Lars I. E. Oddsson and Carlo J. De Luca

J Appl Physiol 94:1410-1420, 2003. First published Dec 13, 2002; doi:10.1152/japplphysiol.01183.2001

You might find this additional information useful...

This article cites 66 articles, 11 of which you can access free at: http://jap.physiology.org/cgi/content/full/94/4/1410#BIBL

This article has been cited by 1 other HighWire hosted article:

Lumbar and cervical erector spinae fatigue elicit compensatory postural responses to assist in maintaining head stability during walking
J. J. Kavanagh, S. Morrison and R. S. Barrett
J Appl Physiol, October 1, 2006; 101 (4): 1118-1126.

[Abstract] [Full Text] [PDF]

Updated information and services including high-resolution figures, can be found at: http://jap.physiology.org/cgi/content/full/94/4/1410

Additional material and information about *Journal of Applied Physiology* can be found at: http://www.the-aps.org/publications/jappl

This information is current as of June 7, 2007.

Activation imbalances in lumbar spine muscles in the presence of chronic low back pain

Lars I. E. Oddsson¹ and Carlo J. De Luca^{1,2}

¹NeuroMuscular Research Center and ²Department of Biomedical Engineering, Boston University, Boston, Massachusetts 02215

Submitted 30 November 2001; accepted in final form 7 December 2002

Oddsson, Lars I. E., and Carlo J. De Luca. Activation imbalances in lumbar spine muscles in the presence of chronic low back pain. J Appl Physiol 94: 1410-1420, 2003. First published December 13, 2002; 10.1152/japplphysiol. 01183.2001.—Paraspinal electromyographic (EMG) activity was recorded bilaterally from three lumbar levels during 30-s isometric trunk extensions [40 and 80% of maximum voluntary contraction (MVC)] in 20 healthy men and 14 chronic low back pain patients in pain. EMG parameters indicating neuromuscular fatigue and contralateral imbalances in EMG root-mean-square amplitude and median frequency were analyzed. Patients in pain showed less fatigue than controls at both contraction levels and produced only 55% of their MVC. Patients in pain likely did not produce a "true" maximum effort. A low MVC estimate would mean lower absolute contraction levels and less neuromuscular fatigue, thus explaining lower scores in the patients. Contralateral rootmean-square amplitude imbalances were present in both categories of subjects although such imbalances, when averaged across lumbar levels, were significantly larger in patients. Median frequency imbalances were significantly larger in the patients, at segmental as well as across lumbar levels. These results suggest that the presence of pain in these patients caused a redistribution of the activation behavior between synergistic muscles of the lumbar back.

chronic low back pain; maximum voluntary contraction; median frequency; muscular imbalance; surface EMG parameters

RECENT STUDIES HAVE ESTIMATED that over 14% of the US population suffers from pain related to joints and the musculoskeletal system (40). Muscular injuries are a common cause of disability in the population, and they are the most common cause of low back pain (LBP) (24, 28). It has been estimated that the $\sim 5-10\%$ of LBP cases that become chronically disabled account for \sim 90% of the costs (29, 45, 70). It is still not known why certain subjects develop a chronic disability, although there are some data to suggest that specific behavioral factors related to fear avoidance may be of importance (34). Improved objective techniques for early diagnosis, treatment, and rehabilitation of patients with LBP may help reduce costs and prevent the development of a chronic disability. The development of such techniques requires a detailed understanding of mechanisms controlling muscle activation in the presence of

Address for reprint requests and other correspondence: L. I. E. Oddsson, 19 Deerfield St., Boston, MA 02215 (E-mail: loddsson@bu.edu).

LBP as well as objective ways of measuring and quantifying muscular function in the presence of LBP.

Surface electromyography is a noninvasive technique for assessing muscle function that has played a major role in our basic understanding of the function of trunk muscles in both normal subjects and LBP patients during specific postures and movements (15, 16, 18, 49–52, 58, 59, 61, 62). For example, the amplitude of the electromyographic (EMG) signal has often been used to assess whether the level of muscle activity is abnormal in patients with pain (1, 9, 30, 38, 39, 54, 64, 77), but the interpretation of the various results have conflicted. Some studies have identified uni- and/or bilateral deviations in muscular activity in back muscles of patients with LBP compared with control subjects (2, 3, 21, 23, 37, 68, 73), whereas others have failed to identify differences in EMG activity in paraspinal muscles of patients with LBP (47, 57, 76). Limitations in these studies, such as poorly described patient populations and electrode locations, as well as inadequately defined tasks performed by the subjects, have also been pointed out (18, 57, 72).

In addition to EMG signal amplitude, the median frequency (MF) or half-power point of the EMG spectrum is a parameter commonly extracted from the EMG signal. Stulen, De Luca, and co-workers (35, 65, 71) were the first to propose the use of this parameter as an indicator of neuromuscular fatigue during constant-force contractions. As a contraction is sustained, there is a compression of the power density spectrum of the EMG signal toward lower frequencies. Because the accompanying decrease in MF is nearly linear during a fatiguing contraction (16), the rate of decrease of the MF offers a convenient means of monitoring this compression over time. Techniques for measuring the MF are restricted to stationary EMG signals, implying that it only works correctly on isometric constant-force contractions. Recently, however, time-frequency analysis techniques have been implemented on the EMG signal, providing a tool to monitor neuromuscular fatigue also under dynamic conditions by using the instantaneous MF (8, 22, 60). A decrease in MF has been associated with muscle metabolic correlates to fatigue, most notably the accumulation of H⁺ ions at the sarcolemma

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

as lactic acid is produced and disassociated (12, 16, 32, 33). In addition, the rate of decline in MF appears to correlate with the subjectively perceived exertion during a constant-force effort (19). Differences in low back muscle fatigability, indicated as a decrease in the MF of the EMG signal of the lumbar back muscles, have been demonstrated between groups of back pain patients and healthy control subjects (13, 31, 61, 63, 64, 66) as well as in patients with myalgia of the trapezius muscle (48). Furthermore, high fatigability of paraspinal muscles has been shown to be associated with the presence as well as the risk of developing LBP (41). For the fatigue during a sustained effort to appear over a reasonably short time, tests monitoring MF are usually conducted at relatively high levels of muscle contraction. Previous work has indicated that levels of 50-60% of the maximal voluntary contraction (MVC) must be maintained for a decrease in the MF to occur during a 30-s contraction (53). Thus the test procedure is commonly based on an initial assessment of a MVC. LBP patients in pain at the time of testing do not comply well with such a protocol because of obvious hesitation and fear of reinjury (75), making the assessment of a reliable MVC difficult (53). Although the MF parameter is commonly used to monitor neuromuscular fatigue, recent in vitro studies have demonstrated the existence of a close relationship between muscle fiber type, size, and the MF of the EMG signal, suggesting that relative changes in MF could be used to indicate activation properties of the underlying muscle (35, 65, 71). Other reports showing that the MF of the EMG signal is closely related to the conduction velocity, the shape of the action potential, and the temperature in the muscle (15, 44) lend further support to these findings.

In the present study, we measured trunk extension strength (MVC) and surface EMG activity of contralateral lumbar spine muscles during a sustained isometric contraction in a cohort of healthy subjects and a group of LBP patients, who reported pain during a test of their back muscle function. We investigated the behavior of several EMG variables at two different force levels and their use as objective indicators of back muscle function. The EMG-based variables included the rate of decrease of the MF of the EMG signal (considered an index of neuromuscular fatigue) and a series of ratios between contralateral pairs of symmetrical measurements of the MF and the root mean square (RMS) amplitude of the two EMG signals (53), considered indicators of muscular imbalances during the sustained isometric effort.

By using these indexes, we addressed specific questions related to muscular strength, neuromuscular fatigue, and muscular imbalances in the two groups of subjects. We hypothesized that the presence of pain in the patient group would be associated with a redistribution of the activation pattern of the lumbar spine muscles apparent as an increased degree of asymmetries indicated by the imbalance ratios extracted from the surface EMG signal compared with healthy subjects (18).

METHODS

The Back Analysis System

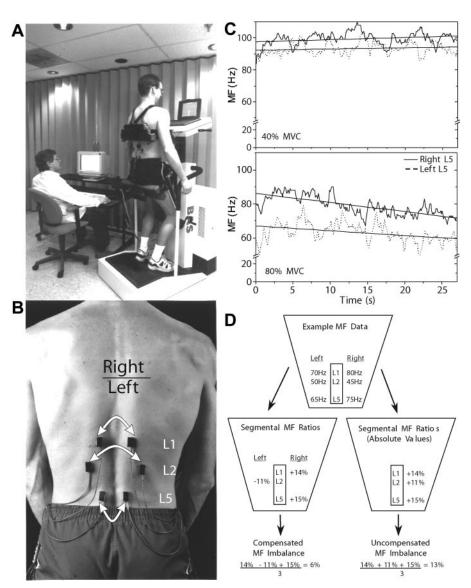
In this study, we used a device referred to as the back analysis system (BAS) to assess lumbar back muscle function (Fig. 1, A and B). Details of the system have been published previously (16, 25, 61, 64). In brief, the system contains four functional elements: I) active differential surface EMG electrodes; 2) a computer-assisted device called the muscle fatigue monitor (MFM), which uses hardware to process the EMG signal in real time; 3) a postural restraint apparatus to constrain the subjects in a defined posture; and 4) a software package to collect and analyze data. The postural restrain apparatus provided means for eliciting isometric contractions from the back muscles. The isometric recording condition is essential for the EMG frequency spectrum analyses that were performed in the study.

The subject was strapped into the postural restraint apparatus to ensure a stable position of the pelvis and the lower limbs during the test. The torque generated by the subject during an isometric trunk extension was measured with two load cells attached at both ends of a nylon harness placed around the upper part of the chest (Fig. 1A). The subject was instructed to maintain the trunk in a symmetrical upright position and to extend the trunk against the harness to raise a cross hair appearing on a video monitor to a target level that was preset to a desired torque level. The cross hair also moved laterally on the screen in proportion to the difference between the forces recorded on each load cell, allowing any asymmetrical efforts to be detected. Lateral targets on the monitor marked a 5% difference between the right and the left load cell. In addition, any visible trunk rotations or lateral bendings were discouraged by the experimenter. The subjects were told to push symmetrically and keep the cross hair at the indicated target level to the best of their ability for the duration of the contraction. Six active surface EMG electrodes were placed bilaterally over sites at L1, L2, and L5 levels of the lower back, corresponding to the anatomical locations of longissimus thoraces, iliocostales lumborum, and multifidus muscle sites (Fig. 1B). The differential electrodes (Delsys 2.1 series) had common mode rejection ratios of >90 dB and a gain of 10 with a 3-dB bandwidth of 20-450 Hz. The EMG signals were further amplified 100-700 times and then processed in real time by customized analog hardware in the MFM controlled through a custom-written software package in a personal computer. The MFM hardware included a specially designed filter that tracked the frequency at which the power of the EMG signal was split in half, thus obtaining the MF, as well as additional electronic circuitry that provided the RMS amplitude of the EMG signal. The MF and RMS for each of the six EMG signals were provided as two separate analog outputs from the MFM system. All 12 analog signals, RMS and MF from each of the six EMG electrodes, were sampled at 10 Hz and stored on hard disk for further off-line processing (cf. Refs. 35, 65, 71). Examples of MF data are shown in Fig. 1C.

Subjects and Protocol

The study was approved by the internal review board (IRB) committees of the local office of the Department of Veterans Affairs (VA) Research and Development and the Charles River Campus IRB at Boston University. Back pain patients were recruited through the Boston-area VA medical system. Written consent was obtained from all subjects. Fourteen male back pain patients were compared with a group of 20 healthy men (Table 1). The patients were in-

Fig. 1. A: back analysis system (BAS) used in this study to monitor surface electromyography (EMG) signals of synergistic muscles of the lower back. B: location of the surface EMG electrodes on the L₁ (longissimus thoraces), L2 (iliocostales), and L5 (multifidus) lumbar levels, respectively. Arrows indicate the contralateral ratios, right divided by left, that were calculated between segmental pairs of EMG data. For further details, see METHODS. C: example of typical raw median frequency (MF) data (270 samples) of the right and left L₅ sites from 1 lower back pain (LBP) patient performing a 40% maximal voluntary contraction (MVC; top traces) and 80% MVC (bottom traces) contraction. The slope of the linear regression line, shown in the graph for each data set, was used as an indicator of neuromuscular fatigue. This individual showed no signs of neuromuscular fatigue at the 40% MVC level (flat slopes). All regressions were highly significant (P < 0.01). Also, note that this individual displayed a positive segmental MF imbalance at the L_5 level (right MF > left MF). D: illustration of how the global imbalance parameters were derived from the original data. The same procedure was used for both the MF and root mean squares (RMS) data.



cluded in the study only if they subjectively reported pain in the lumbar region during the test. Two subjective pain parameters were assessed: a pain drawing to indicate the site of pain and a 0–100 visual analog scale to assess the pain level experienced by the patient. Patients with diagnosed spinal stenosis and other verifiable structural spinal abnormalities

Table 1. Characteristics of LBP patients and control subjects

Category	LBP Patients with Pain	Controls
Number	14	20
Age, yr	39.6 ± 10.1	27.1 ± 6.3
Height, m	1.78 ± 0.07	1.77 ± 0.06
Weight, kg	86.1 ± 12.7	78.4 ± 7.0
Duration of injury,		
mo	$9.3 \pm 8.3(1 - 31)$	N/A
Visual analog scale	$26 \pm 17 (10 - 70)$	N/A
MVC, N	$542 \pm 218 (263 {-} 891)$	$982 \pm 315 (463 {-} 1{,}552)$

Values are means \pm SD; ranges are given in parentheses. LBP, lower back pain; MVC, maximal voluntary contraction; N/A, not applicable.

such as herniated disc, prior back surgery, spondylolisthesis, and cancer were excluded. Thus the subjects matched categories one and two as defined by the Quebec Task Force on Spinal Disorders (67a). In addition, patients were excluded if they had cardiovascular, respiratory, orthopedic, neurological, endocrine, or renal conditions that were contraindications to a sustained isometric resistance exercise. After a period of low-level warm-up contractions, subjects were asked to perform a MVC. The greatest of three attempts was used to indicate trunk extension strength. Subjects were tested at two different contraction levels: 40 and 80% of MVC. The contractions were sustained for 30 s.

Data Analysis

Fatigue parameters. A linear regression analysis was performed on MF data for all muscle sites between 3 and 30 s of the contraction (27 s of data at 10 Hz, i.e., 270 samples for each data set). The initial 3 s were excluded to allow the subjects to stabilize the contraction level and to let the hardware in the MFM to settle the MF of the EMG signal. The slope of the linear regression line was measured in Hz/s for each of the six electrode sites (cf. Table 3, e.g., R-Slope-L₁;

R indicates right and L left sides, respectively). A negative slope indicates the presence of neuromuscular fatigue during the contraction, whereas a flat or positive slope indicates that no neuromuscular fatigue was present during the 30 s of contraction. Examples are shown in Fig. 1.

Ratios and imbalance parameters. The six MF and RMS signals were used to calculate three MF ratios and three RMS ratios; one MF and one RMS ratio for each pair of EMG electrodes at the three lumbar levels (L_1 , L_2 , and L_5 , respectively, cf. Fig. 1, C and D). The parameters were calculated separately for each lumbar level from the sample-by-sample ratio (right-side value divided by left-side value) of the two signals of interest between 3 and 30 s of the contraction (providing 270 ratios from 27 s of data sampled at 10 Hz). Each of the ratio values was then transformed according to the following procedure to provide a time series of 270 corrected ratios (R) with symmetrical properties centered around 0

$$R = egin{cases} ext{ratio} - 1, & ext{ratio} \geq 1 \ - \Big(rac{1}{ ext{ratio}} - 1 \Big), & ext{ratio} < 1 \end{cases}$$

An average of all the transformed ratios between 3 and $30 \mathrm{s} (270 \mathrm{\,epochs})$ of the contraction was used to represent the segmental imbalance behavior between the two EMG signals. The derived ratio was multiplied by 100 to represent percent difference between the right and the left sides. For example, a value of 20 would mean that the right side was 20% larger than the left side, whereas a value of -20 would mean that the left side was 20% larger than the right side. These ratios provided a symmetrical relative comparison between right- and left-sided differences in the underlying EMG parameters.

Two global EMG parameters were then calculated from the local segmental ratio parameters. The procedure is shown in the following equations and further illustrated and exemplified in Fig. 1. For each subject, we defined the "uncompensated" imbalance as the mean across the three lumbar levels of the absolute value of the segmental ratios and the "compensated" imbalance as the mean of the segmental ratios across all lumbar levels. These parameters were calculated for both the MF and RMS imbalances.

$$Uncompensated\ imbalance = \frac{|ratio_{L1}| + |ratio_{L2}| + |ratio_{L5}|}{3}$$

$$Compensated\ imbalance = \frac{ratio_{L1} + ratio_{L2} + ratio_{L5}}{3}$$

The uncompensated imbalance parameter provides a measure of the total muscular imbalances regardless of direction (right or left), whereas the compensated imbalance parameter takes into consideration the direction of the local segmental imbalances, with a positive value indicating that right > left and a negative value indicating that left > right. Consequently, the compensated imbalances represent the residual imbalance after a possible cancellation between the different lumbar levels within each subject (see Fig. 1). To avoid the effect of between-subject cancellation of compensated imbalances of opposite signs, we compared the absolute value of the compensated imbalances between different conditions.

Specific Research Questions and Related Statistical Analysis

The following dependent variables were used in the analysis: MVC as an indicator of muscular strength; individual

mean MF slope based on the six muscle sites to indicate neuromuscular fatigue rate; absolute values of segmental RMS ratios (L_1 , L_2 , and L_5); uncompensated RMS imbalance; absolute values of segmental MF ratios (L_1 , L_2 , and L_5); uncompensated MF imbalance; absolute values of individual compensated RMS and MF imbalances. The independent variables were subject group (LBP and healthy) and contraction level (40 and 80% MVC), respectively. Standard deviation (SD) was used throughout the analysis as the measure of spread in the data.

Recent studies have demonstrated good reliability intraclass correlation (0.65-0.90) of MF-slope and other MF parameters extracted from surface EMG signals of different back extensor muscles during an isometric endurance test both in healthy subjects (20,36,46) and in LBP patients (36). Intraclass correlation exceeding 0.90 have been reported for such parameters extracted from time-frequency analysis of the EMG signal during a fatiguing lifting task (22). Before statistical analysis, a Shapiro-Wilk W test for normality was performed on all dependent variables to justify the use of parametric statistical methods. A significant W statistic would indicate a nonnormal distribution. Significance level was set to P < 0.05.

The following six specific research questions were addressed in this study: 1) Are MVC levels in LBP patients with pain when tested in the BAS similar to those of healthy control subjects? One t-test for independent samples was conducted to address this question (P < 0.05). 2) Is the overall fatigue rate (estimated from MF slope) in LBP patients with pain and in healthy control subjects similar at comparable force levels? Three t-tests were conducted to compare MF slopes between the two groups at 40% MVC, at 80% MVC, and between 40% MVC in the control group and 80% MVC in the LBP group. To maintain an overall significance level of 0.05 when conducting multiple t-tests, the P level was adjusted down by using a Bonferroni correction procedure for correlated dependent measures. 3) Are contralateral activation imbalances of the lumbar back muscles, measured through the RMS amplitude of the EMG signal during a symmetrical effort in the BAS, similar at different force levels in healthy control subjects, and how do they compare to LBP patients with pain? 4) Are contralateral activation imbalances of the lumbar back muscles, measured through the MF of the EMG signal during a symmetrical effort in the BAS, similar at different force levels in healthy control subjects, and how do they compare to LBP patients with pain? 5) Is the residual contralateral RMS imbalance (compensated RMS imbalance) similar at different force levels in healthy subjects, and how does it compare to LBP patients with pain? 6) Is the residual contralateral MF imbalance (compensated MF imbalance) similar at different force levels in healthy subjects, and how does it compare to LBP patients with pain?

Questions 3–6 were addressed with a one-way analysis of variance followed by contrast analysis (planned comparisons) and Bonferroni-corrected P levels for correlated dependent measures to maintain an overall P < 0.05. Pearson's correlation coefficient was used to estimate linear relationships between dependent measures. Statistical analysis was performed with Statistica 5.0 (StatSoft, Tulsa, OK).

RESULTS

All dependent variables were found to be normally distributed according to Shapiro-Wilk W test. This justified the use of parametric statistical analysis methods.

Table 2. Location of pain site, pain rating, and duration of injury for the fourteen LBP patients

Patient No.	Pain Site	VAS	Duration, mo
1*	left L ₅	75	14
2	$left S_1$	15	3
3*	$left L_5$	10	6
4^*	$left L_5$	10	2
5*	$left L_5$	20	8
6^*	$left L_5$	35	20
7	$left S_1$	40	1
8*	$left L_5$	30	8
9	$\operatorname{mid} \operatorname{L}_5$	70	31
10*	$_{\rm right~\dot{L}_{1-2}}$	40	1
11*	$_{ m right}$ $_{ m L_5}$	65	6
12	$_{ m right}$ $_{ m L_5}$	85	12
13	$_{ m right}$ $_{ m L_5}$	20	3
14	$\operatorname{right} \operatorname{S}_1$	40	8

VAS, visual analog score, 0–100. *Subjects with a pain site that directly coincided with the placement of an electromyographic (EMG) electrode (cf. Fig. 6).

Strength and Subjective Pain Parameters

The characteristics of the two groups of subjects are shown in Table 1. The LBP subjects produced 55% of the MVC of the control subjects. The difference between the two groups was highly significant (P <0.001). The left-right force symmetry was maintained within the 5% target provided on the screen in front of the subjects for all test contractions. The patients had been injured for an average of 9.3 mo, ranging from 1 to 31 mo. Two patients had been injured for <2 mo (cf. Table 2). Thus most of the patients could be classified as being in a chronic state of their injury. The mean visual analog scale pain score was 26, with a range between 10 and 70. The individual pain scores as well as the site of pain indicated by the subjects on a pain drawing are shown in Table 2. Eight of the subjects indicated pain on their left side, one in the mid region, and five on the right side of the lumbar paraspinal muscles. Six of the subjects with left-sided pain and two with right-sided pain (denoted with an asterisk in Table 2) indicated that their pain site directly coincided with the location of an EMG electrode (L_1 , L_2 , or L_5).

MF Slopes Indicating Neuromuscular Fatigue

Data from the 40% MVC contraction level of three LBP patients were discarded. The combination of low contraction level, small muscle mass, and amount of adipose tissue between the muscle tissue and the electrode yielded low-level EMG recordings (typically <10 μV RMS). Consequently, the signal-to-noise ratio was typically 4-5. Thus an accurate assessment of the EMG signal was not possible in these individuals. The parameter indicating degree of neuromuscular fatigue at the different muscle sites, the MF slope, is presented in Table 3. Three statistical comparisons were made, two based on a relative force level (mean MF slope at 40 and 80% MVC between the two groups, respectively; A-B and C-D in Table 3) and one based on a comparable absolute force level [40% MVC (393 N) in the control group vs. 80% MVC (433 N) in the LBP group, respectively; B-C in Table 3]. A Bonferroni-corrected P level of <0.025 was used to maintain an overall significance level of 0.05 (three t-tests and a mean correlation of 0.36 between dependent measures). At the 40% MVC level, the patients mostly displayed small positive slope values, indicating no buildup of neuromuscular fatigue. At this contraction level, the healthy control subjects displayed negative slopes at all muscle sites except for the left L₂ level, where the slope value was zero (Table 3). As shown in Table 3, the mean slopes at the 40% MVC as well as the 80% MVC contraction levels were significantly more negative in the control group compared with the LBP group, indicating higher levels of neuromuscular fatigue in the control group when compared at relative force levels (A-B, P = 0.015 and C-D, P = 0.005 in Table 3). Mean MF slopes were not significantly different at comparable absolute force levels (B-C in Table 3, P=0.258), indicating similar levels of neuromuscular fatigue at these contraction levels.

Table 3. Means across all subjects in each category of the slope of the linear regression for the median frequency

Param	A LBP 40%	B Control 40%	C LBP 80%	D Control 80%
R-Slope-L ₁	0.04 ± 0.24	-0.17 ± 0.37	-0.19 ± 0.18	-0.62 ± 0.42
R -Slope- L_2	0.14 ± 0.42	-0.05 ± 0.22	-0.17 ± 0.38	-0.25 ± 0.17
R-Slope-L ₅	-0.03 ± 0.19	-0.30 ± 0.29	-0.31 ± 0.36	-0.73 ± 0.58
L-Slope-L ₁	0.03 ± 0.26	-0.14 ± 0.29	-0.33 ± 0.41	-0.57 ± 0.42
L-Slope-L ₂	0.26 ± 0.26	0.00 ± 0.28	-0.20 ± 0.25	-0.26 ± 0.29
L-Slope-L ₅	0.02 ± 0.31	-0.28 ± 0.33	-0.29 ± 0.35	-0.70 ± 0.53
Mean slope	0.08 ± 0.16	-0.15 ± 0.26	-0.26 ± 0.25	-0.53 ± 0.32
A–B	0.23,	P = 0.015		
В-С	<i>'</i>	0.11, P	= 0.258	
C-D		,		= 0.005

Values are means \pm SD (in Hz/s) for the 6 different electrode sites and the different test conditions. Mean slopes \pm SD across all 6 muscle sites within each test condition are also shown. The last 3 rows show results from contrast analysis between the 2 groups after an analysis of variance to address relevant research questions. Note that the mean slopes for the LBP subjects at 40% MVC were positive, indicating an absence of neuromuscular fatigue during the contraction. See text for details. R, right; L, left.

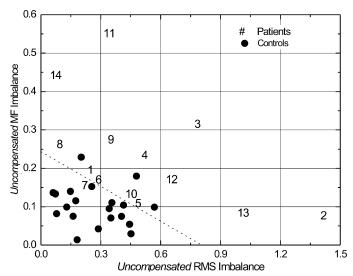


Fig. 2. Mean uncompensated RMS imbalance plotted vs. uncompensated MF imbalance for all control subjects (●) and LBP patients (subjects 1–14, cf. Table 2). The uncompensated imbalance parameter is calculated as the mean of the absolute values of the 3 segmental imbalance ratios, which in turn represent the mean of the 270 samples of RMS and MF data from the 2 contralateral sites of interest (cf. Methods). The dotted line was drawn arbitrarily to demonstrate that a separation between the 2 groups could be achieved on the basis of these 2 variables, reflecting the total presence of EMG-based imbalances in the lumbar paraspinal muscles.

Contralateral Surface EMG Imbalance Parameters

The analysis for the LBP patients was focused on the 80% MVC contraction level because of low signal-tonoise ratios at the lower contraction level. The individual mean uncompensated MF and RMS imbalances for the two groups are plotted against each other in Fig. 2. These parameters indicate the total presence of contralateral MF and RMS imbalances in the surface EMG signals across the L_1 , L_2 , and L_5 lumbar levels. This figure serves to qualitatively illustrate the less variable behavior in the control group compared with the LBP patients with respect to these parameters. Solid circles denote control subjects and LBP patients are represented by *numbers* 1-14 according to Table 2. An arbitrary line was drawn to indicate that, with the exception of one LBP subject (subject 7) and three control subjects, the data separate into two groups. In general, the group of LBP subjects displayed higher levels of uncompensated MF and/or RMS imbalances (cf. Fig. 2). This is further illustrated in Figs. 3 and 4.

Figure 3, A-C, shows the mean +1 SD across all subjects of the absolute values of segmental RMS ratios for each of the segmental levels L_1 , L_2 , and L_5 , respectively. The mean uncompensated RMS imbalance is shown in Fig. 3D. Two statistical contrasts were performed for each of the four parameters, one comparing the control group 40% MVC to the 80% MVC contractions and one to compare the LBP group to the control group. The two segmental RMS ratios at the L_5 level for the control group were almost identical and were not tested (cf. Fig. 3). Thus a total of seven comparisons were performed to address research ques-

tion 3. The mean correlation between the RMS variables was 0.59, resulting in a Bonferroni-corrected significance level of P < 0.023 to maintain an overall level of 0.05. In the control group, the segmental RMS ratios for the 40 and 80% MVC contractions were not statistically different from each other at any of the lumbar levels. This was true also for the mean uncompensated RMS imbalance (Fig. 3D). The LBP group had significantly higher segmental RMS ratio than the control group at the L_5 level (P < 0.005). At the L_1 and L_2 levels as well as for the mean uncompensated RMS imbalance, the LBP group and control group were not statistically different from each other (Fig. 3).

In a similar fashion, Fig. 4, A-C, shows the mean +1SD across all subjects of the absolute values of segmental MF ratios for each of the segmental levels L₁, L₂, and L₅, respectively. The corresponding mean uncompensated MF imbalance is shown in Fig. 4D. The same statistical analysis as described above was performed for the MF parameters. The 40 and 80% MVC values for the control group at the L₂ and L₅ (segmental MF ratios) as well as the mean uncompensated MF imbalance were almost identical and were therefore not included in the contrast analysis (cf. Fig. 4). The segmental MF ratios at the L₁ level were included in the contrast analysis. Thus, in total, five statistical comparisons were performed for these MF parameters. The mean correlation between the dependent measures was 0.44, resulting in a Bonferroni-corrected P <0.021. The segmental MF ratio at 40 and 80% MVC contraction levels for the control subjects were not statistically different from each other. The LBP group displayed significantly higher segmental MF ratios at all lumbar levels (P < 0.0003, P < 0.017, P < 0.0001 for L₁, L₂, and L₅ level, respectively) as well as a higher uncompensated MF imbalance (P < 0.0001).

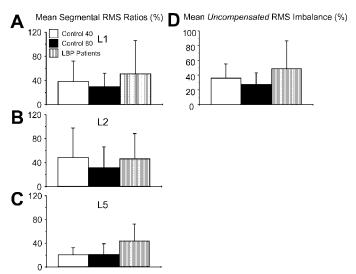


Fig. 3. Mean +1 SD of the absolute value of the segmental RMS ratios (A-C) displayed for each of the lumbar segment levels $(L_1, L_2,$ and L_5 , respectively) and mean +1 SD uncompensated RMS imbalance (D) for all LBP patients $(80\% \ \text{MVC})$ and the control subjects $(40 \ \text{and} \ 80\% \ \text{MVC})$. The uncompensated imbalance indicates the total presence of muscular imbalances as defined here regardless of its direction (right or left).

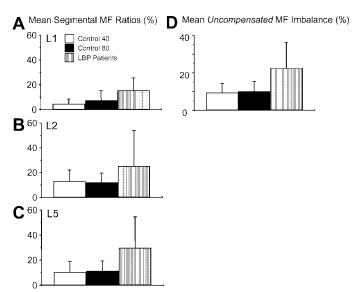


Fig. 4. Mean +1 SD of the absolute value of the segmental MF ratios (A-C) displayed for each of the lumbar segment levels $(L_1, L_2, and L_5, respectively)$ and mean +1 SD uncompensated MF imbalance (D) for all LBP patients $(80\% \ MVC)$ and control subjects $(40 \ and \ 80\% \ MVC)$. The uncompensated imbalance indicates the total presence of muscular MF imbalances as defined here regardless of its direction (right or left).

Figure 5 shows mean values for the absolute values of the compensated RMS (A) and MF imbalances (B). These parameters indicate overall residual imbalances after a within-subject cancellation of segmental imbalances has been taken into account. In general, the segmental imbalances canceled out across lumbar levels to a larger extent in the control subjects compared with the LBP group. Two statistical contrasts were performed to address *question* 5 related to the compensated RMS imbalances, one between the 40 and 80% MVC contraction levels of the control group and one between the LBP and control group. The mean correlation between the dependent variables was 0.4, resulting in a Bonferroni-corrected P < 0.033 for performing two tests. One contrast was performed for the compensated MF imbalance related to question 6 because the 40 and 80% MVC values for the control group were almost identical. Thus a P level of 0.05 was used. The absolute values of the compensated RMS imbalance at the 40 and 80% MVC contraction levels for the control subjects were not statistically different from each other. The LBP group displayed significantly higher absolute compensated RMS as well as MF values (P <0.031 and P < 0.031, respectively).

Figure 6 shows contralateral RMS and MF imbalances plotted vs. each other for patients who had reported a pain site that coincided with the location of at least one of the EMG electrodes. Each patient is identified by his number shown in Table 2. For comparison, the solid circles represent the contralateral MF and RMS imbalances at the L_5 level for the control subjects. The gray area represents an ellipse that was drawn to include all healthy subjects. Note that all LBP subjects with the exception of one (subject 5) fall outside of the

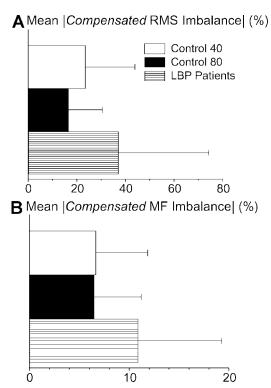


Fig. 5. Mean +1 SD of the absolute values of the compensated RMS and MF imbalances for all control subjects and LBP patients. The compensated imbalances take into consideration the direction of the local segmental imbalances and may thus reflect a within-subject cancellation across the different lumbar levels. The absolute values of the compensated imbalances were compared to avoid the effect of between-subject cancellation of values of opposite signs.

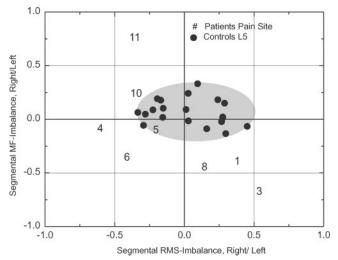


Fig. 6. Segmental RMS imbalance plotted vs. MF imbalance for the 8 LBP subjects who had a pain site that coincided with 1 of the EMG electrode locations (cf. Table 2 for subject numbers). For reference, the corresponding imbalances from the L_5 lumbar level are displayed for the 20 control subjects. The gray area represents an ellipse that was drawn to include all healthy subjects. Note that all LBP patients who had negative MF imbalances had pain on the left side (subjects 1, 3, 4, 5, 6, and 8, cf. Table 2), whereas patients with positive MF imbalances had pain on the right side (subjects 10 and 11).

gray area that incorporates healthy pain-free behavior. Patients who reported pain on the left side (*subjects 1*, 3, 4, 5, 6, and 8, cf. Table 2) displayed negative MF imbalances, whereas patients with pain on the right side (10, 11) displayed positive MF imbalances. Thus, in this group of patients, the injured side consistently displayed higher MF frequencies than the noninjured side. No such relationship was seen for the RMS imbalances.

DISCUSSION

The results of this study support the hypothesis presented by De Luca and co-workers (32, 41, 61, 63). They demonstrate that the subjective presence of the sensation of pain during LBP is associated with altered amplitude and spectral properties of the surface EMG signal from muscles underlying the location of the pain site. These alterations were mainly manifested as skewed contralateral MF ratios and to a lesser degree the RMS ratios of the surface EMG signal. These results will be discussed separately below with respect to the interpretation of extracted imbalances in RMS and MF parameters, respectively.

Strength and Fatigue Parameters

The participating LBP patients were in pain at the time their back muscle function was tested. Therefore, our finding that the patients produced only about half of the force of the control subjects during the assessment of their MVC was not surprising. However, previous work has shown that the back muscle strength of LBP patients in remission at the time of testing is similar to that of healthy individuals (61). Our interpretation of this discrepancy is that muscle force production in our patient group was inhibited because of the presence of pain during the assessment of the MVC and the full force-producing capacity of the muscles was not revealed. In addition, the effort and/or ability of these individuals to produce muscular force was likely limited because of apparent fear of additional pain and/or reinjury. An underestimation of the MVC in the patient group is consistent with the results from our objectively measured EMG parameters. For example, our results demonstrated lower levels of neuromuscular fatigue, i.e., less negative MF slope, in the LBP patients than in the control subjects, at both the 40 and 80% MVC levels. In fact, at the 40% MVC contraction level, the LBP patients displayed no apparent neuromuscular fatigue, i.e., no decrease in MF during the contraction, whereas the control subjects did. These findings are consistent with a low MVC estimate in the patient group. The alternate explanation, namely that the patients produced their "true" MVC and actually were less fatigable than their healthy cohorts is less likely and not supported by previous studies (32, 41, 61, 63). Consequently, when pain is present the performance of the subject is likely to be limited, suggesting that tests of back muscle fatigability based on the assessment of a true maximum effort of the back muscles are unreliable for this population of patients and should be avoided. Our results confirm, as previously proposed, that quantitative and objective indexes of muscle function can still be obtained in the presence of pain by using surface EMG-based imbalance parameters at submaximal force levels (53). As our results in the present study have demonstrated, these parameters have the attractive property, at least in healthy subjects, to remain constant over a large force range (40–80% MVC).

Surface EMG-based Imbalance Parameters

We have introduced the concept of uncompensated and compensated EMG-based imbalance parameters to indicate aspects of how contralateral muscles in the lumbar back are activated during a sustained isometric contraction in a symmetrical task. These parameters combine segmental ratios of either the RMS amplitude or the MF of the EMG signal from two contralateral muscle sites. The values of the segmental ratios reflect relative contralateral load sharing and relative differences in recruitment behavior between contralateral muscle sites, respectively. The physiological rationale for such an interpretation of these parameters is based on the fact that the RMS amplitude of the EMG signal is closely related to the force developed by the muscle (15, 27, 53) and that the MF reflects (in large part) the muscle fiber conduction velocity and therefore contains information about muscle fiber type and size (35). Uncompensated imbalances indicate the global presence of uneven activation behavior across all contralateral muscle sites, whereas the compensated EMG imbalances reflect the residual of uneven activation behavior after segmental directionality (right-left) of these imbalances is taken into consideration. Thus, on an individual level, a positive compensated imbalance indicates that right-sided values are greater than left-sided ones, and vice versa for a negative imbalance. If the uncompensated imbalance is equal to the compensated imbalance, the segmental imbalances, at L_1 , L_2 , and/or L₅, are all in the same direction. When the compensated imbalance is smaller than the uncompensated imbalance, then some cancellation has taken place between different segmental levels, i.e., at least one level is negative and/or one is positive.

RMS Imbalance Parameters

We found similar levels of uncompensated RMS imbalances, in LBP patients and healthy control subjects (Fig. 3) suggesting that the presence of such imbalances during a symmetrical isometric task is normal in a healthy back, at least within the ranges we observed. Interestingly, on a segmental level, our group of patients displayed significantly larger RMS ratios at L_5 , the lumbar level that 10 of the 14 LBP patients reported as their pain site (Table 2). The absolute value of compensated RMS imbalances were significantly higher in the patients compared with the control subjects indicating smaller residual segmental activation imbalances in the healthy group (Fig. 5). Thus the presence of a high compensated RMS imbalance, i.e.,

large residual segmental activation imbalances, may have been an indication of a nonhealthy back muscle function in our patient group. Interestingly, the external forces measured in the BAS device in the patient group were symmetrical during the test despite an asymmetrical activation of the lumbar back muscles as indicated by the high compensated RMS imbalances (Fig. 5). This could occur if these individuals used other muscles, not recorded in the present study, to perform the task. For example, latissimus dorsi, quadratus lumborum, or thoracic parts of the erector spinae would be able to contribute to the torque measured in the BAS system. Alternatively, the contralateral force-RMS relationship may be distorted as a result of chronic injury in these patients such that less force is produced for a certain level of RMS output. The RMSforce relationship is influenced by factors such as temperature, fatigue, subcutaneous tissue, and the geometrical relationship between active muscle fibers and electrode site/configuration as well as motor endplate location with respect to the recording electrodes (7). However, by calculating the ratio between symmetrical muscle sites, these error sources will decrease substantially because they are likely to be similar for the two sites included in the estimation of the parameter. Thus it appears likely that the contralateral imbalances seen in the patient group are related to their injury.

MF Imbalance Parameters

We found significantly larger segmental MF ratios at all three lumbar levels in the LBP group compared with the healthy one (Fig. 4). In addition, uncompensated as well as compensated MF imbalances were significantly larger in our LBP group (Figs. 4 and 5). The presence of some level of uncompensated MF imbalances in both groups (Fig. 4D), however, suggests that there were segmental contralateral differences in the underlying active population of muscle fibers, with respect to type and/or size, in both the LBP patients and the healthy subjects. This interpretation of the MF imbalance parameters is supported by recent in vitro studies conducted on rat muscles showing that the MF reflects information regarding the size as well as the type of fibers that are activated (35). These investigators found that, with all else being equal, larger muscle fiber size as well as faster fiber type was associated with higher conduction velocities and thus higher MF. In addition, the action potential shape, conduction velocity, and temperature in the muscle will influence the MF of the EMG power spectrum (15, 44, 71). However, these factors could be assumed to be similar between the contralateral muscle sites.

An earlier similar observation on lateral differences in the activation of back muscles was made by Merletti et al. (43), who noted differences in the fatigue response of electrically stimulated contralateral muscles in the backs of healthy individuals. They further surmised that the difference was due to fiber-type modifications following the life-long preferential usage due to hand dominance. In the present study, contralateral

lumbar muscles were monitored across multiple levels. Interestingly, our results show that when healthy back muscles are activated synergistically during a symmetrical isometric task, local contralateral imbalances, likely reflecting muscle fiber type and/or size differences, tend to be smaller across multiple segments in healthy subjects compared with the LBP group. Taken together, the interpretation of these previous works suggests that in our study the chronic LBP patients used their back muscles differently than the healthy individuals during a sustained isometric trunk extension effort. One interpretation consistent with these findings is that bilateral differences in muscle fiber size and/or type may be present in chronic LBP patients.

Central and Peripheral Pain Mechanisms

It is possible to further view the results of the present study in light of known physiological mechanisms related to pain. Most of the patients in the present study were in a chronic state of injury. During chronic pain, there are known effects of central as well as peripheral factors that have a bearing on our findings of altered EMG parameters in the LBP patients. For example, central sensitization after extended nociceptor activity from sites of tissue injury is associated with hyperalgesia and increased and persistent sensation of pain even from non-injury-causing stimuli (42, 56, 69). This has been termed a "disease state" of the nervous system (4, 5) during which the primary afferent neurotransmitter, substance P, contributes to a reorganization of spinal cord circuitry that leads to persistent and exacerbated pain (5, 56, 74). This may be of direct relevance to the results of the present study because substances released and modulated during the central sensitization process, including substance P, can modify motoneuron excitability through pre- and postsynaptic actions, which in turn may alter recruitment properties of the motoneuron pool and thus cause imbalances of the kind we have noted in the present study (55). Other work using a model for induced experimental muscle pain in the masseter muscle of the cat found that the proprioceptive signals from the muscle spindles were centrally modulated in the presence of pain (14), a mechanism that could directly cause changes in recruitment behavior that would be detectable in the EMG signal. Yet another mechanism that may be speculated to play a role in the activation balance of back muscles in chronic pain patients relates to the function of nervi nervorum. It has been proposed (10, 11) and recently demonstrated by Sauer et al. (67) that nervi nervorum have a nociceptive function and that they participate in neural inflammation. Furthermore, nervi nervorum appear to have a role in chronic pain related to adhesion effects between different tissue layers due to growth of scar tissue after an injury (26, 78).

In conclusion, our results demonstrate that the presence of pain in our LBP patients, subjectively experienced by the subject, was associated with physiological effects that were objectively measured with surface

EMG techniques. We found that subacute and chronic LBP patients in pain at the time of testing display specific muscle activation imbalances during a symmetrical trunk extension effort that appear to reflect physiological impairments related to their injury. In addition, we have found that, during this symmetrical isometric effort, paraspinal back muscles in healthy subjects may have the ability to globally offset local segmental activation imbalances to a greater extent than those in LBP patients. The present study suggests that surface EMG may be a useful tool to detect these imbalances.

The skilful assistance by Erik-Jan Giphart in the data analysis process is highly appreciated.

This study was supported by funds from the Department of Veterans Affairs Research and Development Administration (award nos. B2029-RA and B872–2RA).

REFERENCES

- Ahern DK, Follick MJ, Council JR, Laser-Wolston N, and Litchman H. Comparison of lumbar paravertebral EMG patterns in chronic low back pain patients and non-patient controls. Pain 34: 153–160, 1988.
- Andersson GBJ, Bogduk N, De Luca CJ, Goldenberg D, Mayer T, Roy S, and Smidt G. Muscle: clinical perspectives. In: New Perspectives on Low Back Pain: Workshop, Airlie, Virginia, May 1988, edited by Frymoyer JW and Gordon SL. Park Ridge, IL: American Academy of Orthopaedic Surgeons, 1989, p. 293-334.
- Andersson GBJ, Ortengren R, and Herberts P. Quantitative electromyographic studies of back muscle activity related to posture and loading. Orthop Clin North Am 8: 85-96, 1977.
- Basbaum AI. Distinct neurochemical features of acute and persistent pain. Proc Natl Acad Sci USA 96: 7739-7743, 1999.
- Basbaum AI. Spinal mechanisms of acute and persistent pain. Reg Anesth Pain Med 24: 59-67, 1999.
- Basmajian JV and De Luca CJ. Muscles Alive. Baltimore, MD: Williams and Wilkins, 1985.
- 8. **Bonato P, Boissy P, Croce UD, and Roy SH.** Changes in the surface EMG signal and the biomechanics of motion during a repetitive lifting task. *IEEE Trans Neural Syst Rehabil Eng* 10: 38–47, 2002
- Boucher JP, King MA, Lefebvre R, and Pepin A. Quadriceps femoris muscle activity in patellofemoral pain syndrome. Am J Sports Med 20: 527–532, 1992.
- Bove GM and Light AR. Calcitonin gene-related peptide and peripherin immunoreactivity in nerve sheaths. Somatosens Mot Res 12: 49-57, 1995.
- Bove GM and Light AR. Unmyelinated nociceptors of rat paraspinal tissues. J Neurophysiol 73: 1752-1762, 1995.
- Brody LR, Pollock MT, Roy SH, De Luca CJ, and Celli B. pH-induced effects on median frequency and conduction velocity of the myoelectric signal. J Appl Physiol 71: 1878–1885, 1991.
- Buijs RJC. Automated spectral EMG evaluation of erector spinae musculature (Master thesis). Boston, MA: NeuroMuscular Research Center, Boston Univ., 1993.
- Capra NF and Ro JY. Experimental muscle pain produces central modulation of proprioceptive signals arising from jaw muscle spindles. *Pain* 86: 151-162, 2000.
- De Luca CJ. Physiology and mathematics of myoelectric signals. IEEE Trans Biomed Eng 26: 313–325, 1979.
- De Luca CJ. Myoelectrical manifestations of localized muscular fatigue in humans. Crit Rev Biomed Eng 11: 251–279, 1984.
- 18. **De Luca CJ.** Use of the surface EMG signal for performance evaluation of back muscles. *Muscle Nerve* 16: 210–216, 1993.
- Dedering A, Nemeth G, and Harms-Ringdahl K. Correlation between electromyographic spectral changes and subjective assessment of lumbar muscle fatigue in subjects without pain from the lower back. Clin Biomech (Bristol, Avon) 14: 103–111, 1999.

- Dedering A, Roos af Hjelmsäter M, Elfving B, Harms-Ringdahl K, and Nemeth G. Between-days reliability of subjective and objective assessments of back extensor muscle fatigue in subjects without lower-back pain. J Electromyogr Kinesiol 10: 151–158, 2000.
- Dolce JJ and Raczynski JM. Neuromuscular activity and electromyography in painful backs: psychological and biomechanical models in assessment and treatment. Psychol Bull 97: 502–520, 1985.
- Ebenbichler GR, Bonato P, Roy SH, Lehr S, Posch M, Kollmitzer J, and Della Croce U. Reliability of EMG timefrequency measures of fatigue during repetitive lifting. Med Sci Sports Exerc 34: 1316–1323. 2002.
- 23. Floyd WF and Silver PHS. The function of the erectores spinae muscles in certain movements and postures in man. *J Physiol* 129: 184–203, 1955.
- 24. Garret W, Bradley W, Byrd S, Edgerton VR, and Gollnick P. Muscle: basic science perspectives. In: New Perspectives on Low Back Pain: Workshop, Airlie, Virginia, May 1988, edited by Frymoyer JW and Gordon SL. Park Ridge, IL: Am. Acad. of Orthopedic Surgeons, 1989, p. 335–372.
- 25. **Gilmore LD and De Luca CJ.** Muscle fatigue monitor (MFM): second generation. *IEEE Trans Biomed Eng* 32: 75–78, 1985.
- 26. Hall TM and Elvey RL. Nerve trunk pain: physical diagnosis and treatment. *Man Ther* 4: 63–73, 1999.
- 27. Herzog W, Sokolosky J, Zhang YT, and Guimaraes AC. EMG-force relation in dynamically contracting cat plantaris muscle. J Electromyogr Kinesiol 8: 147–155, 1998.
- 28. Holbrook TL, Grazier K, Kelsey JL, and Stauffer RN. The Frequency, Occurrence, Impact and Cost Of Selected Musculo-skeletal Conditions in the United States. Park Ridge, IL: American Academy of Orthopedic Surgeons, 1984.
- Indahl A, Velund L, and Reikeraas O. Good prognosis for low back pain when left untampered. A randomized clinical trial [see comments]. Spine 20: 473–477, 1995.
- Jensen C, Nilsen K, Hansen K, and Westgaard RH. Trapezius muscle load as a risk indicator for occupational shoulderneck complaints. Int Arch Occup Environ Health 64: 415–423, 1993.
- Jorgensen K. Human trunk extensor muscles physiology and ergonomics. Acta Physiol Scand Suppl 637: 1–58, 1997.
- 32. **Jorgensen K and Nicolaisen T.** Trunk extensor endurance: determination and relation to low-back trouble. *Ergonomics* 30: 259–267, 1987.
- 33. **Juel C.** Muscle action potential propagation velocity changes during activity. *Muscle Nerve* 11: 714–719, 1988.
- 34. Klenerman L, Slade PD, Stanley IM, Pennie B, Reilly JP, Atchison LE, Troup JD, and Rose MJ. The prediction of chronicity in patients with an acute attack of low back pain in a general practice setting. Spine 20: 478–484, 1995.
- 35. **Kupa EJ, Roy SH, Kandarian SC, and De Luca CJ.** Effects of muscle fiber type and size on EMG median frequency and conduction velocity. *J Appl Physiol* 79: 23–32, 1995.
- 36. Lariviere C, Arsenault AB, Gravel D, Gagnon D, and Loisel P. Evaluation of measurement strategies to increase the reliability of EMG indices to assess back muscle fatigue and recovery. *J Electromyogr Kinesiol* 12: 91–102, 2002.
- 37. **Lariviere C, Gagnon D, and Loisel P.** The comparison of trunk muscles EMG activation between subjects with and without chronic low back pain during flexion-extension and lateral bending tasks. *J Electromyogr Kinesiol* 10: 79–91, 2000.
- 38. Lund JP, Donga R, Widmer CG, and Stohler CS. The painadaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. *Can J Physiol Pharmacol* 69: 683–694, 1991.
- 39. **MacIntyre DL and Robertson DG.** Quadriceps muscle activity in women runners with and without patellofemoral pain syndrome. *Arch Phys Med Rehabil* 73: 10–14, 1992.
- 40. Magni G, Caldieron C, Rigatti-Luchini S, and Merskey H. Chronic musculoskeletal pain and depressive symptoms in the general population. An analysis of the 1st National Health and Nutrition Examination Survey data. *Pain* 43: 299–307, 1990.

- Mannion AF, Connolly B, Wood K, and Dolan P. The use of surface EMG power spectral analysis in the evaluation of back muscle function. J Rehabil Res Dev 34: 427–439, 1997.
- 42. **Mense S.** Nociception from skeletal muscle in relation to clinical muscle pain. *Pain* 54: 241–289, 1993.
- Merletti R, De Luca CJ, and Sathyan D. Electrically evoked myoelectric signals in back muscles: effect of side dominance. J Appl Physiol 77: 2104–2114, 1994.
- 44. **Merletti R, Sabbahi MA, and De Luca CJ.** Median frequency of the myoelectric signal. Effects of muscle ischemia and cooling. *Eur J Appl Physiol* 52: 258–265, 1984.
- Nachemson AL. Newest knowledge of low back pain. Clin Orthop 279: 8–20, 1992.
- 46. **Ng JK and Richardson CA.** Reliability of electromyographic power spectral analysis of back muscle endurance in healthy subjects. *Arch Phys Med Rehabil* 77: 259–264, 1996.
- Nouwen A, Van Akkerveeken PF, and Versloot JM. Patterns of muscular activity during movement in patients with chronic low-back pain. Spine 12: 777-782, 1987.
- Öberg T, Sandsjo L, Kadefors R, and Larsson SE. Electromyographic changes in work-related myalgia of the trapezius muscle. Eur J Appl Physiol 65: 251–257, 1992.
- Oddsson L. Co-ordination of a simple voluntary multi-joint movement with postural demands: trunk extension in standing man. Acta Physiol Scand 134: 109-118, 1988.
- Oddsson L and Thorstensson A. Fast voluntary trunk flexion movements in standing: primary movements and associated postural adjustments. Acta Physiol Scand 128: 341–349, 1986.
- Oddsson L and Thorstensson A. Fast voluntary trunk flexion movements in standing: motor patterns. *Acta Physiol Scand* 129: 93–106, 1987.
- Oddsson LI. Control of voluntary trunk movements in man. Mechanisms for postural equilibrium during standing. Acta Physiol Scand Suppl 595: 1-60, 1990.
- 53. Oddsson LI, Giphart JE, Buijs RJ, Roy SH, Taylor HP, and De Luca CJ. Development of new protocols and analysis procedures for the assessment of LBP by surface EMG techniques. J Rehabil Res Dev 34: 415–426, 1997.
- 54. Pink M, Jobe FW, Perry J, Browne A, Scovazzo ML, and Kerrigan J. The painful shoulder during the butterfly stroke. An electromyographic and cinematographic analysis of twelve muscles. Clin Orthop 288: 60–72, 1993.
- Rekling JC, Funk GD, Bayliss DA, Dong XW, and Feldman JL. Synaptic control of motoneuronal excitability. *Physiol Rev* 80: 767–852, 2000.
- Ren K and Dubner R. Central nervous system plasticity and persistent pain. J Orofac Pain 13: 155–163, 1999.
- 57. **Roland MO.** A critical review of the evidence for a pain-spasm-pain cycle in spinal disorders. *Clin Biomech (Bristol, Avon)* 1: 102–109, 1986.
- Roy SH. The role of muscle fatigue in low back pain. In: Back Pain Rehabilitation, edited by D'Orazio BP. Boston, MA: Andover Medical, 1993, p. 149–179.
- 59. Roy SH. Combined use of surface electromyography and ³¹P-NMR spectroscopy for the study of muscle disorders. *Phys Ther* 73: 892–901, 1993.
- Roy SH, Bonato P, and Knaffitz M. EMG assessment of back muscle function during cyclical lifting. J Electromyogr Kinesiol 8: 233–245, 1998.
- Roy SH, De Luca CJ, and Casavant DA. Lumbar muscle fatigue and chronic lower back pain. Spine 14: 992–1001, 1989.

- 62. Roy SH, De Luca CJ, Emley M, and Buijs RJ. Spectral electromyographic assessment of back muscles in patients with low back pain undergoing rehabilitation. *Spine* 20: 38–48, 1995.
- 63. Roy SH, De Luca CJ, Emley M, Oddsson LI, Buijs RJ, Levins JA, Newcombe DS, and Jabre JF. Classification of back muscle impairment based on the surface electromyographic signal. J Rehabil Res Dev 34: 405–414, 1997.
- 64. Roy SH, De Luca CJ, Snyder-Mackler L, Emley MS, Crenshaw RL, and Lyons JP. Fatigue, recovery, and low back pain in varsity rowers. *Med Sci Sports Exerc* 22: 463–469, 1990.
- 65. Roy SH, Kupa EJ, Kandarian SC, and De Luca CJ. Effects of muscle fiber type and size on EMG median frequency. *Proc Int Congr ISEK 10th*, Charleston, SC, 1994, p. 32–33.
- Roy SH and Oddsson LI. Classification of paraspinal muscle impairments by surface electromyography. *Phys Ther* 78: 838– 851, 1998.
- 67. Sauer SK, Bove GM, Averbeck B, and Reeh PW. Rat peripheral nerve components release calcitonin gene-related peptide and prostaglandin E2 in response to noxious stimuli: evidence that nervi nervorum are nociceptors. *Neuroscience* 92: 319–325, 1999.
- 67a.Scientific approach to the assessment, and management of activity-related spinal disorders. A monograph for clinicians. Report of the Quebec Task Force on Spinal Disorders. Spine 12, Suppl 7: S1–S59, 1987.
- 68. Sihvonen T, Partanen J, Hanninen O, and Soimakallio S. Electric behavior of low back muscles during lumbar pelvic rhythm in low back pain patients and healthy controls. Arch Phys Med Rehabil 72: 1080–1087, 1991.
- Simone DA, Marchettini P, Caputi G, and Ochoa J. Identification of muscle afferents subserving sensation of deep pain in humans. J Neurophysiol 72: 883–889, 1994.
- 70. **Snook SH.** The costs of back pain in industry. *Occup Med* 3: 1–5, 1988.
- 71. **Stulen FB and DeLuca CJ.** Frequency parameters of the myoelectric signal as a measure of muscle conduction velocity. *IEEE Trans Biomed Eng* 28: 515–523, 1981.
- Thompson DA and Biedermann HJ. Electromyographic power spectrum analysis of the paraspinal muscles. Long-term reliability. Spine 18: 2310–2313, 1993.
- 73. **Triano JJ and Schultz AB.** Correlation of objective measure of trunk motion and muscle function with low-back disability ratings. *Spine* 12: 561–565, 1987.
- Urban MO and Gebhart GF. Central mechanisms in pain. Med Clin North Am 83: 585-596, 1999.
- 75. Vlaeyen JW, Seelen HA, Peters M, de Jong P, Aretz E, Beisiegel E, and Weber WE. Fear of movement/(re)injury and muscular reactivity in chronic low back pain patients: an experimental investigation. *Pain* 82: 297–304, 1999.
- 76. Wolf SL and Basmajian JV. Assessment of paraspinal electromyographic activity in normal subjects and in chronic low back pain patients using a muscle feedback device. In: *Biomechanics 6-A*, edited by Asmussen E and Jorgensen K. Baltimore, MD: University Park, 1977, p. 319–324.
- 77. Yuan CX, Xing JH, and Yan CY. Observations on clinical therapeutic effect in treating soft tissue injuries by acupuncture, with pain threshold and electromyography as parameters. J Tradit Chin Med 9: 40–44, 1989.
- 78. **Zochodne DW.** Epineurial peptides: a role in neuropathic pain? *Can J Neurol Sci* 20: 69–72, 1993.