

Rehabilitation of a Young Athlete With Extension-Based Low Back Pain Addressing Motor-Control Impairments and Central Sensitization

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Objective: To describe the conservative management of a young athlete with extension-based (EB) low back pain (LBP).

Background: We present the case of a 15-year-old female high school gymnast with a 4-year history of EB LBP. Magnetic resonance imaging revealed a healed spondylolysis and significant atrophy with fatty infiltrate of the lumbar multifidi muscles (LMM). She had several courses of outpatient orthopaedic rehabilitation that focused on core muscle strengthening (improving activation and strength of the LMM and transversus abdominus muscle in a neutral pelvic position) without long-lasting improvement. She was unable to tolerate higher levels of training or compete.

Differential Diagnosis: The LMM are rich in muscle spindles and provide continuous feedback to the central nervous system about body position. Atrophy and fatty infiltrate of the LMM can compromise neuromuscular function and contribute to dysfunctional movement patterns that place a greater demand on lumbar spine structures. Ongoing motor-control impairments perpetuate nociceptive input, leading to central sensitization.

Treatment: The athlete had difficulty controlling trunk extension during sport-specific activities; she moved early and to a greater extent in the lumbar spine. The aim of the treatment was to teach the athlete how to control her tendency to overload her lumbar spine when bending backward, thus reducing nociceptive input from lumbar spine structures and desensitizing the nervous system.

Uniqueness: Treating EB LBP by addressing motor-control impairments and cognitive-affective factors as opposed to core strengthening.

Conclusions: Activity modification, bracing, and traditional core-strengthening exercises may not be the most appropriate treatment for athletes experiencing EB LBP. Addressing cognitive-affective factors in addition to correcting maladaptive motor behavior and moving in a pain-free range reduces nociceptive input, desensitizes the nervous system, and allows athletes to gain control over their pain.

Key Words: spondylolysis, facet syndrome, lumbar multifidi muscle, core strengthening, motor-control training

Low back pain (LBP) is estimated to occur in 10% to 15% of young athletes, but the prevalence in some sports that require repetitive lumbar extension may be higher.¹ The condition occurs frequently in artistic gymnasts (50%) and rhythmic gymnasts (86%).^{2,3} Approximately half of the young athletes who present with extension-based (EB) LBP have a fracture in the pars interarticularis (spondylolysis).⁴ Management of spondylolysis includes activity modification, bracing, physical therapy, use of a bone stimulator, and sometimes surgery.^{1,5} The primary goal has traditionally been to achieve bony healing.⁶ In some cases, athletes continue to have pain despite a healed fracture or have complete resolution of symptoms with a nonunion.⁶ In addition, a subgroup of athletes with EB LBP pain have negative imaging for a spondylolysis or stress reaction but a clinical presentation identical to those with positive imaging.^{7,8} These conditions may involve the posterior elements of the spine and are often referred to as spondylogenic back pain, hyperlordotic back pain, mechanical LBP, or lumbar facet syndrome.⁹

The lumbar multifidi muscles (LMM) play an important role in stabilizing the lumbar spine. Compared with all lumbar muscles, the LMM are shorter, are more compact, and have a large cross-sectional area and short muscle

fibers.¹⁰ This morphology allows the LMM to generate very large forces over a small distance and makes the LMM well suited for stability as opposed to mobility.¹¹ The LMM are rich in muscle spindles, respond to changes in relative orientations of body parts or vertebrae, and provide continuous feedback to the central nervous system (CNS) about body position.¹² Spinal stability is achieved by the LMM working synergistically with the abdominal muscles, pelvic floor, and diaphragm.^{13,14} The LMM have been shown to atrophy after the first episode of LBP and do not recover automatically.¹⁵ A number of researchers^{16–19} have found greater LMM fat content in patients with chronic LBP compared with asymptomatic volunteers. Fatty infiltration is thought to be a result of local dysfunction rather than disuse.²⁰ Injury to the intervertebral disc or zygapophyseal joints through shared innervation causes a reflex inhibition of the LMM that is followed by atrophy and fatty infiltrate.²¹ Distorted input from the dysfunctional LMM to the CNS can alter proprioception and impair the ability to control trunk motion, thus placing a greater demand on the lumbar spine structures.²²

Motor-control impairment is defined as a deficit in the control of movements during functional daily activities.²³ Motor-control impairment is known to occur secondary to

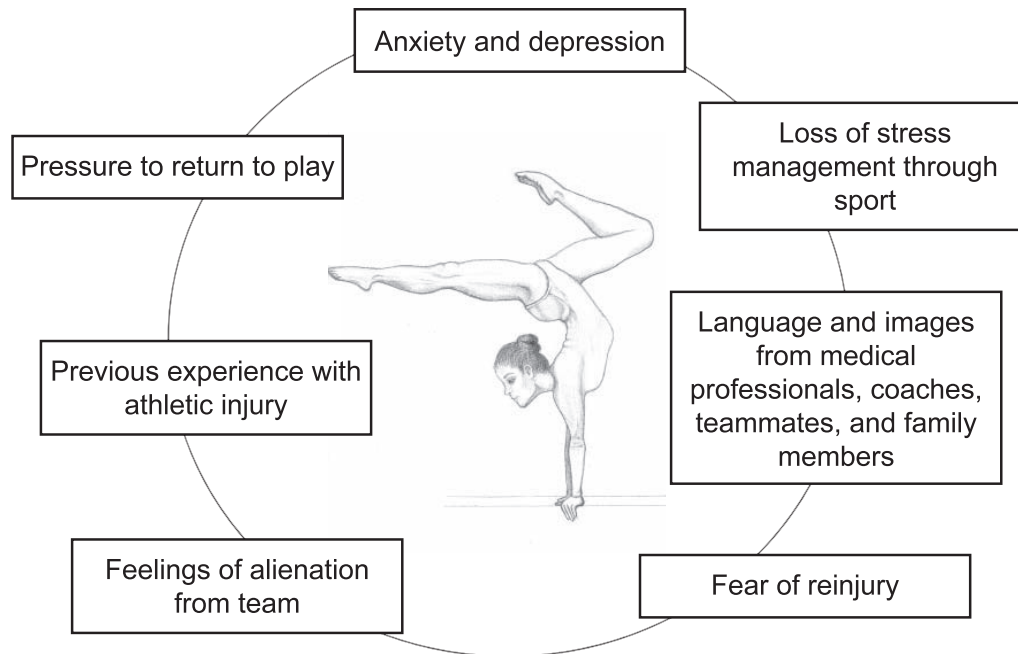


Figure 1. Central drivers of pain in the injured athlete.

pathologic processes and the presence of pain.¹⁴ Psychological processes, such as stress, fear, anxiety, depression, hysteria, and somatization, are also known to disrupt motor behavior.²⁴ After an episode of acute LBP, ongoing maladaptive motor-control behavior provides a basis for ongoing peripherally driven nociceptive sensitization.¹⁴ The nervous system undergoes changes to its cortical mapping and develops a pain memory that may leave it sensitized to the exacerbation and recurrence of pain.²⁵ This phenomenon is referred to as *central sensitization*, whereby the nervous system remains in a state of hyperexcitability even after the initial injury has healed.²⁶ This is due to altered sensory processing in the CNS and dysfunction of pain-inhibitory mechanisms.²⁷ To manage individuals with LBP, several authors^{23,24,27} have described classification systems that focus on identifying dysfunctional control of trunk motion. Both Sahrman²³ and O’Sullivan²⁴ identified excessive lumbar extension during movement as a distinct clinical pattern in their classification systems. This pattern can be reliably recognized.^{25,28} They advocated **correcting the dysfunctional movement pattern via a motor-learning approach (addressing the dysfunctional movement pattern via education and repeated practice with appropriate movement patterns and muscle activation)**. When the athlete is able to demonstrate pain-free control, the training progresses to larger ranges of motion at faster speeds with more complex sport-specific movements rather than solely focusing on activation of the deep trunk muscles while maintaining a neutral pelvic position.^{23,24} O’Sullivan²⁴ also proposed that patients with LBP experience alienation, frustration, anger, and depression that are central drivers of their pain. Athletes often experience these emotions as a result of not being able to participate in their sport (Figure 1). **Addressing cognitive-affective factors in addition to correcting maladaptive motor behavior and moving in a pain-free range reduces nociceptive input and desensitizes the nervous system, thereby allowing athletes to progress with their rehabilitation programs.**

The purpose of this case report is to describe the conservative management of a female athlete with EB LBP. Interventions focused on addressing motor-control impairments after repeated courses of core strengthening (improving activation and strength of the LMM and transversus abdominus muscle while in neutral pelvic position) failed to reduce the athlete’s pain or disability.

CASE DESCRIPTION

The 15-year-old female gymnast had a 4-year history of episodic EB LBP. She described her pain as a deep ache in her low back and rated it at 0/10 at best, 3/10 at the time of the evaluation, and 6/10 at worst. Over 4 years, she had undergone several courses of outpatient orthopaedic rehabilitation that focused on core muscle strengthening but had no long-lasting improvement. Although the pars interarticularis fracture was healed, the athlete referred to her LBP as “the fracture in my back.” She was unable to tolerate higher levels of gymnastic training or competition. At the time of the evaluation, she was unable to practice at all. The athlete’s goal was to return to competitive gymnastics.

Physical examination revealed hyperlordosis in relaxed standing,²⁹ early and excessive lumbar extension when bending backward with a hinge point at L4-L5,^{13,24,25} quadriceps hypertrophy, hip-flexor and quadriceps tightness assessed with a Thomas test,³⁰ stiffness in the midthoracic spine with posterior-anterior spring,³¹ and limited ankle dorsiflexion (10° bilaterally)³² that contributed to increased lumbar extension when squatting. The athlete’s Beighton ligamentous laxity score was 7/9. The Beighton scale is a popular screening technique for hypermobility.³³ Several investigators^{4,33} interpreted a score of 0 to 3 as *normal* and a score of 4 to 9 as representing *ligamentous laxity and associated hypermobility with poor motor control*.

Radiographs (anterior-posterior, lateral, and oblique views) and magnetic resonance imaging were performed.

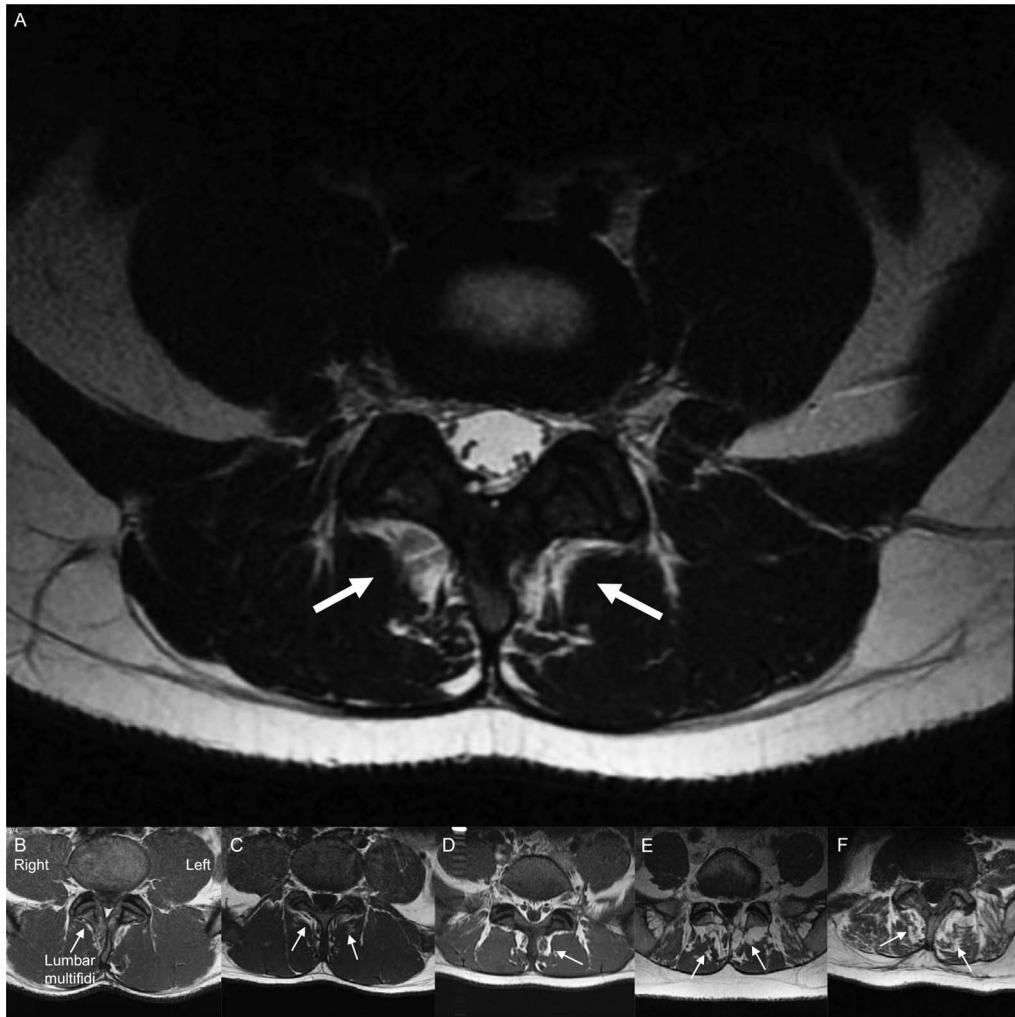


Figure 2. A, Lumbar spine magnetic resonance imaging scan of a 15-year-old high school gymnast demonstrating lumbar multifidus muscle atrophy with fatty infiltration (Goutallier grade 2). Examples of B, Goutallier grade 0 (right unilateral), C, Goutallier grade 1 (bilateral), D, Goutallier grade 2 (bilateral), E, Goutallier grade 3 (bilateral), and F, Goutallier grade 4 (bilateral). Parts B–F are reprinted from Winslow J, Getzin A, Greenberger H, Silbert W. Fatty infiltrate of the lumbar multifidus muscles predicts return to play in young athletes with extension-based low back pain. *Clin J Sport Med*. Published online ahead of print August 18, 2017, <http://journals.lww.com/cjsportsmed/pages/default.aspx>, with permission.

Radiographs demonstrated an L5 spondylolysis; however, there was no evidence of pedicular edema on magnetic resonance imaging suggestive of an old injury. Using the Goutallier classification system (GCS), a musculoskeletal radiologist graded the fatty infiltration of the LMM at 2 (Figure 2). The GCS is defined as grade 0, *normal muscle*; grade 1, *fatty streaks within the muscle*; grade 2, *fat less than muscle*; grade 3, *fat and muscle equal*; and grade 4, *fat greater than muscle*. The GCS has been found to be a reliable and valid method for measuring fatty infiltrate in the LMM.³⁴

This athlete presented with motor impairments consistent with O’Sullivan’s definition of extension-control impairment.²⁴ Previous bouts of outpatient orthopaedic rehabilitation focused on core strengthening rather than directly addressing the dysfunctional movements, both locally and globally, that likely contributed to excessive loads on the spinal elements and nociceptive input to the nervous system. The treating clinician hypothesized that by focusing on the athlete’s tendency to demonstrate early and excessive lumbar extension during sport-specific

movements and improving mobility at hypomobile regions along the kinetic chain, the athlete would be able to reduce strain on sensitive lumbar structures and thereby improve her tolerance of sport-specific movements.

Treatment included manipulation of the thoracic spine, manipulation of the talocrural joints of the ankles, hip-flexor and quadriceps stretching, and motor-control training to control lumbar spine extension during sport-specific movements. The aim of the intervention was to improve ankle dorsiflexion and thoracic and hip extension using manual therapy techniques, reactivate and reeducate the LMM through graded exposure to a progressively larger nonpainful range, and desensitize the nervous system by reducing the fear of movement. The athlete progressed from supine exercises using the Spine Stabilizer (Chattanooga Group, Hixson, TN) to upright functional movements using a wooden dowel. The stabilizer and wooden dowel provided tactile feedback that enabled the athlete to learn how to control lumbar extension (Figure 3). The Spine Stabilizer was placed under her lumbar spine and inflated until she felt pressure on her back. The athlete then



Figure 3. A–D, Motor-control training of lumbar extension with sport-specific movements.

performed combinations of leg slides and arm raises while trying to maintain pressure on the Spine Stabilizer. If she began to extend her lumbar spine, pressure would drop on the Spine Stabilizer, prompting her to self-correct. A wooden dowel was used to provide feedback regarding lumbar extension when performing functional upright activities, such as lunges, backbends, and arm raises. The athlete held the dowel with 1 hand placed over the lumbar lordosis. The pressure felt on the hand, produced by the contact with the lumbar spine, provided feedback during the functional movements and cued the athlete that she was extending her lumbar spine so that she could correct the movement pattern. Strategies to control lumbar extension included increasing hip or thoracic extension and tilting the pelvis posteriorly. Once the athlete had better control of her lumbar extension, gymnastic-specific activities were practiced with external cuing provided by the clinician (Figure 3).

Manual therapy techniques (joint and soft tissue mobilization) were used to facilitate thoracic spine and hip extension and were immediately followed by sport-specific movements. The athlete performed high numbers of repetitions of **each movement (20–30) with few rest breaks and was given a home program that she was instructed to practice often (3–4 times a day).** External cuing by the clinician was limited; the athlete was encouraged to identify when the lumbar spine was moved early and to a greater extent and what adjustments needed to be made. As the athlete's pain decreased and control of lumbar extension improved, she progressed to movements in positions of less stability, larger range, and greater velocity, eventually advancing to more ballistic sport-specific movements. The athlete was involved in selecting the movement progressions to keep herself engaged, improve adherence with the

home program, and increase specificity of the neuromuscular reeducation targeted at meeting her long-term goal of returning to gymnastics. Language regarding movement patterns was kept consistent to ensure good clinician-patient communication. The **clinician was also very positive and avoided terminology that might cause the athlete to worry more about her back pain. To specifically address the athlete's maladaptive thought of her back being "fractured," the clinician redirected the patient to think of her LBP as postural stress, explaining that by moving more efficiently, she would be able to reduce the stress and gain control over her pain.** The clinician never used medical terminology such as pars interarticularis, spondylolysis, or fracture.

The athlete was seen for 13 weeks (16 visits). At the end of the outpatient orthopaedic rehabilitation program, she could control the degree of lumbar extension with sport-specific movements. The athlete rated her pain level as 0/10 at best, 0/10 at the time of discharge, and 1/10 at worst. She demonstrated improved hip-flexor length during the Thomas test and better thoracic segmental mobility with posterior-anterior spring; she was also able to squat while keeping her heels on the ground. The athlete started going to the gymnastics center to practice and slowly progressed to high-level training. She was followed for a full year and had no further episodes of LBP that limited her sport participation. She resumed competition and eventually competed at the national level.

DISCUSSION

When young athletes continue to experience EB LBP, impaired motor control, as opposed to muscle weakness alone, may be the reason. Central sensitization of pain must

also be considered as a result of sustained peripheral nociceptive input resulting in changes at the spinal cord and cortical levels combined with maladaptive cognitive-affective factors.²⁴ Atrophy and fatty infiltrate of the LMM can compromise neuromuscular function and contribute to dysfunctional movement patterns that place a greater demand on lumbar spine structures.²² **Strengthening a muscle alone does not ensure that an athlete will use a muscle when needed to perform an activity.**³⁵

Individuals with LBP related to motor-control impairments may demonstrate a reduction in proprioceptive awareness of the lumbo-pelvic region.³⁶ For these individuals, a program that emphasizes a motor-learning approach, including changing dysfunctional physical movement patterns and cognitive processes, is recommended.²⁴ The goal of this method is to train the patient to control his or her provocative movement patterns, reduce excessive forces to the painful structures, and thus desensitize the nervous system. Emerging evidence for this approach has been provided by case reports^{37,38} and a randomized controlled trial.³⁹

It has been suggested that athletes with generalized joint hypermobility are more prone to developing LBP; however, a study conducted by Roussel et al⁴⁰ demonstrated that generalized joint hypermobility, evaluated with the Beighton score, did not predict LBP. In our patient, it was uncertain if limited hip range of motion, thoracic hypomobility, LMM changes, or dysfunctional movement patterns were present before the injury. The athlete responded well to motor-control training, was able to return to gymnastics after 3 months of therapy, and had no recurrences after a full year of competition. **The long-term improvements may be attributed to neuromuscular reeducation, improved segmental control and body position awareness, and desensitization of the nervous system.**

Future investigators need to examine the relationship between chronic EB LBP in young athletes and LMM atrophy with fatty infiltrate, motor-control impairments, and central sensitization. Activity modification, bracing, and core-strengthening exercises alone may not be the most appropriate treatment for athletes from EB LBP. Mounting evidence⁴¹ indicated that the pain from EB LBP may in fact not be from a pars interarticularis defect. Learning how to control lumbar extension with sport-specific movements and understanding pain may lead to more effective and efficient recovery in these athletes.

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REFERENCES

1. Purcell L, Micheli L. Low back pain in young athletes. *Sports Health*. 2009;1(3):212–222.
2. Hutchinson MR. Low back pain in elite rhythmic gymnasts. *Med Sci Sports Exerc*. 1999;31(11):1686–1688.
3. Kolt GS, Kirkby RJ. Epidemiology of injury in elite and subelite female gymnasts: a comparison of retrospective and prospective findings. *Br J Sports Med*. 1999;33(5):312–318.
4. Micheli LJ, Wood R. Back pain in young athletes. Significant differences from adults in causes and patterns. *Arch Pediatr Adolesc Med*. 1995;149(1):15–18.

5. Petering RC, Webb C. Treatment options for low back pain in athletes. *Sports Health*. 2011;3(6):550–555.
6. Oren JH, Gallina JM. Pars injuries in athletes. *Bull Hosp Jt Dis (2013)*. 2016;74(1):73–81.
7. Semon RL, Spengler D. Significance of lumbar spondylolysis in college football players. *Spine (Phila Pa 1976)*. 1981;6(2):172–174.
8. Kujala UM, Taimela S, Erkinntalo M, Salminen JJ, Kaprio J. Low-back pain in adolescent athletes. *Med Sci Sports Exerc*. 1996;28(2):165–170.
9. Houghton KM. Review for the generalist: evaluation of low back pain in children and adolescents. *Pediatr Rheumatol Online J*. 2010;8:28.
10. Ward SR, Kim CW, Eng CM, et al. Architectural analysis and intraoperative measurements demonstrate the unique design of the multifidus muscle for lumbar spine stability. *J Bone Joint Surg Am*. 2009;91(1):176–185.
11. Rosatelli AL, Ravichandiran K, Agur AM. Three-dimensional study of the musculotendinous architecture of lumbar multifidus and its functional implications. *Clin Anat*. 2008;21(6):539–546.
12. Gildea JE, Van Den Hoorn W, Hides JA, Hodges PW. Trunk dynamics are impaired in ballet dancers with back pain but improve with imagery. *Med Sci Sports Exerc*. 2015;47(8):1665–1671.
13. Hicks GE, Fritz JM, Delitto A, McGill SM. Preliminary development of a clinical prediction rule for determining which patients with low back pain will respond to a stabilization exercise program. *Arch Phys Med Rehabil*. 2005;86(9):1753–1762.
14. Hodges PW, Moseley GL. Pain and motor control of the lumbopelvic region: effect and possible mechanisms. *J Electromyogr Kinesiol*. 2003;13(4):361–370.
15. Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine (Phila Pa 1976)*. 1996;21(23):2763–2769.
16. Kjaer P, Bendix T, Sorensen JS, Korsholm L, Leboeuf-Yde C. Are MRI-defined fat infiltrations in the multifidus muscles associated with low back pain? *BMC Med*. 2007;5:2.
17. Mengiardi B, Schmid MR, Boos N, et al. Fat content of lumbar paraspinal muscles in patients with chronic low back pain and in asymptomatic volunteers: quantification with MR spectroscopy. *Radiology*. 2006;240(3):786–792.
18. Freeman MD, Woodham MA, Woodham AW. The role of the lumbar multifidus in chronic low back pain: a review. *PM R*. 2010;2(2):142–146; quiz 1 p following 67.
19. Teichtahl AJ, Urquhart DM, Wang Y, et al. Fat infiltration of paraspinal muscles is associated with low back pain, disability, and structural abnormalities in community-based adults. *Spine J*. 2015;15(7):1593–1601.
20. Hodges P, Holm AK, Hansson T, Holm S. Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. *Spine (Phila Pa 1976)*. 2006;31(25):2926–2933.
21. Indahl A, Kaigle AM, Reikeras O, Holm SH. Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles. *Spine (Phila Pa 1976)*. 1997;22(24):2834–2840.
22. Tsao H, Druitt TR, Schollum TM, Hodges PW. Motor training of the lumbar paraspinal muscles induces immediate changes in motor coordination in patients with recurrent low back pain. *J Pain*. 2010;11(11):1120–1128.
23. Sahrman S. *Diagnosis and Treatment of Movement Impairment Syndromes*. St Louis, MO: Mosby; 2002:51–105.
24. O’Sullivan P. Diagnosis and classification of chronic low back pain disorders: maladaptive movement and motor control impairments as underlying mechanism. *Man Ther*. 2005;10(4):242–255.
25. Gondhalekar GA, Kumar SP, Eapen C, Mahale A. Reliability and validity of standing back extension test for detecting motor control impairment in subjects with low back pain. *J Clin Diagn Res*. 2016;10(1):KC07–KC11.

26. Puentedura EJ, Louw A. A neuroscience approach to managing athletes with low back pain. *Phys Ther Sport*. 2012;13(3):123–133.
27. Frank C, Kobesova A, Kolar P. Dynamic neuromuscular stabilization & sports rehabilitation. *Int J Sports Phys Ther*. 2013;8(1):62–73.
28. Henry SM, Van Dillen LR, Trombley AR, Dee JM, Bunn JY. Reliability of novice raters in using the movement system impairment approach to classify people with low back pain. *Man Ther*. 2013;18(1):35–40.
29. Salamh PA, Kolber M. The reliability, minimal detectable change and concurrent validity of a gravity-based bubble inclinometer and iphone application for measuring standing lumbar lordosis. *Physiother Theory Pract*. 2014;30(1):62–67.
30. Kim GM, Ha SM. Reliability of the modified Thomas test using a lumbo-plevic stabilization. *J Phys Ther Sci*. 2015;27(2):447–449.
31. Brismee JM, Gipson D, Ivie D, et al. Interrater reliability of a passive physiological intervertebral motion test in the mid-thoracic spine. *J Manipulative Physiol Ther*. 2006;29(5):368–373.
32. Brantingham JW, Lee Gilbert J, Shaik J, Globe G. Sagittal plane blockage of the foot, ankle and hallux and foot alignment-prevalence and association with low back pain. *J Chiropr Med*. 2006;5(4):123–127.
33. Smits-Engelsman B, Klerks M, Kirby A. Beighton score: a valid measure for generalized hypermobility in children. *J Pediatr*. 2011; 158(1):119–123, 123.e1–123.e4.
34. Battaglia PJ, Maeda Y, Welk A, Hough B, Kettner N. Reliability of the Goutallier classification in quantifying muscle fatty degeneration in the lumbar multifidus using magnetic resonance imaging. *J Manipulative Physiol Ther*. 2014;37(3):190–197.
35. Adkins DL, Boychuk J, Remple MS, Kleim JA. Motor training induces experience-specific patterns of plasticity across motor cortex and spinal cord. *J Appl Physiol (1985)*. 2006;101(6):1776–1782.
36. O’Sullivan PB, Burnett A, Floyd AN, et al. Lumbar repositioning deficit in a specific low back pain population. *Spine (Phila Pa 1976)*. 2003;28(10):1074–1079.
37. Harris-Hayes M, Van Dillen LR, Sahrman SA. Classification, treatment and outcomes of a patient with lumbar extension syndrome. *Physiother Theory Pract*. 2005;21(3):181–196.
38. Caneiro JP, Ng L, Burnett A, Campbell A, O’Sullivan PB. Cognitive functional therapy for the management of low back pain in an adolescent male rower: a case report. *J Orthop Sports Phys Ther*. 2013;43(8):542–554.
39. Vibe Fersum K, O’Sullivan P, Skouen JS, Smith A, Kvale A. Efficacy of classification-based cognitive functional therapy in patients with non-specific chronic low back pain: a randomized controlled trial. *Eur J Pain*. 2013;17(6):916–928.
40. Roussel NA, Nijs J, Mottram S, Van Moorsel A, Truijen S, Stassijns G. Altered lumbopelvic movement control but not generalized joint hypermobility is associated with increased injury in dancers. A prospective study. *Man Ther*. 2009;14(6):630–635.
41. Andrade NS, Ashton CM, Wray NP, Brown C, Bartanusz V. Systematic review of observational studies reveals no association between low back pain and lumbar spondylolysis with or without isthmic spondylolisthesis. *Eur Spine J*. 2015;24(6):1289–1295.

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