

## Localized Oxygen Use of Healthy and Low Back Pain Individuals During Controlled Trunk Movements

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**Summary:** Individuals who have low back pain (LBP) have significantly different motion characteristics than healthy individuals. However, the cause of these differences is unknown. Oxygen use of the erector spinae muscle was examined while simultaneously monitoring motion characteristics to determine whether oxygen use differed between healthy and LBP individuals. Thirty volunteers were classified as healthy, structural, or muscular-based LBP. A near-infrared spectrometer monitored oxygen use and blood volume in the lumbar region. Results showed significant differences in oxygen use but not blood volume between healthy and LBP subjects with muscular-based disorders. Inability of the muscular group to use oxygen in a manner similar to the healthy group indicates different processes at the tissue level, indicating that differences in oxygen use may provide insight into why motion patterns differ between healthy and LBP groups. **Key Words:** Back injury—Oximetry—NIRS—Blood volume—Erector spinae—Muscle damage.

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Low back disorders have been recognized as one of the most common problems in the United States today (1). It is estimated that in 1994, 65% of reported injuries affected the back. Additionally, back pain is recognized as the most costly musculoskeletal disorder in industry, with the mean cost for each case at \$8,321 in 1989 (2). Despite the prevalence of this problem, it remains difficult to diagnose (3–5). Patients are classified as having “low back pain syndrome,” or are categorized into groups based on their description of pain (3,6).

Subjective measures are not sufficient in determining the health status of the spinal area. Both the perception of loading and fatigue are determined by the individual, and cannot be quantitatively measured. As coactivity of the muscles increases, so may the loading on the disc (7). It has been shown that there are few receptors in the disc area of the spine capable of recognizing the compressive load placed on the disc (8). Therefore, individuals may severely underestimate the loads that are tolerable on the spine. Furthermore, fatigue is not an adequate measure of

capability; it is possible that injury may occur before fatigue is ever perceived by the individual. To properly assess the safety of returning to work, an objective measurement of capability after a back injury is needed.

Many physical, psychologic, and socioeconomic parameters have been evaluated to objectify the functional improvement of low back pain (LBP) (4). However, with such an abundance of available methods, no particular area has been recognized as being the best measure of low back disability or impairment (5). The ideal measure allows for a quantitative evaluation of function. Physical capability is easily studied, and has included measures of strength, physiological factors (heart rate, oxygen consumption), fatigue, and motion patterns.

Strength testing, physiological factors, and fatigue have not shown much success in differentiating between healthy individuals and those with LBP (9,10). Strength, heart rate, and whole body oxygen use evaluate the total body fitness of the individual rather than focusing on the source of pain for those with LBP. Although electromyography (EMG) has been used frequently (11–15), the use of EMG in determining the functional change to the muscle has not been well defined, and must overcome many complications. EMG is often evaluated under iso-

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metric conditions, which cannot be applied to dynamic activity. Also, maximal voluntary contractions (MVCs), traditionally used to normalize EMG activity, may be influenced by both pain and motivational factors (4). Each of these methods has several limitations that make them unreliable techniques for evaluating patients with LBP.

Little work has been done in the area of motion pattern behaviors. However, Marras et al. have shown this to be a more reliable method. These studies have shown considerable reliability in correctly classifying both subjects with LBP and healthy individuals (16,17). Additionally, this method allows for the evaluation of tasks at various degrees of asymmetry. It was found that the measure of velocity (derived from position) showed significant differences between normal and injured lumbar spines. In the 1993 and 1995 articles of Marras et al. (16,17), one- and two-stage models (respectively) were developed that were highly successful in differentiating between healthy individuals and those with LBP, and also had moderate success in the classification of subjects with LBP into 1 of 10 low-back-disorder classification groups.

Although differences in motion are recognized between those with LBP and healthy individuals, it remains unknown what factor is responsible for these differences. The internal factor that is often considered a driving mechanism of functional motion is the oxygen consumption in the body. Oxygen is fundamental to physical performance. Therefore, the limited physical capabilities of those with LBP may be attributable to a change in their use of oxygen. It has been found that endurance training increases the oxidative metabolism of muscles (18,19). During activity, trained muscles demonstrated lower fatigue rates and a more efficient use of oxygen. For this to occur, there must be a change in the physiologic functions of the muscle. Initial force measurements between untrained and trained muscles showed no significant difference (18). This demonstrates that the change within the muscle results in a different mechanism of oxygen use without altering the strength of the muscle.

Although oxygen levels may be monitored internally, this method is invasive and does not allow for extended monitoring in humans. Another means of measuring the oxygen in a muscle can be performed by using near-infrared spectroscopy (NIRS). NIRS has been shown to be successful in monitoring muscular oxygen levels (20–25).

Blasi et al. (24) examined the levels of oxygen in the muscle with and without occlusion of blood flow. The consumption of oxygen during MVCs declined in both cases, which demonstrated that NIRS could be used successfully in dynamic exercise without occlusion. These results were compatible with those of Hamaoka et al. (21) and Murthy et al. (22), who also noted a decline in oxygen levels during submaximal muscular contraction. As the

level of activity increased, the intensity of oxygen consumption also increased. The use of NIRS during dynamic activity was confirmed by Belardinelli et al. (23), who showed that the oxygen levels of the muscle progressively decreased during incremental exercise.

To determine why there are differences in motion between healthy and LBP individuals, the muscles used for movement of the spine should be considered on a more definable level, in their physiologic capabilities. Although the oxygen use of the whole body does not hold much promise for classifying LBP, the use of oxygen in the active muscles themselves may demonstrate a functional difference between these groups.

Damage to the muscle tissue alters the physiological events within the muscle, thereby altering the function of the muscle while the regeneration process takes place (19,26). If there is an inability to repair the muscle, degeneration will continue (26). A void remains in our knowledge of how oxygen is used by damaged or regenerating tissue. It would be expected that a difference would be seen between the localized oxygen consumption of healthy subjects and those with LBP caused by a muscular injury. With healthy muscle tissue, oxygen levels and blood volume decrease as oxygen is consumed by the exercising muscle (25). Alternately, with damaged tissue the oxidative capacity may be altered, resulting in decreased oxygen consumption (27). Differences in oxygen consumption may not be detectable between healthy subjects and those with LBP caused by a structural problem (such as a herniated or degenerative disc). Although muscle injury may be present in those with structural problems, the source of pain is most likely created by nerve compression or impingement.

This study evaluated the hypothesis that localized oxygen consumption is dissimilar between muscle-based or structural-based LBP and those who are in good health. These differences are indicative of a functional change in muscles driving spinal motion caused by back injury. This may provide insight as to why motion characteristics are altered with injury.

## METHODS

### Subjects

The 30 subjects who volunteered for this study consisted of 18 men and 12 women ages 24–47 (mean = 33.7, SD = 4.0). The mean height of these subjects was 176.3 cm (SD = 1.02), and mean weight was 79.8 kg (SD = 6.9).

Subjects were divided into one of three categories: healthy, structural, or muscular. Healthy subjects had no prior history of back problems, whereas the other two

groups currently had LBP. The structural group consisted of two subjects with spondylolisthesis, three with degenerative disc disorder (DDD), three with a herniated disc, and one with both DDD and spondylolisthesis. The muscular subjects did not have any known cause for pain. Both patient categories were evaluated by a physician before their inclusion in this study. All subjects were non-smokers and in good health at the time of the study. Summary data for each category are shown in Table 1.

### Apparatus

The lumbar motion monitor (LMM) described in Marras et al. (16) was used to monitor trunk motion for all subjects. The LMM is an exoskeleton of the spine that continuously monitors the position of the trunk in three-dimensional space. Velocity and acceleration are also continuously monitored. Data were collected on a laptop computer.

The device used to measure the oxygen level of the muscles was a near-infrared spectrometer, the MRM-96 (NIM, Inc., Philadelphia, PA, U.S.A.). This device measures deoxygenation in the capillary bed of an exercising muscle (25). Deoxygenation is measured using dual-wavelength spectrophotometry. Light is sent out from the sensor at wavelengths of 760 and 850 nm. The sum of these signals received at the detector represents the blood volume change, whereas the difference (760–850) represents the change in deoxygenation. The pattern of photon migration is symmetrical, allowing for localization within a muscle (28). Although no definite measurement of the muscle volume sampled during these measurements can be made, it is known that the average depth of light penetration is 2.5 to 3.0 cm. The linearity of the NIRS in evaluating tissue deoxygenation changes has been previously validated (23,24,29). The NIRCOM software was also used to aid in analyzing the data, and data were stored on a laptop computer. Changes in both oxygen and blood volume level were monitored throughout the entire experiment.

### Independent Variables

The independent variables for this study were subject group and the asymmetry of the subject. The subject group

was healthy, structural, or muscular, and the asymmetries included 0, 15, and 30° of twist to both the right and left (indicated by 15R, 15L, 30R, and 30L).

### Dependent Variables

The dependent variables considered for this experiment consisted of oxygen and blood volume-related parameters and motion characteristics. The motion characteristics analyzed were the same as those described by Marras et al. (17). Position, velocity, and acceleration of the trunk were evaluated in the three planes of the body: sagittal, lateral, and transverse. Based on their motion parameters, a probability of normal was calculated for each subject.

The oxygen-related parameters examined were the change in oxygen level of the muscle (right erector spinae) and the rate of oxygen depletion. Oxygen change was evaluated relative to the oxygen level at the beginning of each task. The maximum change in oxygen level during each trial was evaluated, as well as the change in oxygen level from the beginning to the end of each trial. The rate of oxygen change was also considered, with rate being found as oxygen change between the beginning and ending of each task divided by the total time of the task. Maximum rate was considered as the maximum change in oxygen level (relative to the initial level) divided by the time for the change to occur. Each of these variables was also evaluated for changes in blood volume.

### Procedure

The tasks performed by these subjects followed the procedure described by Marras et al. (17). Before performing the experiment, the experimental procedure was explained to the subject, background information was collected, and a consent form was signed. Each subject was fitted with an appropriately sized LMM, and the oxygen monitor was placed over the right erector spinae muscles in the lumbar spine region. Subjects were asked to flex and extend the trunk as quickly as possible, while maintaining their twisting position at a given asymmetry (0°, and 15 and 30° of twist to both the right and left). The positive twisting direction was to the right, whereas the negative direction twisted to the left. A visual display was provided that the subject watched throughout each task to maintain their position. The velocity and sagittal range of motion were determined by the subjects, and subjects performed all conditions within their capabilities. There were also three tasks in which the subject was asked to move in a particular plane of motion, with no given boundary constraints. These tasks were conducted to evaluate the difference between controlled and uncontrolled movements of the trunk and included: sagittal, in which the subject

**TABLE 1.** Mean and standard deviations of subject characteristics by category

Category	Males/females	Age	Height (cm)	Weight (kg)
Healthy	6/6	30.9 (5.1)	176.8 (12.8)	73.0 (17.9)
Structural	6/3	38.9 (8.3)	176.3 (9.3)	84.6 (18.3)
Muscular	6/3	31.4 (6.7)	175.3 (4.6)	82.8 (8.8)

was asked to flex and extend in the sagittal plane; lateral, in which the subject bent from side to side; and twist, in which the subject was asked to twist the trunk from side to side.

**Data Analysis**

The software developed in the Biodynamics Laboratory at Ohio State University was used to analyze the motion data collected. This software, described by Marras et al. (30), calculated trunk position, velocity, and acceleration to be evaluated from each task. Probability of normal was calculated using the Marras et al. (30) model of statistical analysis. These variables will be compared between the three different groups of subjects. Oxygen and blood volume variables were also compared between the patient groups and the healthy subjects. Oxygen and blood volume are evaluated as changes in optical density. This value multiplied by 100 is equal to the relative percent change in optical density. For each task performed, statistical analysis was performed to determine the significance of each of the dependent variables. The statistical analysis used *t* tests (SAS version 6.09) to determine whether either of the patient groups was significantly different from the healthy group. Results were considered significant for  $\alpha < 0.05$ , and mildly significant for  $\alpha < 0.10$ .

**RESULTS**

**Oxygen and Blood Volume Parameters**

The results of *t* tests for the dependent oxygen variables for the healthy versus structural (H/S) and healthy versus muscular (H/M) groups are shown in Table 2. Only oxygen change and maximum oxygen change showed significance ( $\alpha < 0.10$ ) between the healthy and structural groups for the 15L° condition. No other variables displayed significance over all of the conditions. Two tasks, the 0°

condition and 15R twist, displayed significance between the healthy and muscular groups. For the 0° condition, all variables were significant at the  $\alpha < 0.05$  level. For the 15R asymmetry condition, all oxygen parameters except maximum oxygen change showed significance at the  $\alpha < 0.05$  level, whereas maximum oxygen change was significant at  $\alpha < 0.10$ . It should be considered that during the first three conditions (0, 15R, and 15L), asymmetry was controlled throughout the task, whereas for the latter conditions (sagittal, lateral, and twist) subjects were not constrained to a particular range.

Figure 1 shows the means and standard deviations for oxygen change for all three groups. The 30° asymmetry conditions are not included, because none of the LBP subjects were able to perform these tasks. The trends seen for both of these figures are compatible, and may be considered simultaneously. For each of the tasks that required sagittal flexion (0° controlled, 15R, 15L, and sagittal uncontrolled), the healthy group showed the least amount of oxygen change, whereas the muscular group showed the greatest amount of change in oxygen level. During the purely sagittal flexion tasks (0° controlled and sagittal uncontrolled), the healthy group showed very little change in oxygen level, displaying a minimal increase in oxygen throughout the task. Alternately, the muscular group showed an increase in oxygen during the 0° controlled condition and a decrease in oxygen level for the uncontrolled sagittal condition. The 15R and 15L asymmetry conditions also involved sagittal flexion, but in these cases twisting was incorporated into the motion. For both of these conditions, the healthy group showed a decrease in oxygen level. The muscular group showed a sharp rise in oxygen during the 15R asymmetry condition. The muscular group consistently showed greater oxygen changes than the healthy group, whereas no particular pattern was seen with the structural group.

Maximum oxygen change, rate of oxygen change, and maximum rate of oxygen change all displayed the same

**TABLE 2.** Statistical summary of *p* values for oxygen parameters

Parameter		Task					
		0°	15 Twist	-15 Twist	Sagittal	Lateral	Twist
H/S	O <sub>2</sub> change (OC)	0.66	0.41	0.07*	0.66	0.33	0.13
	O <sub>2</sub> rate (OR)	0.68	0.39	0.13	0.65	0.18	0.27
	Maximum OC	0.82	0.43	0.07*	0.71	0.35	0.23
	Maximum OR	0.95	0.56	0.15	0.69	0.20	0.92
H/M	O <sub>2</sub> change (OC)	0.05**	0.05**	0.71	0.17	0.71	0.68
	O <sub>2</sub> rate (OR)	0.05**	0.01**	0.99	0.29	0.42	1.00
	Maximum OC	0.02**	0.06*	0.51	0.31	0.98	0.92
	Maximum OR	0.04**	0.05**	0.38	0.67	0.80	0.50

H/S, healthy versus structural; H/M, healthy versus muscular.  
 \*\*Indicates significance at the  $\alpha < 0.05$  level.  
 \*Indicates significance at the  $\alpha < 0.10$  level.

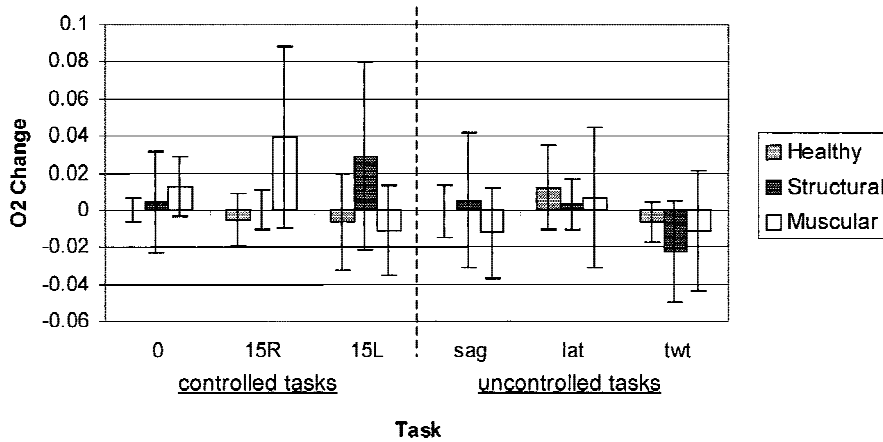


FIG. 1. Oxygen change versus task for all groups.

trends as were seen with oxygen change (Fig. 1). This is confirmed by the significance values seen in Table 2.

The results of *t* tests for the blood volume (BV) parameters for both the structural and muscular groups as compared with the healthy group are shown in Table 3. Contrary to the oxygen parameter results, significance was seen between the healthy and structural groups for each of the blood volume measures during the 0° controlled condition. Additionally, blood volume rate was significant at  $\alpha < 0.10$  during the lateral condition. No particular pattern of significance was seen between the healthy and muscular groups, although the blood volume change (BVC) and maximum BVC showed significance ( $\alpha < 0.10$ ) for the 15° asymmetry and significance at the  $\alpha < 0.05$  level for the sagittal condition. Additionally, BVC and blood volume rate (BVR) were significant for the lateral condition, and BVR was significant at  $\alpha < 0.10$  during the twist condition.

The means and standard deviations for blood volume change are shown in Figure 2. During the sagittal flexion conditions, the healthy group consistently decreased its

blood volume level; this pattern was also seen with the structural group. The muscular group differed in that blood volume increased slightly during the 15R asymmetry condition. Little difference was seen between the BVC and maximum BVC for each of the conditions.

As with the oxygen parameters, maximum BVC, the rate of blood volume change and the maximum rate of BVC all displayed the same trends as BVC (Fig. 2). These trends were also found to be significant, as shown in Table 3.

### Motion Parameters

*T* tests were also performed on the sagittal motion parameters for each condition that involved flexion and extension. The results of these tests can be seen in Table 4.

### DISCUSSION

The objective of this study was to evaluate the effectiveness of the NIRS device in determining the low back health status of an individual. A previously validated pro-

TABLE 3. Statistical summary of *p* values for blood volume (BV) parameters

Parameter		Task					
		0°	15 Twist	-15 Twist	Sagittal	Lateral	Twist
H/S	BV change (BVC)	0.01**	0.77	0.38	0.26	0.27	0.85
	BV rate (BVR)	0.03**	0.59	0.49	0.73	0.07*	0.46
	Maximum BVC	0.01**	0.84	0.37	0.26	0.26	0.97
	Maximum BVR	0.05**	0.94	0.47	0.81	0.16	0.48
H/M	BV change (BVC)	0.82	0.09*	0.62	0.03**	0.03**	0.26
	BV rate (BVR)	0.91	0.16	0.60	0.18	0.01**	0.09*
	Maximum BVC	0.56	0.08*	0.65	0.04**	0.32	0.31
	Maximum BVR	0.80	0.17	0.62	0.31	0.94	0.17

H/S, healthy versus structural; H/M, healthy versus muscular.

\*\*Indicates significance at the  $\alpha < 0.05$  level.

\*Indicates significance at the  $\alpha < 0.10$  level.

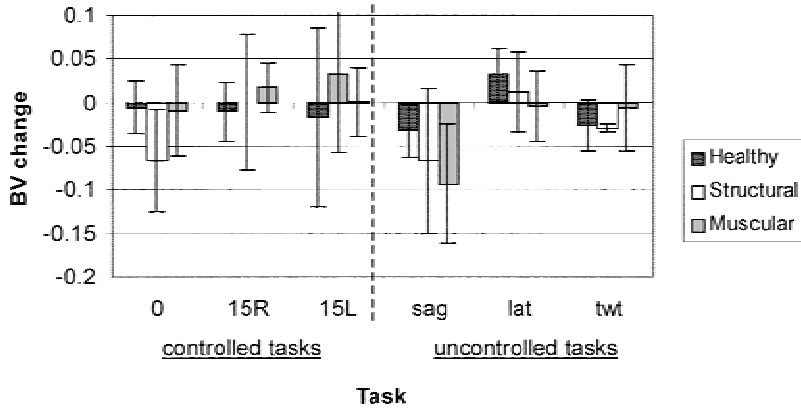


FIG. 2. Blood volume change versus task for all groups.

toloc (30) that evaluated the motion parameters of both healthy subjects and subjects with back pain was used as the criterion standard for quantitatively distinguishing between healthy individuals and those with LBP.

The oxygen parameters evaluated showed significance between the healthy and muscular groups, but not as strongly between the healthy and structural group. This may have been caused by the source of the initial injury. As diagnosed by a physician, all subjects within the muscular group showed no identifiable signs of any structural damage to their lumbar spine, which potentially indicates that the source of pain is located within the muscle itself. However, the structural group showed clear evidence of their particular disorders. It is possible, then, for that group to have pain because of several reasons; although there may be damage to the muscle, it is also likely that pain is being caused by nerve compression or deterioration. If pain is being caused by a multitude of disorders, it is not surprising that the subjects in this group showed very different oxygen consumptions and that no definite pattern could be found.

For the 15R and 15L controlled asymmetry conditions,

TABLE 4. Statistical summary of p values for sagittal motion parameters

Parameter	Task			
	0°	15 Twist	-15 Twist	Sagittal
H/S Range of motion	0.52	0.04	0.02	0.68
H/S Flexion velocity	0.00	0.00	0.00	0.00
H/S Extension velocity	0.00	0.00	0.00	0.00
H/S Flexion acceleration	0.01	0.00	0.01	0.00
H/S Extension acceleration	0.01	0.00	0.01	0.00
H/M Range of motion	0.36	0.50	0.82	0.28
H/M Flexion velocity	0.00	0.00	0.03	0.00
H/M Extension velocity	0.00	0.00	0.05	0.00
H/M Flexion acceleration	0.01	0.01	0.03	0.00
H/M Extension acceleration	0.01	0.01	0.03	0.00

H/S, healthy versus structural; H/M, healthy versus muscular.

the healthy group consistently showed a normal response—oxygen level as well as blood volume decreased. As muscle tissue is exercised, it can be expected that BV and oxygen levels will decrease as blood is blocked from entering the muscle because of contraction and the oxygen is used for energy (25). For the 0° controlled condition, a slight increase in oxygen was seen; however, this increase was minimal, and differed very little from the baseline value taken at the beginning of the task. A slight decrease was seen in BV for this condition. It is likely that in the short duration of these tasks, the exercise was not robust enough to create a large decrease in oxygen. For the controlled 0° and 15R conditions, the muscular group showed a significant increase in oxygen level, whereas BV levels did not differ significantly from those seen with the healthy group. This increase in oxygen indicated that oxygen was not effectively being used by the muscle.

These results can be linked with literature explaining the physiological basis of muscle injury. Several different types of trauma produce similar muscular responses (26). Although information on the regeneration of muscle is incomplete, several things are known about the physical structure of injured muscle (26,27). With acute injury, there is extensive disruption of the structural components of muscle (31). However, injury may be caused in many ways and still result in damage to muscle fibers (26). It is common with any type of injury that further degradation of the muscle occurs after the initial injury (32).

Injury to the muscle is characterized by several traits. The myofibrils of the muscle break down, leading to disruption of the mitochondria, sarcoplasmic reticulum, and sarcolemma (27). With damage to the sarcolemma, the barriers protecting the muscle cells are weakened. Because of the large concentration gradient, calcium is then able to move down the gradient into the muscle cell (26). This increased intracellular calcium is a common mediator in skeletal muscle regeneration (33). Because of the increased concentration of calcium within the cell, the mi-

tochondria work to buffer more calcium. This decreases respiration of the cell, thereby decreasing the amount of ATP present for energy (19,26). With decreased ability to regenerate ATP, the muscle becomes stiffer. If stretching occurs during this state, cytoskeletal or myofibrillar damage may occur (19). Alternate effects of decreased ATP levels include decreased function of ion pumps and decreased contractile function (26).

The link between cell damage and the results seen here is that with mitochondrial damage, the oxidative enzyme activity of the cell is decreased (27). Clearly, the patients seen in this study with muscular back pain did not use the available oxygen during exercise. This inability to consume oxygen is explained if the subjects incurred mitochondrial damage because of their muscle injury and is represented in this study by an increase in the oxygenation measures. Additionally, if no damage to the mitochondria occurred, it is possible that the mitochondria's ability to use oxygen was inhibited by their attempt to buffer calcium within the cell. An additional link can be made between muscle damage and the motion parameter results. With the inability to use oxygen and quickly regenerate ATP, impaired contractile function results (26). This impaired function is observed in the decreased velocity and acceleration of the muscular subjects compared with the healthy subjects.

The results seen in this study agree with those seen in studies examining subjects with metabolic myopathies (34,35). With metabolic myopathy, there is damage to the mitochondria. Although damage in these cases is typically progressive, the results of these studies provide insight about the mechanism of oxygen use in subjects with muscle injury. Bank and Chance found that the oxygen level of the muscle increased while the BV decreased during exercise for subjects with cytochrome c oxidase or myophosphorylase deficiencies. Additionally, Abe et al. (34) found that although oxygen levels decreased during exercise, a much smaller use of oxygen was seen for the subjects with metabolic myopathies than with the normal group. Both studies used NIRS to measure localized oxygen and blood volume.

It was interesting to note that no significant difference in oxygen consumption was detected between the healthy and muscular groups for the 15L asymmetry condition. This may be explained by the pain symptoms described by the muscular subjects. Of the nine subjects, five reported pain across both sides of their back, whereas the remaining four reported a greater amount of back pain on the right side. Because the right side is not as tense during twisting to the left (15L asymmetry condition), these subjects may have been more comfortable while performing this task, thereby allowing them to perform in a manner more similar to the healthy group. The situation with the structural

group may be similarly explained, because five of the nine subjects reported pain on the left side of their back and the 15R asymmetry condition was not found to be significant.

The oxygen-related differences seen between the healthy and muscular groups of subjects agree with the results seen from the motion parameters evaluated. For the controlled 0 and 15R conditions, healthy and muscular LBP subjects were clearly differentiated. Although the LMM data clearly separate these subjects across all conditions, the NIRS data provide more insight about their muscle usage patterns. For the 0 and 15R controlled asymmetry conditions, subjects were forced to cocontract muscles, and could not avoid using the erector spinae muscle. This was represented by the changes in oxygen seen during these conditions. No clear explanation can be made regarding the 15L condition. It is possible that during this task, patients were not forced to use the damaged muscle. The fact that the muscular subjects were not different from healthy subjects for the uncontrolled conditions was also not surprising. For both the lateral and twist conditions, no flexion or extension took place. It is likely that the erector spinae muscle was not being used, and therefore no change in its oxygen or blood volume use was detected. Similarly with the uncontrolled sagittal condition, subjects could have avoided using the damaged muscle, because their movement was not controlled in any way.

The agreement of the NIRS and LMM data indicates that a functional difference exists between healthy subjects and muscular LBP subjects. For those with muscular LBP, it is likely that the individual has sustained an injury at the tissue or cellular level. This injury inhibits the muscle from behaving in a manner similar to that seen for the healthy group.

There are several other possible explanations for the source of differences between healthy and LBP subjects. Pain in the facet joints may cause subjects with LBP to "guard" against pain, thereby cocontracting several muscles during trunk movement. Pain caused by the intersegmental muscles may have similar effects. Patients with LBP may also have altered recruitment of muscles based on the location of their pain. With muscles on the same venous drain, if a particular muscle remains continually contracted, blood supply cannot effectively be delivered. Therefore, an increased amount of blood and oxygen will be delivered to the muscle that is not contracting.

Knowing that an injury exists at the muscular level is an important piece of information when evaluating those with muscular back injuries. An important contribution of this study is that the pattern of oxygen use helps to explain why motion patterns change between LBP and healthy individuals and also why motion patterns of LBP individuals change over time. An assessment of muscular oxy-

gen consumption in conjunction with motion characteristics may be used to successfully determine the improvement of muscular LBP patients. Muscles may be trained to improve their function as well as their oxygen use (18,19). This evaluation may then be used to determine the effectiveness of a particular treatment. Hopefully, with a better understanding of the injury and the status of improvement, individuals may be better advised as to what activities are within their capability. This may, in turn, reduce the risk of reinjury.

### Limitations and Future Research

Using the same method as was used here, a comparison of the oxygen use both before and after injury would provide further insight about oxygen use during muscle injury and regeneration. It would also be useful to perform a prospective study monitoring the oxygen use of patients over time.

Finally, although metabolic deficiency may be the cause for the increased oxygen levels seen with the patient groups, this finding should be validated with a closer examination of the muscle tissue. Muscle biopsy or electron microscopy may be used to better evaluate the damage to the muscle.

### CONCLUSIONS

It has been shown that NIRS is an effective measure for distinguishing between healthy individuals and those with muscular back pain. Because BV was not significantly different between these two groups, it is clear that the structure of the muscle itself is damaged, altering the ability of the muscle to use oxygen in a normal manner. This difference provides insight as to why velocity and acceleration patterns of muscular-based LBP subjects are slower than in healthy subjects during controlled conditions.

### REFERENCES

- Marras WS, Fine LJ, Ferguson SA, et al. The effectiveness of commonly used lifting assessment methods to identify industrial jobs associated with elevated risk of low-back disorders. *Ergonomics* 1999;42:229-45.
- U.S. Department of Health and Human Services (USDHHS). *Musculoskeletal Disorders and Workplace Factors. Second Printing.* Cincinnati: NIOSH, 1997.
- Kelsey JL, White AA. Epidemiology and impact of low-back pain. *Spine* 1980;5:133-42.
- Flores L, Gatchel RJ, Polatin PB. Objectification of functional improvement after nonoperative care. *Spine* 1997;22:1622-33.
- Waddell G, Allan DB, Newton M. Clinical evaluation of disability in low back pain. In: *The Adult Spine: Principles and Practice.* Philadelphia: Lippincott-Raven Publishers, 1997:171-84.
- Spitzer WO, Leblanc FE, Dupuis M, et al. 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* 1987;12:S1-S59.
- Marras WS, Mirka GA. A comprehensive evaluation of trunk response to asymmetric trunk motion. *Spine* 1992;17:318-26.
- Cavanaugh JM. Neural mechanisms of lumbar pain. *Spine* 1995;20:1804-9.
- Leamon TB. Research to reality: a critical review of the validity of various criteria for the prevention of occupationally induced low back pain disability. *Ergonomics* 1994;37:1959-74.
- Riihimaki H. Low-back pain, its origin and risk indicators. *Scand J Work Environ Health* 1991;17:81-90.
- Roy SH, DeLuca CJ, Casavant DA. Lumbar muscle fatigue and chronic lower back pain. *Spine* 1989;14:992-1001.
- Roy SH, DeLuca CJ, Emley M, et al. Spectral electromyographic assessment of back muscles in patients with low back pain undergoing rehabilitation. *Spine* 1995;20:38-48.
- Peach JP, McGill SM. Classification of low back pain with the use of spectral electromyogram parameters. *Spine* 1998;23:1117-23.
- Crenshaw AG, Karlsson S, Gerdle B, et al. Differential responses in intramuscular pressure and EMG fatigue indicators during low- vs high-level isometric contractions to fatigue. *Acta Physiol Scand* 1997;160:353-61.
- Mannion AF, Dolan P. Electromyographic median frequency changes during isometric contraction of the back extensors to fatigue. *Spine* 1994;19:1223-9.
- Marras WS, Parnianpour M, Ferguson SA, et al. Quantification and classification of low back disorders based on trunk motion. *Eur J Phys Med Rehabil* 1993;3:218-35.
- Marras WS, Parnianpour M, Ferguson SA, et al. The classification of anatomic- and symptom-based low back disorders using motion measure models. *Spine* 1995;20:2531-46.
- Ravalec X, LeTallec N, Carre F, et al. Improvement of muscular oxidative capacity by training is associated with slight acidosis and ATP depletion in exercising muscles. *Muscle Nerve* 1996;19:355-61.
- Friden J, Lieber R. Structural and mechanical basis of exercise-induced muscle injury. *Med Sci Sports Exerc* 1992;24:521-30.
- Sahlin K. Non-invasive measurements of O<sub>2</sub> availability in human skeletal muscle with near infrared spectroscopy. *Int J Sports Med* 1992;13:157-60.
- Hamaoka T, Iwane H, Shimomitsu T, et al. Noninvasive measures of oxidative metabolism on working human muscles by near-infrared spectroscopy. *J Appl Physiol* 1996;81:1410-7.
- Murthy G, Kahan NJ, Hargens AR, et al. Forearm muscle oxygenation decreases with low levels of voluntary contraction. *J Orthop Res* 1997;15:507-11.
- Belardinelli R, Barstow TJ, Porszasz J, et al. Changes in skeletal muscle oxygenation during incremental exercise measured with near infrared spectroscopy. *Eur J Appl Physiol* 1995;70:487-92.
- Blasi RA, Cope M, Elwell C, et al. Noninvasive measurement of human forearm oxygen consumption by near infrared spectroscopy. *Eur J Appl Physiol* 1993;67:20-5.
- Rundell KW, Nioka S, Chance B. Hemoglobin/myoglobin desaturation during speed skating. *Med Sci Sports Exerc* 1997;29:248-58.
- Armstrong RB. Muscle damage and endurance events. *Sports Med* 1986;3:370-81.
- Carlson BM, Faulkner JA. The regeneration of skeletal muscle fibers following injury: a review. *Med Sci Sports Exerc* 1983;15:187-98.
- Chance B, Dait MT, Zhang C, et al. Recovery from exercise-induced desaturation in the quadriceps muscles of elite competitive rowers. *Am J Physiol* 1992;262:C766-75.
- Chance B, Nioka S, Kent J, et al. Time-resolved spectroscopy of hemoglobin and myoglobin in resting and ischemic muscle. *Anal Biochem* 1988;174:698-707.



30. Marras WS, Ferguson SA, Gupta P, et al. The quantification of low back disorder using motion measures—methodology and validation. *Spine* 1999;24:2091–100.
31. Tidball JG. Inflammatory cell response to acute muscle injury. *Med Sci Sports Exerc* 1995;27:1022–32.
32. Devor ST, Faulkner JA. Regeneration of new fibers in muscles of old rats reduces contraction-induced injury. *J Appl Physiol* 1999;87:750–6.
33. White TP, Devor ST. Skeletal muscle regeneration and plasticity of grafts. *Exerc Sport Sci Rev* 1993;21:263–95.
34. Abe K, Matsuo Y, Kadekawa J, et al. Measurement of tissue oxygen consumption in patients with mitochondrial myopathy by noninvasive tissue oximetry. *Neurology* 1997;49:837–41.
35. Bank W, Chance B. An oxidative defect in metabolic myopathies: diagnosis by noninvasive tissue oximetry. *Ann Neurol* 1994;36:830–7.