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University of Alberta

Activity of human trunk muscles with and without back pain

by

Milan Zedka 

A thesis submitted to the Faculty of Graduate studies and Research in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

Division of Neuroscience

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ABSTRACT

Back pain is a musculoskeletal disorder whose rational treatment is problematic because its etiology is largely unknown. This thesis contains several discoveries regarding trunk muscle activity in health, fatigue and pain. The functioning of individual muscles can be inferred from electromyographic (EMG) records. Since the recording is not standardized, we first examined the influence of electrode type and configuration on EMG from the erector spinae muscles (ES). We showed that an unorthodox placement of electrodes perpendicularly to the long axis of ES fibres prevents their undesired displacement while a consistent relationship between EMG parameters and force or fatigue is preserved. Investigating trunk muscle coordination in postural tasks revealed that back muscles, but not abdominal muscles, were reciprocally activated instead of showing the general co-contraction frequently observed in other tasks and considered as a common pattern. Our study of ES activation during fast repetitive hand movements indicates that segmental stretch reflexes play an important role in postural stabilization of the trunk. This finding challenges the current view that postural adjustments during voluntary limb movements are performed in a feed-forward manner. Finally, our investigations of reflex and voluntary ES activities during induced pain shed more light on back muscle functioning under pathological conditions. A major finding was that hypertonus of painful paraspinal muscles is not connected to hyperexcitability in the γ-loop. A discovery of differential ES activation depending on the phase of voluntary movements (resulting in a decreased depth of EMG modulation) may partially explain the previous controversy about hyper- or hypofunction of paraspinal muscles in back pain.

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CHAPTER 1.

GENERAL INTRODUCTION

1.1 Back pain problem

Low back pain is a common condition afflicting people throughout the world. While acute pain can totally but temporarily incapacitate the sufferers, persistent symptoms in chronic pain often drastically impair the quality and productivity of their lives. Therefore, back pain is not only a major health problem for the individual but has also its socio-economic dimensions. Proper management of pain remains one of the most pressing issues of society in general and scientific and health workers in particular.

The importance of the back pain problem is underlined with social statistics. It is predicted that 50 - 80% of adults will have low back pain at some time in their lives (Biering-Sorensen, 1983; Heliovaara, 1989). More than 5 million people in the U.S.A. are disabled due to this condition, half of them permanently (Deyo, 1991). Low back pain is the most common reason for disability under 45 years of age (Cunningham & Kelsey, 1984) and the resulting loss in work time accounts for more lost working days than cancer, cardio-vascular diseases and AIDS put together (Rowe, 1983). It has been estimated that in 1991 the cost for the treatment, compensation and the time lost at work due to LBP in the United States was approximately $72 billion. The medical costs alone were $24 billion (Deyo, Cherkin, Conrad & Violinn, 1991). Back and neck pain are the leading causes of sick leave, compensation and early retirement expenditures not only in the U.S.A. but also in most countries of the industrialized world (Nachemson, 1992; Waddell, 1996). In the United Kingdom, Department of Health and Social Security figures show that 46.5 million working days in the financial year 1987/1988 were lost due to spinal problems which represent 12% of all incapacities (Lapsley, 1990). The cost

of back pain in the Netherlands in 1994 was about $5 billion, which is equal to almost 2% of the Gross National Product (van Tulder, Koes & Bouter, 1995). Obviously, a problem of such dimensions should receive more attention. Unfortunately, low back pain is only a symptom and in the majority of cases not a disease with a diagnosis based on an understanding of the underlying pathology which would provide information on rational treatment.

1.2 Etiology of back pain

Low back pain (LBP) is a symptom that can be caused by various disease entities and can be affected by various psychological factors. The pathological basis for pain may lie within the spine (spondylogenic or neurogenic) or outside of the spine (viscerogenic, vascular, or psychogenic).

Spondylogenic back pain may be defined as pain derived from the spinal column and its associated structures (vertebrae, sacroiliac and facet joints, intervertebral discs, ligaments and muscles). Lesions of these structures together with neurogenic lesions (radiculopathy, arachnoid irritation, etc.) constitute the most common source of low back pain seen in clinical practice. Since only 1% of people with acute LBP have radiculopathy (Sinaki & Mokri, 1996), spondylogenic back pain by far dominates among the causes of back pain, when classified according to the pain source. From the etiological point of view, the causes of spinal lesions may be degenerative, inflammatory, infectious, metabolic, neoplastic, traumatic, congenital or developmental.

The painful structures are usually not amenable to direct scrutiny; therefore, a tentative diagnosis is usually arrived at by taking a careful case history and employing a thorough physical examination, with imaging and laboratory procedures as indicated. Imaging techniques, such as conventional radiography, myelography, discography, facet arthrography, computed tomography, magnetic resonance imaging, nuclear medicine, videofluoroscopy, ultrasound or angiography can detect gross structural defects and are useful in excluding life-threatening causes of back pain (tumors, tuberculosis, etc.). However, there are limitations in interpreting the findings in a clinical context since diagnostic tests often correlate poorly with symptoms and even less well with the level

of disability of the subjects (Schwarzer, Wang, O'Driscoll, Harrington, Bogduk & Laurent, 1995; Fraser, Sadhu & Gogan, 1995).

The underlying cause of LBP is often difficult to discern. A specific lesion is found in only 10 - 20 % of persons with acute LBP (Nachemson, 1992). In 80 – 90% of cases the underlying pathology of the back symptoms remains undetected and the syndrome is then referred to as idiopathic low back pain (ILBP). ILBP is a benign condition with recovery from symptoms occurring in several days or weeks from the time of onset in the vast majority of individuals. Some will have recurrent episodes and in about 10% of patients the condition will develop into a chronic state (Bergquist-Ulmann & Larsson, 1977). Our limitation in understanding ILBP is obvious by the inconsistencies in diagnoses for the condition. The diagnostic terms for such conditions are numerous (non-specific back pain, mechanical back pain, myofascial back pain, lumbago, regional musculoskeletal illness of the axial skeleton, etc.).

A short look at the risk factors associated with low back pain (Table 1.1.) evokes a strong impression that most cases of back pain are caused by mechanical, often occupational, factors (Burn & Paterson, 1990; Frymoyer, 1991; Sinaki & Mokri, 1996). Indeed, many authors claim that heavy physical loading and extreme positions are associated with higher incidence of back symptoms (Hult, 1954; Riihimaki, Tola, Videman & Hanninen, 1989; Svensson & Andersson, 1983; Videman *et al.*, 1989). However, conclusions about mechanical factors in the etiology of back pain cannot be final because not all studies have found such associations (Biering-Sorensen & Thomsen, 1986; Sairanen, Brushaber & Kaskinen, 1981; Damkot, Pope, Lord & Frymoyer, 1984).

1.3 Diagnosis of mechanical back pain

The diagnosis of back pain of mechanical origin is made after excluding other known causes. Clinical evaluation starts with the patient's history, which may reveal a mechanical traumatic event in the past or a history of physically demanding activity (occupation). If no strenuous event can be recalled by the patient the symptoms are often interpreted as a result of accumulated micro-lesions, which went unnoticed over a period

Table 1.1. Back Pain Risk Factors.

1. Individual factors:

Age (35 – 55 years)

Gender (females over 60)

Genetic (HLA-B27 in ankylosing spondylitis)

Stature (tall, obese)

Skeletal defects (scoliosis, spondylolisthesis, leg length discrepancy etc.)

Muscular strength and physical fitness

Smoking

Psychological factors (depression, anxiety, hysteria, alcoholism)

2. Occupational factors:

Physically heavy work, overexertion

Static work postures, prolonged position

Frequent bending and twisting

Lifting and forceful movements

Pulling and pushing

Cyclic loading

Slipping, tripping and falling

Vibration

Boring, repetitious, dissatisfying work

of time. Accordingly, the onset of pain may be abrupt or insidious. The syndrome presents as a dull aching pain that affects the lower spine and might spread along the spine or to the buttocks. Strenuous effort, poor posture at work or during sleep and forced movements are often aggravating factors. Aggravation is further caused by internal (digestive, gynecologic, psychological) and external factors (drafts of cold air, etc.). Pain often progressively worsens during the day (activity) and is usually relieved by rest. There are no associated neurological symptoms (paresthesia, numbness).

On inspection there may be paraspinal spasm and postural asymmetry. Physical examination may further reveal local tenderness or pain on palpation, tightness of paraspinal muscles, and sometimes a withdrawal reaction to touch. Often a painful spinal segment with modifications of the texture and sensitivity of the soft tissues – cutaneous (cellulagia), tenoperiosteal or muscular (trigger points) – can be found using segmental palpation maneuvers (Maigne, 1996). Several tests are used to assess physical performance. Active and passive trunk range of motion (ROM) is tested for flexion, extension, lateral flexion and rotation and is usually diminished. It can be measured with a tape measure or inclinometers but often it is only subjectively assessed by the examiner. Trunk muscle strength is usually assessed subjectively by the patient's ability to apply force against the clinician's resistance or against gravity. In pain it is usually reduced. These tests are, in fact, a constituent part of a routine neurological examination, which has to be performed to assess involvement of nerve roots etc. It should further include an evaluation of the strength of leg muscles, their possible atrophy, their stretch reflexes and the straight-leg raising test (Lasegue test). More complex tasks are used for subjective evaluation of gait, posture and coordination. Testing of motor capabilities should be complemented by searching for areas of altered skin sensation.

Unfortunately, physical examination rarely discloses the precise source of back pain, which would allow goal-directed therapy. Rather, the determination of functional losses is considered important for evaluation of the rehabilitation process.

1.4 Biomechanical approach to back pain

Due to the mechanical character of many of the risk factors associated with back pain, most investigators approach back pain as a biomechanical problem (Farfan, 1973). It is supposed that the onset of lower back pain is related to the imbalance in the mechanical components of the back. The determination of the loads imposed on the low back structures is considered to be crucial because by controlling the loading back pain could be prevented or effectively treated.

Because of the relative inaccessibility of spinal tissues to direct load measurement, the goal of the biomechanical approach is the creation of a mathematical model that would simulate real loads in the human low back during daily activities. Entering information obtained on patient's examination into a valid biomechanical model of the lower back could, in principle, identify deficits in the patterns of muscular activity and point to structures that are most probably damaged. The diagnosis of such deficits could then lead to the prescription of individualized exercises selected to optimize load balance. Models of this type have been created and refined over several decades (Andersson & Winters, 1990). Unfortunately, none of them provides satisfactory guidance in the prevention or treatment of back pain.

If we disregard the fact that pain is a complex phenomenon that probably cannot be reduced to tissue mechanics, there are two major requirements that have to be fulfilled before a reliable mathematical model can be formulated: (a) detailed knowledge of the anatomy and (b) understanding of the biomechanics of vertebral function.

It may come as a surprise that such basic questions were not answered a long time ago. Anatomical studies on human cadavers have been performed for hundreds of years and yet it was not until 1980 that a new detailed anatomical description of the back musculature was provided by Bogduk (1980). This fact casts a shadow of doubt on previous models based on the muscle descriptions in the classical anatomy books.

As to the functions of the spine, it participates in voluntary trunk movements and maintains postural stability during limb and head movements and locomotion. Many aspects of how these functions are achieved are poorly understood. Postural stability does not mean only stability of a rigid body in quiescent standing or sitting positions. In

real life, dynamic stability of the trunk is required while the centre of gravity and the base of support constantly change. In addition to static loads the low-back structures have to initiate or resist repeated accelerations and decelerations of the trunk, limbs and head. This task has to be completed in a smooth, coordinated fashion. A loss of the spine's ability to maintain its patterns of displacement under physiological loads so there is no initial or additional neurologic deficit, no major deformity, and no incapacitating pain, is called clinical instability of the spine (White & Panjabi, 1990).

The structures in the lower back that provide spinal stability and come under stress when the trunk performs its function are vertebrae, disks, facet joints, ligaments and muscles. The first four are considered as passive components, while muscles, in addition to their passive properties, can generate torques by active contractions. Biomechanical properties of the tissues have been investigated in numerous studies *in vitro* (Markolf & Morris, 1974; Goel, Goyal, Clark, Nishiyama & Nye, 1985; Panjabi, Krag & Chung, 1984; Abumi, Panjabi, Kramer, Duranceau, Oxland & Crisco, 1990). However, loads applied to isolated tissue out of functional context do not necessarily simulate loading in real life, particularly when many surrounding tissue elements normally interact with the tissue in question.

Invasive *in vivo* experimental investigations into the function of the low-back structures are generally unacceptable for ethical reasons. Therefore, considerable effort has been put into developing indirect methods. The experimental variables that can be obtained *in vivo* are trunk kinematics and forces generated at points of contact with the environment. Such measurements provide information about the external aspect of the trunk activity but reveal little about active, internal force generation by individual trunk muscles. The spine consists of a large number of segments and its global motion requires stability in multiple planes. A significant number of muscles with complex insertions and interactions must function in synergy to properly distribute the loads imparted to the mobile segments. Unfortunately, a comprehensive understanding of the role muscles play in the control of trunk motion and posture is lacking. The development of such understanding could perhaps lead to assessment techniques for determining quantitatively and objectively a given muscular deficiency and the optimal therapeutic regimen to improve such deficiency.

Determining tolerable loads for individual tissues is only the first step towards solving the back pain problem. An equally important issue is our ability to control such loads at will. We can certainly choose a weight and roughly influence the position in which we want to handle it. It is, however, impossible to decide that we want to rotate, for example, the L3 vertebra against the L4 vertebra by 2°. We would neither know what position to choose to let some external load do the work for us nor which muscles to employ to produce such rotation actively. To understand what forces are acting in the low back during daily activities or during prescribed exercises, we have to determine which muscles produce or resist those forces, when and how much they are active. Furthermore, we want to know under what circumstances the muscles are not optimally activated to generate adequate contractions. Besides geometrically unfavorable states of a muscle in certain postures, torque production is dictated by the condition of the muscle fibres and their neural supply. The importance of understanding the neuromuscular basis of such conditions as muscle fatigue or muscle pain is obvious. In this respect, back muscle neurophysiology has a long way to go before it receives as much attention as the biomechanical approach to back pain.

The following section will introduce some problems concerning back muscle functioning from the neurophysiological point of view, which were addressed by my colleagues and myself in the period of the last five years at the University of Alberta. The work has all been published or submitted for publication in scientific journals.

1. 5 Thesis Objectives

Activity of trunk muscles has been estimated using a neurophysiological method called electromyography (EMG). This technique uses metal wire electrodes, placed directly in the investigated muscle or on the overlying skin, to infer various phenomena related to muscular contraction from electric potentials produced by muscle fibres. EMG records provide information on the timing of muscle contractions allowing observations on their relationship to associated movements. Also, since muscle output cannot be measured directly in mechanical terms, EMG signals have been used to predict muscle force. Unfortunately, the relationship between EMG signals from muscles and force is

often non-linear and depends on factors such as muscle length, presence of surrounding tissues etc. The same applies for studies where parameters of the EMG signal have been used to quantify muscle fatigue, since muscle fatigue has been associated with impaired performance and injury (Roy, De Luca & Casavant, 1989). Some studies have relied on the amplitude of the EMG signal as an indicator of muscle fatigue, while others monitor the modifications to the frequency domain of the EMG signal. (Piper, 1912; Cobb & Forbes, 1923; Petrofsky & Lind, 1980; Stulen & De Luca, 1981; Naeije & Zorn, 1981; Palla & Ash, 1981; Arendt-Nielsen & Mills, 1985). The quality of the EMG signal depends on many intrinsic factors and also on the recording technique. Since the technique of recording from back muscles is not standardized, the first paper (Chapter 2 in this thesis) was designed to investigate the effect of the types of electrodes used and their placement on the EMG signal.

The next two studies deal with the role of the back muscles in postural stabilization of the trunk. There is considerable need to address the issue of trunk muscle coordination in the task of providing postural stability. It is assumed that in a healthy trunk, agonistic and antagonistic activities are well coordinated to provide smooth motion but the precise relationships are unknown. We addressed the issue of trunk muscle coordination in a balance perturbation study (Chapter 3). The simplest conceivable response of trunk muscles to trunk perturbation would be co-contraction. Indeed, several studies indicate that co-contractions of trunk muscles are very common, not only in symmetric activities, such as trunk flexion-extension in the sagittal plane, but also in situations of asymmetry, such as lateral flexion (Andersson, Ortengren & Herberts, 1977), rotation (Basmajian, 1978; Morris, Benner & Lucas, 1962; Pope, Andersson, Broman, Svensson & Zetterberg, 1986) or walking (Thorstensson, Carlson, Zomlefer & Nilsson, 1982). Co-contraction may be important from the point of view of mechanical stability, providing appropriate stiffness to the trunk. However, high levels of antagonistic muscle activities and the resultant spinal loads have been considered as the reason for the relationship between twisting and low-back pain (Magora, 1974; Frymoyer, Pope, Constanza, Rosen, Goggin & Wilder, 1980). An alternative response to trunk perturbation would be a coordinated and reciprocal sequence of contractions and relaxations of antagonistic muscles timed so as to counteract the perturbation. However,

previous studies of the trunk musclulature have not revealed an equivalent of the reciprocal inhibition, found between agonists and antagonists in the limbs. We conducted the balance study (Chapter 3) to determine which of the two strategies (co-contraction vs. reciprocal activation) is used in the trunk, and whether this depends on velocity of perturbation, expectation, or visual clues.

In addition to external perturbations, the trunk is subject to internal perturbations generated by moving body parts. For example, muscles in the trunk are involved in trunk stabilization during arm movement as the trunk is coupled to the upper limbs by muscles of the shoulder girdle. Beevor (1903) first observed that the erector spinae contracts when an arm is elevated forwards. The voluntary movement would have a perturbing effect on equilibrium and contraction of postural muscles would exert correction forces necessary for maintaining the equilibrium. Belenkii et al. (1967) demonstrated that postural muscles (ipsilateral hamstrings) were activated prior to a voluntary raising of one arm. This and later studies of voluntary arm movements (Lee, Buchanan & Rogers, 1987; Bouisset & Zattara, 1981; Gurfinkel, Lipshits & Lestienne, 1988; Cordo & Nashner, 1982) refuted the general opinion of the first half of this century that posture was controlled purely by stretch reflexes in postural muscles elicited by deviations of the body from vertical alignment (Liddell & Sherrington, 1924; Fulton & Liddell, 1925, Magnus, 1926). Thus the current concept on postural control is that voluntary movements are accompanied by postural adjustments that are largely organized centrally in a feed-forward mode. In Chapter 4 we examine EMG activity in back muscles during hand movements and argue that the emphasis on central control of postural adjustments should not lead to an underestimation of the importance of trunk muscle stretch reflexes in trunk stability.

Stretch reflexes may have several functions, one of which is to regulate muscle tone (Liddell & Sherrington, 1924). Increased stretch reflex gain has been, therefore, considered as the underlying reason for muscle hypertonus (taut spastic muscle on palpation), which is a frequent finding in painful muskuloskeletal conditions, including back pain. This notion has been supported by numerous studies, which have found excitation of γ-motoneurones upon stimulation of muscle nociceptors or pain-conducting small-diameter afferents (Jovanovic, Anastasijevic & Vuco, 1990; Ljubisavljevic,

Jovanovic & Anastasijevic, 1992; Johansson, Djupsjobacka & Sjolander, 1993; Djupsjobacka, Johansson, Bergenheim & Wenngren, 1995). Surprisingly, the stretch reflex has never been compared in normal and painful back muscles. Chapter 5 is devoted to the stretch reflex in the erector spinae during back muscle pain and during back skin pain.

Motor dysfunction in back pain patients is a common occurrence. However, the link between pain and motor output has not been established, partially because in patients it is rarely possible to locate the origin of back pain. The changed motor patterns observed in the clinical setting might be the result of multiple injuries and it is hard to make conclusions about dysfunction of individual back structures. Therefore, in order to investigate paraspinal muscle activity during voluntary trunk movements (Chapter 6), back pain confined to the erector spinae muscle was induced by infusion of a hypertonic saline solution. The study was aimed at elucidating controversial findings regarding the level of back muscle activity in back pain. While some authors have suggested higher paraspinal activity in low-back pain, others have reported unchanged or smaller activity (Alston, Carlson, Feldman & Grimm, 1966; Jayasinghe, Harding, Anderson & Sweetman, 1978; Hoyt *et al.*, 1981; Collins, Cohen, Nailboff & Schandler, 1982; Thorstensson & Arvidson, 1982; Wolf, Nacht & Kelley, 1982; Nouwen & Bush, 1984; Ahern, Follick, Council, Laser-Wolston & Litchman, 1988; Arena, Sherma, Bruno & Young, 1989). Also, overloading of muscles compensating for weak painful muscles has been considered as a reason for changed motion patterns and perpetuation of pain (Janda, 1986). Therefore, we examined the possibility of an increased activity in the contralateral erector spinae during unilateral muscle pain.

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CHAPTER 2.

Comparison of surface EMG signals between electrode types, interelectrode distances and electrode orientations in isometric exercise of the erector spinae muscle[1]

2.1 Introduction

Surface electromyographic (SEMG) signal processing techniques allow a good assessment of muscle activity. The amplitude and frequency characteristics of the SEMG have been used for prediction of force and also for quantification of muscle fatigue. The quality of the signal depends on a number of factors, including the type and placement of the electrodes. Various types of commercially available surface electrodes differ in the shape, size and material of their active surfaces and in other prolperties. The position of the electrodes over the muscle is also a variable of major importance. Previous studies have shown an effect of the interelectrode distance (IED) on the signal characteristics from the recorded muscle as well as on the amount of cross talk (Lynn, Bettles, Hughes & Johnson, 1978; Zipp, 1982). The IED is usually considered as the distance between the electrodes at the time of their placement. However, IED rarely remains the same when the underlying muscle changes its length. As the skin stretches or folds, the electrodes placed in the direction of the muscle fibres move considerably. It has been noticed in our laboratory that the displacement of the surface electrodes is particularly

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noticeable over the erector spinae (ES) muscle. The IED, determined in upright standing, may change by several centimeters during flexion or extension of the trunk. This displacement depends on the initial distance between the bipolar electrodes and also on their orientation in regard to the course of the muscle fibres. It is possible that in some cases the displacement could significantly alter the SEMG recordings and result in different conclusions about the muscle activity. Therefore, this study investigated the effects of the electrode type, IED and orientation on the electrode travel and the recorded SEMG characteristics.

2.2 Methods

2.2.1 Subjects

Ten male subjects between 20 and 31 years of age with no history of low-back pain volunteered for the study. Their average height was 178 cm (SD $\pm$ 16 cm) and their weight was 81 kg (SD $\pm$ 9 kg). All subjects participating in the experiment signed an informed consent form.

2.2.2 Experimental Paradigm

Isometric contractions of ES were performed in a partially stooped posture by pulling upward on a handle attached to a chain anchored in the floor. The length of the chain was adjusted for each subject such that the trunk was flexed at 40°, the legs were straight and the elbows were at 90° of flexion. The handle allowed a firm grip with both hands (Figure 2.1.). The subjects were instructed to perform maximal voluntary contraction (MVC) of the ES for 5 seconds. The mean value of the MVC during the 5 s period was taken as 100%. Subsequently, the subjects were asked to exert 20%, 40%, 60% and 80% of their MVC in a random order for a period of 5 s, with 10 minutes of recovery between the tasks. The last task in the series was an isometric prolonged holding at 80% MVC till fatigue. This fatigue trial was terminated by the subject when

the maintenance of the required force level shown on a digital display of a force monitor became unsustainable (failure point).

2.2.3 Recording and analysis

Electrodes were placed on the subjects while they were standing upright. After appropriate skin preparation three types of surface electrodes were placed according to the configuration shown in Figure 2.2. The Ag/AgCl Miniature Biopotential Skin Electrodes had a round surface and a diameter of 2 mm. After application of gel, these electrodes were affixed to the skin with a collar-shaped double adhesive disc. One Miniature electrode was placed over the left ES 2 cm laterally to the L3 spinous process. The other four electrodes were placed in a row cranially to this common electrode 2 cm apart from each other. Each of these electrodes was paired with the common electrode forming interelectrode distances of 2, 4, 6 and 8 cm. This cranio-caudal arrangement of the Miniature electrodes was considered as the orientation "in series" and the channels were referred to as MS2, MS4, MS6 and MS8, for Miniature electrodes "in series" at 2, 4, 6, and 8 cm apart, respectively. In addition, two Miniature electrodes were placed lateral to the common electrode, in a row perpendicular to the array described above. In this way, pairs with interelectrode distances of 2 and 4 cm "in parallel" were obtained (MP2, MP4).

On the right side the disposable 14445C Hewlett-Packard monitoring electrodes (HP) were used. Their Ag/AgCl active surface had a diameter of 1 cm and was covered with a thin sponge containing gel. The common electrode was placed on ES 2 cm lateral to L3. Two electrodes were placed cranial to it to create pairs "in series" 3 cm and 6 cm apart (HPS3, HPS6). One electrode was placed "in parallel" 3 cm lateral to the common electrode (HPP3).

The third electrode type used in this experiment were the bipolar Neuromuscular Research Center electrodes (NMRC) developed in Boston. The active surfaces of these electrodes are two parallel bars (1 cm x 1 mm) of a constant separation 1 cm, housed in a single unit. No gel was used with this electrode type. One electrode was placed "in series" over the right ES cranial to the HP row (NMRCS). Lateral to NMRCS, another electrode was placed "in parallel" (NMRCP).

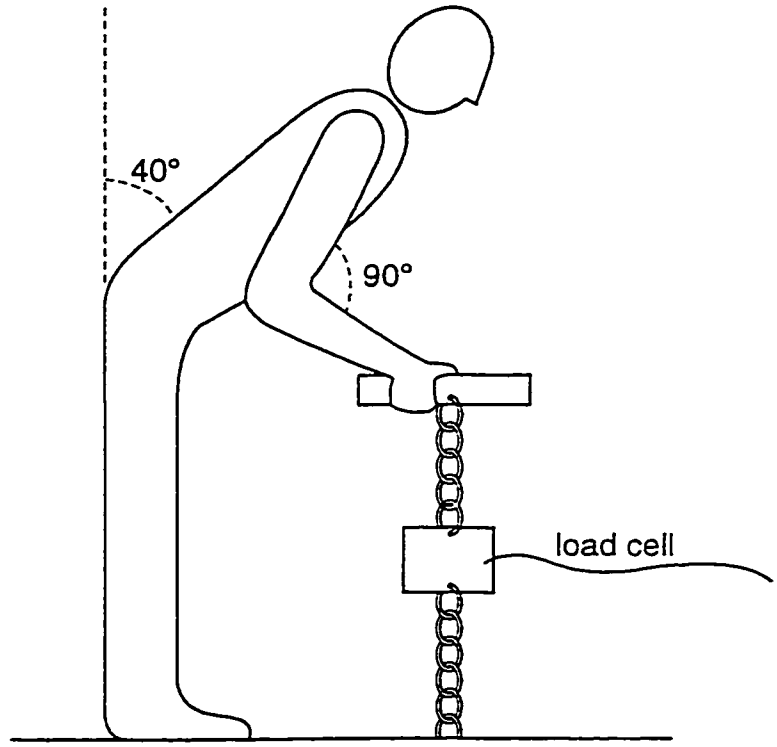

Figure 2.1. Posture of subjects during isometric pulling on a handle attached with a chain to the floor.

The signals picked up by the Miniature and HP electrodes were amplified with a gain 1000 (Measurement Systems Inc. - Ann Arbor, Michigan). SEMG signals from the NMRC electrodes were amplified separately (gain 2000). The SEMG signals from the graded loading trials (20%, 40%, 60%, 80%, and 100% MVC) were directly fed to a computer-based data acquisition system (MetraByte DAS 20 analogue to digital converter) operating at a sampling frequency 1000 Hz. The signal was high-pass filtered at 5 Hz and RMSed (RC 0.025 ms time constant). The average amplitude (AA) of the RMS EMG signal during the 5 s interval of the graded conditions was calculated for each channel. The power spectra were derived from the raw EMG signals using the fast Fourier transform algorithm. The total power (TP) and median frequency (MF) were analyzed. The fatigue trials were first recorded on a magnetic tape and subsequently played back into the computer. In order to obtain comparable cross-sectional data from the trials of various durations, the contractions were sampled at the 10th, 20th, 30th, ..., 100th percentiles, each for 2.1 s at 1000 Hz. Due to the limited capacity of the tape recorder only 8 channels were recorded in the fatigue trial. The raw data underwent the Fourier transform and the whole power spectra were analyzed for changes in TP and MF. Also, in order to investigate the changes in more detail, each power spectrum was divided into 10 frequency bands (5 - 30 Hz, 30 - 60 Hz, 60 - 90 Hz, etc.). In each frequency band the TP values at the failure point (9th time segment) were compared to the initial TP values (1st time segment). The difference was expressed as a percentage of the initial values.

The generated force was measured with a strain gauge force transducer (SM 500) attached to a digital display (ST-1, Prototype Design Fabrication Company) and the signal was also stored in the computer.

In order to test for significant differences between the variables (interelectrode distance, electrode type and electrode orientation), one-way ANOVA was performed on groups of channels with one dependent variable. Thus, for the influence of the distance, comparisons were made separately for the group of miniature electrodes "in series" (MS2, MS4, MS6, MS8), miniature electrodes "in parallel" (MP2, MP4) and the HP electrodes "in series" (HPS3, HPS6).

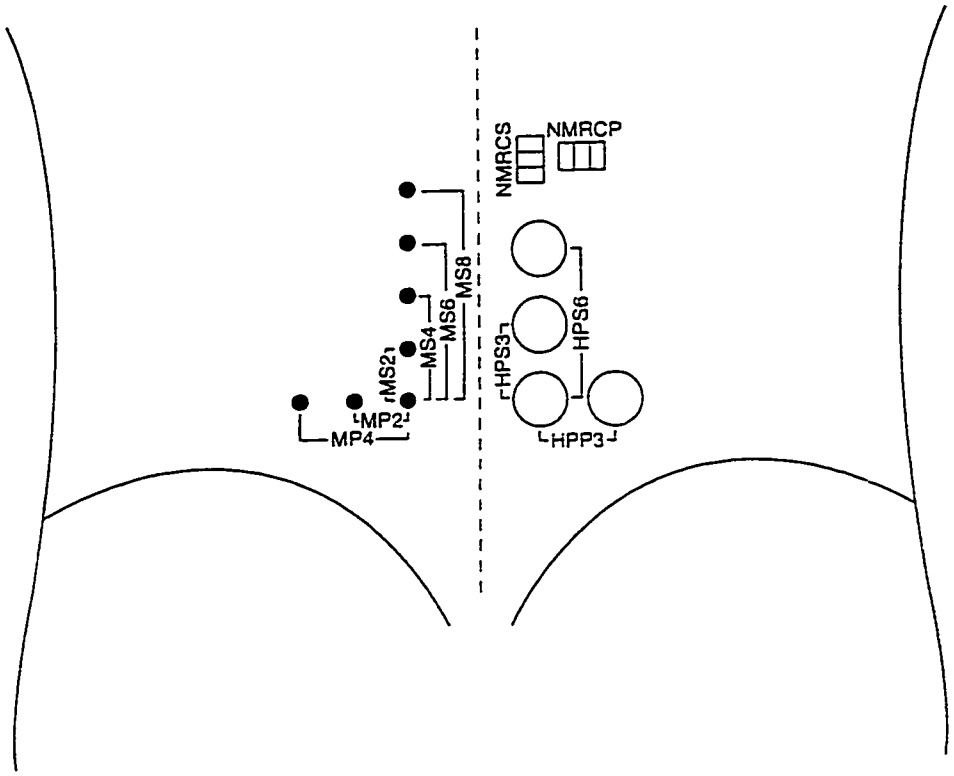

Figure 2.2. Placement of electrodes on the back of subject over the left and right erector spinae. NMRCP - NMRC electrodes in parallel, NMRCS - NMRC electrodes in series, HPP3 - HP electrodes in parallel 3 cm apart, HPS3 - HP electrodes in series 3 cm apart, HPS6 - HP electrodes in series 6 cm apart, MP2 - miniature electrodes in parallel 2 cm apart, MP4 - miniature electrodes in parallel 4 cm apart, MS2 - miniature electrodes in series 2 cm apart, MS4 - miniature electrodes in series 4 cm apart, MS6 - miniature electrodes in series 6 cm apart, MS8 - miniature electrodes in series 8 cm apart.

Given the placement of the electrodes, a pure effect of the electrode type could be investigated only between the miniature and the HP electrodes "in series", 6 cm apart (MS6, HPS6). However, for practical use, all three types were compared when each electrode pair was placed at a distance most frequently used with that type (1 cm for NMRC, 2 cm for Miniature, 3 cm for HP). Finally, the effect of the orientation was determined comparing channels "in series" with those "in parallel" (MS2-MP2, MS4-MP4, HPS3-HPP3, NMRCS-NMRCP). Significance of p was set at 0.05.

2.3 Results

2.3.1 Graded loading

EMG - Force relationship

With increasing grade of contraction AA and TP rose in all channels (Figures 2.3.A. and 2.3.B.). The correlation between AA and force was significant ($p < 0.001$) in all channels ($0.60 \leq r \leq 0.72$), except for NMRCS ($r = 0.40$). The relationship between TP and force followed a similar pattern. The correlation was significant in all cases ($0.59 \leq r \leq 0.68$), except NMRCS ($r = 0.39$). MF did not change significantly with increasing load (Figure 2.3.C.). However, at each contraction level MF differed significantly between the channels ($p < 0.001$).

Interelectrode distance

Comparisons between various channels revealed that with greater IED the AA and TP increased and MF decreased for both the miniature and HP electrodes. These trends reached significance for AA and TP at higher contraction levels (60% - 100% MVC) with miniature electrodes "in parallel" ($p < 0.01$). Significant difference in MF compared between IEDs was found with miniature electrodes "in series" ($p < 0.01$), except at 20% MVC.

Electrode orientation

AA and TP had greater values "in series" than "in parallel", with the exception of the NMRC electrodes where the greater values were found "in parallel". These trends, however, never reached significance. The orientation affected MF in the miniature and the HP electrodes. The values were smaller "in parallel" than "in series" for 20 - 60% MVC ($p < 0.01$). No influence of the orientation on the MF was observed in the NMRC channels.

As compared to the upright standing, at 40° trunk flexion the HP and the miniature electrodes "in series" were considerably displaced. The displacement was greater between electrodes with greater initial IED. Electrodes "in parallel" did not change their IED (Table 2.1.).

Electrode type

Comparison between the miniature and the HP electrodes "in series" 6 cm apart revealed no difference between these two electrode types. However, the difference in AA, TP and MF between all three types was significant ($p < 0.001$) if they were placed at the distance commonly used with each type (1 cm for NMRC, 2 cm for Miniature, 3 cm for HP).

2.3.2 Fatigue trial

MF decreased during the course of the fatigue trial in all channels (Figure 2.4.). The slopes ranged between 0.02 and 0.07 Hz per % of task duration for all electrode types and orientations. However, the drop was statistically significant only in the NMRCP channel with a slope of 0.18 ($p < 0.001$). As in graded loading, the channels arranged "in parallel" recorded lower MF than those "in series" and MF was lower in channels with greater IED

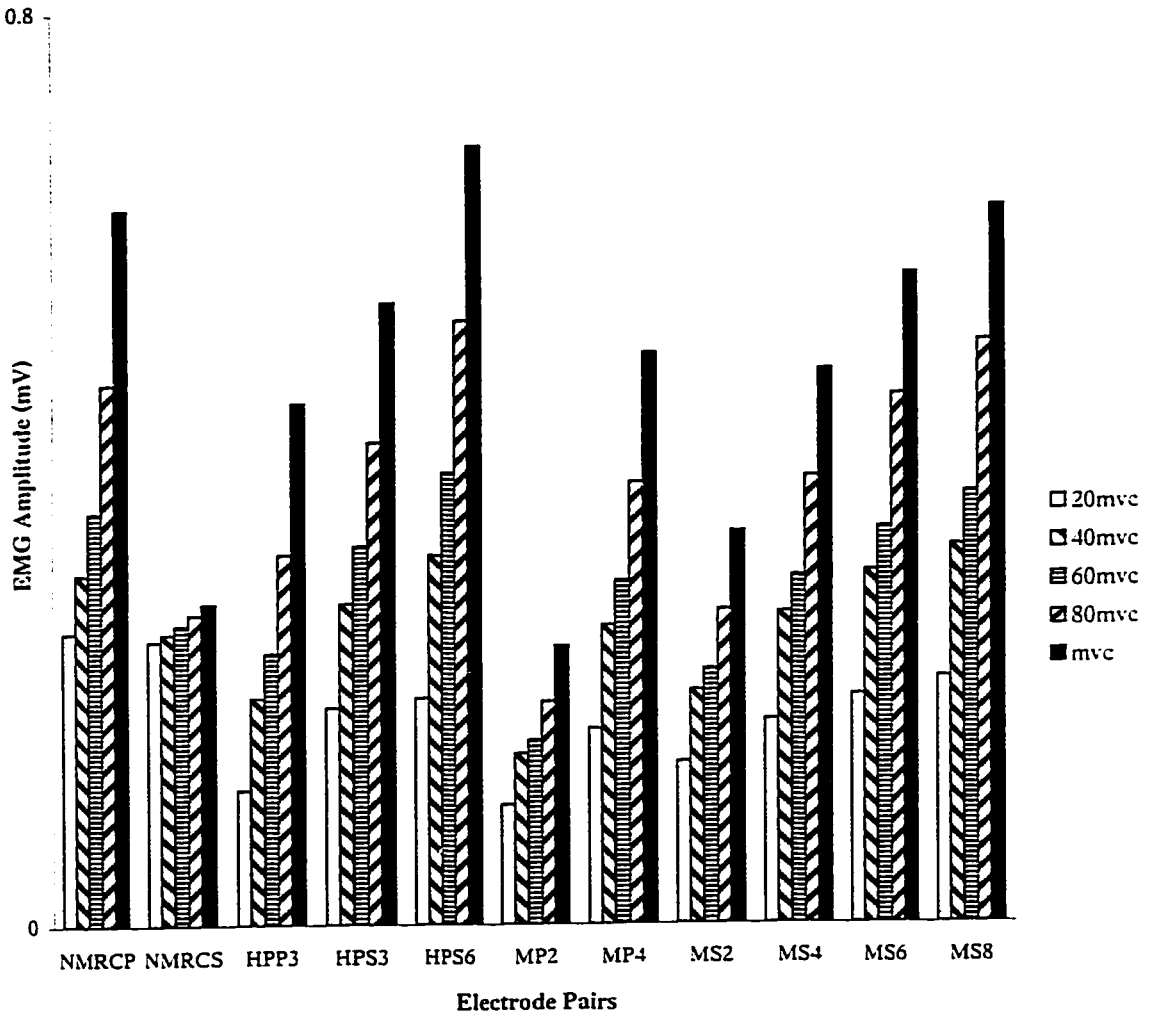

Figure 2.3.A. Mean average EMG amplitude of the 10 subjects during graded isometric contractions. NMRCP - NMRC electrodes in parallel, NMRCS - NMRC electrodes in series, HPP3 - HP electrodes in parallel 3 cm apart, HPS3 - HP electrodes in series 3 cm apart, HPS6 - HP electrodes in series 6 cm apart, MP2 - miniature electrodes in parallel 2 cm apart, MP4 - miniature electrodes in parallel 4 cm apart, MS2 - miniature electrodes in series 2 cm apart, MS4 - miniature electrodes in series 4 cm apart, MS6 - miniature electrodes in series 6 cm apart, MS8 - miniature electrodes in series 8 cm apart, 20mvc - 20% of maximum voluntary contraction, 40mvc - 40% of maximum voluntary contraction, 60mvc - 60% of maximum voluntary contraction, 80mvc - 80% of maximum voluntary contraction, mvc - maximum voluntary contraction.

Mean Total Power

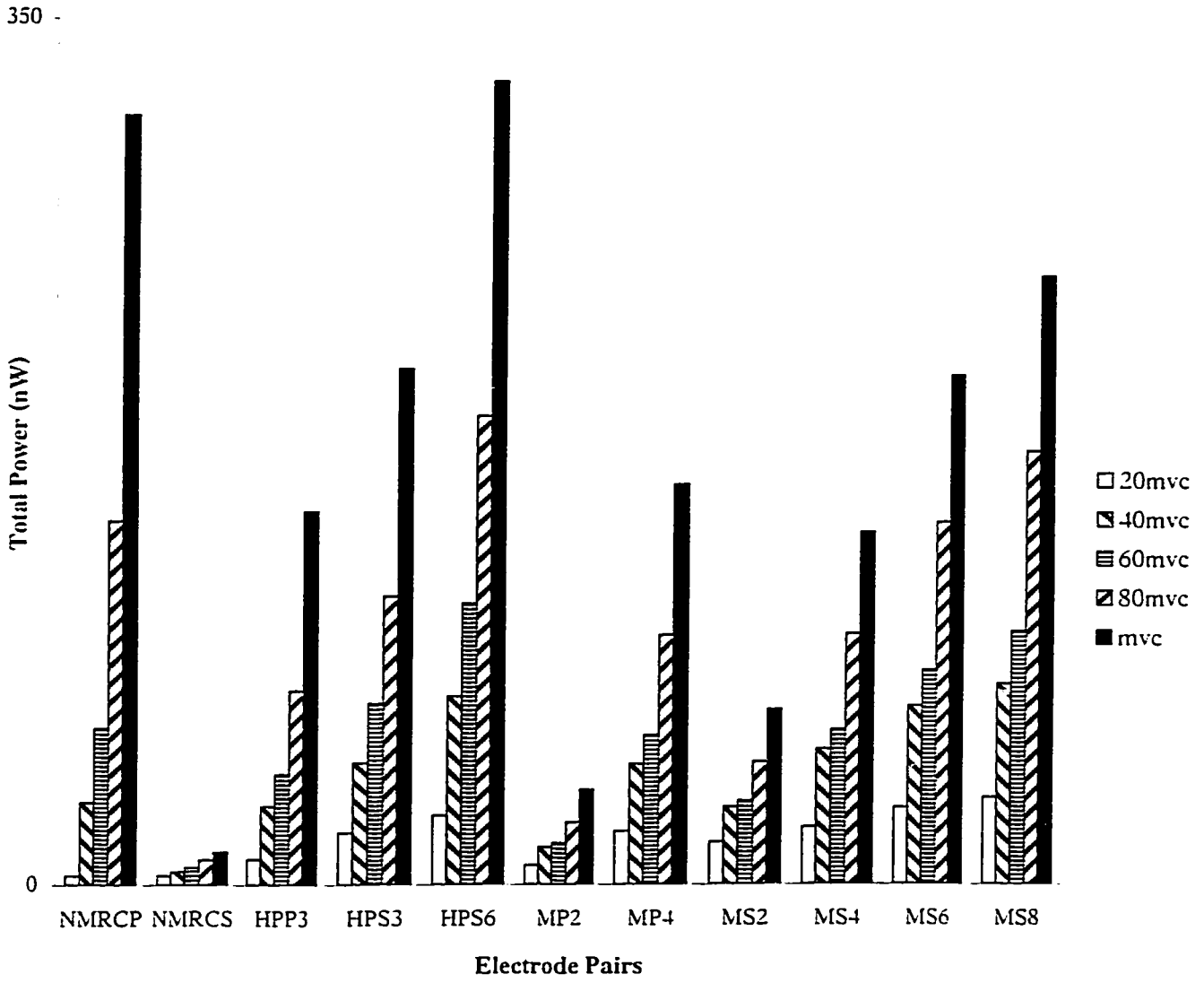

Figure 2.3.B. Mean total power of the 10 subjects during graded isometric contractions. NMRCP - NMRC electrodes in parallel, NMRCS - NMRC electrodes in series, HPP3 - HP electrodes in parallel 3 cm apart, HPS3 - HP electrodes in series 3 cm apart, HPS6 - HP electrodes in series 6 cm apart, MP2 - miniature electrodes in parallel 2 cm apart, MP4 - miniature electrodes in parallel 4 cm apart, MS2 - miniature electrodes in series 2 cm apart, MS4 - miniature electrodes in series 4 cm apart, MS6 - miniature electrodes in series 6 cm apart, MS8 - miniature electrodes in series 8 cm apart, 20mvc - 20% of maximum voluntary contraction, 40mvc - 40% of maximum voluntary contraction, 60mvc - 60% of maximum voluntary contraction, 80mvc - 80% of maximum voluntary contraction, mvc - maximum voluntary contraction.

Mean Median Frequency

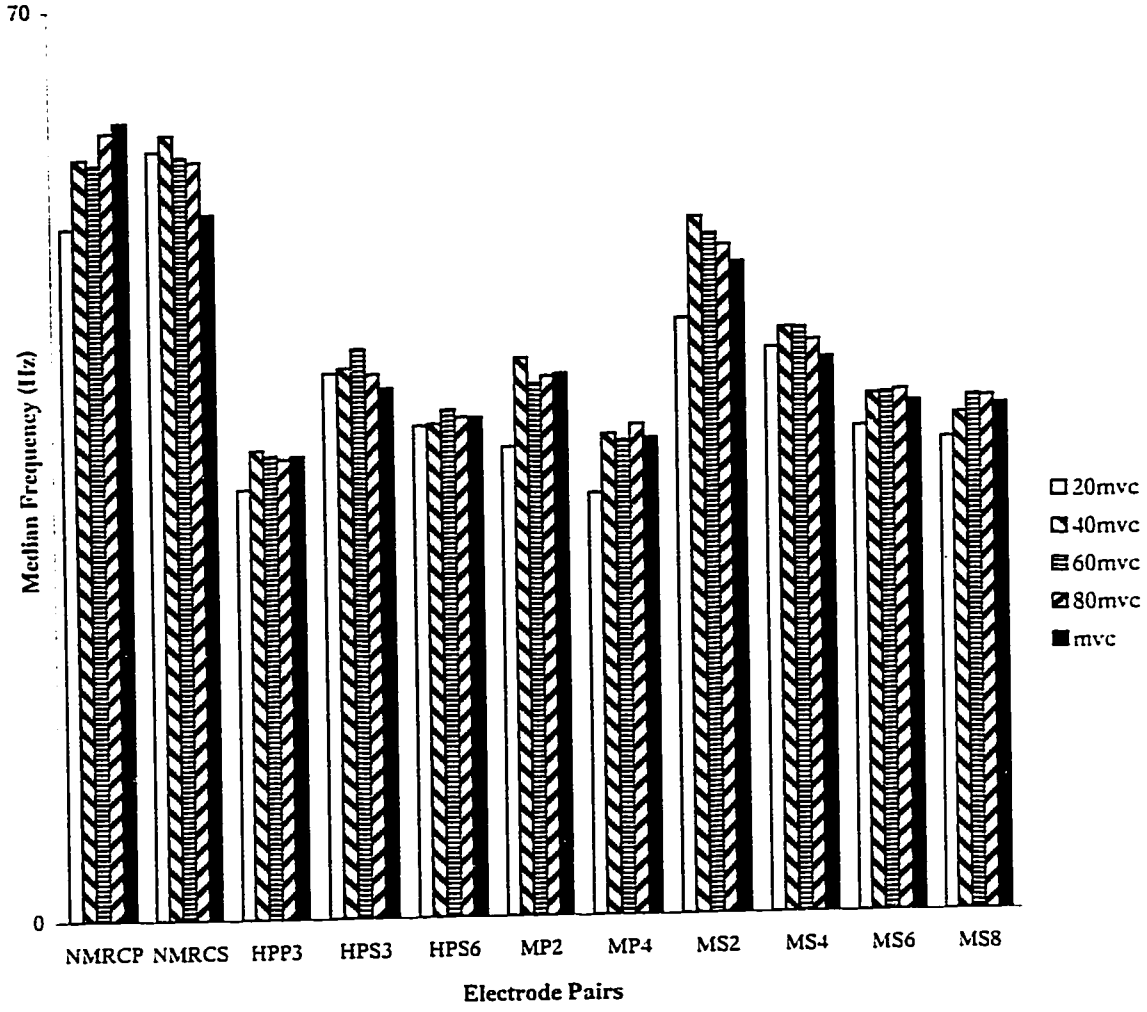

Figure 2.3.C. Mean median frequency of the 10 subjects during graded isometric contractions. NMRCP - NMRC electrodes in parallel, NMRCS - NMRC electrodes in series, HPP3 - HP electrodes in parallel 3 cm apart, HPS3 - HP electrodes in series 3 cm apart, HPS6 - HP electrodes in series 6 cm apart, MP2 - miniature electrodes in parallel 2 cm apart, MP4 - miniature electrodes in parallel 4 cm apart, MS2 - miniature electrodes in series 2 cm apart, MS4 - miniature electrodes in series 4 cm apart, MS6 - miniature electrodes in series 6 cm apart, MS8 - miniature electrodes in series 8 cm apart, 20mvc - 20% of maximum voluntary contraction, 40mvc - 40% of maximum voluntary contraction, 60mvc - 60% of maximum voluntary contraction, 80mvc - 80% of maximum voluntary contraction, mvc - maximum voluntary contraction.

Table 2.1. Displacement of electrodes in cm during change from upright position into 40° trunk flexion.

Electrodes	IEDup (cm)	IED40 (cm)	IED40 - IEDup (cm)
NMRCP	1.0	1.0	0
NMRCS	1.0	1.0	0
HPP3	3.0	3.0	0
HPS3	3.0	4.5	1.5
HPS6	6.0	8.9	2.9
MP2	2.0	2.0	0
MP4	4.0	4.0	0
MS2	2.0	2.6	0.6
MS4	4.0	5.1	1.1
MS6	6.0	7.6	1.6
MS8	8.0	10.3	2.3

IEDup - interelectrode distance in the initial upright position.
IED40 - interelectrode distance at 40° trunk flexion.
Electrode abbreviations are explained in text.

TP increased during the course of the prolonged holding. The increase was more prominent towards the end of the trial (Figure 2.5.). A closer analysis revealed that during prolonged holding the changes in TP were not equally distributed among the ten frequency bands (Figure 2.6.). Although in most channels TP rose by increasing the power over the whole range of the spectra, the largest relative increase was found below 60 Hz. In channel NMRCP the energy increase took place only below 60 Hz while the power content in the upper section of the power spectra decreased. In NMRCS the augmentation of power in the lower frequencies did not compensate sufficiently for the power drop in the high-frequency bands resulting in a slight decrease in total power.

2.4 Discussion

2.4.1 Graded loading

EMG - force relationship

Although the measured force undoubtedly resulted from activation of many muscles, AA and TP registered from ES showed to be a linear function of the changing force. A relationship between force and the amplitude of an RMS EMG signal in short isometric contractions has been documented for various muscles (Lippold, 1952; Moritani & De Vries, 1978; Woods & Bigland-Ritchie, 1983; Lawrence & De Luca, 1983), including the erector spinae (Rosenburg & Seidel, 1989).

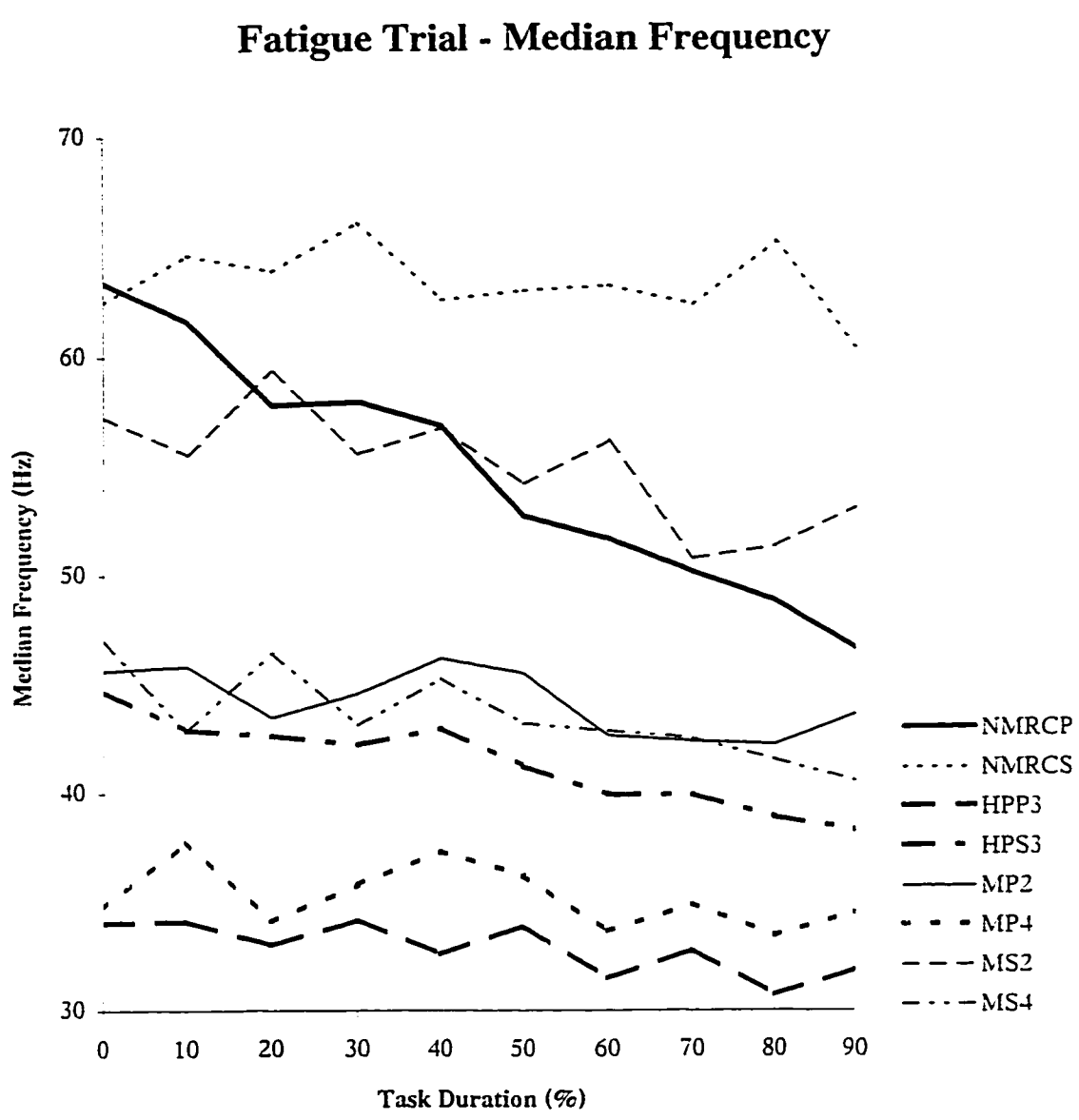

Figure 2.4. Mean median frequency during prolonged holding. NMRCP - NMRC electrodes in parallel, NMRCS - NMRC electrodes in series, HPP3 - HP electrodes in parallel 3 cm apart, HPS3 - HP electrodes in series 3 cm apart, MP2 - miniature electrodes in parallel 2 cm apart, MP4 - miniature electrodes in parallel 4 cm apart, MS2 - miniature electrodes in series 2 cm apart, MS4 - miniature electrodes in series 4 cm apart.

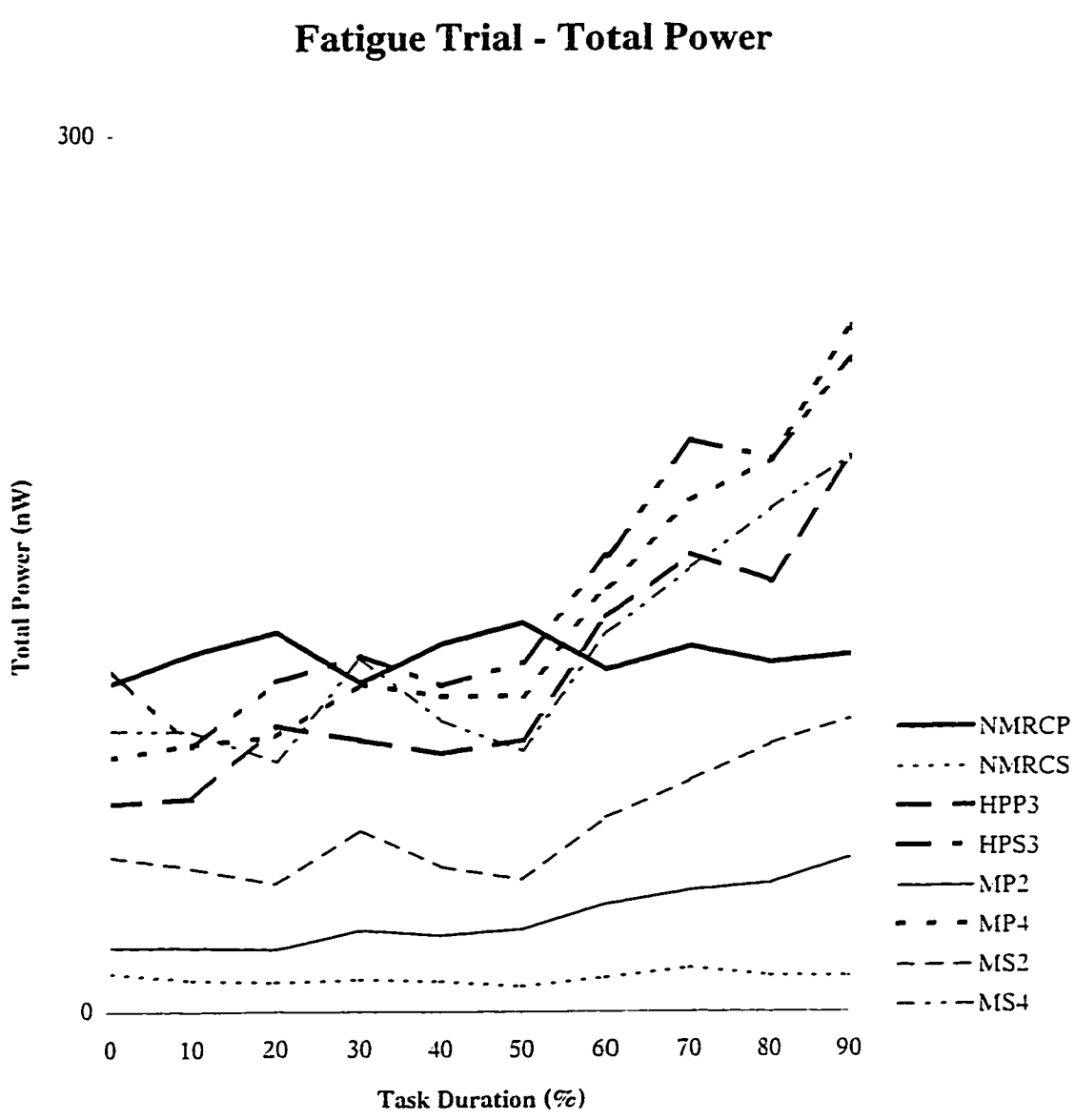

Figure 2.5. Mean total power during prolonged holding. NMRCP - NMRC electrodes in parallel, NMRCS - NMRC electrodes in series, HPP3 - HP electrodes in parallel 3 cm apart, HPS3 - HP electrodes in series 3 cm apart, MP2 - miniature electrodes in parallel 2 cm apart, MP4 - miniature electrodes in parallel 4 cm apart, MS2 - miniature electrodes in series 2 cm apart, MS4 - miniature electrodes in series 4 cm apart.

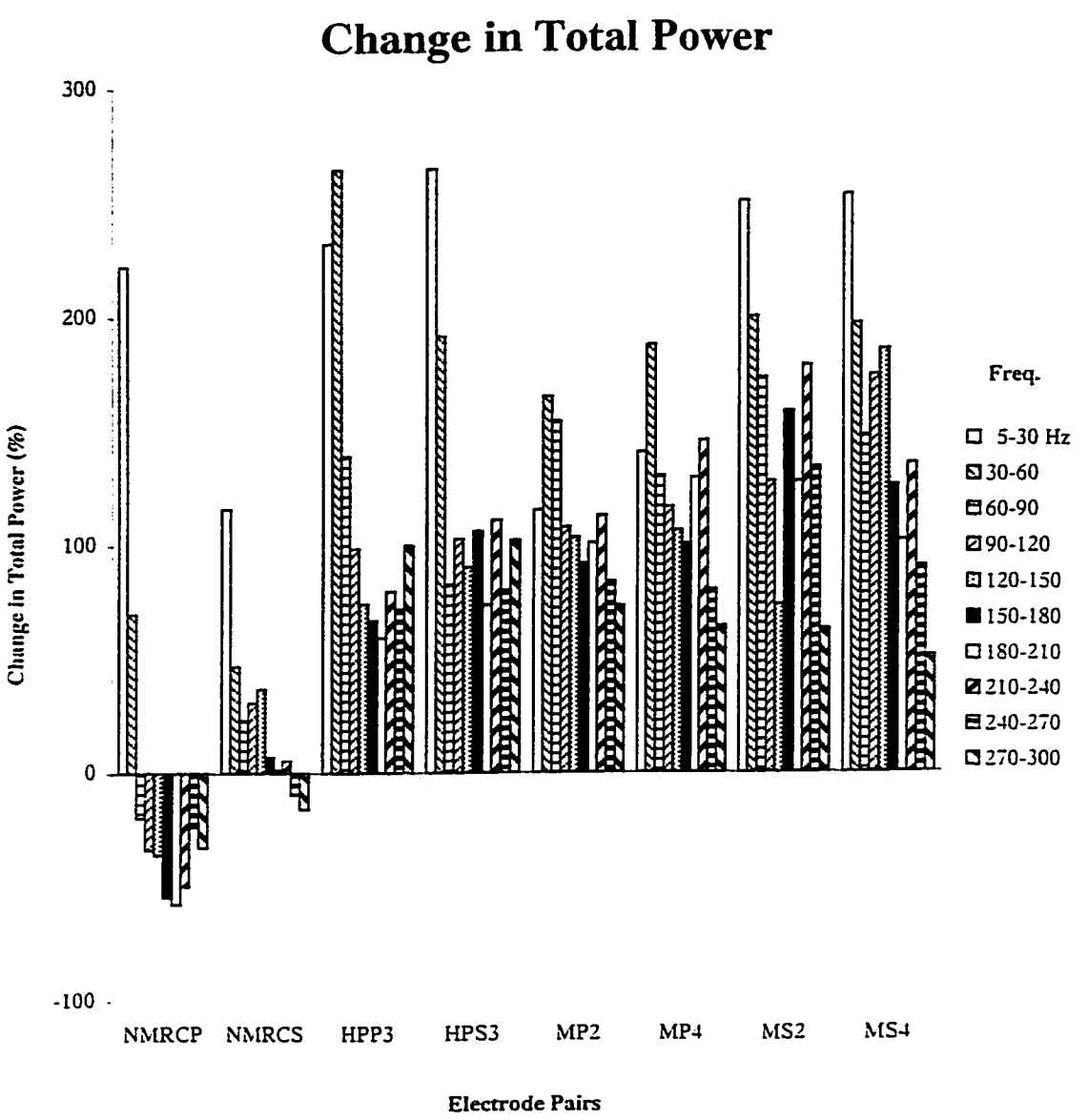

Figure 2.6. Relative change in the mean total power for each electrode pair and frequency band during prolonged holding. The values represent change (in percent) of the total power at the failure point compared to the initial values. NMRCP - NMRC electrodes in parallel, NMRCS - NMRC electrodes in series, HPP3 - HP electrodes in parallel 3 cm apart, HPS3 - HP electrodes in series 3 cm apart, MP2 - miniature electrodes in parallel 2 cm apart, MP4 - miniature electrodes in parallel 4 cm apart, MS2 - miniature electrodes in series 2 cm apart, MS4 - miniature electrodes in series 4 cm apart.

Also frequency spectra have been found to reflect the level of muscle contraction. Most studies have been performed on the limbs where MF of the power spectrum increased linearly with force, due to the fact that the largest units with highest conduction velocities were recruited last (Bolodeau, Arsenault, Gravel & Bourbonais, 1990; Broman, Bilotto & De Luca, 1985). However, an opposite pattern has been observed in the erector spinae (Roy, De Luca & Casavant, 1989). Here MF decreased with increasing force. Furthermore, some studies on the limb muscles report no dependence of MF on load (Bazzy, Korten & Haddad, 1986). Therefore, the relationship between MF and force is rather controversial. Dolan and colleagues (Dolan, Mannion & Adams, 1995) have found no reduction of MF with increasing force in ES and ascribed this to the fact that the muscle was tested in a flexed position. It has been documented before that changes in muscle length affect the mechanism of force generation (Rosenburg & Seidel, 1989). Also in the present study, performed at 40° flexion, no direct relationship between MF and force was discovered. It has been proposed that MF changes with the level of contraction only as long as the muscular tension is graded mainly by recruitment of fresh fibres, while a change in firing rates has little effect (Solomonow *et al.,* 1990; Hagberg & Ericson, 1982). If change in MF closely reflects a recruitment pattern of motor units, our finding indicates that ES may be different from other large muscles, where new motor units are recruited even at forces close to MVC (Kukulka & Clamann, 1981; De Luca, Le Fever, McCue & Xenakis, 1982).

Interelectrode distance

A larger IED allows recording from a larger volume of tissue, the vicinity of electrodes being judged as a hemispheric space with a radius equal to the electrode separation (Basmajian & Blumenstein, 1980; Lynn, Bettles, Hughes & Johnson, 1978; Zipp, 1982). Therefore, a closer spacing between the electrodes reduces the amount of the recorded EMG signal which is reflected in a lower mean voltage (Laurig, 1970). Lindstrom and Magnusson (1977) have postulated that bipolar electrodes act as a high-pass filter whose cut-off frequency is determined by IED. The closer the electrodes, the

higher the cut-off frequency. Broadening of the power spectra on the right side results in the increase in MF.

Electrode orientation

The dipole created by the electrical source is a vector parallel to the direction of muscle fibres and bipolar electrodes function as an antenna. The orientation of the antenna axis in relation to the dipole axis determines the recorded signal. Theoretically, for records from a single muscle fibre the highest amplitude is registered if the electrode axis lies parallel to the dipole axis (i.e. configuration "in series" in the present study). Conversely, if these axes are perpendicular to each other, no potential gradient is registered. The surface electrodes record a summed signal from many fibres firing at different rates with a phase shift which is further modified depending on the distance between the muscle fibres and the recording electrodes. Thus, a placement of electrodes "in parallel" does not lead to a total cancellation but only an attenuation of the EMG signal.

2.4.2 Fatigue trial

MF was used to monitor muscle activity during the prolonged holding in this study, as it is a more reliable measure of muscle fatigue than EMG amplitude (De Luca, 1984). A shift of the EMG power spectrum to lower frequencies has been considered as evidence of neuromuscular fatigue (Piper, 1912; Cobb & Forbes, 1923; Petrofsky & Lind, 1980; Stulen & De Luca, 1981; Naeije & Zorn, 1981; Palla & Ash, 1981; Arendt-Nielsen & Mills, 1985). Among the factors dictating the shape of the power spectrum are the conduction velocity, the fibre radius, the number of fibres per motor unit, the position of the innervation zone, the electrode-to-muscle distance etc. (Lindstrom & Magnusson, 1977). The shift of MF to the left is supposed to be due mainly to the reduced conduction velocity of the muscle fibres during fatigue. The relationship between the median frequency and the conduction velocity has been found to be linear (Stulen & De Luca, 1981; Sadoyama, Masuda & Miyamo, 1983). It is believed that the decrease in the conduction velocity of the muscle fibres may be an effect of a local increase in the

production of metabolites accompanying exercise. The accumulation of metabolites is further enhanced by the relative ischemia under prolonged isometric contractions (Richardson, 1981). The change in the conduction velocity is probably not the only reason for the frequency shift. Other factors like synchronization of the motor units or decrease in their firing rate also play a part (Bigland-Ritchie, Donovan & Roussos, 1981; Stulen & De Luca, 1978).

It has been observed that TP of the EMG spectrum increases during a fatiguing contraction at a constant force level (Kwatny, Thomas & Kwatny, 1970; Kadefors, Kaiser & Petersen, 1968). In the present study, TP increased in two distinguishable phases in most channels. The abrupt increase in the total power in the second half of the prolonged holding took place mainly in the lower range of the spectrum. Because the tissue acts as a low-pass filter, more energy is transferred to the electrodes as the frequency content of the signal shifts to lower frequencies. Another possible argument is that recruitment of new units occurred as the employed units were no longer capable of maintaining constant force (Clamann & Broecker, 1979).

2.5 Concluding comments

The goal of this study was to discover which type of electrodes is most appropriate for surface recording and how should the electrodes be placed in order to find the optimum combination between the accuracy of the SEMG signal and the electrode displacement. Good results were obtained with miniature and the HP electrodes independently of their placement. The orientation "in parallel" gave smaller amplitude and lower frequencies but the changes during the graded loading and fatigue were similar to the more usual and generally accepted orientation "in series". Moreover, the constant IED of electrodes "in parallel" during the trunk flexion suggests that this placement should not be automatically disregarded as improper. The SEMG signals obtained with the NMRC electrodes were less consistent than the signals from the miniature and HP electrodes. Several factors may be responsible. For "lack of space" over the erector spinae the NMRC electrodes were placed at the level of the tenth thoracic vertebra. As

there are functional differences between various levels of ES, recordings from the thoracic level are hardly comparable with those from the lumbar portion. Another observation made with the NMRC electrodes was that they picked up a considerable amount of contact noise. These were the only electrodes affixed to the skin with adhesive tapes and no gel. The distance between the two active surfaces was fixed and could not follow the stretching of the skin during movement. Considering these facts, the NMRC electrodes may not be suitable for studies where their sliding on the skin cannot be prevented.

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CHAPTER 3.

Electromyographic response of the trunk muscles to postural perturbation in sitting subjects[1]

3.1 Introduction

Equilibrium reactions are automatic compensatory head, trunk and limb movements used to retain or regain vertical alignment of the subject's centre of gravity with the base of support. These reactions make upright sitting, stance and gait possible and provide the background postural set necessary for development and execution of all skilled motor responses.

In the past, studies on equilibrium reactions using objective measuring methods have been performed on standing subjects and have concentrated on activation strategies and recruitment patterns in the muscles of the lower limb (Mauritz, Dietz & Haller, 1980; Horak & Nashner, 1986; Horak, Nashner & Diener, 1990). Little is known about the function of the human trunk muscles as stabilizers of the upper body during sudden balance perturbations, although their role in locomotion has been studied during various activities (Biederman, Shanks, Forrest & Inglis, 1991; Grabiner, Koh & El Ghazawi, 1992; Cassisi, Robinson, O'Conner & MacMillan, 1993; Cooper, Stokes, Sweet, Taylor & Jayson, 1983; De Luca, 1993; Haig, Talley, Grobler & Le Breck, 1993; van Dieen, Toussaint, Thissen & van de Ven, 1993). Since trunk muscles are often co-activated during asymmetric voluntary efforts (Andersson, Ortengren & Herberts, 1977;

[1] A version of this chapter has been published. Zedka, M., Kumar, S. & Narayan, Y. (1998). Electromyographic response of the trunk muscles to postural perturbation in sitting subjects. Journal of Electromyography and Kinesiology 8, 3-10.

Basmajian, 1978; Morris, Benner & Lucas, 1962; Pope, Andersson, Broman, Svensson & Zetterberg, 1986), it is important to find out whether they respond to postural perturbation in the same way. Trunk muscle activity during unexpected loading and unloading of the upper torso in a standing position was studied by Carlson and colleagues (Carlson, Nilsson, Thorstensson & Zomlefer, 1981). The standing posture might have been responsible for the observed variabilities in the activation of paraspinal muscles. Cresswell et al. (Cresswell, Oddsson & Thorstensson, 1994) used a similar experimental design to further study the role of the trunk muscles in the increase of intra-abdominal pressure, presumably used for stabilizing the trunk.

Despite the fact that sitting is a common everyday activity, there is a lack of electromyographic data on balance tasks in this position. The sitting position also allows to study more directly the balance reactions of the trunk without compensatory movements of the lower limbs. Forssberg and Hirschfeld (Forssberg & Hirschfeld, 1994) presented a study on sitting adult subjects exposed to balance perturbations in the sagittal plane. A movable platform was used to deliver translations and rotations in the sagittal plane. They found that the threshold for postural adjustments was higher during sitting than standing (8° at 50°. s^{-1} for backward rotations). Comparison between the electromyographic data from the forward translation and the backward rotation showed similar activation patterns, although the kinematics of the recorded body segments were different. The only common element in both types of perturbation was the backward rotation of the pelvis which occurred early enough (10 - 15 ms after the platform acceleration) to be considered as a possible triggering moment of postural adjustments in sitting.

The present study was conducted to obtain more information concerning responses to balance perturbation both in the sagittal and the frontal planes. The goal was to quantitatively describe the human equilibrium reactions in sitting healthy subjects in terms of electromyographic activity of their trunk muscles as a result of: a) velocity of perturbation, b) direction of perturbation, c) expectation of perturbation, and d) visual feedback.

3. 2 Methods

3.2.1 Subjects

Five healthy subjects (2 females and 3 males), with no history of back injury, back pain or any other affliction of the trunk muscles participated in the study. They were recruited from student population and ranged between 21 and 29 years of age. The demographic data are presented in Table 3.1.

3.2.2. Experimental paradigm

A special balance seat allowing rotations in the sagittal (forward-backward) and frontal (left-right) planes was designed and fabricated for generating perturbations (Figure 3.1.). The top platform (60 cm x 80 cm) was situated 35 cm above the ground. In the axis of rotation of the seat a potentiometer (5% accuracy for full-scale deflection of 180°) measuring the angular displacement was placed. A rotatory table mounted on the seat platform allowed to switch between perturbations in the sagittal and frontal planes by turning the sitting subject by 90°. A lever for manual delivery of tilts was attached to the seat. Subjects were centred on the platform sitting comfortably with their knees slightly flexed, heels resting on a low friction dolly to reduce the support due to the feet by reducing friction forces with the ground. Their pelves were fastened to the seat with a safety belt. The subjects were asked to look straight forward and let their hands rest in their laps. The response to each perturbation was supposed to be spontaneous. Therefore, the subjects received no instruction to maintain equilibrium.

The influence of the speed and direction of the perturbation on the magnitude of the EMG signals was investigated. The effect of vision was determined by conducting trials with subjects blindfolded and with eyes open. It was also tested whether information about the speed and direction of the upcoming perturbation given to a blindfolded subject changed the EMG response. This variable was called expectation.

Table 3.1. Anthropometric data of the experimental sample

Subject	Gender	Height (cm)	Weight (kg)	Age (years)
E.Z.	F	170	74	24
L.W.	F	172	67	29
C.C.	M	176	72	24
K.W.	M	177	74	24
M.F.	M	178	74	21

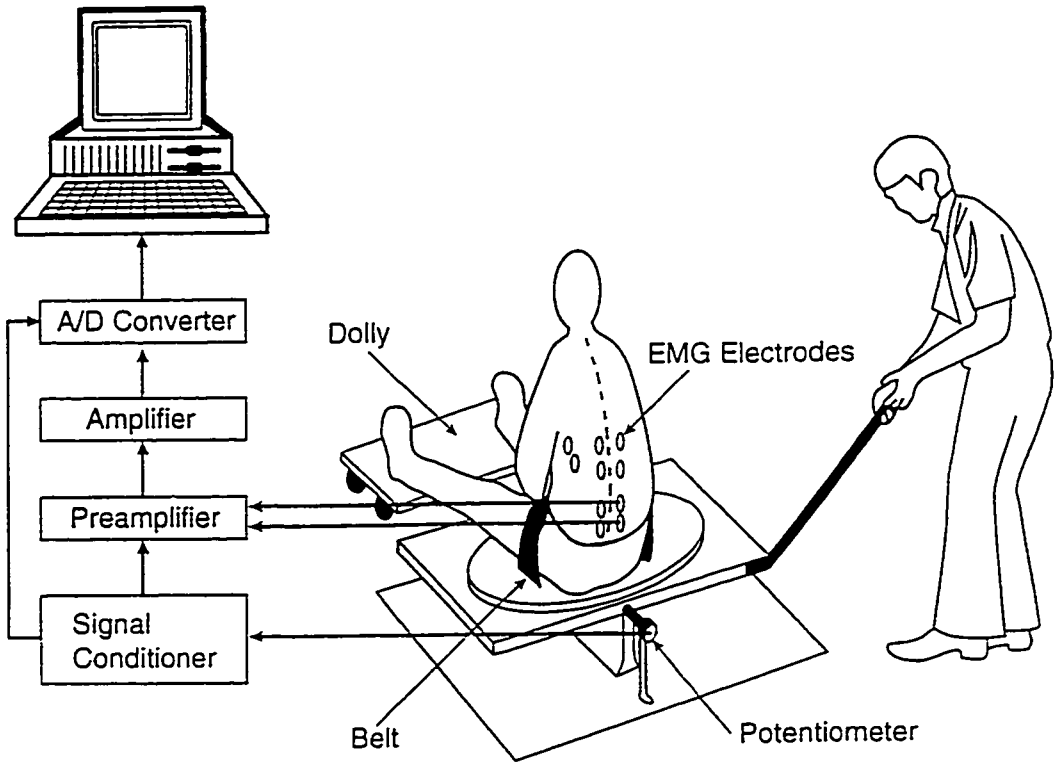

Figure 3.1. Schematic drawing of the experimental setup.

Manual perturbations at fast and slow velocities were delivered. The experimenter practiced delivery of perturbation till consistent velocity motion was achieved. In the fast conditions the angular velocity of the perturbation was 26°. s^{-1} (SD ± 6°. s^{-1}) and its amplitude was 10° For the slow perturbations the angular velocity was 8°. s^{-1} (SD ± 4°. s^{-1}) and the displacement was 20°. At both velocities rotational perturbations were directed forward, backward, left and right. Following the initial displacement, the seat was left in the tilted position. Only the slow conditions were used to compare responses with and without visual feedback. The fast conditions were always performed with a blindfold and were separated into expected and unexpected trials. In the expected trials, the subjects were told the direction and speed of the next tilt. Then the experimenter counted to three and displaced the seat. In unexpected trials no clues were given to the subject prior to the perturbation. All conditions were tested in a random order.

3.2.3 Recording and analysis

Electromyographic activity was recorded bilaterally from the musculus erector spinae at two levels (T10, L3), the obliquus abdominis externus (EO), the obliquus abdominis internus (IO), the rectus abdominis (RA) and the latissimus dorsi (LD). After appropriate skin preparation with isopropyl alcohol, gelled HP Adult Monitoring Disposable Electrodes (14445 C) were placed in the direction of the muscle fibres in bipolar array, 3 cm apart (Figure 3.2.). The EMG signals were preamplified and amplified (total gain 1000), fed to the computer through Metrabyte DAS 20 analogue to digital converter (sampling frequency 500 Hz), band pass filtered between 5 Hz - 250 Hz, and RMSed (25 ms RC time constant). Preliminary tests have shown that in the trunk muscles the frequency components of the EMG signals rarely surpassed 200 Hz. The cut-off frequency 5 Hz was chosen to eliminate movement artifact. In the fast conditions a two-second activity window was displayed on a screen for quantitative analysis. The window encompassed 0.2 s preceding the seat displacement and 1.8 s following it.

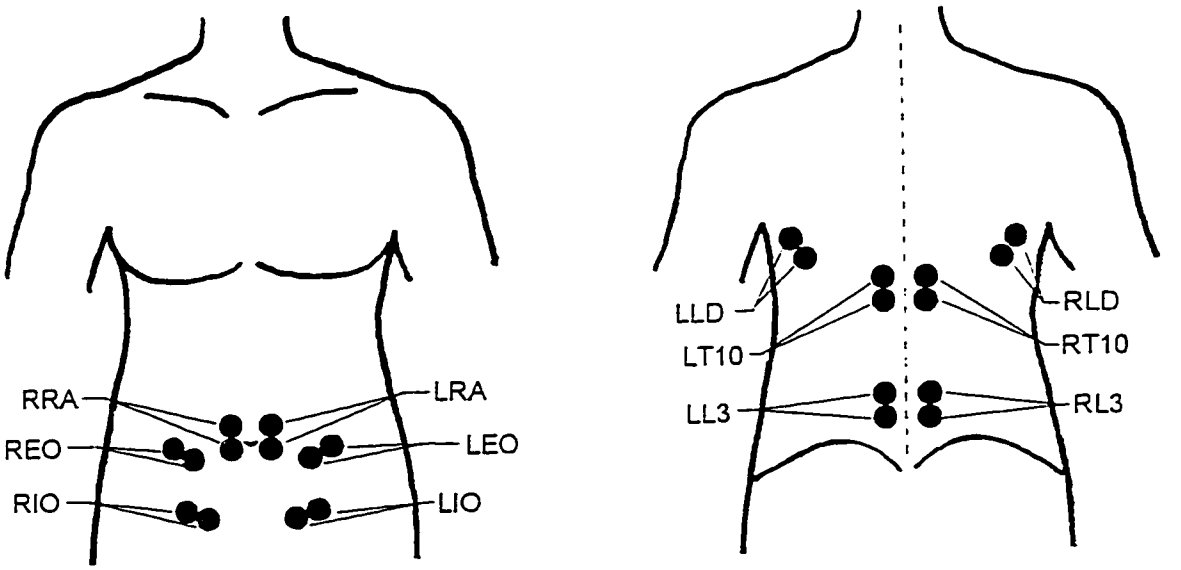

Figure 3.2. Placement of electrodes. RRA, right rectus abdominis; REO, right external oblique; RIO, right internal oblique; LRA, left rectus abdominis; LEO, left external oblique; LIO, left internal oblique; RLD, right latissimus dorsi; RT10, right erector spinae at T10 level; RL3, right erector spinae at L3 level; LLD, left latissimus dorsi; LT10, left erector spinae at T10 level; LL3, left erector spinae at L3 level.

This time interval was chosen to cover EMG activity shortly before, during, and after the seat perturbation. In the slow conditions the activity window corresponded with the onset and offset of the seat displacement which lasted three seconds. All EMG activity related to the seat perturbation started and ended within this interval. For each channel the magnitude of the EMG signal was plotted against time.

The electrical signal of the rotary perturbation was fed to the computer together with the EMG signals. The slope of the angular displacement curve served for the calculation of the angular velocities of the fast and slow perturbations.

A statistical analysis of the EMG parameters was carried out to calculate descriptive statistics and analysis of variance. The simple factorial ANOVA was used to test for significant differences between variables (velocity, direction, expectation, visual feedback). The significance of p was set at the 5% level.

3.3 Results

The magnitude analysis did not reveal any significant effect of expectation or visual feedback on the EMG patterns. Therefore, the data from expected and unexpected trials were pooled. Similarly, the data from trials with eyes open were pooled with those without visual feedback.

The direction of rotation specifically influenced the EMG patterns. For each direction certain muscles generated similar EMG patterns according to their location on the trunk. All muscles could be grouped into four quadrants, each quadrant containing three muscles (Figure 3.3.). In order to show the overall EMG response of the muscles at ten equal time segments of the activity window, the magnitude was plotted over time. Before the perturbation some background EMG activity was present in the trunk muscles, which was higher in the back than in the abdominal muscles. Fast backward rotations of the seat evoked a contraction of the abdominals and a simultaneous relaxation of the back muscles (Figure 3.4.A.). The back muscles resumed their activity after the tilt was finished and new levels of EMG were established. The opposite was seen during the fast forward tilts (Figure 3.4.B.). Here the abdominal muscles retained an amplitude near

their resting values whereas the back muscles responded with a contraction which decreased after the seat reached the final position. During rotations in the sagittal plane, there was a marked symmetry between the EMG activities on the right and left sides.

A phasic antagonistic pattern was observed in the paraspinal muscles (T10, L3) and occasionally also in the latissimus dorsi during fast rotations to the left or right. When a perturbation to the left was delivered, the right back muscles contracted, whereas the left back muscles relaxed. A later increase in the left back EMG after the initial relaxation corresponded with a decrease in activity on the right side (Figure 3.4.C.). An opposite pattern was seen during right perturbations (Figure 3.4.D.). In no condition an alternating EMG activity was observed between the right and left abdominal muscles (EO, IO, RA).

During slow backward rotations, the EMG magnitude in the abdominal muscles gradually increased with the ongoing seat displacement. In the initial response to the perturbation a shallow dip was present in the back muscle EMG (Figure 3.5.A.). Slow rotations forward resulted in a slight activity increase in both dorsal and abdominal muscles (Figure 3.5.B.). Rotations sideways led to an asymmetrical activation of the right and left back muscles but a symmetrical activation of the abdominal wall (Figures 3.5.C. and 3.5.D.). In response to slow perturbations the muscles showed rather tonic activation with no phasic patterns.

3.4 Discussion

The goal of this study was to quantitatively describe the responses of the trunk muscles to balance perturbations during sitting. The EMG analysis showed that in various conditions different muscles responded with different intensities. Their activation patterns regularly reflected the location of the muscle on the trunk with respect to the direction of perturbation.

The EMG patterns found during the rotations in the sagittal plane were marked for their left-right symmetry. This finding was expected in the healthy population. During backward rotations the initial relaxation of the back muscles and contraction of

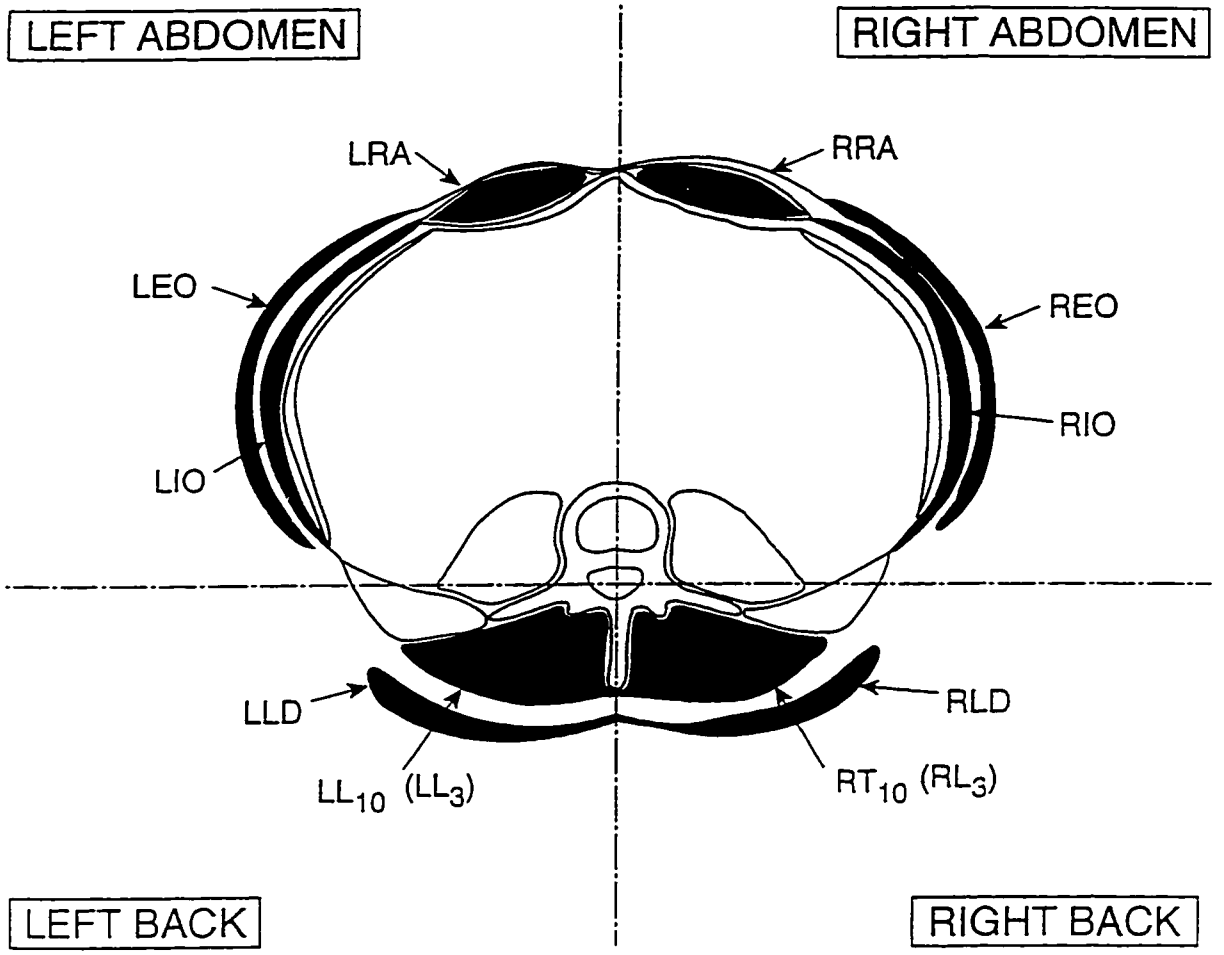

Figure 3.3. Division of the trunk muscles into four quadrants. RRA, right rectus abdominis; REO, right external oblique; RIO, right internal oblique; LRA, left rectus abdominis; LEO, left external oblique; LIO, left internal oblique; RLD, right latissimus dorsi; RT10, right erector spinae at T10 level; RL3, right erector spinae at L3 level; LLD, left latissimus dorsi; LT10, left erector spinae at T10 level; LL3, left erector spinae at L3 level.

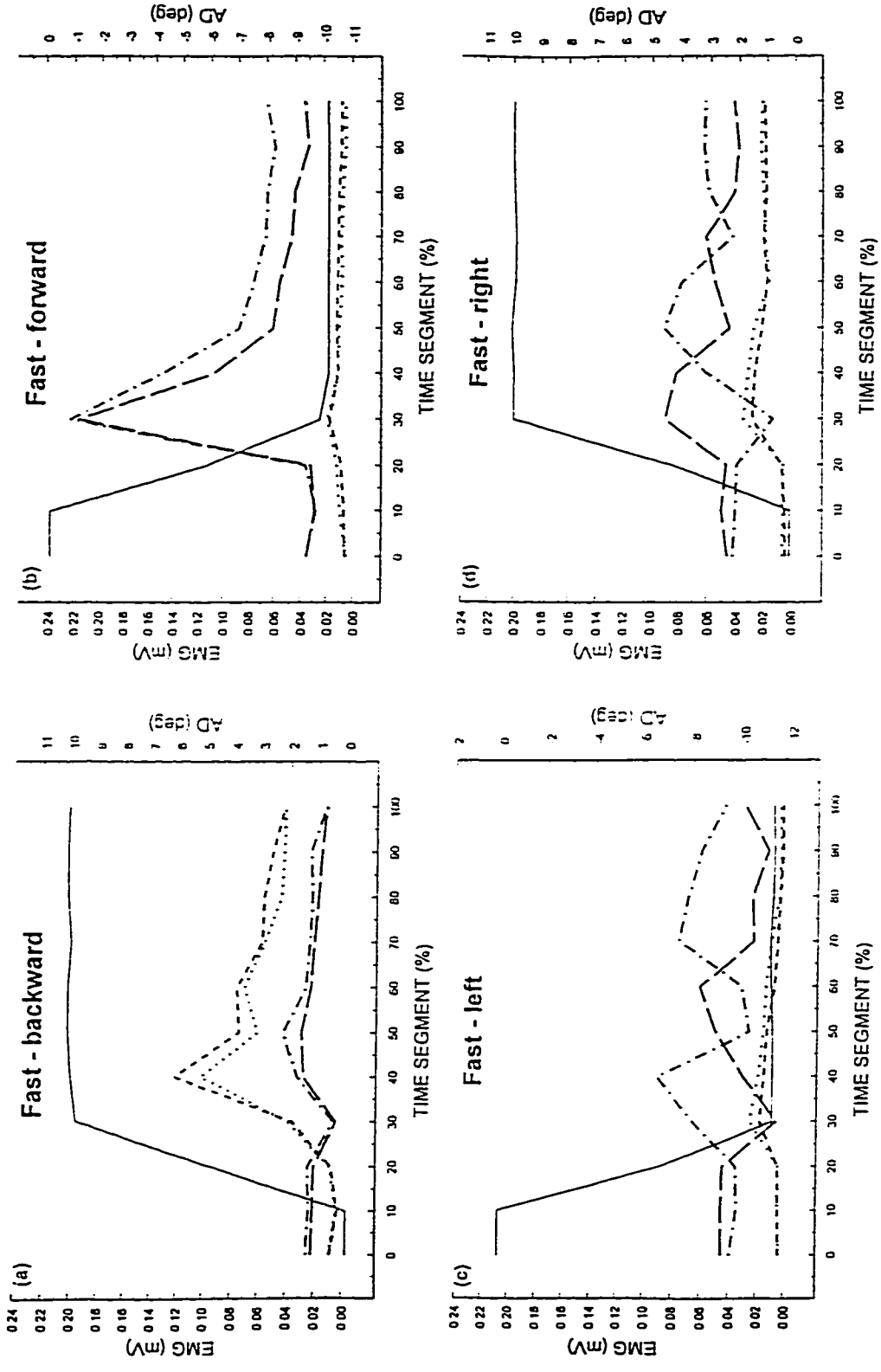

Figure 3.4. A-D. EMG magnitude during fast perturbations in four directions (mean of 5 subjects). Traces represent angular displacement of the seat (——) and the muscle quadrants as defined in Figure 3.3.: right back (— · —), left back (— —), right abdomen (· · · ·), left abdomen (- - -).

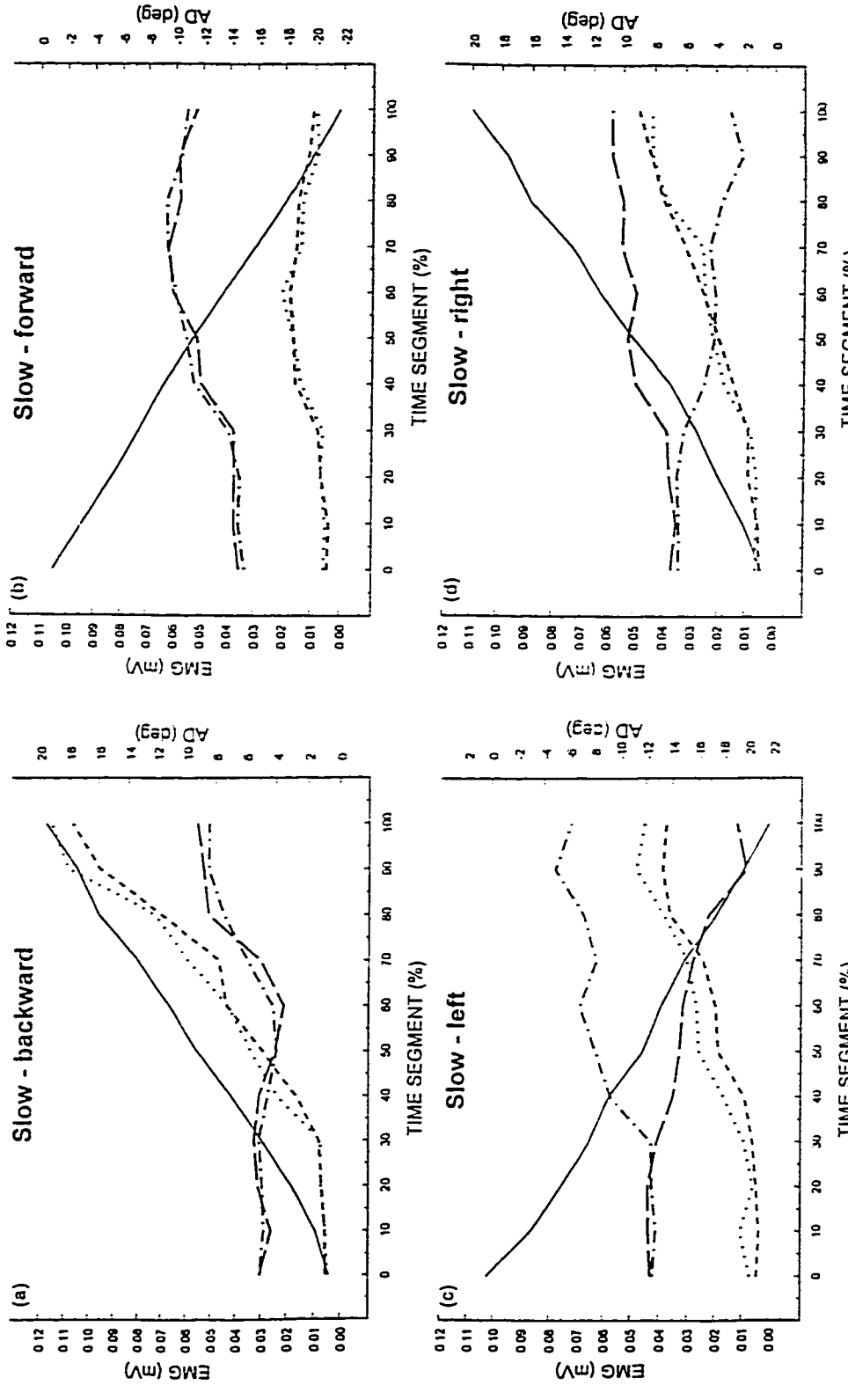

Figure 3.5. A-D. EMG magnitude during slow perturbations in four directions (mean of 5 subjects). Traces represent angular displacement of the seat (——) and the muscle quadrants as defined in Figure 3.3.: right back (— · —), left back (— —), right abdomen (· · · · ·), left abdomen (– – –).

the abdominal muscles corresponded with an observed trunk flexion, which was an appropriate behaviour in order to maintain balance. The resumed activity of the back muscles in a later phase may be considered as a postural adjustment to the new rotated position. In contrast, the forward tilt rotated the pelvis forward and moved the centre of gravity in this direction. Any stronger contraction of the abdominals would have been undesirable, as a flexion of the trunk would have brought the torso further away from its vertical alignment. In this task a contraction of the back muscles was used to minimise the excursion of the centre of gravity through the activation of the trunk extensors. The sharp initial burst of back muscle activity during the fast forward tilt helped decelerate the trunk. The increase in the activity of the abdominal muscles during the fast forward rotations was negligible. In fact, in one subject a bilateral decrease in the abdominal muscles EMG from its resting level was observed. During slow tilts the EMG gradually increased in both dorsal and abdominal muscles not opposing any sudden acceleration of the torso. The asymmetrical contractions of the right and left back muscle groups during frontal tilts were an important discovery. When the seat and the pelvis were displaced to one side the contraction of the contralateral back muscles reduced the displacement of the upper torso. In fast perturbations the right and left back muscles responded phasically, such that an increased activity on one side corresponded with a decrease on the other. It has been shown that during rapid limb movements initiated by the subject, the activity of both agonist and antagonist muscles display a phasic pattern (Angel, 1977; Jacobs, Andrews, Iannone & Greninger, 1980; Waters & Strick, 1981). Explosive force generated by sudden contraction of only the agonist would compromise the precision of the movement and could even result in tissue damage. On the other hand, muscle co-contraction of antagonists would compromise the speed of movement. The phasic employment of the antagonistic muscle groups seems to be the most efficient control of ballistic movements. In the present experiment the torso was accelerated passively through the displacement of the seat. In order to keep the torso upright in the fast perturbations, the subject had to respond with a rapid contraction similar to initiation of a ballistic movement. It has been reported before that a reciprocal activation of antagonists for postural correction allows effective, directionally specific movement of the centre of gravity back toward equilibrium (Dunbar, Horak, Macpherson & Rushmer, 1986). The

reciprocal antagonist activity may be seen as an attempt to compensate for initial errors in scaling response magnitude.

In contrast to the back, the response in the abdominal muscles had always a similar trend on both sides. Relaxation of one side and contraction on the other was never observed. This finding fits well with the hypothesis that co-contraction of the abdominal muscles is used for regulation of the intra-abdominal pressure which helps stiffen the torso (Grillner, Nilson & Thorstensson, 1978). The intra-abdominal pressure was not measured in this study.

Horak and colleagues demonstrated in a study of standing subjects balancing on a hydraulic platform that expectation of postural stimulus amplitude or velocity affected the EMG magnitude in leg muscles (Horak, Diener & Nashner, 1989). The present study did not show a significant role of expectation, perhaps because expectation was defined differently. Horak and colleagues defined an "unexpected" perturbation as the one that differed in character (amplitude, velocity) from a row of invariable prior perturbations. After adaptation of the neuromuscular system to the row of predictable trials a novel task resulted in significantly different responses from the leg muscles. In contrast, in the present study the conditions were randomized and any perturbation was considered as "unexpected" unless the information about its direction and velocity was given. This paradigm did not allow sufficient adaptation to expected trials. Therefore, the subjects could not "rely" on their previous experience and responded to expected and unexpected perturbation in the same way. Verbal information about the character of the upcoming trial was obviously not sufficient to change the subject's strategy.

Visual feedback tested in slow perturbations was not found significant for the EMG magnitude. The presence of intact proprioceptive and vestibular apparatus probably provided compensation for the blindfold.

3.5 Conclusions

Several observations about the trunk muscles during balance perturbations during sitting were made. The pattern of the EMG magnitude depended on the direction of the

perturbation and was characteristic for each condition. A marked symmetry between the right and left sides was found for the rotations in the sagittal plane. The frontal tilts showed asymmetry mainly in the back muscles. The back muscles, but not the abdominal muscles, exhibited an alternating phasic activation. Expectation or vision played no role in the magnitude of the EMG signal.

The EMG patterns found in healthy subjects showed regularities which might not be present in the population with changed trunk motility. For example, impaired equilibrium reactions have been observed in back-pain patients (Byl & Sinnott, 1991). Therefore, further studies should include back-pain sufferers to determine changes in the recruitment pattern of their trunk muscles. The testing of the equilibrium reactions in sitting patients could help in the diagnostic process as well as in rehabilitation.

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CHAPTER 4.

Phasic activity in the human erector spinae during repetitive hand movements[1]

4.1 Introduction

Movements of individual body segments – "focal movements" - are accompanied by activity of distant musculature – "associated postural adjustments" (Belenkii, Gurfinkel & Paltsev, 1967; Lee, Buchanan & Rogers, 1987; Bouisset & Zattara, 1981; Gurfinkel, Lipshits & Lestienne, 1988). The site of coordination between the postural muscles and the muscles executing focal movements is still a matter of speculation. Studies of ballistic voluntary movements showed that postural EMG activity sometimes precedes the focal muscle EMG or occurs simultaneously with it. These observations have led to the notion of two principal modes of postural control (Massion, 1992). In the "hierarchical mode" the pathways responsible for focal movement send collaterals to the postural networks. In this mode the onset of the movement and the postural activity are closely coupled and time-locked (Gahery & Massion, 1981; Paulignan, Dufosse, Hugon & Massion, 1989). In the "parallel mode" there are separate pathways to the focal and the postural muscles and so uncoupled activation can occur (Lee et *al.*, 1987). In both modes the execution of ballistic focal movements and the accompanying postural adjustments is considered to be preprogrammed (Cordo & Nashner, 1982). It has been suggested that postural adjustments are generated in close connection with focal ballistic movements and an intact motor cortex is indispensable for their execution (Palmer, Cafarelli & Ashby, 1994; Palmer, Downes & Ashby, 1996).

[1] A version of this chapter has been published. Zedka, M. & Prochazka, A. (1997). Phasic activity in the human erector spinae during repetitive hand movements. Journal of Physilogy 504.3, 727-734.

Postural adjustments can also be evoked by unexpected perturbations of the limbs. These involuntary responses occur at relatively short latencies and it has therefore been inferred that subcortical brain areas may provide for certain aspects of postural control (Traub, Rothwell & Marsden, 1980; Cordo & Nashner, 1982; Massion, 1992). Afferent signals from the moving limb were assumed to be critical for this postural activity.

In the present paper we studied cyclical activity in a back muscle, the erector spinae (ES), during fast cyclical hand movements. To our knowledge, the activation of postural musculature during repetitive focal movements of the upper extremities has not been studied. Its mechanism may be different from discrete voluntary ballistic movements or discrete externally imposed limb perturbations. If speed is of the essence in this task and each new cycle of the hand movement can be expected to be similar to the previous cycle, higher centres could in principle delegate a substantial part of the postural activity to the spinal circuitry. Mechanical perturbation transferred to the trunk from the moving limb segment may be strong enough to evoke a local stretch reflex in the ES which would stabilize the trunk. Stretch reflexes in the lumbar ES in response to tapping have been described in the past (Dimitrijevic, Gergoric, Sherwood & Spencer, 1980). Tapping the lumbar ES in our subjects with a servo device evoked a short latency EMG response at about 15 ms and a medium latency response at 50 ms. We argue in this paper that the periodic ES EMG activity seen during repetitive hand movements is to a large extent produced by local stretch reflexes as it corresponds with lumbar movement and occurs even in the absence of afferent input from the upper limb or voluntary commands descending from the brain.

4.2 Methods

4.2.1 Subjects

A total of five healthy subjects (4 males, 1 female) aged between 25 and 50 years participated in the study with their informed consent. The study was performed

according to the Declaration of Helsinki. Ethical approval for these experiments was granted by the University of Alberta Hospital Research Ethics Board.

4.2.2 Experimental paradigm

After appropriate skin preparation a pair of adhesive surface EMG electrodes was placed over the left ES 2 cm lateral to the L3 spinous process, 4 cm apart, parallel to the muscle fibres. An accelerometer was taped to the skin on the back between the EMG electrodes. Its axis was parallel to that of the vertebral column (i.e. it responded to rostro-caudal accelerations). In some experiments a second accelerometer, sensitive to movement in the antero-posterior direction, was stuck to the subject's forehead. The wrist angle was measured by a purpose-made length gauge (linear variable displacement transducer) attached to the back of the right hand and the right forearm.

To test the hypotheses that the rhythmical activity in ES associated with rhythmical hand movements is not generated in the brain and that afferent input from the hand-moving muscles is not critical, several experimental conditions were designed. In each case the same background ES EMG level was maintained (10% MVC). This was achieved by showing to the subject a low-pass-filtered (10 Hz), full-wave-rectified version of the ES EMG signal on an oscilloscope.

Experiment A:

Subjects standing with their feet 20 cm apart and their pronated right arm held forward and horizontal, repetitively extended and flexed the wrist as fast as possible in the vertical plane. This task was also performed while the subjects were sitting upright in a chair with their knees flexed at a right angle, their feet supported on the ground. Finally, the repetitive voluntary hand movements were performed while lying prone on a table with the arm hanging down at a right angle to the trunk.

Experiment B:

The experimenter produced hand excursions by passively flexing and extending the standing subject's wrist with the forearm bound to a horizontal support. Additionally, passive wrist movements were produced by an electromagnetic length servo through a handle which the subject was holding. The handle moved cyclically at 8 Hz, 12 Hz and 20 Hz. The subject was sitting during this task.

Experiment C:

The wrist flexor and extensor muscles were electrically stimulated (trains of 25 Hz stimuli amplitude-modulated at 8 Hz) in a reciprocating manner to evoke alternating contractions which produced hand movements similar to the cyclical voluntary movements. Also, in order to assess a possible direct effect of afferent excitation by the electrical stimulation, control trials were performed in which the flexor and extensor groups were stimulated simultaneously (also at 8 Hz) to produce as little movement as possible, while still activating the muscle nerves.

Experiment D:

To further investigate the role of the limb afferents, the wrist extensor muscles were mechanically vibrated. Parameters (100 Hz, amplitude 1mm) were chosen to stimulate muscle spindle primary endings, on average once per cycle (Prochazka & Trend, 1988). The vibrator was turned on and off at the frequency of the individual's voluntary wrist movements (8 Hz) to reproduce approximately the pattern of activation of the muscle spindle afferents during the hand movements.

Experiment E:

The subjects were asked to focus their attention on their back muscles and repeatedly contract and relax them as fast as possible while standing, sitting or lying prone.

4.2.3 Recording and analysis

The EMG signals from the left ES and the right wrist extensors were high-pass filtered at 300 Hz, full-wave rectified, low-pass filtered at 30 Hz and sampled at 500/s by a CED 1401 laboratory interface and a personal computer. Signals from the wrist angle sensor and the back and head accelerometers were also sampled at 500/s. SIGAVG 5.41 software (Cambridge Electronic Design Ltd.) was used for data collection. Five 0.5 s windows from the total of 5 s data were averaged for each trial using custom software (DASA21). To visualize the frequency content of each channel, fast Fourier transforms were performed using MATLAB 4.2c.1 software (The Math Works, Inc.). Microsoft Excel 5.0a (Microsoft Corporation) was used to generate the figures and also for statistical analysis. The mean and standard deviation of the frequency at which the peaks in the frequency spectra occurred was calculated for five subjects. Student's t-test was performed to determine whether the ES activation frequency was significantly different during maximum speed cyclical voluntary hand movements from the frequency during maximum speed voluntary ES contraction (significance of $p = 0.01$).

4.3 Results

Experiment A:

Repetitive voluntary hand movements at a given frequency were accompanied by regular ES EMG bursting at the same frequency while standing, sitting or lying prone.

Some subjects were able to move their hands cyclically at frequencies as high as 8.6 Hz (7.8 Hz mean ± 0.4 Hz standard deviation, n = 5). Data from one subject are presented in Figure 4.1. Both from the time plots in the left part of the figure and the frequency plots in the right part it is obvious that the frequency of changes in wrist angle, wrist extensor EMG and head and back acceleration was identical. There was no statistical difference in the frequency of hand movement and associated ES activation between standing, sitting and lying.

The wrist extensor EMG frequency spectra had a prominent second component at twice the fundamental frequency. The peak is partly attributable to small EMG bursts occurring between the main bursts (see time plot Figure 4.1.) and partly to the non-sinusoidal nature of the signal as a whole. A similar peak at the first harmonic is also visible in the wrist angle spectrum (relatively small compared to the peak at the fundamental frequency) and this is also attributable to the non-sinusoidal nature of the wrist angle signal.

Experiment B:

When the forearm was constrained by tying it to a horizontal support, and the hand was moved passively by the experimenter, a different picture was seen. Figure 4.2. presents data from the same subject as in Figure 4.1. A small peak in the ES EMG frequency spectrum occurred at 11.2 Hz which corresponded to the dominant component in the back accelerometer frequency spectrum. However, this frequency was double that of the dominant components of hand and head motion (5.6 Hz). This finding was consistent in all subjects and trials. Across subjects the mean frequency of the hand and head movement was 4.9 ± 1.0 Hz (n = 5), while the principal frequency of the lower back accelerometer and ES EMG was 9.9 ± 1.8 Hz (n = 5).

In other experiments the hand was moved passively by a length servo motor driven by a signal generator producing sine waves at three target frequencies: 8, 12 and 20 Hz. The extended forearm was unconstrained. Figure 4.3.A. shows spectra from one such trial where the movement frequency was 12.4 Hz. Note that the spectra of wrist angle, back acceleration and ES EMG all had peaks at the movement frequency. This was

true in all trials for all five subjects, regardless of imposed movement frequency. There were small inaccuracies in setting the target frequency of the signal generator from one set of trials to the next. The mean frequencies of the spectral components in the three sets of experiments therefore deviated slightly from the target frequencies (8.3 $\pm$ 0.1 Hz, 12.4 $\pm$ 0.1 Hz and 20.0 $\pm$ 0.1 Hz, n = 5).

Experiment C:

Figure 4.3.B. presents the data from reciprocal electrical stimulation of the wrist extensors and flexors of one subject at 8.2 Hz. Across subjects the stimulation produced hand movement at 8.2 $\pm$ 0.1 Hz (n = 5) which was also precisely matched in the frequency spectra of the back acceleration and the ES EMG. In contrast, simultaneous stimulation of the flexors and extensors at the same modulation frequency did not produce detectable hand movement and neither was any significant back acceleration or cyclical ES EMG present (figure not shown).

Experiment D:

100 Hz vibration of the forearm extensors modulated at 8.2 Hz evoked reflex EMG bursts at 8.2 $\pm$ 0.1 Hz (n = 5) in the vibrated muscle with no detectable hand movement. Neither the back accelerometer nor the ES EMG registered any rhythmical activity in the lower back (Figure 3C).

Experiment E:

When instructed to rhythmically contract the ES as fast as possible, the subjects were not able to produce ES EMG bursts at frequencies greater than 6.6 Hz (5.8 $\pm$ 0.6 Hz, n = 5). There was no significant difference between trials while standing, sitting or lying. The EMG burst frequency during voluntary ES activation was significantly lower than that during voluntary hand movements ($p < 0.01$).

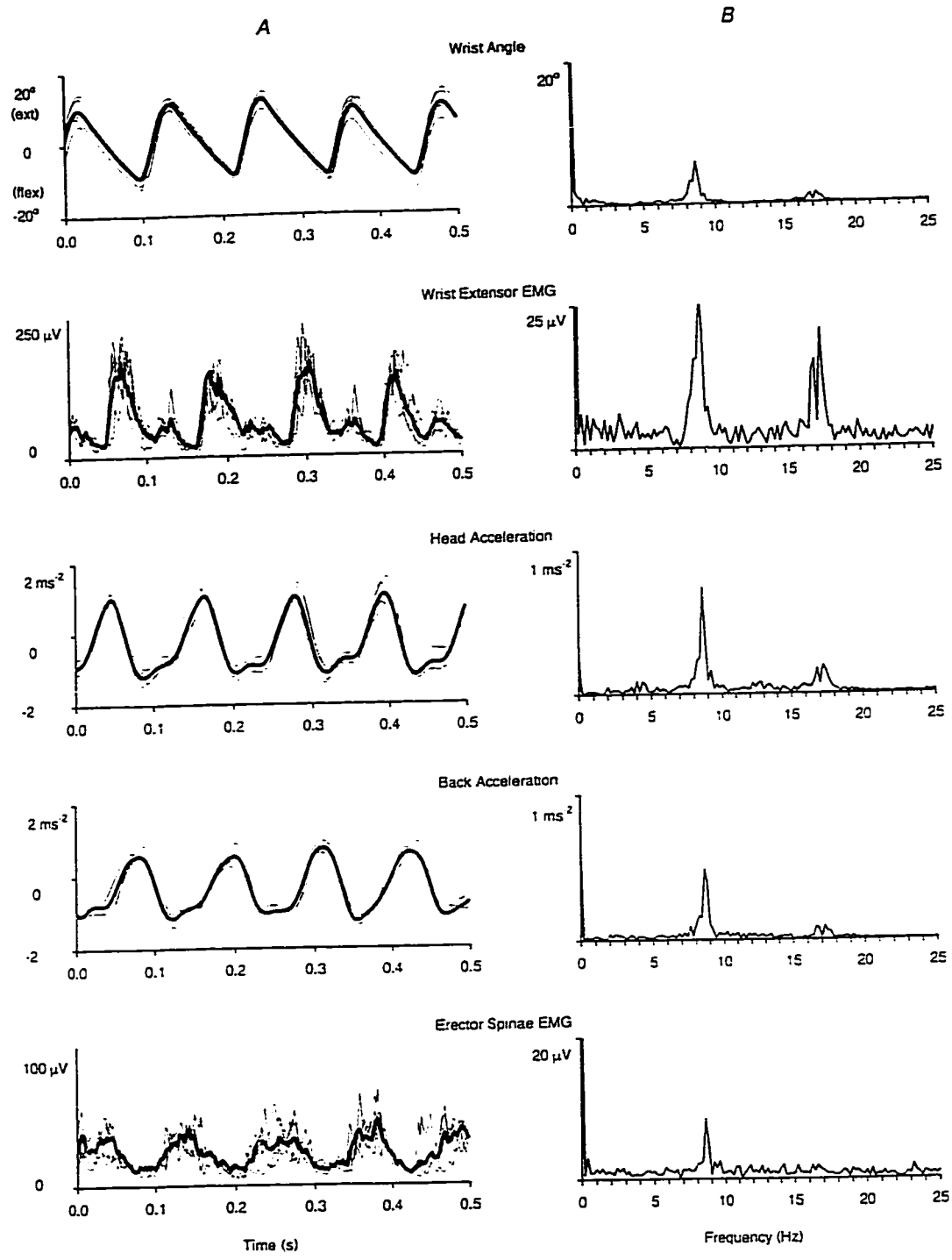

Figure 4.1. Unconstrained forearm - voluntary cyclical hand movement. A repetitive hand movement at 8.6 Hz resulted in cyclical accelerations of the head and lower back which was accompanied by cyclical erector spinae (ES) muscle activation at the same frequency. A) superimposed time plots of wrist angle, wrist extensor EMG, head and back acceleration and ES EMG respectively. Each plot shows 5 single traces (thin lines), each 0.5 seconds long, and superimposed averages (thick lines). The traces were aligned at t=2.5 s. B) frequency spectra of the data shown in (A). Each spectral plot was computed from a total of 5 seconds of recording.

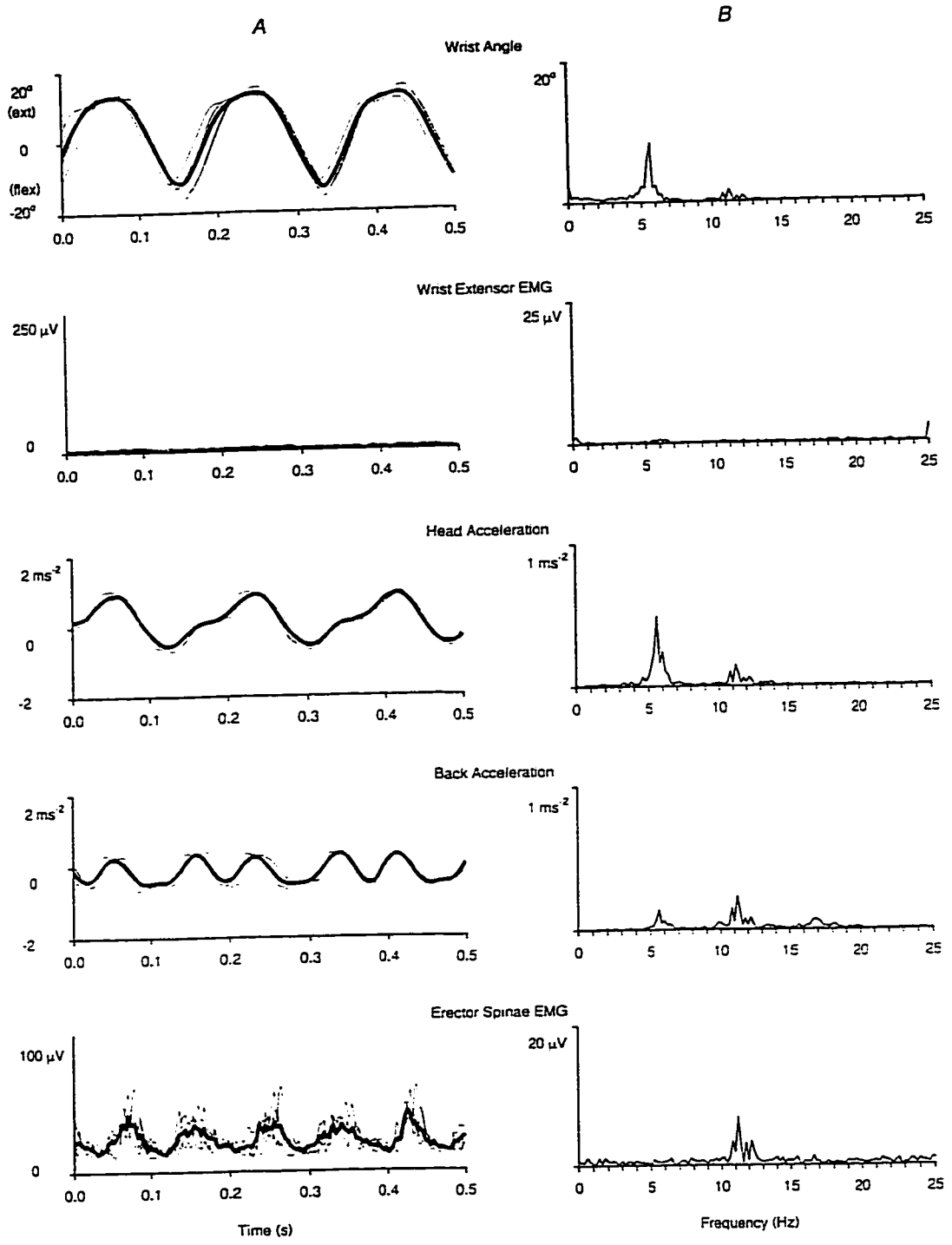

Figure 4.2. Constrained forearm - passive cyclical hand movement. Binding the subject's forearm to a horizontal constraint resulted in a different coupling of the mechanical waves to the trunk (as compared to Figure 4.1.). While movements imposed on the hand at 5.6 Hz resulted in principal spectral components of 5.6 Hz for the head, they produced prominent components at 11.2 Hz for back acceleration and ES EMG. The concurrence of the latter components suggests that ES was activated in response to the lower back movement, rather than by descending commands. Same layout of plots as in Figure 4.1.

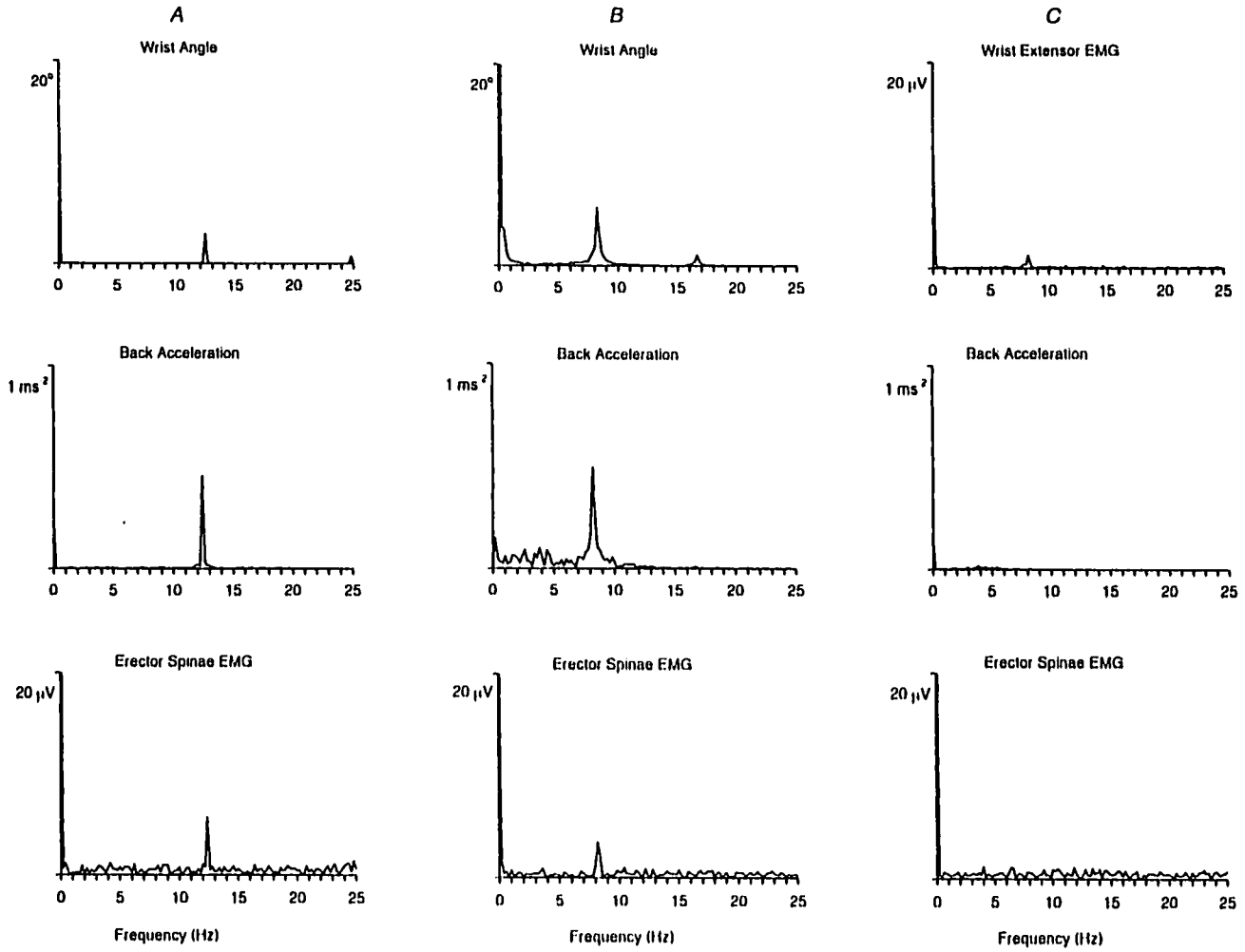

Figure 4.3. Frequency spectra of wrist angle (wrist extensor EMG in C), back acceleration and ES EMG signals obtained during A) passive hand movements produced by a servo motor; B) hand movements produced by electrical stimulation of the forearm muscles; C) amplitude-modulated vibration of the wrist extensor muscles to evoke cyclical input to the CNS from forearm afferents. A) spectra show peaks at 12.4 Hz, the frequency of the imposed movements. B) spectra show peaks at 8.2 Hz, the frequency of the modulated electrical stimulation. These results show that cyclical ES EMG is generated in response to movement of the hand in the absence of central commands. C) no detectable direct propriospinal effect of input from forearm muscle afferents on the ES.

4.4 Discussion

The function of the back muscles is to move voluntarily the trunk as well as to provide appropriate postural adjustments during movement of other body parts. Several neural pathways have been identified that connect (directly or indirectly) to motoneurons of the back muscles in animals as well as in humans (Alstermark, Lundberg, Pinter & Sasaki, 1987; Ghez, 1991; Bolton & Tracey,1992; Robbins, Schwartz-Giblin & Pfaff, 1992; Gracies, Meunier & Pierrot-Deseilligny, 1994; Mouton & Holstege, 1994; Vanderhorst & Holstege, 1995). Is this descending input critical for the cyclical activation of trunk muscles during fast cyclical hand movements? Our results lead us to conclude that it is not and that the cyclical ES EMG accompanying rhythmical hand movements we observed was due to stretch reflexes originating in the back muscles.

The ES EMG bursting frequency during rapid voluntary hand movements corresponded with the fundamental frequency of the hand movement if the forearm was not constrained (experiment A). While the cortical origin of the voluntary commands for the forearm muscles could hardly be disputed, the source of the accompanying activation of the trunk muscles was unclear. The relatively low EMG bursting frequency during selective voluntary activation of the ES (experiment E) makes it improbable that the same descending pathway could be responsible for ES activation during repetitive hand movements at higher frequencies. Moreover, the results of trials where the hand movements were non-voluntary, i.e. produced by the experimenter, by the servo motor or through electrical stimulation (experiments B and C), indicate that cyclical ES activity could be generated even in the absence of voluntary commands for cyclical hand movements. Also the fact that ES EMG burst frequencies in these passive cases (up to 20 Hz) could by far exceed the rate of activation seen during any cyclical voluntary movement (hand or back) supports the notion that the rhythm was generated in the periphery rather than in the cortex.

In conditions where the forearm was unconstrained, the ES EMG burst frequency corresponded with the oscillation frequency of the moving hand. Therefore, proprioceptive neuronal pathways from the forearm muscles had to be considered as a

source of input to the ES α-motoneurons. We tried to evoke ES EMG bursting by stimulating the forearm extensor afferents both electrically and through vibration. Reciprocal electrical stimulation of the forearm muscles at 8 Hz evoked wrist movements and ES EMG bursting of the same frequency. Cyclical stimulation of the same intensity but administered in phase to the wrist flexors and extensors elicited what we assume was comparable cyclical afferent input without significant hand movement. No ES EMG bursting was present in this case. Admittedly the afferent activity evoked in flexors and extensors would have been in phase in this situation, though probably not of identical levels in the two muscle groups. Modulated vibration of the wrist extensors evoked regular EMG bursting in the vibrated muscle but not in ES. These findings all indicate that afferent input from the forearm muscles was not responsible for the generation of the rhythmical ES activity.

From our result we would argue that movement of the hand was necessary to cause the cyclical ES activity and that this movement caused a referred mechanical perturbation of the trunk which in turn elicited local reflex responses in the back muscles. This argument is strongly supported by the trials where the subject's hand was moved passively by the experimenter while the forearm was tied to a horizontal support. This created a new coupling of the mechanical waves to the trunk. The trunk now oscillated at twice the frequency of the hand as registered by the accelerometer mounted on the lower back. The ES EMG burst frequency corresponded with the back accelerometer readings and not with the hand or head movements.

Is the organization of postural adjustments during repetitive voluntary hand movements different from that during single voluntary ballistic movements? In his review article Massion (1992) concluded that studies of posture and equilibrium led to two models of organization of focal movements and their associated postural adjustments. The "hierarchical" model assumes one common command descending to both focal and postural muscles and proposes a tight linkage between the focal movement and postural adjustments. The "parallel" model favors a looser connection between the circuits for the focal and the associated movements. Since the focal movements are voluntary, both models emphasize a central origin of the input commands to the postural network. In contrast, we demonstrated in this paper that postural activity in ES was

present during rhythmical hand movements even if the central command was missing (passive hand movements). The apparent dependence of ES activation on mechanical trunk perturbations is reminiscent of the automatic postural reactions to external perturbations described by Marsden *et al.* (1977) and Traub *et al.* (1980). A model reconciling the organization of anticipatory postural adjustments during focal movements with automatic postural reactions has been presented by Cordo & Nashner (1982). The idea was that the anticipatory and reflexive adjustments had similar organizational properties and were therefore routed through common neural pathways. In this light it is conceivable that postural adjustments during repetitive voluntary focal movements could use the same neural circuitry as postural adjustments during single ballistic voluntary focal movements. The former would use mainly peripheral, the latter mainly central input. It is tempting to speculate that the postural system switches between peripheral and central inputs depending on the predictability of the destabilizing effect of the focal movement. "Postural set" could play a role in the selection between the central and peripheral modes of control (Nashner 1976; Cordo & Nashner 1982; Horak, Diener & Nashner 1989). On this view, in single ballistic limb movements, which do not cause regular body oscillations, postural adjustments are carried out in a feed-forward mode in close relation to the focal command (Belenkii *et al.*, 1967; Lee *et al.*, 1987; Bouisset & Zattara, 1981; Gurfinkel *et al.*, 1988). On the other hand, in repetitive movements of the upper extremity, the higher nervous centres may decouple the generation of postural activity from central focal commands and use peripheral feedback to take advantage of mechanical events in the trunk rather than to repetitively generate descending commands for each cycle. We showed that the trunk always oscillated during repetitive hand movements whether these were voluntary or not. Since the degree of trunk perturbation during each new cycle is easily predictable, it may be sufficient for the brain to provide tonic input to ES α-motoneurons allowing afferent input to evoke rapid compensatory contractions of ES. It is likely that free hand movements have only negligible impact on the net displacement of the body centre of gravity, and therefore the rhythmical ES activity probably serves to stabilize trunk segments rather than to maintain whole-body equilibrium. This notion is supported by the fact that rhythmical ES EMG bursting during hand movements was present in standing as well as in sitting and lying.

Delegating corrective responses to spinal levels would make motor control of the trunk segments easier, especially in cases where the rhythm of the trunk does not correspond to the rhythm of the moving hand.

4.5 Conclusion

We conclude that cyclical ES activation seen during repetitive hand movements is to a large extent due to mechanical events transmitted from the arm to the trunk. Oscillations of body segments elicit proprioceptive reflexes in the lower back muscles which contribute to trunk stabilization.

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CHAPTER 5.

Different effects of cutaneous and muscle pain on the human erector spinae stretch reflex[1]

5.1 Introduction

Compared to limb muscles, the reflex activity1 of human back muscles has received very little attention in the past. Considering the frequency of musculoskeletal disorders in the lower back region, this fact is quite surprising. The question of how pain influences the function of the spinal circuitry responsible for back muscle activity is quite relevant since back pain is accompanied by changes in motor patterns (Cassisi, Robinson, O'Conner & McMillan, 1993; Grabiner, Koh & El Ghazawi, 1992; Price, Clare & Ewerhardt, 1948; Paquet, Malouin & Richards, 1994). As in limb muscles, the interaction between sensory input and motor output is likely to take place at many levels of the neuraxis. Before we can understand the impact of pain on more complex motor activities and behaviour controlled by the higher centres, it is important to elucidate the interaction of pain and basic spinal reflexes.

To our knowledge, only one study concerned with human back muscle reflexes and pain has been published (Kugelberg & Hagbarth, 1958). It described trunk muscle responses to cutaneous stimuli from the trunk region. However, the back pain syndome is usually characterized by deep ligamentous, articular or muscular pain. Cutaneous

[1] A version of this chapter has been submitted for publication in the Journal of Physiology. Zedka, M., Prochazka, A., Knight, B., Gillard, D & Gauthier, M. Different effects of cutaneous and muscle pain on the human erector spinae stretch reflex.

signs, if present, occur secondarily (Kraft, Johnson & Leban, 1968; Travell & Simons, 1983).

It has been noticed that deep somatic or visceral pain leads to local increases in muscle tone (Travell & Simons, 1983; Maigne, 1996; Vecchiet, Giamberardino & Saggini, 1991). Although never confirmed experimentally, an opinion that muscular hypertonus is a result of increased muscle stretch reflexes is encountered in the literature (Berberich, Hoheisel, Mense & Skeppar, 1987; Johansson & Sojka, 1991). In scoliosis it has been suggested that unilaterally increased stretch reflexes may underlie back muscle hypertonus (Trontelj, Pecak & Dimitrijevic, 1979). Note that the mechanisms of hypertonus in scoliosis and chronic back pain are not necessarily the same.

The amplitude of the stretch reflex is determined by the excitability of the skeletomotor neurones, fusimotor neurones and/or muscle spindles. A classical manoeuvre to test the stretch reflex gain is the muscle tendon jerk. Since the 19th century it has been widely used in testing limb reflexes and, in isolated cases, it has also been described for the back muscles (Dimitrijevic, Gregoric, Sherwood. & Spencer, 1980; Tani, Yamamoto, Ichimiya & Kimura, 1997). If the activity of α-motoneurones is held constant by maintaining a constant level of voluntary electromyogram (EMG) during muscle taps, it is possible to test the hypothesis that painful input to the central nervous system leads to muscular hypertonus through increased spindle sensitivity to stretch or increased gain of central transmission.

Since both deep and superficial pain could affect muscle tone, in this study we investigated whether a stretch reflex evoked by tapping the lumbar portion of the erector spinae (ES) muscle is changed by superficial (cutaneous) or deep (muscle) pain. Superficial pain was induced by mechanical pricking or electrical stimulation of the lumbar skin while deep pain was induced by infusing hypertonic saline into the right ES muscle.

5.2 Methods

5.2.1 Subjects

Five healthy volunteers (1 female and 4 males, age 20 – 40 years) participated in the study. They were familiarized with the protocol and signed a consent form in accordance with the requirements of the University of Alberta, Faculty of Medicine Research Ethics Board. The study was performed according to the Declaration of Helsinki.

5.2.2 EMG

After preparation of the skin with ethyl alcohol, bipolar surface EMG from the right and left ES at the level of the 3rd lumbar vertebra (L3) was monitored with self-adhesive Ag/AgCl electrodes (Electrotrace ET 301, Huntington Beach, California, USA) placed in the cranio-caudal direction, 2 cm from the midline, 4 cm apart (Figure 5.1.). The EMG signal was amplified (gain 1000), band-pass filtered (30Hz – 50 kHz), full-wave rectified, low-pass filtered at 300 Hz, and sampled at 1000 s^{-1} by a Cambridge Electronic Design 1401 laboratory interface (CED, England) and personal computer. CED SIGAVG 6.30 software was used for data collection.

5.2.3 Stretch reflex

Stretch reflexes of the right lumbar ES were elicited by sudden rapid indentations delivered by a prodder fixed to an electromagnetic length servo, which was driven by rectangular waveforms. The end of the metal prodder was provided with a hard rubber cap. The cap was in permanent contact with the skin and the mechanical perturbation of the muscle was produced by indentations of a constant amplitude and duration (5 mm, 1 s). The displacement and the impact force of the prodder were recorded using the SIGAVG 6.30 software.

With the EMG electrodes attached, the subjects were seated on a stool with their legs on the ground and positioned so that the prodder was touching the skin over the right

ES between the two EMG electrodes, i.e. adjacent to L3 spinous process, 2 cm from the midline. To ensure that during the whole experiment the taps would be delivered to the same site, adjustable upholstered pads positioned against each side of the body were used to restrict lateral movements of the trunk. The sitting subjects produced constant background ES EMG activity by stretching both arms forward while watching a low-pass filtered (10 Hz) version of the rectified EMG signal on an oscilloscope in front of them. The position of the lumbar spine in the antero-posterior direction was maintained constant by monitoring the prodder force trace on the oscilloscope. Under these conditions 20 control taps were delivered.

5.2.4 Stimulation

Mechanical Cutaneous Stimulation

Superficial pain was first elicited by pressing the tip of a metal woodscrew into the lumbar skin 1 cm lateral to the tapped site. The screw was connected to a shaft in series with a spring (compliance 2.4 $mm.N^{-1}$). Application of constant pressure during a series of 20 taps was ensured by pressing on the shaft so that the spring was compressed by a constant amount. The radius of curvature of the screwtip was about 0.5 mm, and the skin was typically indented by about 5 mm during the procedure. After collection of 20 sweeps the pin was removed.

Electrical Cutaneous Stimulation

For superficial pain elicited by electrical stimulation of the skin, two surface self-adhesive gel electrodes (Chattanooga Corp., diameter 1 cm) were placed over the right ES. The cathode was centered 1.5 cm lateral to the tapped site and the anode was centered 2.7 cm lateral to the tapped site. Each tap was preceded by a 500 ms 25 s^{-1} train of 0.1 ms constant current pulses. The tap was delivered 50 ms after the end of the train of stimuli. The delay was chosen to avoid interference of the electrical stimulation artifact with the EMG response to the tap. The threshold of perception of the pulses was determined for each subject in terms of pulse amplitude (mA). Responses to 20 taps were

collected after trains of stimuli at a stimulation intensity equal to double the perceptual threshold (2xT). The subjects never reported this stimulation as painful. After 5 minutes of rest the same procedure was repeated using intensities 4xT, which were painful. After each tap the subjects verbally rated the pain on a 0 - 10 scale, where 0 represented "no pain" and 10 was "unbearable pain".

Deep (muscle) pain

Muscle pain was induced by an infusion of hypertonic saline into the right ES. A small area of skin over the muscle 3 cm lateral to the L3 spinous process was cleaned with alcohol and an intravenous catheter/needle (1.1 mm diameter, 5.1 cm long) was inserted perpendicularly to the surface of the back, 4 cm into the muscle. An intravenous infusion set attached to a 10 ml disposable syringe was filled with 5% aqueous solution of NaCl . The needle was pulled out and the infusion set was connected to the flexible intravenous catheter, which remained in the muscle, secured to the skin by tape. The syringe with saline was placed in an infusion pump (Harvard Apparatus, model 22). The pump was controlled digitally by a computer, which regulated the infusion rate according to a pre-determined time course so that the pain was kept approximately constant (see Appendix). Verbalized pain ratings after each muscle tap revealed that the feed-forward compensator mode of control of the infusion rate kept perceived pain to within 1 or 2 rating points throughout the trial (Figure 5.2.). The perceived intensities of all three forms of elicited pain were reasonably constant over the test period. Both mechanical and electrical stimulation of the skin (4xT) were perceived as pricking, burning pain confined to a small area around the site of stimulation, without spread. In contrast, saline infusion into the right ES muscle resulted in deep, dull pain spreading from the site of injection downward to the ipsilateral superior gluteal region (Figure 5.3.).

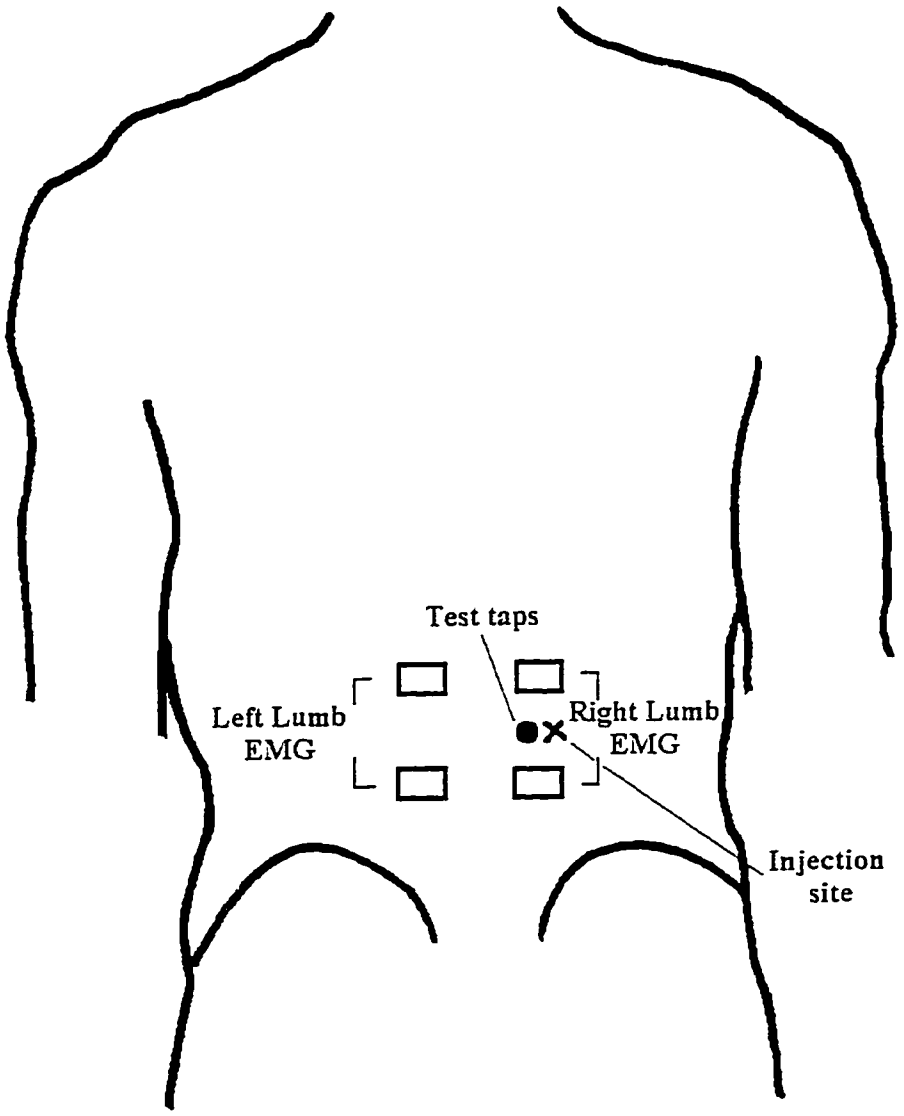

Figure 5.1. Placement of EMG electrodes over the right and left lumbar erectores spinae with tap site (●) and painful stimulation site (X).

5.2.5 Experimental paradigm

20 control taps were delivered before any stimulation. After 5 minutes of rest 20 taps were delivered during painful mechanical skin stimulation. When the pain completely subsided, the responses to 20 taps were recorded during non-painful electrical stimulation (2xT). Data for 20 taps were then collected for painful (4xT) electrical stimulation of the skin. After pain subsided, the infusion catheter was introduced into ES. The initial insertion pain usually subsided to a zero rating 10 – 20 minutes after the catheter was in place and a second set of control taps was delivered. Only if the stretch reflex monitored on a computer screen at this point was not different from the pre-insertion control (later confirmed by off-line analysis), the effect of muscle pain was tested.

Student's t-test (paired two-sample test for differences of means) was used to compare the amplitudes of the stretch reflex components between conditions with stimulation and control conditions without stimulation. The significance level was set at $\alpha = 0.05$.

5.3 Results

Control indentations of the right lumbar ES elicited an EMG response consisting of two peaks followed by a trough (Figure 5.4.). This pattern is similar to that reported by Dimitrijevic *et al.* (1980) and Tani *et al.* (1997), using pulsatile tap stimuli. These authors ascribed the terms R1 and R2 to the first and second peaks, and though the latencies we observed were somewhat longer, conceivably because of a slightly longer rise time of our step indentations (12.5 ms, 0 to 63.2% of maximum, N.B. rise times were not stated in references 13 and 14), we will adhere to the R1 and R2 terminology. The mean latency of R1 was 19.3 ± standard deviation 2.1 ms and that of R2 was 44.6 ± 2.5 ms. In the left ES the taps to the right ES elicited a different response. Instead of the R1 peak there was a trough with onset latency 26.2 ± 3.2 ms. This trough was followed by a peak (46.4 ± 4.3 ms) which corresponded to R2 in the right ES.

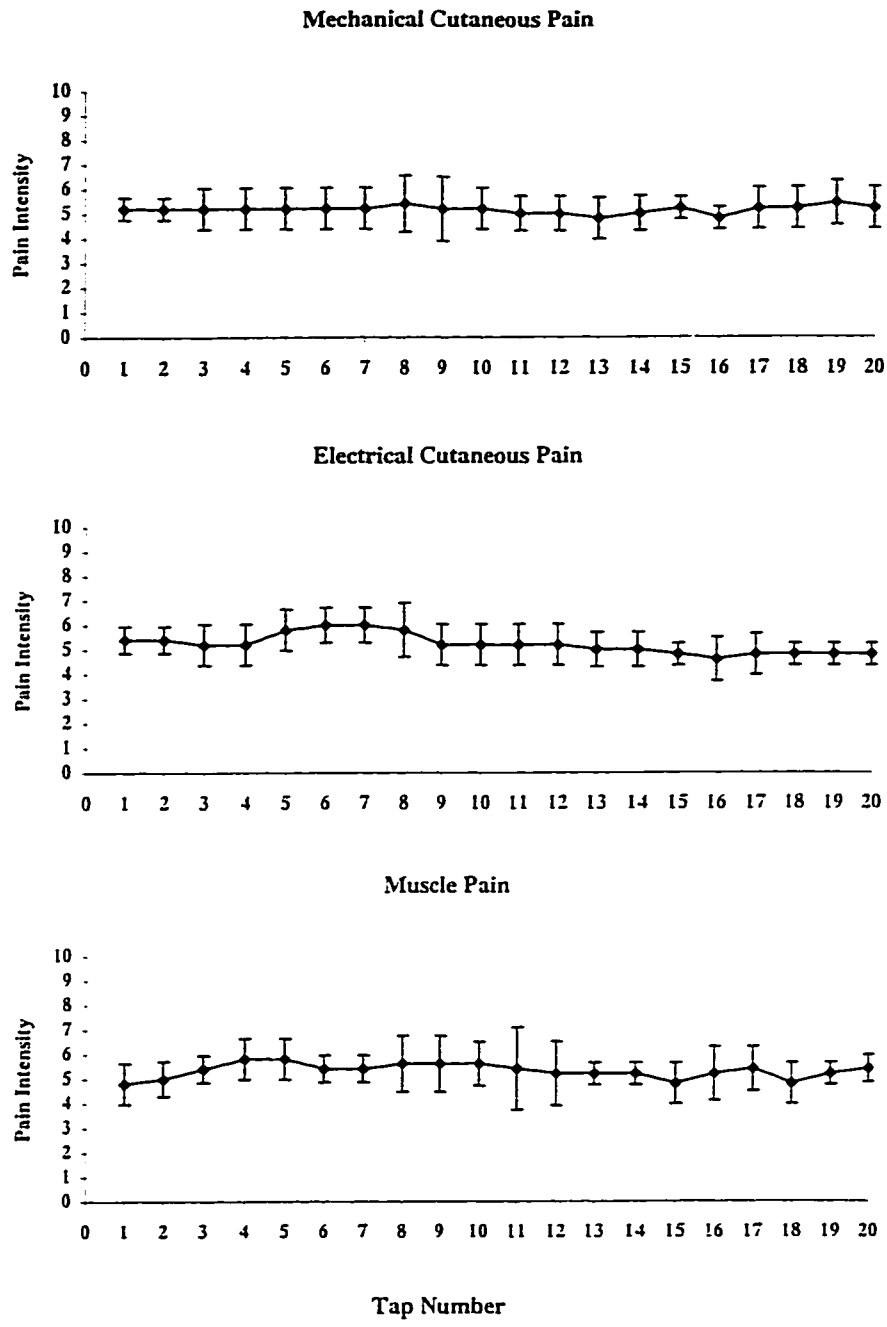

Figure 5.2. Pain intensity rating (mean ± standard deviation) of 5 subjects during 20 taps under painful stimulation. 0 - no pain, 10 – unbearable pain.

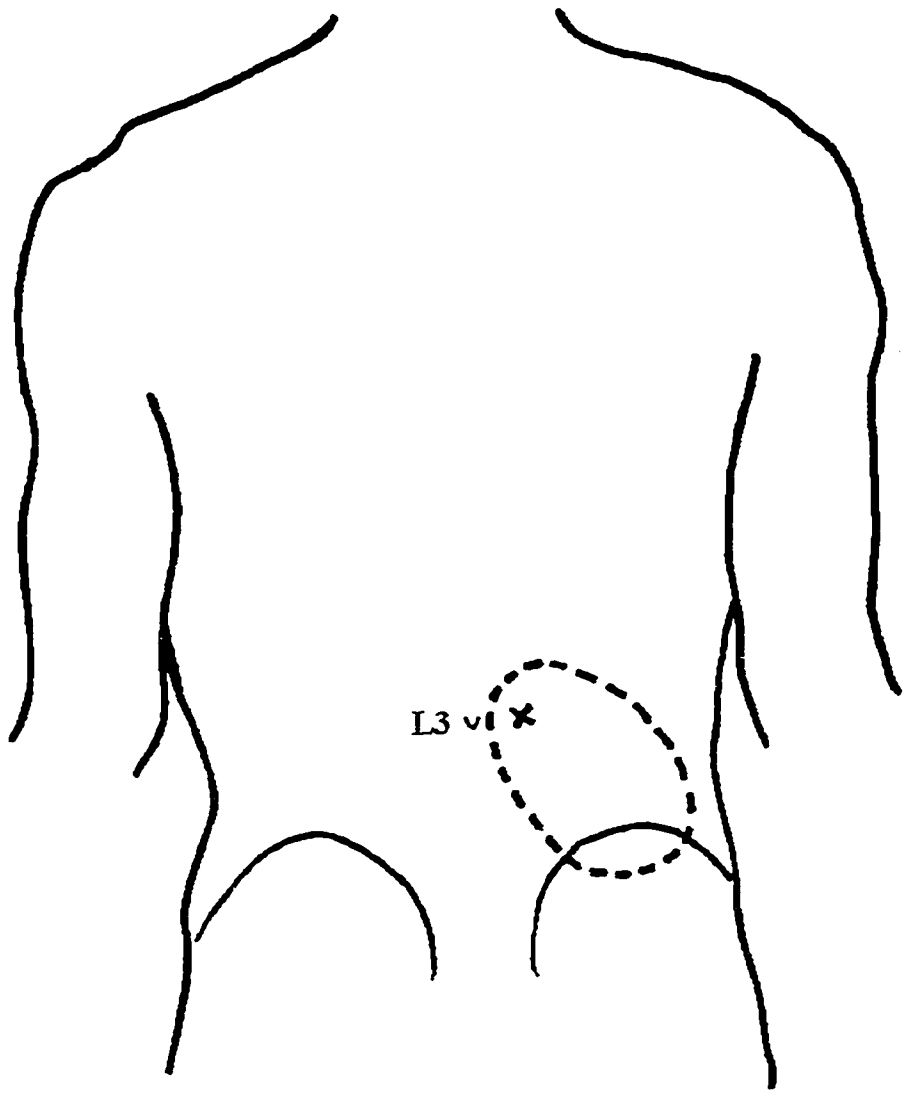

Figure 5.3. Distribution of muscle pain during infusion of 5% NaCl into the right ES at the 3rd lumbar vertebral level (X).

EMG responses to ES muscle indentations under different non-painful and painful conditions are summarized in Figures 5.5. and 5.6. For control trials the mean amplitude and standard deviation of R1 in the right ES computed over the post-stimulus interval 15 ms to 35 ms across all subjects was 34 ± 19 μV (after subtraction of the mean pre-stimulus amplitude calculated between –25 and 0 ms), while the mean amplitude of R2 computed in a similar way over the interval 45 to 70 ms was 22 ± 9 μV (Figure 5.6.). In the left ES in control trials the mean amplitude and standard deviation of the R1 trough in the interval between 25 and 45 ms (after subtraction of the mean pre-stimulus amplitude) was –12 ± 5 μV and the amplitude of R2 between 45 and 70 ms was 31 ± 18 μV.

During mechanical cutaneous pain the amplitudes of R1 and R2 in the right ES were 27 ± 22 μV and 68 ± 25 μV, respectively. Pain had a statistically significant effect only on the R2 amplitude, which was tripled compared to the controls ($p < 0.05$). No significant difference in EMG amplitude was seen in the left ES.

A similar effect was found with painful (4xT) electrical stimulation. The mean EMG values were 29 ± 21 μV and 68 ± 29 μV for R1 and R2, respectively. Again R2 tripled, which was a significant increase ($p < 0.05$) while R1 did not change significantly. In contrast to the painful stimulation, a non-painful electrical stimulation at 2xT did not produce any changes to the EMG response compared to controls. The respective values of R1 and R2 were 22 ± 14 μV and 18 ± 10 μV. No significant effect of electrical stimulation at either 2xT or 4xT was seen in the left ES.

Contrary to expectation, the stretch reflex during deep muscle pain elicited by NaCl infusion was not significantly different from the stretch reflex in control subjects. The mean rating across subjects during infusion was 5.3 ± 0.8, which was similar to that during the mechanical (5.2 ± 0.7) and electrical (5.2 ± 0.8) cutaneous pain trials. The EMG amplitude of R1 was 20 ± 17 μV and the amplitude of R2 was 23 ± 15 μV. Again, the deep pain did not influence the response in the left ES.

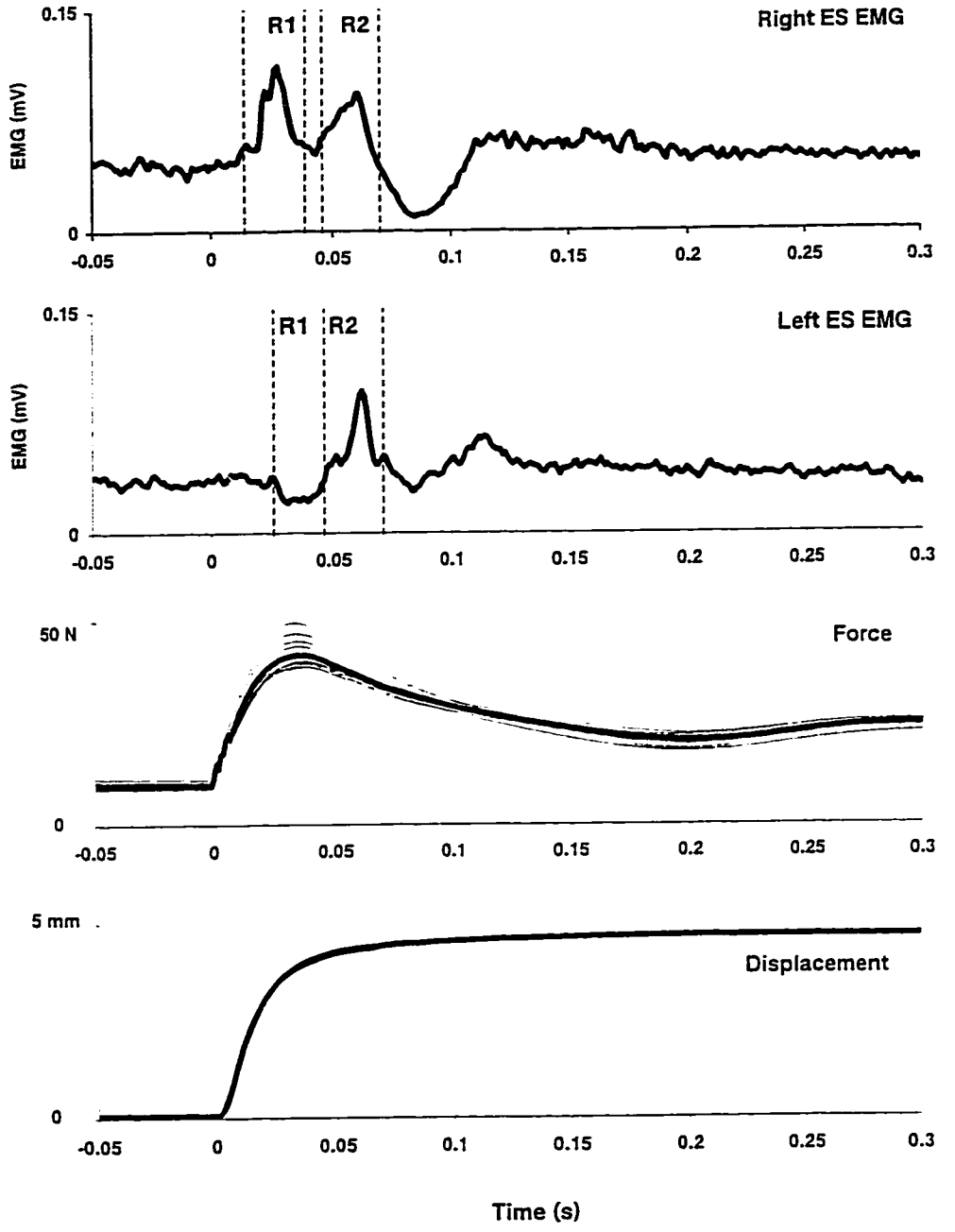

Figure 5.4. Mean EMG responses of the right and left lumbar erectores spinae (ES) to taps delivered to the right ES (average of 20 trials in one subject). In the right ES the response consists of two peaks (R1, R2). In the left ES a short-latency trough is followed by a peak. The vertical dashed lines indicate the intervals used for computation of mean amplitudes of the R1 and R2 responses (shown in Figure 5.6. after subtraction of pre-stimulus background EMG). Impact force and displacement of the prodder are shown in the lower two traces (thin lines show individual trials, thick lines show means). Variability in displacement profiles was very small, so thin and thick lines merged in this case.

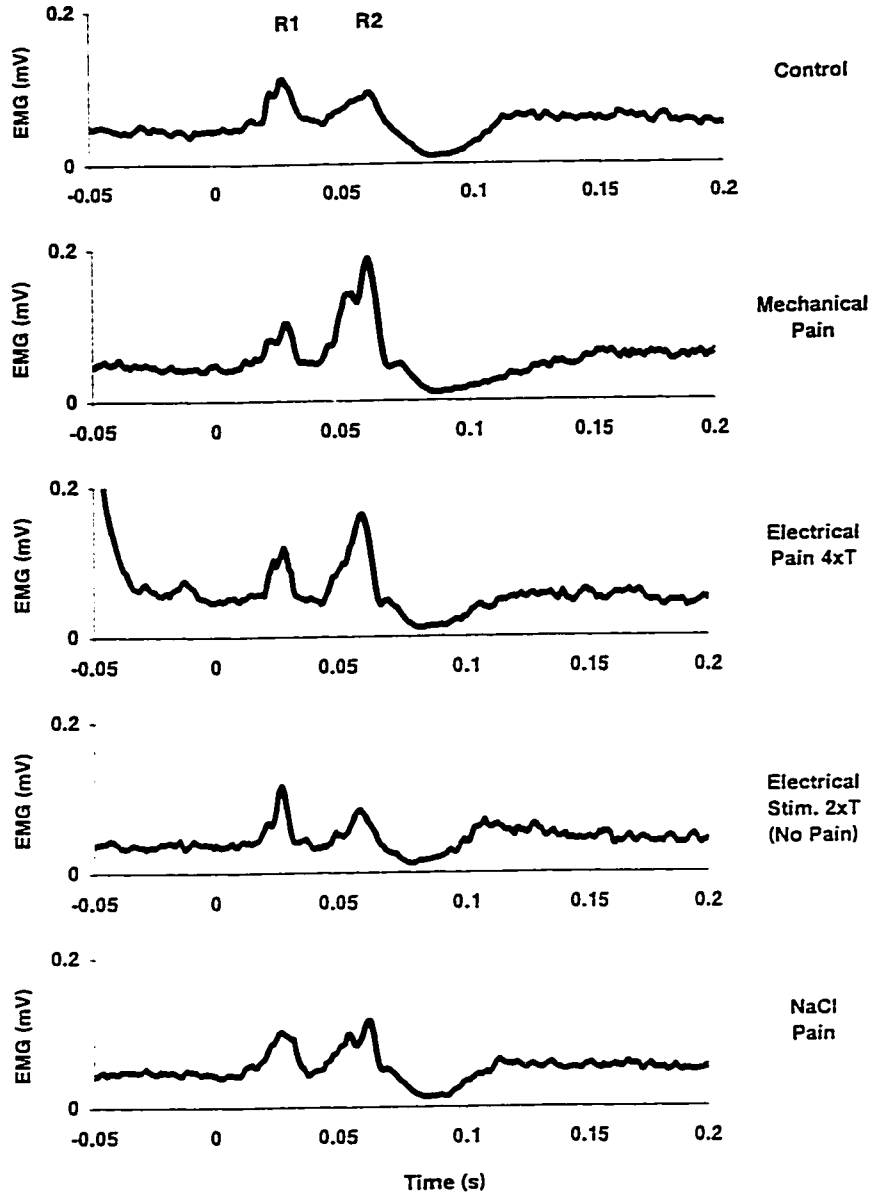

Figure 5.5. EMG responses of the right ES to indentations under non-painful and painful conditions (average of 20 trials in one subject). Note the increased R2 amplitude with painful cutaneous stimulation (mechanical, electrical 4xT) while non-painful electrical stimulation (2xT) and deep muscle pain had no effect on either R1 or R2 responses.

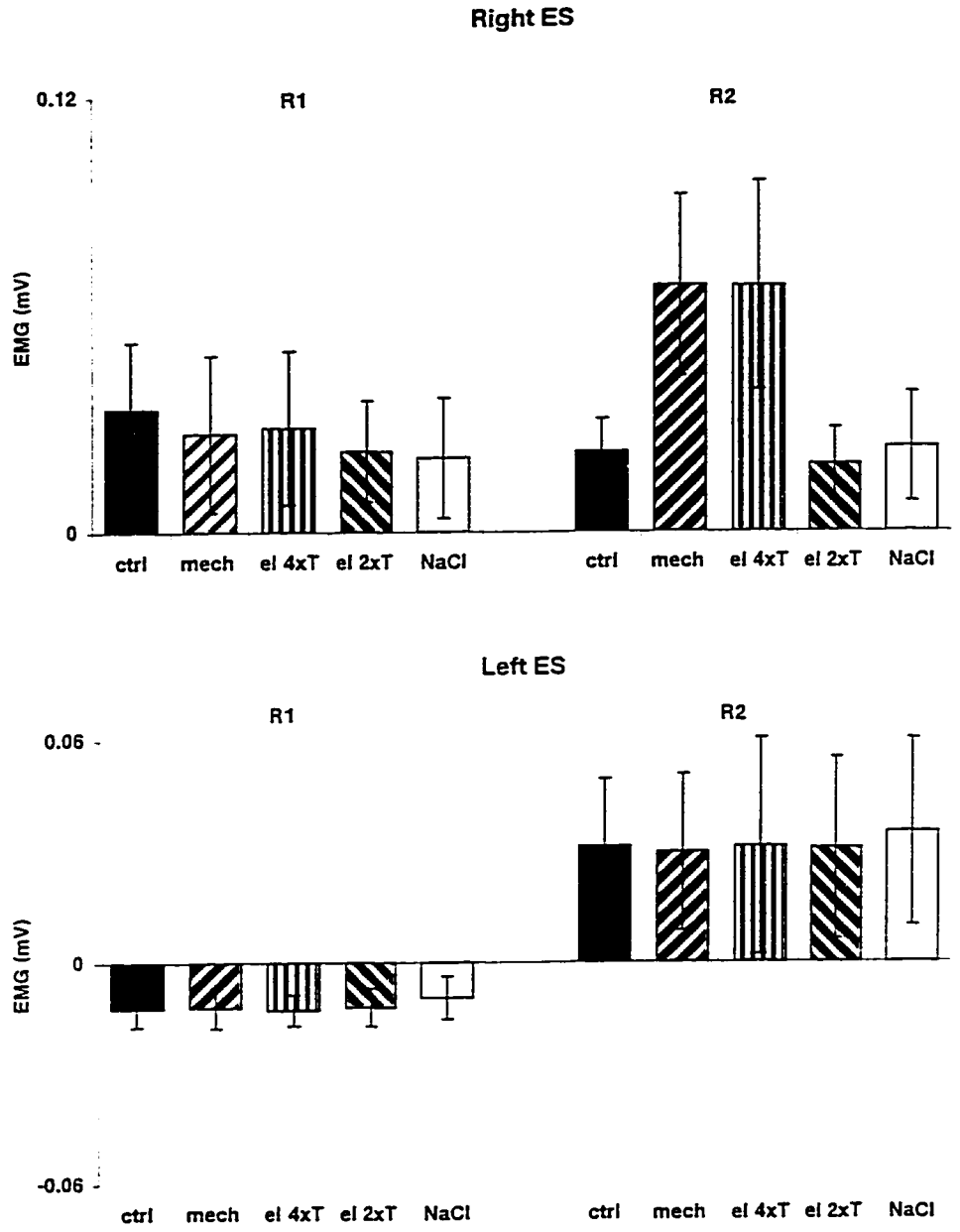

Figure 5.6. EMG amplitude (mean ± one standard deviation bars) of the short (R1) and long (R2) latency responses from the right and left ES after subtraction of the mean pre-stimulus background EMG values. Right ES – right erector spinae; Left ES – left erector spinae; ctrl – painless control condition; mech – painful mechanical stimulation on the skin; el 4xT – painful electrical stimulation of the skin; el 2xT – non-painful electrical stimulation of the skin; NaCl – deep muscle pain.

5.4 Discussion

In this study we investigated the effect of muscle and cutaneous pain on the back muscle stretch reflex on the hypothesis that an increased stretch reflex and therefore increased muscle tone occurs during pain (Berberich *et al.*, 1987; Johansson & Sojka, 1991).

The stretch reflex in ES was first described in detail by Dimitrijevic *et al.* (1980). Tapping ES at the level of L5 revealed a short latency response (R1) at 12 ± 1.6 ms and a long latency response (R2) between 30 and 50 ms, which seemed to be independent of each other. R1 was more constant than R2 and showed vibratory-induced suppression, postvibratory facilitation and facilitation during a Jendrassik manoeuvre. These observations and the reflex latency led the authors to the conclusion that the R1 response was probably oligosynaptic. In contrast, R2 was more variable and was increased during muscle vibration. Its latency and its bilateral presence during unilateral tapping suggested a polysynaptic pathway.

A further description of the ES stretch reflex was provided recently by Tani *et al.* (1997). The reflex was elicited by taps in the interspinous space at various thoracic and lumbar levels. R1 was more reproducible than R2 and its latency was shortest when recorded at the tapped level. The latency increased in the caudal direction but not as steeply as the R2 latency, which resulted in a greater temporal separation between R1 and R2 caudally. Also, in contrast to R1, R2 recorded at L4-5 showed a progressive decrease in latency with more rostral stimuli. The authors suggested that R2 propagates rostrad from the stimulus site and may include supraspinal transmission. Based on rough calculations of conduction times, the authors implicated fast-conducting dorsal column pathways as a possible part of the loop.

There is also evidence from limb studies of segmental mechanisms contributing to long latency responses, such as polysynaptic spinal transmission (Ghez & Shinoda, 1978; Upton, McComas & Sica, 1971), slowly conducting afferents (Kirkwood & Sears, 1974; Matthews, 1984), or repetitive spindle discharge (Hagbarth, Hagglund, Wallin & Young, 1981; Eklund, Hagbarth, Hagglund & Wallin, 1982). It seems likely that more than one pathway or mechanism is involved in generating long latency reflex activity.

In connection with the present study, it is appropriate to mention that cutaneous input has also been proposed to participate in the generation of the long latency stretch reflex. Marsden, Merton and Morton (1972) showed that anesthesia of the thumb suppressed the late response in the long flexor of the thumb. Although subsequent studies of various muscles, including ES, have shown that local anesthesia did not abolish the long latency response, a possibility that the stretch reflex is modified by cutaneous input cannot be ruled out (Datta & Stephens, 1981; Darton, Lippold, Shahani & Shahani, 1985). Deuschl, Schenck and Lucking (1985) showed that stimulation of cutaneous afferents evoked long latency reflexes in the thenar muscles similar to the response to simulation of a motor nerve suggesting that R2 may be a product of mixed proprioceptive and cutaneous input. Thus, while not necessary to generate the long latency response, cutaneous input may play a modulatory role. Conditioning a tap-elicited stretch reflex in back muscles with trains of electrical stimuli to the saphenous nerve in decerebrate cats demonstrated a facilitatory effect on the reflex amplitude if the conditioning-test interval was longer than 20 ms (Carlson & Lindquist, 1976). In a human study dealing with cutaneous reflexes Kugelberg and Hagbarth (1958) found facilitation of tonic ES activity by painful stimulation of the skin overlying the muscle. This response was considered as a withdrawal reflex because activation of ES would increase lumbar lordosis and thus remove the skin from the painful stimulus. Similar effects were observed in cats following mechanical stimulation of the skin of the trunk and electrical stimulation of nerves supplying the lumbar back skin (Carlson & Lindquist, 1976). The significant increase in R2 amplitude found in the present study reflects increased excitability of polysynaptic reflex pathways and is consistent with the concept of avoidance of painful stimulation. The noxious character of the cutaneous stimulation seems to be important since no effect was present if the stimuli were not painful.

In contrast to the facilitatory effect of skin stimulation, deep muscle pain did not change the amplitude of the long latency stretch reflex. This fact demonstrates that the differences between deep and superficial pain observed in the sensory domain (perceptual quality, pattern of spread etc.) can be extended to the motor output. From the present study it is not possible to identify the neural circuits responsible for the different effects of skin and muscle pain on the long latency response. It has been established that

cutaneous pain is signaled to the CNS through small diameter myelinated Aδ and unmyelinated C afferent fibres which terminate in laminae I, IIo and V in the dorsal horn and also in lamina X . Afferent fibres excited by noxious stimuli in deep tissues terminate mostly outside lamina IIo, particularly in laminae I and V (Willis, 1986; Mense, Light & Pearl, 1981; Craig & Mense, 1983; Sigiura, Terui, Hosoya & Kohno, 1989). Little is known about the connections of the second-order neurones in the dorsal horns with the α-motoneurones in the ventral horns but the different termination of the primary afferents suggests separate processing of the two types of pain. The interaction between the sensory and motor systems is polysynaptic and probably occurs at all levels of the CNS hierarchy. At the spinal level, the γ-motoneurones and interneurones synapsing onto the α-motoneurones have been considered as possible links between pain input and altered motor output (Johansson & Sojka, 1991; Lund, Donga, Widmer & Stohler, 1991). Γ (fusimotor) neurones receive a complex input from the skin, joints and muscles (Appelberg, Johansson & Sojka, 1986; Johansson, Sjolander & Sojka, 1988; Johansson, Sjolander, Sojka & Wadell, 1989). Stimuli that excite small diameter muscle afferents have been shown to increase the excitability of γ–motoneurones (Jovanovic, Anastasijevic & Vuco, 1990; Ljubisavljevic, Jovanovic & Anastasijevic, 1992; Johansson, Djupsjobacka & Sjolander, 1993; Djupsjobacka, Johansson & Bergenheim, 1994; Djupsjobacka, Johansson, Bergenheim & Wenngren, 1995, but cf. Mense & Skeppar, 1991). Johansson (1981) formulated a "final common output hypothesis" according to which γ-motoneurones integrate descending and multimodal reflex input and transmit the result to α-motoneurones via the spindle reflex arc. On this view, muscle spindle afferents are seen as the "final common output" to α-motoneurones. Pain-evoked fusimotor sensitization of the muscle spindles would increase the gain of the stretch reflex, which in turn would result in muscle hypertonus. According to this hypothesis muscle pain elicited in the present experiment was therefore expected to change the amplitude of the EMG response to muscle tap. Surprisingly, the reflex gain remained unchanged. This implies that spindle responses to indentation were unchanged and that there was therefore no pain-induced change in fusimotor action on spindles. There are caveats to this conclusion. First, it is just conceivable that fusimotor action did indeed increase with pain, but the incremental response to muscle indentation in our experiments

was unchanged either because the effects on spindle gain of increased static and dynamic fusimotor action happened to be equal and opposite (Prochazka, 1996), or spindles were taut throughout, so their afferent responses to indentation were "saturated" and therefore unresponsive to fusimotor changes (Wood, Morgan, Gregory & Proske, 1994). On balance however, the parsimonious conclusion is that deep back pain of the type we studied does not cause significant changes in fusimotor action. This is a fairly important point, because the notion of a fusimotor involvement in chronic muscle pain is based on several lines of evidence from acute experiments and is widely entertained as being a plausible mechanism.

The demonstration of a lack of pain effect on the short latency R1 response confirmed the conclusions of previous studies in limbs that pain has no influence on the monosynaptic spinal pathway as tested through the H- reflex (Willer & Bussel, 1980; Grossi & Arner, 1984; Willer, Bergeret, De Broucker & Gaudy, 1988; Leroux, Belanger & Boucher, 1995). Although the short latency stretch reflex evoked by muscle tap may not be exclusively monosynaptic (Burke, Gandevia & McKeon, 1984), evidence from the limb muscles shows that the monosynaptic component is dominant. Notwithstanding presynaptic inhibitory mechanisms, the monosynaptic pathway is probably exposed to less modulation than polysynaptic pathways. However, the number of synapses involved in the short latency transmission in human back muscles is not established. Although monosynaptic connections of ipsilateral low-threshold afferents onto back muscle α-motoneurones were demonstrated by intracellular recordings in cats (Jankowska & Odutola, 1980), an earlier report considered the monosynaptic pathway for the stretch reflex in cats as exceptional and suggested a polysynaptic route instead (Carlson, 1978).

The tap to the right ES resulted in a short-latency attenuation of EMG in the contralateral muscle. Although reciprocal inhibition between trunk muscles has been observed in both animals and humans (Kugelberg & Hagbarth, 1958; Trontelj *et al.*, 1979; Eble, 1961; Zedka, Kumar & Narayan, 1998), the pathways connecting the human axial muscles have not been satisfactorily described. Latency measurements during intracellular recordings from cat motoneurons revealed that, unlike limb antagonists, reciprocal inhibition between contralateral paraspinal muscles is not mediated through a disynaptic pathway (Jankowska & Odutola, 1980). Similar information on reciprocal

inhibition between human axial muscles is not available. Even though the results of the present study were not precise enough for accurate estimates of the number of synapses involved in the inhibition, it demonstrated for the first time that the delay between ipsilateral excitation and contralateral inhibition can be as short as 5 ms, suggesting an oligosynaptic pathway.

5.5 Conclusion

In conclusion, increased stretch reflex gain (of either monosynaptic or polysynaptic pathways) does not seem to be the mechanism of increased muscle tone during back pain originating from deep tissues. Our results do not support the idea that muscle spindles are sensitized by fusimotor action elicited by deep pain, at least of the type evoked by saline infusion. In contrast to the lack of effect of deep pain on ES stretch reflex, cutaneous pain did increase the amplitude of the long latency response, suggesting facilitation of polysynaptic reflex pathways.

5.6 References

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CHAPTER 6.

Voluntary trunk movement during induced back muscle pain in humans[1]

6.1 Introduction

Low back pain is a common disabling musculoskeletal disorder. Both prevention and treatment of this condition are problematic due to insufficient understanding of the underlying pathological processes. Unfortunately, back pain can reflect any tissue pathology in the low back or pelvic region (bones, ligaments, joints, muscles, viscera etc.). Since current imaging techniques do not identify the source of pain in the vast majority of cases (Arangio, Hratzell & Reed, 1990; Hoffmann, Kent & Deyo, 1991; Wiesel, Tsourmas, Feffer, Citrin & Patronas, 1984; Boden, Davis, Dina, Patronas & Wiesel, 1990), the diagnosis of low back disorder is often based on non-specific signs, such as deep tissue pain and altered motor patterns (McCombe, Fairbank, Cokersone & Pynsent, 1989). Although these are often the only diagnostic indicators, the relationship between them remains unclear. Many studies have investigated the functionality of back muscles and discovered that their output is abnormal in back pain. The force generated by the muscles is consistently diminished but electromyographic (EMG) recordings are equivocal – both hyper- and hypoactivity have been reported (Alston, Carlson, Feldman & Grimm, 1966; Jayasinghe, Harding, Anderson & Sweetman, 1978; Hoyt *et al.*, 1981; Collins, Cohen, Nailboff & Schandler, 1982; Thorstensson & Arvidson, 1982; Wolf, Nacht & Kelley, 1982; Nouwen & Bush, 1984; Ahern, Follick, Council, Laser-Wolston & Litchman, 1988; Arena, Sherma, Bruno & Young, 1989).

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The cause and extent of the muscle weakness, whether it is present in all types of movement, and how to reconcile it with the contradictory EMG findings, are issues that remain to be determined. Unfortunately, an important factor responsible for the contradictory results from clinical studies seems to be the non-homogeneity of their low-back pain population. As a direct consequence of the difficulty with localizing the primary tissue damage, the clinical studies may contain patients with different primary afflictions. Also, in clinical studies there is no healthy "norm" to which the patients could be compared. Data from the individual's pain-free history are rarely available and there is a certain inter-individual variability in motion patterns even within the healthy population (Gomez, 1994). Factors such as these are responsible for the current poor understanding of the complex pathology of back pain.

The role of lumbar structures in back pain and the influence of pain on their function may be better studied by an alternative approach that bypasses some problems encountered in clinical back pain studies. Kellgren's experiments in the 1930s showed that injections of hypertonic saline into muscles or ligaments cause deep pain comparable to spontaneous musculoskeletal pain (Kellgren, 1938a; Kellgren, 1938b). The advantage of this method is that both the intensity and location of the pain can be controlled. By selectively targeting a chosen structure one can determine the impact of pain on the surrounding tissues and general motor patterns. An obvious candidate for studying the link between pain and altered movement is skeletal muscle. Inappropriate neural control of a given muscle has been suggested to bring about compensatory changes in synergistic muscles disposing them to overload and injury (Janda, 1986; Edgerton, Wolf, Levendowski & Roy, 1996). In our study we therefore examined the effect of pain on the activity of a back muscle, the erector spinae (ES), and its neighboring muscles. In recent studies pain induced by saline infusion in jaw and neck muscles did not cause changes in muscle output in the sense of general hyper- or hypoactivity, as had often been posited. Any changes observed depended rather specifically on the functional context (Stohler, Ashton-Miller & Carlson 1988; Ashton-Miller, McGlashen, Herzenberg & Stohler, 1990; Arendt-Nielsen, Graven-Nielsen, Svarrer & Svensson, 1995; Stohler, Zhang & Lund, 1996; Svensson, Arendt-Nielsen & Houe, 1995; Svensson, Houe & Arendt-Nielsen, 1997). A "pain adaptation" model of muscle function in pain has been proposed,

according to which muscles acting as agonists become less active while those acting as antagonists become more active (Lund, Donga, Widmer & Stohler, 1991). This arrangement would, it was argued, decrease forces in the painful structures and act as a protective mechanism to prevent further damage. The present study investigates whether this model can be applied to voluntary trunk movements during back pain since both muscle "weakness" and "hyperactivity" have been frequently reported in back pain (Alston *et al.*, 1966; Jayasinghe *et al.*, 1978; Hoyt *et al.*, 1981; Collins *et al.*, 1982; Thorstensson & Arvidson, 1982; Wolf *et al.*, 1982; Nouwen & Bush, 1984; Ahern *et al.*, 1988; Arena *et al.*, 1989). Our results led us to ask whether the altered motor patterns we observed could be the result of conscious avoidance of imminent pain. We will argue that if this were the case, the motor patterns and responses should have returned to normal when pain was no longer experienced. We conclude that the enduring sensorimotor changes that outlast the experience of pain point to a slow neuromodulatory mechanism.

6.2 Methods

6.2.1 Subjects

Five healthy volunteers (1 female and 4 males, age 20 – 40 years) participated in the study. They were familiarized with the protocol and signed a consent form in accordance with the requirements of the University of Alberta, Faculty of Medicine Research Ethics Board. The study was conducted according to the Declaration of Helsinki.

6.2.2 EMG

After preparation of the skin with ethyl alcohol, bipolar surface EMG from the right and left ES at the third lumbar (L3) and tenth thoracic (Th10) vertebral levels was monitored with self-adhesive Ag/AgCl electrodes (Figure 6.1.; ElectroTrace® ET 301,

Huntington Beach, California, USA). The EMG signal was amplified (gain 1000), band-pass filtered (30Hz – 50 kHz), full-wave rectified, low-pass filtered (300 Hz), and sampled at 500 s^{-1} by a 1401 laboratory interface (Cambridge Electronic Design, England). SIGAVG 6.30 software running on a personal computer was used to store and average the recordings. As part of the off-line analysis the EMG signal was digitally smoothed for plotting purposes using a second-order low-pass (forward and reverse, zero-phase) filter with a cutoff frequency at 1.6 Hz (Matlab software, The Mathworks Inc.).

6.2.3 Motion analysis

Body motion was quantified using the 6D-RESEARCH™ Motion Capture and Analysis System (Skill Technologies Inc., Phoenix, Arizona, USA). The system's hardware includes two stationary electromagnetic transmitters and sensors responding to motion within the magnetic field. Sensor # 1 was taped to the subject's forehead. Sensors # 2,3 and 4 were taped over the spinous processes of the 7th cervical vertebra (C7), the 10th thoracic vertebra (Th10) and the 3rd lumbar vertebra (L3), respectively. Sensor # 5 was placed on the lateral side of the right knee (RK). The 3D-motion data were captured and stored in a high speed personal computer using the 6D-RESEARCH™ software. Kinematic images were generated from the sensor trajectories displayed on the computer screen. To enhance the contrast of the markers, the figures were edited with Corel PHOTO-PAINT™ 6.0 software.

6.2.4 NaCl infusion

Subjects were seated on a chair. A small area of skin over the right ES 3 cm lateral to the L3 spinous process was cleaned with alcohol and an intravenous catheter/needle (1.1 mm in diameter, 5.1 cm long) was inserted perpendicularly to the surface of the back, 4 cm into the muscle. An intravenous infusion set attached to a 10 ml disposable syringe was filled with 5% NaCl. The needle was pulled out and the infusion set was connected to the intravenous catheter, which remained in the muscle. The syringe with saline was placed in an infusion pump (Harvard Apparatus, model 22).

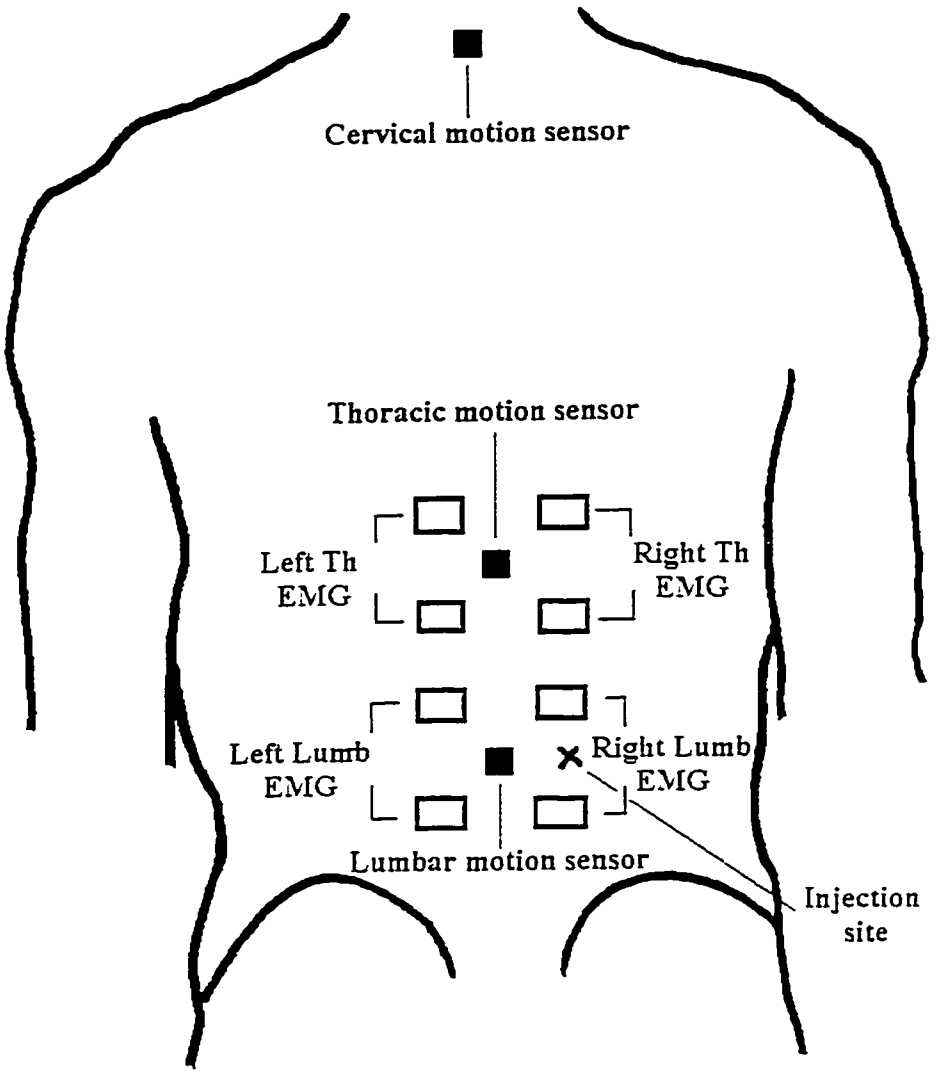

Figure 6.1. Placement of EMG electrodes and motion markers on the back. Left Lumb – left lumbar erector spinae, Right Lumb – right lumbar erector spinae, Left Th– left thoracic erector spinae, Right Th – right thoracic erector spinae.

The pump was controlled digitally by a computer, which regulated the infusion rate in a feed-forward compensation manner in order to keep the pain approximately constant. The profile of the infusion rate over approximately 25 min was an inverse function of the mean pain rating profile obtained from two subjects during preliminary tests when 5% NaCl was infused at several constant rates (50, 60, 100, 140, 150 and 200 $\mu l.\ min^{-1}$) for 12 min. The subjects verbally rated their pain every 15 seconds on a 0 –10 scale, where 0 represented "no pain" and 10 was "unbearable pain". Pain rating profiles during the main experiment revealed that the feed-forward regulation of the infusion rate provided minimal fluctuations in perceived pain (see Appendix).

6.2.5 Experimental paradigm

With the EMG and the motion analysis systems in place the subjects were asked to stand upright with their eyes closed. All tasks were performed with a stabilized pelvis, which was achieved by firmly strapping the subject about the hips to a wall. Preliminary testing during which a sensor was placed on the right posterior superior iliac spine confirmed that this arrangement kept pelvic movement below 5° (angle measured between the principal axes of the sensors placed on the pelvis and the right knee). The subject's knees were also stabilized in extension with straps attached to the wall.

Each of the following tasks was performed three times before the saline infusion, three times during the infusion, and finally three times after the infusion was terminated and there had been a verbal pain rating of zero for 30 minutes.

Trunk flexion-extension

On a "ready-steady-go" signal the subjects bent their trunk forward from the waist as far as they could without bending their knees. They remained in the flexed position for 1 second, then returned to the upright position. The whole task was typically completed in 8 seconds.

Lateral flexion

The subjects bent from the upright position to the left as far as possible, then back to the upright position, to the right and again back to the upright position. With short pauses between each phase of the task, the duration was again about 8 sec.

Axial rotation

Starting from the upright position facing forward the subjects first rotated to the left from the waist, then back to the starting position, then to the right and back to the starting position with similar timing to the above. Their hands were clasped horizontally in front of the body and did not touch the trunk.

6.2.6 Data analysis

The mean amplitude of the rectified EMG signal was calculated for defined phases of the task cycle (see dashed vertical lines in Figures 6.2., 6.3. and 6.4.). These phases were defined on the basis of discrete events in the EMG signal in non-painful conditions. EMG was chosen rather than the angular displacement signal because it was the EMG modulation that was of major interest. The start and end of the whole sequence was defined in terms of the onset and the end, respectively, of EMG activity in the injected muscle, the right lumbar ES. The transitions between flexion and extension, between the left and right lateral flexion and between the left and right axial rotation were defined as a one-second-long interval of the lowest EMG activity. The timing of divisions defined with respect to the right lumbar ES (no pain) was applied to all other muscles. Student's t-test (paired two-sample test for difference of means) was used to compare between the mean of the EMG signals within corresponding phases before, during and after pain. The significance level was set at $\alpha = 0.05$. Analysis was performed with Microsoft® Excel 97 software.

6.3 Results

6.3.1 Before pain

The EMG patterns of pain-free flexion-extension of one subject are shown in Figure 6.2. The right and left ES muscles participated in both flexion and extension. The muscles were activated in the early phase of flexion (sometimes preceded by initial inhibition, if resting background activity was present – not shown) and remained active until a certain lumbar angle (mean ± standard deviation: 65° ± 11°) was reached where they became silent. The final phase of flexion was completed with minimal ES contribution. In the static flexed position there was virtually no ES EMG activity (relaxation). Trunk re-extension was accomplished with strong ES activation, which started before the movement and continued until the upright position was reached.

During lateral flexion ES muscles on both sides were active (Figure 6.3.). The thoracic portion of each ES produced more EMG during the ipsilateral bend than during the contralateral bend. This difference was not so marked in the lumbar portion. The maximal angular displacement in the frontal plane of the lumbar (L3) motion sensor was 19° ± 8° to the left and 17° ± 5° to the right. The thoracic sensor (Th10) was displaced 56° ± 10° to the left and 45° ± 8° to the right.

The EMG pattern during axial rotation was similar to that in lateral flexion. While the thoracic portion of ES was strongly involved in rotation to the ipsilateral side, it was little active during the contralateral rotation. The lumbar portion was more evenly active during rotation in both directions (Figure 6.4). The L3 motion sensor showed angular displacement of 19° ± 5° to the left and 14° ± 7° to the right in the transverse plane.

6.3.2 During pain

Unilateral back pain changed the EMG patterns bilaterally in a direction-specific manner (Figures 6.2. and 6.5.). While the mean ES EMG amplitude during trunk flexion was not significantly changed, the ES relaxation observed during the final phase of flexion in painless conditions was absent in pain ($p < 0.05$). This ES activity persisted while flexion was maintained. During the extension phase the mean EMG amplitude was

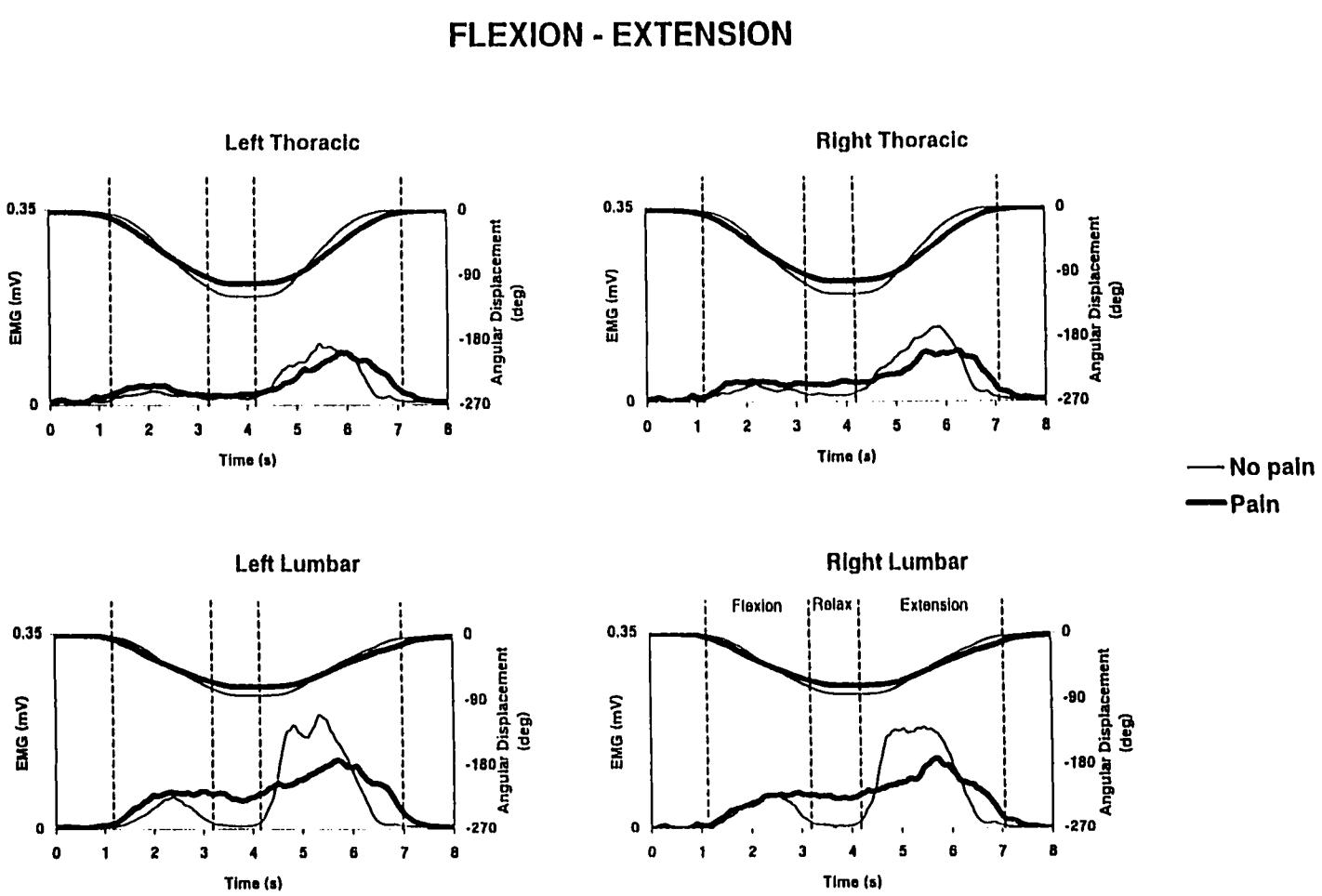

Figure 6.2. EMG patterns from the back muscles during painless and painful flexions-extensions of one subject (average of 3 trials for each). The angular displacement in the sagittal plane of the 3rd lumbar vertebra is shown together with EMG traces of the lumbar erector muscles and the displacement of the 10th thoracic vertebra is shown with thoracic EMG traces. Vertical dashed lines demarcate the three intervals over which mean EMG amplitude was calculated (see section 6.2.6.). Note the smaller depths of modulation of EMG with pain.

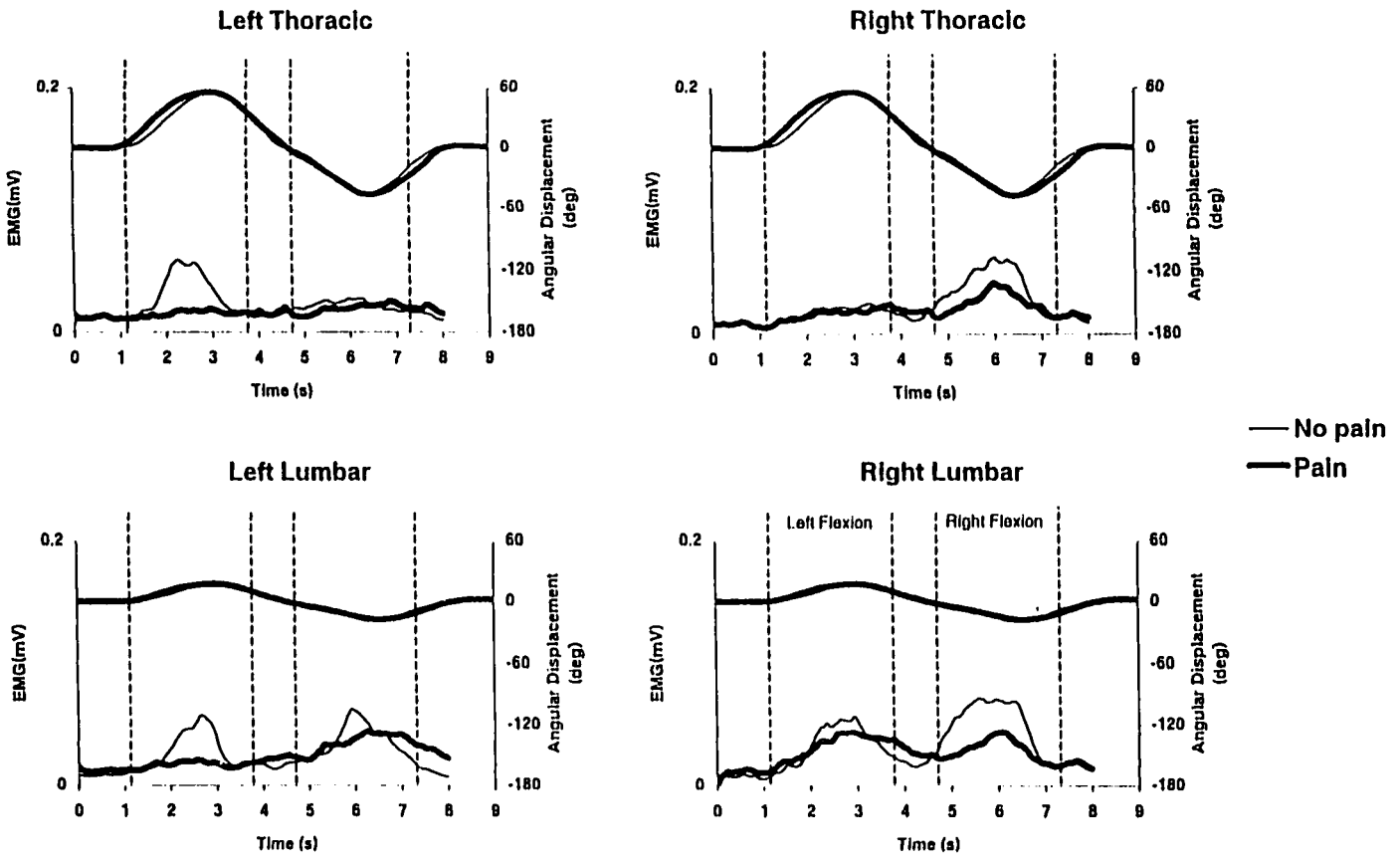

Figure 6.3. EMG patterns from the back muscles during painless and painful lateral flexion of the same subject as in Figure 6.2. The angular displacement in the frontal plane of the 3rd lumbar vertebra is shown with the EMG traces from the lumbar erectores spinae and the displacement of the 10th thoracic vertebra is shown with the thoracic erector spinae EMG traces.

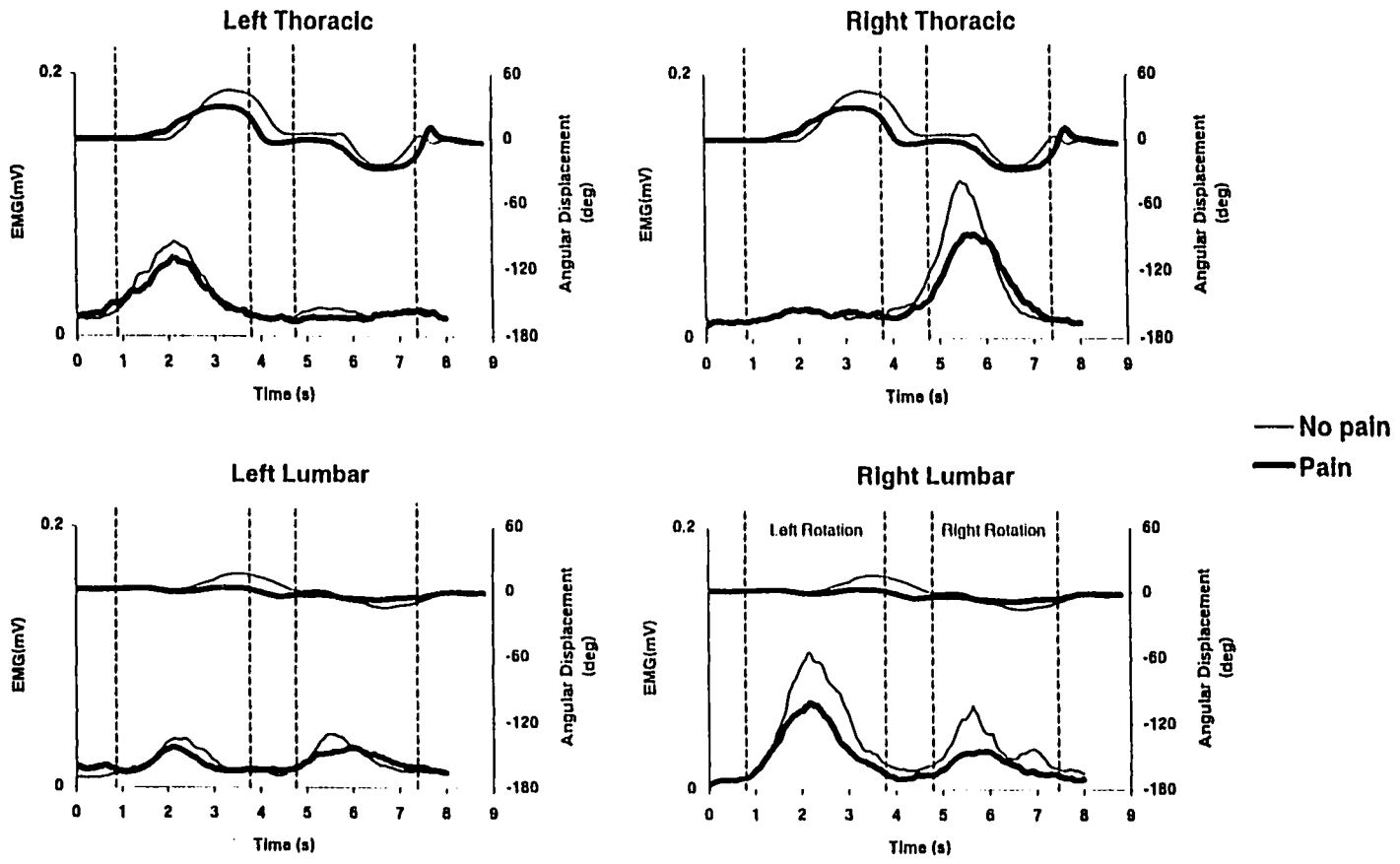

Figure 6.4. EMG patterns from the back muscles during painless and painful axial rotation of the same subject as in Figure 6.2. The angular displacement in the transverse plane of the 3rd lumbar vertebra is shown with lumbar EMG traces and the displacement of the 10th thoracic vertebra is shown with thoracic EMG traces.

significantly decreased compared to the painless extension ($p < 0.05$). Lateral flexion and axial rotation during pain were associated with decreased ipsilateral ES EMG during both left and right bends. This pattern was more pronounced in the thoracic portion of ES. In the lumbar portion pain decreased the mean amplitude in both the ipsilateral and contralateral muscles (Figures 6.3. and 6.4.). The influence of pain on EMG is summarized in Table 6.1., where the relative changes are presented as a ratio of the mean EMG amplitude during pain and the mean EMG amplitude in controls.

As illustrated in Figures 6.2.,6.3.,6.4. and 6.6., pain somewhat decreased the range of motion and speed in all types of movements, even though the subjects had been instructed to produce similar excursions. Interestingly, lumbar excursions were less affected in lateral flexion than in flexion-extension or axial rotation. Figure 6.6. also shows that the effect of unilateral back muscle pain on the range of motion to the left and to the right was approximately symmetrical. It is important to notice that during flexion-extension unilateral back pain did not cause major deviations of the trunk from the sagittal plane, indicating that whatever the changes in muscle activation, the kinematic outcome was not markedly asymmetrical.

6.3.3 After pain

Figure 6.7. shows the flexion-extension EMG pattern of one subject 30 minutes after he started rating the pain as zero. The same pattern was found in three out of five subjects. Even in the absence of pain the EMG amplitude during dynamic extension in the injected muscle (right lumbar ES) was significantly different from the control. At the other recording sites there was no significant difference from the trials before injection. In the right lumbar ES it is also noticeable that while the extension EMG was still smaller than control 30 min after pain, the flexion-relaxation phenomenon was fully recovered.

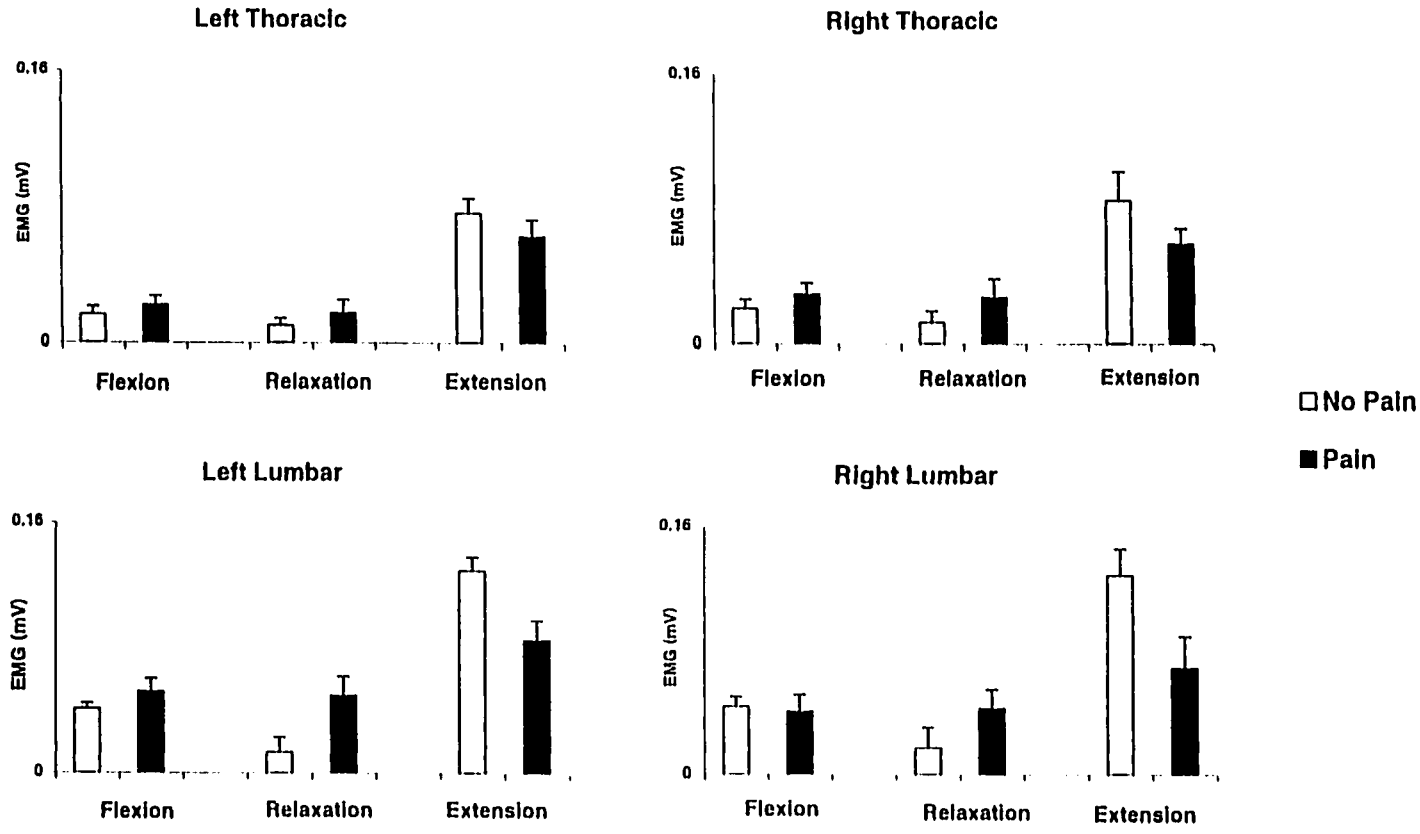

Figure 6.5. EMG amplitudes (mean and standard deviation bars) from the four back muscles monitored of all 5 subjects during three phases of movement (flexion, relaxation, extension) under painful and painless conditions.

Table 6.1. Relative changes of the mean back muscle EMG amplitude with pain, expressed as a percentage of the EMG amplitude before pain. RL3 – right lumbar erector spinae, LL3 – left lumbar erector spinae, RT10 – right thoracic erector spinae, LT10 – left thoracic erector spinae.

	Flexion - Extension			Lateral Flexion		Axial Rotation	
	Flexion	Relax	Extension	Left	Right	Left	Right
RL3	93.2	251.6	53.2	106.3	69.2	67.5	57.8
LL3	121.4	380.7	65.6	68.1	104.3	90.3	101.1
RT10	141.8	221.0	69.7	94.0	80.3	101.0	83.9
LT10	133.4	174.2	82.6	59.6	97.0	89.6	87.3

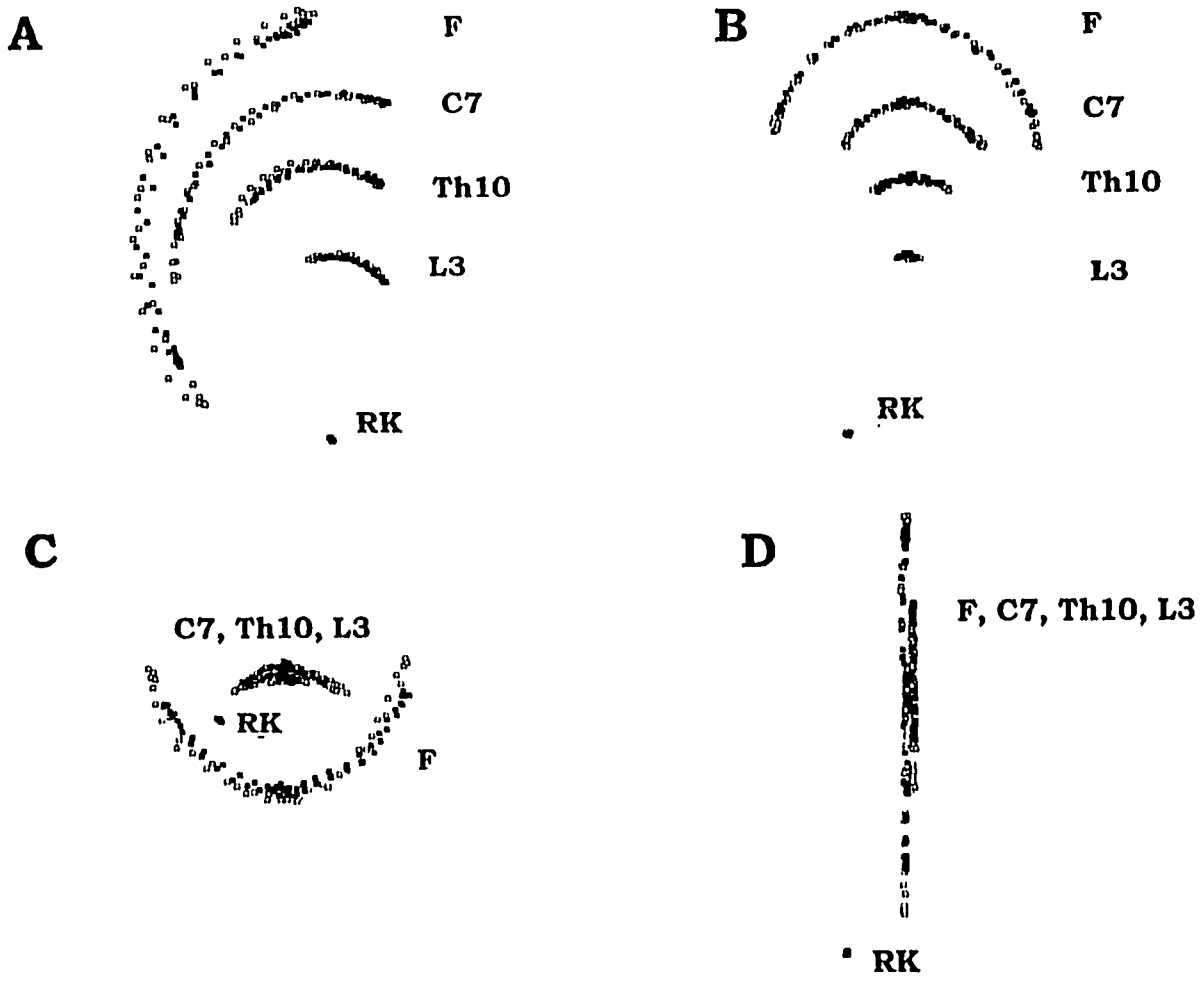

Figure 6.6. Trajectories of kinematic sensors during flexion-extension (A – lateral view, D – frontal view), lateral flexion (B – frontal view) and axial rotation (C – view from above). F- forehead sensor, C7 – neck sensor, Th10 – thoracic sensor, L3 – lumbar sensor, RK – right knee sensor. Painful trial (■), painless trial (□).

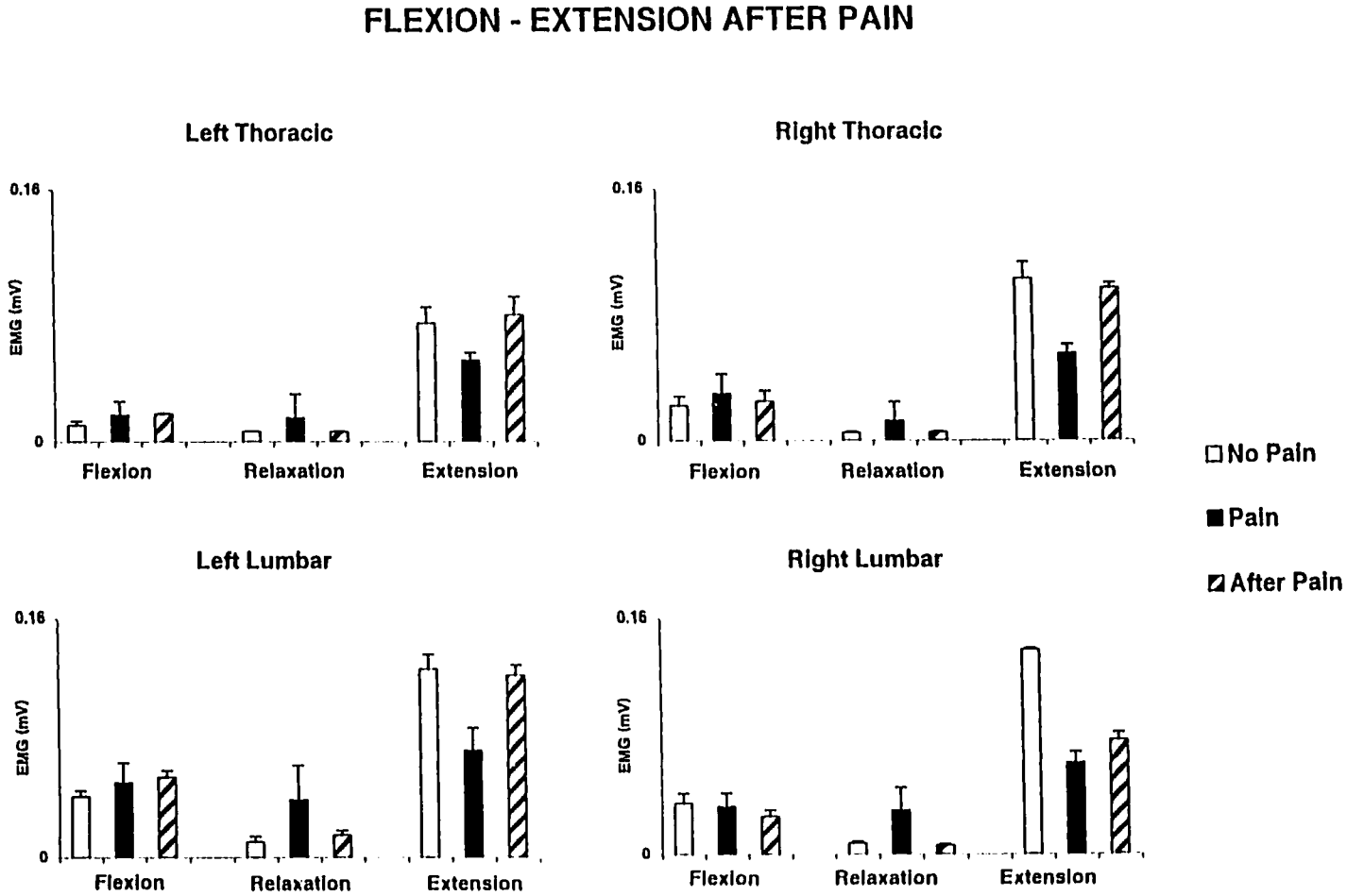

Figure 6.7. EMG amplitudes (mean and standard deviation bars) from the four back muscles during flexion-extension of one subject before, during, and 30 minutes after pain (average of 3 trials for each condition).

6.4 Discussion

The findings of this study indicate that unilateral injection of hypertonic saline into the lumbar portion of ES produced symptoms similar to those encountered in low back pain patients. The pain was described by the subjects as deep and dull, as expected for pain originating from deep tissues (Mense, 1991). Another characteristic feature similar to spontaneous back pain was the irradiation. The pain spread from the site of injection 3 cm to the right of the L3 spinous process down the right ES into the right superior gluteal region. The thoracic portion of the right ES and the left ES were never described as painful. The gluteal pain was probably of reflex origin since spontaneous lumbar pain follows the same pattern and referred pain has been reported after administration of hypertonic saline (Kellgren, 1938a; Kellgren, 1938b; Travell, 1983; Arendt-Nielsen, Graven-Nielsen, Svensson & Jensen, 1997; Graven-Nielsen, Arendt-Nielsen, Svensson & Jensen, 1997; Graven-Nielsen, Arendt-Nielsen, Svensson, & Jensen, 1997). It cannot be ruled out, however, that saline spread into the gluteal region due to gravity and/or intramuscular pressure gradients since injected fluid has a tendency to diffuse through the muscle along its longitudinal fascicular planes (Amis, Prochazka, Short, Trend & Ward, 1987).

Although pain was strictly unilateral in the lumbar region and below, the motor symptoms were bilateral at both lumbar and thoracic levels, at least for flexion-extension. This finding suggests that weakening of a muscle by pain does not always result in an increased activity of its synergists to compensate for the lost force production, as has been suggested (Janda, 1986; Edgerton *et al.*, 1996). On the contrary, the simultaneous inhibition of both the left and right ES during trunk re-extension or the ipsilateral ES inhibition during lateral flexion (even to the uninjected side) decreased the velocity and range of trunk motion without kinematic asymmetry, which supports the guarding theory consistent with the "pain adaptation" model (Lund *et al.*, 1991). It seems that EMG output can be changed even in non-painful muscles, if their action results in movement of the affected region, as in the case of the left ES in left lateral flexion. The limited range and velocity of movement have a "splinting effect" presumably resulting in reduced

afferent input from the painful muscle. It is possible that the pain-adapted movement trajectory provides a minimal mean-square jerk of the trunk (jerk being defined as the rate of change of acceleration, or equivalently, the third derivative of displacement), which has been found to characterize smooth movements (Flash & Hogan, 1985).

The "pain adaptation" model predicts that during the antagonistic phase of movement muscle activity should be increased. In the case of ES this was true for the flexed position where EMG activity was normally absent or very small. During most of the dynamic phase of flexion, however, the increase in ES EMG in pain did not reach significance even though the angular velocity was smaller with pain. This may mean that muscles other than ES had an altered activation profile. For example, it has been shown that fast trunk flexion is initiated and controlled by contraction of abdominal muscles (Thorstensson, Oddsson & Carlson, 1985). If the abdominal muscles were less active in back pain (notice the less active left ES during left lateral flexion in this paper), an increased braking action of ES would not be necessary to explain the slowed dynamic flexion. Another possible explanation for the absence of pain effect on the EMG during dynamic flexion could be a reflex origin of the ES activation. During re-extension ES has to be sufficiently voluntarily activated to overcome initial trunk inertia and to maintain movement momentum against gravity. Trunk flexion requires less EMG activity since gravitational force acts in the direction of the movement. Also, during eccentric contractions significantly larger forces are generated for a given level of activation (EMG) due to visco-elastic properties of the muscle potentiated by its stretch reflexes. If the EMG during flexion reflected predominantly a stretch reflex rather than voluntary activity, the non-significant effect of pain on this phase would be in line with our finding that deep muscle pain has no effect on the stretch reflex (please see accompanying paper). It would also suggest that pain influences descending control much more than it does segmental control.

The persisting ES activity in static flexion is difficult to explain. Although the flexion-relaxation phenomenon was noticed by Allen (1948) as early as 1948 and its absence in low back patients has since been reported by many authors (Ahern *et al.*, 1988; Floyd & Silver, 1951; Floyd & Silver, 1955; Valencia & Munro, 1985; Triano & Schultz, 1987; Paquet, Malouin & Richads, 1994; Shirado, Ito, Kaneda & Strax, 1995;

Gracovetsky, 1988), its origin is still unknown. It seems that the increased ES activity reflects more than the patient's unwillingness to flex fully due to pain. The range of trunk motion is indeed smaller in the absence of relaxation but the patients are still able to flex beyond the angle where relaxation appears in healthy subjects (Triano & Schultz, 1987). It has been suggested that from the point where the ES muscles become silent, passive lumbar structures such as interspinous ligaments etc. take over the trunk load. The function of ES in flexion is in fact to ensure adequate loading of these structures through the control of lumbar lordosis (Gracovetsky, 1988). An absence of the flexion-relaxation phenomenon may indicate that the injured ligaments cannot sustain these forces and have to be protected by ES contraction (Floyd & Silver, 1955). However, in the present study it is hard to see why the (painful) muscles should remain contracted to reduce loading of uninjured, painless ligaments. It can be speculated that pain in any lumbar structure (ligaments, joints or muscles) is transmitted to ES motoneurones through a common element in the central neuronal circuitry. The muscle then works in a "pain mode" to protect the spine from extreme movement whenever pain is signaled, without distinguishing the tissue of origin. Candidates for such a common element are dorsal horn neurons known to receive mixed input from deep tissues (Schaible, Schmidt & Willis, 1987; Hoheisel & Mense, 1989). Ascending signals from these neurons can interact with motor commands at higher levels as well as at the spinal level. It has also been shown that signals from small-diameter afferents reach group II interneurons (Lundberg, Malmgren & Schomburg, 1987) and γ-motoneurones (Johansson, Sjolander & Sojka, 1988; Johansson, Sjolander, Sojka & Wadell, 1989; Appelberg, Johansson, Sojka, 1986; Jovanovic, Anastasijevic & Vuco, 1990; Ljubisavljevic, Jovanovic & Anastasijevic, 1992; Johansson, Djupsjobacka & Sjolander, 1993; Djupsjobacka, Johansson & Bergenheim, 1994; Djupsjobacka, Johansson, Bergenheim & Wenngren, 1995; Mense, Skeppar, 1991; Johansson, 1981) located more ventrally in the spinal cord. Excitatory and inhibitory populations of group II interneurones are intercalated in descending pathways to α-motoneurones of the limbs (Lundberg *et al.*, 1987). It is not known whether the same applies in trunk segments but descending pathways could reach phylogenetically old axial α-motoneurones to a large extent indirectly (Rothwell, 1994). Therefore, one of the sites of modulation of motor signals by pain may be here. In fact,

Lund and his co-workers drew attention to groups of excitatory and inhibitory interneurones in the spinal cord when they proposed possible circuitry for their "pain-adaptation" model (Lund *et al.*, 1991). They explained the increase of antagonist muscle EMG activity by reflex facilitation of the excitatory pathway and inhibition of the inhibitory pathway. Another site of motor output modulation might be the γ-loop since γ-motoneurones have been shown to receive reflex action from small-diameter fibres as well as from fibres signaling length or tension changes in ligaments and muscles (Johansson, Sjolander & Sojka, 1991; Sojka, Sjolander, Johansson & Djupsjobacka, 1991). It is conceivable that increased input from high-threshold afferents could increase fusimotor drive, making muscle spindle endings more sensitive to input from stretched muscles. However, if such an increase in spindle gain occurs, compensatory reductions in reflex transmission at motoneurones would be required to explain the lack of an increase in stretch reflexes (see accompanying paper).

The reduced modulation depth of EMG in the painful muscle could conceivably be due to a segmental inhibition by nociceptive input of descending voluntary commands to α-motoneurones. On the other hand, a possibility that the observed changes reflect a purely voluntary avoidance of pain was also considered. If conscious perception of pain were necessary for a modulation of the EMG patterns, no modulation should be seen when pain has dissipated. This was not the case, at least in the injected portion of ES (Figure 7, Right Lumbar). While the EMG pattern of two subjects returned to normal 30 minutes after pain, three subjects showed significantly decreased EMG amplitudes in the injected muscle during trunk re-extension. This reduction was probably due to a local inhibitory reflex action, since the EMG signal from non-injected sites had returned to control values and a selective voluntary inhibition of just the injected portion of ES during trunk extension is rather difficult without training. In Chapter 5 we observed that during an infusion of hypertonic saline well-motivated subjects found it difficult isometrically to generate ES EMG levels comparable to those without pain (unpublished observation). Our notion that a decreased voluntary drive alone is not responsible for decreased EMG amplitude in agonistic contractions is also supported by animal studies in which noxious muscle stimulation evoked specific modulation of masticatory movements

driven by electrical stimulation of descending pathways (Schwartz, Stohler & Lund, 1993; Westberg, Clavelou, Schwartz & Lund, 1997).

Whatever the reason for the decreased modulation depth, it seems to deprive painful muscles of sufficient relaxation. Interestingly, lack of relaxation due to repetitive movements or sustained contractions (muscle "overuse") has been considered as a possible reason for development of chronic musculoskeletal pain (Elert, Rantapaa Dahlqvist, Henriksson-Larsen & Gerdle, 1989; Veiersted, Westgaard & Andersen, 1990; Elert, Rantapaa Dahlqvist, Henriksson-Larsen, Lorentzon & Gerdle, 1992; Middaugh, Kee & Nicholson, 1994). Most attention has been devoted to the ergonomic arrangement of the workplace since pain has been associated with poor posture, monotonous movements and psychological stress. However, as several studies, including the present one, have shown, a paucity of EMG silent periods is also characteristic of painful muscles or muscles surrounding painful tissues (Stohler *et al.*, 1988; Ashton-Miller *et al.*, 1990; Arendt-Nielsen *et al.*, 1995; Stohler *et al.*, 1996; Svensson *et al.*, 1995; Svensson *et al.*, 1997). Though the widespread view that painful muscles are hyperactive even when subjects are at rest has not been substantiated in recent controlled studies, the question remains, how often during the day are painful muscles actually allowed to rest? A study of low-back pain patients has found almost continuous activity in their back muscles during the night (Fischer & Chang, 1985). Therefore there is a possibility that, paradoxically, the beneficial "splinting" muscle activity present within minutes after acute injury could in the long term lead to chronic musculoskeletal pain.

6.5 Conclusion

Back muscle pain induced a phase-specific alteration of trunk motor patterns. ES EMG activity was increased in phases where the muscles acted as antagonists and decreased where they acted as agonists, which is in agreement with the "pain adaptation" model. Unilateral back pain produced bilateral EMG changes leading to a reduction in velocity and range of the trunk movement. No compensatory activity in synergists of the injected muscle was seen. Although pain very probably alters voluntary commands,

changes in the motor output have an involuntary component. Our results from this and the accompanying paper indicate that deep pain changes descending motor commands but has surprisingly little effect on segmental stretch reflexes.

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CHAPTER 7.

GENERAL DISCUSSION

Compromised muscle function in patients with LBP has been frequently documented (Biering-Sorensen, 1984; Karvonen, Viitasalo, Komi, Nummi & Jarvinen, 1980; McNeill, Warwick, Andersson & Schultz, 1980; Smidt, Herring, Amundsen, Rogers, Russell & Lehmann, 1983). It is commonly held that the passive tissues of the spine are increasingly stressed with increasing muscle functional insufficiency, although the mechanism associating muscle insufficiency to LBP is not understood (Gracovetsky, 1988; Seidel, Beyer & Brauer, 1987). In the two concluding sections I will summarize my research on back muscles and will point out main problems, which, in my opinion, should be tackled in order to gain a better understanding of back pain.

7. 1 Contribution of the thesis

In order to discover the role of muscle insufficiency in LBP, it is necessary to study the functional capacity of the trunk muscles in appropriate tasks under normal conditions, as well as during fatigue and controlled back pain. The most popular non-invasive technique presently available to assess functional capacity of trunk muscles is surface EMG. Since EMG recording from trunk muscles is not standardized, the EMG signals require cautious interpretation. Chapter 2 was devoted to the influence of electrode type, distance and orientation on some parameters of the EMG signals from the erector spinae muscles. We found a linear relationship between two parameters of the surface EMG signal – the EMG amplitude and the total EMG power - and the force generated by isometric extension of the trunk. This finding confirms the usefulness of EMG measurements for estimates of relative force increments during this type of

exercise. On the other hand, no relationship between the median frequency of the EMG signal and force was discovered, which further supports the controversy about using the median frequency for estimates of muscle force (Bolodeau, Arsenault, Gravel & Bourbonais, 1990; Broman, Bilotto & De Luca, 1985; Roy, De Luca & Casavant, 1989; Bazzy, Korten & Haddad, 1986; Dolan, Mannion & Adams, 1995). In the course of the fatiguing isometric contraction the median frequency of the ES EMG power spectra had a consistently decreasing tendency while the total EMG power increased. The results suggest that both parameters objectively reflect muscle fatigue. While absolute values of the investigated EMG parameters varied with the type, distance and orientation of the electrodes, the trends observed during graded contraction and fatigue were the same in most configurations. This finding was especially important for the pairs of electrodes placed perpendicularly to the long axis of the fibres of ES. The effect of this placement on the parameters of the EMG signal has never been studied, although this orientation prevents electrode displacement during trunk flexion, which is a common source of signal noise. Sensitivity of the EMG signal parameters to minor electrode displacements has been documented (Roy, De Luca & Schneider, 1986).

In Chapter 3, the EMG activity of trunk muscles was recorded to address the issue of muscle coordination in postural tasks. A number of previous studies have concluded that trunk muscles are co-activated in asymmetric tasks. When the trunk is flexed laterally, EMG activity increases in the posterior back muscles on both sides of the spine (Andersson, Ortengren & Herberts, 1977). Bilateral co-contraction of lumbar back muscles at heel strike has been observed both in walking (Battaye & Joseph, 1966; Waters & Morris, 1970; Thorstensson, Carlson, Zomlefer & Nilsson, 1982) and running (Thorstensson *et al.*, 1982). Basmajian (1978) and Morris with associates (Morris, Benner & Lucas, 1962) reported antagonistic activity of the deep trunk muscles during axial rotation. Pope and his co-workers (Pope, Andersson, Broman, Svensson & Zetterberg, 1986) found antagonistic activity in both abdominal and posterior back muscles during twisting in the standing position. The degree of co-activation symmetry between the left and right sides may vary depending on the conditions under which rotation is performed. Unresisted trunk rotations show a smaller degree of co-contraction symmetry (Kumar, Narayan & Zedka, 1996), while more symmetrical co-contractions are

seen in isometric tasks or in tasks where trunk rotation is combined with trunk flexion (Kumar, Zedka & Narayan, submitted). In the asymmetrical postural tasks studied in Chapter 3, the abdominal muscles were usually activated together but co-activation was not a usual pattern for the right and left ES. Contractions of the paraspinal muscles on one side coincided with relaxations on the contralateral side. This clear difference between the back and the abdominal muscles may reflect a functional difference. While abdominal co-contraction presumably stiffens the trunk, reciprocal activation of the back muscles allows a more direction-specific response to perturbation. The asymmetric activation of back muscles may be necessary to keep the head upright while the pelvis is being tilted. This strategy might provide stable gaze, although blindfolded subjects responded in the same way. Another finding which has not been described before was the phasic character of the reciprocal activation of the right and left ES evoked at the higher tilt velocity. No matter whether the muscle activation was reflex or central in origin, this observation suggested that reciprocal inhibition between human back muscles might be organized in a way similar to that in limb muscles.

Phasic activity in the erector spinae was studied in Chapter 4, where the EMG bursts were elicited by hand movements. The frequency of activation achievable in this manner was never achieved by a direct voluntary effort to contract the erector spinae muscle. After a systematic consideration of various descending neural influences in different experiments we adopted the view that the EMG bursts in the ES were due to a local segmental proprioceptive reflex which presumably contributed to trunk stabilization. To our knowledge, this is the first time that a functional role has been proposed for the proprioceptive reflexes in the back muscles. This paper is a challenge to the prevailing opinion that postural activity during voluntary movements is centrally generated in a feed-forward manner (Belenkii, Gurfinkel & Paltsev, 1967; Bouisset & Zattara, 1981; Gurfinkel, Lipshits & Lestienne, 1988). While descending signals certainly influence back muscle activity, their role may be mostly modulatory, at least for some types of movements.

Although increased stretch reflex gain has been often associated with muscle hypertonus in musculoskeletal pain (Johansson & Sojka, 1991), the role of proprioceptive reflexes in back pain has never been studied. The study presented in Chapter 5 seems to

be the first to investigate this issue. It came as a surprise that the stretch reflex response to a mechanical indentation of the ES was not changed by induced deep muscle pain, while its long-latency component was increased during cutaneous pain. The results suggest that increased tone in paraspinal muscles – a common finding in back pain – may not be associated with increased excitability in the γ-loop. It has to be kept in mind, however, that the mechanism of saline-induced pain could be different from that of naturally occurring back pain.

In Chapter 6 back muscle pain induced a phase-specific alteration of trunk voluntary motor patterns. ES EMG activity was increased in phases where the muscles acted as antagonists and decreased where they acted as agonists, which is in agreement with the "pain adaptation" model of Lund and his colleagues (Lund, Donga, Widmer & Stohler, 1991). Unilateral back muscle pain produced bilateral EMG changes leading to a reduction in velocity and range of the trunk movement. thus possibly providing a physiological splint to the painful low-back structure (muscle). On the other hand, it is possible that the less modulated continuous EMG activity contributes to perpetuation of the muscle pain. Whether a similar muscle pattern would be observed as a secondary reaction to pain (e.g. after injection of a spinal ligament) remains to be determined. Contrary to expectations, no compensatory activity in synergists of the injected muscle was seen. Our results from the last two papers indicate that deep pain has surprisingly little effect on segmental stretch reflexes but changes descending motor commands. The persistence of EMG changes limited to only one segment of the ES after pain had dissipated suggests that changes in the motor output may have an involuntary component. It should be further investigated to what extent the observed changes reflect different voluntary commands and to what extent the descending commands are modulated by a pain reflex.

7. 2 Future directions

Back pain remains mysterious despite its ubiquitous and disturbing character. Our inability to accurately evaluate morphological and functional changes makes

etiological diagnosis impossible, which in turn often results in unsuccessful treatment. As long as low back pain remains a complaint in search of a clear diagnosis, progress in prevention and medical management will be limited. The situation can be improved by (a) introducing already available, more objective evaluation tools into clinical practice and (b) focusing on back pain research.

7. 2. 1. Clinical evaluation

If we disregard limitations of imaging and biochemical assessment techniques, considerable progress has to be made in the physical evaluation of patients. Currently, there is a lack of consensus on appropriate assessment and treatment methods (Field & Lohr, 1992; Deyo, Cherkin, Conrad & Violinn, 1991; Keller, Soule, Wennberg & Hanley, 1990; Harwood *et al.*, 1997). In the clinical setting, functional deficits in muscuoskeletal disorders are still often assessed subjectively. The usually tested variables are muscle strength and joint range of motion. Such measures may point to individual muscle deficiencies in the limbs but they are rather crude for testing individual muscles and joints in the trunk. Ideally, more objective methods, such as force gauges, EMG and motion analyzing systems, should be used in a battery of standardized tests comprising voluntary and reflex movements of the trunk and limbs. Clinicians who do not have access to sophisticated equipment should at least start routinely using measuring tapes and inclinometers to monitor spinal flexibility and sets of weights to quantify trunk strength. Also standardized measurements of trunk muscle endurance may be of value because back pain has been connected to increased fatigability of trunk muscles (Roy, De Luca & Casavant, 1989).

Discrepancies related to non-standardized measuring techniques, different back pathologies, poor quantification of pain, and unreliable controls may be the reason why some studies have found back muscle strength unaffected in LBP patients (Roy, De Luca & Casavant, 1989; Jorgensen & Nicolaisen, 1987), whereas most studies have reported deficits in trunk strength (Addison & Schultz, 1980; Smith, Mayer, Gatchel & Becker, 1985; Mayer, Smith, Keeley & Mooney, 1985; McNeill, Warwick, Andersson & Schultz,

1980). Obviously, all measurements of voluntary activities reflect psychophysical performance (motivation) and not true physiological abilities.

7. 2. 2. Research

In order to explain and efficiently fight back pain, more intensive research is needed. Scientific studies concerned with precise morphology and function of the low back region did not start until two decades ago. Until then, theories about back pain pathology were based on assumptions that often turned out to be inaccurate. For example, Bogduk (1980) revised the gross anatomy of the back muscles and thereby supplied those modelling back pain with unexpected new data. Even the most accurate and complete knowledge of muscle orientation in static positions does not allow one to infer their orientation during movement. Adequate imaging techniques are necessary to study the dynamic anatomy of the spine. Another example of the further need for macroanatomical studies is the controversial existence of meniscoid formations, whose entrapment allegedly causes intra-articular blockage of facet joints which can be relieved by manipulation (Kos & Wolf, 1972). Even less morphological information is available from histological studies, for instance in relation to the controversy about sensory innervation of vertebral structures (Maigne, 1996). Although some recent histological data have been presented by Giles & Singer (1997), there are still conflicting reports regarding the cross-section, length and type of back muscle fibres (Sirca & Kostevc, 1985; Mannion, Dumas, Cooper, Espinosa, Faris & Stevenson, 1997). Muscle spindles in the lumbar ES also deserve more attention. Although their presence has been confirmed, they have not been closely studied.

From a functional point of view, it is essential to obtain information on spinal neuronal circuitry involved in trunk muscle activity. Knowing the mechanism through which pain interferes with the relay of somatosensory signals to motoneurones would perhaps explain many motor deficits observed in back pain. Currently, all speculations on the functioning of the thoracic spinal circuitry would have to be based on information extrapolated from limb muscles. But do phenomena, such as I a or I b inhibition exist also between the trunk muscles? What is the reflex connection between muscles,

ligaments and skin? Answers to these questions could help in elucidating the formation and spread of paraspinal muscle spasms, pain concentration in trigger points and its referral into remote areas, as well as cutaneous pain and induration, which are frequent phenomena accompanying back pain. Regarding motor output, the mechanism of force generation in trunk muscles and its alteration during fatigue and injury needs investigation. An evaluation of the interaction between voluntary and reflex mechanisms in pain is important for the rehabilitation process. For instance, the common opinion that "weak muscles" have to be strengthened by voluntary exercises has to be substantiated. The relationship between fatigue, muscle fibre type and pain and its manifestation in EMG may also shed more light on back pain. Furthermore, it should be determined how frequently back muscles perform eccentric contractions since these have been found more painful and damaging to the muscle tissue than isometric or concentric contractions. Imunohistochemical examinations have revealed structural disruption of the cytoskeleton within muscle fibres (Lieber, Thornell & Friden, 1996). Some studies indicate that eccentric exercise is more damaging to fast fibre types (Lieber & Friden, 1988). Is there any connection between their higher fatigability and pain? What is the effect on sensorimotor function of proteolytical enzymes released by inflammatory cells invading traumatized muscle fibres? Another question out of many concerns the role of algogenic substances produced both peripherally and centrally by the neural tissue. How do they contribute to the development of chronic pain?

Before we find satisfying answers, both the prevention and treatment of back pain will not be based on a solid ground and treatment will remain in its current ad hoc and empirical state. We do not know how we are interfering when using heat, cold, electricity, acupuncture, massage, pressure, manipulation, stretching, etc. A relatively high success rate in treating acute cases of low back pain can not be ascribed to our knowledge of the problem. Most people with acute back pain recover spontaneously within a few weeks and require minimal medical care. Such results should be contrasted with the low success rate in treating those 10% of patients, whose condition becomes chronic. Their lives remain miserable despite the fact that they account for almost 90% of society's total expenditure on back pain (Cats-Baril & Roth, 1985).

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APPENDIX

An open-loop control of experimental muscle pain

(Developed in collaboration with Michel Gauthier, Debby Gillard and Arthur Prochazka)

The infusion pump used in experiments in Chapters 5 and 6 was controlled digitally by a computer which regulated the infusion rate in a feed-forward manner in order to keep the pain approximately constant. The main program was written in Delphi (Borland International Inc.) which linked to the compensating filter running in real time with the use of Matlab Simulink software (The Mathworks Inc.) and controlled the infusion pump via the computer's serial port.

Originally, our intention was to use the closed-loop system described by Zhang, Ashton-Miller and Stohler (1993). The software of this system was kindly supplied to us by Dr. J. Lund. However, due to difficulties implementing their software on our equipment we opted for a simpler, open-loop control. Preliminary tests showed that in terms of the variability of the subjects' pain response our results with simple open-loop control were comparable to those of the above mentioned paper. The characteristics of the open-loop system are described below.

In order to achieve a desired pain response which closely resembles a step response, the unknown response dynamics of the subject must first be identified. The input-output relationship between the pain response and a known infusion rate input was obtained in preliminary tests on two subjects. A 5% NaCl solution was infused at six different constant rates (50,60,100,140,150, 200 μl/min) in each case for 12 minutes. The subjects verbally rated their pain every 15 seconds on a 0 - 10 scale, where 0 represented "no pain" and 10 was "unbearable pain". A block diagram of the setup is shown in Figure A.1.A.

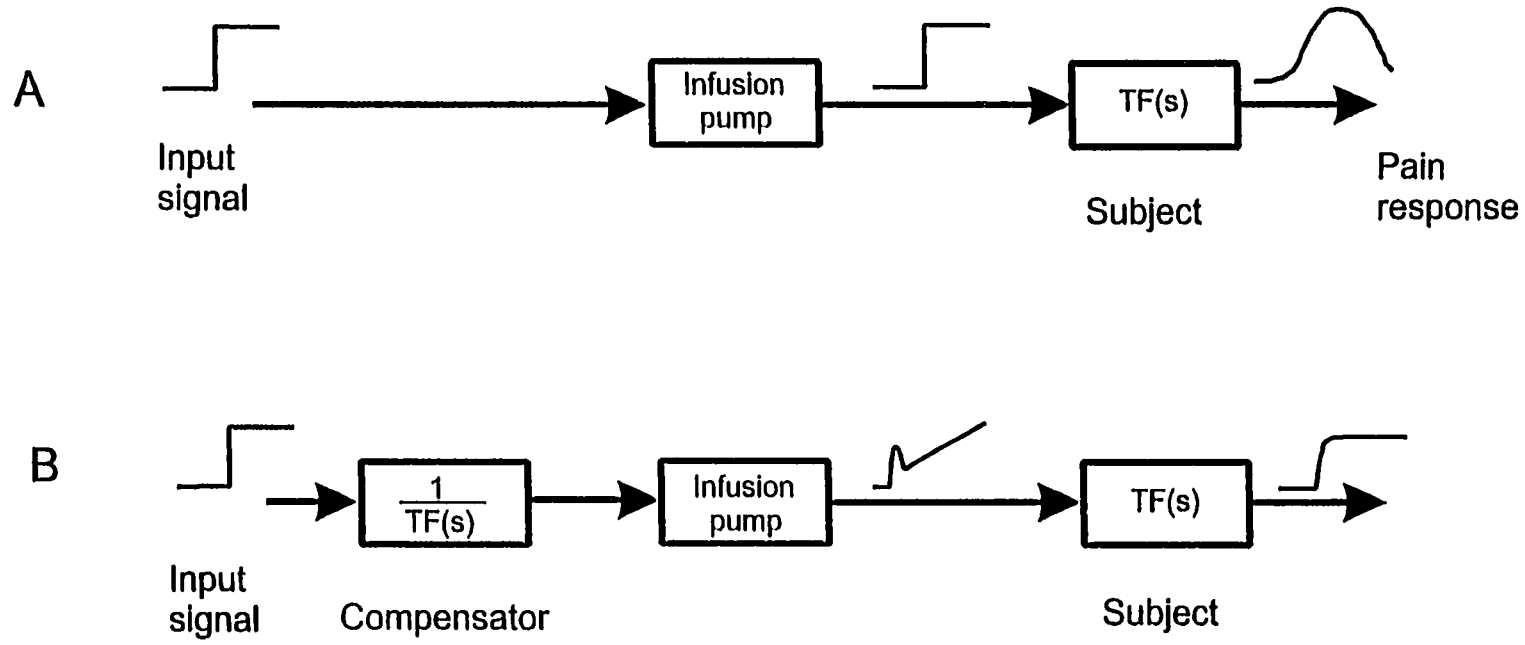


Figure A1. Block diagram of the pain control system. (A) Identifying the subject's pain response to a step rate of infusion. (B) Compensated controller: input signal and pain response are nearly identical.

The input signal, a step function, was fed to the digital controller of the infusion pump resulting in a step change in infusion rate. Eight trials were averaged to obtain the mean step response which was then fitted by eye with the following transfer function:

$$TF(s) = \frac{9.72\times10^{-8} e^{-45s} (s+.0001)}{(s+.009)^2 (s^2+.018s+.0001)}$$

The exponential term e^{-45s} corresponds to a delay and the other s-terms correspond to differential and integral components. The mean and modelled step responses are shown in Figure A.2.

Once the subject's response dynamics were known, a compensator was designed so that a given input signal would be propagated through the system with the net result that the desired pain response would closely correspond to the input signal. The inverse of the subject's transfer function was used as the compensation element "cancelling out" the effects of the subject's delayed and sluggish response so that the output signal (i.e. perceived pain) closely followed the input signal (Figure A.1.B.). The infusion rate controller was therefore modelled as the inverse transfer function of the mean pain rating step response (neglecting the delay term) as given by:

$$\frac{1}{TF(s)} = \frac{.00387 (s+.009)^2 (s^2+.018s+.0001)}{(s+.0001)(s+.03)^4}$$

The denominator term $(s + .03)^4$ was purposefully added, partly to avoid overshoot and ringing in the response and partly because the Matlab software requires that the denominator be of a higher order than the numerator. In reality, due to approximations in the identification of subject's response dynamics, the input and output

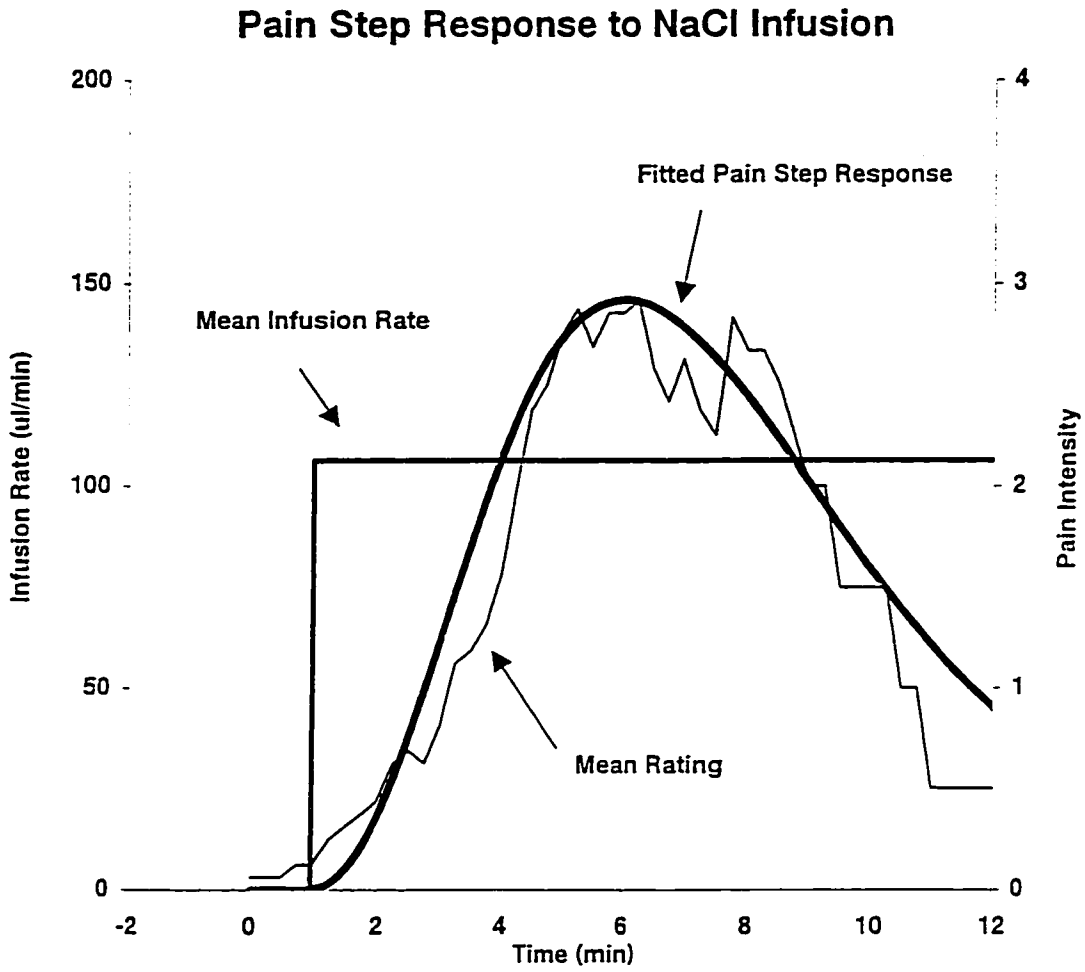


Figure A2. Measured and modelled pain responses to step inputs of infusion rate. The mean pain rating profile (thin line) in response to a step change in mean infusion rate and the fitted response of the model (thick line). Experimental data are from 2 subjects, 8 trials.

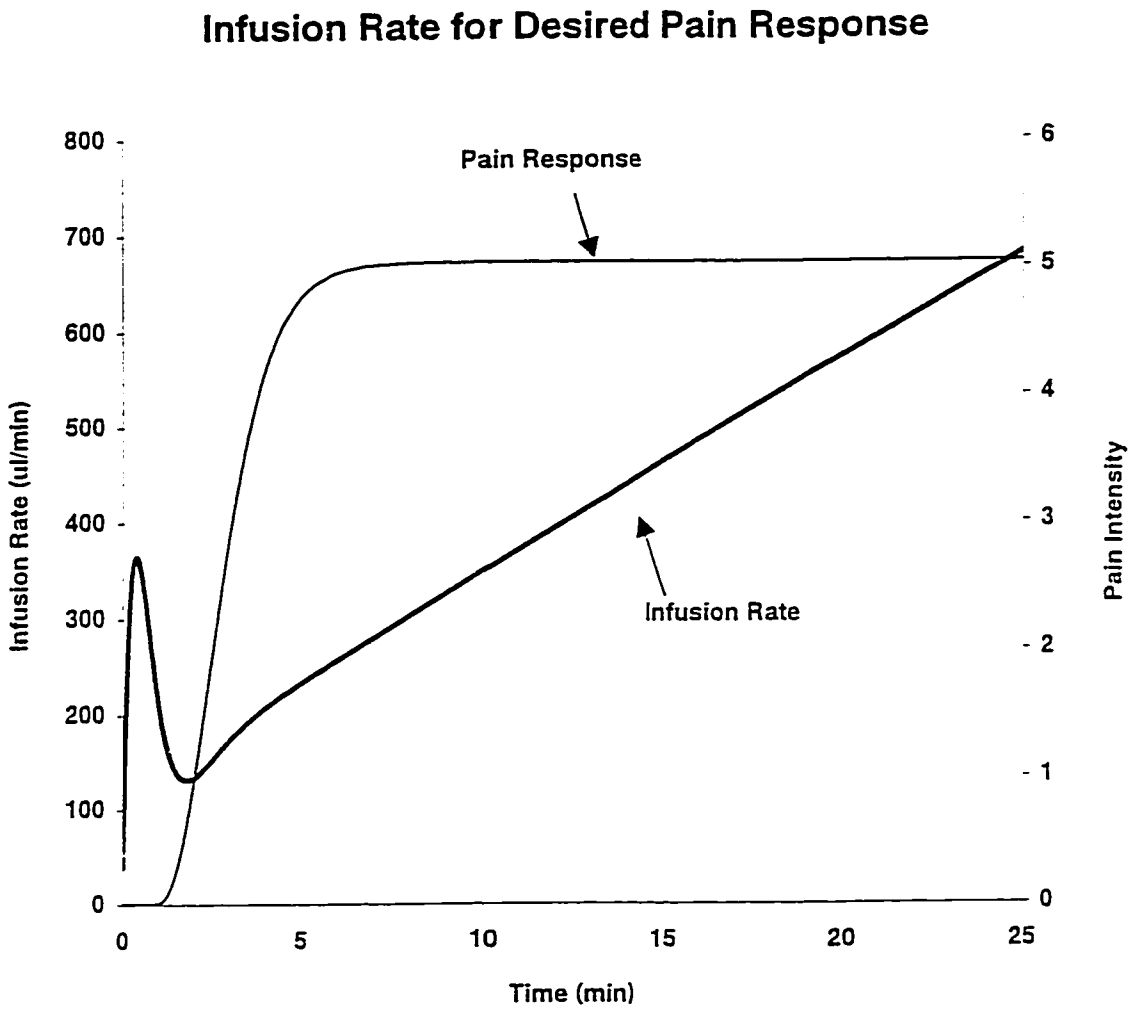


Figure A3. The profile of infusion rate (thick line) computed by the compensator to achieve a step change in pain rating of 5 on a subjective rating scale extending between 0 (no pain) and 10 (unbearable pain). The predicted pain response is a damped step function (thin line).

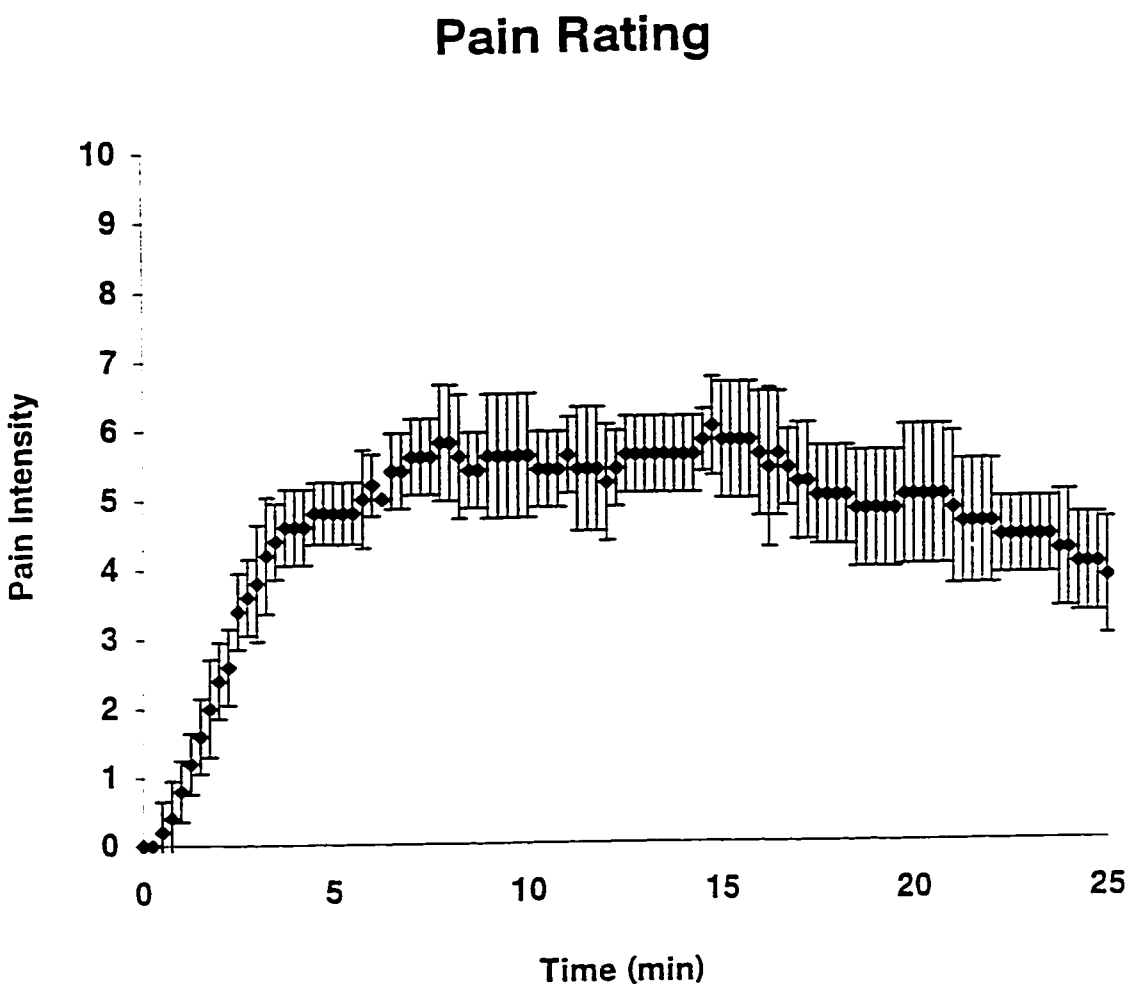


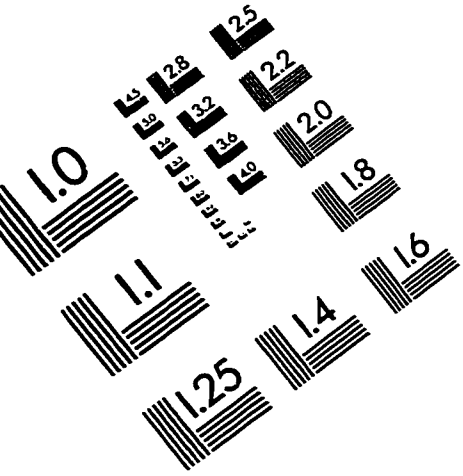
Figure A4. Pain intensity rating of 5 subjects (mean and standard deviation bars) during 25 minutes of hypertonic saline infusion using the open-loop compensated controller and a step input.

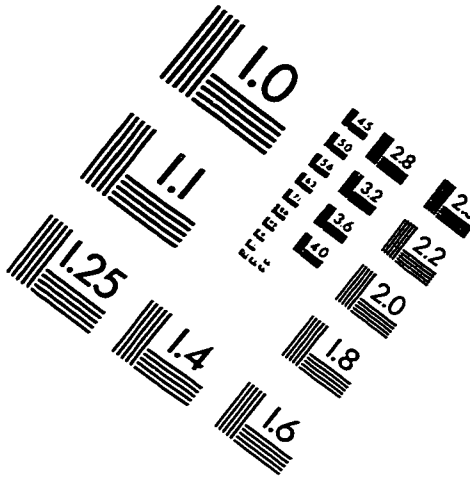
signal are not exactly the same, but do closely resemble each other. Plots of the predicted response to a step input (pain intensity level of 5) and the required control action (flow rate) to achieve this response are shown in Figure A.3. Please compare the predicted response to the actual mean response obtained across all subjects in the experiment (Figure A.4.).

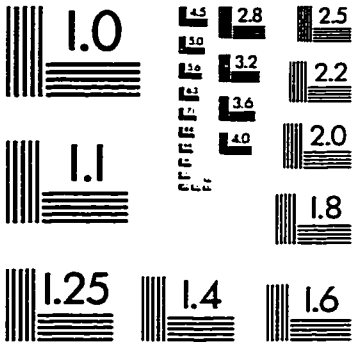
References

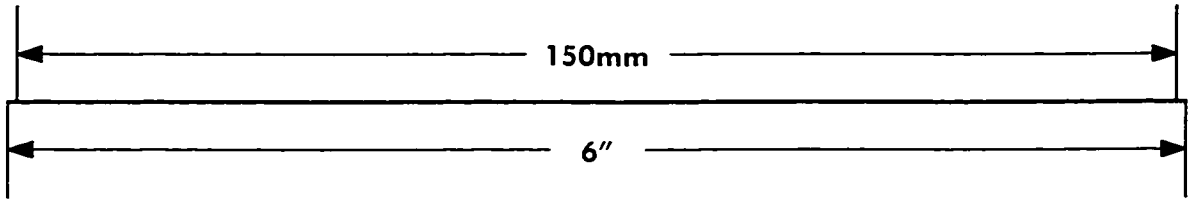
ZHANG, X., ASHTON-MILLER, J. A. & STOHLER, C. S. (1993). A closed loop system for maintaining constant experimental muscle pain in man. *IEEE Transactions on Biomedical Engineering* **40**, 344-352.

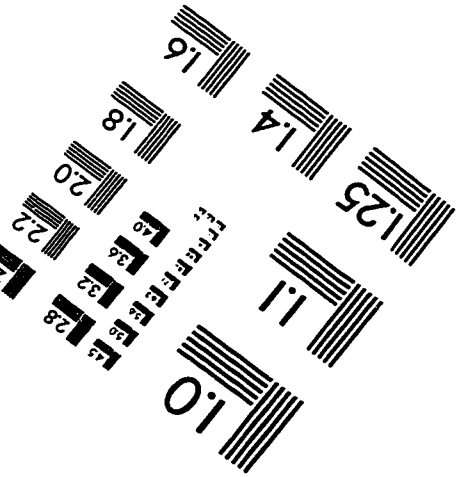
IMAGE EVALUATION
TEST TARGET (QA-3)

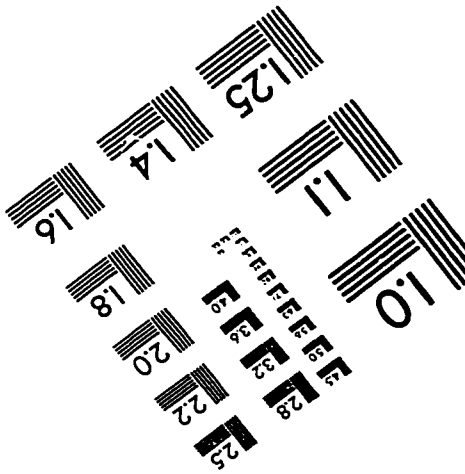












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