KUOPION YLIOPISTON JULKAISUJA D. LÄÄKETIEDE 314
KUOPIO UNIVERSITY PUBLICATIONS D. MEDICAL SCIENCES 314

VILLE LEINONEN

Neuromuscular Control in Lumbar Disorders

Doctoral dissertation

To be presented by permission of the Faculty of Medicine of the University of Kuopio for public examination in Auditorium L3, Canthia building, University of Kuopio, on Thursday 6th November 2003, at 12 noon

Departments of Physical and Rehabilitation Medicine,
Physiology, Neurosurgery, Clinical Neurophysiology
University of Kuopio

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ISBN 951-781-354-6
ISSN 1235-0303

Kopijyvä
Kuopio 2003
Finland

Leinonen, Ville. Neuromuscular Control in Lumbar Disorders. Kuopio University Publications D. Medical Sciences 314. 2003. 59 p.
ISBN 951-781-354-6
ISSN 1235-0303

ABSTRACT

Impaired motor and sensory functions have been associated with low back pain (LBP). This includes disturbances in a wide range of sensorimotor control e.g. sensory dysfunctions, impaired postural responses and psychomotor control. However, the physiological mechanisms, clinical relevance and characteristics of these findings in different spinal pathologies require further clarification.

The purposes of this study were to investigate postural control, lumbar muscle function, movement perception and associations between these findings in healthy volunteers (n=35), patients with lumbar disc herniation (n=20) and lumbar spinal stenosis (LSS, n=26).

Paraspinal muscle responses for sudden upper limb loading and muscle activation during flexion-extension movement and the lumbar endurance test were measured by surface electromyography (EMG). Postural stability was measured on a force platform during two- and one-footed standing. Lumbar movement perception was assessed in a motorised trunk rotation unit in the seated position. In addition, measurements of motor- (MEP) and somatosensory evoked potentials (SEP) and needle EMG examination of lumbar multifidus muscles were performed in the LSS patients. Clinical and questionnaire data were also recorded.

A short latency paraspinal muscle response (~50 ms) for sudden upper limb loading was observed. The latency of the response was shortened by expectation ($p=0.017$). The response latency for unexpected loading was similar in healthy persons and disc herniation patients but the latency was not shortened by expectation in the patients ($p=0.014$). Also impaired postural control ($p<0.05$) and lumbar movement perception ($p=0.012$) were observed in disc herniation patients. The impaired lumbar movement perception ($p=0.054$) and anticipatory muscle activation ($p=0.043$) tended to be restored after successful surgery but postural control had still not recovered after 3 months of follow-up. The majority of LSS patients were unable to sense a rotational movement in the lumbar area and thus had clearly impaired lumbar movement perception ($p=0.006$). Abnormal MEPs had only inconsistent and SEPs showed no associations with impaired movement perception and postural stability in LSS. Abnormal needle EMG findings and flexion-extension activation of paraspinal muscles were frequently observed in LSS patients. Lumbar paraspinal muscle endurance was better than in previously evaluated healthy subjects and chronic LBP patients ($p<0.001$).

The results demonstrated clearly impaired lumbar sensory and motor function in sciatica and LSS patients. The pure reflex activation of paraspinal muscles was not affected in sciatica but a difference was found in the premotoneuronal response control. The impaired proprioceptive functions and premotoneuronal response control seem to recover at least partially but the maintenance of postural stability is a complex activity which does not seem to recover automatically in operated sciatica patients at least in three months follow-up. Paraspinal muscle denervation and dysfunction were clearly detectable in LSS but lumbar paraspinal muscle endurance was unexpectedly good.

National Library of Medicine Classification: WE 750

Medical subject headings: low back pain; intervertebral disk displacement; spinal stenosis; electromyography; muscle, skeletal; evoked potentials, motor; evoked potentials, somatosensory; posture; psychomotor performance; comparative study; prospective study

To Leena

ACKNOWLEDGEMENTS

This work was carried out in the Department of Physical and Rehabilitation Medicine, Kuopio University Hospital and in the Department of Physiology, University of Kuopio in collaboration with the Departments of Neurosurgery and Clinical Neurophysiology, Kuopio University Hospital during the years 1999-2003.

I express my sincere gratitude to my principal supervisor Docent Olavi Airaksinen, M.D., Ph.D., Head of the Department of Physical and Rehabilitation Medicine, Kuopio University Hospital for enthusiastic support and guidance during this work. In addition to the never failing encouragement, he provided the necessary facilities for this study.

I am greatly impressed for the endless ideas of my supervisor, Professor Osmo Hänninen, M.D., Ph.D., Head of the Department of Physiology, University of Kuopio. I thank him for introducing me to the world of science and the encouraging discussions at any time of the day.

I am deeply grateful to my supervisor Docent Simo Taimela, M.D., Ph.D., CEO & Medical Director of DBC International Ltd., for his special expertise and essential help in this work. He always pointed out the key issues and confirmed the finalisation of the studies.

I express my warm thanks to Markku Kankaanpää, M.D., Ph.D., for introducing me this work and the constructive guidance especially in the initiation of the study.

I owe my sincere thanks to Docent Arto Herno, M.D., Ph.D., for interesting discussions and recruiting and managing the stenosis patients, Matti Luukkonen, M.D., for recruiting and managing the disc herniation patients.

I thank my other co-authors Sara Määttä, M.D. and Professor Juhani Partanen, M.D., Ph.D., Head of the Department of Clinical Neurophysiology, for interesting discussions and expertise in the field of neurophysiology and Martti Kansanen M.D., Ph.D. from the department of Otorhinolaryngology.

I thank Jari Arokoski, M.D., Ph.D., for interesting discussions and Sakari Savolainen, M.D., Ph.D., and Veli Turunen, M.D., for introducing me the spinal surgery. I owe sincere respect to Ville Westerlund and Tommi Kääriäinen for fruitful thoughts and continuing this work.

I wish to express special thanks to the entire staff of Departments of Physical and Rehabilitation Medicine, Physiology and Neurosurgery.

I thank all persons who participated to this study.

I thank the official referees of this study, Docent Heikki Hurri M.D., Ph.D., and Docent Uolevi Tolonen, M.D., Ph.D., for they thorough review and constructive criticism.

I thank Pirjo Halonen, M.Sc., for expert statistical assistance, Heikki Aalto, Ph.D., from the Department of Otorhinolaryngology, Helsinki University Central Hospital for his valuable help in the body sway analysis, Ewen MacDonald, Ph.D., for correcting the language of both this summary and the original publications. I owe a special thank to Mega Electronics Ltd., especially Juha Kylliäinen, M.Sc., Rainer Mustonen, M.Sc. and Kari Tiihonen, M.Sc., for their indispensable technical support.

I am grateful to all my co-workers and friends for their support and encouragement.

I express my deep feelings and gratitude to my parents Marja and Simo Leinonen and my parents in law Pirjo and Risto Hirvonen for loving support.

Finally, my deepest and warmest loving thank I give to my dear wife Leena whose love and support has kept me alive during these years and to whom I dedicate this thesis.

The research was financially supported by grants from Kuopio University Hospital, the Ministry of Education and Academy of Finland (TULES Graduate School), the Finnish Medical Society Duodecim, Savo Foundation for Advanced Technology and Finnish Cultural Foundation.

Kuopio, October, 2003 Ville Leinonen

ABBREVIATIONS

ANOVA	Analysis of variance
CL	Cortical latency
CLBP	Chronic low-back pain
CMRR	Common mode rejection ratio
CNS	Central nervous system
CPFV	Center point of force velocity
CT	Computed tomography
EEG	Electroencephalography
EMG	Electromyography
ES	Erector spinae
LBP	Low back pain
LSS	Lumbar spinal stenosis
MEG	Magnetoencephalography
MEP	Motor evoked potentials
MF	Multifidus
MRI	Magnetic resonance imaging
MPF	Mean power frequency
ODI	Oswestry disability index
PL	Peripheral latency
RBDS	Rimon's brief depression scale
RCT	Randomised controlled trial
ROS	Reactive oxygen species
SEP	Somatosensory evoked potentials
SD	Standard deviation
TNF	Tumour necrosis factor
VAS	Visual analogue scale

LIST OF ORIGINAL PUBLICATIONS

This thesis is based on the following original publications referred to in the text by the Roman numerals:

I Leinonen V, Kankaanpää M, Hänninen O, Airaksinen O, Taimela S. Paraspinal muscle responses during sudden upper limb loading. Eur J Appl Physiol 2002;88:42-9.

II Leinonen V, Kankaanpää M, Luukkonen M, Hänninen O, Airaksinen O, Taimela S. Disc herniation-related back pain impairs feed-forward control of paraspinal muscles. Spine 2001;26:E367-72.

III Leinonen V, Kankaanpää M, Luukkonen M, Kansanen M, Hänninen O, Airaksinen O, Taimela S. Lumbar paraspinal muscle function, perception of lumbar position and postural control in disc herniation-related back pain. Spine 2003;28:842-8.

IV Leinonen V, Määttä S, Taimela S, Herno A, Kankaanpää M, Partanen J, Kansanen M, Hänninen O, Airaksinen O. Impaired lumbar position sense in association with postural stability and motor and somatosensory evoked potential findings in lumbar spinal stenosis. Spine 2002;27:975-83.

V Leinonen V, Määttä S, Taimela S, Herno A, Kankaanpää M, Partanen J, Hänninen O, Airaksinen O. Paraspinal muscle denervation, paradoxically good lumbar endurance and abnormal flexion-extension cycle in lumbar spinal stenosis. Spine 2003;28:324-31.

CONTENTS

1 INTRODUCTION

Low back pain (LBP) is the most common musculo-skeletal disorder causing huge humanitarian and economical costs (Andersson 1999, Aromaa et al. 2002). The pain may arise from injured structures within the lumbar spine, however the anatomical cause and functional consequences of the LBP often remain undefined. In chronic pain, the precise aetiology is even less well understood. Imaging studies such as MRI visualise the macroscopic structural changes but similar changes are often found in healthy subjects and frequently the correlation with the clinical condition seems to be rather low (Jensen et al. 1994, Jarvik et al. 2001).

The chronic LBP is a multidimensional problem including pain and functional disability with its associated socioeconomical consequences. Impairments in neuromuscular control have often been associated with chronic LBP and are considered a probable link between pain and disability (Luoto 1999, Ebenbichler et al. 2001, Holm et al. 2002, Hodges and Moseley 2003, van Dieen et al. 2003b). There is increasing evidence that the impaired functions can recover with treatment and be restored by active rehabilitation (Ebenbichler et al. 2001).

The appropriate muscular control and movement perception is of vital importance in preventing low back injury. The protection against injury requires anticipation of events and adequate muscular responses. Both abnormal and missing protective reflexes could possibly lead to trauma or microtrauma of muscles, nerves, intervertebral discs and ligamentous spine during loading (McGill 1997).

The impaired neuromuscular control becomes particularly emphasised in conditions of chronic pain, which are also the most expensive for the society and the most demanding for the health care system. The etiological research is important for developing more effective treatment modalities especially in prolonged and chronic pain conditions. Unfortunately we have only a limited understanding of the time-course, appearance and clinical relevance of changes in motor control in LBP attributable to different pathophysiological mechanisms.

This study has investigated the postural control and lumbar motor and proprioceptive functions in specific lumbar disorders of disc herniation and lumbar spinal stenosis. The purpose was to evaluate the phenomena involved in motor control in LBP and patient groups with specific back disorders were used as experimental models.

2 GENERAL OVERVIEW

WHO has developed the International Classification of Functioning, Disability and Health (ICF) as a framework for the description of health and health-related states (WHO 2001). Functioning encompasses all body functions, activities and participation, and disability includes impairments, activity limitations and participation restrictions. These functions interact with environmental and personal factors defining the health condition (Figure 1). This taxonomy model can be used in the creation of assessment methods of lumbar disorders (WHO 2001). The current study is focused on "Sensory functions and pain" (b2) and "Neuromusculoskeletal and movement-related functions" (b7).

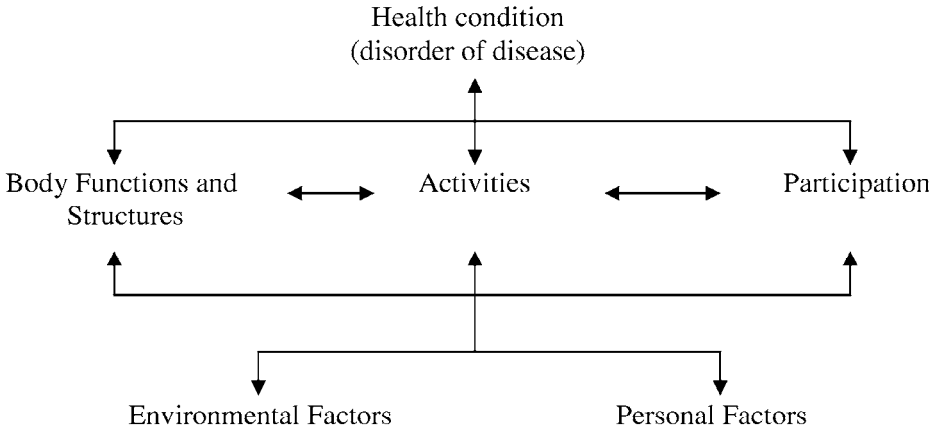

Figure 1. WHO model of interactions between the components of ICF.

Pain is defined as an unpleasant sensation and emotional experience associated with actual or potential tissue damage, or described in terms of such damage (IASP 1979, Merskey and Bogduk 1994). The acute pain usually eases during tissue healing or after the impending threat of tissue damage has passed. In conditions of chronic neuropathic pain, the sensory processing of the affected body region becomes abnormal (Watkins and Maier 2002) and there may become detectable changes in central information processing (Peyron et al. 2000, Juottonen et al. 2002, Farina et al. 2003) and altered pain experience.

Low back pain (LBP) is defined as pain in the lumbar area. It is a common problem which affects the majority of the population. The lifetime prevalence of LBP varies from 60 to 90 percent with an annual incidence of 5% (Frymoyer 1988, Aromaa et al. 2002). In the majority of cases, the back problems tend to show the first symptoms before the age of twenty (Leboef-Yde and Kyvik 1998). Usually the pain is acute and heals by itself in less than two months, but most of these cases will experience relapses with each episode becoming worse and worse. Approximately 5 to 10% of cases

become chronic, lasting over two months and creating a major medical challenge (Frymoyer 1988).

Despite the progress in diagnostic methods, the aetiology of back pain remains uncertain in most of the cases. It has been estimated that a precise diagnosis can be determined for less than 20% of low back pain patients (White and Gordon 1982, Nachemson 1985, Deyo and Weinstein 2001), depending partly on the interpretation of radiographic findings. Furthermore, the radiological and clinical findings and symptoms do not invariably correlate. As many as half of the asymptomatic subjects exhibited abnormal changes in their discs according to the MRI study by Jensen et al. (1994) and the MRI findings alone have only limited importance (Videman et al. 2003).

Nerve-root symptoms occur only in one percent of acute LBP patients, but they are often associated with enduring and persistent pain. Sciatica is defined as pain in a distribution area of a lumbar nerve root, often accompanied by sensory and motor deficits. Sciatic pain requires that there must be mechanical and inflammatory stimuli to lumbar nerve roots. The most common cause of sciatica is a herniated lumbar disc but in the ageing population, there is now an increased prevalence of spinal stenosis as a cause of sciatica (Frymoyer 1988).

This study defines the LBP syndrome as a multidimensional problem including pain, functional disability and socioeconomical consequences. The low back syndrome seems to be a continuous process, and the functional disorders vary with respect to the stage and duration of the illness and physical and psychological stress. However, the time course of the LBP and the relation between time and symptoms are poorly understood. According to experimental studies, the spatial or temporal overloading of spinal structures leads to micro-injuries, inflammation, pain and neuromuscular dysfunction (Claude et al. 2003, Solomonow et al. 2003a). This dysfunction has been claimed to be associated with clinical pain (Indahl 1999, Solomonow et al. 2003b). Although in chronic pain, the psychosocial component often seems to be important, the origin of the syndrome is organic and thus an evaluation of spinal function is of clear value.

3 REVIEW OF THE LITERATURE

3.1 Lumbar disorders

Classification of lumbar disorders

Classification of the LBP is still a challenging problem. Lumbar diseases are often divided into specific and non-specific conditions. In non-specific (idiopathic) cases the origin of the pain is unknown, however, there are several potential sources of pain like muscles, tendons, ligaments, nerves, discs etc. The (rare) specific spinal diseases are attributable to fractures, tumours and infections etc. The more common specific conditions are disc herniation and lumbar spinal stenosis, which are the main causes of sciatica (Frymoyer 1988, Deyo and Weinstein 2001). The LBP is also divided by its duration, usually the pain lasting less than 6 weeks is called acute, 6-12 weeks is subacute and over 3 months is chronic LBP (Bigos et al. 1994).

3.1.1 Low back pain

3.1.1.1 Pathophysiology

The underlying mechanisms of LBP are usually unknown but there are several potential sources and causes of pain. Currently, there seems to be only concepts but little hard evidence for the development of the LBP. One potential course of the syndrome could be as follows: first the micro-injury e.g. to the muscles, tendons, ligaments, intervertebral disc or endplate. Injury is followed by macrophage and neutrophil accumulation and inflammation (Ahn et al. 2002, Burke et al. 2002). Static lumbar flexion has been indicated to cause the development of creep and micro-injuries of the collageneous structures in the lumbar ligaments. This elicits acute inflammation and hyperexcitability of the multifidus muscle (Claude et al. 2003, Solomonow et al. 2003a). This reflexive muscular stiffness is thought to protect the injured structures and enabling tissue healing (Claude et al. 2003, van Dieen et al. 2003, Solomonow et al. 2003a).

Reactive oxygen species (ROS) seem to contribute to muscle fatigue (Reid 2000) and therefore they may have role also in the pathophysiology of LBP. Dorsal ramus neuropathy is thought to be one potential cause of LBP (Sihvonen 1995). Experimental studies have indicated that pain arising from the passive or active structures can lead to pathological activation of the paraspinal muscles at the same and adjacent segments (Indahl 1999, Solomonow et al. 2003a). Despite this important finding, one still requires unequivocal clinical evidence about these protective reflexes. In the clinical course of a long-lasting spinal disorder, ultimately, the chronic pain may lead to deconditioning of the muscles and neural modulation of the central nervous system (Flor et al. 1997, Kankaanpää 1999, Luoto 1999).

3.1.1.2 Symptoms and findings

The primary diagnostic evaluation includes perusal of the medical history and a physical examination. This aims to exclude or diagnose serious causes of pain and nerve root compression. In addition, the social and psychological factors causing distress possibly complicating the recovery should be clarified (Bigos et al. 1994, Waddell et al.

1996, Deyo and Weinstein 2001). Patients suffering prolonged pain and when there is suspicion of a serious illness or persisting radicular symptoms need further evaluation.

In the acute condition, imaging is often unnecessary (Deyo and Weinstein 2001). The plain radiograph is currently the basic examination procedure for prolonged LBP lasting over six weeks (Jarvik and Deyo 2002). The primary supplementary examination is MRI, which reveals also macroscopic soft tissue changes (Herzog et al. 1995) and can even be considered as the primary procedure in the patients with radicular symptoms when surgery is potentially indicated. Disc degeneration is a usual finding also in asymptomatic subjects (Boden et al. 1990, Tertti et al. 1991, Jensen et al. 1994, Jarvik et al. 2001) and does not appear to have a cause-effect relationship with pain. Disc disruptions seems to have clinical significance (Kuslich et al. 1991, Moneta et al. 1994), but the clinical relevance of the indicative MRI findings of the disc disruptions is rather weak (Videman et al. 2003) and the need for their invasive evaluation (discography) and treatment is not clear. Therefore, evaluation of the functional ability is often more important than anatomic findings (Bigos et al. 1994, Waddell et al. 1996, Deyo and Weinstein 2001).

3.1.1.3 Treatment and prognosis

LBP is not a self-limiting condition (Hestbaek et al. 2003). Effective pain medication is essential in the acute condition, and it may prevent the pain from becoming chronic (Bigos et al. 1994, Waddell 1996, van Tulder et al. 1997). It is important to inform the patient about the benign nature of the syndrome, the need to avoid bed rest and to encourage the patient to maintain normal daily activities (Malmivaara et al. 1995). In acute benign pain it is crucial that the diagnosis and interpretation of the radiological findings are undertaken to ensure that the patients do not progress to illness behaviour and fear-avoidance (Koes et al. 2001), which are often complicating factors promoting chronic LBP and disability.

In prolonged pain, activating procedures are recommended (van Tulder 1997), and in the case of radiating pain surgeon should be consulted at the latest after six weeks of observation (Deyo and Weinstein 2001). The patient should be encouraged to exercise and make active use of the back. Spinal manipulation and physical therapy are potential alternatives to treatment (Deyo and Weinstein 2001). If not started before, then an active rehabilitation process including guided and self-motivated exercises should be added to the treatment of pain lasting over six weeks. In chronic pain, a comprehensive evaluation of the condition and an intensive integrated rehabilitation should be undertaken (Bigos et al. 1994, Guzman et al. 2002).

The good prognosis of the acute pain should be emphasised from the beginning but also the patient should be informed about the frequent recurrence of the pain. Only five to 10 percent of the cases become chronic, lasting over three months but those cases result in the most severe medical and economical hardship.

3.1.2 Disc herniation

3.1.2.1 Pathophysiology

In the herniated disc, the nucleus pulposus has gone through the ruptured annulus fibrosus. Disc herniation causes mechanical compression of the nerve roots and

chemical irritation mainly by activation of the inflammatory processes (Kuslich et al. 1991, Olmarker et al. 1993). The tumor necrosis factor-alpha (Olmarker and Larsson 1998, Olmarker and Rydevik 2001) and several other cytokines (Watkins and Maier 2002) appear to be clearly associated with the inflammatory process in sciatica. The use of specific anti-TNF-α treatment may alter the treatment strategy of acute sciatica in the future (Watkins and Maier 2002, Karppinen et al. 2003).

3.1.2.2 Symptoms and findings

The disc rupture itself can be painful and, in addition to the local LBP, they can also mimic the radicular symptoms evoked by a herniated disc (Karppinen et al. 2001). The radicular symptoms include pain, numbness, sensory disturbances, paresthesia and motor weakness. In severe cases (cauda equina syndrome) urinary retention, anal incontinence or sensory loss in a saddle distribution may be present (Frymoyer 1988). The disc herniation is not visible in the plain radiograph but can be detected by MRI (Herzog et al. 1995) or optionally by CT.

3.1.2.3 Treatment and prognosis

The radicular symptoms usually disappear in three months by conservative treatment. In those cases with persistant severe symptoms over six weeks with a clear radiological finding, surgery is usually considered. Intolerable pain, cauda equina syndrome or acute paresis/paralysis may require emergency surgery (Kostuik et al. 1986, Bigos et al. 1994). Due to the good spontaneous recovery, only approximately ten percent of the patients need to undergo surgery (Frymoyer 1988), however, the result of surgery is better if it is initiated within three months from the beginning of the symptoms (Hurme and Alaranta 1987). One year after surgery 50-85% of the operated patients are free of sciatica (Hoffman et al. 1993, Junge et al. 1995). However, there is a considerable recurrence of the syndrome. The need for re-operation may be up to 20% during 13 years follow-up (Nykvist et al. 1995), according to Finnish Hospital Discharge Register 14% of patients needed re-operation during a 10-year follow-up (Österman et al. 2003).

According to an early randomised trial comparing surgery and conservative treatment, the surgery was more effective for two years follow-up, but from two until ten years the results were statistically equivalent (Weber 1983), though a later reanalysis has found a slight benefit favouring surgery also during a longer follow-up. The recent RCT showed faster recovery after surgery but only minor differences between conservative care and surgery at two-year follow-up (Österman et al. 2002). The current conservative treatment includes pain medication and patient information. Bed rest should be avoided and the patient should be encouraged to resume normal daily activities (within the limits imposed by the pain) as in non-specific LBP. New promising treatments, such as therapy with TNF-alpha antibodies, are also on the horizon (Karppinen et al. 2003).

3.1.3 Lumbar spinal stenosis

LSS is a notable degenerative disorder of the lumbar spine, responsible for low back and lower extremity pain. The symptoms of the disorder arise from compression of the cauda equina due to degenerative processes leading to anatomical stenosis of the lumbar spinal canal and intervertebral foramina (Verbiest 1954).

3.1.3.1 Pathophysiology

The anatomical narrowness of spinal canal is the basic aetiology for LSS but the syndrome is caused by degeneration of lumbar spine leading to facet arthrosis and disc degeneration with osteophytes and thickening and calcification of ligamentum flavum (Rauschning 1993, Fritz et al. 1998, Spivak 1998). The pathophysiology of the symptoms is poorly known, but it is thought that the mechanical compression irritates the nerve roots and causes the symptoms of the disease. However, the symptoms do not clearly correlate with the degree of the stenosis, and there is a considerable fluctuation in the symptoms (Amundsen et al. 1995, Jönsson et al. 1997). One potential explanation is vascular compression theory based on the venous congestion caused by the mechanical compression of the cauda equina veins, leading to decreased blood flow and subsequent dysfunction of the lumbar nerve roots (Porter 2000). Recent experimental studies have supported that theory by indicating ectopic firing caused by venous stasis of lumbar nerve roots and inhibition of ectopic firing after decompression (Ikawa et al. 2003).

3.1.3.2 Symptoms and findings

The symptoms include LBP, intermittent claudication and radicular symptoms such as pain, numbness, weakness and loss of sensation in the lower limbs. Claudication and radicular symptoms are often provocated by spine extension and revealed by flexion and the claudication eases slowly, when the subject stops walking (Spivak 1998). The plain radiograph can indicate LSS but the diagnosis is based on MRI or CT findings correlating with the symptoms (Katz et al. 1994).

3.1.3.3 Treatment and prognosis

After diagnosis of LSS, the options are follow-up, conservative treatment or surgery. The mild symptoms are usually treated conservatively, but severe stenosis often requires decompressive surgery. Conservative treatment includes analgesic medication, physical therapy, spinal support and calcitonin therapy (Eskola et al. 1992), however, there is still need for the RCT based evidence on the effect of conservative treatment modalities. If the symptoms and disability are intolerable despite conservative treatment, then surgery is indicated. The symptoms are eased, and the extent of disability decreased after surgery (Atlas et al. 2000), but the long-term outcome shows large variation (Herno 1995, Hurri et al. 1998). The natural course of LSS is unclear but according to a recent RCT, surgery seems to be better than conservative care in moderately severe LSS at one-year follow-up (Slätis et al. 2002).

3.2 Motor control

3.2.1 Classification of motor control to voluntary and automatic actions

Human motor control includes schematically three different co-operating levels (Figure 2). The highest level is planning of the movement, the lowest level is spinal reflexes, and the intermediate level connects the highest and lowest levels for the execution of the task (Kalaska and Drew 1993). The terms voluntary and reflex are widely used in scientific papers and in everyday conversation, but the exact meaning of

these terms is not clear and need some definition (Prochazka et al. 2000). This study defines focused attention encompassing conscious (voluntary) control and subsidiary activation encompassing automatic motor programs (including reflexes). Isolated voluntary and automatic actions are rarely seen in real life, the normal functions are combinations of different levels of control. Reflexes assist in voluntary functions and cortical activation may regulate reflexes. The primary motor cortex is involved in reflex inhibition via the descending pathways, which vary their activity level according to the attention, arousal and emotional state of the subject.

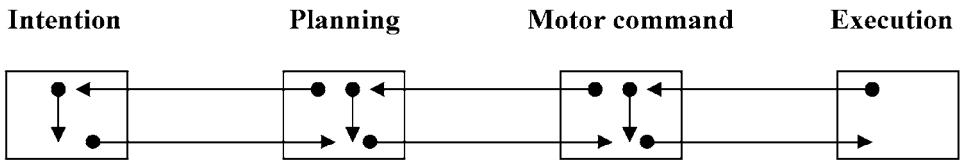

Figure 2. The concept of motor control.

3.2.2 Focused attention

Voluntary movements are programmed in the brain. Several cortical areas e.g. supplementary motor cortex, basal ganglia, cerebellum, thalamus, anterior gyrus cinguli and primary and supplementary somatosensory cortex are linked to the primary motor cortex. These centres are involved in the planning of the movement. The final command to perform the motion is initiated by the pyramidal cells of primary motor cortex and transmitted through the corticospinal pathway to the ventral horn of the spinal cord. At a specific spinal level, the upper motor neuron synapses with the alpha motor neuron which then transmits the command to muscle fibers of its own motor unit (Rothwell 1994).

3.2.3 Subsidiary movement

The subsidiary muscle activations are often associated with voluntary movements and are function of servo actions needed e.g. in postural control (Rothwell 1994). These actions include anticipatory postural adjustments and are usually automatic i.e. unconsciously controlled but are more complex than simple reflexes. The responsible level controlling these fast actions is intermediate, also called the extrapyramidal system. In addition to thalamus and basal nerve nuclei, this involve the cerebellum and basal ganglia, which are involved in the tuning of the fine movements, coordination, timing, motor learning and muscle tone.

Automatic motor programs

Reflexes are the fastest way to control movements. They are categorically divided into mono-, di- and polysynaptic reflexes. The monosynaptic reflex is the simplest and

fastest of the three reflex types. The electrical reflex latency varies from ~10 ms in spinal muscles to ~50 ms in distal lower limb muscles depending on the distance of respective muscle from the spinal cord or cranial nerve nuclei. The latency consists of the sensory impulse conduction in sensory neurons, synaptic transmission and motor impulse conduction in the motor neurons (Rothwell 1994, Schmidt and Lee 1999). In addition, there is a notable time lag from the electrical activation of the muscle cells to the mechanical force produced by the muscle (Rothwell 1994, Schmidt and Lee 1999). In complex responses, the relative time for information processing is longer.

In the simple monosynaptic stretch reflex, muscle spindles sense the stretching of the muscle fibres. The information is mediated by the Ia sensory afferent nerves into the dorsal horn at the respective level of the spinal cord. These sensory nerve cells synapse with the motor neurons activating the corresponding muscle. In more complex reflexes, an increasing number of interneurons are involved in mediating the reflex (Rothwell 1994). The muscle activation is modulated also by the gamma motor neuron system. These types of reflexes are usually involved in voluntary and subsidiary movements e.g. permitting the fluency of movements by controlling the agonist-antagonist muscle activation.

3.3 Postural control

The equilibrium of the body is essential for locomotion and performing other types of limb movements. The purpose of the postural control system is to support, stabilize and balance with the aim of maintaining postural stability. Postural control functions at three tightly connected levels i.e. postural reflexes, triggered reactions and voluntary movements these being listed from fastest to slowest level of action and from the lowest to highest level of cognition, respectively. The somatosensory, vestibular and visual receptors provide the sensory information needed in this process (Rothwell 1994). The sensory information is transmitted by the ascending pathways of the spinal cord and the motor commands are transmitted by the descending pathways which synapse with the lower motor neurons. The major descending pathways controlling body posture are the vestibulospinal tracts. The neurons of the vestibulospinal tracts synapse with the same alpha motor neuron as the corticospinal tract (Rothwell 1994).

Postural responses are modulated by feedback information but also pre-programmed functions are needed (Schmidt and Lee 1999, Hodges 2003). The cerebellum is important in maintaining the balance and adaptation and learning of postural reflexes and feed-forward responses (Horak and Diener 1994, Rothwell 1994). Postural control is a complex procedure and vulnerable to disruption by a wide variety of disorders (Horak and Nashner 1986, Rothwell 1994). In addition to the neurological and vestibular diseases, postural control plays also a significant role in several musculo-skeletal impairments including back and joint disorders (Alaranta et al. 1994). Increasing age starts to have detrimental effects on postural control after the fifth decade of life (Alaranta et al. 1994, Aalto 1997).

3.4 Motor control of the lumbar spine

Lumbar spinal anatomy has been described in detail by Bogduk and Twomey (1991) and reviewed in the association with LBP by Indahl (1999) and Kankaanpää (1999). According to Panjabi (1992), the spinal stability system consists of three subsystems, which are the passive spinal column, the active spinal muscles and the neural control unit. An overloading or dysfunction of any of these subsystems may lead to injury if there is a failure of compensation mechanisms (Panjabi 1992). Intra-abdominal pressure, diaphragm, pelvic floor and abdominal muscle activation have been shown to have a significant role in spinal control (Hodges 2003).

The simple stability and instability concept in the low back syndrome has recently been challenged and the importance of neural control system has been emphasised. Hodges (2003) has shown that CNS does not simply stiffen the spine and restrict the spinal motion, but actively uses movements to maintain equilibrium in the posture. Lund (2003) has indicated that mechanical LBP is associated with restricted but painful motion rather than mechanical instability. This indicates that a potential pathomechanism of "the instability" is dysfunction of neuromuscular control system.

3.4.1 Segmental function of the lumbar spine

Two vertebrae, their intervertebral disc and facet joints form the functional spinal unit (FSU, Pope et al. 1993). The ligamento-muscular reflex between multifidus muscles, zygapophysial joints and intervertebral ligaments has been demonstrated in the control of the intricate neuromuscular balance in the lumbar motion segment (Solomonow et al. 1998, Indahl 1999). The possible interactive responses between injured or diseased spinal structures, i.e., disc or facet joints, and the paraspinal musculature may lead to segmental dysfunction of the lumbar spine (Sihvonen et al. 1991, Indahl et al. 1995 and 1997, Kaigle et al. 1998).

3.4.2 Kinetic chain

Thoracolumbar fascia connects the spine, upper limbs and pelvis and via the sacrotuberous ligament the pelvis to the lower limbs. This allows dynamic load transfer from spine to legs (Vleeming et al. 1995). Paraspinal, gluteal and hamstring muscles exhibit sequential activation during sagittal trunk flexion and extension (Paquet et al. 1994, Leinonen et al. 2000). Dysfunction of any part of the integrated system may lead to abnormal load transfer between low back and pelvis. Therefore, it seems rational that the hip extensor muscles, in addition to the spinal and abdominal musculature, will be subjected to deconditioning in CLBP patients, if the use of these muscles is avoided (Kankaanpää et al. 1998).

3.4.3 Feedback control

The appropriate proprioceptive information is essential for motor control. In unpredictable postural perturbation, the control strategy is based on sensory information transmitted from the receptive structures and during intended movements, feedback

information is needed for error correction (Schmidt and Lee 1999). The proprioceptive information of the body and limb movements originate from the muscle spindles, the Golgi tendon organs, joint and the cutaneous receptors (Rothwell 1994, Schmidt and Lee 1998). Muscle receptors seem to play a major role in joint position sense (McCloskey 1978, Pedersen et al. 1998). The increased repositioning error after paraspinal muscle vibration and impaired lumbar position sense after lumbar paraspinal muscle fatigue emphasize the importance of muscle spindles in the positioning of lumbosacral spine (Brumagne et al. 1999 and 2000, Taimela et al. 1999).

Tactile sensations and conscious proprioceptive information about the body are mediated to the somatosensory cortex via the dorsal columns, the fasciculus gracilis from lower limbs and the lower part of the body and by the fasciculus cuneatus from the upper limbs and the upper part of the body. The dorsal spinocerebellar and probably also the spinothalamic tracts transmit the proprioceptive information needed in the maintenance of postural stability (Rothwell 1994). The precise neurophysiological mechanisms processing the proprioceptive input in conscious sensory perception remain to be determined but probably involve coordinated inputs from cerebellum, thalamus and somatosensory cortex.

3.4.4 Feed-forward control

In predictable postural perturbation, the CNS can plan control strategies in advance. According to the recognition schema theory, muscle activation can be pre-programmed based on initial condition, environmental outcome and expected sensory consequences (Schmidt 1975, Schmidt and Lee 1999).

Upper (Cordo and Nashner 1982, Zattara and Bouisset 1988, Hodges and Richardson 1996) or lower (Hodges and Richardson 1998) limb voluntary movements evoke non-conscious muscle activation in the trunk muscles via a feed-forward mechanism. This refers to the activities of the central movement control system, which maintains postural stability and prepares the trunk to bear a potentially increasing load by activating certain trunk muscles. These trunk muscles which maintain the dynamic spine stability, are activated before the activation of the prime muscles responsible for the gross limb movement without an afferent input from the respective trunk movement (Belen'kii et al. 1967, Cordo and Nashner 1982, Friedli et al. 1988, Zattara and Bouisset 1988, Aruin and Latash 1995, Hodges et al. 1999).

3.5 Measurements of lumbar function

The clinical examination include inspection, palpation, range of spinal motion, testing of lower limb sensory function, muscles and reflexes. These examinations with the functional and provocation tests form the basics of the evaluation of lumbar function in LBP (Deyo and Weinstein 2001). The test results are usually non-specific and evaluated subjectively, but their repeatability is acceptable and they are very useful, especially when they are correlating with the subjective symptoms. Neurophysiologic assessments include neurography, electromyographic examination and motor and somatosensory evoked potential measurements. They are used in the assessment of nerve injury and in the diagnosis of the level of nerve injury (Dvorak et al. 2000).

Several functional measurements can be used in the study of LBP. Surface electromyography measurements are used in the assessment of muscle function (Sihvonen 1995) and endurance (Kankaanpää 1999). Muscle strength (Rantanen 2001) and lumbar kinematics are measured with a wide variety of methods (Lund 2003). Postural control can be measured on a force-platform either with or without simultaneous EMG and trunk motion measurements (Luoto 1999). They have been mainly used in assessing physiological and pathophysiological mechanisms of lumbar function (Hodges 2003). The test results usually overlap with values obtained from back healthy subjects. Therefore they are not of any great benefit in the diagnosis but can be used for example in the planning of rehabilitation procedures and in the objective evaluation of the effectiveness of the rehabilitation. The diagnostic value of the functional measurement is often rather poor and their clinical use is also hindered by a lack of standardisation.

3.6 Pain and sensory-motor control

3.6.1 Pain and proprioception

Impaired lumbar proprioception is associated with LBP (Taimela et al. 1999). An increased repositioning error during trunk movement has been observed in LBP patients (Gill and Callaghan 1998, Brumagne et al. 2000, Newcomer et al. 2000). Paraspinal muscle spindles seem to be important in the correct positioning of the lumbosacral spine and the muscle spindle input may be decreased in lumbar pain (Brumagne et al. 1999, Taimela et al. 1999). In addition, experimental muscle pain has been shown to affect the central modulation of proprioceptive signals of jaw muscle spindles (Capra and Ro 2000). However, there is experimental evidence that the nociceptive input can actually enhance the central sensitivity to the mechanoreceptor input (Torebjörk et al. 1992).

3.6.2 Pain and trunk muscle function

Pain has clear effects on motor control. LBP can induce changes in neuromuscular control and motor performance. The function and co-ordination of the muscles stabilising the lumbar spine seem to be impaired in patients suffering from LBP (Hodges et al. 1996, Wilder et al. 1996). Delayed or absent trunk muscle activation has been observed in low back pain patients during upper (Hodges and Richardson 1996) and lower limb movements (Hodges and Richardson 1998) and after experimental pain (Hodges et al. 2003). A different muscle response pattern to sudden load release (Radebold et al. 2000 and 2001) and during expected and unexpected upper limb and trunk loading (Wilder et al. 1996, Magnusson et al. 1996) has been found in CLBP patients compared with healthy controls.

Psychosocial stress can change spinal muscle activation and loading. However, notable individual differences have been observed in its importance (Marras et al. 2000, Davis et al. 2002). CLBP patients have been claimed to exhibit an increased paraspinal (Roy et al. 1989) and gluteal (Kankaanpää et al. 1998) muscle fatigability. During trunk flexion-extension, CLBP patients had increased paraspinal muscle activity when acting as antagonist (Ahern et al. 1988, Sihvonen et al. 1991, Sihvonen 1995) and decreased

activity when acting as agonist (Sihvonen et al. 1991), with similar activation patterns being observed during experimental pain (Zedka et al. 1999). The CLBP and experimental muscle pain induced increased paraspinal muscle activity in the normally silent period and decreased activity in the normally active period of the gait (Arendt-Nielsen et al. 1996).

3.6.3 LBP and general motor control

The impaired postural control is associated with chronic lumbar disorders (Byl and Sinnot 1991, Luoto et al. 1996). A slow psychomotor reaction time is also associated with back pain (Taimela et al. 1993). Poor postural and psychomotor control (Taimela 1992) is a known risk factor for accidental injuries, but in lumbar disorders it is not yet known whether the poor postural control is a consequence of pain or neuromuscular impairment or a potential major source of the disorder. Thus, there appears to be little prospective evidence on whether the impaired motor control is pre-existing (risk factor) or a secondary phenomenon (consequence) of the musculoskeletal disorder. The muscle dysfunction and deconditioning seem to be rather a consequence of the tissue injury and pain. However, this claim needs further prospective evaluation.

Pain seems to decrease the muscle activation amplitude during voluntary contractions and increase the muscle activation during automatic contractions (Zedka et al. 1999). One potential explanation for this is the hypothesis based on the model of fear-avoidance behaviour, where the decrease in central output is revealed as decreased primary motor activation and decreased inhibition of reflexes.

Active physical rehabilitation has been claimed to restore the impaired neuromuscular functions (Wilder et al. 1996, Kankaanpää et al. 1999). However, impaired postural control failed to improve after rehabilitation and after unsuccessful treatment the condition may even deteriorate (Luoto et al. 1998). Despite the widespread acceptance of the importance of stabilising functions of the spine, there is still a need of evidence based on randomised controlled trials on the effect of stabilising exercises in LBP.

In conclusion extensive variety of changes in motor and sensory control and trunk muscle function have been reported. However, they occurrence and clinical relevance in LBP with different pathophysiological causes remains undefined. In addition, the time-course of the changes and the effect of interventions are rarely evaluated.

4 AIMS OF THE STUDY

The purpose of this study was to evaluate involuntary motor control in patients with specific lumbar disorders of disc herniation and lumbar spinal stenosis in relation to healthy controls.

The specific aims

To investigate the latency and magnitude of the reflex responses in paraspinal muscles after unexpected and expected upper limb loading and to investigate the effect of anticipation and external spinal support on these responses
- in healthy subjects
- in patients with disc herniation before and after discectomy surgery

To assess lumbar movement perception
- in healthy subjects
- in patients with disc herniation before and after discectomy surgery
- in patients with LSS

To evaluate postural stability
- in healthy subjects
- in patients with disc herniation before and after discectomy surgery
- in patients with LSS

To determine paraspinal muscle function, innervation and endurance in patients with LSS

5 SUBJECTS AND METHODS

5.1 Subjects

Total of 81 subjects volunteered to participate in the study (Table 1). All subjects provided informed consent prior to their participation. The study was approved by the Kuopio University Hospital Research Ethics Board, and it was performed according to the Declaration of Helsinki. The subjects had not had any previous spine surgery or cervical radicular symptoms. Additional exclusion criteria were neurological, metabolic or severe cardiovascular disease.

Table 1. Characteristics of the subjects

	Healthy subjects		Sciatica patients	LSS
Study	I	II, III	II, III	IV, V
Number of subjects	20	15	20	26
Age (y)	23±1	37±12	39±10	56±7
Height (cm)	173±10	175±9	175±7	168±10
Weight (kg)	68±11	74±13	78±16	79±14
Disability index (ODI 0-100)			38.4±17	24.7±7
Depression score (RBDS 0-21)			5.6±4.4	5.0±4
LBP intensity (VAS 0-100)			60.8±19	44.6±23
Leg pain intensity (VAS 0-100)			64.2±25	39.6±29

5.1.1 Healthy subjects

Thirty-five healthy subjects were recruited in the study (I-III).

5.1.2 Patients with herniated lumbar disc

The study included 20 patients selected for surgery due to lumbar disc herniation (15 males and 5 females; II, III). The patients underwent microdiscectomy performed by a neurosurgeon (III). Baseline measurements were done one day before the surgery and the follow-up measurements 3 months after the surgery (II, III).

5.1.3 Patients with lumbar spinal stenosis

The study included 26 LSS patients (11 men, 15 women; IV, V). The diagnosis was based on the patients' symptoms and signs and was confirmed by computed tomography (CT) or magnetic resonance imaging (MRI).

5.2 Assessment of paraspinal muscle function

5.2.1 Surface EMG

Bipolar surface electromyography (EMG) was recorded bilaterally over the paraspinal muscles at T12-L1 and L5-S1 levels by a four channel ME 3000P EMG system (Mega Electronics Ltd, Kuopio, Finland) with disposable Ag/AgCl surface electrodes (Medicotest, Olstykke, Denmark). The electrodes were placed on the erector spinae (ES, T12-L1 level) and multifidus (MF, L5-S1 level) muscles as recommended by Biedermann et al. (1991). The raw EMG signal was recorded at the sampling rate of 2 kHz and band-pass filtered between 7-500 Hz with an analogue filter, amplified (differential amplifier, CMRR>110 dB, gain 1000, noise<1μV), analogue-to-digital converted (12-bit), and stored in a personal computer for later analysis. (I-III, V)

5.2.2 Paraspinal muscle responses for sudden upper limb loading

Back muscle reaction time for unexpected (eyes closed) and expected (eyes open) upper limb loading was measured by a method modified from one previously described (Wilder et al. 1996). During the procedure, the subject was standing and holding a box in the hands while a weight of 0.68 kg was suddenly dropped from the height of the subject's eyes into the box equipped with a marker switch indicating the moment of impact. Twelve consecutive measurements were performed in sequences of three trials with the eyes open and three with the eyes closed in supported (first six) and unsupported (last six) standing positions (I-III).

The muscle activation onsets and offsets were determined visually from the rectified EMG. The determination was made without reference points in order to exclude observer bias (I-III).

5.2.3 Dynamic function

Subjects performed sagittal trunk flexion and extension in the standing position with knees extended and feet 10 cm apart while raw surface EMG was recorded bilaterally over the paraspinal muscles from the abovementioned placements. The subjects were instructed to flex their body to the limit of full flexion and to extend back to the upright position (forward flexion and extension lasting from 5 to 10 seconds, Leinonen et al. 2000). The muscle activation amplitudes were calculated from a 1 sec period during the flexion movement, full flexion and extension movement, respectively. The full-trunk flexion and extension activation were related with flexion activation modified from the previously described method (Ahern et al. 1988, Watson et al. 1997). The range of motion was determined by an accelerometer/goniometer (Mega Electronics Ltd, Kuopio, Finland) placed on the T6 vertebra. (V)

5.2.4 Lumbar endurance

The back muscle endurance was measured by a previously demonstrated isoinertial back endurance test (Kankaanpää et al. 1997, 1999). Briefly, subjects were seated in a

back extensor training unit (DBC International, Helsinki, Finland), which the fixation mechanism (lower limbs and hips were mechanically bound) focused the dynamic movement to the upper back. The subjects performed repetitive upper trunk extensions with a continuous isoinertial load (30 repetitions per min controlled by a metronome) and the subjects were instructed to hold the tension at both ends of the movement range. The hyperextension of the spine was prevented to avoid the provocation of claudication symptoms. The test was performed until exhaustion or pain, and the endurance time was measured.

Spectral mean power frequency (MPF) slopes of the first 90 seconds of the endurance time were calculated by performing a fast Fourier transformation to assess the muscle fatigue (percentage change of MPF per minute). The average MPF was calculated from the first 5 seconds of the performance (initial MPF). The LSS patients (pooled MPF change and initial MPF at L5-S1 level) were compared with the subjects of previous studies by t-test [10 healthy controls (Kankaanpää et al. 1997) and also with 59 chronic LBP patients who did not suffer any symptoms of LSS (Kankaanpää et al. 1999)]. (V)

5.2.5 Muscle denervation

Electromyography (EMG) of the paraspinal muscles was performed at the L3 to S1 levels bilaterally with Viking IV EMG equipment (Dantec, USA) using a concentric needle electrode (Neuroline, 50x0.45 mm). Amplification was set at 50 μV/div and the high and low-pass filters at 10 kHz and 20 Hz, respectively.

At least 20 insertions were analysed from each multifidus muscle and the aim of the examination was to detect any abnormal spontaneous activity indicative of a lower motor neuron disorder and thus as a sign of denervation. Abnormal activities indicative of denervation were considered to be fibrillation potentials, positive sharp waves and complex repetitive discharges (Kimura 2001). The analysis was performed on-line (V).

5.3 Lumbar movement perception

Lumbar proprioception was assessed in a previously described trunk rotation measurement unit (DBC International Ltd, Helsinki, Finland; Taimela et al. 1999), which targeted the rotation on the lumbar/thoracic spine. In the test, the subject was placed in the device in the seated position, ears and eyes covered, while the seat was rotated with an angular velocity of 1°/sec and the subject indicated the initiation of the movement by releasing a finger switch. The magnitude of the lumbar rotation was recorded. In addition, the subject was asked to indicate the direction of movement. The results of five consecutive trials were pooled. (III,IV)

5.4 Postural stability

Postural control was measured with a vertical force platform (Smart Balance Master, NeuroCom^{R}, Clackamas, OR, USA), which is based on the principle introduced by Terekhov (1974). The load cell signals were recorded by a personal computer with a sampling frequency of 50 Hz and were low-pass filtered. The postural control was

evaluated during eight 20 sec trials (one two-footed trial with eyes open and one with eyes closed; six one-footed trials, of which three were performed while standing with the right foot and three while standing with the left foot. The center point of force velocity (CPFV, cm/sec) was calculated for each trial (III, IV).

5.5 Motor and somatosensory evoked potentials

MEPs were elicited by transcranial and lumbar root stimulation using a MES-10 magnetic stimulator (Cadwell Laboratories Inc., Kennewick, WA, USA) with a circular 9-cm-diameter coil producing 2 Tesla. The motor responses and SEPs were recorded by using standard EMG equipment (Nicolet Viking I, Dantec, Wisconsin, USA). The responses were high and low-pass filtered.

MEPs were recorded bilaterally from the anterior tibial muscles using bipolar Ag-AgCl cup electrodes. The latencies of the MEP after cortical stimulation (cortical latency, CL) and after lumbar root stimulation (peripheral latency, PL) were measured from the response with the shortest latency.

During the SEP procedure, the tibial nerves were stimulated at the ankle behind the medial malleolus with a surface electrode. The cortical P40-N50 responses were recorded 2 cm posterior from the vertex referenced to the forehead with platinum needle electrodes. Peripheral responses were recorded from the popliteal space with Ag/AgCl cup electrodes. P40 latencies and P40-N50 amplitudes were measured.

The MEP and SEP latencies were compared with the laboratory reference values. The P-value was used as a measure of normality and $p<0.05$ was classified as abnormal. SEP amplitudes <1 μV were also considered as abnormal. (IV)

5.6 Questionnaires and clinical signs

Low back and lower extremity pain intensities were determined by Visual Analogue Scale (VAS, Scott and Huskinsson 1976) and functional disability by Oswestry disability index (ODI, Fairbanks 1980). The depressive symptoms were evaluated by Rimon's brief depression scale questionnaire (RBDS, Keltikangas-Järvinen and Rimon 1987). Clinically observed sensory deficit, motor weakness and limited range of movement (fingertips over the level of the knee during forward bending) were recorded. (II-V)

5.7 Statistical analysis

A repeated measures analysis of variance with three (four) within factors [control of position, level and expectance (and operation)] was used to analyze the effects of supported position, measurement level (ES, T12-L1 and MF, L5-S1), anticipation (and operation) for the short latency response of paraspinal muscles (I-III). The degree of lumbar rotation and CPFV were compared in the patients and controls by independent samples t-test (III), and the effect of surgery was assessed by paired t-test (III). Correlation coefficients were calculated to assess the associations between questionnaire data, degree of lumbar rotation, CPFV and paraspinal reflex latencies in the sciatica patients (III) and SEP and MEP (IV) and muscle endurance and flexion-

extension activation (V) in the LSS patients. Discriminant or multiple linear regression analysis was performed (IV, V). The statistical analyses were performed with SPSS 10.0 software (SPSS, Chicago, IL) and statistical significance was set as $p<0.05$.

6 RESULTS

6.1 Reliability of the measurements

Twenty healthy volunteers (I) were tested twice within a 1-week interval to assess the reliability of the postural control, lumbar movement perception and paraspinal muscle reaction measurements. In at least 18 (usually 19) of the 20 patients, the two tests yielded measurements that were within 95% of the deviation for all parameters. Thus, the reliability of these measurements was acceptable according to the Bland and Altman method (Bland and Altman 1986).

6.2 Paraspinal muscle responses

6.2.1 Expectation

A short latency response of ~50 ms was observed in paraspinal muscles. On average the latency was ~3 ms shorter ($p=0.017$) during expected trials and the latency shortened during the first three expected trials ($p=0.02$). Visual expectation decreased also the magnitude of the response ($p<0.05$). Trunk movement was initiated ~35 ms and ~50 ms after the impact of the load at T6 and T12 levels, respectively. (I)

6.2.2 LBP with sciatica

The short latency response for the unexpected upper limb loading was similar in the patients and controls. The response latency was shortened by expectation both in healthy controls and sciatica patients in the controlled standing position, i.e., while the pelvis was supported. However, expectation did not decrease the response latency of patients in the uncontrolled position ($p=0.014$, Figure 3). In other words, there was a significant difference between the groups with respect to the effect of control of the position on the effect of expectance of the load. (II)

6.2.3 Surgery in sciatica

After discectomy, the reflex latency was shortened to a greater extent by anticipation more than had been the case prior to the operation ($p=0.043$, Figure 3). Patients with clinically observed motor weakness had less shortened latencies by expectation at baseline but responded similarly to the others after surgery but had longer latencies during the unexpected trials ($p=0.005$). (III)

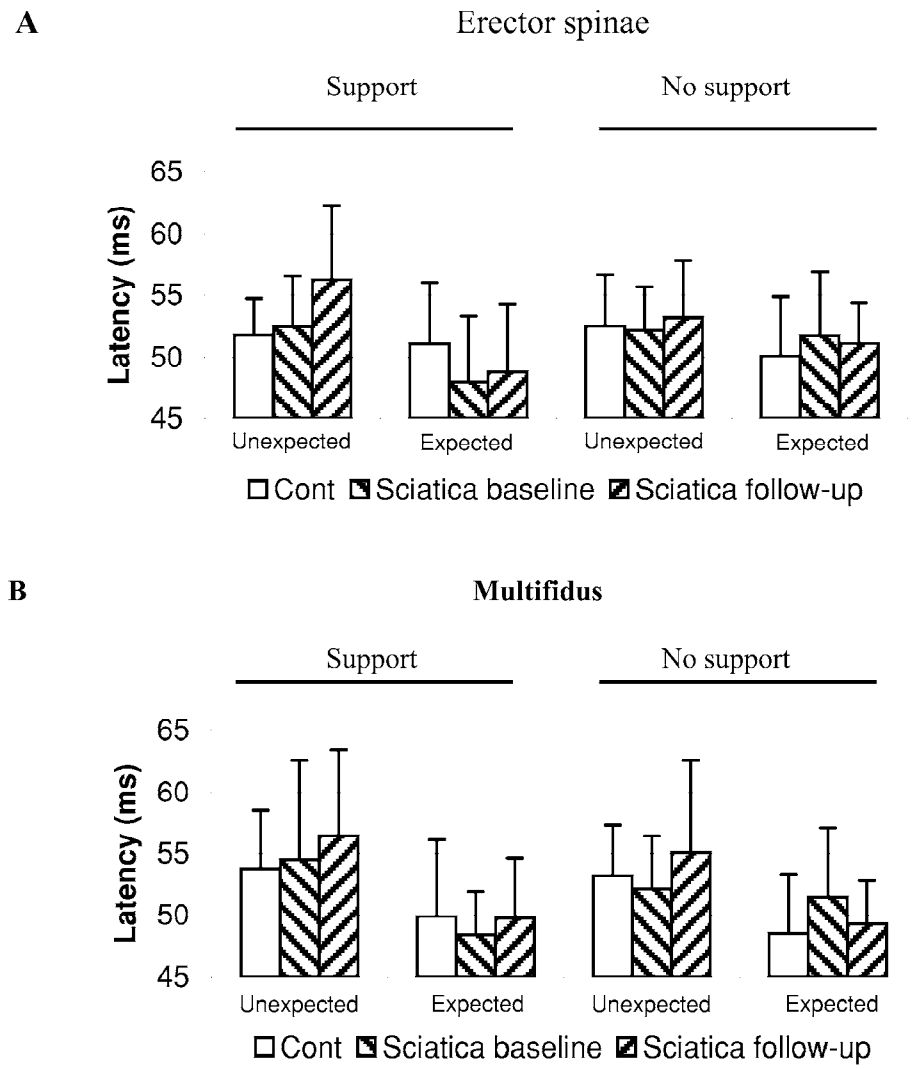

Figure 3. Paraspinal muscle reflex latencies in supported and unsupported standing positions in healthy control subjects and sciatica patients at baseline and 3 months after surgery (follow-up) during unexpected and expected upper limb loading trials from erector spinae (A) and mulfidus (B) muscles. Error bars are SDs.

6.3 Impaired lumbar movement perception

6.3.1 LBP with sciatica

The threshold to detect a change in the position during lumbar rotation was ~2.5 and ~1.0 degrees (p=0.012, Figure 4) in the patients and controls, respectively, and it tended to decrease in the patients after surgery (p=0.054, Figure 4). The degree of lumbar rotation and self-reported disability correlated moderately (r=0.455, p<0.05). The improved self-reported disability correlated with improved movement perception (r=0.589, p<0.05). (III)

6.3.2 LSS

The majority of LSS patients (76.9%) reported the movement direction incorrectly (p=0.006). Furthermore, these patients consistently localised the movement sensation elsewhere in their body than in lumbar region, usually in the shoulder region. The mean±SD lumbar rotations ranged from 7.2°±8.8 to 3.9°±5.9 from the first to the fifth consecutive trials, respectively (Figure 4). The LSS patients had a significantly poorer ability to sense a change in their lumbar position than previously evaluated healthy controls (p<0.0002) and LBP patients (p=0.0005) not suffering from symptoms of LSS. (IV)

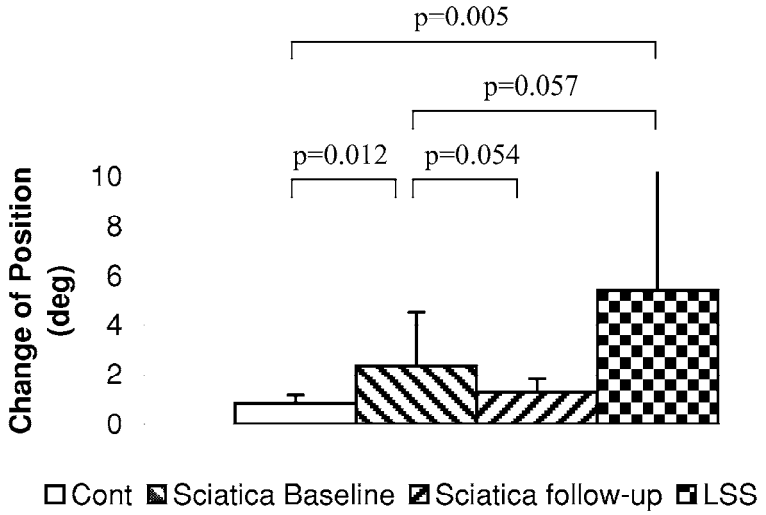

Figure 4. The measurements for the sensation of a change in lumbar position in control subjects and in sciatica patients at baseline and 3 months after surgery (follow-up) and in LSS patients. Five consecutive trials are pooled. Error bars are SDs.

6.4 Postural stability

6.4.1 LBP with sciatica

Patients exhibited larger CPFV values than controls in the two-footed stance with eyes open (p=0.022) and with eyes closed (p=0.001) and in the one-footed balance test (p=0.004, Figure 5). Motor weakness (R^2=0.355; p=0.006) was related with larger two-footed CPFV with eyes open and limited range of movement (R^2=0.465; p=0.001) was related with larger two-footed CPFV with eyes closed at baseline. Patients with motor weakness (p=0.078) and a limited range of movement (p=0.051) tended to have larger one-footed CPFV, but there was no difference in the CPFV values between healthy side and leg with sciatica. The postural control remained unchanged in the patients after (Figure 5) the microdiscectomy but the improved self-reported disability correlated (r=0.524, p<0.05) with improvements in two-footed CPFV with eyes open. (III)

6.4.2 LSS

The mean±SD CPFV values were 4.1±1.5 and 3.6±1.3 cm/s on right and left leg, respectively (Figure 5). Abnormal cortical MEPs were observed in 11 patients (44%) and abnormal SEPs in 16 patients (61.5%). The association between motor and sensory conduction and postural stability was inconsistent. A significant association was found between increased CPFV and prolonged cortical MEP latencies on the left side but no association was detected on the right side. No significant associations were found between abnormal SEPs and CPFV. The associations between impaired lumbar sensation and increased CPFV as well as lumbar sensation and cortical left side MEP were of borderline significance. (IV)

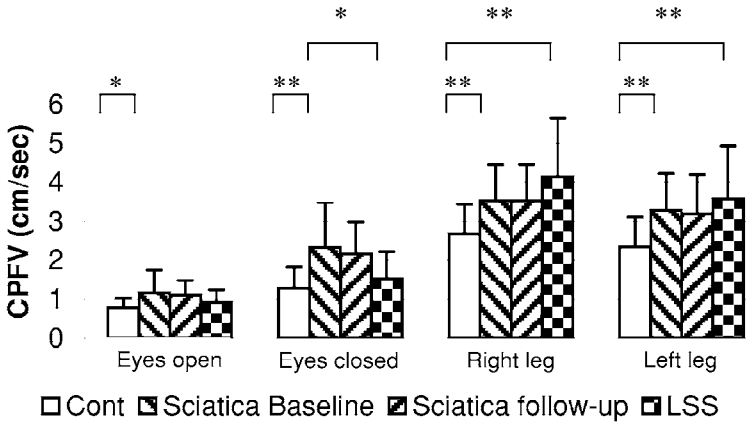

Figure 5. The measurements for postural control (center point of force velocity, CPFV) in control subjects and in sciatica patients at baseline and 3 months after surgery (follow-up) and in LSS patients. Error bars are SDs. *p<0.05, **p<0.01.

6.5 Paraspinal muscle function and endurance

6.5.1 Parapinal muscle denervation

Abnormal findings in the needle EMG of the paraspinal muscles indicative of denervation, were observed in 18 out of the 22 (81.8%) LSS patients. Increasing age and lower extremity pain were related with the probability of abnormal findings in paraspinal needle EMG. Stepwise discriminant analysis entered the subject's age, lower extremity pain and relative extension activation at T12-L1 level as independent variables. Wilks' lambda was 0.184 ($P<0.001$) and these variables explained 81.6% of the variability between the subgroups of the patients with and without denervation. (V)

6.5.2 Flexion-extension function

The average paraspinal muscle activations during full flexion were ~60 and ~65 percent of flexion activation at T12-L1 and L5-S1 levels, respectively. The full flexion-relaxation was achieved by none of the patients despite the fact that all but one patient had a range of flexion movement covering at least 85 degrees. The full-flexion muscle activation associated poorly with denervation, muscle endurance, pain and disability, however, the associations between extension activation and pain and disability were more obvious. The relative extension activation at T12-L1 level was smaller in the patients with paraspinal denervation than in those without denervation. Stepwise linear regression analysis entered lower extremity numbness and paraspinal muscle denervation as independent variables accounting for ~46% of the variability of the relative extension activation (R^2=0.462, p=0.003). (V)

6.5.3 Lumbar endurance

Paraspinal muscle fatigue (pooled mean±SD MPF change at L5-S1 level) was significantly smaller in LSS patients (-6.8±7.1 %/min) than that observed in previously evaluated healthy subjects (-20.4±11.3 %/min) and LBP patients (-21.1±8.7 %/min, p<0.001) not suffering from the symptoms of LSS. The mean endurance time (~172 sec) was similar to that of healthy controls. Paraspinal muscle endurance (MPF change) was not associated with the denervation of the muscles but the endurance time tended to be shorter (p=0.059) in those patients who had denervation. Endurance time, but not the MPF change, was related with disability and low back and lower extremity pain. Stepwise linear regression analysis entered lower extremity pain as an independent variable which explained ~62% of the variability of the endurance time (R^2=0.624, p<0.001). The initial MPF values of LSS patients (84.4±20.9 Hz) were similar as in the previously evaluated healthy subjects (79.1±14.9 Hz, p=0.488) and higher than in LBP patients (71.1±13.2 Hz, p=0.0009). (V)

7 DISCUSSION

7.1 Main novel findings

This study was aimed at detecting the sensory and motor function of the lumbar spine and postural control in healthy subjects and two specific lumbar disorders. The lumbar disc herniation and spinal stenosis were selected to represent two different stages of the LBP syndrome. Disc herniation represents an early and more acute stage whereas spinal stenosis encompasses the late and perhaps the final stage.

Impaired postural control, lumbar movement perception and defective effect of visual expectation on lumbar muscle control were seen in patients with sciatica. Movement perception and the defect in anticipatory processing seem to recover in short term after successful surgery, but postural control did not recover

Nearly total loss of lumbar movement perception was observed in lumbar spinal stenosis, indicative of a clear impairment of lumbar proprioception associated with LSS. The observations of paraspinal muscle denervation and disturbed muscle function during dynamic movement were expected but the exceptionally good lumbar endurance was surprising. The associations between different pathological findings were inconsistent and not necessarily related with each other.

7.2 Subjects and methods

The sciatica patients were selected by a specialist neurosurgeon to meet the criteria for microdiscectomy surgery. They represented a specific condition of low back syndrome with sciatica due to a radiologically confirmed disc herniation and the dropouts did not diverge from the others. LSS patients were selected by a specialist in physical and rehabilitation medicine representing a specific condition of low back syndrome with radicular symptoms due to radiologically confirmed LSS. The patients of both groups were suffering considerable pain and disability.

The repeatability of paraspinal muscle responses for sudden upper limb loading, lumbar movement perception and postural stability was assessed in healthy subjects. The measurements were indicated to be repeatable and the methods were valid for assessing the studied issues. However, according to current knowledge it would have been an advantage to perform all of the measurements in both groups of patients and also on a larger sample of healthy individuals. The sciatica patients had a healthy control group comparable with respect to age and height but LSS patients did not and the use of historical and internal control limits the value of the results.

7.3 Motor control

Anticipation in lumbar motor control

A short latency (~50 ms) paraspinal muscle response for sudden upper limb loading was observed (I), the response being shortened by visual expectation (I). The effect of anticipation can be explained by feed-forward control, where the visual system alerted the reflex mechanisms about incoming sensory impulses. Thus, in the anticipated condition, cortical pre-programming could well be involved in the response modulation.

The response latency was comparable with the earlier findings (Carlson et al. 1981, Marsden et al. 1981, Carpenter et al. 1999, Hodges et al. 2001). The shortening of the latency during expected upper limb loading (I, II) has been observed also in a later study (Moseley et al. 2003) confirming the finding.

The visual expectation did not make the reflex faster in sciatica patients, in contrast to control subjects in the experiments where the spine and pelvis were not supported (II). This indicates that prolonged sciatic pain can impair the lumbar feed-forward control. This is in line with the earlier findings of impaired feed-forward postural responses of the trunk muscles in CLBP (Hodges and Richardson 1996 and 1998, Hodges 2001) and during experimental muscle pain (Hodges et al. 2003). The unaffected paraspinal muscle latency during unexpected upper limb loading differs from the earlier findings of delayed trunk muscle activation during expected and unexpected upper limb and trunk loading (Wilder et al. 1996, Magnusson et al. 1996) and sudden load release (Radebold et al. 2000 and 2001) in CLBP patients. The difference can be explained by dissimilarity of patient groups and methodological variation. Wilder et al. (1996) used computer based wavelet analysis in their assessment of muscle response latencies and therefore the latencies are not comparable. During direct trunk loading and load releasing, the triggering mechanism may be different (I). The duration of the symptoms in sciatica patients (II, III) were usually from three to six months and thus perhaps the variable changes in trunk muscle control were dependent on the pathophysiology and duration of the LBP syndrome.

Since the latency of the responses for unexpected stimulus was unaffected, the limited effect of anticipation in CLBP patients may be due to impairment in the processing of visual feedback information in the short-term memory. Short-term memory is not a distinct cortical area, but an abstract concept representing the co-operation and recruitment of neural networks in several cortical areas involved in information processing. It is an information storage process regulating movement output by processing sensory input between short-term sensory store and long-term memory (Schmidt and Lee 1999).

According to the recognition schema theory, the initial conditions, environmental outcomes, and expected sensory consequences serve as the basis for movement evaluation, i.e. a schema for movement control in the short-term memory by sensory information enables motor learning (Schmidt 1975, Schmidt and Lee 1999). Therefore, the altered short-term memory is probably the explanation for the diminished effect of anticipation, revealing the impairment of feed-forward control system in CLBP patients. It has been hypothesised that CLBP can impair also the higher level information processing i.e. functioning of short-term memory. This hypothesis was based on the observation of missing differences between preferred and non-preferred upper limbs during hand reaction time test in CLBP patients (Luoto et al. 1999).

The possible explanations for the difference in the effect of anticipation between controlled and uncontrolled positions may be the initial condition where the supports in the controlled position function as reference points (Schmidt and Lee 1999). The controlled initial condition, therefore, enhances the affected feed-forward control. The possible enhancing effect of these reference points is in line with the observation that in back healthy subjects there was a small but significant learning effect to reduce the

response latency during three consecutive trials in the controlled position but not in the uncontrolled position (I).

LBP and depression have been shown to interfere with voluntary motor control. The impaired feed-forward control values did not correlate significantly with pain, functional disability and depression scores in the whole patient group (II) but the anticipatory trunk muscle control does seem to be affected by severe LBP. CLBP and pain-related depression could be related to the motor behaviour at a higher level involved in anticipatory behaviour. However, the assessment of a causal relationship between depression, pain and anticipatory control requires further studies.

7.4 Sensory contributions to motor control

The impaired lumbar movement perception was observed in the patients with sciatica (III), however, the impairment was not as obvious as that seen in the patients with lumbar spinal stenosis (IV). The perception of lumbar movement tended to recover after discectomy, indicating that at least in sciatica patients, the impaired lumbar proprioception is a reversible phenomenon (III). The impaired lumbar movement perception may be due to feedback error as a result of sensory loss or a deficit in information processing or a combination of both mechanisms. CLBP seems to reorganise the somatosensory cortex (Flor et al. 1997), being in line with the idea of reversible pain related dysfunction of central sensory information processing.

The majority of LSS patients failed to sense the direction of lumbar rotation correctly and, in addition, they localised the movement sensation elsewhere than in lumbar region. This is evidence of a peripheral sensory loss in LSS but does not exclude the possibility of altered central sensory-motor processing (IV). The proprioceptive deficit in LSS was more severe in LSS than in previously observed LBP patients with no LSS (Taimela et al. 1999). Since ligament mechanoreceptors (e.g. Sjölander et al 2002) and muscle receptors (e.g. McCloskey 1978, Sjölander et al 2002) are important in propioception and denervation with impaired muscle function (V) and ligament calcification is typically associated with LSS, the impaired movement sensation in LSS patients was to be expected. The local loss of lumbar proprioception appears to be more important in spinal function than the changes in long ascending nerve tracts evaluated by SEPs.

The current results are in line with the earlier observations of a large repositioning error in trunk movement in idiopathic LBP (Gill and Callaghan 1998, Brumagne et al. 2000, Newcomer et al. 2000) and in lumbar segmental instability (O'Sullivan et al. 2003). Since muscle spindles have a major role in lumbar proprioception (Brumagne et al. 1999) and the γ-muscle spindle system is thought to have a significant function in feed-forward control (Sjölander et al. 2002), dysfunction of the muscle spindle system may contribute to both impaired lumbar proprioception and feed-forward control.

7.5 Postural control

Impaired postural stability was observed in sciatica (III) being comparable with the previous findings in LBP patients and controls (Byl and Sinnot 1991, Luoto et al. 1998). In the current study, the two-footed body sway was also increased in the patients with

clinically observed motor weakness and a limited range of movement. Furthermore, one-footed body sway was not associated with the side of sciatica in the one-footed test (III). The two-footed postural stability was weaker also with eyes open, but the difference between patients and controls was more prominent when the eyes were closed during testing (III). This is in agreement with the previous findings indicating that the impaired sensory input from muscles and joints is more severely challenged during the absence of visual input (Byl and Sinnot 1991, Mientjes et al. 1999, Radebold et al. 2001).

The unsuccessful recovery of impaired postural control after surgery in sciatica is not surprising according to a previous prospective evaluation in CLBP patients. After active rehabilitation, the impairment in postural control remained unchanged in the patients with good outcome but deteriorated further in the patients with poor outcome (Luoto et al. 1998). In addition, low performance in a test of standing balance was associated with future back disorders (Takala and Viikari-Juntura 2000).

According to the previous studies in conservatively (Balague et al. 2001, Dubourg et al. 2002) and surgically (Dubourg et al. 2002) treated sciatica patients, the recovery of impaired muscle performance was not a matter of course even at six months of follow-up. Therefore, the recovery of postural control may have been still taking place after three months follow-up. However, many postural reactions are not triggered by lower leg proprioception, but probably by receptors in the proximal body segments (Bloem et al. 2000). Thus, the impaired postural control seems to be related with the information processing in addition to the lower leg muscle function. A longer follow-up duration, perhaps with postoperative intervention, would help in clarifying this issue.

The postural stability usually improves during the repetitive measurements indicating motor learning. In LSS patients, the postural stability had a weak negative correlation with lower extremity pain intensity during standing i.e. those patients with more severe pain seemed to have better postural stability (IV). Further analysis showed that the one-footed postural stability improved during the repetitive measurements in the patients with less pain, but it seemed to become even worse in the patients with severe pain. This is further evidence that pain may impair motor learning.

Postural control is a complex procedure involving integrated sensory and motor function. An association between poor balance performance during unstable sitting with eyes closed and delayed trunk muscle response times during quick force release, reflecting the relationship between impaired postural control and delayed muscle activation, has been reported previously in idiopathic CLBP (Radebold et al. 2001). The current results in sciatica patients are in accordance with those findings. Additionally, the postural control and muscle response times correlated with impaired proprioception (III). Impaired postural control was associated with impaired lumbar perception both in sciatica and LSS patients (III, IV). In LSS patients, according to the evoked potential findings, the one-footed postural control seemed to be more dependent on motor than sensory conduction between the lower limbs and the central nervous system (IV).

7.6 Function of lumbar muscles

Impaired lumbar muscle function and denervation were frequently observed in LSS patients. The lumbar endurance time of LSS patients was associated with low back and lower extremity pain but not with power frequency change. The limiting factor of endurance in some LSS patients was not the paraspinal muscle fatigue but the pain intensity, especially the intensity of radicular symptoms (V). I also attempted to measure lumbar endurance in sciatica patients, but because half of the first eight patients were unable to perform the test due to intolerable pain, further analyses were discontinued.

The value of dynamic lumbar endurance assessments and power frequency analysis as a measurement of muscle function must be questioned in patients with muscle denervation and radicular symptoms (V). However, the endurance test could be useful in monitoring the functional ability and the fatigue resistance of paraspinal muscles in LSS patients indicate indirect evidence for selective type 2 muscle fiber atrophy possibly associated with LSS (V). These results are in accordance with an earlier proposal of the low diagnostic value of the isometric trunk strength test (Rantanen 2001). That test was indicated to be a potentially hazardous for high-risk patients due the cardiovascular stress (Rantanen 2001), which could be particularly dangerous in LSS patients who often are elderly people.

The paraspinal muscle dysfunction during flexion-extension movement was similar in LSS patients (V) as previously observed in CLBP patients (Ahern et al. 1988, Sihvonen et al. 1991, Shirado et al. 1995, Sihvonen et al. 1997) and during experimental pain (Zedka et al. 1999). This was evident as increased paraspinal muscle activity when acting as an antagonist and decreased activity when acting as an agonist (V). The different recruitment patterns found in CLBP patients increased spinal stability and therefore may represent a beneficial compensation mechanism (van Dieen et al. 2003a and b). Decreased extension activation but not the increased flexion-relaxation activation was related with the denervation and subjective low back and lower extremity symptoms and were not correlated with each other (V). Thus, these could be independent phenomena reflecting different changes in motor control properties.

Abnormal paraspinal needle EMG findings indicate that denervation of paraspinal muscles was frequent in LSS patients (V). In previous studies, a denervation of multifidus muscles was often observed in sciatica patients (Mattila et al. 1986, Rantanen et al. 1993). Although the muscle receptors seem be important in lumbar perception (Brumagne et al. 1999 and 2000, Taimela et al. 1999), the impaired lumbar perception did not have any direct relationship with the muscle denervation findings in LSS. However, the denervation findings often represent only a limited area of the paraspinal muscles, which may explain their poor relationship with both lumbar muscle endurance and proprioception. Since the extensive spinal degeneration and abnormal paraspinal muscles are common in LSS the dorsal ramus neuropathy (Sihvonen 1995) may be associated also with LSS and therefore their evaluation would be beneficial in the assessment of pathophysiology of LSS.

7.7 Clinical significance and hypothesis for further investigation

The chronic pain may modulate neuronal activity at the spinal, intermediate and cortical levels (Flor et al. 1997, Watkins and Maier 2002, Juottonen et al. 2002, Farina et al. 2003) and it may alter both central tactile and motor processing (Juottonen et al. 2002). The impaired motor control can be restored by rehabilitation procedures. In rehabilitation, the aim is to enhance the motor performance. In painful conditions, the central information processing can be disturbed by the sensation of pain. This may lead to disuse of muscles by fear-avoidance behaviour (Vlaeyen et al. 1999, Vlaeyen and Linton 2000). During exercise, the brain receives alternative information in addition to pain signals, this probably being explained by gate control theory, decreased sensitivity of receptive structures or CNS remodelling. Thus, this pain-related CNS remodelling may be reversed by rehabilitation (Flor 2003). This may lead to relief of pain and break the vicious cycle of pain, disuse and de-conditioning. The apparently partly reversible phenomenon of impaired feed-forward control in sciatica (II, III) indicate a possible site of motor control to focus rehabilitation of LBP patients.

The effect of rehabilitation may elicit better neuronal function as well as improved muscle properties. The cognitive effect may at least in certain situations be even more important than the effect at the muscle level. The crucial factor is to enhance muscular function by improving the muscle control. After successful rehabilitation of CLBP patients (Mannion et al. 2001b) no change in histologically analysed structural muscle anatomy was observed (Kaser et al. 2001) and the changes in muscle performance were mainly related with psychological factors (Mannion et al. 2001a and b), however, the histological changes seem to appear slowly (Rissanen et al. 1995). The current study supports the idea that co-ordination and muscle control are perhaps more important in the rehabilitation of LBP patients than muscle strength.

The functional brain imaging has provided new insight into the anatomical structures and physiological functions of pain processing (Flor et al. 1997, Peyron et al. 2000). In addition to the imaging of neuronal function, other methods used in cognitive neuroscience could be advantageous in assessing the chronic pain. The current results point to impaired sensory function and information processing being associated with both LSS and sciatica.

In future studies the representative cohorts of LBP patients with various spinal pathologies should be tested with the full measurement repertoire in prospective settings. This kind of simultaneous testing would clarify the associations between the findings. The prospective setting with an adequate follow-up time would clarify the still remaining question, which phenomena are the cause and which are the consequence of the problem. Furthermore, it is important to clarify which phenomena are reversible and through which kind of intervention.

This work does not alter the current clinical practice but does broaden our view of the LBP syndrome and in the terms of WHO can provide new insights into sensory and neuromusculoskeletal functions associated with lumbar disorders and has provided novel hypotheses which can be evaluated in further experiments.

8 CONCLUSIONS

Impaired anticipation related lumbar motor control, proprioception and postural control were observed in LBP patients with sciatica. Anticipation related lumbar control and proprioception improved after surgery but postural stability remained unchanged. These results indicate that, in addition to lumbar nerve root irritation, there seem to be changes of the central motor control in LBP with disc herniation and that the recovery of these impairments is not simply a matter of course.

Extensive loss of lumbar proprioception, lumbar paraspinal muscle denervation and impaired muscle function were observed in lumbar spinal stenosis. These findings indicate that there appears to be not only notable structural pathologies in the lumbar spine but also clearly abnormal lumbar function in LSS.

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