Original Article

J. Phys. Ther. Sci.
26: 323–329, 2014

Properties of Force Output and Spectral EMG in Young Patients with Nonspecific Low Back Pain during Isometric Trunk Extension

Tatsuhiro Miura, PT, MS[1)*], Keishoku Sakuraba, MD, PhD[2)]

[1)] *Department of Sports Medicine, Graduate School of Medicine, Juntendo University: Bunkyo-ku, Tokyo, Japan*
[2)] *Department of Sports Medicine, Graduate School of Health and Sports Science, Juntendo University, Japan*

Abstract. [Purpose] To clarify the influence of nonspecific low back pain (NSLBP) on force fluctuation and the myoelectric data of back muscles during isometric trunk extension at low to high force levels. [Subjects] Fourteen male subjects with NSLBP and 14 healthy male control subjects participated in this study. [Methods] All participants extended their trunk isometrically maintaining 10 levels of target force [2, 5, 10, 15, 20, 30, 50, 70, 80 and 90% of maximal voluntary contraction (MVC) in a random order] for about 4 seconds with visual feedback. A force transducer and tri-axis force sensor were positioned at the 7th thoracic vertebra to measure force output and the direction of force. Myoelectric activities of the back muscles (longissimus thoracis, L2 level; multifidus, S1 level) were recorded by surface electromyography. [Results] Force output of NSLBP subjects fluctuated more than that of healthy subjects at 30% and 50%MVC. Higher median power frequency in the multifidus was observed in NSLBP subjects at moderate to high force levels. [Conclusion] These results show that the properties of force output in NSLBP subjects differ from those in healthy subjects, suggesting that the assessment of force fluctuation of back muscles at moderate force levels is a useful index for evaluating and discriminating NSLBP.
Key words: Force fluctuation, Back muscle, Nonspecific low back pain

(This article was submitted Aug 5, 2013, and was accepted Sep. 22, 2013)

INTRODUCTION

Low back pain (LBP) is one of the most prevalent conditions afflicting people today, and most people experience LBP at some point in their lives. The lifetime prevalence of LBP is reported to be as high as 84%, and best estimates suggest that the prevalence of chronic LBP is about 23%, with 11–12% of the population being disabled by it[1)]. More than 85% of patients who present to primary care have low back pain that cannot reliably be attributed to a specific disease or spinal abnormality; this condition is called nonspecific low back pain (NSLBP)[2–4)]. Studies of the causes of LBP and strategies for its prevention have been important themes in recent times. Despite many researchers' efforts, there remain a large number of unanswered questions and disagreements about which risk factors are the most influential. Previous studies of individual factors have reported that previous experience of LBP and aging are high risk factors[5, 6)], as well as an association between genetic factors and LBP[7, 8)]. Gender, obesity, and smoking are reported to have a weak association with LBP[9–11)]. In studies of biomedical factors, prolonged bending and twisting[12, 13)], poor fitness and muscle strength are reported as risk factors[14, 15)]. Recent studies of the association between muscular structure and LBP have established a general consensus that muscles play an important role in LBP[16–19)].

In the modern lifestyle, people spend much time sitting. A Dutch population-based study reported that a sedentary lifestyle posed a higher risk of chronic NSLBP because of low activity levels[20)]. Actually, activity levels of trunk muscles in sitting postures and spinal pelvic movement in sitting is very low (less than 15% maximal voluntary contraction; MVC) as previously reported by Miura et al[21, 22)]. Moreover it has been reported that muscle activity levels of the trunk extensors in daily living is 20−30%MVC[23)]. If LBP is associated with low to moderate levels of muscle activity, the study of muscle properties at these activity levels is required.

As for the properties of back muscles, many surface electromyography (EMG) studies have demonstrated differences in activation patterns[17)], and asymmetrical activation[24)] between LBP subjects and healthy controls. Especially, power spectrum analysis has been increasingly used for the purpose of diagnosis and assessment of fatigability of LBP patients. Median frequency changes with fatigue have also been used to discriminate between healthy subjects and people with LBP[25–28)]. In these studies, initial median fre-

*Corresponding author. Tatsuhiro Miura (E-mail: miura@stf.teu.ac.jp)

quency (IMF) was used, which has been demonstrated as being satisfactorily reliable[29]. Patients with LBP had higher IMF at the L5 site at 80%MVC[30], and at the L4/5 site at 66.6%MVC[27] than healthy subjects; however, the IMF of patients with LBP during low to moderate force output is unclear. The fact that the IMF of patients with LBP is different from healthy subjects indicates that the motor control of patients with low back pain is also different from that of healthy subjects.

There is another view of motor control in muscle function, which focuses on force fluctuation. It has been reported that force output during static contractions are not constant but fluctuate around a mean value, and the standard deviation (SD) of force increases as the target force increases[31, 32]. Force fluctuation is a natural characteristic of human motor control[33], and is mainly influenced by the excitation and discharge behavior of the motoneuron pool[34]. Increased force fluctuation has been associated with muscle fatigue[35], aging[36], musculoskeletal disorders and pain[37]. Force fluctuations are known to depend on muscle architecture and on the level of the exerted force[34]. With regard to back muscles, only a few studies have been conducted. These studies showed that fatigue increases force fluctuation, but the force levels investigated are unclear[38]. However, there have been no studies of the influence back muscles have on force fluctuation in LBP subjects. Therefore, the aim of the current study was to clarify the influence of NSLBP on force fluctuation and the myoelectric data of back muscles during isometric trunk extension from low to high force levels.

SUBJECTS AND METHODS

Fourteen male subjects with NSLBP and 14 healthy male control subjects participated in this study. The criteria for NSLBP subjects were defined in accordance with a previous study with some modifications[27]. Subjects with NSLBP had a history of at least two episodes in the previous year severe enough to prevent their performance of activities of daily living (ADL), and at least one episode in the last 3 months. Pain levels were set at a minimum of 3 out of 10 on a visual analogue scale. Healthy subjects had no history of LBP in the last 5 years. None of the subjects had any structural disorder in the lumbar region. The characteristics of the subjects are shown in Table 1. There were no statistical differences in age, height, body weight or BMI (body mass index) between the NSLBP group and the healthy control group. The purpose and methods of this study were explained to all the participants who read and signed an informed consent revealing all the details of the study protocol, which were approved by the ethics committee of the School of Health and Sports Science, Juntendo University (No. 24-14).

During force measurements, subjects were positioned in a custom-designed setup designed to allow isometric trunk extensions to be performed prone, with the pelvis stabilized with belts to avoid movement. Trunk extension force was measured with a force transducer (TU-BR; TEAC, Tokyo, Japan) that was adjusted to make contact with the midline of each subject at the T7 level. The force output was low-pass filtered at 1 kHz and recorded at a sampling frequency of 2 kHz.

Table 1. Characteristics of subjects

	Healthy (n=14)	NSLBP (n=14)
Age (year)	21.6±2.3	21.1±1.1
Height (cm)	173.8±5.3	172.2±5.5
Weight (kg)	65.5±5.6	62.2±4.4
BMI	21.7±2.0	21.0±1.9

NSLBP: nonspecific low back pain, BMI: body mass index

EMG activity of the back muscles was measured with pairs of self-adhesive disposable Ag/AgCl disc surface electrodes (Blue Sensor, N-00-S; Ambu, Denmark) that were attached to the skin. These electrodes were placed at an inter-electrode distance of 20 mm, parallel to the following muscles on the left side: longissimus thoracis (2–3 cm lateral to the L2 spinous process); and the lumbar multifidus (S1 level, parallel to a line connecting the posterior superior iliac spine and L1–L2 interspinous space). The placement of the electrodes varied slightly according to the shape of the subjects' bodies. A ground electrode was placed over the left iliac crest. The electrodes were taped securely to avoid excessive movement of the leads. Before electrode placement, the skin was shaved and abraded with fine sandpaper to reduce skin impedance to below 5 kΩ. Surface EMG signals were recorded at a sampling frequency of 2,000 Hz using a Noraxon MyoSystem 1200 (Noraxon USA Inc., Scottsdale, AZ, USA). The EMG system bandwidth was 10–1,000 Hz, the common mode rejection ratio was greater than 100 dB at 60 Hz, and the differential input impedance was greater than 10 MΩ. All raw myoelectric signals were amplified with a gain of 1,000. Data were collected and processed using MyoResearch XP Ver.1.07 computer software (Noraxon USA Inc., Scottsdale, AZ, USA). First, the raw surface EMG data were visually checked for electrocardiac artifacts, and when artifacts were observed, they were removed using a program in MyoResearch[39]. EMG characteristics such as the root mean square (RMS) and median power frequency (MdPF) were extracted for each muscle. Fast Fourier Transforms (FFT) utilizing the Hanning window were used to calculate MdPF.

Subjects performed isometric contractions with the back muscles so that the trunk exerted an extension force to match a series of ten target forces. To determine the target forces, each subject first performed two or more MVCs until the peak force of at least two of the MVCs were within 5% of each other. The peak force exerted during an acceptable MVC trial was taken as the MVC force. The target forces were performed in a random order with three trials at each of 2, 5, 10, 15, 20, 30, 50, 70, 80 and 90% of MVC force. Before beginning the series, subjects adequately practiced the task at each target force; however, practice was not performed at 70, 80 and 90% of MVC in order to avoid fatigue. Subjects were instructed to increase trunk extension force within 2 seconds to match the target, indicated on a visual

Table 2. Standard deviation and coefficient of variation of force of healthy and nonspecific low back pain subjects

		Target force (%MVC)	2	5	10	15	20	30	50	70	80	90	
SD of force	Healthy	mean	0.29	0.50	0.70	0.97	1.03	1.23	2.40	3.06	3.82	4.97	
		(SE)	(0.02)	(0.04)	(0.06)	(0.06)	(0.08)	(0.12)	(0.16)	(0.33)	(0.36)	(0.63)	# ****
	NSLBP	mean	0.29	0.49	0.73	0.95	1.20	1.64	2.96	3.03	4.25	5.30	
		(SE)	(0.03)	(0.04)	(0.07)	(0.11)	(0.16)	(0.20)	(0.23)	(0.36)	(0.50)	(0.70)	
CV of force (%)	Healthy	mean	2.34	1.62	1.15	1.04	0.86	0.68	0.80	0.72	0.81	0.94	
		(SE)	(0.17)	(0.11)	(0.09)	(0.05)	(0.07)	(0.06) *	(0.04) **	(0.07)	(0.07)	(0.11)	# ****
	NSLBP	mean	2.51	1.69	1.26	1.12	1.04	0.94	1.05	0.76	0.95	1.04	¶ *
		(SE)	(0.15)	(0.10)	(0.10)	(0.13)	(0.11)	(0.08)	(0.07)	(0.07)	(0.10)	(0.12)	

*p<0.05, **p<0.01, ****p<0.0001
MVC: maximal voluntary contraction, NSLBP: nonspecific low back pain, SD: standard deviation, CV: coefficient of variation, SE: standard error of the mean
# indicates a significant main effect of target force
¶ indicates a significant main effect of group

display positioned 50 cm away from the face, and to hold that force as steadily as possible for 4 seconds, in each extension at 50% or less of target force (Experiment 1). In extensions at 70% or more of target force, subjects increased the trunk extension force within 1 second and held that force for 2 seconds or as close as possible to 2 seconds. For visual feedback, a horizontal line was placed on a computer monitor to indicate the target force for each trial. The gain of the force display was adjusted so that the target-force line was always at the same location on the monitor. Moreover, subjects were instructed to maintain a constant direction of trunk extension, which was monitored by examiners on a three-axis force sensor. Adequate rest was given between trials to minimize fatigue. The rest lasted a minimum of 30 seconds, or until the subject felt fully recovered from the previous trial, with considerably longer rest periods after high-force contractions.

At the end of these trials, subjects were asked to perform 30 isometric trunk extensions with visual feedback at a target force of 30%MVC (Experiment 2). The task consisted of repeated 12 seconds cycles subdivided into 1 second of progressive rise to reach the target force, 5 seconds to sustain this force level as steadily as possible, and 6 seconds of rest. The task lasted 6 minutes (30 cycles).

For each submaximal contraction, the absolute mean forces, the standard deviation (SD) of the force and the coefficient of variation (CV) of force (SD/mean force×100) were assessed in a time window of 2 seconds (in trials at 70%, 80% and 90%MVC, the time window was 1 second), which was determined as the period when the CV was smallest. Then, RMS and MdPF of EMG were calculated in the same time window. The RMS value was normalized to %MVC.

In statistical analysis for Experiment 1, two-factor, repeated-measures analysis of variance (RM-ANOVA) was used to compare the NSLBP and the healthy control group (between-subject factor) at each of the ten target force levels (repeated-measures factor) for SD, CV, RMS and MdPF. Post hoc tests, including the Bonferroni test and the unpaired t-test, were conducted as appropriate. Regression analysis was also performed on the MdPF of the multifidus muscle using the least-squares method. For Experiment 2, two-factor RM-ANOVA was used to compare the NSLBP and the healthy control group (between-subject factor) during the 30 trials (repeated-measures factor) for CV, RMS and MdPF. Statistical analysis was performed using Dr. SPSS II for Windows (version 11.0.1 J; SPSS, Inc., Chicago, IL). P values less than 0.05 were considered significant. All results are reported as mean ± standard error of the mean (SE) except for the force at MVC.

RESULTS

The results of Experiment 1 were as follows. Maximal voluntary contraction force output in the NSLBP group was 606.4±68.8 N (mean ± SD) and 575.8±101.8 N in the healthy group. MVC force output per weight was 9.3±1.1 N/kg in the NSLBP group, 9.2±1.4 N/kg in the healthy group. No significant differences between the groups were observed.

There was a main effect of target force in the RM-ANOVA of the SD of force indicating a general increase with higher target force (Table 2, $F_{9, 234}$=78.796, p<0.0001). As for SD of force, there was neither a significant main effect of group nor an interaction between group and target force ($F_{1, 26}$=0.787, p=0.383; $F_{9, 234}$=0.367, p=0.727). Regarding CV of force, there was a main effect of target force indicating a general decrease with higher target force up to 30%MVC, and a leveling off from 30% to 90%MVC in general (Table 2, $F_{9, 234}$=52.351, p<0.0001). Moreover, there was a main effect of group indicating a significant difference between the NSLBP and the healthy group ($F_{1, 26}$=4.698, p<0.05), and the post hoc test showed that CV of the NSLBP group was significantly higher than that of the healthy group at 30% and 50%MVC (p<0.05 and p<0.01, respectively). There was no significant interaction between group and target force ($F_{9, 234}$=0.490, p=0.770).

There was a main effect of target force in the RM-ANOVA of the RMS of both muscles (longissimus thoracis and multifidus) indicating a monotonic increase with higher target force (Table 3, $F_{9, 234}$=351.285, p<0.0001; $F_{9, 234}$=469.307, p<0.0001). As for RMS, there were no signifi-

Table 3. Root mean square of EMG and median power frequency values of the longissimus thoracis and multifidus of healthy and nonspecific low back pain subjects

			Target force (%MVC)	2	5	10	15	20	30	50	70	80	90	
RMS (%MVC)	Longissimus thoracis	Healthy	mean	18.1	20.5	23.6	27.7	29.9	37.9	55.1	72.4	85.3	89.3	
			(SE)	(2.3)	(2.8)	(1.5)	(2.4)	(2.3)	(3.0)	(2.9)	(3.7)	(4.2)	(3.3)	# ****
		NSLBP	mean	15.6	18.5	22.3	25.2	28.2	35.3	50.0	69.6	74.6	93.8	
			(SE)	(2.1)	(2.3)	(2.9)	(2.6)	(3.3)	(2.7)	(3.5)	(4.1)	(4.1)	(3.4)	
	Multifidus	Healthy	mean	20.4	22.8	27.6	30.4	33.6	40.1	56.4	70.0	81.1	87.7	
			(SE)	(2.5)	(2.8)	(2.1)	(2.2)	(3.0)	(2.9)	(3.2)	(2.9)	(3.6)	(3.2)	# ****
		NSLBP	mean	23.7	25.8	30.0	33.1	37.2	44.6	64.2	78.5	84.8	97.1	
			(SE)	(2.3)	(2.9)	(2.9)	(2.7)	(3.2)	(2.9)	(3.3)	(3.7)	(2.6)	(3.7)	
MdPF (Hz)	Longissimus thoracis	Healthy	mean	75.9	76.4	77.2	77.9	76.6	80.1	73.9	69.1	68.3	67.1	
			(SE)	(2.8)	(2.7)	(3.8)	(3.1)	(2.6)	(2.8)	(3.1)	(3.4)	(3.8)	(3.5)	# ****
		NSLBP	mean	76.3	74.7	76.0	76.3	75.0	75.6	75.6	73.0	71.7	65.9	
			(SE)	(3.1)	(2.9)	(3.7)	(3.2)	(3.2)	(3.1)	(3.5)	(3.2)	(3.0)	(4.0)	
	Multifidus	Healthy	mean	126.6	132.4	132.0	130.9	134.3	130.8	122.8	109.5	107.0	103.8	
			(SE)	(5.7)	(6.2)	(5.3)	(5.3)	(5.0)	(3.9)	(4.8)	(4.6)	(4.4)	(4.8)	* # ****
		NSLBP	mean	131.0	131.2	131.9	132.6	132.9	135.1	132.6	127.2	121.5	119.4	§ ***
			(SE)	(4.2)	(5.3)	(4.2)	(4.5)	(4.5)	(5.5)	(4.8)	(5.4)	(4.9)	(5.1)	

*p<0.05, ***p<0.001, ****p<0.0001
MVC: maximal voluntary contraction, NSLBP: nonspecific low back pain, RMS: root mean square, MdPF: median power frequency, SE: standard error of the mean
# indicates a significant main effect of target force
§ indicates a significant interaction between group and target force

cant main effects of group in either the longissimus thoracis or multifidus ($F_{1, 26}$=0.647, p=0.429; $F_{1, 26}$=1.908, p=0.179), and there were no significant interactions between group and target force in both muscles ($F_{9, 234}$=1.632, p=0.169; $F_{9, 234}$=1.198, p=0.316).

Regarding MdPF, there was a main effect of target force indicating a stable value up to 30%MVC and a modest decrease from 30% to 90%MVC in the longissimus thoracis muscle (Table 3, $F_{9, 234}$=11.266, p<0.0001). There was neither a significant main effect of group nor an interaction between group and target force in the longissimus thoracis muscle ($F_{1, 26}$=0.003, p=0.954; $F_{9, 234}$=1.397, p=0.239). On the other hand, there was a main effect of target force in the RM-ANOVA of the MdPF of multifidus muscle indicating a stable value up to 50%MVC and a decrease from 50% to 90%MVC (Table 3, $F_{9, 234}$=25.226, p<0.0001). Moreover, there was a significant group × target force interaction in the MdPF of multifidus muscle ($F_{9, 234}$=5.997, p<0.001). A post hoc test showed a simple main effect that MdPF of the NSLBP group was significantly higher than that of the healthy group at 70%, 80% and 90%MVC (p<0.05). There was no significant main effect of group in the MdPF of multifidus ($F_{1, 26}$=1.095, p=0.305). Regression analysis by the least squares method of the MdPF of the multifidus muscle and the target force revealed that MdPF decreased after 50%MVC of the target force in the NSLBP group (r=0.269, p<0.05) and after 30%MVC in the healthy group (r=0.529, p<0.001).

The results of Experiment 2 were as follows. Regarding CV, there was a significant main effect of group, but neither a significant main effect of trial number nor an interaction between group and trial number was observed ($F_{1, 26}$=8.190, p<0.01; $F_{29, 754}$=1.036, p=0.415; $F_{29, 754}$=0.520, p=0.883). There were neither significant main effects (group, trial number) nor interactions (group × trial number) in the RM-ANOVA analysis of the RMS of both muscles (longissimus thoracis: $F_{1, 26}$=0.139, p=0.712; $F_{29, 754}$=1.049, p=0.398; $F_{29, 754}$=0.896; multifidus: $F_{1, 26}$=0.898, p=0.352, $F_{29, 754}$=1.104, p=0.398, $F_{29, 754}$=0.716, p=0.659). There was a main effect of trial number in the RM-ANOVA analysis of the MdPF of longissimus thoracis muscle indicating a modest decrease up to 5 trials and leveling off after that ($F_{29, 754}$=5.906, p<0.0001). The same main effect of trial number was observed for the multifidus up to 4 trials ($F_{29, 754}$=10.934, p<0.0001). There was neither a significant main effect of group nor an interaction between group and trial number for the MdPF of either muscle (longissimus thoracis: $F_{1, 26}$=0.031, p=0.862; multifidus: $F_{1, 26}$=0.082, p=0.777).

DISCUSSION

The current study assessed the back muscle properties of NSLBP subjects in detail focusing on force fluctuation during isometric trunk extension, and EMG characteristics such as RMS and MdPF. Our present results show a difference between NSLBP and healthy subjects in force fluctuations at moderate target forces and in MdPF changes at high target forces.

No significant difference in MVC force output was observed between the NSLBP group and the healthy control.

MVC force output per weight was also the same. Previous studies have shown that LBP patients often do not produce a "true" MVC for fear of pain, resulting in LBP patients having less fatigable back muscles than healthy control subjects in fatiguing protocols[24, 30, 40]. It may be possible to compare the results between the two groups in the present study, because the NSLBP subjects exerted MVC force at the same level as those of healthy subjects, probably due to the fact that the NSLBP subjects had no pain while participating in the experiment. It should also be possible to adequately compare the results between the two groups since there were no differences in the height, body weight and BMI of the two groups of subjects.

The SD of force generally increased as the target force increased. This result is in agreement with previous studies of finger, elbow, knee and ankle muscles[41–43]. The SD of force also generally increased as the RMS of EMG increased. This increase in SD with higher target force is related to the orderly recruitment of motor units. When muscle activity increases, larger motor units are recruited[44], which produce larger and unfused twitches, causing an increase in force fluctuation[45].

The CV of force generally decreased as the target force rose to 30%MVC, and leveled off at higher than 50%MVC. This result is almost the same as those reported in previous studies of finger, elbow, knee and ankle muscles[38, 41, 46, 47]. At low target forces, few motor units are active, so any recruitment or change in the discharge rates of the active units causes higher fluctuations. As the target force increases, the relative increment of each motor unit recruitment declines and the fluctuations reach a plateau as the force level further increases[46]. CV values in the present study were generally lower than those reported in previous studies. This may be because the data analyzed in this study was sampled from the steadiest portion of the data collected in each of three trials. Comparing the NSLBP and the healthy group, there was no difference in the SD of force, but there was a difference in the CV of force at 30% and 50%MVC. Since the CV of force is a normalized measure of force output fluctuation, it is sensitive to motor unit recruitment and discharge behavior[41, 46]. Previous studies have reported that experimental muscle pain increased force fluctuation during isometric contractions of the elbow, knee and ankle muscles at 2.5, 20, 50 and 70%MVC force, with no changes in the overall force level[47, 48]. The higher CV of the NSLBP group at 30% and 50%MVC in this study indicates changes in the motor unit population, and the influence of a history of low back pain on motor control.

One might question whether a higher CV of force in the NSLBP group at 30% and 50%MVC was due to the influence of fatigue. However, this was disproved by the results of Experiment 2, in which no change was observed in the level of CV during 30 trials at 30%MVC target force in both groups. Also, the CV of the healthy group was always consistently lower than that of the NSLBP group, which indicates that the higher CV of NSLBP at 30%MVC is not due to fatigue. Moreover, EMG values such as RMS and MdPF showed no influence of fatigue, for example increasing RMS or decreasing MdPF[49, 50]. A previous study reported that healthy male subjects could repeat isometric trunk extension at 40%MVC of force more than 50 times, with 8-second cycles (1.5 seconds of progressive rise to reach the target force, 5 seconds to sustain this force level, and 1.5 seconds of rest)[51]. In Experiment 1 of this study, subjects rested sufficiently between trials so they should not have been tired. Therefore, the data collected in Experiment 1 is unlikely to have been influenced by fatigue. We propose that the same would be true at 50%MVC, however this was not confirmed by experimental investigation.

Regarding the EMG data, first, the RMS of both muscles in both groups almost linearly increased as the level of force increased, suggesting that EMG data were properly collected. Second, MdPF was higher in the multifidus of the NSLBP group at high force levels than in the healthy group, as reported in previous studies[27, 30]. It is unclear how these EMG data relate to basic muscle physiology, but there are three possible explanations: 1. Previous studies have reported that there are differences in muscle fiber types between LBP patients and healthy subjects. Mannion AF et al. demonstrated that the lumbar paraspinal muscle of LBP patients is made up of more type IIB fibers, which are relatively larger than type I fibers, compared to normal controls[52, 53]. Motor units in muscles made up of type I fibers show the characteristics of firing in a more orderly manner and are able to discharge at longer inter-spike intervals, while motor units in muscle made up of type II muscle fibers fire at shorter intervals[54]. The differences of muscle fiber type between the two groups might cause MdPF differences; 2. The synchronization of motor unit firing might be another explanation. At high force levels, motor unit firing is more in-phase and the motor units are more synchronized with each other[55, 56], resulting in a decrease in MdPF. The motor unit firing patterns of the multifidus of the NSLBP group would most likely be less synchronized; 3. One more possible explanation may be the low-pass filter effect of body skin and subcutaneous tissues[57]. As the level of force increases, large and deeper motor units with higher conduction velocities are recruited, which are attenuated by these tissues. The deep fibers of the multifidus in the NSLBP group might be relatively less activated than those in the healthy group, a hypothesis that seems to be supported by previous research. The multifidus muscle has been shown to make a major contribution to the control and segmental stabilization of the lumbar spine[58] and is associated with LBP[59]. Previous research shows that the fatigue rate of this muscle is greater in LBP patients than in healthy subjects[60], and that select atrophy of this muscle can be observed in LBP patients[61].

From the results of CV of force we expected to find differences between the groups in muscle activities at 30% and 50%MVC when analyzing the EMG values, but no significant differences were detected in the RMS or MdPF of EMG of either the longissimus thoracis or multifidus muscles at these moderate force levels. However, the three possible explanations given above may explain the differences in MdPF of the multifidus at high forces. Although a similar trend appeared to occur at moderate forces, the results were not significant. Regression analysis found a decrease

in MdPF of the multifidus from 30%MVC in the healthy group, while there was a decrease from 50%MVC in the NSLBP group, indicating differences in muscle activation properties. These differences are highlighted by the fact that the CV of force was different between the two groups at these same moderate force levels, a result which seems to be associated with MdPF differences in the multifidus.

Although there were differences in MdPF between the two groups at high force levels, no differences were observed in the CV of force. The reason for this result seems to be that at high force levels the recruitment pattern and synchronization of motor unit firing caused the fluctuation of force to reach a plateau, making it difficult to observe a difference between the two groups as was observed for MdPF.

Finally, we would like to consider the influence of NSLBP on EMG and force fluctuation, the main purpose of this study. Previous studies have reported that there are relatively more type I fibers in back muscles[62, 63]. However, the recruitment and firing rate patterns of motor units of back muscles are unclear. If what we know about the deltoid muscle, which has almost the same ratio of muscle fiber composition[62], is referred to, it seems that the recruitment and synchronization of motor units is a more important contractile mechanism than firing rate modulation in back muscles[64]. Again, it is possible that the properties of the multifidus of NSLBP, observed through EMG, indicate relatively few type I fibers, less synchronized activation patterns, and less activated deep fibers, which are detected at high force levels but are difficult to detect at low to moderate force levels. However, the CV of force detected a difference between NSLBP and healthy subjects at moderate force levels, which indicates the usefulness of the CV of force in the assessment of NSLBP patients, because NSLBP patients cannot perform well at higher levels of force, and ADL often require tasks to be performed at moderate levels of muscle power.

The current results should be considered in light of the factor that all subjects were young men. The CV of force has been shown to be affected by aging[36]. Therefore, in order to limit the effects of this variable, the subjects recruited to participate in this study were of a similar age. In the case of this study, this age group happened to be young men due to subject availability. Although this limitation should be investigated further in the future, we believe the results of this study are meaningful.

In conclusion, the current study investigated the influence of NSLBP on force fluctuation and the myoelectric data of back muscles during isometric trunk extension at low to high force levels. The results of this study demonstrate that NSLBP increases force fluctuation at moderate force levels, and these changes are accompanied by changes in MdPF of the multifidus muscle. We suggest that the assessment of force fluctuation of back muscles, especially the multifidus, at moderate force levels is a useful index for the evaluation and discrimination of NSLBP.

REFERENCES

1) Airaksinen O, Brox JI, Cedraschi C, et al.: Chapter 4. European guidelines for the management of chronic nonspecific low back pain. Eur Spine J, 2006, 15: S192–S300. [Medline] [CrossRef]
2) Chou R, Qaseem A, Snow V, et al.: Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med, 2007, 147: 478–491. [Medline] [CrossRef]
3) van Tulder MW, Assendelft WJ, Koes BW, et al.: Spinal radiographic findings and nonspecific low back pain. A systematic review of observational studies. Spine, 1997, 22: 427–434. [Medline] [CrossRef]
4) Balagué F, Mannion AF, Pellise F, et al.: Non-specific low back pain. Lancet, 2012, 379: 482–491. [Medline] [CrossRef]
5) Adams MA, Mannion AF, Dolan P: Personal risk factors for first-time low back pain. Spine, 1999, 24: 2497–2505. [Medline] [CrossRef]
6) Gilgil E, Kacar C, Butun B, et al.: Prevalence of low back pain in a developing urban setting. Spine, 2005, 30: 1093–1098. [Medline] [CrossRef]
7) Omair A, Holden M, Lie BA, et al.: Treatment outcome of chronic low back pain and radiographic lumbar disc degeneration are associated with inflammatory and matrix degrading gene variants: a prospective genetic association study. BMC Musculoskelet Disord, 2013, 14: 105. [Medline] [CrossRef]
8) Seki S, Kawaguchi Y, Chiba K, et al.: A functional SNP in CILP, encoding cartilage intermediate layer protein, is associated with susceptibility to lumbar disc disease. Nat Genet, 2005, 37: 607–612. [Medline] [CrossRef]
9) Stevenson JM, Weber CL, Smith JT, et al.: A longitudinal study of the development of low back pain in an industrial population. Spine, 2001, 26: 1370–1377. [Medline] [CrossRef]
10) Shiri R, Karppinen J, Leino-Arjas P, et al.: The association between obesity and low back pain: a meta-analysis. Am J Epidemiol, 2010, 171: 135–154. [Medline] [CrossRef]
11) Shiri R, Karppinen J, Leino-Arjas P, et al.: The association between smoking and low back pain: a meta-analysis. Am J Med, 2010, 123: 87e7–87e35. [Medline] [CrossRef]
12) Hoogendoorn WE, Bongers PM, de Vet HC, et al.: High physical work load and low job satisfaction increase the risk of sickness absence due to low back pain: results of a prospective cohort study. Occup Environ Med, 2002, 59: 323–328. [Medline] [CrossRef]
13) Punnett L, Fine LJ, Keyserling WM, et al.: Back disorders and nonneutral trunk postures of automobile assembly workers. Scand J Work Environ Health, 1991, 17: 337–346. [Medline] [CrossRef]
14) Batti'e MC, Bigos SJ, Fisher LD, et al.: Isometric lifting strength as a predictor of industrial back pain reports. Spine, 1989, 14: 851–856. [Medline] [CrossRef]
15) Verbunt JA, Smeets RJ, Wittink HM: Cause or effect? Deconditioning and chronic low back pain. Pain, 2010, 149: 428–430. [Medline] [CrossRef]
16) Geisser ME, Ranavaya M, Haig AJ, et al.: A meta-analytic review of surface electromyography among persons with low back pain and normal, healthy controls. J Pain, 2005, 6: 711–726. [Medline] [CrossRef]
17) Hubley-Kozey CL, Vezina MJ: Differentiating temporal electromyographic waveforms between those with chronic low back pain and healthy controls. Clin Biomech (Bristol, Avon), 2002, 17: 621–629. [Medline] [CrossRef]
18) Krismer M, van Tulder M: Strategies for prevention and management of musculoskeletal conditions. Low back pain (non-specific). Best Pract Res Clin Rheumatol, 2007, 21: 77–91. [Medline] [CrossRef]
19) van Dieën JH, Cholewicki J, Radebold A: Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine, 2003, 28: 834–841. [Medline] [CrossRef]
20) Heneweer H, Vanhees L, Picavet HS: Physical activity and low back pain: a U-shaped relation? Pain, 2009, 143: 21–25. [Medline] [CrossRef]
21) Miura T, Sakuraba K: Trunk muscle activation during changing the posture from slump to upright in sitting. Professionalism Physiotherapy, 2010, 4: 1–6.
22) Miura T, Sakuraba K: Influence of different spinal alignments in sitting on trunk muscle activity. J Phys Ther Sci, 2013, 25: 483–487. [CrossRef]
23) Sawai S, Sanematsu H, Kanehisa H, et al.: Evaluation of muscular activity in daily actions. Jpn J Phys Fit Sprts Med, 2004, 53: 93–106.
24) Oddsson LI, De Luca CJ: Activation imbalances in lumbar spine muscles in the presence of chronic low back pain. J Appl Physiol, 2003, 94: 1410–1420. [Medline]
25) Roy SH, De Luca CJ, Casavant DA: Lumbar muscle fatigue and chronic lower back pain. Spine, 1989, 14: 992–1001. [Medline] [CrossRef]
26) Kramer M, Ebert V, Kinzl L, et al.: Surface electromyography of the paravertebral muscles in patients with chronic low back pain. Arch Phys Med Rehabil, 2005, 86: 31–36. [Medline] [CrossRef]
27) Humphrey AR, Nargol AV, Jones AP, et al.: The value of electromyography of the lumbar paraspinal muscles in discriminating between chronic-low-

back-pain sufferers and normal subjects. Eur Spine J, 2005, 14: 175–184. [Medline] [CrossRef]
28) Heydari A, Nargol AV, Jones AP, et al.: EMG analysis of lumbar paraspinal muscles as a predictor of the risk of low-back pain. Eur Spine J, 2010, 19: 1145–1152. [Medline] [CrossRef]
29) Nargol AV, Jones AP, Kelly PJ, et al.: Factors in the reproducibility of electromyographic power spectrum analysis of lumbar paraspinal muscle fatigue. Spine, 1999, 24: 883–888. [Medline] [CrossRef]
30) Elfving B, Dedering A, Nemeth G: Lumbar muscle fatigue and recovery in patients with long-term low-back trouble–electromyography and health-related factors. Clin Biomech (Bristol, Avon), 2003, 18: 619–630. [Medline] [CrossRef]
31) Enoka RM, Christou EA, Hunter SK, et al.: Mechanisms that contribute to differences in motor performance between young and old adults. J Electromyogr Kinesiol, 2003, 13: 1–12. [Medline] [CrossRef]
32) Galganski ME, Fuglevand AJ, Enoka RM: Reduced control of motor output in a human hand muscle of elderly subjects during submaximal contractions. J Neurophysiol, 1993, 69: 2108–2115. [Medline]
33) Stein RB, Gossen ER, Jones KE: Neuronal variability: noise or part of the signal? Nat Rev Neurosci, 2005, 6: 389–397. [Medline] [CrossRef]
34) Tracy BL, Mehoudar PD, Ortega JD: The amplitude of force variability is correlated in the knee extensor and elbow flexor muscles. Exp Brain Res, 2007, 176: 448–464. [Medline] [CrossRef]
35) Missenard O, Mottet D, Perrey S: Factors responsible for force steadiness impairment with fatigue. Muscle Nerve, 2009, 40: 1019–1032. [Medline] [CrossRef]
36) Tracy BL: Force control is impaired in the ankle plantarflexors of elderly adults. Eur J Appl Physiol, 2007, 101: 629–636. [Medline] [CrossRef]
37) Hortobágyi T, Garry J, Holbert D, et al.: Aberrations in the control of quadriceps muscle force in patients with knee osteoarthritis. Arthritis Rheum, 2004, 51: 562–569. [Medline] [CrossRef]
38) Salomoni SE, Graven-Nielsen T: Muscle fatigue increases the amplitude of fluctuations of tangential forces during isometric contractions. Hum Mov Sci, 2012, 31: 758–771. [Medline] [CrossRef]
39) Hu Y, Mak JN, Luk KD: Effect of electrocardiographic contamination on surface electromyography assessment of back muscles. J Electromyogr Kinesiol, 2009, 19: 145–156. [Medline] [CrossRef]
40) Larivière C, Arsenault AB, Gravel D, et al.: Surface electromyography assessment of back muscle intrinsic properties. J Electromyogr Kinesiol, 2003, 13: 305–318. [Medline] [CrossRef]
41) Moritz CT, Barry BK, Pascoe MA, et al.: Discharge rate variability influences the variation in force fluctuations across the working range of a hand muscle. J Neurophysiol, 2005, 93: 2449–2459. [Medline] [CrossRef]
42) Hong SL, Lee MH, Newell KM: Magnitude and structure of isometric force variability: mechanical and neurophysiological influences. Motor Control, 2007, 11: 119–135. [Medline]
43) Svendsen JH, Madeleine P: Amount and structure of force variability during short, ramp and sustained contractions in males and females. Hum Mov Sci, 2010, 29: 35–47. [Medline] [CrossRef]
44) Henneman E, Somjen G, Carpenter DO: Functional significance of cell size in spinal motoneurons. J Neurophysiol, 1965, 28: 560–580. [Medline]
45) Jones KE, Hamilton AF, Wolpert DM: Sources of signal-dependent noise during isometric force production. J Neurophysiol, 2002, 88: 1533–1544. [Medline]
46) Taylor AM, Christou EA, Enoka RM: Multiple features of motor-unit activity influence force fluctuations during isometric contractions. J Neurophysiol, 2003, 90: 1350–1361. [Medline] [CrossRef]
47) Salomoni SE, Graven-Nielsen T: Experimental muscle pain increases normalized variability of multidirectional forces during isometric contractions. Eur J Appl Physiol, 2012, 112: 3607–3617. [Medline] [CrossRef]
48) Salomoni SE, Ejaz A, Laursen AC, et al.: Variability of three-dimensional forces increase during experimental knee pain. Eur J Appl Physiol, 2013, 113: 567–575. [Medline] [CrossRef]
49) Ng JK, Richardson CA, Jull GA: Electromyographic amplitude and frequency changes in the iliocostalis lumborum and multifidus muscles during a trunk holding test. Phys Ther, 1997, 77: 954–961. [Medline]
50) Olson MW: Trunk muscle activation during sub-maximal extension efforts. Man Ther, 2010, 15: 105–110. [Medline] [CrossRef]
51) Larivière C, Gravel D, Gardiner P, et al.: A submaximal test to assess back muscle capacity: evaluation of construct validity. J Electromyogr Kinesiol, 2009, 19: e422–e429. [Medline] [CrossRef]
52) Mannion AF, Kaser L, Weber E, et al.: Influence of age and duration of symptoms on fibre type distribution and size of the back muscles in chronic low back pain patients. Eur Spine J, 2000, 9: 273–281. [Medline] [CrossRef]
53) Mannion AF, Weber BR, Dvorak J, et al.: Fibre type characteristics of the lumbar paraspinal muscles in normal healthy subjects and in patients with low back pain. J Orthop Res, 1997, 15: 881–887. [Medline] [CrossRef]
54) Grimby L, Hannerz J: Firing rate and recruitment order of toe extensor motor units in different modes of voluntary conraction. J Physiol, 1977, 264: 865–879. [Medline]
55) Solomonow M, Baten C, Smit J, et al.: Electromyogram power spectra frequencies associated with motor unit recruitment strategies. J Appl Physiol, 1990, 68: 1177–1185. [Medline]
56) Hermens HJ, Bruggen TA, Baten CT, et al.: The median frequency of the surface EMG power spectrum in relation to motor unit firing and action potential properties. J Electromyogr Kinesiol, 1992, 2: 15–25. [Medline] [CrossRef]
57) Qi L, Wakeling JM, Green A, et al.: Spectral properties of electromyographic and mechanomyographic signals during isometric ramp and step contractions in biceps brachii. J Electromyogr Kinesiol, 2011, 21: 128–135. [Medline] [CrossRef]
58) Danneels LA, Vanderstraeten GG, Cambier DC, et al.: A functional subdivision of hip, abdominal, and back muscles during asymmetric lifting. Spine, 2001, 26: E114–E121. [Medline] [CrossRef]
59) MacDonald DA, Moseley GL, Hodges PW: The lumbar multifidus: does the evidence support clinical beliefs? Man Ther, 2006, 11: 254–263. [Medline] [CrossRef]
60) Biedermann HJ, Shanks GL, Forrest WJ, et al.: Power spectrum analyses of electromyographic activity. Discriminators in the differential assessment of patients with chronic low-back pain. Spine, 1991, 16: 1179–1184. [Medline] [CrossRef]
61) Danneels LA, Vanderstraeten GG, Cambier DC, et al.: CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. Eur Spine J, 2000, 9: 266–272. [Medline] [CrossRef]
62) Johnson MA, Polgar J, Weightman D, et al.: Data on the distribution of fibre types in thirty-six human muscles. An autopsy study. J Neurol Sci, 1973, 18: 111–129. [Medline] [CrossRef]
63) Mannion AF, Dumas GA, Cooper RG, et al.: Muscle fibre size and type distribution in thoracic and lumbar regions of erector spinae in healthy subjects without low back pain: normal values and sex differences. J Anat, 1997, 190: 505–513. [Medline] [CrossRef]
64) Sale DG: Influence of exercise and training on motor unit activation. Exerc Sport Sci Rev, 1987, 15: 95–151. [Medline] [CrossRef]