

EGE JOHANSON

Back extensor muscle fatigability and postural
control in people with low back pain



TARTU UNIVERSITY PRESS

Institute of Exercise Biology and Physiotherapy, University of Tartu, Tartu, Estonia

Dissertation is accepted for the commencement of the Degree of Doctor of Philosophy in Exercise and Sport Sciences on 16 May 2011 by the Council of the Faculty of Exercise and Sport Sciences, University of Tartu, Tartu, Estonia

Supervisors: Mati Pääsuke, PhD, Professor, University of Tartu, Estonia
Simon Brumagne, PhD, Associate professor, University of Leuven, Belgium

Opponent: Professor Alvis Paeglitis, Dr. biol., Latvian Academy of Sport Education, Latvia

Commencement: Room 203 at Jakobi 5 Street, Tartu on June 28, 2011 at 14.15. p.m.

Publication of this thesis is granted by the Institute of Exercise Biology and Physiotherapy, University of Tartu and by the Doctoral School of Behavioral, Social and Health Sciences created under the auspices of European Union Social Fund.



European Union
European Social Fund



Investing in your future

ISSN 1406–1058
ISBN 978–9949–19–697–5 (trükis)
ISBN 978–9949–19–698–2 (PDF)

Autoriõigus Ege Johanson, 2011

Tartu Ülikooli Kirjastus
www.tyk.ee
Tellimus nr 339

To my little daughter Elise

CONTENTS

LIST OF ORIGINAL PUBLICATIONS	8
ABBREVIATIONS.....	9
1. INTRODUCTION.....	10
2. REVIEW OF LITERATURE.....	12
2.1. Functional anatomy of the lumbar spine	12
2.2. Definition, epidemiological and etiological aspects of low back pain	13
2.3. Lumbar muscle dysfunction in low back pain	15
2.3.1. Back muscle weakness.....	15
2.3.2. Back muscle fatigability.....	17
2.4. Postural control and low back pain	19
3. AIMS OF THE DOCTORAL PROJECT.....	21
4. MATERIALS AND METHODS	22
4.1. Subjects	22
4.2. Study design.....	23
4.3. Methods.....	24
4.3.1. Sørensen back endurance test.....	24
4.3.2. Electromyography.....	25
4.3.3. Muscle vibration	26
4.3.4. Postural sway analysis.....	28
4.3.5. Dynamometry.....	28
4.4. Data reduction and statistical analysis.....	28
5. RESULTS	30
5.1. Low back muscle fatigue during Sørensen endurance test in people with and without chronic low back pain	30
5.2. Low back muscle strength in females with chronic low back pain ..	36
5.3. Postural stability and proprioceptive control strategies in people with recurrent low back pain and healthy subjects.....	36
6. DISCUSSION	40
6.1. Low back muscle fatigue during Sørensen endurance test in people with chronic low back pain: relationship between electromyography power spectrum changes, back muscle strength and antropometric characteristics	40
6.2. Proprioceptive postural control in people with recurrent low back pain: The effect of back muscle fatigue on postural stability and postural control strategies.....	44
CONCLUSIONS.....	48
REFERENCES.....	49
SUMMARY IN ESTONIAN	61
ACKNOWLEDGEMENTS	63
PUBLICATIONS	65
CURRICULUM VITAE.....	103
ELULOOKIRJELDUS	104

LIST OF ORIGINAL PUBLICATIONS

The thesis is based on the following original papers, which are referred to in the text by their Roman numerals:

- I. **Pääsuke M, Johanson E, Proosa M, Ereline J, Gapeyeva H.** Back extensor muscle fatigability in chronic low back pain patients and controls: Relationship between electromyogram power spectrum changes and body mass index. *Journal of Back and Musculoskeletal Rehabilitation*, 2002, 16: 17–24.
- II. **Süüden E*, Ereline J, Gapeyeva H, Pääsuke M.** Low back muscle fatigue during Sørensen endurance test in patients with chronic low back pain: relationship between electromyographic spectral compression and anthropometric characteristics. *Electromyography and Clinical Neurophysiology*, 2008, 48: 185–192.
- III. **Johanson E, Brumagne S, Janssens L, Pijnenburg M, Claeys K, Pääsuke M.** The effect of acute back muscle fatigue on postural control strategy in people with and without recurrent low back pain. *European Spine Journal* (2011 In Press).
- IV. **Johanson E, Ereline J, Gapeyeva H, Pääsuke M.** Back extensor muscle strength and fatigability in female patients with idiopathic chronic low back pain. In: Battistella L.R, Imamura M. (Eds) *3rd World Congress of Physical and Rehabilitation Medicine, ISPRM, Bologna: Medimond, 2005, 419–422.*

*–2006–2009-surname was Süüden

The contribution of the dissertant to the compiling of the doctoral thesis:

Paper I. The dissertant conducted the experimental part of the studies, had responsibility for collecting and analysing data, as well as outcome assessment and participated in the writing of the paper (the chapters of results and discussion have been written by the dissertant independently).

Paper II, III and IV. The dissertant had primary responsibility for protocol development, subjects screening, performing measurements, preliminary and final data analyses, and writing of the manuscripts.

ABBREVIATIONS

absMF	–	Absolute value of mean centre of pressure displacement during multifidus muscle vibration
absTS	–	Absolute value of mean centre of pressure displacement during triceps surae muscle vibration
BM	–	Body mass
BMI	–	Body mass index
CNS	–	Central nervous system
COP	–	Centre of pressure
EMG	–	Electromyography
F_z	–	Vertical ground reaction force
IL	–	Iliocostalis lumborum muscle
LBP	–	Low back pain
MF	–	Multifidus muscle vibration
MPF	–	Mean power frequency
MRI	–	Magnetic resonance imaging
MVC	–	Maximal voluntary contraction
M_x	–	Moment of force (torque) around the frontal axis
M_y	–	Moment of force (torque) around the saggital axis
NRS	–	Numerical rating scale
ODI	–	Oswestry Disability Index
RMS	–	Root mean square
RW	–	Relative weighting
SD	–	Standard deviation
T_{endur}	–	Endurance time
TS	–	Triceps surae muscle

I. INTRODUCTION

Approximately 80% of the world's population will develop low back pain (LBP) at some point in their adult life (Andersson, 1997) and it is one of the most common types of musculoskeletal pain (Frymoyer and Cats-Baril, 1991, van Tulder et al., 2000). Furthermore, 34% of people who experience acute LBP will have recurrent episodes (Wasiak et al., 2006). Recurrence of back pain did not have a more specific medical diagnosis than non-specific medical LBP for more than 6 months and had at least three self-reported recurrent episodes of LBP (most liberal definition of LBP recurrence) (Marras et al., 2007). Only about 10% of cases have a specific and identified but most cases are still unclear (Bouter et al., 1998, Krismer and van Tulder, 2007). Hence, it is difficult to find clinical guidelines on how best to manage LBP the best and why some people experience recurrences and others do not.

It has been suggested that LBP is associated with several structural and functional abnormalities in neuromuscular system such as atrophy of low back muscles or alteration of muscle fibres (Mannion, 1999) and altered muscle coordination patterns (Van Dieen et al., 2003). These alterations can lead to decreased back extensor muscle strength and endurance (Nicolaisen and Jørgensen, 1985, Hultman et al., 1993).

The assessment of low back muscle fatigue has been of interest for investigators over many years because of its tight association with LBP. Numerous studies have identified an association between patients with LBP and easily fatigued low back muscles, either based on subjective (endurance time) (Biering- Sørensen, 1984, Hultman et al., 1993, Alaranta et al., 1995) or objective (electromyographic spectral analysis) (Kankaanpää et al., 1998, Elfving et al., 2003, Da Silva et al., 2005) assessment methods on muscle fatigue. Surface electromyography (EMG) is a noninvasive technique for assessing muscle function that has played a major roll in basic understanding of the low back muscle fatigue in both people with and without LBP during specific postures and movements. The erector spinae muscle group is important for maintaining upright posture of the trunk (Bogduk, 1991, Bogduk et al., 1992). Therefore, the evaluation of erector spinae muscle function may improve rehabilitation strategies and the effectiveness of specific exercise interventions for patients with LBP.

Although early studies of trunk muscle function focused on strength and endurance of the trunk muscles in people with LBP (Thorstensson and Arvidson, 1982, Suzuki and Endo, 1983), more recently the focus has shifted to issues of motor control. Growing number of studies report changes in motor control of the trunk muscles in people with LBP (Hodges and Richardson, 1996, Sihvonen et al., 1997, Radebold et al., 2000). Postural control can partly be viewed as a dynamic feedback control system (Gresty, 1987, Johansson and Magnusson, 1991). To maintain optimal postural control in daily activities the central nervous system (CNS) must identify and selectively focus on the sensory inputs (visual, vestibular, proprioceptive) that are providing the functionally most

reliable signals (Carver et al., 2006). For feedback control a stimulus is needed which has its primary effect on sensory input and is well-defined in time (Söderström and Stoica, 1989, Johansson, 1993). Because of that muscle vibration is used as a powerful stimulus of muscle spindles which can evoke illusory sensations of joint displacement (Goodwin et al., 1972, Roll and Vedel, 1982, Cordo et al., 2005). People with LBP have been observed to have altered lumbosacral proprioceptive acuity (Newcomer et al. 2000, Brumagne et al., 2000), dysfunction in trunk muscle control (Hides et al., 1996, Hodges and Richardson, 1996) and altered postural balance (Mientjes and Frank, 1999, Henry et al., 2006). Some studies showed that lumbar extensor muscle fatigue resulted in an increased postural sway in healthy individuals (Davidson et al., 2004, Vuillerme et al., 2007). However, the possible relationship between back muscle fatigue and selection of a proprioceptive postural control in people with and without LBP is poorly understood.

The main goal of present study was to evaluate back extensor muscle fatigability and the effect of acute back muscle fatigue on postural control in people with and without LBP.

2. REVIEW OF LITERATURE

2.1. Functional anatomy of lumbar spine

A thorough knowledge of the anatomy of lumbar spine is needed to aid in understanding of the mechanisms that cause LBP and to provide rationale of management. Complex central and peripheral elements control the bio-mechanics of the lumbar spine and ensure the optimal spinal loading in normal everyday life situations (Adams and Dolan, 2005, Cifrek et al., 2009).

To interpret possible changes in motor control, local and global muscle system can be used (Bergmark, 1989, Comerford and Mottram, 2001). The so called local system consists of deep intrinsic muscles and controls intervertebral motion. The global system consists of large superficial muscles with origin on the pelvis and insertions on the thoracic cage, controls gross movements of the spine and balances external loads (Cholewicki et al., 1997).

The lumbar back muscles lie behind the vertebral transverse processes, cover the posterior elements of the lumbar spine and exert their actions on the lumbar spine. The intrinsic muscles of the back are concerned with the maintenance of posture and movements of the vertebral column and head. The main mass of the lumbar back muscles is formed by lumbar erector spinae muscles and multifidus muscles. According to Bogduk lumbar erector spinae muscle is divided into two parts: (1) longissimus thoracis: (a) longissimus thoracis pars lumborum, (b) longissimus thoracis pars thoracis and (2) iliocostalis lumborum: (a) iliocostalis lumborum pars lumborum, (b) iliocostalis lumborum pars thoracis (Bogduk, 1980). The erector spinae muscles are the chief extensor of the vertebral column. They straighten the flexed column and can bend it posteriorly (Floyd and Silver, 1955, Bogduk, 1980).

When the massive erector spinae muscles are removed, several short muscles (semispinalis, multifidus, and rotators) are visible in the groove between the transverse and spinous processes of the vertebrae. Collectively, this group of muscles is known as deep layer of intrinsic back muscles or transversospinal muscle because their fibres run from the transverse processes to the spinous processes of the vertebrae. The multifidus muscle consists of short, triangular muscular bundles that are thickest in the lumbar region. The fibres of multifidus are centred on each spinous process, fibres radiate inferiorly in a systematic order to assume a variety of attachments inferiorly (Adams et al., 2002). This arrangement allows the multifidus muscle to act on each spinous process individually and separately. By some authorities in the past the multifidus muscle has been regarded as a rotator of the lumbar spine. It has no such action. The obliquity of the fibres provides them with only a minor transverse action, the predominant action of the multifidus muscle is to pull downwards on the spinous processes (Adams et al., 2002). The mechanical role of the multifidus muscle is more on transfer of forces and to act as a mover, controlling the

lordosis (Bergmark, 1989). These muscles are supplied by the dorsal rami of the spinal nerves (Bergmark, 1989, Ebraheim et al., 2004, Cilroy et al., 2008).

Transverse and interspinal muscles as a deep segmental back muscles are small-paired muscles that connect the spinal processes between their lateral surfaces. These muscles in spite of their comparatively small muscle force but due to their short length, give an increased stiffness and extrinsic mechanical stability to the spine (Bogduk, 1997, Bergmark, 1989).

The quadratus lumborum muscle is a wide rectangular shaped muscle that arises from the 12th rib and lumbar transverse processes. Caudally it attaches to the top margin of the ilium. The main function of the muscle is the control (stabilization) of the pelvis and produce the lateral flexion of the trunk. Furthermore, it has also been claimed to take a part in respiration (Bogduk, 1980, Bergmark, 1989). The lateral muscles of lumbar spine are innervated by the ventral rami of the spinal nerves (Ebraheim et al., 2004).

The gross anatomy of the lumbar muscles has been (re)investigated in great detail over the last 10–30 years, and the most salient findings are reported in a series of excellent manuscripts (Bogduk, 1980, Bogduk and Macintosh, 1984, Macintosh and Bogduk, 1987; 1991, Bergmark, 1989, Bogduk et al., 1992, Ebraheim et al., 2004).

2.2. Definition, epidemiological and etiological aspects of low back pain

LBP is defined as pain and discomfort localised between the 12th rib and the inferior gluteal folds, with or without leg pain. Most cases are non-specific, but in 5–10% of cases a specific cause is identified. Non-specific (common) LBP is defined as back pain with no known underlying pathology (e.g. infection, tumour, osteoporosis, ankylosing spondylitis, fracture, inflammatory process, radicular syndrome or cauda equina syndrome) (Krismer and Tulder, 2007). Back pain may be classified based on its duration. Acute LBP occurs suddenly after a period of minimum of 6 months without LBP and lasts for less than 6 weeks. Subacute LBP is low back pain persisting between 6 weeks and 3 months, chronic LBP as low back pain persisting for 3 months or more. Mechanical LBP is defined as pain secondary to trauma or deformity of an anatomic structures. Recurrent LBP is defined as a new episode after a symptom-free period, not an exacerbation of persistent LBP (van den Hoogen et al., 1998).

LBP is a common condition affecting a large percentage of the population. It is estimated that between 70–85% of the population in industrialised countries (one-year prevalence 15% to 45%, adult incidence 5% per year) will experience LBP at some point in their lives (Kelsey and White, 1980, Biering-Sørensen, 1983, Waddell, 1987). LBP is not only limited to adults, but it occurs widely even during childhood and adolescence (Balague et al., 1999) peaking ages

between 35 and 55 years (Andersson, 1997). Pain cannot be attributed to pathology or neurological encroachment in about 85% of people. A role of genetic influence on liability to back pain is suggested from recent research (Hestbaek et al., 2004, MacGregor et al., 2004).

Acute LBP is usually considered to be self-limiting (recovery rate 90% within 6 weeks) but 2–7% of people develop chronic pain. Around two-thirds of people are likely to experience relapses of pain over 1 year and around a third are likely to have relapses of work absence (Hestbaek et al., 2003). Recurrent and chronic back pain is widely acknowledged to account for a substantial proportion of total worker's absenteeism (Nachemson et al., 2000).

The dysfunction of the lumbar spine including impaired back muscle endurance, motor and postural control has a pivotal role in etiology of LBP. In many cases, the pathology or the organic basis behind LBP remains unclear (Bouter et al., 1998, Turk and Okifuji, 1999) and the origin of LBP has been explained in several ways. The most powerful risk factors for a new episode of back pain are altered muscle control (Hides et al., 1996, Hodges, 2001, Moseley et al., 2002), altered postural control (Brumagne et al., 2004, 2008) and increased back muscle endurance (Biering-Sørensen 1984, Kankaanpää et al., 1997). Beyond that, the most frequently reported environmental risk factors are heavy physical work and frequent lifting, stooping, postural stress and vibration (Andersson, 1997). Psychosocial risk factors include stress, distress, anxiety, depression, cognitive functioning and pain behaviour job dissatisfaction and mental stress at work (Andersson, 1997, Hoogendoorn et al., 2000, Linton, 2000). Pain may arise from the degeneration of intervertebral discs (Frymoer et al., 1984, Mooney, 1989) or from abnormalities in other tissues, such as facet joints, vertebral bodies, ligaments, muscles (Cavanaugh, 1995, Siddall and Cousins, 1997). The changes in different structures of the spine may produce spinal instability, which may cause the sensation of pain (Panjabi, 1992). Pain may be associated with compression load, damage to the nerves (Mooney, 1989, Siddall and Cousins, 1997) or abnormal chemical events that occur with tissue damage, as well as the release of chemical mediators (Mooney, 1989, Cavanaugh, 1995). Psychological factors may also produce pain and the pain may then be of psychogenic origin (Viikari-Juntura et al. 1991, Liebenson, 1992).

In brief a source of LBP can arise from any part of the lumbosacral region that is innervated, such as vertebral periosteum, intervertebral discs, back extensor muscles, tendons, ligaments, vessels, zygapophyseal joints and sacroiliac joints or it can arise from the visceral organs (Schwarzer et al., 1995, Bogduk, 1997, Coppes et al., 1997, Freemont et al., 1997).

2.3. Lumbar muscle dysfunction in low back pain

LBP has been shown to be associated with histomorphological and structural changes in the paraspinal muscles, i.e. the back muscles are smaller, contain more fat, show a degree of selective muscle fiber atrophy (Verbunt et al., 2003) and their blood circulation may be restricted because calcific deposits in the abdominal aorta and vertebral arteries (Kauppila et al., 1997, 2004). In consequence, the lumbar paraspinal muscles are weaker (Häkkinen et al., 2003) and exhibit excessive fatigability (Mannion et al., 1997, Greenough et al., 1998, Humphrey et al., 2005). Also poor co-ordination of paraspinal muscles has been related with chronic LBP and with excess lumbar muscle fatigability (Wilder et al., 1996, Taimela et al., 1999, Leinonen, 2003). These changes are widely thought to be a result of disuse and deconditioning, secondary to pain and illness, a process called the deconditioning syndrom (Nachemson and Lindh, 1969, Thorstensson and Arvidson, 1982). Behavioural avoidance can cause decrease in physical activity, which can result in reduced lumbar mobility and loss of muscle strength and endurance because of muscle atrophy (Biering-Sørensen, 1984, Laasonen, 1984, Airaksinen et al., 1996) i.e. physical deconditioning. As apart of the deconditioning syndrome, reduced endurance capacity of paraspinal muscles has been related to chronic LBP (Suzuki and Endo, 1983, Biering-Sørensen, 1984, Hultman et al., 1993). Its important to prevent recurrence of LBP, because alterations in motor control, proprioceptive acuity may lead to weakness and fatigability and these two back muscle impairments recognized as a potential cause of the recurrent LBP (Mannion, 1999). Therefore, the evaluation of the back extensor muscle strength and fatigability, motor control has important applications in assessment of people with LBP during rehabilitation.

2.3.1. Back muscle weakness

Chronic LBP is associated with several anatomical or structural abnormalities such as atrophy of back muscle mass or alteration of muscle fibres characteristics (Ng et al., 1998, Mannion, 1999), several impairments in structure (Mattila et al., 1986, Hultman et al., 1993, Sihvonen et al., 1993, Venna et al. 1994) and limitations in functions such as in muscle strength (Biering-Sørensen, 1984, Mayer et al., 1985) and endurance (Roy et al., 1989, Kankaanpää et al., 1998b, Latimer et al., 1999, Mannion, 1999). There are many studies where have been reported the impairments in structure like atrophy of type II (fast) motor units in the trunk musculature (Mattila et al., 1986, Rissanen et al., 1995), changes in type I (slow) motor units (Mattila et al., 1986), sensory and neurological deficits (Venna et al., 1994) or abnormal EMG activity (Sihvonen et al., 1993) and impairments in muscle function such as decreased muscle strength (Hultman et al., 1993, Kankaanpää, 1999) and endurance (Nicolaisen and Jørgensen, 1985, Hultman et al., 1993, Kankaanpää, 1999). The chronic pain

situation itself may cause alterations of back muscles which might lead to weakness and fatigability and these two back muscle impairments are recognized as a potential cause of the recurrent nature of LBP (Panjabi, 1992).

To maintain stability of the lumbar spine antagonistic flexor and extensor muscles must be simultaneously active (Cholewicki et al, 1997). Decreased back extensor muscle strength has often been associated with LBP (Biering-Sørensen, 1984, Hultman et al., 1993). There seems to be an agreement that people with LBP especially ones with chronic problems have weaker back extensor muscles than healthy persons (Nicolaisen and Jørgensen, 1985, Klein et al., 1991). Not only are the back muscles weaker but there are also modifications in extensor/flexor muscle strength ratio (Mayer et al., 1985, Shirado et al., 1995). People with LBP have a significant loss of both flexor and extensor muscle strength, but the main loss of strength is found in the back extensors (Mayer et al., 1985, Hultman et al., 1993, Shirado et al., 1995, Kankaanpää, 1999). Chronicity and severity of LBP may be supplementary factors for the reduction in back extensor muscle strength. Numerous investigations have shown that people with chronic LBP are weaker than the subjects with acute or even intermittent LBP (Hultman et al., 1993, Kankaanpää, 1999). The decrease in back muscle performance (strength and endurance) following a first episode of LBP, the so-called „deconditioning syndrome” is proposed as a potential cause of recurring LBP (Mannion, 1999, Verbunt et al., 2003). In people with more frequent LBP, magnetic resonance imaging (MRI) studies showed a slightly smaller cross-sectional area of the paraspinal muscles and greater signal intensities, possibly due to muscle atrophy, which may be one of the causing factors of back muscle dysfunction (Cooper et al., 1992, Hultman et al., 1993, McGregor et al., 1999). However, the association between paraspinal muscle cross-sectional area and back function related factors, such as disability and back pain, have been controversial (Mannion et al., 2000, Käser et al., 2001).

Isometric, isokinetic and isoinertial trunk strength measurements are common methods in the assesment of trunk muscle performance (Mayer et al., 1989a, Mayer et al., 1995, Hupli et al, 1997, Rätty et al., 1999). The strength measurements impose heavy loads on the lumbar spine structures and may predispose the patients to injury and further aggravating pain (Kankaanpää, 1999). Therefore the back extensor muscle strength measurements pose an ethical consideration in people with LBP. The results of these tests are influenced greatly by motivation and current level of pain (Newton and Waddell, 1993, Moony and Andersson, 1994). It is not clear in every LBP case whether decreased back extensor strength is caused by current pain or fear of pain, or whether it is due to actual disturbances in nerve or muscle functions. These reasons severely limit the use of back extensor muscle strength measurement in clinical practice.

2.3.2. Back muscle fatigability

Neuromuscular fatigue is generally defined as the failure to maintain the required or expected force (De Luca, 1984, Gandevia, 2001). It may arise not only because of peripheral changes at the level of the muscle, but also because the central nervous system (CNS) fails to drive the motoneurons adequately (Bigland-Ritchie et al., 1978, Gandevia, 2001). In physically demanding occupations, back muscle fatigue is easily developed during repetitive lifting, bending and twisting maneuvers, which have been shown to be occupational risk factors for LBP (Frymoyer et al., 1983). The assessment of low back muscle fatigue has been of interest for investigators over many years because of its tight association with LBP. Numerous studies have identified an association between people with LBP and easily fatigued low back muscles, either based on subjective (endurance time, Borg scale) (Biering-Sørensen, 1984, Borg, 1990, Hultman et al., 1993, Alaranta et al., 1995, Kankaanpää et al., 1997) or objective (electromyographic spectral analysis) (Klein et al., 1991, Mannion et al., 1997, Kankaanpää et al., 1998a, Pääsuke et al., 2002, Elfing et al., 2003, Da Silva et al., 2005) assessment methods of muscle fatigue.

Various test positions have been used in studies of isometric back muscle fatigue. Most common is the Sørensen test, i.e. prone unsupported trunk in horizontal position (Mannion et al., 1997, Ng et al., 1997, Kankaanpää et al., 1998a, Koumantakis et al., 2001), which is about 40–50% of maximal voluntary contraction (MVC) force (Mannion and Dolan, 1994). The change in parameters of the EMG spectrum obtained during this test has been shown to be a better predictor of first-time LBP acquisition than the simple measure of endurance time (Mannion et al., 1997). On the other hand, Adams et al. (1999) reported that the median frequency (MF) parameters were not significant predictors of first-time LBP, although endurance time during the Biering-Sørensen was significant at some of their follow-ups. Several studies however have shown that people with chronic LBP often suffer from excessively fatigable back extensors muscles (Biering-Sørensen, 1984, Jørgensen and Nicolaisen, 1987, Mayer et al., 1989b, Roy et al., 1989, Tsuboi et al., 1994).

Surface EMG is a noninvasive technique for assessing muscle function that has played a major role in basic understanding of low back muscle fatigue in both normal subjects and in people with LBP. It has been suggested that muscle fatigue is present as soon as muscle contraction starts (Bigland-Ritchie et al., 1981) and can be measured by a shift of the EMG power spectrum to lower frequencies (spectral compression) caused by neural and metabolic factors in the muscle (Lindström et al., 1970). The fatigue-induced EMG spectral compression has been related to the action potential conduction velocity propagation. This is most likely due to an accumulation of metabolites (e.g. H^+ and extracellular K^+) (Bigland-Ritchie et al., 1981, Tesch et al., 1983) reducing intracellular pH (Brody et al., 1991) and, thus, decreasing sarcolemma excitability. Although the exact mechanisms underlying the EMG spectral compression are not fully understood, the resultant shift to lower frequencies during sustained contraction

is recognized as an electrophysiological monitoring of fatigue process (Hägg, 1992, Mannion and Dolan, 1994, Umezu et al., 1998, Kankaanpää et al., 1998a).

A decrease in MF and the mean power frequencies (MPF) of the EMG power spectrum (the slope) is an indicator of muscle fatigue commonly used for back muscles, while its initial MF and MPF may indicate muscle fibre composition (Roy et al., 1989, Biedermann et al., 1991). Differences in EMG power spectrum parameters in people with LBP compared with those in healthy individuals have been usually shown a steeper slope (Roy et al., 1989, Mayer et al., 1995). However, conversely, a less steep slope for people with LBP has also been reported (Peach and McGill, 1998). Studies in people with LBP have shown difference in MPF and MF slope between the right and left side of bilateral recordings of an isometric contraction of back extensor muscles (Roy et al., 1995, Oddsson et al., 1997), but for healthy subjects difference between both sides (Tsuboi et al., 1994) and no difference (Oddsson et al., 1991, Mannion et al., 1997) have been shown.

Physical characteristics (age and BMI) have a significant influence on lumbar paraspinal muscle fatigability in the isometric Sørensen test (Kankaanpää et al., 1998a). Despite the wide-spread use of EMG power spectrum parameters to monitor of the back extensor muscle fatigue, its relationship with subjects BMI, has received only little attention (Kankaanpää et al., 1998a), however a few studies have reported that increased BMI is positively associated with CLBP (Orvieto et al., 1994, de Leboeuf-Yde et al., 1999, Bayramoglu et al., 2001, Pääsuke et al., 2002). The relationship between the changes in EMG power spectrum during fatiguing sustained isometric contractions of the back extensor muscles and BMI in people with chronic LBP is poorly understood.

Concerning gender, several studies have reported that women performed the back endurance test longer and showed less progressive decreases in spectral indices (MF slope) than did the men (Mannion and Dolan, 1994, Mayer et al., 1995, Kankaanpää et al., 1998a). This suggests that women fatigue more slowly than men in the Sørensen back endurance test (Biering-Sørensen, 1984, Oddsson et al., 1991). The gender differences in lumbar muscle fatigability during the Sørensen test can most likely be explained by the differences in muscle anatomic and functional characteristics or it can partially by the higher weight of the torso/upper limbs of men compared to women and therefore back muscle activity at a higher percentage of maximal voluntary contraction. It has been shown that back muscles in women have a greater relative cross-sectional area of fatigue-resistant type I fibres (women 73% vs men 56%), and as much as a twofold higher type I type II fibre area ratio than back muscles in men (Thorstensson and Calson, 1987). In addition, men have a 17% larger total erector spinae cross-sectional area than women (Parkkola et al., 1993).

Despite the wide-spread use of Sørensen back endurance test to monitor the lumbar back muscle fatigue, the relationship between the EMG power spectrum compression during sustained isometric contraction and anthropometric charac-

teristics in people with chronic LBP and healthy individuals and its gender differences are not well understood.

2.4. Postural control and low back pain

Optimal postural control is essential to perform daily activities. Postural control involves two main functions: postural orientation and postural balance (Massion, 1994). The ability to control of body balance during standing is dependent on the activity of central nervous system (CNS) (Winter et al., 1998). The CNS regulates the body stability while standing or during locomotion mainly by means of afferent signals from the visual system (Merger et al., 2005), proprioceptors (Bove et al., 2003, Tresch, 2007) and changes in vestibular input (Bacsi and Colebatch, 2005). These signals, which allow us to assess the position and motion of the body in space, are constantly reweighted so as to generate the appropriate forces to control and maintain balance in a wide range of situations (Massion, 1992). Alterations in postural control (Nies and Sinnott, 1991, Luoto et al., 1996, Mientjes and Frank, 1999, Mok et al., 2004, Moseley et al., 2004, Moseley and Hodges, 2005), impairments in motor control (Hodges and Richardson, 1996) and altered lumbosacral proprioceptive acuity (Brumagne et al., 2000, Newcomer et al., 2000) have been observed in people with recurrent LBP, which might be a causative factors in their postural instability. Pain also may be a confounding factor to maintain postural stability, but it is not certain whether pain causes changes in motor control or whether motor control changes lead to pain, or both (Arendt-Nielsen et al., 1996, Hodges et al., 2001, Hodges and Moseley, 2003).

There are several studies of trunk muscle function focused on the strength and endurance of the trunk muscles in people with chronic LBP (Thorstensson and Arvidson, 1982, Suzuki and Endo, 1983), but more recently the focus has shifted to issues of motor control. Muscle activity must be coordinated to maintain control of the spine and the efficacy of the muscle system is dependent on its controller, the CNS (Panjabi, 1992). Numerous studies have reported impaired balance in people with LBP when standing on one (Luoto et al., 1998) or two legs (Nies and Sinnott, 1991) and people with poor performance in a test of standing balance have an increased risk for LBP (Takala and Viikari-Juntura, 2000).

Proprioceptive input from the muscles of the legs and trunk plays an important role in maintaining postural stability (Bloem et al., 2000), suggesting that sensory deficitis from either location might result in instability. Muscle vibration is known as a powerful stimulus of muscle spindles (Roll and Vedel, 1982, Cordo et al., 2005) and can evoke illusory sensations of joint displacement, which most of the time correspond with a perceived lengthening of the vibrated muscle (Goodwin et al., 1972, Cordo et al., 2005). Vibration is a potent stimulus for muscle spindle Ia afferents (Brown et al., 1967). Muscle spindles are respon-

sible for the sense of position and movement (Gandevia, 1992). Reduced proprioception in the spine in people with chronic LBP has been established for standing posture (Brumagne et al., 2004), sitting (Brumagne et al., 2000) and four-point kneeling (Gill and Callaghan, 1998). When postural muscles are vibrated and when the CNS uses these signals for postural control, the kinaesthetic illusions will cause excessive corrective displacement of the center of mass to avoid falling. For example, during standing vibration of triceps surae muscles can give the illusion of forward leaning and therefore the subject will compensate with a backwards shift of the center of mass, even to the point of falling (Eklund, 1972, Brumagne et al., 2004). Healthy individuals normally maintain postural stability using a “multi-segmental” control strategy (Allum et al., 1998, Morasso and Schieppati, 1999, Brumagne et al., 2004). In this model postural control is seen as a more dynamic process whereby muscle activation occurs in a proximal to a distal sequence (Allum et al., 1998, Morasso and Schieppati, 1999). In contrast, people with LBP seem to use a more rigid postural strategy (i.e. ankle steered strategy) to control postural balance resulting in postural instability when postural demands increase (Mok et al., 2007, Brumagne et al., 2008).

Postural control might be negatively influenced by muscle fatigue. Muscle fatigue can be defined as a decreased force-generating capacity (Bigland-Ritchie et al., 1983) and may be caused by peripheral changes or by a failure of the CNS to drive the motoneurons adequately (Brumagne et al., 2008). It possibly influences postural control due to altered muscle contractile efficiency (Bigland-Ritchie et al., 1983, Duchateau and Hainaut, 1985), proprioceptive acuity (Allen and Proske, 2006) and cortical control (Taylor et al., 1996, Gandevia, 2001). Excessive fatigability of back extensor muscles is common among people with chronic LBP (Biering-Sørensen, 1984, Mannion et al., 1997, Latimer et al., 1999). The fatigue-related changes in muscle stiffness may reduce the capacity of the paraspinal muscles to stabilize the spine (Granata et al., 2004). Furthermore, Taimela et al. (1999) concluded that lumbar muscle fatigue impaired lumbar position sense in people with LBP and healthy subjects. Some studies showed that lumbar extensors fatigue resulted in increased postural sway in healthy individuals (Davidson et al., 2004, Vuillerme et al., 2007). However, the possible relationship between back muscle fatigue and the selection of a proprioceptive postural control strategy in healthy individuals and in people with LBP are poorly understood.

3. AIMS OF THE DOCTORAL PROJECT

The general aim of the present doctoral thesis was to evaluate back extensor muscle fatigability and postural control in people with and without low back pain.

Accordingly, the specific aims of the present investigation were:

- (1) To assess back extensor muscle fatigability during sustained submaximal isometric contraction condition and its associations with anthropometric characteristics in people with and without chronic low back pain. This specific aim is addressed in Paper I and II;
- (2) To evaluate back extensor muscle isometric strength in people with and without chronic low back pain. This specific aim is addressed in Paper IV;
- (3) To assess the effect of acute back muscle fatigue on postural control in people with and without recurrent low back pain. This specific aim is addressed in Paper III.

4. MATERIALS AND METHODS

4.1. Subjects

In total, 57 subjects with LBP and 58 subjects without LBP as controls gave informed consent and participated in this study. Eighty-three subjects (53 women and 30 men) from Tartu (Estonia) and 32 individuals (22 women and 10 men) from Leuven (Belgium) were participated. Table 1 displays the division of the subjects and then mean age and anthropometric characteristics in different studies.

Table 1. Anthropometric characteristics and age of the subjects (mean±SE).

Papers	n	Age (yrs)	Height (cm)	Body mass (kg)	BMI (kg·m⁻²)
Paper I					
Chronic LBP group *	12	47.4±4.4	169.8±2.9	74.9±4.2	25.9±1.2
Healthy controls*	12	46.7±1.4	168.8±2.4	74.3±3.5	25.3±1.4
Paper II					
<u>Women</u>					
Chronic LBP group	10	50.3±3.4	164.7±2.2	71.9±3.9	26.9±1.4
Healthy controls	10	49.6±1.3	163.2±1.1	67.2±3.9	24.6±1.4
<u>Men</u>					
Chronic LBP group	10	50.7±11.9	177.8±2.5	81.2±3.7	25.6±1.2
Healthy controls	10	49.3±6.5	177.5±1.7	80.5±3.4	25.6±1.3
Paper III					
Recurrent LBP subjects #	16	22.7±1.7	174.7±9.6	66.8±12.5	21.9±2.2
Healthy controls #	16	22.0±1.1	172.0±10.7	65.5±9.6	22.1±2.0
Paper IV					
Female chronic LBP group	9	47.3±1.7	164.3±1.6	69.9±2.6	25.9±1.8
Female healthy controls	10	45.6±1.2	163.9±1.3	66.7±2.7	23.6±1.7

Abbreviations: LBP– low back pain; BMI – body mass index.

* 7 women and 5 men; # 11 women and 5 men.

A medical screening by a physician was performed to include and exclude subjects. In Paper I, II, IV in the initial clinical examination at the hospital, the cause of the back pain was performed to be non-specific. People with nerve root compression or disc prolapsed, severe scoliosis, spondyloarthritis, previous back surgery, and other serious and specific causes of back pain were excluded. The chronic LBP diagnosis included the criteria that people had LBP for longer than 3 months (on the average for 6.8 ± 2.1 yrs) and they did not have radicular symptoms. Individuals were recruited through the Tartu University Hospital.

In Paper III individuals were included in the recurrent LBP group if they had experienced non-specific mechanical LBP for more than 6 months, reported at least 6/100 on the Oswestry Disability Index, version 2 (ODI) (Fairbank and Pynsent, 2000) and had at least three self-reported recurrent episodes of LBP (most liberal definition of LBP recurrence) (Marras et al., 2007). None was undergoing regular medical treatment or physical therapy for their LBP at the time of testing or in the last 6 months. Study participants in Paper III were excluded if they had LBP with a non-musculoskeletal etiology, musculoskeletal injuries of the lower limbs, previous spinal surgery, history of neurological disease, specific balance or coordination problems, a history of cerebral trauma or if they were using any pain relieving medication. Subjects were recruited from the University Hospitals of Leuven, where they had sought medical attention for LBP. Individuals were included in the control group if they had no history of LBP and an ODI equal to 0/100. All procedures were approved by the institutional Medical Research Ethical Committee and were applied with respect to the Declaration of Helsinki (Ethical Principles for Medical research Involving Human Subjects).

4.2. Study design

One part of the present study was performed during the period of 2002–2005 in University of Tartu in the Laboratory of Kinesiology and Biomechanics. The second experimental part was carried out in the Department of Rehabilitation Sciences, University of Leuven (Belgium) from 2006 to 2007. Papers (I, II, IV) describes work done in Estonia, which include back extensor muscle fatigability and strength measurements. Paper III presents work done in Belgium, postural control measurements in two conditions.

In papers I and II, low back muscle fatigue was assessed during Sørensen back isometric endurance test in people with and without chronic LBP. Relationship between EMG spectral compression and anthropometric characteristics was found. Sørensen back endurance test till exhaustion was used to evaluate back muscle fatigue. Surface EMG was recorded bilaterally from the erector spinae muscle at the level of L3 to monitor the EMG power spectrum changes during fatiguing isometric contraction.

In paper III, acute back muscle fatigue was used as a mechanism to induce or sustain a suboptimal proprioceptive postural control strategy in people with and without recurrent LBP. Experimental protocol is presented in Table 2.

In paper IV, strength and fatigability characteristics of back extensor muscles were assessed in female chronic LBP patients compared to healthy subjects. MVC force of the back extensor muscles was measured by standard back dynamometer.

Table 2. Experimental postural control protocol (Paper III)

1. Control (day 1)
1.A. Upright stance – stable support surface
1.A.1. Without vision
1.A.2. Without vision, bilateral triceps surae muscle vibration
1.A.3. Without vision, bilateral lumbar multifidus muscle vibration
1.B. Upright stance – unstable support surface (foam)
1.B.1. Without vision
1.B.2. Without vision, bilateral triceps surae muscle vibration
1.B.3. Without vision, bilateral lumbar multifidus muscle vibration

2. Back muscle fatigue (day 2)
2.A. Upright stance – stable support surface
2.A.1. Without vision
2.A.2. Without vision, bilateral triceps surae muscle vibration
2.A.3. Without vision, bilateral lumbar multifidus muscle vibration
2.B. Upright stance – unstable support surface (foam)
2.B.1. Without vision
2.B.2. Without vision, bilateral triceps surae muscle vibration
2.B.3. Without vision, bilateral lumbar multifidus muscle vibration

4.3. Methods

4.3.1. Sørensen back endurance test

Back extensor muscle isometric endurance was evaluated using Sørensen test (Biering-Sørensen, 1984) (Paper I; II; IV). In Paper III, Sørensen test was used for inducing the back muscle fatigue condition. During the Sørensen test the subject lay in a prone position on a treatment bench with the lower half of the body below the level of the anterior superior iliac spines strapped to the couch at three positions: at the ankles as close to the malleoli as possible, at the knee creases, and at the level of the greater trochanter of the femur. The seat belts were tightened as firmly as possible while considering the subject's level of comfort. The subject's hands were placed at the sides of the trunk (Paper I, II, IV) or crossed over the chest (Paper III), and the chest was supported at a 45° angle downward from the horizontal plane with the head and neck in neutral position. While the subjects performed Sørensen back endurance test, they were instructed at the beginning of the test to lift the upper trunk clear of the chair and maintain unsupported upper body in the horizontal plane as long as possible (until exhaustion). The horizontal position during the test was controlled by a small sack (hanging from the ceiling), which was placed between the scapulae. The test was terminated when the subject could no longer maintain upper body

in the horizontal plane (defined as > 2 cm reduction in height for 2 s) despite strong verbal encouragement. The endurance time was recorded in seconds by using a stopwatch and was taken as indicator of back extensor muscle isometric endurance (Fig. 1).



Figure 1. Sørensen back endurance test.

4.3.2. Electromyography

In Paper I, II and IV, surface EMG was recorded bilaterally from the centre of the lumbar erector spinae muscle during Sørensen back endurance test. After the skin was shaved, abraded, and then cleaned with alcohol, pairs of bipolar surface EMG electrodes (Ag-AgCl, 8-mm diameter, 20-mm interelectrode distance) were attached bilaterally over the lumbar erector spinae muscle at the level of L3 (approximately 3 cm laterally to the center of the spinous process). As a reference electrode a large carbon rubber plate (Nemectron, Germany, 7×12.5 cm) was placed on the iliac crest. The EMG signals were amplified and displayed with Medicor MG-440 preamplifiers with the frequency band ranging 1 Hz – 1 kHz. The output signals from EMG preamplifiers were digitized on-line (sampling frequency 1 kHz) by analogue-to-digital converter installed in personal computer. The digitized signals were stored on a hard disk for further analysis. EMG power spectrum MF was calculated by using Fast Fourier Transform Algorithms (Lindström et al., 1970), where a 1024 data point window (1 s) slides over the whole recorded signal area with a 512 point shift (50% overlap). During Sørensen back isometric endurance test the MF was determined and averaged over each period of 5 s, whereas the following characteristics were calculated: initial MF as the mean of the first 5 s, end MF as the mean of the last

5 s and MF slope as the percent change from initial value ($\% \cdot \text{min}^{-1}$). MF slope was taken as indicator of the erector spinae muscle fatigability.

In paper III, surface electromyography (EMG) during Sørensen endurance test was recorded from the iliocostalis lumborum pars thoracis (IL) and MF (multifidus) muscles (Myosystem, USA). The pairs of surface-electrodes (Medicotest blue sensor, INC, USA) were placed 2 cm apart, over the muscle belly, and following the direction of the muscle fibers. To reduce cross-talk signals from adjacent muscles, the electrode positions of the IL and MF muscles were at the intersection of the line corresponding to the muscle fiber orientation and horizontal lines through the spinous process of L2 (IL) and L5 (MF), respectively. A ground electrode was placed over the right malleolus lateralis. The EMG data were amplified ($\times 1000$), band-pass filtered (10–500 Hz) and sampled at 2000 Hz using a Micro1401 data acquisition system and Spike2 software (Cambridge Electronic Design, UK).

4.3.3 Muscle vibration

In paper III, muscle vibration, as a strong stimulation for muscle spindles (Roll and Vedel, 1982, Cordo et al., 2005), was used to appraise the role of proprioception in postural control. During vibration, an illusion of muscle-lengthening alters the proprioceptive sense (Goodwin et al., 1972, Cordo et al., 2005). When postural muscles are vibrated and when the CNS uses these signals for postural control, the kinaesthetic illusions will cause excessive corrective displacement of the center of mass to avoid falling. For example during standing in healthy individual, vibration of triceps surae (TS) muscles induces an involuntary body sway in backward direction, whereas lumbar multifidus (MF) muscle vibration produces a forward body sway (Brumagne et al., 2004, Brumagne et al., 2008). Displacement of the center of pressure (COP) specifies how the subject makes use of proprioceptive signals from the vibrated muscle to control posture. Therefore, two muscle vibrators (Maxon motors, Switzerland) were used. Vibration was applied bilaterally to TS muscles or MF muscle, respectively (Fig. 2). The frequency of vibration was 60 Hz and the amplitude was approximately 0.5 mm. Trials lasted 60 s. Muscle vibration started 15 s after the start of trial and lasted for 15 s. Activation and deactivation of the vibrators were manually controlled.

The subjects stood barefoot on the force plate with the feet separated by the width of the hips, arms hanging loosely at the sides. The subjects were instructed to remain still and relaxed. Two test conditions were used to evaluate postural control: (1) control condition (Day 1) and (2) back muscle fatigue condition (Day 2). Each condition involved six trials (Table 2). All trials were performed on stable and unstable support surface. For the unstable support surface trials a “foam” (Airex balance pad) was used to decrease the reliability of ankle proprioception, so the CNS should rely on other proprioceptive signals for

postural control such as from the back muscles (Ivanenko et al., 2000, Bru-magne et al., 2008). Back muscle fatigue was used to evaluate if this may be a mechanism to induce changes in proprioceptive postural strategies. During the trials subject had to stand barefoot on a force plate (or on “foam” on the force plate) with the arms loosely hanging along the body (Fig. 2). The heels were 10 cm apart with the forefeet in a free splayed out position. Vision was occluded by non-transparent glasses and subjects were instructed to remain immobile, but relaxed. In the muscle vibration trials, data was collected for 15 s prior to the start of vibration, muscle vibration lasted for 15 s and data collection continued for 30 s after cessation of vibration.

In the back muscle fatigue condition a modified Biering-Sørensen back endurance test (Biering-Sørensen, 1984, Latimer, 1999) was performed and immediately followed by the same six postural control trials used in the first (control) condition. To ensure that the force platform measurements were obtained in a genuine fatigued state, the fatiguing test took place beside the force platform.

Participants were asked to rate their back pain on a Numerical Rating Scale (NRS) (0–10) and their perceived effort on an adapted Borg scale (0–10). While the back endurance test was performed, surface EMG was recorded.

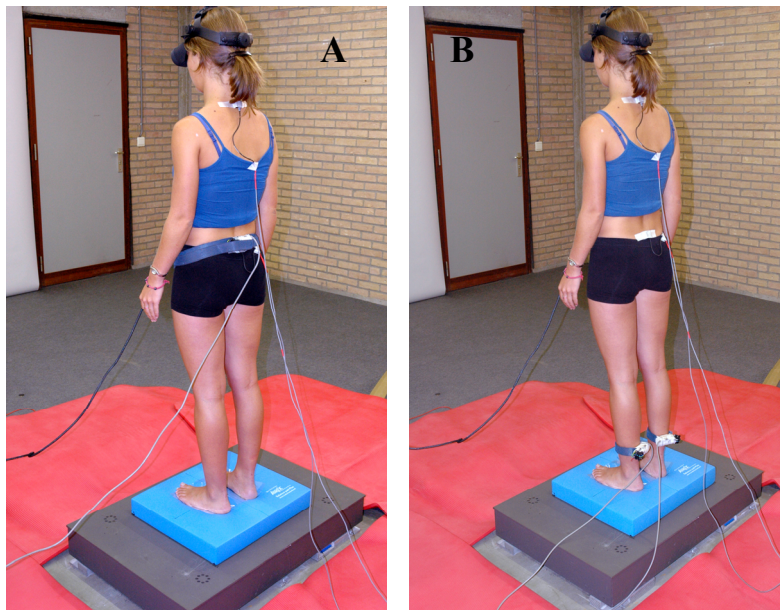


Figure 2. Lumbar multifidus (A) and triceps surae (B) muscle vibration during upright standing on an unstable support surface (“foam”).

4.3.4 Postural sway analysis

In paper III, postural sway characteristics (mean center of pressure COP and root mean square (RMS) during upright of standing subject were measured using a six-channel force plate (Bertec Corporation, OH, USA). It recorded the moments of force around the frontal (M_x) and sagittal (M_y) axes and the vertical ground reaction force (F_z). Force plate data were sampled at 500 Hz using a Micro 1401 data acquisition system and Spike2 software (Cambridge Electronic Design, UK) and low pass filtered with a cut-off frequency of 5 Hz. To evaluate trunk position in space two piezo-resistive accelerometers (ICSensors, UK) also connected with the data-acquisition system were placed on the spinosus processes of T1 and S1 vertebra in upright posture.

4.3.4 Dynamometry

MVC force of the back extensor muscles was measured by standard back dynamometer DC-200 (Russia) (Paper IV). Each individual was instructed to stand on a platform with knees fully extended and head and trunk erect. The participant grasped the hand bar using an alternating grip and the hand bar was positioned across the thighs. The participant was instructed to pull the hand bar straight upward using the back muscles and to roll the shoulders backward during the pull, without leaning backward. Each pull lasted approximately 3 seconds (Heyward, 2000). A plumb line was hung from the ceiling directly behind the participant. If his/her back came in contact with this line, indicating that the participant was starting to lean backward, the test was terminated immediately. Three trials were administered with a 1-minute recovery period between each (Heyward, 2000). The highest of the three measurements was recorded. Each participant was encouraged to exhale throughout the entire contraction to avoid the Valsalva maneuver. The dynamometer was calibrated prior to the start of data collection to ensure that each measurement was accurate.

4.4. Data reduction and statistical analysis

Standard statistical methods were used for the calculation of means and standard errors of the means (\pm SE) (Papers I, II and IV). During Sørensen back endurance test, the MPF was determined and averaged over each period of 5 s. The MPF was defined as the weighted mean value of the data points forming the single spectrum. The following characteristics were calculated: initial MPF (first 5 s), end MPF and MF (last 5 s), MPF and MF slopes (% change/min) for right and left side (Paper I) (mean of the right and left side data). MF slope was taken as indicator of the erector spinae muscle fatigability (Papers I, II and IV). One-way analysis of variance (ANOVA) followed by Tukey post hoc compari-

sons was used to evaluate differences between the groups and between body sides. Pearson's correlation coefficient was used to estimate linear relationships between subject's anthropometric characteristics, endurance time and EMG power spectrum parameters. Statistical significance was accepted at $p < 0.05$ (Papers I, II and IV).

In paper III, postural sway characteristics were recorded from the force plate readings using Spike2 and Microsoft Excel software. Displacements of the COP in the anterior-posterior direction were calculated from the raw force plate data using the equation: $COP = M_x/F_z$. Further data reduction was performed by calculating the RMS values of the COP displacements as a measure of postural stability and the mean values in order to appraise the directional effect of muscle vibration on COP displacement. The COP displacements in the muscle vibration trials were analyzed over two epochs: the 15 s preceding and the 15 s during muscle vibration. Positive values correspond to forward COP displacement and negative values with backward COP displacement. In addition, ratios of the COP displacement during the TS muscle vibration trials versus MF muscle vibration trial were calculated to determine the proprioceptive postural control strategy using the equation: $RW_{TS/MF} = \text{absolute TS} / (\text{abs TS} + \text{abs MF})$. Where RW is the relative proprioceptive weighting, abs TS is the absolute value of mean COP displacement during TS muscles vibration and abs MF is the absolute value of mean COP displacement during MF vibration. A score of zero means 100% reliance on lumbar muscles proprioception in postural control. In contrast, a score of one means 100% reliance on proprioception of the ankle muscles in postural control (Brumagne et al., 2008).

To determine back muscle fatigue MPF of the EMG was calculated (Ng and Richardson, 1996). Differences between the two groups, conditions and trials were analyzed using a repeated measures analysis of variance (ANOVA/MANOVA). Post hoc analysis (Tukey) was performed on significant main and interaction effects to calculate specific effects. The level of statistical significance was set at $p < 0.05$. The statistical analysis was performed with Statistica 9.0 (Statsoft, OK, USA) (Paper III).

5. RESULTS

5.1. Low back muscle fatigue during Sørensen endurance test in people with and without chronic low back pain

Endurance time

People with chronic LBP had significantly shorter ($p < 0.05$) endurance time (235.0 ± 30.7 s) of the Sørensen test as compared to healthy controls (352.0 ± 42.2 s) (Fig. 3A, Paper I). Male with chronic LBP had shorter ($p < 0.05$) endurance time (230.2 ± 42.0 s) in the Sørensen test compared to the healthy female (308.3 ± 30.0 s) and male subjects (314.0 ± 53.0 s) (Fig. 3B, Paper II). No significant differences in endurance time were observed between the healthy male and female subjects, and female with chronic LBP.

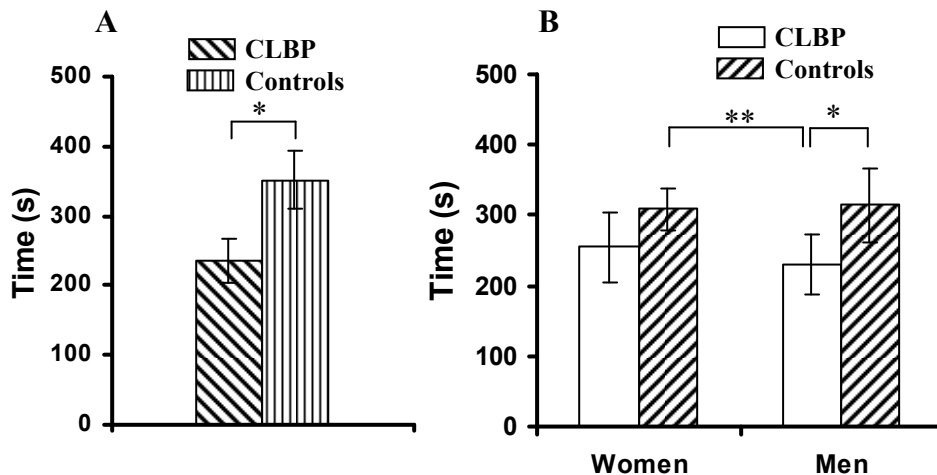


Figure 3. Endurance time in people with chronic low back pain (CLBP) and healthy controls (mean±SE). A – Paper I; B – Paper II. * $p < 0.05$; ** $p < 0.01$.

Changes in surface EMG spectral parameters

The MPF significantly declined as time of isometric contraction progressed. No significant differences ($p > 0.05$) in initial MPF during first 5 s and end MPF of right or left side during last 5 s of the Sørensen back isometric endurance test between the measured groups were observed (Fig. 4A, Paper I). People with chronic LBP had significantly higher ($p < 0.05$) MPF slope for left and right side compared to healthy controls (Fig. 4B, Paper I).

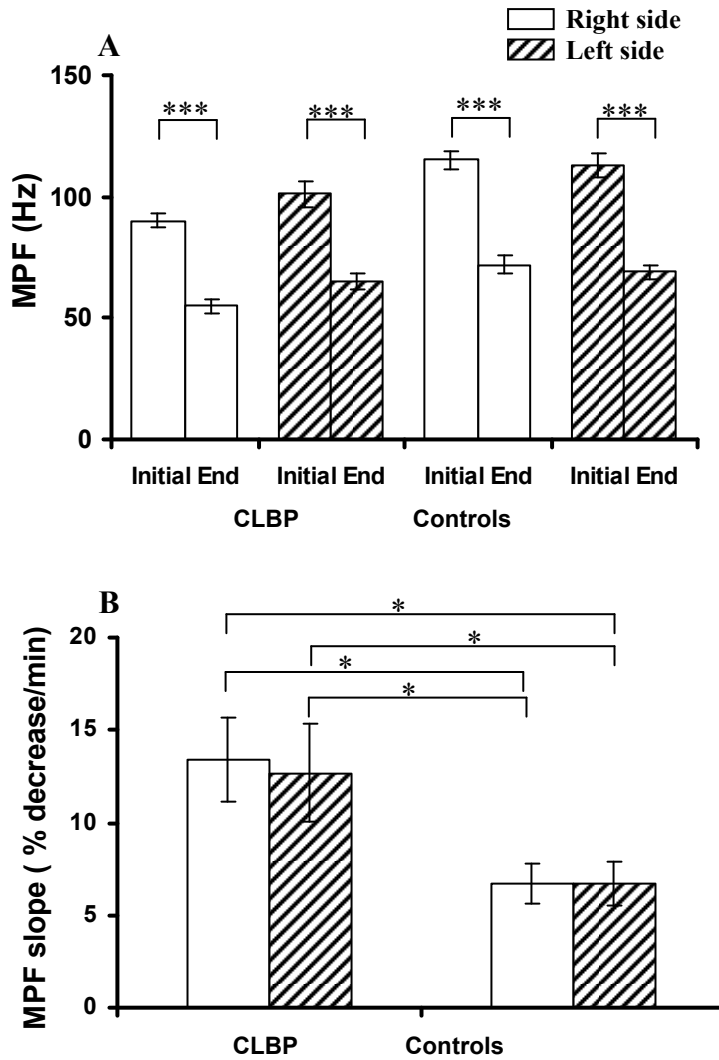


Figure 4. Initial and end mean power frequency (MPF) of EMG power spectrum (A) and MPF decrease over time (MPFslope) (B) of right and left side during the Sørensen back endurance test in people with chronic low back pain (CLBP) and controls (mean±SE). * $p < 0.05$; *** $p < 0.001$.

Healthy male subjects had higher ($p<0.05-0.01$) MF slope than healthy female subjects (Fig. 5, Paper II). No significant differences in MF slope were found between the female and male with chronic LBP, and between people with chronic LBP and healthy subjects.

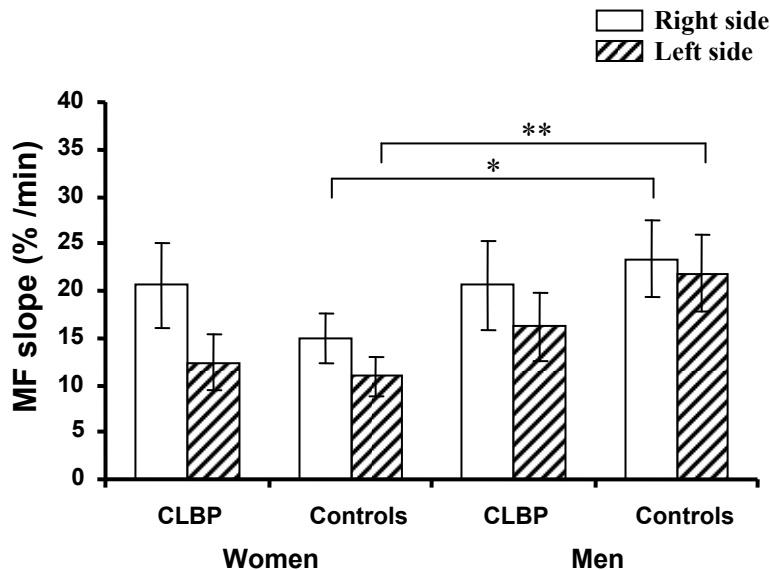


Figure 5. EMG power spectrum median frequency decrease over time (MFslope) of the erector spinae muscle during the Sørensen back endurance test in people with chronic low back pain (CLBP) and healthy controls (mean \pm SE). * $p<0.05$; ** $p<0.01$.

Correlation between EMG power spectrum changes and anthropometric changes

In people with chronic LBP the endurance time of the Sørensen test correlated significantly negatively with BMI ($r=-0.71$; $p<0.01$), and MPF slope of right side ($r=-0.85$; $p<0.001$), MPF slope of left side ($r=-0.65$; $p<0.05$) (Table 3, Paper I). In controls, BMI correlated significantly positively with initial MPF of right side ($r=0.58$; $p<0.05$) and left side ($r=0.72$; $p<0.01$), MPF slopes of right side ($r=0.57$; $p<0.05$) and left side ($r=0.68$; $p<0.05$). A significant negative correlation between endurance time, MPF slopes of right side ($r=-0.80$; $p<0.01$) and left side ($r=-0.73$; $p<0.01$) was also observed (Paper I).

Table 3. Pearson correlation coefficients between BMI, endurance time and EMG spectral parameters during the Sørensen endurance test in people with chronic LBP (n=12) and controls (n=12).

Parameters	BMI	t _{endur}	Initial MPF (right)	Initial MPF (left)	MPF slope (right)	MPF slope (left)
LBP group						
BMI	X	-0.71*	0.17	0.45	0.47	0.20
t _{end}		X	0.26	-0.22	-0.85*	-0.65*
Initial MPF (right)			X	0.78*	-0.42	-0.32
Initial MPF (left)				X	0.01 _v	-0.06
MPF slope (right)					X	0.90*
MPF slope (left)						X
Controls						
BMI	X	-0.28	0.58*	0.72*	0.57*	0.68*
t _{end}		X	-0.12	-0.23	-0.80*	-0.73*
Initial MPF (right)			X	0.77*	0.32	0.32
Initial MPF (left)				X	0.48	0.49
MPF slope (right)					X	0.95*
MPF slope (left)						X

Abbreviations: LBP – low back pain, BMI – body mass index, t_{endur} – endurance time, initial MPF – EMG mean power frequency during first 5 s of the Sørensen endurance test; MPFslope – EMG mean power frequency decrease over time during the Sørensen endurance test, *p<0.05.

Table 4 and Table 5 (Paper II) provide the correlation coefficients between anthropometric parameters and low back muscle fatigue characteristics in female subjects and in male subjects, respectively. In female and male with chronic LBP and healthy female subjects the endurance time correlated moderately to strongly negatively ($r = -0.46$ to -0.75) with MF slope. In male with chronic LBP and in healthy female subjects body mass and BMI correlated moderately negatively with endurance time ($r = -0.44$ to -0.69). Female with chronic LBP had strong positive correlation and healthy control subjects had moderate positive correlation between endurance time and initial MF for right side during the Sørensen endurance test ($r = 0.72$ and 0.49 , respectively). Body mass and BMI correlated moderately positively with MF slope ($r = 0.40$ – 0.67) in all measured subject groups. In healthy female subjects MF slope correlated moderately positively with initial MF ($r = 0.45$ – 0.61) (Paper II).

Table 4. Correlations between anthropometric parameters and low back muscle fatigue characteristics during the Sørensen back endurance test in female with chronic LBP and healthy subjects.

Parameters	Height	BM	BMI	t _{endur}	Initial MF (right)	Initial MF (left)	MF slope (right)	MF slope (left)
LBP group (n=10)								
Height	X	0.23	-0.28	-0.20	-0.38	-0.38	-0.01	0.16
BM		X	0.85*	-0.21	-0.01	0.30	0.64*	0.52
BMI			X	-0.12	0.15	0.50	0.57	0.44
t _{endur}				X	0.72*	0.29	-0.63*	-0.75*
Initial MF (right)	MF				X	0.70*	-0.38	-0.15
Initial MF (left)						X	0.22	0.22
MF slope (right)							X	0.45
MF slope (left)								X
Controls (n=10)								
Height	X	0.01	0.07	0.08	-0.37	0.28	-0.15	-0.01
BM		X	0.88*	-0.44	0.27	0.46	0.40	0.52
BMI			X	-0.65*	0.42	0.68*	0.47	0.64*
t _{endur}				X	-0.33	-0.35	-0.16	-0.59
Initial MF (right)	MF				X	0.47	0.60	0.58
Initial MF (left)						X	0.45	0.61
MF slope (right)							X	0.51
MF slope (left)								X

Abbreviations: LBP – low back pain; BM – body mass; BMI – body mass index; t_{endur} – endurance time; MF – median frequency; MF slope – median frequency decrease over time.

* p < 0.05.

Table 5. Correlations between anthropometric parameters and low back muscle fatigue characteristics during the Sørensen back endurance test in male with chronic LBP and healthy subjects.

Parameters	Height	BM	BMI	t _{endur}	Initial MF (right)	Initial MF (left)	MF slope (right)	MF slope (left)
LBP group (n=10)								
Height	X	0.28	-0.39	0.19	-0.40	-0.68	0.23	0.22
BM		X	0.77	-0.58	0.07	0.16	0.67*	0.64*
BMI			X	-0.69*	0.32	0.61	0.65*	0.57
t _{endur}				X	-0.26	-0.57	-0.46	-0.66*
Initial MF (right)					X	0.78*	0.02	0.10
Initial MF (left)						X	0.14	0.39
MF slope (right)							X	0.30
MF slope (left)								X
Controls (n=10)								
Height	X	0.51	0.43	0.07	0.39	0.35	0.29	0.22
BM		X	0.90*	0.22	0.65*	0.91*	0.61	0.46
BMI			X	0.02	0.41	0.71*	0.56	0.59
t _{endur}				X	0.49	0.42	-0.31	-0.36
Initial MF (right)					X	0.83*	0.11	0.01
Initial MF (left)						X	0.01	0.06
MF slope (right)							X	0.97*
MF slope (left)								X

Abbreviations: LBP – low back pain; BM – body mass; BMI – body mass index; t_{endur} – endurance time; MF – median frequency; MF slope – median frequency decrease over time.

* p < 0.05

5.2. Low back muscle strength in females with chronic low back pain (Paper IV).

MVC force of back extensor muscles was significantly higher ($p < 0.05$) in controls compared to female with chronic LBP (Fig. 6). Back extensor muscle isometric strength was 24.1% less in female with chronic LBP compared to healthy controls.

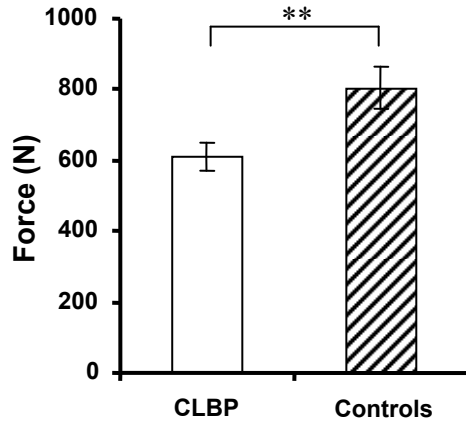


Figure 6. Isometric maximal voluntary contraction force of back extensor muscles in female with chronic low back pain (CLBP) and in healthy controls (mean \pm SE). ** $p < 0.01$.

5.3. Postural stability and proprioceptive control strategies in people with recurrent low back pain and healthy subjects (Paper III)

Control condition (non-fatigued back muscles)

People with recurrent LBP showed significantly larger posterior sways than controls during ankle muscle vibration when standing on an unstable support surface ($p < 0.001$) in the control condition (i.e. non-fatigued back muscles). However, back muscle vibration induced significantly smaller anterior sways in the unstable support surface trials in people with recurrent LBP compared to healthy controls ($p < 0.001$) (Fig. 7).

Based on the proprioceptive weighting ratios people with LBP showed a significantly more ankle-steered proprioceptive control strategy compared to healthy subjects (Table 6).

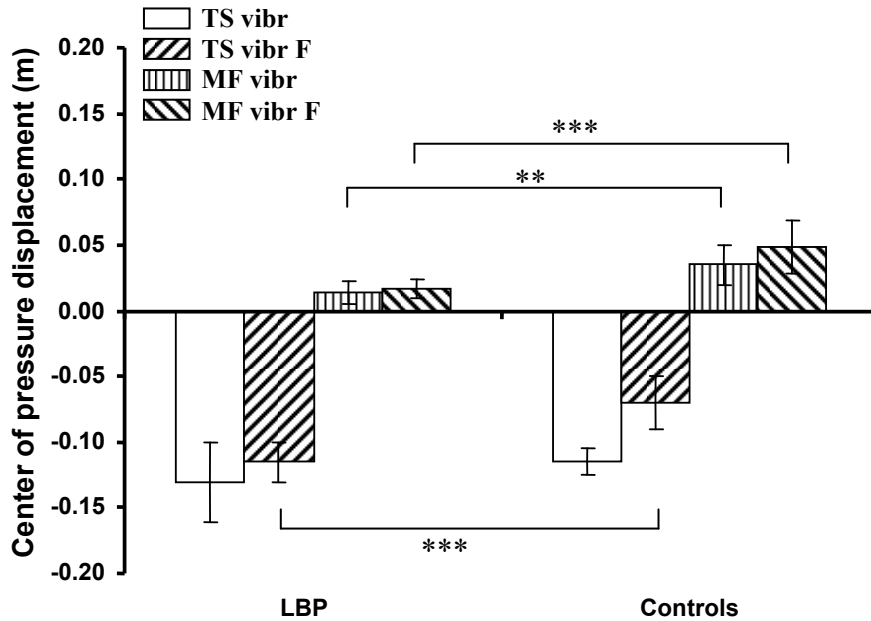


Figure 7. Anterior-posterior sways for the muscle vibration trials in the control condition TS vibr – triceps surae muscles vibration; TS vibr F – triceps surae muscles vibration on foam; MF vibr – lumbar multifidus muscles vibration; MF vibr F – lumbar multifidus muscles vibration on foam; LBP – low back pain; (mean \pm SD) ** $p < 0.01$; *** $p < 0.001$.

Table 6. Relative proprioceptive weighting ratios of the people with recurrent LBP and healthy subjects (mean \pm SD).

Condition		LBP patients (n=16) (%)	Controls (n=16) (%)	F-value	p-value
Control	RW TS/MF	0.85 \pm 0.07	0.73 \pm 0.11	12.31	0.002
	RW TS/MF F	0.86 \pm 0.07	0.52 \pm 0.16	58.69	0.000
Fatigued	RW TS/MF	0.86 \pm 0.09	0.78 \pm 0.11	6.11	0.007
	RW TS/MF F	0.86 \pm 0.09	0.72 \pm 0.10	30.67	0.000

Abbreviations: RW – relative weighting; TS – triceps surae muscles vibration; MF – lumbar multifidus muscles vibration; F – foam; LBP – low back pain.

Back muscle fatigue condition

Postural stability and proprioceptive control strategies after back muscle fatigue

Back muscle fatigue induced a significant decrease in postural stability in healthy subjects when standing on an unstable support surface compared to the control condition (mean RMS values: 0.131 ± 0.037 m vs. 0.081 ± 0.025 m, respectively; $p < 0.05$). People with LBP maintained their decreased postural stability after back muscle fatigue was induced (mean RMS values: 0.136 ± 0.038 m (on foam) and 0.123 ± 0.033 m; $p > 0.05$).

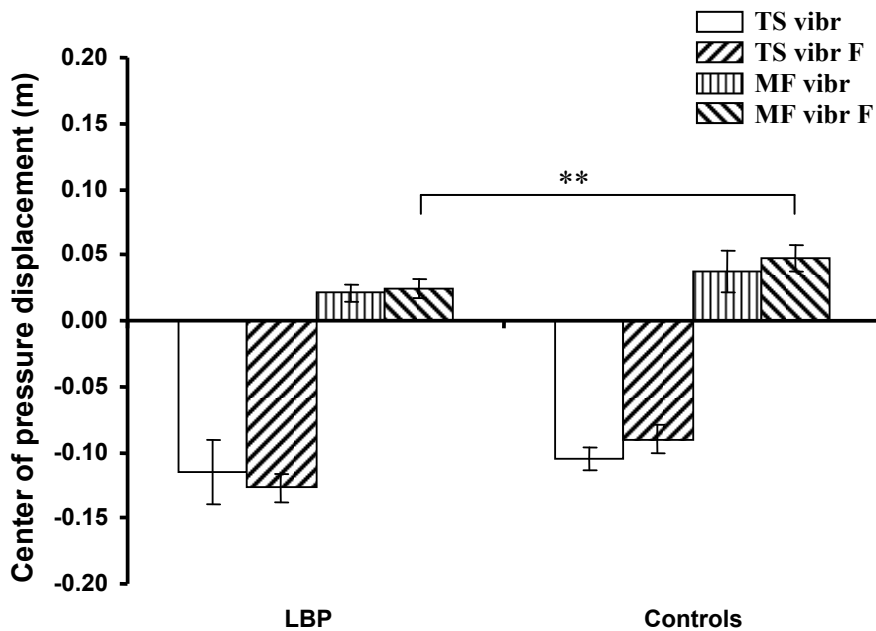


Figure 8. Anterior-posterior sways for the muscle vibration trials in the fatigue condition (mean \pm SD). TS vibr – triceps surae muscles vibration; TS vibr F – triceps surae muscles vibration on foam; MF vibr – lumbar multifidus muscles vibration; MF vibr F – lumbar multifidus muscles vibration on foam; LBP – low back pain patients. ** $p < 0.01$.

Back muscle fatigue had no significant influence on proprioceptive control during both the TS muscle vibration and MF muscle vibration trials in healthy subjects and people with recurrent LBP while standing on a stable support surface ($p > 0.05$). However, when standing on an unstable support surface, acute back muscle fatigue induced an increased backward sway during ankle muscle vibration in healthy subjects compared to the control condition ($p < 0.001$). Moreover, back muscle fatigue induced a significant decrease in anterior sway during MF vibration in the healthy controls compared to the

control condition when standing on the “foam” support (0.039 ± 0.020 m vs. 0.054 ± 0.022 m, respectively; $p < 0.05$). Despite this decrease, people with LBP still showed significantly smaller forward sways during MF vibration compared to the healthy individuals ($p < 0.01$) (Fig. 8).

The proprioceptive weighting ratios showed that when healthy subjects were standing on an unstable support surface, and their back muscles were fatigued, they relied significantly more on ankle proprioception for postural control than they did when their back muscles were not fatigued ($p < 0.001$) (Table 6).

In contrast, back muscle fatigue did not have a significant additional influence on relative proprioceptive weighting ratios in people with recurrent LBP compared to healthy controls when standing on an unstable support surface ($p > 0.05$).

6. DISCUSSION

6.1. Low back muscle fatigue during Sørensen endurance test in people with chronic low back pain: relationship between electromyography power spectrum changes, back muscle strength and antropometric characteristics

EMG power spectrum changes during Sørensen endurance test

In this thesis, the lumbar erector spinae muscle MPF decreases during the Sørensen back isometric endurance test. This is in agreement with the results of previous studies of back extensor muscle isometric contractions (Smidt and Blanpied, 1987, Roy et al., 1989, van Dieen et al., 1993, Mannion and Dolan, 1994, Kankaanpää et al., 1998a,b). The EMG spectrum shift to lower frequencies (spectral compression) caused by neural and metabolic factors during fatiguing contractions (Lindström et al., 1970), i.e. an intracellular pH decrease due to lactate accumulation and H^+ concentration (Brody et al., 1991) or extracellular K^+ accumulation (Linssen et al., 1991, Sjøgaard, 1991). In the investigated moderate contractions, lactate production was expected to have been minimal, especially in view of the high percentage of type I (slow twitch) muscle fibres shown to be present in erector spinae muscle (Jørgensen and Nicolaisen, 1991). Extracellular K^+ accumulation might, therefore, be the important factor limiting erector spinae muscle endurance at moderate contraction levels. The exact physiological mechanisms behind the EMG spectral changes are believed to be multifactorial, where a number of factors has been suggested to influence the rate of EMG spectral shifts toward lower frequencies during fatiguing contractions. These factors include: (1) slowing of action potential velocity, (2) synchronization of motor units, (3) slowing of firing frequency, (4) recruitment of new motor units during the fatiguing contraction (DeLuca, 1984). Although the exact mechanisms underlying the EMG spectral compression are not fully understood, the resultant shift to lower frequencies during sustained contraction is recognized as an electrophysiological monitoring of fatigue process (Hägg, 1992, Mannion and Dolan, 1994, Umezue et al., 1998, Kankaanpää et al., 1998a).

In addition to changes in EMG power spectrum parameters the present study indicated that people with chronic performed a shorter sustained isometric contraction of the back extensor muscles till exhaustion (endurance time of Sørensen test) and showed greater lumbar erector spinae muscle MPF slopes for right and left side, than did age-and gender-matched healthy controls. This suggests that people with chronic LBP fatigued faster than controls in the Sørensen back endurance test. This finding is in agreement with several earlier studies (Biering-Sørensen, 1984, Nicolaisen and Jørgensen, 1985, Mayer et al.,

1989, Roy et al., 1989, Hultman et al., 1993). These results discussed above are from the Paper I.

The results from Paper II suggested that male with chronic LBP had a reduced back extensor muscle isometric endurance compared to the healthy female subjects when performed the Sørensen back endurance test until exhaustion, but not with female subjects with LBP. Male with chronic LBP had shorter endurance time, i.e. fatigued faster than healthy control subjects despite the subjects having been strongly verbally encouraged to continue throughout the sustained isometric contraction. Several previous studies demonstrated a reduced back extensor muscle isometric endurance in people with chronic LBP compared to the healthy control subjects (Nicolaisen and Jørgensen, 1985, De Luca, 1993, Da Silva et al., 2005). Many studies comparing men to women during the Sørensen back isometric endurance test reported shorter endurance time in men (Mannion, 1999, Oddsson and De Luca, 2003, Da Silva et al., 2005). However, the relative weight of the trunk is generally lower for women and consequently the trunk holding task is at a lower level of MVC, may resulting in a longer endurance time in this test (Frymoyer et al., 1983, Roy et al., 1998).

The most commonly used EMG variable for assessing low back muscle fatigue is MF or half-power point of the EMG (Smidt and Blanpied, 1987). The initial value of MF was associated to the distribution of the muscle fibre type recruited (Sung et al., 2005), while MF slope, i.e. the rate of change over time, was associated to the fatigability properties of the active motor units (Tesch et al., 1983). In the present study, healthy male subjects had a higher initial EMG power spectrum MF of the erector spinae muscle compared to the healthy female subjects when performing Sørensen back endurance test (Paper II). This is an indicator of the greater pre-fatigue loading of the erector spinae muscle during sustained isometric contraction in healthy men than in women. Concerning gender, several studies have reported that women performed the endurance contraction longer and showed less progressive decreases in spectral indices (MF slope) than did the men (Mannion and Dolan, 1994, Mayer et al., 1995, Kankaanpää et al., 1998a). This suggests that women fatigue more slowly than men in the Sørensen back endurance test (Biering-Sørensen, 1984, Oddsson et al., 1991).

A steeper MF slope of the erector spinae muscle during the Sørensen back endurance test in healthy male subjects was observed compared to the healthy female subjects, indicating greater fatigability in men. A steeper MF slope obtained from the lumbar erector spinae muscle in healthy men compared to the women during an unsupported trunk holding test has been reported previously (Mannion and Dolan, 1994), which is in good agreement with the present results. The gender differences in lumbar muscle fatigability during the Sørensen test can most likely be explained by the differences in muscle anatomic and functional characteristics or it can partially by the higher weight of the torso/upper limbs of men compared to women and therefore back muscle activity at a higher percentage of maximal voluntary contraction. It has been shown that

back muscles in women have a greater relative cross-sectional area of fatigue-resistant type I fibres (women 73% vs men 56%), and as much as a twofold higher type I/type II fibre area ratio than back muscles in men (Thorstensson and Calson, 1987). In addition, men have a 17% larger total erector spinae cross-sectional area than women (Parkkola et al., 1993). In several previous studies, low back muscle fatigue has been assessed by using EMG power spectrum indices (Mannion, 1999, Oddson and De Luca, 2003). It has been suggested that EMG power spectrum MF slope during fatigue reflects the changes in the action potential propagation of individual muscle fibers that are a result of the underlying accumulation of metabolic by-products (lactate and extracellular K^+) during the fatiguing contraction (Lindström et al., 1970, van Dieen et al., 2003).

Back muscle strength

Decreased back extensor muscle strength has often been associated with LBP (Biering-Sørensen, 1984, Hultman et al., 1993). In the present thesis back extensor muscle isometric strength was 24.1% less in female with chronic LBP compared to healthy controls. There seems to be an agreement that people with LBP especially ones with chronic problems have weaker back extensor muscles than healthy persons (Nicolaisen and Jørgensen, 1985, Klein et al., 1991). Not only are the back muscles weaker but there are also modification in extensor /flexor muscle strength ratio (Mayer et al., 1985, Shirado et al., 1995). In literature people with LBP have a significant loss of both flexor and extensor muscle strength, but the main loss of strength is found in the back extensors (Mayer et al., 1985, Hultman et al., 1993, Shirado et al., 1995, Kankaanpää, 1999). Chronicity and severity of LBP may be supplementary factors for the reduction in back extensor muscle strength. Numerous investigations have shown that people with chronic LBP are weaker than the subjects with acute or even intermittent LBP (Hultman et al., 1993, Kankaanpää, 1999).

LBP has been shown to be associated with histomorphological and structural changes in the paraspinal muscles, i.e. the back muscles are smaller, contain more fat, show a degree of selective muscle fibre atrophy (Verbunt et al., 2003) and their blood circulation may be restricted because calcific deposits in the abdominal aorta and vertebral arteries (Kauppila et al., 1997, 2004). In consequence, the lumbar paraspinal muscles are weaker (Häkkinen et al., 2003) and exhibit excessive fatigability (Mannion et al, 1997, Greenough et al., 1998, Humphrey et al., 2005).

Correlations between antropometric parameters and low back pain muscle fatigue characteristics

The results of correlation analysis from Paper I showed that endurance time of the Sørensen test correlated negatively with MPF slopes of the erector spinae muscle in people with chronic LBP and healthy controls. High correlations have

been reported between MPF or MF slopes during fatiguing contractions of the back extensor muscles and endurance time by several authors (van Dieen et al., 1993, Mannion and Dolan, 1994). The initial MPF of the erector spinae muscle during first the 5 s of the Sørensen back endurance test did not differ significantly between the groups of chronic LBP pain and healthy controls, which indicates the similar back extensor muscle loading in both subject groups in pre-fatigue condition. This confirms the group differences in fatiguability to be real and not caused by group differences in muscle loading. No right-left side differences were found for initial MPF and MPF slope in people with chronic LBP and controls. Thus, the muscle loading and rate of decrease of muscle activation during fatiguing contraction was similar for both sides. The differences between people with chronic LBP and controls in back extensor muscle fatigability during the Sørensen test can be explained by several factors. Subjects with LBP often avoid using their back in everyday situations, because of fear of pain and its consequences (Waddell et al., 1993). Also poor co-ordination of paraspinal muscles has been related with CLBP and with excess lumbar muscle fatigability (Wilder et al., 1996, Taimela et al., 1999, Leinonen et al., 2003). These changes are widely thought to be a consequence of disuse and deconditioning, secondary to pain and illness, a process called the deconditioning syndrome (Nachemson and Lindh, 1969, Thorstensson and Arvidson, 1982). It has been shown that lumbar back muscle fatigue leads to abnormal spinal movements due to loss of precise muscle co-ordination which increase mechanical loading of passive elements, such as ligaments and intervertebral discs, and may cause back injury and pain (Wilder et al., 1996). Poor back muscle endurance may predict future occurrence of LBP (Biering-Sørensen, 1984, Mannion et al., 1997).

The data from present thesis suggest that BMI has a significant influence on back extensor muscle fatigability in the Sørensen isometric endurance test. The correlation analysis indicated that in people with chronic LBP with high BMI the endurance time was shorter than in people with low BMI (Paper I). Healthy control subjects with high BMI had greater lumbar erector spinae muscle initial MPF as well as MPF slope for right and left side. This suggest that subjects with high BMI had greater back extensor muscle loading and fatigued faster during Sørensen test than subjects with low BMI. In literature, there are some suggestions, that subject body mass (weight) may influence the Sørensen isometric endurance test result (Biering-Sørensen, 1984, Mannion and Dolan, 1994, Alaranta et al., 1994). Kankaanpää et al. (1998a) investigated the influence of BMI on paraspinal muscle fatigability (endurance time, EMG spectral indices) by using Sørensen test and found a strong influence of this factor. BMI showed a strong negative correlation, endurance time a strong positive correlation with paraspinal muscle fatigability (MF slope). Multiple regression analysis indicated that MF slope (fatigue) during the test was dependent on BMI in both sexes, but the effect of BMI was more pronounced in women than men. Several previous studies showed rising of LBP prevalence with increasing BMI

(de Leboeuf-Yde et al., 1999, Bayramoglu et al., 2001). This association may suggest a role of body weight and height in the pathogenesis of LBP. These findings support the previously reported need for education regarding weight reduction as useful implement in LBP prevention (Orvieto et al., 1994). Correlation data from Paper II showed differences in EMG power spectrum compression parameters during fatiguing contraction in people with chronic LBP compared with those in healthy control subjects have usually shown a steeper slope (Hägg, 1992, Vestgaard-Poulsen et al., 1995). However, no significant differences in MF slope between female and male with chronic LBP were found in the present study and this finding is somewhat surprising for us. These differences between our findings and previous studies may be caused by the differences in the experimental settings and the number of the subjects who participated in this study. It has been indicated that MF slope obtained from the low back muscles during a sustained submaximal contraction is approximately linear and strongly negatively correlated with endurance time, suggesting being a sensitive, objective, and motivation-independent indicator providing information regarding the degree of muscle fatigue (Alaranta et al., 1995, Mannion, 1999, Pääsuke et al., 2002). In the present study, MF slope of the erector spinae muscle during Sørensen back endurance correlated moderately to strongly with endurance time in female and male with chronic LBP and healthy female control subjects. One purpose of this study was to correlate objective patterns of the low back muscle fatigue with subject's anthropometric parameters. A moderate to strong negative correlation between body mass and BMI, and endurance time evaluated during Sørensen test was observed in male with chronic LBP and in healthy female control subjects. Body mass and BMI correlated moderately negatively with MF slope in all measured groups of subjects. Thus, the correlation analysis indicated that subjects with higher body mass and BMI appeared to fatigue faster during Sørensen back endurance test than that of subjects with lower body mass and BMI. However, the relationship between anthropometric characteristics and low back muscle fatigability in different loading conditions and its association with gender and chronic LBP need further clarification.

6.2. Proprioceptive postural control in people with recurrent low back pain:

The effect of back muscle fatigue on postural stability and postural control strategies (Paper III)

Acute back muscle fatigue may be a mechanism to induce changes in proprioceptive postural strategy. The main result of this study is that healthy individuals after back muscle fatigue were significantly more dependent on ankle proprioception while standing on an unstable support surface in comparison

with the control condition. This suggests that in healthy subjects back muscle fatigue induced a shift to a more ankle-steered proprioceptive postural control strategy when standing on an unstable support surface, as used by people with recurrent LBP.

People with recurrent LBP were more dependent on ankle signals in comparison to healthy subjects during the control condition. An explanation for this reliance could be reduced lumbosacral proprioception (Brumagne et al., 2000, Newcomer et al., 2000). This probably leads to a refocusing of proprioceptive sensitivity from the trunk to the ankles (Brumagne et al., 2004, Brumagne et al., 2008, Claeys et al. 2011). The CNS regulates the body stability while standing or locomotion mainly by means of afferent signals from the visual system (Mergner et al., 2005), proprioceptors (Bove et al., 2003, Tresch, 2007) and changes in vestibular input (Bacsi and Colebatch, 2005). Another possible but not mutually exclusive explanation is increased antagonistic cocontraction of the trunk muscles to stabilize the spine (Granata et al., 2004), which might lead to a reduced multi-segmental control strategy (Mok et al., 2007). They will restore and maintain their equilibrium by moving around the ankles and keeping the rest of their body stiff. This way of controlling posture is in line with the inverted pendulum model (Winter et al., 1998). Alterations in postural control (Nies and Sinnott, 1991, Luoto et al., 1996, Mientjes and Frank, 1999, Mok et al., 2004, Moseley et al., 2004, Moseley and Hodges, 2005), impairments in motor control (Hodges and Richardson, 1996) and altered lumbosacral proprioceptive acuity (Brumagne et al., 2000, Newcomer et al., 2000) have been observed in people with recurrent LBP, which might be a causative factors in their postural instability. Pain also may be a confounding factor to maintain postural stability, but it is not certain whether pain causes changes in motor control or whether motor control changes lead to pain, or both (Arendt-Nielsen et al., 1996, Hodges et al., 2001, Hodges and Moseley, 2003).

Moreover, our results demonstrated that people with recurrent LBP sustained their reliance on ankle proprioception for controlling posture while standing on an unstable support surface. An unstable support surface decreases the acuity of ankle proprioceptive signals (Ivanenko et al., 2000). Therefore, their sustained reliance on ankle proprioceptive showed their inability to switch to a more appropriate proprioceptive postural control strategy, as demonstrated by other studies (Mok et al., 2007, Brumagne et al., 2008, Claeys et al. 2011), leading to decreased postural stability. Muscle activity must be coordinated to maintain control of the spine and the efficacy of the muscle system is dependent on its controller, the CNS (Panjabi, 1992). Numerous studies have reported impaired balance in people with LBP when standing on one (Luoto et al., 1998) or two legs (Nies and Sinnott, 1991) and people with poor performance in a test of standing balance have an increased risk for LBP (Takala and Viikari-Juntura, 2000). In contrast, based on the lower proprioceptive weighting ratios, healthy controls seemed to make more use of other proprioceptive signals, in addition to those from the ankles, which is more in line with the multi-segmental control

model (Allum et al., 1998, Morasso and Schieppi, 1999). These results confirmed our previous findings (Brumagne et al., 2004, Brumagne et al., 2008).

In back muscle fatigue condition healthy individuals had a longer endurance time of the back extensor muscles in comparison to people with recurrent LBP. These results are in agreement with previous studies (Biering-Sørensen, 1984, Mannion et al., 1997, Latimer et al., 1999). Both the significant decline in MPF of the back muscles and the very high perceived effort scores after the back endurance test in both groups suggested that real fatigue of the back muscles was induced.

However, despite the very high perceived effort scores, it is still important to take the possibility of a submaximal performance regarding back muscle endurance into consideration. Pain-related factors might contribute to the perception of a maximal effort in people with recurrent LBP (Tam and Yeung, 2006). Moreover, the rate of decline in mean MPF is similar between the groups suggesting that the patients may not have reached the same level of fatigue as the healthy individuals. In addition, most people with recurrent LBP reported significantly more pain after the back muscle fatigue test, so we cannot exclude that they stopped earlier due to this increase in pain.

Back muscle fatigue in healthy individuals resulted in a significantly stronger reliance on proprioceptive signals from the ankles for controlling posture during quiet standing on “foam”, resulting in a decreased postural stability. Vibration of triceps surae muscles can give the illusion of forward leaning and therefore the subject will compensate with a backwards shift of the center of mass, even to the point of falling (Eklund, 1972, Brumagne et al., 2004). In comparison with the control condition, significantly larger posterior sways have been shown during triceps surae muscle vibration while standing on an unstable support surface. An increased sway due to lumbar muscle fatigue has already been shown by some studies (Davidson et al., 2004, Vuillerme et al., 2007). Vuillerme et al. suggested that the significant interaction between fatigue and support surface could be attributable to the sensory reweighting hypothesis. It is possible that healthy individuals reweight their sensory input from the trunk to the ankles due to the back muscle fatigue. This can be explained by the negative influence of fatigue on the muscle receptors and thereby on proprioception (Gandevia, 2001, Allen and Proske, 2006). Muscle fatigue may be caused by peripheral changes or by a failure of the CNS to drive the motoneurons adequately (Gandevia et al., 1996, Taylor and Gandevia, 2008). It possibly influences postural control due to altered muscle contractile efficiency (Bigland-Ritchie et al., 1983, Duchateau and Hainaut, 1985), proprioceptive acuity (Allen and Proske, 2006) and cortical control (Taylor et al., 1996, Gandevia, 2001, Taylor and Gandevia, 2008). Excessive fatigability of back extensor muscles is common among people with chronic LBP (Biering-Sørensen, 1984, Mannion et al., 1997, Latimer et al., 1999). Due to lumbar muscle fatigue proprioceptive acuity can decrease, which leads to inaccurate signals about lumbar spine position and movement (Taimela et al., 1999). Under simple (non-fatigued) postural

conditions greater dependence upon proprioceptive input from the ankles is the norm, and increased input from back muscle spindles only becomes important when the stance is unstable, in which case healthy controls adapt their strategy accordingly but people with recurrent LBP do not. It is the ability to adapt postural control strategy in unstable conditions which then appears to be lost in the healthy subjects when back muscle is fatigued. However, the possible relationship between back muscle fatigue and the selection of a proprioceptive postural control strategy in healthy individuals and in people with recurrent LBP need more investigations.

A limitation of this present thesis was a relatively small number of subjects in the measured groups of people with LBP and age- and gender matched controls. Besides, gender differences in MVC force of back extensor muscles in people with and without LBP were not analyzed. Because of the young age, low disability and moderately challenging postural tasks of the studied subjects, the collected results may underestimate the postural control impairment that can be observed in a patient population of older age and with higher disability during the more demanding activities of daily life. Therefore, future studies with an older population and with more expressed disabilities have to be conducted. Despite the short rest periods between the trials in testing postural control strategies, the learning effects and general fatigue cannot be ruled out from affecting the results. In addition, back muscle fatigue might be recovered when performing postural control trials. However, the total duration of these trials was about 10 min. So, complete recovery from back muscle fatigue cannot be expected in that time frame. Moreover, significant differences in postural strategy were observed when standing on an unstable support surface in the last trials. In future more research is needed on the physiological basis of postural control strategies in people with and without chronic or recurrent LBP.

CONCLUSIONS

1. People with chronic low back pain fatigued faster than healthy controls during sustained submaximal isometric contraction of back extensor muscles. No gender differences were found in back extensor muscle isometric endurance in people with chronic low back pain and in healthy subjects.
2. People with and without chronic low back pain who had higher body mass and body mass index fatigued faster during sustained submaximal isometric contraction of back extensor muscles.
3. Females with chronic low back pain demonstrated lowered back extensor muscle isometric strength compared with age- and gender-matched healthy subjects.
4. People with recurrent low back pain relied strongly on ankle proprioception independent of the postural demands resulting in a decreased postural stability.
5. Back muscle fatigue in healthy subjects impaired the ability to adapt their postural control strategy and the healthy individuals were resorting to a similar postural strategy to that observed in people with recurrent low back pain when postural demands increased.

REFERENCES

1. Adams MA, Bogduk N, Burton K, Dolan P. (2002) The biomechanics of back pain. Elsevier Science.
2. Adams MA, Dolan P. (2005) Spine biomechanics. *J Biomech* 38: 1972–1983.
3. Adams MA, Mannon AF, Dolan P. (1999) Personal risk factors for first-time low back pain. *Spine* 24: 2497–2505.
4. Airaksinen O, Herno A, Kaukanen E, Saari T, Sihvonen T, Suomalainen O. (1996) Density of lumbar muscles 4 years after decompressive spinal surgery. *Eur Spine J* 5: 193–197.
5. Alaranta H, Hurri H, Heliövaara M, Soukka A, Harju R. (1994) Non-dynamometric trunk performance tests: reliability and normative data. *Scand J Rehabil Med* 26: 211–215.
6. Alaranta H, Luoto S, Heliövaara M, Hurri H. (1995) Static back endurance and the risk of low-back pain. *Clin Biomech* 10: 323–324.
7. Allen TJ, Proske U. (2006) Effect of muscle fatigue on the sense of limb position and movement. *Exp Brain Res* 170: 30–38.
8. Allum JH, Bloem BR, Carpenter MG, Hulliger M, Hadders-Algra M. (1998) Proprioceptive control of posture: a review of new concepts. *Gait Posture* 8: 214–242.
9. Andersson GBJ. (1997) The epidemiology of spinal disorders. In: Frymoyer JW (ed.). *The Adult Spine: Principles and Practice*. 2nd end. New York: Raven Press, pp. 93–141.
10. Arendt-Nielsen L, Graven-Nielsen T, Sværre H, Svensson P. (1996) The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. *Pain* 64: 231–240.
11. Bacsı AM, Colebatch JG. (2005) Evidence for reflex and perceptual vestibular contributions to postural control. *Exp Brain Res* 160: 22–28.
12. Balagué F, Troussier B, Salminen JJ. (1999) Non-specific low back pain in children and adolescents: risk factors. *Eur Spine J* 8: 429–438.
13. Bayramoğlu M, Akman MN, Kiliç S, Cetin N, Yavuz N, Ozker R. (2001) Isokinetic measurement of trunk muscle strength in women with chronic low-back pain. *Am J Phys Med Rehabil* 80: 650–655.
14. Bergmark A. (1989) Stability of the lumbar spine. A study in mechanical engineering. *Acta Orthop Scand Suppl* 230: 1–54.
15. Biedermann HJ, Shanks GL, Forrest WJ, Inglis J. (1991) Power spectrum analyses of electromyographic activity. Discriminators in the differential assessment of patients with chronic low-back pain. *Spine* 16: 1179–1184.
16. Biering-Sørensen B. (1984) Physical measurement as risk indicators for low back trouble over one year period. *Spine* 9: 106–119.
17. Biering-Sørensen F. (1983) A prospective study of low back pain in a general population. I. Occurrence, recurrence and aetiology. *Scand J Rehabil Med* 15: 71–79.
18. Bigland-Ritchie B, Donovan EF, Roussos CS. (1981) Conduction velocity and EMG power spectrum changes in fatigue of sustained maximal efforts. *J Appl Physiol* 51: 1300–1305.

19. Bigland-Ritchie B, Johansson R, Lippold OC, Woods JJ. (1983) Contractile speed and EMG changes during fatigue of sustained maximal voluntary contractions. *J Neurophysiol* 50: 313–324.
20. Bigland-Ritchie B, Jones DA, Hosking GP, Edwards RH. (1978) Central and peripheral fatigue in sustained maximum voluntary contractions of human quadriceps muscle. *Clin Sci Mol Med* 54: 609–614.
21. Bloem BR, Allum JH, Carpenter MG, Honegger F. (2000) Is lower leg proprioception essential for triggering human automatic postural responses? *Exp Brain Res* 130: 375–391.
22. Bogduk N, Macintosh JE, Percy MJ. (1992) A universal model of the lumbar back muscles in the upright position. *Spine* 17: 897–913.
23. Bogduk N, Macintosh JE. (1984) The applied anatomy of the thoracolumbar fascia. *Spine* 9: 164–170.
24. Bogduk N, Twomey LT. (1991) *Clinical Anatomy of Lumbar Spine*. Churchill Livingstone, Melbourne, 65–107.
25. Bogduk N. (1980) A reappraisal of the anatomy of the human lumbar erector spinae. *J Anat* 131: 525–540.
26. Bogduk N. (1991) The lumbar disc and low back pain. *Neurosurg Clin N Am* 2: 791–806.
27. Bogduk N. (1997) *Clinical Anatomy of the Lumbar Spine and Sacrum*. 3rd ed. Churchill Livingstone, Edinburgh, UK.
28. Borg G. (1990) Psychophysical scaling with applications in physical work and the perception of exertion. *Scand J Work Environ Health* 16: 55–58.
29. Bouter LM, van Tulder MW, Koes BW. (1998) Methodologic issues in low back pain research in primary care. *Spine* 23: 2014–2020.
30. Bove M, Nardone A, Schieppati M. (2003) Effects of leg muscle tendon vibration on group Ia and group II reflex responses to stance perturbation in humans. *J Physiol* 550: 617–630.
31. Brody LR, Pollock MT, Roy SH, De Luca CJ, Celli B. (1991) pH-induced effects on median frequency and conduction velocity of the myoelectric signal. *J Appl Physiol* 71: 1878–1885.
32. Brown MC, Engberg I, Matthews PB. (1967) The relative sensitivity to vibration of muscle receptors of the cat. *J Physiol* 192: 773–800.
33. Brumagne S, Cordo P, Lysens R, Verschuere S, Swinnen S. (2000) The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. *Spine* 15: 989–994.
34. Brumagne S, Cordo P, Verschuere S. (2004) Proprioceptive weighting changes in persons with low back pain and elderly persons during upright standing. *Neurosci Lett* 5: 63–66.
35. Brumagne S, Janssens L, Knapen S, Claeys K, Suuden-Johanson E. (2008) Persons with recurrent low back pain exhibit a rigid postural control strategy. *Eur Spine J* 17: 1177–1184.
36. Carver S, Kiemel T, Jeka JJ. (2006) Modeling the dynamics of sensory reweighting. *Biol Cybern* 95: 123–134.
37. Cavanaugh J. (1995) Mechanisms of lumbar spine . *Spine* 20, 1804–1809.

38. Cholewicki J, Panjabi MM, Khachatryan A. (1997) Stabilizing function of trunk flexor-extensor muscles around a neutral spine posture. *Spine* 22: 2207–2212.
39. Cifrek M, Medved V, Tonković S, Ostojić S. (2009) Surface EMG based muscle fatigue evaluation in biomechanics. *Clin Biomech* 24: 327–340.
40. Claeys K, Brumagne S, Dankaerts W, Kiers H, Janssens L. (2011) Decreased variability in postural control strategies in young people with non-specific low back pain is associated with altered proprioceptive reweighting. *Eur J Appl Physiol* 111: 115–123
41. Comerford MJ, Mottram SL. (2001) Movement and stability dysfunction – contemporary developments. *Man Ther* 6: 15–26.
42. Cooper RG, St Clair Forbes W, Jayson MIV. (1992) Radiographic demonstration of paraspinal muscle wasting in patients with chronic low back pain. *Br J Rheumatol* 31: 389–394.
43. Coppes MH, Marani E, Thomeer RT, Groen GJ. (1997) Innervation of “painful” lumbar discs. *Spine* 22: 2342–2350.
44. Cordo PJ, Gurfinkel VS, Brumagne S, Flores-Vieira C. (2005) Effect of slow, small movement on the vibration-evoked kinesthetic illusion. *Exp Brain Res* 167: 324–334.
45. da Silva RA Jr, Arsenault AB, Gravel D, Larivière C, de Oliveira E Jr. (2005) Back muscle strength and fatigue in healthy and chronic low back pain subjects: a comparative study of 3 assessment protocols. *Arch Phys Med Rehabil* 86: 722–729.
46. da Silva RA Jr, Arsenault AB, Gravel D, Larivière C, de Oliveira E Jr. (2005) Back muscle strength and fatigue in healthy and chronic low back pain subjects: a comparative study of 3 assessment protocols. *Arch Phys Med Rehabil* 86: 722–729.
47. Davidson BS, Madigan ML, Nussbaum MA. (2004) Effects of lumbar extensor fatigue and fatigue rate on postural sway. *Eur J Appl Physiol* 93: 183–189.
48. De Luca CJ. (1984) Myoelectrical manifestations of localized muscular fatigue in humans. *Crit Rev Biomed* 11: 251–279.
49. De Luca CJ. (1993) Use of the surface EMG signal for performance evaluation of back muscles. *Muscle Nerve* 16, 210–216.
50. Duchateau J, Hainaut K. (1985) Electrical and mechanical failures during sustained and intermittent contractions in humans. *J Appl Physiol* 58: 942–947.
51. Ebraheim NA, Hassan A, Lee M, Xu R. (2004) Functional anatomy of lumbar spine. *Semin Pain Med* 2: 131–137.
52. Eklund G. (1972) General features of vibration-induced effects on balance. *Ups J Med Sci* 77: 112–124.
53. Elfving B, Dederich A, Németh G. (2003) Lumbar muscle fatigue and recovery in patients with long-term low-back trouble – electromyography and health-related factors. *Clin Biomech* 18: 619–630.
54. Fairbank JC, Pynsent PB. (2000) The Oswestry Disability Index. *Spine* 25: 2940–2952.
55. Floyd WF, Silver PH. (1955) The function of the erector spinae muscles in certain movements and postures in man. *J Physiol* 129: 184–203.

56. Freemont AJ, Peacock TE, Goupille P, Hoyland JA, O'Brien J, Jayson MI. (1997) Nerve ingrowth into diseased intervertebral disc in chronic back pain. *Lancet* 350: 178–181.
57. Frymoyer JW, Cats-Baril WL. (1991) An overview of the incidences and costs of low back pain. *Orthop Clin North Am* 22: 263–271.
58. Frymoyer JW, Newberg A, Pope MH, Wilder DG, Clements J, MacPherson B. (1984) Spine radiographs in patients with low-back pain. An epidemiological study in men. *J Bone Joint Surg Am* 66: 1048–1055.
59. Frymoyer JW, Pope MH, Clements JH, Wilder DG, MacPherson B, Ashikaga T. (1983) Risk factors in low-back pain. An epidemiological survey. *J Bone Joint Surg Am* 65: 213–218.
60. Gandevia SC, Allen GM, Butler JE, Taylor JL. (1996) Supraspinal factors in human muscle fatigue: evidence for suboptimal output from the motor cortex. *J Physiol* 15: 529–536.
61. Gandevia SC. (1992) Some central and peripheral factors affecting human motoneuronal output in neuromuscular fatigue. *Sports Med* 13: 93–98.
62. Gandevia SC. (2001) Spinal and supraspinal factors in human muscle fatigue. *Physiol Rev* 81: 1725–1789.
63. Gill KP, Callaghan MJ. (1998) The measurement of lumbar proprioception in individuals with and without low back pain. *Spine* 23: 371–377.
64. Gilroy AM, MacPherson BR, Ross LM. (2008) *Atlas of Anatomy* (1st ed.). New York: Thieme Med Pub.
65. Goodwin GM, McCloskey DI, Matthews PBC. (1972) The contribution of muscle afferents to kinesthesia shown by vibration induced illusions of movement and by the effects of paralysing joint afferents. *Brain* 95: 705–748.
66. Granata KP, Slota GP, Wilson SE. (2004) Influence of fatigue in neuromuscular control of spinal stability. *Hum Factors* 46: 81–91.
67. Greenough CG, Oliver CW, Jones AP. (1998) Assessment of spinal musculature using surface electromyographic spectral color mapping. *Spine* 23: 1768–1774.
68. Gresty M. (1987) Stability of the head: studies in normal subjects and in patients with labyrinthine disease, head tremor, and dystonia. *Mov Disord* 2: 165–185.
69. Henry SM, Hitt JR, Jones SL, Bunn JY. (2006) Decreased limits of stability in response to postural perturbations in subjects with low back pain. *Clin Biomech* 21: 881–892.
70. Hestbaek L, Iachine IA, Leboeuf-Yde C, Kyvik KO, Manniche C. (2004) Heredity of low back pain in a young population: a classical twin study. *Twin Res* 7: 16–26.
71. 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: 149–165.
72. Heyward VH. (2000) *Advanced Fitness Assessment and Exercise Prescription* (4th ed). Champaign: Human Kinetics.
73. Hides JA, Richardson CA, Jull GA. (1996) Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine* 21: 2763–2769.

74. Hodges P, Moseley G, Gabrielsson A, Gandevia S. (2001) Acute experimental pain changes postural recruitment of the trunk muscles in pain-free humans. *Society for Neuroscience Abstracts*.
75. Hodges PW, Moseley GL. (2003) Pain and motor control of the lumbopelvic region: effect and possible mechanisms. *J Electromyogr Kinesiol* 13: 361–370.
76. Hodges PW, Richardson CA. (1996) Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. *Spine* 15: 2640–2650.
77. Hodges PW. (2001) Changes in motor planning of feedforward postural responses of the trunk muscles in low back pain. *Exp Brain Res* 141: 261–266.
78. Hoogendoorn WE, van Poppel MN, Bongers PM, Koes BW, Bouter LM. (2000) Systematic review of psychosocial factors at work and private life as risk factors for back pain. *Spine* 25: 2114–2125.
79. Hultman G, Nordin M, Saraste H, Ohlsèn H. (1993) Body composition, endurance, strength, cross-sectional area, and density of MM erector spinae in men with and without low back pain. *J Spinal Disord* 6: 114–123.
80. Humphrey AR, Nargol AV, Jones AP, Ratcliffe AA, Greenough CG. (2005) The value of electromyography of the lumbar paraspinal muscles in discriminating between chronic-low-back-pain sufferers and normal subjects. *Eur Spine J* 14: 175–184.
81. Hupli M, Sainio P, Hurri H, Alaranta H. (1997) Comparison of trunk strength measurements between two different isokinetic devices used at clinical settings. *J Spinal Disord* 10: 391–397.
82. Hägg GM. (1992) Interpretation of EMG spectral alterations and alteration indexes at sustained contraction. *J Appl Physiol* 73: 1211–1217.
83. Häkkinen A, Ylinen J, Kautiainen H, Airaksinen O, Herno A, Tarvainen U, Kiviranta I. (2003) Pain, trunk muscle strength, spine mobility and disability following lumbar disc surgery. *J Rehabil Med* 35: 236–240.
84. Ivanenko YP, Solopova IA, Levik YS. (2000) The direction of postural instability affects postural reactions to ankle muscle vibration in humans. *Neurosci Lett* 292: 103–106.
85. Johansson R, Magnusson M. (1991) Human postural dynamics. *Crit Rev Biomed Eng* 18: 413–437.
86. Johansson R. (1993) *System modeling and identification*. Englewood Cliffs, NJ: Prentice Hall.
87. Jørgensen K, Nicolaisen T. (1987) Trunk extensor endurance: determination and relation to low-back trouble. *Ergonomics* 30: 259–267.
88. Jørgensen K, Nicolaisen T. (1991) Isometric trunk extensor endurance in young females. *Abstract Book of the 23th International Congress on Biomechanics*. Perth, pp. 16–17.
89. Kankaanpää M, Laaksonen D, Taimela S, Kokko SM, Airaksinen O, Hänninen O. (1998a) Age, sex, and body mass index as determinants of back and hip extensor fatigue in the isometric Sørensen back endurance test. *Arch Phys Med Rehabil* 79: 1069–1075.

90. Kankaanpää M, Taimela S, Laaksonen D, Hänninen O, Airaksinen O. (1998b) Back and hip extensor fatigability in chronic low back pain patients and controls. *Arch Phys Med Rehabil* 79: 412–417.
91. Kankaanpää M, Taimela S, Webber CL Jr, Airaksinen O, Hänninen O. (1997) Lumbar paraspinal muscle fatigability in repetitive isoinertial loading: EMG spectral indices, Borg scale and endurance time. *Eur J Appl Physiol Occup Physiol* 76: 236–242.
92. Kankaanpää M. (1999) Lumbar muscle endurance in the assessment of physical performance capacity of low back patients. Doctoral Dissertation. Kuopio University.
93. Kauppila LI, McAlindon T, Evans S, Wilson PW, Kiel D, Felson DT. (1997) Disc degeneration/back pain and calcification of the abdominal aorta. A 25-year follow-up study in Framingham. *Spine* 15: 1642–1647.
94. Kauppila LI, Mikkonen R, Mankinen P, Peltö-Vasenius K, Mäenpää I. (2004) MR aortography and serum cholesterol levels in patients with long-term nonspecific lower back pain. *Spine* 29: 2147–2152.
95. Kelsey JL, White AA. (1980) Epidemiology and impact of low-back pain. *Spine* 5: 133–142.
96. Klein AB, Snyder-Mackler L, Roy SH, DeLuca CJ. (1991) Comparison of spinal mobility and isometric trunk extensor forces with electromyographic spectral analysis in identifying low back pain. *Phys Ther* 71: 445–454.
97. Koumantakis GA, Arnall F, Cooper RG, Oldham JA. (2001) Paraspinal muscle EMG fatigue testing with two methods in healthy volunteers. Reliability in the context of clinical applications. *Clin Biomech* 16: 263–266.
98. Krismer M, van Tulder M. (2007) Strategies for prevention and management of musculoskeletal conditions. Low back pain (non-specific). *Best Pract Res Clin Rheumatol* 21: 77–91.
99. Käser L, Mannion AF, Rhyner A, Weber E, Dvorak J, Müntener M. (2001) Active therapy for chronic low back pain: part 2. Effects on paraspinal muscle cross-sectional area, fiber type size, and distribution. *Spine* 26: 909–919.
100. Laasonen EM. (1984) Atrophy of sacrospinal muscle groups in patients with chronic, diffusely radiating lumbar back pain. *Neuroradiology* 26: 9–13.
101. Latimer J, Maher CG, Refshauge K, Colaco I. (1999) The reliability and validity of the Biering-Sorensen test in asymptomatic subjects and subjects reporting current or previous nonspecific low back pain. *Spine* 15: 2085–2089.
102. Leboeuf-Yde C, Kyvik KO, Bruun NH. (1999) Low back pain and lifestyle. Part II – Obesity. Information from a population-based sample of 29,424 twin subjects. *Spine* 24: 779–784.
103. Leinonen V, Kankaanpää M, Luukkonen M, Kansanen M, Hänninen O, Airaksinen O, Taimela S. (2003) Lumbar paraspinal muscle function, perception of lumbar position, and postural control in disc herniation-related back pain. *Spine* 28: 842–848.
104. Liebenson CS. (1992) Pathogenesis of chronic back pain. *J Manipul Physiol Ther* 15: 299–308.

105. Lindström L, Magnusson R, Petersén I. (1970) Muscular fatigue and action potential conduction velocity changes studied with frequency analysis of EMG signals. *Electromyography* 10: 341–356.
106. Linszen WH, Stegeman DF, Joosten EM, Binkhorst RA, Merks MJ, ter Laak HJ, Notermans SL. (1991) Fatigue in type I fiber predominance: a muscle force and surface EMG study on the relative role of type I and type II muscle fibers. *Muscle Nerve* 14: 829–837.
107. Linton SJ. (2000) A review of psychological risk factors in back and neck pain. *Spine* 25: 1148–1156.
108. Luoto S, Aalto H, Taimela S, Hurri H, Pyykkö 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: 2081–2089.
109. Luoto S, Taimela S, Hurri H, Aalto H, Pyykkö I, Alaranta H. (1996) Psychomotor speed and postural control in chronic low back pain patients A controlled follow-up study. *Spine* 21: 2621–2627.
110. MacGregor AJ, Andrew T, Sambrook PN, Spector TD. (2004) Structural, psychological and genetic influences on low back and neck pain: A study of adult female twins. *Arthr Rheum* 51: 160–167.
111. Macintosh JE, Bogduk N. (1987) The morphology of the lumbar erector spinae. *Spine* 1: 658–668.
112. Macintosh JE, Bogduk N. (1991) The attachments of the lumbar erector spinae. *Spine* 16: 783–792.
113. Mannion AF, Connolly B, Wood K, Dolan P. (1997) The use of surface EMG power spectral analysis in the evaluation of back muscle function. *J Rehabil Res Dev* 34: 427–439.
114. Mannion AF, Dolan P. (1994) Electromyographic median frequency changes during isometric contraction of the back extensors to fatigue. *Spine* 19: 1223–1229.
115. Mannion AF, Käser L, Weber E, Rhyner A, Dvorak J, Müntener M. (2000) Influence of age and duration of symptoms on fibre type distribution and size of the back muscles in chronic low back pain patients. *Eur Spine J* 9: 273–281.
116. Mannion AF. (1999) Fibre type characteristics and function of the human paraspinal muscles: normal values and changes in association with low back pain. *J Electromyogr Kinesiol* 9: 363–377.
117. Marras WS, Ferguson SA, Burr D, Schabo P, Maronitis A. (2007) Low back pain recurrence in occupational environments. *Spine* 32: 2387–2397.
118. Massion J. (1992) Movement, posture and equilibrium: interaction and coordination. *Prog Neurobiol* 38: 35–56.
119. Massion J. (1994) Postural control system. *Curr Opin Neurobiol* 4: 877–887.
120. Mattila M, Hurme M, Alaranta H, Paljärvi L, Kalimo H, Falck B, Lehto M, Einola S, Järvinen M. (1986) The multifidus muscle in patients with lumbar disc herniation. A histochemical and morphometric analysis of intraoperative biopsies. *Spine* 11: 732–738.

121. Mayer T, Gatchel R, Betancur J, Bovasso E. (1995) Trunk muscle endurance measurement. Isometric contrasted to isokinetic testing in normal subjects. *Spine* 20: 920–927.
122. Mayer TG, Gatchel RJ, Kishino N, Keeley J, Capra P, Mayer H, Barnett J, Mooney V. (1985) Objective assessment of spine function following industrial injury. A prospective study with comparison group and one-year follow-up. *Spine* 10: 482–493.
123. Mayer TG, Kondraske G, Mooney V, Carmichael TW, Butsch R. (1989) Lumbar myoelectric spectral analysis for endurance assessment. A comparison of normals with deconditioned patients. *Spine* 14: 986–991.
124. Mayer TG, Vanharanta H, Gatchel RJ, Mooney V, Barnes D, Judge L, Smith S, Terry A. (1989a) Comparison of CT scan muscle measurements and isokinetic trunk strength in postoperative patients. *Spine* 14: 33–36.
125. McGregor AH, Anderton L, Vedi V, Johnson J, Hughes SPF, Gedroyc WMW. (1999) The influence of position and pathology on measurements of the cross-sectional area of the multifidus and erector spinae muscle groups. Presented to the International Society for the Study of the Lumbar Spine, Hawaii.
126. Mergner T, Schweigart G, Maurer C, Blümle A. (2005) Human postural responses to motion of real and virtual visual environments under different support base conditions. *Exp Brain Res* 167: 535–556.
127. Mientjes MI, Frank JS. (1999) Balance in chronic low back pain patients compared to healthy people under various conditions in upright standing. *Clin Biomech* 14: 710–716.
128. Mok NW, Brauer SG, Hodges PW. (2004) Hip strategy for balance control in quiet standing is reduced in people with low back pain. *Spine* 29: E107–112.
129. Mok NW, Brauer SG, Hodges PW. (2007) Failure to use movement in postural strategies leads to increased spinal displacement in low back pain. *Spine* 32: E537–543.
130. Mooney V, Andersson GB. (1994) Trunk strength testing in patient evaluation and treatment. *Spine* 19: 2483–2485.
131. Mooney V. (1989) Where is the lumbar pain coming from? *Ann Med* 21: 373–379.
132. Morasso PG, Schieppati M. (1999) Can muscle stiffness alone stabilize upright standing? *J Neurophysiol* 82: 1622–1626.
133. Moseley GL, Hodges PW, Gandevia SC. (2002) Deep and superficial fibers of the lumbar multifidus muscle are differentially active during voluntary arm movements. *Spine* 27: E29–36.
134. Moseley GL, Hodges PW. (2005) Are the changes in postural control associated with low back pain caused by pain interference? *Clin J Pain* 21: 323–329.
135. Moseley GL, Nicholas MK, Hodges PW. (2004) Pain differs from non-painful attention-demanding or stressful tasks in its effect on postural control patterns of trunk muscles. *Exp Brain Res* 156 : 64–71.
136. Nachemson A, Lindh M. (1969) Measurement of abdominal and back muscle strength with and without low back pain. *Scand J Rehabil Med* 1: 60–63.

137. Nachemson AL, Waddell G, Norlund AI. (2000) Epidemiology of neck and low back pain. In *Neck and back pain: The scientific evidence of causes, diagnosis and treatment*: 165–188.
138. Newcomer KL, Laskowski ER, Yu B, Johnson JC, An KN. (2000) Differences in repositioning error among patients with low back pain compared with control subjects. *Spine* 25: 2488–2493.
139. Newton M, Waddell G. (1993) Trunk strength testing with iso-machines. Part 1: Review of a decade of scientific evidence. *Spine* 18: 801–811.
140. Ng JK, Richardson CA, Jull GA. (1997) Electromyographic amplitude and frequency changes in the iliocostalis lumborum and multifidus muscles during a trunk holding test. *Phys Ther* 77: 954–961.
141. Ng JK, Richardson CA, Kippers V, Parnianpour M. (1998) Relationship between muscle fiber composition and functional capacity of back muscles in healthy subjects and patients with back pain. *J Orthop Sports Phys Ther* 27: 389–402.
142. Ng JK, Richardson CA. (1996) Reliability of electromyographic power spectral analysis of back muscle endurance in healthy subjects. *Arch Phys Med Rehabil* 77: 259–264.
143. Nicolaisen T, Jørgensen K. (1985) Trunk strength, back muscle endurance and low-back trouble. *Scand J Rehabil Med* 17: 121–127.
144. Nies N, Sinnott PL. (1991) Variations in balance and body sway in middle-aged adults. Subjects with healthy backs compared with subjects with low-back dysfunction. *Spine* 16: 325–330.
145. Oddsson LI, De Luca CJ. (2003) Activation imbalances in lumbar spine muscles in the presence of chronic low back pain. *J Appl Physiol* 94: 1410–1420.
146. Oddsson LI, Giphart JE, Buijs RJ, Roy SH, Taylor HP, De Luca CJ. (1997) Development of new protocols and analysis procedures for the assessment of LBP by surface EMG techniques. *J Rehabil Res Dev* 34: 415–426.
147. Oddsson LIE, Moritani T, Andersson E, Thorstensson A. (1991) Differences between males and females in EMG and fatigability of lumbar back muscles. In: *Electromyographical Kinesiology. Proceedings of the 8th Congress of the International Society of Electrophysiological Kinesiology*. Amsterdam: Elsevier Science, pp. 295–298.
148. Orvieto R, Rand N, Lev B, Wiener M, Nehama H. (1994) Low back pain and body mass index. *Mil Med* 159: 37–38.
149. Panjabi MM. (1992) The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *J Spinal Disord* 5: 383–389.
150. Parkkola R, Rytökoski U, Kormano M. (1993) Magnetic resonance imaging of the discs and trunk muscles in patients with chronic low back pain and healthy control subjects. *Spine* 18: 830–836.
151. Peach JP, McGill SM. (1998) Classification of low back pain with the use of spectral electromyogram parameters. *Spine* 23: 1117–1123.
152. Pääsuke M, Johanson E, Proosa M, Ereline J, Gapeyeva H. (2002) Back extensor muscle fatigability in chronic low back pain patients and controls: relationship between electromyogram power spectrum and body mass index. *J Back Musculoskel Rehabil* 16: 17–24.

153. Radebold A, Cholewicki J, Panjabi MM, Patel TC. (2000) Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. *Spine* 25: 947–954.
154. Rissanen A, Kalimo H, Alaranta H. (1995) Effect of intensive training on the isokinetic strength and structure of lumbar muscles in patients with chronic low back pain. *Spine* 20: 333–340.
155. Roll JP, Vedel JP. (1982) Kinaesthetic role of muscle afferents in man, studied by tendon vibration and microneurography. *Exp Brain Res* 47: 177–190.
156. Roy SH, De Luca CJ, Casavant DA. (1998) Lumbar muscle fatigue and chronic lower back pain. *Spine* 12: 992–1001.
157. Roy SH, De Luca CJ, Emley M, Buijs RJ. (1995) Spectral electromyographic assessment of back muscles in patients with low back pain undergoing rehabilitation. *Spine* 20: 38–48.
158. Rätty HP, Kujala U, Videman T, Koskinen SK, Karppi SL, Sarna S. (1999) Associations of isometric and isoinertial trunk muscle strength measurements and lumbar paraspinal muscle cross-sectional areas. *J Spinal Disord* 12: 266–270.
159. Schwarzer AC, Aprill CN, Bogduk N. (1995) The sacroiliac joint in chronic low back pain. *Spine* 20: 31–37.
160. Shirado O, Ito T, Kaneda K, Strax TE. (1995) Concentric and eccentric strength of trunk muscles: influence of test postures on strength and characteristics of patients with chronic low-back pain. *Arch Phys Med Rehabil* 76: 604–611.
161. Siddall PJ, Cousins MJ. (1997) Spinal pain mechanisms. *Spine* 22: 98–104.
162. Sihvonen T, Herno A, Paljärvi L, Airaksinen O, Partanen J, Tapaninaho A. (1993) Local denervation atrophy of paraspinal muscles in postoperative failed back syndrome. *Spine* 18: 575–581.
163. Sihvonen T, Lindgren KA, Airaksinen O, Manninen H. (1997) Movement disturbances of the lumbar spine and abnormal back muscle electromyographic findings in recurrent low back pain. *Spine* 22: 289–295.
164. Sjøgaard G. (1991) Role of exercise-induced potassium fluxes underlying muscle fatigue: a brief review. *Can J Physiol Pharmacol* 69: 238–245.
165. Smidt GL, Blanpied PR. (1987) Analysis of strength tests and resistive exercises commonly used for low-back disorders. *Spine* 12: 1025–1034.
166. Sung PS, Zurcher U, Kaufman M. (2005) Nonlinear analysis of electromyography time series as a diagnostic tool for low back pain. *Med Sci Monit* 11:1–5.
167. Suzuki N, Endo S. (1983) A quantitative study of trunk muscle strength and fatigability in the low-back-pain syndrome. *Spine* 8: 69–74.
168. Söderström T, Stoica P. (1989) *System Identification*. Englewood Cliffs, NJ: Prentice Hall.
169. Taimela S, Kankaanpää M, Luoto S. (1999) The effect of lumbar fatigue on the ability to sense a change in lumbar position. A controlled study. *Spine* 24: 1322–1327.
170. Takala EP, Viikari-Juntura E. (2000) Do functional tests predict low back pain? *Spine* 25: 2126–2132.
171. Tam GY, Yeung SS. (2006) Perceived effort and low back pain in non-emergency ambulance workers: implications for rehabilitation. *J Occup Rehabil* 16: 231–240.

172. Taylor JL, Butler JE, Allen GM, Gandevia SC. (1996) Changes in motor cortical excitability during human muscle fatigue. *J Physiol* 490: 519–528.
173. Taylor JL, Gandevia SC. (2008) A comparison of central aspects of fatigue in submaximal and maximal voluntary contractions. *J Appl Physiol* 104: 542–550.
174. Tesch PA, Komi PV, Jacobs I, Karlsson J, Viitasalo JT. (1983) Influence of lactate accumulation on EMG frequency spectrum during repeated concentric contractions. *Acta Physiol Scand* 119: 61–67.
175. Thorstensson A, Arvidson A. (1982) Trunk muscle strength and low back pain. *Scand J Rehabil Med* 14: 69–75.
176. Thorstensson A, Carlson H. (1987) Fibre types in human lumbar back muscles. *Acta Physiol Scand* 131: 195–202.
177. Thorstensson A, Nilsson J. (1982) Trunk muscle strength during constant velocity movements. *Scand J Rehabil Med* 14: 61–68.
178. Tresch MC. (2007) A balanced view of motor control. *Nat Neurosci* 10: 1227–1228.
179. Tsuboi T, Satou T, Egawa K, Izumi Y, Miyazaki M. (1994) Spectral analysis of electromyogram in lumbar muscles: fatigue induced endurance contraction. *Eur J Appl Physiol Occup Physiol* 69: 361–366.
180. Turk DC, Okifuji A. (1999) Assessment of patients' reporting of pain: an integrated perspective. *Lancet* 353: 1784–1788.
181. Umezu Y, Kawazu T, Tajima F, Ogata H. (1998) Spectral electromyographic fatigue analysis of back muscles in healthy adult women compared with men. *Arch Phys Med Rehabil* 79: 536–538.
182. Waddell G, Newton M, Henderson I, Somerville D, Main CJ. (1993) A Fear-Avoidance Beliefs questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain* 52: 157–168.
183. Waddell G. (1987) A new clinical model for the treatment of low-back pain. *Spine* 12: 632–644.
184. van den Hoogen HJ, Koes BW, van Eijk JT, Bouter LM, Devillé W. (1998) On the course of low back pain in general practice: a one year follow up study. *Ann Rheum Dis* 57: 13–19.
185. van Dieën JH, Oude Vrielink HH, Housheer AF, Lötters FB, Toussaint HM. (1993) Trunk extensor endurance and its relationship to electromyogram parameters. *Eur J Appl Physiol Occup Physiol* 66: 388–396.
186. van Dieën JH, Selen LP, Cholewicki J. (2003) Trunk muscle activation in low-back pain patients, an analysis of the literature. *J Electromyogr Kinesiol* 13: 333–351.
187. van Tulder M, Malmivaara A, Esmail R, Koes B. (2000) Exercise therapy for low back pain: a systematic review within the framework of the cochrane collaboration back review group. *Spine* 25:2784–2796.
188. Wasiak R, Kim J, Pransky G. (2006) Work disability and costs caused by recurrence of low back pain: longer and more costly than in first episodes. *Spine* 31: 219–225.
189. Venna S, Hurri H, Alaranta H. (1994) Correlation between neurological leg deficits and reaction time of upper limbs among low-back pain patients. *Scand J Rehabil Med* 26: 87–90.

190. Verbunt JA, Seelen HA, Vlaeyen JW, van de Heijden GJ, Heuts PH, Pons K, Knotterus JA. (2003) Disuse and deconditioning in chronic low back pain: concepts and hypotheses on contributing mechanisms. *Eur J Pain* 7: 9–21.
191. Vestergaard-Poulsen P, Thomsen C, Sinkjaer T, Henriksen O. (1995) Simultaneous ³¹P-NMR spectroscopy and EMG in exercising and recovering human skeletal muscle: a correlation study. *J Appl Physiol* 79: 1469–1478.
192. Viikari-Juntura E, Vuori J, Silverstein BA, Kalimo R, Kuosma E, Videman T. (1991) A life-long prospective study on the role of psychosocial factors in neck-shoulder and low-back pain. *Spine* 16: 1056–1061.
193. Wilder DG, Aleksiev AR, Magnusson ML, Pope MH, Spratt KF, Goel VK. (1996) Muscular response to sudden load. A tool to evaluate fatigue and rehabilitation. *Spine* 21: 2628–2639.
194. Winter DA, Patla AE, Prince F, Ishac M, Gielo-Perczak K. (1998) Stiffness control of balance in quiet standing. *J Neurophysiol* 80: 1211–1221.
195. Vuillerme N, Anziani B, Rougier P. (2007) Trunk extensor muscles fatigue affects undisturbed postural control in young healthy adults. *Clin Biomech* 22: 489–494.

SUMMARY IN ESTONIAN

Selja sirutajalihaste väsimus ja posturaalkontroll alaseljavalude korral

Sissejuhatus

Inimese lülisammas on kõikvõimalike komprimeerivate koormuste suhtes väga tundlik. Seljavaevusi on ulatuslikult uuritud, kuid vaatamata sellele on seljavalud üks töövõimetus sagedasemaid põhjusi. Enamasti tekivad alaseljavalud staatiliste pingutuste tõttu sundasendites, tingituna lülisamba halvast funktsionaalsest seisundist, kerelihaste nõrkusest või lihaste neuraalse kontrolli häiretest kesknärvisüsteemi tasandil. Ka kõrgetasemeliste diagnostikameetoditega pole paljudel juhtudel võimalik alaseljavalude põhjust kindlaks määrata. Seljalihaste funktsionaalse seisundi hindamiseks kasutatakse vastupidavusteste, millega hinnatakse väsimuse teket staatilise ja dünaamilise lihastöö tingimustes. Palju on uuritud seljalihaste jõu ja vastupidavuse seoseid alaseljavaevustega patsientidel võrreldes tervetega, kuid väsimuse lokalisatsiooni iseärasused sõltuvalt indiviidi vanusest, soost ja kehaehituslikest iseärasustest pole veel lõplikult selged. Viimasel kümnendil on suuremat tähelepanu hakatud pöörama nn. motoorsele kontrollile, mida juhib kesknärvisüsteem ja mis peab tagama optimaalse kehaasendi igapäevatoimingutes. Kesknärvisüsteemi poolt vastuvõetava sensoorse sisendi häirunud identifitseerimine põhjustab kehaasendi muutumist ruumis. Sensorne sisend proprioretseptiivse signaali näol lihastest võib olla häirunud mitmel põhjusel. Käesolevas doktoritöös kasutati lihasväsimust kui mehhanismi uurimaks, missuguseid strateegiaid kasutavad alaseljavaludega vaatlusalused võrreldes tervetega oma kehaasendi säilitamiseks selle häirumise korral erinevates tingimustes.

Doktoritöö põhieesmärk oli hinnata selja sirutajalihaste väsimust ja posturaalkontrolli alaseljavaevustega indiviididel võrreldes tervetega.

Uurimistöö ülesanded

Põhieesmärgist lähtuvalt püstitati uurimistöös järgmised ülesanded:

1. Uurida selja sirutajalihaste väsimust submaksimaalse staatilise lihas-kontraktsiooni tingimustes ja seoseid antropomeetriliste karakteristikute ning väsimuse näitajate vahel alaseljavaludega vaatlusalustel võrreldes tervetega.
2. Võrrelda selja sirutajalihaste maksimaalset tahtelist jõudu alaseljavaludega vaatlusalustel ja tervetel.
3. Hinnata akuutse lihasväsimuse mõju kehaasendi kontrollile alaseljavaludega vaatlusalustel võrreldes tervetega.

Uuritavad ja kasutatav metoodika

Uuringutes osales kokku 57 alaseljavaludega ja 58 alaseljavaludeta inimest (kontrollgrupina). Seljalihaste jõu ja vastupidavuse uuringud viidi läbi Tartu Ülikooli kinesioloogia ja biomehaanika laboris aastatel 2002–2005. Kehasendi kontrolli uuringud viidi läbi Leuveni Ülikooli füsioteraapia laboris aastatel 2006–2007. Seljalihaste isomeetrilist vastupidavust hinnati Sørenseni testiga, mille käigus registreeriti seljalihaste bioelektriline aktiivsus *erector spinae* lihasel. Seljalihaste isomeetrilise maksimaaljõu testimisel kasutati standartset seljadünamomeetrit. Lihavibratsiooni kasutati lihaskävide I a afferentide stimuleerimiseks, et kutsuda esile kehasendi häirumine ruumis.

Järeldused

1. Selja sirutajalihaste kestva submaksimaalse staatilise pingutuse tingimustes väsisid alaseljavaludega inividid kiiremini võrreldes tervetega. Soolisi erinevusi seljalihaste staatilises vastupidavuses alaseljavaludega inivididel ja tervetel ei täheldatud.
2. Selja sirutajalihaste tahteline maksimaaljõud oli alaseljavaludega inivididel võrreldes tervetega väiksem.
3. Suurema kehamassi ja kehamassiindeksiga inivididel arenes selja sirutajalihaste väsimus kestva submaksimaalse staatilise pingutuse tingimustes kiiremini.
4. Alaseljavaludega inivididel domineeris kehasendi stabiilsuse säilitamisel raskendatud tingimustes hüppeliigese strateegia.
5. Seljalihaste akuutse väsimuse tingimustes kasutasid terved ja alaseljavaludega inividid sarnast kehasendi säilitamise strateegiat.

ACKNOWLEDGEMENTS

I wish to express my sincere gratitude and appreciation to all the people who helped me during this effort, and in particular:

- prof. *Mati Pääsuke*, my academic supervisor of this thesis, for the ideas to get started and full support during preparation of the thesis until completion; he has provided me with enough freedom to make my own decisions, but at the same time, he has strongly kept me on the right path;
- prof. *Simon Brumagne*, my co-supervisor from Katholieke Universiteit Leuven, Belgium; with his expertise and modern ideas, he has helped me to widen and deepen my understanding of the postural control using muscle vibration in people with low back pain;
- dr. *Helena Gapeyeva*, PhD, for help in conducting the studies;
- I am deeply grateful to dr. *Jaan Ereline*, PhD, for the help in statistical analysis of this study and patiently guided me through the many and varied statistical problems, for careful and patient help with the layouts of figures and tables in this thesis:
- *Mare Vene* for editing the English language;
- my family for their positive attitude and moral support through my doctorate study;
- my friends who helped me in any way to complete the thesis.

PUBLICATIONS

CURRICULUM VITAE

Ege Johanson

Date of birth: 05. 08 1979

Nationality: Estonian

Address: Siili 6–12, 50104 Tartu

Phone: +372 56 914 277

E-mail: egejoh@ut.ee

Education

- 2003–2011 PhD student in Faculty of Exercise and Sport Sciences, University of Tartu
- 2006–2007 Katholieke Universiteit Leuven, Belgium
- 2001–2003 Faculty of Exercise and Sport Sciences, University of Tartu, MSc *cum laude*
- 1997–2001 Faculty of Exercise and Sport Sciences, University of Tartu, BSc *cum laude*
- 1994–1997 Parksepa Secondary School

Employment

- 2005–2010 OÜ BioDesign, physiotherapist
- 2010 University of Tartu, Faculty of Medicine, Institute of Anatomy, assistant
- 2006 University of Tartu, Faculty of Exercise and Sport Sciences, Institute of Exercise Biology and Physiotherapy, assistant
- 2002–2005 East Tallinn Central Hospital, physiotherapist

Retraining

- 2011 4th Baltic Conference in Exercise and Sport Sciences, oral presentation
- 2010 „International Baltic arthroscopy and Sports medicine Conference”, Tartu, Estonia
- 2010 Baltic and North Sea Conference on PRM”, Stockholm, Sweden
- 2006–2010 Leuven Katholieke Universitet
- 2005 „International Society of Physical and Rehabilitation Medicine ISPRM”, Sao Paulo, Brasil, (oral presentation)
- 2004 Symposium: “State of the Art in Chronic Low Back Pain” Bodrumis, Turkey (oral presentation)

Main Research Interests:

- Back muscle fatigability
- Proprioceptive control of muscle and its effect on postural balance

Publications:

- Papers in international refereed journals – 5
- Other scientific articles – 13
- Abstracts – 10

ELULOOKIRJELDUS

Ege Johanson

Sünniaeg: 05. 08 1979
Kodakondsus: Eesti
Aadress: Siili 6–12, 50104 Tartu
Telefon: +372 56 914 277
Email: egejoh@ut.ee

Haridus

2003-2011 Tartu Ülikooli kehakultuuriteaduskond, doktoriõpe
2006–2007 Katholieke Universiteit Leuven, Belgia, doktoriõpe
2001–2003 Tartu Ülikooli kehakultuuriteaduskond, MSc *cum laude*
1997–2001 Tartu Ülikooli kehakultuuriteaduskond, BSc *cum laude*
1994–1997 Parksepa Keskkool

Töökogemus

2005– BioDesign OÜ, füsioterapeut
2010 Tartu Ülikool, Arstiteaduskond, Anatoomia instituut, assistent
2006 Tartu Ülikool, Kehakultuuriteaduskond, Spordibioloogia ja füsioteraapia instituut; erakorraline teadur
2002–2005 Ida-Tallinna Keskhaigla, füsioterapeut

Erialane enesetäiendus

2011 4th Baltic Conference in Exercise and Sport Sciences, suuline ettekanne, Tartu, Eesti
2010 „International Baltic arthroscopy and Sports medicine Conference”, Tartu, Eesti
2010 Baltic and North Sea Conference on PRM”, Stockholm, Rootsi
2006–2010 Leuveni Katoliiklik Ülikool, valikuliselt erialane täiendkoolitus ülikooli kliinikus
2005 „International Society of Physical and Rehabilitation Medicine ISPRM”, Sao Paulo, Brasiilia (suuline ettekanne)
2004 Sümpoosium nimega: “State of the Art in Chronic Low Back Pain” Bodrumis, Türgi (suuline ettekanne).

Peamised uurimisvaldkonnad

- Seljalihaste väsimus: ealised, soolised ja alaseljavaludega seotud aspektid
- Proprioretseptiivne posturaalne kontroll alaseljavaludega patsientidel

Publikatsioonid:

- Teaduslikud artiklid rahvusvahelise levikuga ajakirjades – 5
- Muud teaduslikud artiklid – 13
- Konverentside teesid – 10

DISSERTATIONES KINESIOLOGIAE UNIVERSITATIS TARTUENSIS

1. **Lennart Raudsepp.** Physical activity, somatic characteristics, fitness and motor skill development in prepubertal children. Tartu, 1996, 138 p.
2. **Vello Hein.** Joint mobility in trunk forward flexion: methods and evaluation. Tartu, 1998, 107 p.
3. **Leila Oja.** Physical development and school readiness of children in transition from preschool to school. Tartu, 2002, 147 p.
4. **Helena Gapeyeva.** Knee extensor muscle function after arthroscopic partial meniscectomy. Tartu, 2002, 113 p.
5. **Roomet Viira.** Physical activity, ecological system model determinants and physical self-perception profile in early adolescence. Tartu, 2003, 167 p.
6. **Ando Pehme.** Effect of mechanical loading and ageing on myosin heavy chain turnover rate in fast-twitch skeletal muscle. Tartu, 2004, 121 p.
7. **Priit Kaasik.** Composition and turnover of myofibrillar proteins in volume — overtrained and glucocorticoid caused myopathic skeletal muscle. Tartu, 2004, 123 p.
8. **Jarek Mäestu.** The perceived recovery-stress state and selected hormonal markers of training stress in highly trained male rowers. Tartu, 2004, 109 p.
9. **Karin Alev.** Difference between myosin light and heavy chain isoforms patterns in fast- and slow-twitch skeletal muscle: effect of endurance training. Tartu, 2005, 117 p.
10. **Kristjan Kais.** Precompetitive state anxiety, self-confidence and athletic performance in volleyball and basketball players. Tartu, 2005, 99 p.
11. **Aire Leppik.** Changes in anthropometry, somatotype and body composition during puberty: a longitudinal study. Tartu, 2005, 161 p.
12. **Jaan Ereline.** Contractile properties of human skeletal muscles: Association with sports training, fatigue and posttetanic potentiation. Tartu, 2006, 133 p.
13. **Andre Koka.** The role of perceived teacher feedback and perceived learning environment on intrinsic motivation in physical education. Tartu, 2006, 137 p.
14. **Priit Purge.** Performance, mood state and selected hormonal parameters during the rowing season in elite male rowers. Tartu, 2006, 101 p.
15. **Saima Kuu.** Age-related contractile changes in plantarflexor muscles in women: associations with postactivation potentiation and recreational physical activity. Tartu, 2006, 101 p.
16. **Raivo Puhke.** Adaptive changes of myosin isoforms in response to long-term strength training in skeletal muscle of middle-aged persons. Tartu, 2006, 99 p.

17. **Eva-Maria Riso.** The effect of glucocorticoid myopathy, unloading and reloading on the skeletal muscle contractile apparatus and extracellular matrix. Tartu, 2007, 114 p.
18. **Terje Sööt.** Bone mineral values in young females with different physical activity patterns: association with body composition, leg strength and selected hormonal parameters. Tartu, 2007, 94 p.
19. **Karin Tammik.** Neuromuscular function in children with spastic diplegic cerebral palsy. Tartu, 2007, 102 p.
20. **Meeli Saar.** The relationships between anthropometry, physical activity and motor ability in 10–17-year-olds. Tartu, 2008, 96 p.
21. **Triin Pomerants.** Ghrelin concentration in boys at different pubertal stages: relationships with growth factors, bone mineral density and physical activity. Tartu, 2008, 80 p.
22. **Tatjana Kums.** Musculo-skeletal function in young gymnasts: association with training loads and low-back pain. Tartu, 2008, 128 p.
23. **Maret Pihu.** The components of social-cognitive models of motivation in predicting physical activity behaviour among school students. Tartu, 2009, 116 p.
24. **Peep Päll.** Physical activity and motor skill development in children. Tartu, 2009, 102 p.
25. **Milvi Visnapuu.** Relationships of anthropometrical characteristics with basic and specific motor abilities in young handball players. Tartu, 2009, 114 p.
26. **Rita Gruodytė.** Relationships between bone parameters, jumping height and hormonal indices in adolescent female athletes. Tartu, 2010, 82 p.
27. **Ragnar Viir.** The effect of different body positions and of water immersion on the mechanical characteristics of passive skeletal muscle. Tartu, 2010, 142 p.
28. **Iti Määrsepp.** Sensorimotor and social functioning in children with developmental speech and language disorders. Tartu, 2011, 90 p.