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Outcome of Back Exercise for Flexion-and Extension-Provoked Low Back Pain

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Abstract

Objectives: To evaluate therapeutic exercise-induced hemodynamic changes in patients with flexion- and extension-provoked low back pain (LBP).

Methods: Men with LBP (n=106) were divided into 2 groups: flexion-provoked LBP (n=61) and extension-provoked LBP (n=45). The Japanese Orthopaedic Association (JOA) score, Visual Analogue Scale (VAS), SF-36 and intramuscular oxygenation measured by near-infrared spectroscopy on the paraspinal muscle during lumbar extension and flexion were evaluated at 2 and 4 weeks.

Results: Deoxygenated hemoglobin during lumbar flexion was significantly lower in flexion-provoked LBP than in extension-provoked LBP. VAS and the SF-36 domains of physical functioning, role physical, bodily pain were significantly improved in the flexion-provoked LBP group. Oxygenated hemoglobin during lumbar extension was increased significantly in the flexion-provoked LBP group. Flexion-provoked LBP patients who exhibited favorable effects from therapeutic exercise also showed better lumbar muscle oxygenation after exercise.

Conclusions: A stretching mechanism in the posterior lumbar elements appeared to have a promising therapeutic effect on extension-provoked LBP. Although LBP attributed to hypoperfusion and oxygenation is involved in flexion-provoked LBP, therapeutic exercise was effective in only 50% of these patients. Evaluating muscle blood flow in flexion-provoked LBP patients is critical for improving the therapeutic effect in these subjects.

Keywords: Low Back Pain (LBP); Therapeutic exercise; Near-infrared spectroscopy

Introduction

Low back pain (LBP) is a common malady that affects approximately 80-90% of people at some point in their lives. [1,2]. The origin of LBP remains uncertain in most cases, and the precise cause of the pain is identified in only 15% of cases even after careful assessment and specific measurements [3]. LBP is typically classified as specific or non-specific, the latter of which is defined as symptoms without a specific cause and accounts for 85-90% of people with LBP [4,5]. From a clinical point of view, disc and/or facet joint-related disorders of the spine are estimated to occur in a high percentage of the LBP population. However, a single structure is not solely responsible for LBP because it is a multifactorial process that depends on constitutional, somatic, psychological, and environmental factors. Thus, a definitive diagnosis cannot be achieved by current radiographic methods, and despite systematic reviews that described several conservative treatment methods including therapeutic exercise for LBP, evidence supporting a specific type of treatment for LBP is lacking [6,7]. Patient-specific treatment based on assessment findings should be included in the management of LBP.

Regarding the use of therapeutic exercise to control LBP, Richardson and Jull hypothesized that controlling back pain and preventing its recurrence can be assisted by enhancing muscle control in the spinal segment [8]. This exercise approach focused on retraining a co-contraction pattern of the deep trunk muscles, the transverse abdominis, and the lumbar multifidus, providing local muscle support for the lumbar motion segment. Several researchers have demonstrated dysfunctions in the multifidus muscle of LBP patients by EMG [9], pathological findings [10], and imaging study [11]. We previously suggested that muscular LBP involves paraspinal muscle blood flow because decreased oxygenated hemoglobin (Oxy-Hb) was detected by near-infrared spectroscopy (NIRS) in the trunk muscles of LBP patients [12].

On the other hand, LBP is often aggravated by flexion or extension movements. The directional nature of instability and clinical presentation is poorly understood in the lumbar spine. O'Sullivan classified non-specific LBP patients on the directional basis of back pain provocation [13]. Flexion-provoked LBP is thought to be induced by lumbar muscle dysfunction in terms of increased muscle tone [13], intramuscular pressure [14], back muscle endurance [15], and muscle fatigability [16], however; the effect of therapies on LBP has not been investigated from the perspective of the directional basis of pain. The purpose of this study was to evaluate the hemodynamic changes in patients with flexion- and extension-provoked LBP before and after therapeutic exercise for LBP.

Methods

This study was carried out over a 2-year period in a single suburban general practice. Ethical approval was given by the Nagoya University Hospital Ethics Committee. Consecutive patients presenting for treatment of their LBP without radiation to the lower limbs were asked to participate in our study. The study subjects were male patients who presented with LBP that had lasted more than 3 month as their main symptom at the outpatient clinic of the Department of

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Orthopedic Surgery. Potential subjects were excluded if they had spinal fracture, tumor, infection, spondylolisthesis, spondylolysis, deformities including scoliosis and lumbar kyphosis greater than 10°, osteophyte formation more than grade 2 of Nathan's classification (0-4) [17] on a radiograph, previous spinal surgery, previous medication for LBP, sciatica, or muscle weakness. Before entering the current study, patients with LBP were assessed by an orthopedic surgeon. Patients who fulfilled the inclusion criteria were classified into the flexion- or extension-provoked LBP group in which back pain was characterized by intermittent pain produced only at end-range in a single direction of lumbar flexion or extension, respectively. Patients with LBP induced by both flexion and extension and patients with LBP induced by neither flexion nor extension were excluded from this study. All patients were classified as flexion dysfunction syndrome or extension dysfunction syndrome based on the McKenzie method assessment, and participated in lumbar flexion and extension exercises derived from McKenzie therapy [18] under the supervision of a single physical therapist. Treatment was aimed at intentionally reproducing the symptoms at end-range as an indicator that the painful structure was being adequately stretched. The therapist who performed the treatment had completed the 4 basic courses arranged by the McKenzie Institute International and had passed a credential examination. The physical therapist performed the exercises at the outpatient clinic once a week and instructed the patients to carry out their exercise routine each day at home for 4 weeks. Patients did not receive medications such as NSAIDs or anti-inflammatory agents for the study period.

Lumbar spine radiographs were obtained for all patients at the first visit. The lordotic angle (L1 to S1 Cobb angle) and the sacral inclination angle (angle between the superior end plate of S1 and a horizontal line) were measured on a lateral lumbar radiograph in a standing position.

Primary outcome variables were observed at the first visit and at 2 and 4 weeks after treatment. These outcome variables included the Japanese Orthopedic Association (JOA) score for back pain (LBP, 0-3) [19], the Visual Analogue Scale (VAS, 0-10 cm horizontal scale), and the SF-36 that consists 36-item general health instrument, which measures 4 domains of physical health: physical functioning (PF), role physical (RP), bodily pain (BP), general health (GH) [20].

Intramuscular oxygenation was evaluated non-invasively using a near-infrared spectrophotometer (OM-220, SHIMADZU Co., Kyoto, Japan). The probe, which was combined with a light transmission fiber and 2 detectors, was positioned with a bandage on the left posterior aspect of the lumbar muscle at the level of the iliac crest. The lumbar spine was extended gradually 45° backward for 15 seconds within a tolerable range of pain in the standing posture, followed by forward bending at 30° for 15 seconds within a tolerable range of pain after a 15-second rest in a neutral standing position. The exercise was started following a stable wave pattern in a resting, standing position for approximately 10 seconds. Oxy-Hb increases in the paraspinal muscle during lumbar extension and deoxygenated hemoglobin (Deoxy-Hb) decreases during lumbar flexion. These values return to those of a resting, standing position quickly after stopping extension or flexion [12]. Bending angle degrees were determined based on a preliminary verification demonstrating that intramuscular hemodynamics had reached a plateau. Parameters such as the relative changes in Oxy-Hb and Deoxy-Hb were compared in arbitrary units. Obese subjects (over 80 kg) and female volunteers were not included because NIRS measurements are difficult to obtain in these subjects.

Means were compared using a Mann-Whitney U test for continuous variables. A 1-way analysis of variance (ANOVA) for repeated measures and Fischer's PLSD as a post hoc test were used to compare the dependent measurements obtained before and after performing the exercise. Differences were considered statistically significant at $p<0.05$. Data were analyzed with StatView 5.0 software (ABACUS, Berkeley, CA).

Results

A total of 106 subjects were assigned to the flexion- and extension-provoked LBP groups. Follow-up information was obtained 2 weeks later for 95 subjects (89.6%) and 4 weeks later for 57 subjects (53.8%). At this point, 39 subjects were missing, and 10 subjects wished to receive a treatment other than therapeutic exercise.

Of the 106 patients selected for treatment, 61 (57.5%) in the flexion-provoked LBP group and 45 (42.5%) in the extension-provoked LBP group have participated in the study.

Significant differences were not found for any of the variables at baseline (Table 1). Although NIRS measurements did not reveal significant differences in the relative change in Oxy-Hb and Deoxy-Hb during lumbar extension between both groups, the relative change in Deoxy-Hb was significantly increased during lumbar flexion in the flexion-provoked LBP group ($p<0.05$).

Of the 57 patients who were followed completely for 4 weeks, 30 (52.6%) patients in the flexion-provoked LBP group and 27 (47.3%) patients in the extension-provoked LBP group were compared at the 2-week and 4-week periods. The average ages were 46.5 ± 12.9 and 43.7 ± 13.2 in the flexion-provoked LBP and 43.7 ± 13.2 extension-provoked LBP groups, respectively, and were not significantly different. VAS improvement was significantly greater in the extension-provoked LBP group than in the flexion-provoked LBP group at 4 weeks ($p<0.05$), but no significant differences were observed at 2 weeks (Figure 1). Similarly, the JOA score improved at 4 weeks in both LBP groups, but significant differences were not observed ($p=0.082$). Regarding the SF-36, significant improvement was found at 4 weeks in the extension-provoked LBP group in PF ($p=0.003$), RP ($p<0.0001$), and BP ($p=0.0017$) compared with the flexion-provoked LBP group (Figure 2).

NIRS measurements revealed that the change in Oxy-Hb during lumbar extension increased significantly at 4 weeks in the flexion-provoked LBP group compared with the extension-provoked LBP

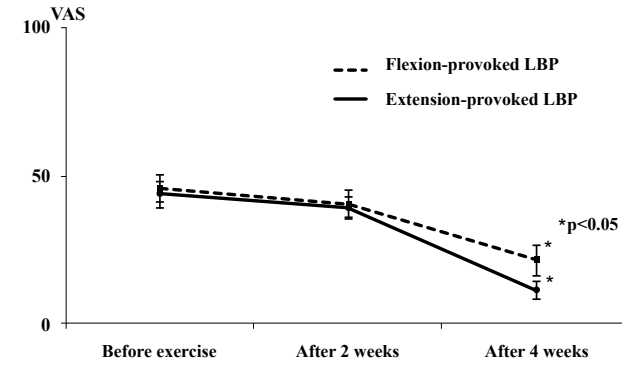

Figure 1: VAS before and after exercise (n=57).
Improvement in VAS was significantly greater in the extension-provoked LBP than in the flexion-provoked LBP at 4 weeks. ($p<0.05$)

(p<0.01), while no significant changes were seen in Deoxy-Hb (Figure 3). Change in Oxy-Hb and Deoxy-Hb during lumbar flexion were not significantly different between the flexion and extension-provoked LBP groups (Figure 4). To investigate the hemodynamic effect on the improvement of LBP in the flexion-provoked LBP (n=30), subjects in which Oxy-Hb increase more than 0.018 arbitrary units (equivalent to 25 percentiles of the increase of Oxy-Hb during lumbar extension at 4 weeks) were defined as the Oxy-Hb-increased group. Oxy-Hb-increased subjects in the flexion-provoked LBP group (n=17, 56.7%) showed a significant VAS improvement from before exercise to 4 weeks after exercise compared with the non-Oxy-Hb-increased group (n=13, 43.3%) (p<0.05) (Figure 5).

	Flexion-provoked LBP (n=61)	Extension-provoked LBP (n=45)
Age (years)	43.9 ± 13.9	42.9 ± 14.0
Duration of symptom (day)	52.0 ± 59.6	41.4 ± 35.4
Height (cm)	168.7 ± 5.4	168.6 ± 6.3
Weight (kg)	66.1 ± 10.0	64.4 ± 8.6
Smoking (/day)	8.1 ± 12.3	10.8 ± 10.9
VAS	40.7 ± 28.3	41.8 ± 28.2
JOA score (LBP; 0-3)	1.2 ± 0.6	1.3 ± 0.6
SF-36 PF	78.7 ± 19.5	77.3 ± 27.1
RP	81.2 ± 22.9	87.2 ± 23.8
BP	48.3 ± 19.6	54.3 ± 23.6
GH	56.4 ± 15.5	59.6 ± 18.8
L1-S1lordotic angle (degree)	34.2 ± 7.3	34.1 ± 7.6
S1 inclination angle (degree)	27.6 ± 6.3	25.3 ± 6.2

VAS¬=Visual Analogue Scale; JOA=Japanese Orthopaedic Association; SF-36; PF=Physical Functioning; RP=Role Physical; BP=Bodily Pain; GH=General Health
Values represent means ± S.D
No statistically significant differences between groups were shown between any variables at baseline.

Table 1: Demographic and initial assessment data.

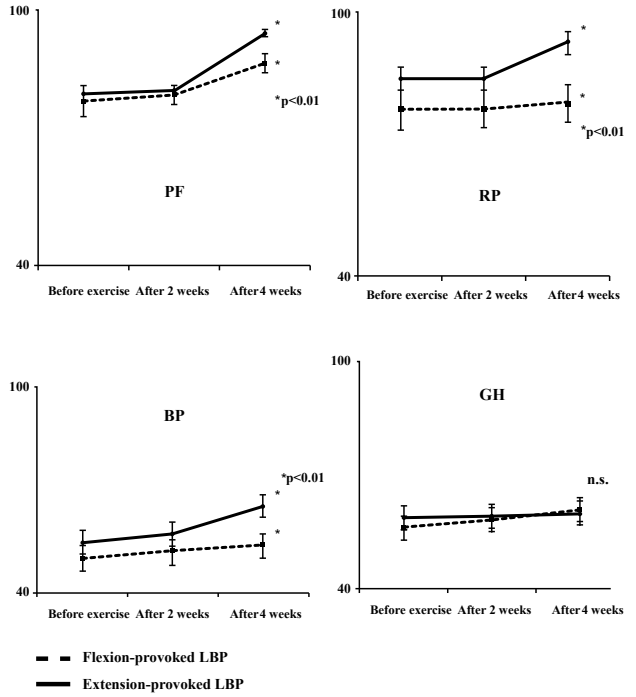

Figure 2: SF-36 before and after exercise (n=57).
Significant improvement was found at 4 weeks in the extension-provoked LBP in PF (p=0.003), RP (p<0.0001), BP (p=0.0017) compared with the flexion-provoked LBP.
Physical Functioning (PF), Role Physical (RP), Bodily Pain (BP), General Health (GH)

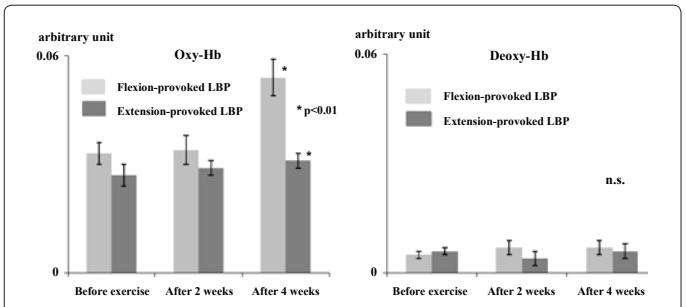

Figure 3: Oxy-Hb and Deoxy-Hb change during lumbar extension before and after exercise.
Oxy-Hb during lumbar extension increased significantly at 4 weeks in the flexion-provoked LBP compared with the extension-provoked LBP. (p<0.01)

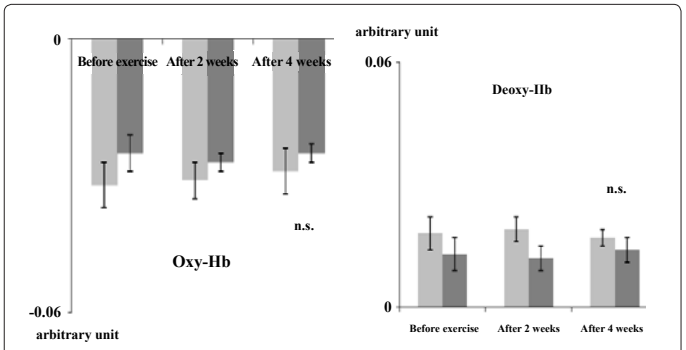

Figure 4: Oxy-Hb and Deoxy-Hb change during lumbar flexion before and after exercise.
Both Oxy-Hb and Deoxy-Hb change during lumbar flexion had no significant differences between the flexion and the extension-provoked LBP.

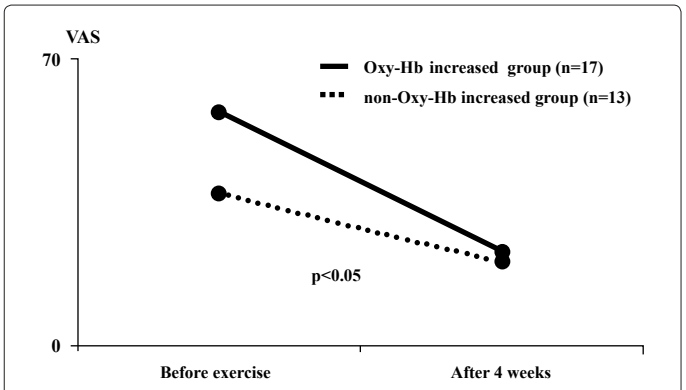

Figure 5: VAS before and after exercise according to Oxy-Hb increase during lumbar extension in the flexion-provoked LBP.
The Oxy-Hb increased group in the flexion-provoked LBP (n=17, 56.7%) significantly improved compared with the non-Oxy-Hb increased group (n=13, 43.3%). (p<0.05)

Discussion

NIRS measurements obtained before treatment demonstrated that the increase in Deoxy-Hb was significantly greater in the flexion-provoked LBP group than in the extension-provoked LBP group. These findings support the study of Konnno et al. [14], which showed that intramuscular pressure increases and blood flow decreases as spinal alignment changes from lordosis to kyphosis. O'Sullivan et al. [13] classified flexion strain pain disorders as disorders in which LBP is reproduced by sustained flexion of the lumbar spine, is relieved by

extension of the lumbar spine, and is associated with loss of the lumbar multifidus muscles rather than spinal mobility impairment. A muscular component is presumably more predominant in flexion-provoked LBP than in extension-provoked LBP. Kunogi [21] stated that LBP with flexion disturbance usually involves backache of discal, ligamentous, or muscular origin and that LBP with extension disturbance involves backache from facet joints, including spondylolisthesis and spondylolysis. However, no reports have described the pathophysiological and therapeutic effects in motion-induced LBP. In muscular LBP and tissue oxygenation during isometric contraction of the multifidus muscle, increases in intramuscular pressure result in tissue hypoxia, and tissue hypoxia responsible for muscular fatigue and loss of function have been addressed [22]. Flexion-provoked LBP is presumably associated with dysfunction of the back muscles, which is characterized by an increase in pressure and fatigability in a manner similar to compartment syndrome [14,16]. Low back intramuscular pressure, which represents capillary blood flow, tissue oxygenation, and muscle function, has been measured in LBP patients [23,24]. Muscle contraction leads to increased intramuscular pressure and significantly decreased pressure in the paravertebral muscles [25]; however, several studies have been skeptical of intramuscular pressure and hypoxia of the multifidus [22,26]. Because satisfactory evidence is lacking about tissue oxygenation and intramuscular pressure, factors other than intramuscular pressure may affect microcirculation and pain generation [27].

Tissue oxygenation and oxygen use by the paraspinal muscles has been investigated only sporadically to elucidate the mechanism of LBP [28-30]. A muscle's oxygen levels progressively decrease during incremental exercise [31]. Trained muscles show lower fatigue and a more efficient use of oxygen; training increases the oxidative metabolism of muscles [32]. Therefore a limited physical capability of oxygen consumption in the paravertebral muscles may be consistent with the alteration in aerobic muscle performance associated with LBP. Kovacs et al. used NIRS to demonstrate that patients with muscular back pain did not use available oxygen during exercise compared with normal subjects [28]. Lumbar paravertebral muscles are dominated by slow twitch fibers, each of which is in contact with a considerable number of capillaries, enabling a better blood flow distribution to the area of local demand during submaximal contraction [33]. The paravertebral muscle can maintain a steady-state level of oxygenation during active lumbar extension [26], which leads to increasing Oxy-Hb by increasing the bulk muscle volume in a less extensible fascia and increasing blood flow to the working skeletal muscle accompanied by the cardiac output response. Regarding localized oxygen use in healthy subjects and those with LBP, Kovacs et al. speculated that the inability to consume oxygen is attributed to mitochondrial damage and to the decreased oxidative enzyme activity of the cell because of the breakdown in the myofibrils of the muscle [28]. Kell and Bhambhani used NIRS to demonstrate that the initial increase in muscle blood volume and oxygenation at the onset of exercise in the lumbar spine is important for oxygen availability and susceptibility to muscle fatigue and pain [34]. The rapid increase of muscle blood flow during lumbar extension is associated with blood distribution to the active motor unit [35,36] and/or increased blood flow in the microcirculation [25]. The paravertebral muscles, which are dominated by fatigue-resistant slow twitch fibers, can maintain the blood supply at higher contraction intensities because of a greater capillary density than other skeletal muscles [33]. Previous studies have suggested that decreased muscle oxygenation is one of the causes of muscle fatigue [37,38], and that muscles can be trained to improve their function and oxygen use [32,39].

On the other hand, exercise therapy is widely used and is the most common conservative intervention for LBP [40,41]. However, how to select the appropriate form of exercise therapy from among aerobic exercises, strengthening exercises, coordination exercises, and specific exercises for LBP treatment is unclear for the clinician [42]. Clear guidance whether it is more effective to administer exercises on an individual basis or in groups are lacking. This is attributable to uncertainty regarding how exercise effects are mediated by exercise type: furthermore, the optimal type of exercise for LBP is not known because the effects of specific exercises have not been systematically assessed [42]. It was suggested that McKenzie therapy, which was employed in this study, was more effective than other strength training, spinal mobilization, and general mobility exercises at short-term follow-up [43]. The subjects in the current study had dysfunction syndrome, which is characterized by intermittent pain produced only at end-range in a single direction of restricted movement, including overstretching of soft tissues that are shortened or contain contracted scar tissue [44]. For dysfunction syndrome, symptoms elimination requires treatment aimed at intentionally reproducing the symptoms at end-range, because adequate stretching of the painful structure has a beneficial effect on pain. Although several investigations showed that muscle stretching affects muscle function and blood flow in the lower limbs [45,46], no previous studies have objectively evaluated the lumbar muscle in flexion- and extension-provoked LBP after exercise therapy. The superior therapeutic effect on extension-provoked LBP showed that the passive motion generated by the McKenzie method improved contracted joint capsules and soft tissues. Yang and King used data obtained from tests on isolated spinal segments of the lumbar spine to demonstrate that the facet load increases with increasing extension moment and excessive facet loads stretch the joint capsule and can be a cause of LBP [47]. Adams and Hutton noted that as the extension of an intervertebral joint increase, the compressive force transmitted across the facet joints increases and hyperextension movements could cause spondylosis and LBP [48]. Because extension-provoked LBP seems predominantly to involve pain derived from the facet joints, McKenzie therapy may be a more valid therapeutic indication for extension-provoked LBP than for flexion-provoked LBP.

The effects of therapeutic exercise on flexion-provoked LBP were inferior to those of extension-provoked LBP. Because the NIRS measurements in the present study suggested muscular involvement in flexion-provoked LBP, we were prompted to evaluate hemodynamic improvement by NIRS and pain relief after therapeutic exercise in flexion-provoked LBP. Those flexion-provoked LBP patients who showed favorable effects from therapeutic exercise had presented with better lumbar muscle oxygenation after exercise. The results indicate that therapeutic exercise has a considerable impact on improved muscle blood flow, and suggests that muscular LBP involves back pain affected by lumbar muscle blood flow and oxygenation. However, a post-therapeutic improvement of the muscle blood flow was observed in 50% of the flexion-provoked LBP cases. Hypoperfusion and oxygenation in the trunk muscle do not always cause flexion-provoked LBP. Accurate awareness of the potential for improvement in muscle blood flow after exercise in individuals with flexion-provoked LBP would be important for better outcome of the therapeutic effects.

The main limitation of the current investigation was the small number of patients included and short observational periods. Further longitudinal studies with larger samples are required to clarify the association between lumbar movement and LBP, the effect of therapeutic exercise.

In conclusion, a stretch mechanism in the posterior lumbar elements such as the capsules of the facet joints and soft tissues appears to have a promising therapeutic effect on extension-provoked LBP. Although LBP attributed to hypoperfusion and oxygenation is involved in flexion-provoked LBP, therapeutic exercise is effective in only about 50% of these subjects. Evaluating the muscle blood flow in individuals with flexion-provoked LBP is critical for improving the therapeutic effect in flexion-provoked LBP.

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