P., BURNETT, A. & STRAKER, L. 2006b. Differences in sitting postures are associated with nonspecific chronic low back pain disorders when patients are subclassified. *Spine (Phila Pa 1976),* 31**,** 698-704.

DANKAERTS, W., O'SULLIVAN, P., BURNETT, A., STRAKER, L., DAVEY, P. & GUPTA, R. 2009. Discriminating healthy controls and two clinical sub-groups of nonspecific chronic low back pain patients using trunk muscle activation and lumbosacral kinematics of postures and movements: a statistical classification model. *Spine (Phila Pa 1976),* 34**,** 1610-8.

DANKAERTS, W., O'SULLIVAN, P. B., BURNETT, A. F. & STRAKER, L. M. 2007. The use of a mechanism-based classification system to evaluate and direct management of a patient with non-specific chronic low back pain and motor control impairment--a case report. *Man Ther,* 12**,** 181-91.

DANKAERTS, W., O'SULLIVAN, P. B., STRAKER, L. M., BURNETT, A. F. & SKOUEN, J. S. 2006c. The inter-examiner reliability of a classification method for non-specific chronic low back pain patients with motor control impairment. *Manual therapy,* 11**,** 28-39.

DIONNE, C. E., DUNN, K. M., CROFT, P. R., NACHEMSON, A. L., BUCHBINDER, R., WALKER, B. F., WYATT, M., CASSIDY, J. D., ROSSIGNOL, M., LEBOEUF-YDE, C., HARTVIGSEN, J., LEINO-ARJAS, P., LATZA, U., REIS, S., GIL DEL REAL, M. T., KOVACS, F. M., OBERG, B., CEDRASCHI, C., BOUTER, L. M., KOES, B. W., PICAVET, H. S., VAN TULDER, M. W., BURTON, K., FOSTER, N. E., MACFARLANE, G. J., THOMAS, E., UNDERWOOD, M., WADDELL, G., SHEKELLE, P., VOLINN, E. & VON KORFF, M. 2008. A consensus approach toward the standardization of back pain definitions for use in prevalence studies. *Spine (Phila Pa 1976),* 33**,** 95-103.

DONELSON, R., APRILL, C., MEDCALF, R. & GRANT, W. 1997. A prospective study of centralization of lumbar and referred pain. A predictor of symptomatic discs and anular

competence. *Spine (Phila Pa 1976),* 22**,** 1115-22.

ENGERS, A., J., JELLEMA, P., WENSING, M., VAN DER WINDT DANIËLLE, A. W. M., GROL, R. & VAN TULDER MAURITS, W. 2008. Individual patient education for low back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD004057/frame.html.

ESSENDROP, M., MAUL, I., LAUBLI, T., RIIHIMAKI, H. & SCHIBYE, B. 2002. Measures of low back function: A review of reproducibility studies. *Clinical Biomechanics,* 17**,** 235-249.

FERREIRA, P., FERREIRA, M., MAHER, C., HERBERT, R. & REFSHAUGE, K. 2006. Specific stabilisation exercise for spinal and pelvic pain: a systematic review. *Aust J Physiother,* 52**,** 79-88.

FERSUM, K. V., DANKAERTS, W., O'SULLIVAN, P. B., MAES, J., SKOUEN, J. S., BJORDAL, J. M. & KVALE, A. 2009. Integration of sub-classification strategies in RCTs evaluating manual therapy treatment and exercise therapy for non-specific chronic low back pain (NSCLBP): a systematic review. *Br J Sports Med*.

FLOR, H. 2000. The functional organization of the brain in chronic pain. *Prog Brain Res,* 129**,** 313-22.

FLOR, H. 2002. Phantom-limb pain: characteristics, causes, and treatment. *Lancet Neurol,* 1**,** 182-9.

FLOR, H. 2003a. Cortical reorganisation and chronic pain: implications for rehabilitation. *J Rehabil Med***,** 66-72.

FLOR, H. 2003b. Remapping somatosensory cortex after injury. *Adv Neurol,* 93**,** 195-204.

FLOR, H. & BIRBAUMER, N. 2000. Phantom limb pain: cortical plasticity and novel therapeutic approaches. *Curr Opin Anaesthesiol,* 13**,** 561-4.

FLOR, H., BRAUN, C., ELBERT, T. & BIRBAUMER, N. 1997. Extensive reorganization of primary somatosensory cortex in chronic back pain patients. *Neurosci Lett,* 224**,** 5-8.

FLOR, H. & DIERS, M. 2009. Sensorimotor training and cortical reorganization. *NeuroRehabilitation,* 25**,** 19-27.

FLOR, H., ELBERT, T., KNECHT, S., WIENBRUCH, C., PANTEV, C., BIRBAUMER, N., LARBIG, W. & TAUB, E. 1995. Phantom-limb pain as a perceptual correlate of cortical reorganization following arm amputation. *Nature,* 375**,** 482-4.

FLYNN, T., FRITZ, J., WHITMAN, J., WAINNER, R., MAGEL, J., RENDEIRO, D., BUTLER, B., GARBER, M. & ALLISON, S. 2002. A clinical prediction rule for classifying patients with low back pain who demonstrate short-term improvement with spinal manipulation. *Spine (Phila Pa 1976),* 27**,** 2835-43.

FRITZ, J. M., BRENNAN, G. P., CLIFFORD, S. N., HUNTER, S. J. & THACKERAY, A. 2006. An examination of the reliability of a classification algorithm for sub-grouping patients with low back pain. *Spine,* 31**,** 77-82.

FRITZ, J. M., PIVA, S. R. & CHILDS, J. D. 2005. Accuracy of the clinical examination to predict

radiographic instability of the lumbar spine. *European spine journal : official publication of the European Spine Society,* 14**,** 743-750.

FURLAN, A., D., IMAMURA, M., DRYDEN, T. & IRVIN, E. 2008. Massage for low-back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD001929/frame.html.

FURLAN, A., D., VAN TULDER, M., W., CHERKIN, D., TSUKAYAMA, H., LAO, L., KOES, B., W. & BERMAN, B., M. 2005. Acupuncture and dry-needling for low back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD001351/frame.html.

GIBSON, J. N. A. & WADDELL, G. 2007. Surgical interventions for lumbar disc prolapse. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD001350/frame.html.

GIESECKE, T., GRACELY, R. H., GRANT, M. A., NACHEMSON, A., PETZKE, F., WILLIAMS, D. A. & CLAUW, D. J. 2004. Evidence of augmented central pain processing in idiopathic chronic low back pain. *Arthritis Rheum,* 50**,** 613-23.

HAGEN, K., BIRGER, HILDE, G., JAMTVEDT, G. & WINNEM, M. 2004. Bed rest for acute low-back pain and sciatica. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD001254/frame.html.

HARRIS-HAYES, M. & VAN DILLEN, L. R. 2009. The inter-tester reliability of physical therapists classifying low back pain problems based on the movement system impairment classification system. *PM R,* 1**,** 117-26.

HAYDEN, J., VAN TULDER, M., MALMIVAARA, A. & KOES, B. 2005a. Exercise therapy for treatment of non-specific low back pain. *Cochrane Database Syst Rev***,** CD000335.

HAYDEN, J. A., VAN TULDER, M. W., MALMIVAARA, A. V. & KOES, B. W. 2005b. Meta-analysis: exercise therapy for nonspecific low back pain. *Ann Intern Med,* 142**,** 765-75.

HESTBAEK, L., LEBOEUF-YDE, C. & MANNICHE, C. 2003. Low back pain: what is the long-term course? A review of studies of general patient populations. *Eur Spine J,* 12**,** 149-65.

HEYMANS, M., W., VAN TULDER, M., W., ESMAIL, R., BOMBARDIER, C. & KOES BART, W. 2004. Back schools for non-specific low-back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD000261/frame.html.

HICKS, G., FRITZ, J., DELITTO, A. & MCGILL, S. 2005. Preliminary development of a clinical

prediction rule for determining which patients with low back pain will respond to a stabilization exercise program. *Arch Phys Med Rehabil,* 86**,** 1753-62.

HICKS, G. E., FRITZ, J. M., DELITTO, A. & MISHOCK, J. 2003. Interrater reliability of clinical examination measures for identification of lumbar segmental instability. *Archives of Physical Medicine and Rehabilitation,* 84**,** 1858-1864.

HIDES JA, RICHARDSON CA & GA, J. 1996. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine,* 21**,** 2763-2769.

HIDES, J. A., JULL, G. A. & RICHARDSON, C. A. 2001. Long-term effects of specific stabilizing exercises for first-episode low back pain. *Spine (Phila Pa 1976),* 26**,** E243-8.

HIDES, J. A., RICHARDSON, C. A. & JULL, G. A. 1995. Magnetic resonance imaging and ultrasonography of the lumbar multifidus muscle. Comparison of two different modalities. *Spine (Phila Pa 1976),* 20**,** 54-8.

HIDES, J. A., RICHARDSON, C. A. & JULL, G. A. 1996. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine (Phila Pa 1976),* 21**,** 2763-9.

HIDES, J. A., STOKES, M. J., SAIDE, M., JULL, G. A. & COOPER, D. H. 1994. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. *Spine; Spine,* 19**,** 165-172.

HODGES, P. W. 1999. Is there a role for transversus abdominis in lumbo-pelvic stability? *Manual Therapy,* 4**,** 74-86.

HODGES PW & CA, R. 1997. Contraction of the abdominal muscles associated with movement of the lower limb. *Physical Therapy,* 77**,** 132-144.

HODGES PW, RICHARDSON C & G, J. 1996. Evaluation of the relationship between laboratory and clinical tests of transverses abdominus function. *Physiotherapy Research International,* 1**,** 30-40.

HODGES, P. W. & MOSELEY, G. L. 2003. Pain and motor control of the lumbopelvic region: effect and possible mechanisms. *J Electromyogr Kinesiol,* 13**,** 361-70.

HODGES, P. W., MOSELEY, G. L., GABRIELSSON, A. & GANDEVIA, S. C. 2003. Experimental muscle pain changes feedforward postural responses of the trunk muscles. *Exp Brain Res,* 151**,** 262-71.

IBRAHIM, T., TLEYJEH, I. M. & GABBAR, O. 2008. Surgical versus non-surgical treatment of chronic low back pain: a meta-analysis of randomised trials (Structured abstract). *International Orthopaedics* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/cldare/articles/DARE-12008104608/frame.html.

IGARASHI, T., KIKUCHI, S., SHUBAYEV, V. & MYERS, R. R. 2000. 2000 Volvo Award winner in basic science studies: Exogenous tumor necrosis factor-alpha mimics nucleus pulposus-

induced neuropathology. Molecular, histologic, and behavioral comparisons in rats. *Spine (Phila Pa 1976),* 25**,** 2975-80.

INTERPHARMA 2007. Gesundheitswesen Schweiz.

JACKSON, M., SOLOMONOW, M., ZHOU, B., BARATTA, R. V. & HARRIS, M. 2001. Multifidus EMG and tension-relaxation recovery after prolonged static lumbar flexion. *Spine (Phila Pa 1976),* 26**,** 715-23.

JENSEN, M. C., BRANT-ZAWADZKI, M. N., OBUCHOWSKI, N., MODIC, M. T., MALKASIAN, D. & ROSS, J. S. 1994. Magnetic resonance imaging of the lumbar spine in people without back pain. *N Engl J Med,* 331**,** 69-73.

JULL, G. A. & RICHARDSON, C. A. 2000. Motor control problems in patients with spinal pain: a new direction for therapeutic exercise. *J Manipulative Physiol Ther,* 23**,** 115-7.

KAILA-KANGAS, L., KIVIMAKI, M., HARMA, M., RIIHIMAKI, H., LUUKKONEN, R., KIRJONEN, J. & LEINO-ARJAS, P. 2006. Sleep disturbances as predictors of hospitalization for back disorders-a 28-year follow-up of industrial employees. *Spine (Phila Pa 1976),* 31**,** 51-6.

KANKAANPAA, M., COLIER, W. N., TAIMELA, S., ANDERS, C., AIRAKSINEN, O., KOKKO-ARO, S. M. & HANNINEN, O. 2005. Back extensor muscle oxygenation and fatigability in healthy subjects and low back pain patients during dynamic back extension exertion. *Pathophysiology*.

KARJALAINEN, K., A., MALMIVAARA, A., VAN TULDER, M., W., ROINE, R., JAUHIAINEN, M., HURRI, H. & KOES, B., W. 2003. Multidisciplinary biopsychosocial rehabilitation for subacute low-back pain among working age adults. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD002193/frame.html.

KELLER, A., HAYDEN, J., BOMBARDIER, C. & VAN TULDER, M. 2007. Effect sizes of non-surgical treatments of non-specific low-back pain. *Eur Spine J,* 16**,** 1776-88.

KENDALL N, MAIN C 1997. Guide to assessing psychosocial yellow flags in acute low back pain. *In:* N, K. (ed.) *Commitee ARCICoNZatNH.* Wellington, New Zealand.

KENT, P. & KEATING, J. L. 2005. Classification in nonspecific low back pain: what methods do primary care clinicians currently use? *Spine,* 30**,** 1433-1440.

KILPIKOSKI, S., AIRAKSINEN, O., KANKAANPÄÄ, M., LEMINEN, P., VIDEMAN, T. & ALEN, M. 2002. Interexaminer reliability of low back pain assessment using the McKenzie method. *Spine (Phila Pa 1976),* 27**,** E207-14.

KING, K., DAVIDSON, B., ZHOU, B. H., LU, Y. & SOLOMONOW, M. 2009. High magnitude cyclic load triggers inflammatory response in lumbar ligaments. *Clin Biomech (Bristol, Avon),* 24**,** 792-8.

KJAER, P., LEBOEUF-YDE, C., KORSHOLM, L., SORENSEN, J. S. & BENDIX, T. 2005. Magnetic resonance imaging and low back pain in adults: a diagnostic imaging study of 40-year-old men and women. *Spine (Phila Pa 1976),* 30**,** 1173-80.

KLEIN-VOGELBACH, S. 2001. *Funktionelle Bewegungslehre,* Berlin, Heidelberg**,** Springer Verlag.

KOOL, J., DE BIE, R., OESCH, P., KNÜSEL, O., VAN DEN BRANDT, P. & BACHMANN, S. 2004b. Exercise reduces sick leave in patients with non-acute non-specific low back pain: a meta-analysis. *J Rehabil Med,* 36**,** 49-62.

LABRY, R., SBRICCOLI, P., ZHOU, B. H. & SOLOMONOW, M. 2004. Longer static flexion duration elicits a neuromuscular disorder in the lumbar spine. *J Appl Physiol,* 96**,** 2005-15.

LANDIS, J. & KOCH, G. 1977a. An application of hierarchical kappa-type statistics in the assessment of majority agreement among multiple observers. *Biometrics,* 33**,** 363-74.

LANDIS, J. & KOCH, G. 1977b. The measurement of observer agreement for categorical data. *Biometrics,* 33**,** 159-74.

LE, B., DAVIDSON, B., SOLOMONOW, D., ZHOU, B. H., LU, Y., PATEL, V. & SOLOMONOW, M. 2009. Neuromuscular control of lumbar instability following static work of various loads. *Muscle Nerve,* 39**,** 71-82.

LEINONEN, V., AIRAKSINEN, M., TAIMELA, S., KANKAANPAA, M., KUKKA, A., KOIVISTO, T. & AIRAKSINEN, O. 2007. Low back pain suppresses preparatory and triggered upper-limb activation after sudden upper-limb loading. *Spine (Phila Pa 1976),* 32**,** E150-5.

LEINONEN, V., KANKAANPAA, M., HANNINEN, O., AIRAKSINEN, O. & TAIMELA, S. 2002a. Paraspinal muscle responses during sudden upper limb loading. *Eur J Appl Physiol,* 88**,** 42-9.

LEINONEN, V., KANKAANPAA, M., LUUKKONEN, M., HANNINEN, O., AIRAKSINEN, O. & TAIMELA, S. 2001. Disc herniation-related back pain impairs feed-forward control of paraspinal muscles. *Spine (Phila Pa 1976),* 26**,** E367-72.

LEINONEN, V., KANKAANPAA, M., LUUKKONEN, M., KANSANEN, M., HANNINEN, O., AIRAKSINEN, O. & TAIMELA, S. 2003. Lumbar paraspinal muscle function, perception of lumbar position, and postural control in disc herniation-related back pain. *Spine (Phila Pa 1976),* 28**,** 842-8.

LEINONEN, V., MAATTA, S., TAIMELA, S., HERNO, A., KANKAANPAA, M., PARTANEN, J., KANSANEN, M., HANNINEN, O. & AIRAKSINEN, O. 2002b. Impaired lumbar movement perception in association with postural stability and motor- and somatosensory-evoked potentials in lumbar spinal stenosis. *Spine (Phila Pa 1976),* 27**,** 975-83.

LINTON, S. J. 2000. A review of psychological risk factors in back and neck pain. *Spine (Phila Pa 1976),* 25**,** 1148-56.

LINTON, S. J. & ANDERSSON, T. 2000. Can chronic disability be prevented? A randomized trial of

a cognitive-behavior intervention and two forms of information for patients with spinal pain. *Spine (Phila Pa 1976),* 25**,** 2825-31; discussion 2824.

LINTON , S. T. 2005. *Understanding pain for better clinical practice. A psychological perspective,* London, Elsevier.

LUNDY EKMAN, L. 2007. *Neuroscience, Fundamentals for rehabilitation,* St. Louis, Saunders Elsevier.

LUOMAJOKI, H. A. 2002. Was ist die Evidenz für Training und Übungen bei LBP. *Manuelle Therapie (German).*

LUOTO, S., AALTO, H., TAIMELA, S., HURRI, H., PYYKKO, I. & ALARANTA, H. 1998. One-footed and externally disturbed two-footed postural control in patients with chronic low back pain and healthy control subjects. A controlled study with follow-up. *Spine,* 23**,** 2081-9; discussion 2089-90.

LUOTO, S., TAIMELA, S., HURRI, H., AALTO, H., PYYKKO, I. & ALARANTA, H. 1996. Psychomotor speed and postural control in chronic low back pain patients A controlled follow-up study. *Spine (Phila Pa 1976),* 21**,** 2621-7.

LUOTO, S., TAIMELA, S., HURRI, H. & ALARANTA, H. 1999. Mechanisms explaining the association between low back trouble and deficits in information processing. A controlled study with follow-up. *Spine (Phila Pa 1976),* 24**,** 255-61.

MAITLAND G, H. E., BANKS K, ENGLISH K, 2006. *Maitland Manipulation der Wirbelsäule*, Springer Medizin Verlag.

MALUF, K. S., SAHRMANN, S. A. & VAN DILLEN, L. R. 2000. Use of a classification system to guide nonsurgical management of a patient with chronic low back pain. *Physical Therapy,* 80**,** 1097-1111.

MANNION, A. F., KNECHT, K., BALABAN, G., DVORAK, J. & GROB, D. 2004. A new skin-surface device for measuring the curvature and global and segmental ranges of motion of the spine: reliability of measurements and comparison with data reviewed from the literature. *European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society,* 13**,** 122-136.

MARTIMO, K.-P., VERBEEK JOS, H., KARPPINEN, J., FURLAN ANDREA, D., KUIJER, P. P. F. M., VIIKARI-JUNTURA, E., TAKALA, E.-P. & JAUHIAINEN, M. 2007. Manual material handling advice and assistive devices for preventing and treating back pain in workers. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD005958/frame.html.

MATTILA, V., SAHI, T., JORMANAINEN, V. & PIHLAJAMÄKI, H. 2008. Low back pain and its risk indicators: a survey of 7,040 Finnish male conscripts. *Eur Spine J,* 17**,** 64-9.

MCGILL, S. 2007. *Low back disorders: evidence based prevention and rehabilitation,* Champaign, USA,

Human Kinetics.

MOBERG, E. 1990. Surgical rehabilitation of the upper limb in tetraplegia. *Paraplegia,* 28**,** 330-4.

MOSELEY, G. L. 2003. A pain neuromatrix approach to patients with chronic pain. *Man Ther,* 8**,** 130-40.

MOSELEY, G. L. 2004a. Graded motor imagery is effective for long-standing complex regional pain syndrome: a randomised controlled trial. *Pain,* 108**,** 192-8.

MOSELEY, G. L. 2004b. Impaired trunk muscle function in sub-acute neck pain: etiologic in the subsequent development of low back pain? *Man Ther,* 9**,** 157-63.

MOSELEY, G. L. 2004c. Why do people with complex regional pain syndrome take longer to recognize their affected hand? *Neurology,* 62**,** 2182-6.

MOSELEY, G. L. 2005a. Distorted body image in complex regional pain syndrome. *Neurology,* 65**,** 773.

MOSELEY, G. L. 2005b. Widespread brain activity during an abdominal task markedly reduced after pain physiology education: fMRI evaluation of a single patient with chronic low back pain. *Aust J Physiother,* 51**,** 49-52.

MOSELEY, G. L. 2006. Graded motor imagery for pathologic pain: a randomized controlled trial. *Neurology,* 67**,** 2129-34.

MOSELEY, G. L. 2007. Using visual illusion to reduce at-level neuropathic pain in paraplegia. *Pain,* 130**,** 294-8.

MOSELEY, G. L. 2008a. I can't find it! Distorted body image and tactile dysfunction in patients with chronic back pain. *Pain,* 140**,** 239-43.

MOSELEY, G. L. 2008b. Pain, brain imaging and physiotherapy--opportunity is knocking. *Man Ther,* 13**,** 475-7.

MOSELEY, G. L. & BARNETT, C. 2009. Motor imagery for peripheral injury. *Arch Phys Med Rehabil,* 90**,** 1443; author reply 1443-4.

MOSELEY, G. L., GALLACE, A. & SPENCE, C. 2008a. Is mirror therapy all it is cracked up to be? Current evidence and future directions. *Pain,* 138**,** 7-10.

MOSELEY, G. L. & GANDEVIA, S. C. 2005. Sensory-motor incongruence and reports of 'pain'. *Rheumatology (Oxford),* 44**,** 1083-5.

MOSELEY, G. L. & HODGES, P. W. 2005. Are the changes in postural control associated with low back pain caused by pain interference? *Clin J Pain,* 21**,** 323-9.

MOSELEY, G. L., HODGES, P. W. & GANDEVIA, S. C. 2002. Deep and superficial fibers of the lumbar multifidus muscle are differentially active during voluntary arm movements. *Spine (Phila Pa 1976),* 27**,** E29-36.

MOSELEY, G. L., HODGES, P. W. & GANDEVIA, S. C. 2003. External perturbation of the trunk in standing humans differentially activates components of the medial back muscles. *J Physiol,* 547**,** 581-7.

MOSELEY, G. L., MCCORMICK, K., HUDSON, M. & ZALUCKI, N. 2006. Disrupted cortical proprioceptive representation evokes symptoms of peculiarity, foreignness and swelling, but not pain. *Rheumatology (Oxford),* 45**,** 196-200.

MOSELEY, G. L., PARSONS, T. J. & SPENCE, C. 2008b. Visual distortion of a limb modulates the pain and swelling evoked by movement. *Curr Biol,* 18**,** R1047-8.

MOSELEY, G. L. & WIECH, K. 2009. The effect of tactile discrimination training is enhanced when patients watch the reflected image of their unaffected limb during training. *Pain,* 144**,** 314-9.

MOSELEY, G. L., ZALUCKI, N. M. & WIECH, K. 2008c. Tactile discrimination, but not tactile stimulation alone, reduces chronic limb pain. *Pain,* 137**,** 600-8.

MOSELEY, G. L., ZALUCKI, N., BIRKLEIN, F., MARINUS, J., VAN HILTEN, J. J. & LUOMAJOKI, H. 2008d. Thinking about movement hurts: the effect of motor imagery on pain and swelling in people with chronic arm pain. *Arthritis Rheum,* 59**,** 623-31.

MURPHY, D. R., BYFIELD, D., MCCARTHY, P., HUMPHREYS, K., GREGORY, A. A. & ROCHON, R. 2006. Interexaminer reliability of the hip extension test for suspected impaired motor control of the lumbar spine. *Journal of manipulative and physiological therapeutics,* 29**,** 374-377.

NIEMISTO, L., KALSO EIJA, A., MALMIVAARA, A., SEITSALO, S. & HURRI, H. 2003. Radiofrequency denervation for neck and back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD004058/frame.html.

O'SULLIVAN, P. 2005. Diagnosis and classification of chronic low back pain disorders: Maladaptive movement and motor control impairments as underlying mechanism. *Manual Therapy,* 10**,** 242-255.

O'SULLIVAN, P., PHYTY, G., TWOMEY, L. & ALLISON, G. 1997. Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine,* 22**,** 2959-67.

O'SULLIVAN, P. B. 2000. Masterclass. Lumbar segmental `instability': clinical presentation and specific stabilizing exercise management. *Manual Therapy,* 5**,** 2-12.

O'SULLIVAN, P. B., BURNETT, A., FLOYD, A. N., GADSDON, K., LOGIUDICE, J., MILLER, D. & QUIRKE, H. 2003. Lumbar repositioning deficit in a specific low back pain population. *Spine,* 28**,** 1074-1079.

OESCH, P., KOOL, J., HAGEN, K. B. & BACHMANN, S. 2010. Effectiveness of exercise on work disability in patients with non-acute non-specific low back pain: Systematic review and meta-analysis of randomised controlled trials. *J Rehabil Med,* 42**,** 193-205.

OLSON, M. W., LI, L. & SOLOMONOW, M. 2009. Interaction of viscoelastic tissue compliance with

lumbar muscles during passive cyclic flexion-extension. *J Electromyogr Kinesiol,* 19**,** 30-8.

OSTELO, R., W. J. G., VAN TULDER, M., W., VLAEYEN, J., W. S., LINTON, S., J., MORLEY, S. & ASSENDELFT, W., J. J. 2005. Behavioural treatment for chronic low-back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD002014/frame.html.

OSTELO, R. W., DE VET, H. C., WADDELL, G., KERCKHOFFS, M. R., LEFFERS, P. & VAN TULDER, M. W. 2002. Rehabilitation after lumbar disc surgery. *Cochrane Database Syst Rev***,** CD003007.

PAALANNE, N., KORPELAINEN, R., TAIMELA, S., REMES, J., MUTANEN, P. & KARPPINEN, J. 2008. Isometric trunk muscle strength and body sway in relation to low back pain in young adults. *Spine (Phila Pa 1976),* 33**,** E435-41.

PANJABI, M. M. 1992. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *Journal of spinal disorders; Journal of spinal disorders,* 5**,** 383-9; discussion 397.

PANJABI, M. M. 2003. Clinical spinal instability and low back pain. *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology; Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology,* 13**,** 371-379.

PANJABI, M. M., LYDON, C., VASAVADA, A., GROB, D., CRISCO, J. J., 3RD & DVORAK, J. 1994. On the understanding of clinical instability. *Spine; Spine,* 19**,** 2642-2650.

PENGEL, L., HERBERT, R., MAHER, C. & REFSHAUGE, K. 2003. Acute low back pain: systematic review of its prognosis. *BMJ,* 327**,** 323.

PENNICK, V. & YOUNG, G. 2007. Interventions for preventing and treating pelvic and back pain in pregnancy. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD001139/frame.html.

PLEGER, B., TEGENTHOFF, M., RAGERT, P., FORSTER, A. F., DINSE, H. R., SCHWENKREIS, P., NICOLAS, V. & MAIER, C. 2005. Sensorimotor retuning [corrected] in complex regional pain syndrome parallels pain reduction. *Ann Neurol,* 57**,** 425-9.

PORTNEY, L. G. & WATKINS, M. P. 2000. *Foundations of clinical research: applications to practice,* New Jersey, Prentice Hall Health.

RACKWITZ, B., DE BIE, R., LIMM, H., VON GARNIER, K., EWERT, T. & STUCKI, G. 2006. Segmental stabilizing exercises and low back pain. What is the evidence? A systematic review of randomized controlled trials. *Clin Rehabil,* 20**,** 553-67.

RADEBOLD, A., CHOLEWICKI, J., POLZHOFER, G. K. & GREENE, H. S. 2001. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients

with chronic idiopathic low back pain. *Spine (Phila Pa 1976),* 26**,** 724-30.

RASMUSSEN-BARR, E., NILSSON-WIKMAR, L. & ARVIDSSON, I. 2003. Stabilizing training compared with manual treatment in sub-acute and chronic low-back pain. *Man Ther,* 8**,** 233-41.

RICHARDSON C, J. G. H. P. H. J. 1999. *Therapeutic exercise for spinal segmental stabilisation in low back pain, scientific basis and clinical approach,* London., Churchill Livingstone.

RICHARDSON CA & JULL, G. 1995. Muscle control – pain control. What exercises would you prescribe? *Manual Therapy,* 1**,** 2-10.

RICHARDSON CA, JULL GA & BA, R. Year. A dysfunction of the deep muscles exists in low back pain patients. *In:* Proceedings of the World Confederation for Physical Therapy, Washington, WCPT, London, 1995.

RICHARDSON, C. H., P. HIDES, J. 2004. *Therapeutic Exercise for Lumbopelvic Stabilization: A Motor Control Approach for the Treatment and Prevention of Low Back Pain,* London, Churchill Livingstone.

RICHARDSON, P. W. H. A. C. A. 1999. Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. *Archives of Physical Medicine and Rehabilitation***,** 1005-1012.

ROELOFS, P., D. D. M., DEYO, R., A., KOES, B., W., SCHOLTEN, R., J. P. M. & VAN TULDER, M., W. 2008. Non-steroidal anti-inflammatory drugs for low back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD000396/frame.html.

ROLAND, M. & MORRIS, R. 1983. A study of the natural history of back pain. Part I: development of a reliable and sensitive measure of disability in low-back pain. *Spine,* 8**,** 141-4.

SACKETT, D., ROSENBERG, W., GRAY, J., HAYNES, R. & RICHARDSON, W. 1996. Evidence based medicine: what it is and what it isn't. *BMJ,* 312**,** 71-2.

SACKETT, D. L. & HAYNES, R. B. 2002. The architecture of diagnostic research. *BMJ (Clinical research ed.),* 324**,** 539-541.

SAHRMANN, S. A. 2002. *Diagnosis and treatment of movement impairment syndromes,* St.Louis, Mosby.

SBRICCOLI, P., SOLOMONOW, M., ZHOU, B. H. & LU, Y. 2007. Work to rest durations ratios exceeding unity are a risk factor for low back disorder; a feline model. *J Electromyogr Kinesiol,* 17**,** 142-52.

SBRICCOLI, P., YOUSUF, K., KUPERSHTEIN, I., SOLOMONOW, M., ZHOU, B. H., ZHU, M. P. & LU, Y. 2004. Static load repetition is a risk factor in the development of lumbar cumulative musculoskeletal disorder. *Spine (Phila Pa 1976),* 29**,** 2643-53.

SCHAAFSMA, F., SCHONSTEIN, E., WHELAN KARYN, M., ULVESTAD, E., KENNY DIANNA,

T. & VERBEEK JOS, H. 2010. Physical conditioning programs for improving work outcomes in workers with back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD001822/frame.html.

SCHMID, A., BRUNNER, F., WRIGHT, A. & BACHMANN, L. M. 2008. Paradigm shift in manual therapy? Evidence for a central nervous system component in the response to passive cervical joint mobilisation. *Man Ther,* 13**,** 387-96.

SCHMIDHAUSER, S. E. A. 2008. Economic costs of low back pain in Switzerland. Report for Swiss National Research program NRP 53 "Musculoskeletal Health - chronic pain". Winterthur.

SCHNEIDER, M., ERHARD, R., BRACH, J., TELLIN, W., IMBARLINA, F. & DELITTO, A. 2008. Spinal palpation for lumbar segmental mobility and pain provocation: an interexaminer reliability study. *J Manipulative Physiol Ther,* 31**,** 465-73.

SCHOLTES, S. A., GOMBATTO, S. P. & VAN DILLEN, L. R. 2009. Differences in lumbopelvic motion between people with and people without low back pain during two lower limb movement tests. *Clin Biomech (Bristol, Avon),* 24**,** 7-12.

SCHULZ, S. 1999. Measurement of shape and mobility of the spinal column: Validation of the SpinalMouse by comparison with functional radiographs. *Doctoral Dissertation. Ludwig-Maximilians University, München*.

SHIRI, R., KARPPINEN, J., LEINO-ARJAS, P., SOLOVIEVA, S. & VIIKARI-JUNTURA, E. 2010a. The association between obesity and low back pain: a meta-analysis. *Am J Epidemiol,* 171**,** 135-54.

SHIRI, R., KARPPINEN, J., LEINO-ARJAS, P., SOLOVIEVA, S. & VIIKARI-JUNTURA, E. 2010b. The association between smoking and low back pain: a meta-analysis. *Am J Med,* 123**,** 87 e7-35.

SHIRI, R., SOLOVIEVA, S., HUSGAFVEL-PURSIAINEN, K., VIIKARI, J., RAITAKARI, O. T. & VIIKARI-JUNTURA, E. 2010c. Incidence of nonspecific and radiating low back pain: followup of 24-39-year-old adults of the Young Finns Study. *Arthritis Care Res (Hoboken),* 62**,** 455-9.

SHUMWAY-COOK, A. W. M. H. 2007. *Motor control. Translating research into clinical practice,* Baltimore.

SMEETS, R. J., WITTINK, H., HIDDING, A. & KNOTTNERUS, J. A. 2006. Do patients with chronic low back pain have a lower level of aerobic fitness than healthy controls?: are pain, disability, fear of injury, working status, or level of leisure time activity associated with the difference in aerobic fitness level? *Spine (Phila Pa 1976),* 31**,** 90-7; discussion 98.

SOLOMONOW, D., DAVIDSON, B., ZHOU, B. H., LU, Y., PATEL, V. & SOLOMONOW, M. 2008. Neuromuscular neutral zones response to cyclic lumbar flexion. *J Biomech,* 41**,** 2821-8.

SOLOMONOW, M. 2006. Sensory-motor control of ligaments and associated neuromuscular

disorders. *J Electromyogr Kinesiol,* 16**,** 549-67.

SOLOMONOW, M. 2009. Ligaments: a source of musculoskeletal disorders. *J Bodyw Mov Ther,* 13**,** 136-54.

SOLOMONOW, M., BARATTA, R. V., BANKS, A., FREUDENBERGER, C. & ZHOU, B. H. 2003a. Flexion-relaxation response to static lumbar flexion in males and females. *Clin Biomech (Bristol, Avon),* 18**,** 273-9.

SOLOMONOW, M., BARATTA, R. V., ZHOU, B. H., BURGER, E., ZIESKE, A. & GEDALIA, A. 2003b. Muscular dysfunction elicited by creep of lumbar viscoelastic tissue. *J Electromyogr Kinesiol,* 13**,** 381-96.

SOLOMONOW, M., EVERSULL, E., HE ZHOU, B., BARATTA, R. V. & ZHU, M. P. 2001. Neuromuscular neutral zones associated with viscoelastic hysteresis during cyclic lumbar flexion. *Spine (Phila Pa 1976),* 26**,** E314-24.

SPITZER, W. O. 1987. Scientific approach to the assessment and management of activity-related spinal disorders. A monograph for clinicians. Report of the Quebec Task Force on Spinal Disorders. *Spine,* 12**,** S1-S59.

STAAL, J. B., DE BIE, R., DE VET HENRICA, C. W., HILDEBRANDT, J. & NELEMANS, P. 2008. Injection therapy for subacute and chronic low-back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD001824/frame.html.

STRADFORD, P., GILL, C., WESTWAY, M. & BINKLEY, J. 1995. Assessing Disability and Change on Individual Patients: A Report of a Patient Specific Measure. *Physiotherapy Canada,* 47**,** 258-263.

STUGE, B., LAERUM, E., KIRKESOLA, G. & VØLLESTAD, N. 2004. The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy: a randomized controlled trial. *Spine,* 29**,** 351-9.

TAIMELA, S., KANKAANPAA, M. & LUOTO, S. 1999. The effect of lumbar fatigue on the ability to sense a change in lumbar position. A controlled study. *Spine (Phila Pa 1976),* 24**,** 1322-7.

TAIMELA, S., KUJALA, U., SALMINEN, J. & VILJANEN, T. 1997. The prevalence of low back pain among children and adolescents. A nationwide, cohort-based questionnaire survey in Finland. *Spine (Phila Pa 1976),* 22**,** 1132-6.

TAIMELA, S. & LUOTO, S. 1999. Does disturbed movement regulation cause chronic back trouble? *Duodecim; laaketieteellinen aikakauskirja,* 115**,** 1669-1676.

TAKATALO, J., KARPPINEN, J., NIINIMAKI, J., TAIMELA, S., NAYHA, S., JARVELIN, M. R., KYLLONEN, E. & TERVONEN, O. 2009. Prevalence of degenerative imaging findings in lumbar magnetic resonance imaging among young adults. *Spine (Phila Pa 1976),* 34**,** 1716-21.

VAN DIEEN, J. H., CHOLEWICKI, J. & RADEBOLD, A. 2003a. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. *Spine (Phila Pa 1976),* 28**,** 834-41.

VAN DIEEN, J. H., SELEN, L. P. & CHOLEWICKI, J. 2003b. Trunk muscle activation in low-back pain patients, an analysis of the literature. *J Electromyogr Kinesiol,* 13**,** 333-51.

VAN DILLEN, L. R., SAHRMANN, S. A., NORTON, B. J., CALDWELL, C. A., FLEMING, D. A., MCDONNELL, M. K. & WOOLSEY, N. B. 1998. Reliability of physical examination items used for classification of patients with low back pain. *Physical Therapy,* 78**,** 979-988.

VAN DILLEN, L. R., SAHRMANN, S. A., NORTON, B. J., CALDWELL, C. A., MCDONNELL, M. K. & BLOOM, N. 2003a. The effect of modifying patient-preferred spinal movement and alignment during symptom testing in patients with low back pain: a preliminary report. *Archives of Physical Medicine and Rehabilitation,* 84**,** 313-322.

VAN DILLEN, L. R., SAHRMANN, S. A., NORTON, B. J., CALDWELL, C. A., MCDONNELL, M. K. & BLOOM, N. J. 2003b. Movement system impairment-based categories for low back pain: stage 1 validation. *The Journal of orthopaedic and sports physical therapy,* 33**,** 126-142.

VAN DILLEN, L. R., SAHRMANN, S. A. & WAGNER, J. M. 2005. Classification, intervention, and outcomes for a person with lumbar rotation with flexion syndrome. *Physical Therapy,* 85**,** 336-351.

VAN TRIJFFEL, E., ANDEREGG, Q., BOSSUYT, P. M. & LUCAS, C. 2005. Inter-examiner reliability of passive assessment of intervertebral motion in the cervical and lumbar spine: a systematic review. *Manual therapy,* 10**,** 256-269.

VAN TULDER, M., BECKER, A., BEKKERING, T., BREEN, A., DEL REAL, M. T., HUTCHINSON, A., KOES, B., LAERUM, E. & MALMIVAARA, A. 2006. Chapter 3. European guidelines for the management of acute nonspecific low back pain in primary care. *Eur Spine J,* 15 Suppl 2**,** S169-91.

VAN TULDER, M., MALMIVAARA, A., ESMAIL, R. & KOES, B. 2000a. Exercise therapy for low back pain: a systematic review within the framework of the cochrane collaboration back review group. *Spine,* 25**,** 2784-96.

VAN TULDER, M. W., OSTELO, R., VLAEYEN, J. W., LINTON, S. J., MORLEY, S. J. & ASSENDELFT, W. J. 2000b. Behavioral treatment for chronic low back pain: a systematic review within the framework of the Cochrane Back Review Group. *Spine (Phila Pa 1976),* 25**,** 2688-99.

VIBE FERSUM, K., O'SULLIVAN, P. B., KVALE, A. & SKOUEN, J. S. 2009. Inter-examiner reliability of a classification system for patients with non-specific low back pain. *Man Ther,* 14**,** 555-61.

VIDEMAN, T. & NURMINEN, M. 2004. The occurrence of anular tears and their relation to lifetime back pain history: a cadaveric study using barium sulfate discography. *Spine (Phila Pa*

1976), 29**,** 2668-76.

VLAEYEN, J. W. & LINTON, S. J. 2000. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain,* 85**,** 317-32.

WADDELL, G. 2004. *Back pain revolution,* Churchill-Livingstone.

WADDELL, G. 2005. Sub-groups within "nonspecific" low back pain. *J Rheumatol,* 32**,** 395-6.

WALKER, B., F., FRENCH, S., D., GRANT, W. & GREEN, S. 2010. Combined chiropractic interventions for low-back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD005427/frame.html.

WALKER, B., MULLER, R. & GRANT, W. 2004. Low back pain in Australian adults. health provider utilization and care seeking. *J Manipulative Physiol Ther,* 27**,** 327-35.

WAND, B. & O'CONNELL, N. 2008. Chronic non-specific low back pain - sub-groups or a single mechanism? *BMC Musculoskelet Disord,* 9**,** 11.

WAND, B. M., PARKITNY, L., O'CONNELL, N. E., LUOMAJOKI, H., MCAULEY, J. H., THACKER, M. & MOSELEY, G. L. 2010. Cortical changes in chronic low back pain: Current state of the art and implications for clinical practice. *Man Ther*.

WEIR, J. P. 2005. Quantifying test-retest reliability using the intraclass correlation coefficient and the SEM. *Journal of strength and conditioning research / National Strength & Conditioning Association,* 19**,** 231-240.

WHITE, L. J. & THOMAS, S. T. 2002. The rater reliability of assessments of symptom provocation in patients with low back pain. *Journal of Back and Musculoskeletal Rehabilitation,* 16**,** 83-90.

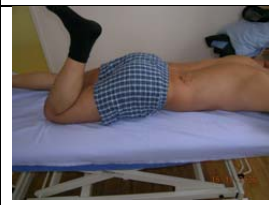
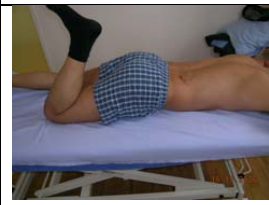
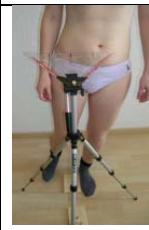
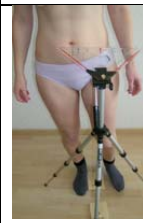
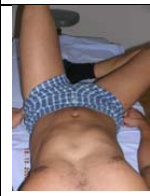
WILLIAMS, M., SOLOMONOW, M., ZHOU, B. H., BARATTA, R. V. & HARRIS, M. 2000. Multifidus spasms elicited by prolonged lumbar flexion. *Spine (Phila Pa 1976),* 25**,** 2916-24.

YOUSEFI-NOORAIE, R., SCHONSTEIN, E., HEIDARI, K., RASHIDIAN, A., PENNICK, V., AKBARI-KAMRANI, M., IRANI, S., SHAKIBA, B., MORTAZ HEJRI, S., JONAIDI, A.-R. & MORTAZ-HEDJRI, S. 2008. Low level laser therapy for nonspecific low-back pain. *Cochrane Database of Systematic Reviews* [Online]. Available: http://www.mrw.interscience.wiley.com/cochrane/clsysrev/articles/CD005107/frame.html.

YOUSSEF, J., DAVIDSON, B., ZHOU, B. H., LU, Y., PATEL, V. & SOLOMONOW, M. 2008. Neuromuscular neutral zones response to static lumbar flexion: muscular stability compensator. *Clin Biomech (Bristol, Avon),* 23**,** 870-80.

Appendix 1. Results of the test reliability between the raters and within raters.

Kappa values for the average rating between two tester pairs and between 2 different intra-tester persons

	Intra-tester	Inter-tester		Intra-tester	Inter-tester
 **Pelvic tilt**	**0.80**	**0.65**	 **Prone knee bend extension**	**0.70**	**0.47**
 **Waiters bow**	**0.88**	**0.62**	 **Prone knee bend rotation**	**0.78**	**0.58**
 **Sitting knee extension**	**0.95**	**0.72**	 **One leg stance left**	**0.84**	**0.65**
 **Rocking backw.**	**0.72**	**0.57**	 **One leg stance right**	**0.67**	**0.43**
 **Rocking forw.**	**0.51**	**0.68**	 **Rotation supine**	**0.86**	**0.38**

Appendix 2.

Exercise program for movement control impairment of the low back

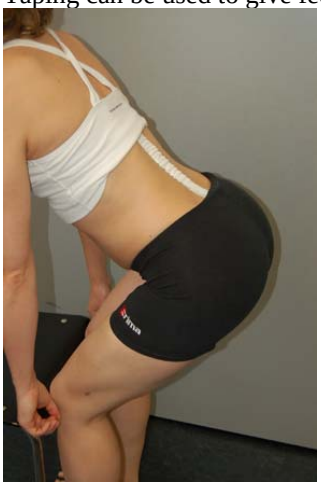
Basic progression and global remarks

- **The exercises are impairment direction specific**
- **First priority is to learn to control the movement: to keep the lumbar spine in neutral whilst moving the neighbouring area (eg. the hip)**
- **Then exercises with loading can be implemented: spine kept in neutral whilst moving and using weights**
- **After the movement control has been regained in the neutral spine position, stretching / lengthening can be started, of the muscles which are too short specifically in this impairment direction**
- **Once a good control of the back is reached, normal sports and strength training can be started**

1. Movement control impairment in Flexion

1.a. Movement control exercises (Flexion)

Taping can be used to give feedback of the correct, neutral spine position



"Waiters bow": " Bend forwards, don't let your spine move, move only in the hips"

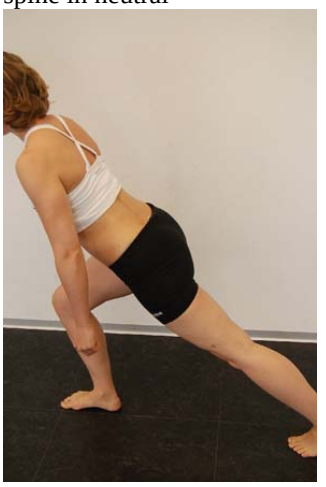
Squatting: you can put a chair against your knees so that the knees don't slide forwards. You have to push your pelvis backwards. "Keep your back in neutral, don't let it bend"

Appendix 2. Exercises for movement control impairment of the lumbar spine

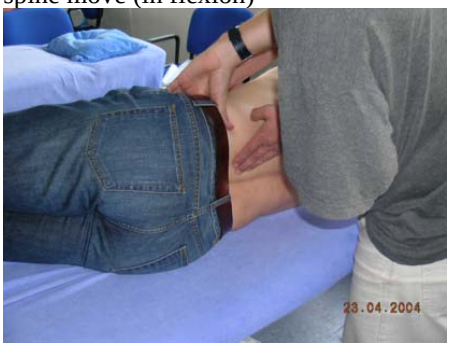
"Bend forwards, keep your spine in neutral"

"Move your pelvis backwards, keep your spine in neutral"

Sit straight. Extend your knee. Don't let your spine move (in flexion)



Launch forwards, don't let your spine move



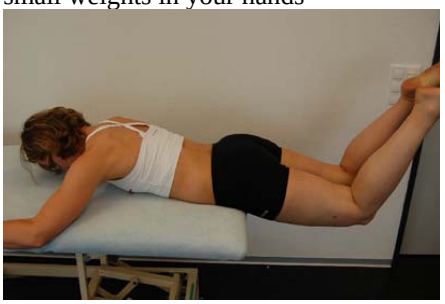
Sidelying: try to find the neutral lordosis

All fours: try to find the neutral lordosis

1. b. Strenghtening exercises (for flexion impairment)

Squatting with spine in neutral, start with small weights in your hands

Squatting with weights in your neck



Prone, lift your legs to strengthen your erector spinae muscles

1. c. Streching / lengthening exercises

Try to actively extend your knee to strech your hamstring muscles

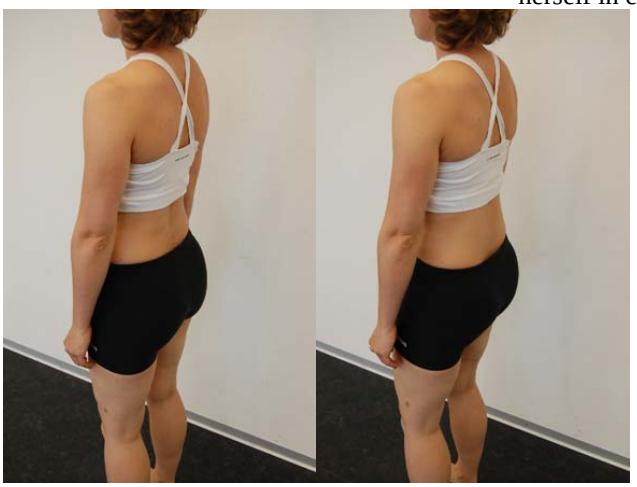
Pull your hip to maximal flexion adduction to strech your gluteus muscles

2. Exercises for extension control impairment

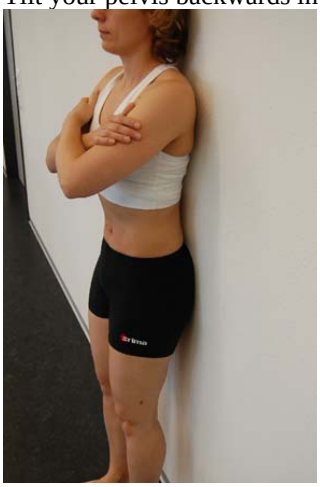
2.a. Movement control exercises

Tilt your pelvis backwards

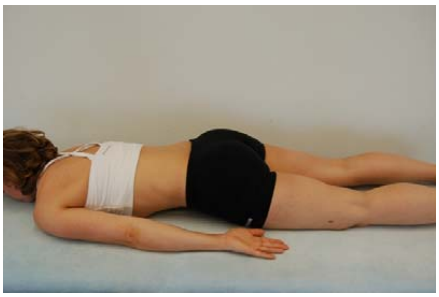
Relax in flexion position and breath deeply with your diagraph (in “active Extension” problem: if the patient is actively keeping herself in extended position)



Tilt your pelvis backwards in standing position

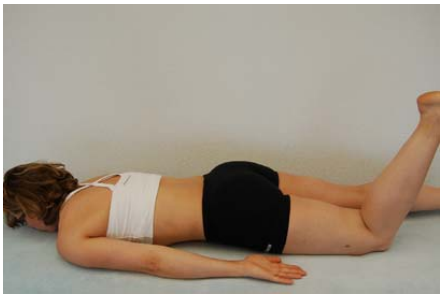


Pelvis tilt against the wall

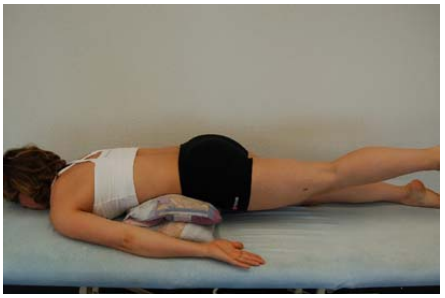


Pelvic tilt prone lying

Appendix 2. Exercises for movement control impairment of the lumbar spine

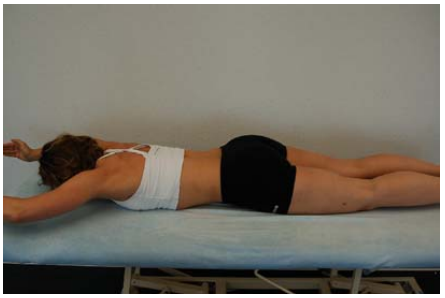


Bend your knee and keep your spine in neutral. Don’t let your spine extend.



Lift your leg whilst keeping your spine in neutral. Don’t let your spine extend (advanced)

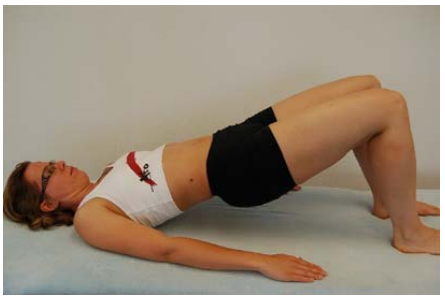
Tilt your pelvis backwards. Extend your thoracic spine. Don’t let your lumbar spine extend.



Tilt your pelvis backwards, extend your arms forwards. Don’t let your lumbar spine extend (advanced)

2.b. Strenghtening exercises

Lower abdominals. Keep your spine in neutral 20 repetitions and upwards



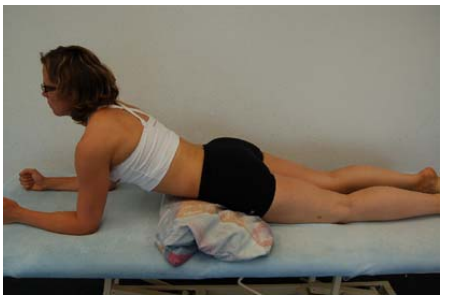
Gluteus muscles. 20 repetitions and upwards

Appendix 2. Exercises for movement control impairment of the lumbar spine

Iliopsoas muscle (keep the position 10 x 10 seconds)

2.c. Streching / lengthening exercises

Rectus femoris muscle



Upper abdominal muscles

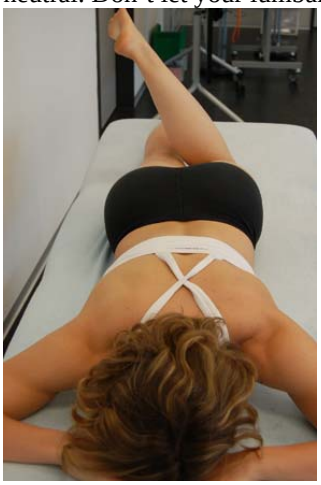
Rectus femoris sidelying strech

3. Rotation / lat.fx control impairment

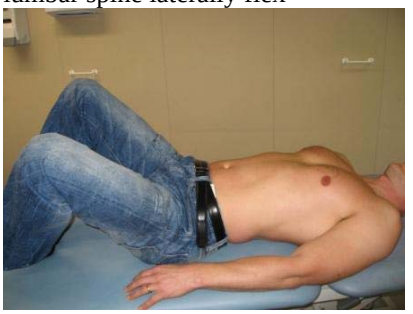
3.a. Movement control exercises

Stand on one leg stance. Keep your pelvis neutral. Don't let your lumbar spine rotate

Side bend you thoracic spine. Don't let your lumbar spine laterally flex



Move one leg to rotation whilst keeping your lumbar spine and pelvis in neutral. Don't let your lumbar spine rotate

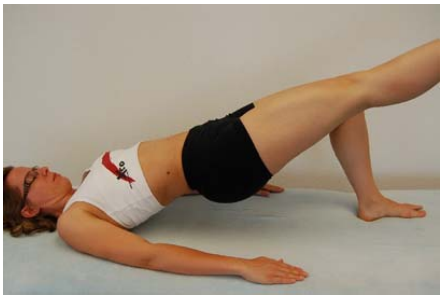


Rotate your leg whilst keeping your back in neutral. Don't let your spine or pelvis rotate

Lift your leg in abduction whilst keeping your spine in neutral. Don't' let your spine rotate

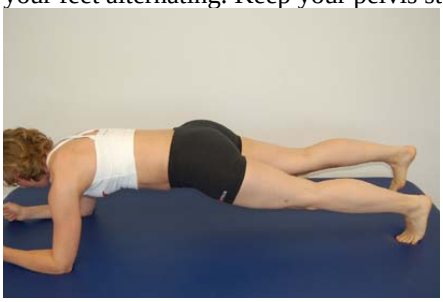
3.b. Strenghtening exercises

Gluteus medius muscle. Keep the position 10 x 10 sec or do 20 repetitions and upwards



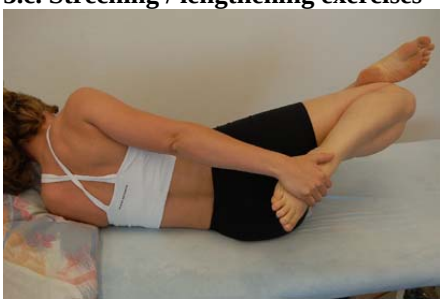
Lift your pelvis up, stay up whilst lifting your feet alternating. Keep your pelvis steady

Quadratus lumborum and oblique abdominal muscle strenghtening



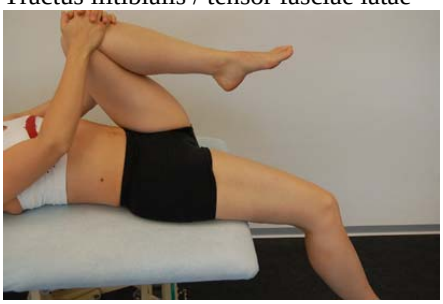
Lift your legs alternating. Strenghtening abdominal muscles

3.c. Streching / lengthening exercises



Tractus ilitibialis / tensor fasciae latae

Piriformis muscle strech



Iliopsoas / rectus femoris / tractus ilitibialis strech

Hannu Luomajoki

Movement Control Impairment as a Sub-group of Non-specific Low Back pain

Evaluation of Movement Control Test Battery as a Practical Tool in the Diagnosis of Movement Control Impairment and Treatment of this Dysfunction

The majority of patients with Low Back Pain (LBP) have non-specific pain (NSLBP). The identification of different sub-groups of patients with NSLBP has high priority in improving assessment and developing tailored, more efficient treatments. A movement control test battery was developed to evaluate movement control ability, which might be a clear sub-group of NSLBP. This doctoral thesis evaluates the test battery in the diagnosis of MCI and in the treatment of this dysfunction. The developed test set provides physiotherapists and medical doctors with an easy and reliable tool for examining whether a patient has normal or disturbed movement control and specific, targeted individual exercises are presented.

UNIVERSITY OF EASTERN FINLAND

Publications of the University of Eastern Finland
Dissertations in Health Sciences

ISBN 978-952-61-0191-0