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Multifidus Muscle Recovery Is Not Automatic After Resolution of Acute, First-Episode Low Back Pain

[Exercise and Functional Testing]

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Outline

Graphics

Abstract^

Study Design: A clinical study was conducted on 39 patients with acute, first-episode, unilateral low back pain and unilateral, segmental inhibition of the multifidus muscle. Patients were allocated randomly to a control or treatment group.

Objectives: To document the natural course of lumbar multifidus recovery and to evaluate the effectiveness of specific, localized, exercise therapy on muscle recovery.

Summary of Background Data: Acute low back pain usually resolves spontaneously, but the recurrence rate is high. Inhibition of multifidus occurs with acute, first-episode, low back pain, and pathologic changes in this muscle have been linked with poor outcome and recurrence of symptoms.

Methods: Patients in group 1 received medical treatment only. Patients in group 2 received medical treatment and specific, localized, exercise therapy. Outcome measures for both groups included 4 weekly assessments of pain, disability, range of motion, and size of the multifidus cross-sectional area. Independent examiners were blinded to group allocation. Patients were reassessed at a 10-week follow-up examination.

Results: Multifidus muscle recovery was not spontaneous on remission of painful symptoms in patients in group 1. Muscle recovery was more rapid and more complete in patients in group 2 who received exercise therapy ($P = 0.0001$). Other outcome measurements were similar for the two groups at the 4-week examination. Although they resumed normal levels of activity, patients in group 1 still had decreased multifidus muscle size at the 10-week follow-up examination.

Conclusions: Multifidus muscle recovery is not spontaneous on remission of painful symptoms. Lack of localized, muscle support may be one reason for the high recurrence rate of low back pain following the initial episode.

An episode of acute low back pain (LBP) resolves in only 2-4 weeks for 90% of patients.[4-6] Yet in the

year following a first episode of LBP, the pain recurs in a staggering 60-80% of patients.[3,41] These recurrences incur major human and financial costs.[8] Successful prevention of recurrent LBP remains a challenge.

There is not a complete understanding of this vulnerability to recurrence. Instability of the lumbar motion segment is considered to be one important cause.[24] Panjabi [25,26] proposed that instability is a loss of control or excessive motion in the spinal segment's neutral zone, which is associated with injury, degenerative disc disease, and muscle weakness. It has been shown in *in vitro* studies that muscles can provide segmental stabilization by controlling motion in the neutral zone, and the neutral zone can be returned to within physiologic limits by effective muscle control.[9,27,42]

All muscles that traverse the lumbar region have the potential to impart stability to the lumbar spine.[2] Some muscles are more specialized than others, however, and they have characteristics that enable them to contribute more stability. Recent *in vitro*, biomechanical studies have shown that the lumbar multifidus is an important muscle for lumbar segmental stability. It is able to provide segmental stiffness and control motion in the neutral zone.[9,27,37,42] Wilke et al [42] showed that when compared with other muscles in close proximity to the L4-L5, the multifidus muscle contributed two thirds of the increased stiffness imparted by contraction of the muscles. The local stabilizing effects of the multifidus muscle on the lumbar motion segment has been confirmed further in recent animal research.[18] For these reasons, any injury to the multifidus muscle could be expected to have direct effects on lumbar segmental stability.

There is evidence for a strong relationship between multifidus muscle dysfunction and poor functional outcome and recurrence of LBP after disc surgery.[30,36] In two studies, patients were allocated to either a positive or negative outcome group on the basis of functional outcome. Results of multifidus muscles biopsies of the patients with poor outcome showed muscle atrophy and an increase in frequency of pathologic changes in the multifidus, especially for moth-eaten type I fibers. In the positive outcome group, the internal type I muscle fiber changes decreased.[30] Functional recovery following disc surgery was associated with curtailment of structural abnormalities in the multifidus muscle. Sihvonen et al [36] proposed that poor functional outcome was secondary to loss of functional muscle support, disturbed segmental mobility, and increased mechanical strain and disability. It is important to note that structural multifidus changes are reversible with adequate surgical and physiotherapeutic intervention.[30]

Because internal structural changes are present in type I multifidus fibers in patients who have experienced pain for only 3 weeks, it may be important to examine for multifidus muscle dysfunction in patients with acute LBP.[7] Multifidus muscle dysfunction in patients with acute LBP also could be related to outcome and recurrence of LBP symptoms.

Recent studies have shown localized segmental dysfunction of the multifidus muscle to occur after a first episode of acute or subacute, unilateral LBP.[14] Rapid multifidus atrophy was demonstrated

ipsilateral to the location of pain with ultrasound imaging.[14] Although the pain associated with an episode of acute LBP may resolve within 2-4 weeks, it is unknown whether multifidus muscle spontaneously recovers at that time.

A prospective clinical study was undertaken to investigate multifidus recovery. Patients with first-episode LBP who had right-left asymmetry of multifidus cross-sectional area (CSA) were studied. Asymmetry between each side of the lumbar spine had to be greater than 11% for patients to be considered for this study. This limit represents two standard deviations above the mean difference in multifidus CSA measured in a previous study of healthy individuals.[14] Patients were allocated randomly to a control or treatment group to investigate the natural course of multifidus muscle recovery and to evaluate the effectiveness of a specific, localized, exercise program. The present study considers the recovery in the immediate, postinjury period (10 weeks). Nine-month and 1-year follow-up examinations were in progress at the time of this report to monitor any long-term effects on recurrence of pain.

[black small square] Methods^

Subjects. Patients were recruited from an accident and emergency department in a hospital during a 6-month period. Men and women were eligible for the study in the first instance if they were aged 18-45 years and were experiencing their first episode of unilateral, mechanical LBP for less than 3 weeks. Pain had to be located between T12 and the gluteal fold (with or without pain radiation into the lower limb) and had to restrict range of lumbar motion. All patients underwent a full medical screening examination, which included a lumbar x-ray and a neurologic examination. Exclusion criteria were previous history of LBP or injury, previous lumbar surgery, spinal abnormalities indicated on radiographs, neuromuscular or joint disease, reflex and/or motor signs of nerve root compression or cauda equina compression, evidence of systemic disease, carcinoma or organ disease, pregnancy, and any sports or fitness training involving the low back muscles done in the past 3 months. Using these criteria, 14 patients were excluded. Reasons for their exclusion included unwillingness to undergo spinal lumbar x-ray examination (two patients), abnormalities found on radiographs (four patients; pars defect, lumbarization, spina bifida occulta, and scoliosis of 24°), no restriction of lumbar range of motion (three patients), neurologic deficit (three patients), and pregnancy (two patients).

Forty-one patients were accepted provisionally into the study. All patients gave their consent, and the study was approved by the Medical Ethical Review Committees of the University of Queensland, and the Mater Adult Hospital, Brisbane, Australia. Random assignment to the control (group 1, medical management) or the treatment group (group 2, medical management and specific, exercise therapy) was achieved by selecting the group number (one or two) from sealed, shuffled envelopes. Twenty patients were allocated randomly into group 1, and 21 patients were allocated into group 2. There were 10 men and 10 women in group 1, and 8 men and 13 women in group 2. The mean age of patients in group 1 was 31 years ± 7.9 years (range, 17-45 years), and the mean age for patients in group 2 was 30.9 years ± 6.5 years (range, 22-44 years).

Assessment Procedures. Assessments were performed by two independent examiners who were

blinded to group allocation and patient presentation. The following assessment measures were used.

1. Pain. The location, quality, and perceived intensity of pain were assessed using the McGill Pain Questionnaire (MPQ), visual analogue scales (VAS), and daily pain diaries.[15,21] All components of the MPQ were calculated, including total, sensory, affective, evaluative, and miscellaneous pain rating indices (PRI); the number of words chosen, and the present pain intensity. Pain diaries were used to record the number of analgesics taken.
2. Disability. The Roland Morris Disability Index was used to assess disability.[33]
3. Range of Motion. Lumbar motion was measured using a two-inclinometer method for lumbar flexion and extension and a single-inclinometer method for lumbar lateral flexion.[20,28] Oil-filled Rippstein goniometers were used for all measurements of lumbar motion. Straight leg raise (SLR) also was measured using the oil-filled goniometer.[29]
4. Habitual activity levels. All patients completed a habitual activity questionnaire to determine premorbid activity levels in the areas of work, sport, and leisure.[1]
5. Muscle CSA. Measurement of multifidus CSA using real-time ultrasound imaging has been described in detail in previous reports.[11] All ultrasound imaging was performed by the blinded ultrasonographer using real-time ultrasound apparatus (Toshiba Sonolayer V SSA-100A, Toshiba Medical Systems, Japan) equipped with a 5-MHz convex array transducer. Bilateral measurements of multifidus CSA were made at each vertebral level from L2 to S1. The validity of the ultrasound to measure the CSA of multifidus at different lumbar levels was established by comparing these measurements obtained with ultrasound with those obtained by magnetic resonance imaging (MRI) in healthy patients.[12] Repeatability of the ultrasound technique has been demonstrated started, and interobserver reliability with this technique also has been reported.[11,14,12]

All patients were assessed initially to provide baseline data, and then were reassessed weekly for 4 weeks. Patients were deemed symptom-free when they could perform all functional activities with no difficulty, when the pain rating measured on the MPQ and visual analogue scale was very low (either 1 or 0), and when lumbar movement and SLR examinations elicited no pain.[6] Patients who did not attain full pain-free function were removed from the study at 4 weeks and, for ethical reasons, received complete physiotherapy treatment. The remaining patients were reassessed during the 10th week of the study, after they had experienced 6 weeks of pain-free activity. At this time, habitual activity levels were reassessed to determine if premorbid activity levels had been resumed during the previous 6-week period, and muscle CSA was measured.

Intervention and Patient Treatment.[^]

Medical Management. Medical management of the low back pain included advice on bed rest and absence from work and prescription of medication. Minimal bed rest (1-3 days) and only minor analgesics were prescribed. These analgesics included aspirin; paracetamol (8-mg codeine tablets); combinations of low doses of codeine and aspirin (as many as eight tablets per day); nonsteroidal, anti-inflammatory agents; Digesic (Dista Products Pty. Ltd., Australia); and Capadex (Fawns and McAllen Pty. Ltd., Victoria, Australia). Prescription of valium was also allowable.

Exercise Therapy. The therapeutic exercises were designed to re-educate the multifidus muscle in its

stabilizing role. They involved facilitating an active, isometric multifidus contraction in co-contraction with the deep abdominal muscles. Patients performed the contraction in the standing position with the lumbar spine in a neutral position. This specific training was based on the approach of Jull and Richardson.[17,31] The exercise was enhanced by using real-time ultrasound imaging to ensure that the inhibited multifidus was activated specifically.[13]

Statistical Analysis. Comparability of baseline measurements between the groups was assessed using one-way analysis of variance (ANOVA) to examine differences in all baseline measurements. Analysis of variance also was used to examine differences between groups over time for all outcome measures used. For ultrasound imaging data, the percentage difference between the painful and nonpainful side was calculated for each vertebral level measured. Analysis of muscle recovery was conducted using the data from the most affected vertebral level (*i.e.*, the vertebral level with the largest percentage difference between sides). If patients did not have localized, segmental, muscle asymmetry at the time of this first assessment, their data were not included in further analyses. Analysis of variance also was used to examine the data from the other vertebral levels measured during the first week. Correlation analyses were done to test for any relationships among muscle recovery, pain, disability, and range of motion.

[black small square] Results^

Study Sample^

Of the 41 patients included in the study, four patients (all from group 1) did not achieve pain-free status after 4 weeks. They were excluded from further participation in the study, but the data for the first 4 weeks have been included. Two patients (one from each group) did not have localized, multifidus asymmetry according to ultrasound images. The data from these patients have not been included in the analyses presented. One patient missed the 10-week follow-up examination because of the illness of a family member. The drop-out rate after 10 weeks, therefore, was 2.4%.

Baseline Characteristics: Group Comparability^

Comparability between groups was shown to be satisfactory. The mean ages, heights, and weights for patients in group 1 and group 2 were 30.9 years and 30.65 years ($F_{(1,37)} = 0.01$; $P = 0.918$); 173.3 cm and 170.1 cm ($F_{(1,37)} = 1.28$; $P = 0.265$), and 73.53 kg and 71.05 kg ($F_{(1,37)} = 0.23$; $P = 0.633$), respectively. Premorbid habitual activity levels were not significantly different for the two groups. The mean duration of symptoms for patients in group 1 and group 2 was 9.16 days and 8.10 days, respectively ($F_{(1,37)} = 0.19$; $P = 0.665$). Further, no significant differences were found in baseline measurements between groups for any of the components of the MPQ or the VAS, any of the lumbar movements measured, or the SLR. The only baseline measure that just reached significance was the disability score obtained from the RMQ (group 1 = 13.6, group 2 = 10.3; $F_{(1,37)} = 4.82$; $P = 0.045$). On a clinical basis, however, three points does not represent a large difference, and the mean overall score was similar to that reported by Roland and Morris,[33] which was 11.4 for patients with LBP. The baseline disability scores therefore can be considered representative of a typical group of patients with

acute LBP. Medication use and area of pain also were similar for the two groups. Actual baseline scores for the MPQ (total PRI), the VAS, and the RMQ are shown in Figures 1-3. Baseline lumbar mobility and SLR values are presented in Table 1.

Figure 1. Pain scores obtained using the visual analogue scale for patients in group 1 (control group) and group 2 (exercise group) for the baseline measurement (week 0) and at weeks 1-4 of the study.

Figure 2. Pain scores from the Total pain rating index of the McGill Pain Questionnaire for patients in group 1 (control group) and group 2(exercise group) for the baseline measure (week 0) and for weeks 1-4 of the study.

Figure 3. Disability scores obtained from the Roland Morris Disability Index for patients in group 1 (control group) and group 2 (exercise group) for the baseline measure (week 0) and for weeks 1-4 of the study.

Table 1. Lumbar Mobility in the Sagittal and Frontal Planes, and SLR Values Recorded for Baseline Measurements and at the End of the 4-Week Assessment Period

Compliance With Treatment^

All patients in group 1 and 2 received the assigned therapy. Patients in group 2 recorded their performance of exercises done at home on sheets that were collected by the authors each week.

Primary Outcomes for Weeks 1-4.^

1. Pain analysis over time. Pain decreased significantly for patients in both groups over time, and complete recovery from pain occurred in all but four patients (Figures 1, 2). There was no group by week interaction for any of the pain measures, indicating that over time the decrease in pain was parallel in both groups (total PRI, $F_{(4,148)} = 0.37$, $P = 0.83$; VAS, $F_{(4,148)} = 0.16$, $P = 0.96$). No significant difference was found between groups at the end of the 4-week period, indicating that although four patients in group 2 had some residual painful symptoms, almost total remission of painful symptoms occurred in patients from both groups overall.
2. Disability analysis over time. Similar to the results of the pain analysis, disability was found to have decreased greatly over time for patients in both groups (Figure 3). Analysis of variance showed that there was no week by group interaction ($F_{(4,148)} = 0.63$; $P = 0.64$), indicating that over time the decrease in disability was parallel in both groups. Further, disability at the end of the 4-week period

was minimal (group 1 mean score, 2.3, group 2 mean score, 0) and was not significantly different for the two groups.

3. Range of motion analysis over time. Range of motion increased over time in all directions measured for patients in both groups. Lumbar mobility in the sagittal and frontal planes and SLR values recorded for baseline measurements and at the end of the 4-week period are shown in Table 1. Lumbar range of motion and SLR measurements for group 1 were not significantly different from those of group 2 at the end of the 4-week period.

4. Ultrasound imaging. Ultrasound imaging revealed that asymmetry of muscle size was limited to one vertebral level in all patients except one in whom marked asymmetry was seen at two adjacent vertebral levels (L5 and S1). Diminished muscle size was seen on the patients' nominated painful side in all cases. The difference between the sides at the most affected vertebral level was expressed as a percentage of the CSA for the unaffected side at that level. The mean of these percentages was 24.03% ± 8.67% (range, 12-46%). The vertebral level with the greatest asymmetry was L5 in 34 patients, S1 in four patients, and L4 in one patient. The results of ultrasound imaging for the majority of patients, those whose asymmetry of their sides occurred at L5, are illustrated in Figure 4 to show the localized effect of inhibition. Results of Duncan's multiple range test confirmed that asymmetry at L5 was significantly different from that at L2-L4 and S1. Patients with symptoms at other vertebral levels demonstrated the same phenomenon.

Figure 4. Ultrasound imaging results showing the difference between sides (expressed as a percentage) in cross-sectional area of the multifidus muscle for vertebral levels L2-S1 in 34 patients who demonstrated muscle asymmetry at the L5.

Muscle recovery over the 4-week period for groups 1 and 2 is shown in Figure 5. Multifidus muscle recovery was more rapid and more complete in patients who received specific, localized exercise therapy. Analysis of variance revealed that total muscle recovery ($F_{(1,140)} = 103.5$; $P = 0.0001$) and weekly muscle recovery ($F_{(4,140)} = 34.75$; $P = 0.0001$) differed significantly between groups.

Figure 5. Ultrasound imaging results showing multifidus muscle recovery for patients in groups 1 (control group) and group 2 (exercise group) for the baseline measure (week 0), weeks 1-4 of the study, and the 10-week follow-up examination. Muscle size is presented as the difference between sides (expressed as a percentage) in cross-sectional area (CSA) at the most affected vertebral level.

Correlations Between Outcome Variables^

Correlation analyses for both groups showed that pain (total PRI) was correlated with disability (group 1, r = 0.83, $P = 0.0001$; group 2, r = 0.85, $P = 0.0001$), indicating that pain and disability decreased simultaneously in the patients with acute LBP. Results of assessments of patients from group 1 showed that pain and disability were not correlated with muscle recovery. Muscle recovery did not

occur at the time of remission of pain and disability in the control group.

10-Week Follow-up Examination^

Analysis of variance was conducted on the data from the 10-week follow-up examination of 35 patients. Results showed that multifidus CSA measurements at the most affected vertebral level were not significantly different from the measurements made at the end of the 4-week period for either group ($F_{(1,31)} = 1.69$; $P = 0.20$). The mean percentage by which sides differed for patients in group 1 at week 4 was 16.84% ± 9.26%, and at the 10-week assessment was 14.02% ± 6.31%. For group 2, the week 4 mean was 0.71% ± 2.49%. At the 10-week follow-up examination this mean percentage was 0.24% ± 3.29%.

Habitual activity baseline data (premorbid activity) was compared with data taken during the six weeks of pain-free activity that some patients experienced between assessments. There was no significant difference between premorbid activity levels and the activity levels achieved between assessments for either group. The premorbid total activity scores and the those for the six-week pain-free period were 7.81 and 7.44, respectively, for group 1 ($F_{(1,32)} = 0.42$; $P = 0.52$) and 8.45 and 8.68, respectively, for group 2 ($F_{(1,38)} = 0.28$; $P = 0.60$).

[black small square] Discussion^

The most important finding of the current study is that multifidus muscle recovery does not occur spontaneously on remission of painful symptoms. Possible mechanisms for the decrease in muscle size are reflex inhibition or inhibition from perceived pain via a long, loop reflex.[14] As the indirect effects of inhibition (decreased muscle size) were seen in the absence of pain, the most likely mechanism by which muscles decreased was reflex inhibition. This phenomenon is very similar to previously reported reflex inhibition of surrounding musculature in peripheral joints such as the knee.[40]

Reflex inhibition occurs when sensory stimuli impede the voluntary activation of a muscle.[23] Reflex inhibition causes weakness and rapid muscle atrophy.[38] It has been reported that reflex inhibition hampers alpha motor neurone activity in the anterior horn of the spinal cord, but findings from He et al [10] may implicate the gamma motor neurone system in inflamed joints.[39] Reflex inhibition can occur in the absence of pain, and its persistence has been demonstrated using electromyography in patients 2, 3, and 12 weeks after knee injury or surgery regardless of whether patients were pain-free and fully weight-bearing.[19,35,40] These results are similar to those found for the patients in group 1 in this study who did not receive specific exercise and in whom a persistent decrease in multifidus muscle size was seen at the 10-week follow-up examination.

The clinical significance of this finding is that although these patients with LBP appeared fully recovered after their initial acute pain subsided, their muscle system certainly had not recovered. Results of the initial 4-week examination of patients in group 1 and group 2 showed that there were no differences between groups in any of the outcome measures of pain, disability, or range of motion. The

only difference was in muscle recovery. Further, even after 6 weeks of normal pain-free activity (at the 10-week follow-up assessment), the multifidus muscles of the patients in group 1 had not improved significantly. The multifidus muscle has been shown to be an important provider of segmental, spinal stability, and dysfunction in the multifidus is correlated with poor functional outcome in patients who undergo disc surgery.[9,18,27,30,36,37,42] The patients with acute LBP in this study whose pain had resolved had resumed participating in a normal level of activity, but it is possible that they did so with a predisposition to further injury and recurrence of LBP.

The results of examination of patients in group 2 showed that specific, localized, holding contractions of the lumbar multifidus helped to restore symmetry of muscle size. The technique stimulated early, localized, segmental activation and isometric holding (endurance) of the multifidus muscle within the co-contraction pattern with the deep abdominal muscles.[17] Several reports of general stabilization training involving co-contraction of trunk muscles have appeared in the literature, and the efficacy of this approach at least in short-term treatment has been demonstrated in patients with herniated lumbar discs.[16,22,32,34] The approach used in this study focused very specifically on nonresisted, isometric co-contraction of multifidus with the deep abdominal muscles; the occurrence of a multifidus contraction was verified in the treatment session using feedback from real-time ultrasound imaging. Multifidus inhibition in this study was related to one vertebral segment in patients with first-episode acute LBP. It is proposed that this localized physical training, which can restore muscle size at the segmental level, may be a necessary first stage of rehabilitation, before more generalized stabilization training, so that muscle control at the segmental level might be better assured.

[black small square] Conclusion^

Multifidus recovery from inhibition associated with first-episode, acute LBP does not occur automatically with resolution of pain and disability. Even when functional levels of activity returned to normal (assessed at the 10-week follow-up examination), muscle size did not return to normal. This may be one factor that contributes to the high recurrence rate of LBP after an acute episode because a high proportion of patients may have a deficit in their lumbar muscular stabilizing capacity despite their lack of pain. This inhibition can be reversed with exercises that focus on activating the multifidus of the segmental level. Further follow-up studies on the recurrence rate of LBP in patients in this study will determine if early muscle activation and return of muscle size alters the incidence of LBP recurrence.

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