Developing clinical procedures to diagnose specific motor control impairments associated with low back pain: prone hip extension (PHE), active straight leg raise (ASLR), and gait variability

Paul Bruno, DC, PhD[1]

(JCCA. 2017;61(3):207-211)

KEY WORDS: motor control, low back pain, chiropractic, gait, prone hip extension, active straight leg raise

Motor control can be defined as the ability to regulate and direct the mechanisms essential to movement.[1] It is well-established that the coordination of muscle activity around the lumbopelvic region is vital to the generation of mechanical spinal stability during static postures and dynamic activities.[2-4] Models illustrating mechanisms by which dysfunctional motor control strategies may serve as a potential cause and/or effect of low back pain (LBP) have been described by Panjabi[5-7] and Hodges[8,9], and chronic LBP patients have been shown to demonstrate a variety of motor control impairments. Two decades ago, a series of studies by Hodges and Richardson[10-12] demonstrated altered anticipatory control of the transversus abdominis during voluntary upper and lower limb movements, which has led to an emphasis on targeting this muscle in many rehabilitation programs. However, the validity of this approach has been questioned[13-15], and motor control impairments have also been reported for the gluteus maximus[16-19], lumbar paraspinal muscles[17,19,20], and abdominal muscles[17,20] in LBP patients during a variety of movements. The findings of recent reviews suggest that rehabilitation programs targeting specific motor control impairments in chronic LBP patients are superior to

[1] Associate Professor, Faculty of Kinesiology and Health Studies, University of Regina

Corresponding author:
Paul Bruno, Associate Professor, Faculty of Kinesiology and Health Studies, University of Regina, 3737 Wascana Parkway, Regina, SK, S4S 0M2
Tel: (306) 337-3343
E-mail: paul.bruno@uregina.ca

The author has no conflicts of interest to declare regarding this paper or the material described therein.

minimal intervention and general exercise[21-24] and generally comparable to other conservative interventions[23-26] in improving clinical outcomes. However, as highlighted by Hodges[8,9], current evidence suggests that the motor control impairments associated with LBP are highly variable (i.e., they do not appear to be uniform within or between individuals) and involve multiple levels of the motor control system. The ability to diagnose the presence of specific motor control impairments would allow clinicians to more effectively individualize and target therapy aimed at correcting specific impairments in their patients.[14,27,28]

To this end, the long-term objective of my research program is to establish evidence-based clinical procedures that can be used to diagnose the presence of specific motor control impairments in individuals with LBP. The remainder of this commentary will describe two projects currently being conducted in our lab that are focused on assessing the clinical utility of the prone hip extension (PHE) test, active straight leg raise (ASLR) test, and gait variability in diagnosing specific motor control impairments associated with LBP.

Prone Hip Extension (PHE) Test & Active Straight Leg Raise (ASLR) Test

The PHE test was originally described as a means of assessing for the presence of a motor control impairment associated with the gluteus maximus in individuals with LBP.[29,30] The test is performed with the patient lying prone and alternately lifting each leg away from the table while the clinician observes and/or palpates the gluteus maximus, hamstrings, and lumbar paraspinal muscles to determine their relative order of activation.[29-31] It was suggested that the "normal" motor control strategy for the movement was for the gluteus maximus to be recruited first, and that delayed recruitment of this muscle represented a motor control impairment that may lead to the development and/or exacerbation of LBP.[29-31] However, several studies have since demonstrated that it is "normal" for the gluteus maximus to be recruited after the hamstrings and paraspinal muscles in both LBP patients and asymptomatic individuals.[16,32-35] As a result, Murphy *et al*.[36] proposed that, rather than attempt to determine the muscle activation order, clinicians should instead observe for specific "abnormal" patterns of lumbopelvic motion during the test, and that the presence of these patterns represented a motor control impairment.

The ASLR test has also evolved over time in the literature. It was originally described as a means of assessing the ability of the sacroiliac joints to effectively transfer loads between the pelvis and legs in females with pregnancy-related pelvic pain.[37,38] More recently, it has been suggested that the test may be useful in diagnosing the presence of motor control impairments in the general LBP population.[39,40] The test is similar to the PHE test, with the patient lying supine (rather than prone) and asked to alternately lift each leg away from the table while the clinician observes whether the pelvis maintains a neutral alignment during the test. An inability to maintain a neutral alignment of the pelvis represents a motor control impairment.[41,42]

Two studies have demonstrated good inter-examiner agreement in classifying LBP patients as "positive" or "negative" based on the presence or absence, respectively, of the previously-described "abnormal" lumbopelvic motion patterns during the PHE test and ASLR test.[36,43] However, there are currently no published studies that have: 1) objectively quantified the lumbopelvic motion patterns demonstrated by LBP patients during these tests; and 2) determined whether any "abnormal" motion patterns demonstrated by LBP patients during these tests are associated with specific underlying dysfunctional muscle recruitment strategies. Additionally, there are currently no published studies to support the notion that the motor control strategies used during these tests are associated with the strategies used during dynamic activities (e.g., gait), as has been suggested by some authors.[29-31,37,38] Despite the similarity in hip joint motion between these tests and the hip extension (PHE) and hip flexion (ASLR) phases of gait, these tasks are generally quite different. The PHE test and ASLR test are non-weight bearing, open kinetic chain movements with a stationary base of support, while walking is weight bearing and consists of both open and closed kinetic chain phases and a continually changing base of support. It is therefore likely that the motor control strategies used to effectively perform these movements would be different.

To provide further clarity on these gaps in the literature, we are currently conducting a study supported by a grant co-funded by the Canadian Chiropractic Research Foundation (CCRF) and Saskatchewan Health Research Foundation (SHRF). Using recently-published motion capture models to measure pelvic and regional lumbar

motion[44,45], the two primary objectives of this study are to: 1) compare the lumbopelvic motion patterns and muscle recruitment strategies demonstrated during the PHE test and ASLR test by LBP patients and asymptomatic controls; and 2) compare the lumbopelvic motion patterns and muscle recruitment strategies demonstrated during the tests and those used during gait. A secondary objective of the study is to determine whether sub-groups of LBP patients with clinical signs of lumbar-related LBP and sacroiliac-related LBP demonstrate different motor control strategies during the tests.[46]

Gait Variability

Human locomotion involves a repeating cyclical sequence of events that take place between the initial contact of one foot and the initial contact of the same foot (i.e., one gait cycle or stride). Gait analysis commonly involves measuring the spatiotemporal parameters of an individual's gait pattern (e.g., stride length, stride time). There is an emerging body of research demonstrating that the stride-to-stride fluctuations (i.e., variability) in these parameters provide unique insights regarding the status of an individual's locomotor control system. Variability is an inherent feature of many human movement patterns, and a certain amount of variability is considered to be a feature of a healthy and adaptable motor control system.[47,48] Gait variability is commonly quantified by calculating the standard deviation (SD) or coefficient of variation (CV) of the parameter of interest over a series of consecutive strides (e.g., stride length SD, stride time CV). Gait patterns have also been shown to possess fractal properties that can be quantified using a fractal scaling index (FSI), which provides a measure of the long-range, self-similar patterns that are associated with healthy physiological systems.[47,49,50] There is a substantial body of evidence that gait variability is altered in individuals with a variety of neurodegenerative diseases (e.g., Huntington's disease, Parkinson's disease, amyotrophic lateral sclerosis, multiple sclerosis)[51], and there is emerging evidence that they have the potential to serve as a tool to clinically screen for an increased risk of falls in older adults[52-54].

There is also preliminary evidence that gait variability is affected by musculoskeletal conditions such as osteoarthritis of the knee[55,56] and chronic LBP[57,58]. However, there are limitations in the current body of evidence regarding gait variability changes associated with LBP, including small patient sample sizes (i.e., n=12) and a lack of FSI measurements due to the short duration of walking trials that have been conducted in the published studies to date. One of my Faculty colleagues has recently developed a method to calculate gait variability over longer duration walking trials using data collected from body-mounted accelerometers.[59] He is also in the process of developing a Smartphone app that can calculate gait variability using data collected from the accelerometer contained in the device. Using this accelerometer-based method, we are currently conducting a study that will provide further insight into the potential usefulness of gait variability in assessing for the presence of impairments associated with the locomotor control system in individuals with LBP.

Future Directions

Depending on the findings of these studies, future lines of inquiry may include an assessment of the effect of interventions on restoring normal motor control strategies during the PHE test and ASLR test, and normal spatiotemporal variability patterns during gait, in LBP patients and the association between such changes and changes in clinical outcomes (e.g., pain, disability).

References

1. Shumway-Cook A, Woollacott M. Motor Control: Translating Research into Clinical Practice. 5th Ed. Philadelphia: Wolters Kluwer, 2016.
2. Bergmark A. Stability of the lumbar spine. A study in mechanical engineering. Acta Orthop Scand Suppl. 1989; 230: 1-54.
3. Cholewicki J, McGill S. Mechanical stability of the in vivo lumbar spine: implications for injury and chronic low back pain. Clin Biomech. 1996; 11: 1-15.
4. McGill S, Grenier S, Kavcic N, Cholewicki J. Coordination of muscle activity to assure stability of the lumbar spine. J Electromyogr Kinesiol. 2003; 13: 353-359.
5. Panjabi M. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. J Spinal Disord. 1992; 5: 383-389.
6. Panjabi M. The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. J Spinal Disord. 1992; 5: 390-396.
7. Panjabi M. Clinical spinal instability and low back pain. J Electromyogr Kinesiol. 2003; 13: 371-379.
8. Hodges P. Pain and motor control: From the laboratory to rehabilitation. J Electromyogr Kinesiol. 2011; 21: 220-228.
9. Hodges P, Tucker K. Moving differently in pain: a new

theory to explain adaptation to pain. Pain . 2011; 152: S90-S98.
10. Hodges P, Richardson C. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. Spine. 1996; 21: 2640-2650.
11. Hodges P, Richardson C. Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb. J Spinal Disord. 1998; 11: 46-56.
12. Hodges P, Richardson C. Altered trunk muscle recruitment in people with low back pain with upper limb movements at different speeds. Arch Phys Med Rehabil. 1998; 80: 1005-1012.
13. Standaert C, Herring S. Expert opinion and controversies in musculoskeletal and sports medicine: core stabilization as a treatment for low back pain. Arch Phys Med Rehabil. 2007; 88: 1734-1736.
14. Standaert C, Weinstein S, Rumpeltes J. Evidence-informed management of chronic low back pain with lumbar stabilization exercises. Spine J. 2008; 8: 114-120.
15. Stokes I, Gardner-Morse M, Henry S. Abdominal muscle activation increases lumbar spinal stability: analysis of contributions of different muscle groups. Clin Biomech. 2011; 26: 797-803.
16. Bruno P, Bagust J. An investigation into motor pattern differences used during prone hip extension between subjects with and without low back pain. Clin Chiropr. 2007; 10: 68-80.
17. Hungerford B, Gilleard W, Hodges P. Evidence of altered lumbopelvic muscle recruitment in the presence of sacroiliac joint pain. Spine. 2003; 28: 1593-1600.
18. Leinonen V, Kankaanpaa M, Airaksinen O, Hanninen O. Back and hip extensor activities during trunk flexion/extension: effects of low back pain and rehabilitation. Arch Phys Med Rehabil. 2000; 81: 32-37.
19. Vogt L, Pfeifer K, Banzer W. Neuromuscular control of walking with chronic low-back pain. Man Ther. 2003; 8: 21-28.
20. Ferguson S, Marras W, Burr D, Davis K, Gupta P. Differences in motor recruitment and resulting kinematics between low back pain patients and asymptomatic participants during lifting exertions. Clin Biomech. 2004; 19: 992-999.
21. Bystrom M, Rasmussen-Barr E, Grooten W. Motor control exercises reduces pain and disability in chronic and recurrent low back pain. Spine. 2013; 38: E350-358.
22. Gomes-Neto M, Lopes J, Conceição C, Araujo A, Brasileiro A, Sousa C, et al. Stabilization exercise compared to general exercises or manual therapy for the management of low back pain: a systematic review and meta-analysis. Phys Ther Sport. 2017; 23: 136-142.
23. Macedo L, Maher C, Latimer J, McAuley J. Motor control exercise for persistent, nonspecific low back pain: a systematic review. Phys Ther. 2009; 89: 9-25.
24. Saragiotto B, Maher C, Yamato T, Costa L, Costa L, Ostelo R, et al. Motor control exercise for nonspecific low back pain: a Cochrane review. Spine. 2016; 41: 1284-1295.
25. Smith B, Littlewood C, May S. An update of stabilisation exercises for low back pain: a systematic review and meta-analysis. BMC Musculoskelet Disord. 2014; 15: 416.
26. Wang X, Zheng J, Yu Z, Bi X, Lou S, Liu J, et al. A meta-analysis of core stability exercise versus general exercise for chronic low back pain. PLoS One. 2012; 7: e52082.
27. Ferreira P, Ferreira M, Maher C, Refshauge K, Herbert R, Hodges P. Changes in recruitment of transversus abdominis correlate with disability in people with chronic low back pain. Br J Sports Med. 2010; 44: 1166-1172.
28. Vasseljen O, Unsgaard-Tøndel M, Westad C, Mork P. Effect of core stability exercises on feed-forward activation of deep abdominal muscles in chronic low back pain: a randomized controlled trial. Spine. 2012; 37: 1101-1108.
29. Janda V. Evaluation of Muscular Imbalance. In: Liebenson C, editor. Rehabilitation of the Spine: A Practitioner's Manual. Baltimore: Lippincott Williams & Wilkins, 1996.
30. Jull G, Janda V. Muscles and Motor Control in Low Back Pain: Assessment and Management. In: Twomey L, Taylor J, editors. Physical Therapy of the Low Back. New York: Churchill Livingstone, 1987.
31. Chaitow L, DeLany J. Clinical Application of Neuromuscular Techniques. Volume 2 – The Lower Body. Edinburgh: Churchill Livingstone, 2002.
32. Bruno P, Bagust J. An investigation into the within-subject and between-subject consistency of motor patterns used during prone hip extension in subjects without low back pain. Clin Chiropr. 2006; 9: 11-20.
33. Guimaraes C, Sakamoto A, Laurentino G, Teixeira-Salmela L. Electromyographic activity during active prone hip extension did not discriminate individuals with and without low back pain. Rev Bras Fisioter. 2010; 14: 351-357.
34. Lehman G, Lennon D, Tresidder B, Rayfield B, Poschar M. Muscle recruitment patterns during the prone leg extension. BMC Musculoskelet Disord. 2004; 5: 3.
35. Sakamoto A, Teixeira-Salmela L, de Paula-Goulart F, de Morais Faria C, Guimaraes C. Muscular activation patterns during active prone hip extension exercises. J Electromyogr Kinesiol. 2009; 19: 105-112.
36. Murphy D, Byfield D, McCarthy P, Humphreys K, Gregory A, Rochon R. Interexaminer reliability of the hip extension test for suspected impaired motor control of the lumbar spine. J Manipulative Physiol Ther. 2006; 29: 374-377.
37. Mens J, Vleeming A, Snijders C, Stam H, Ginai A.

The active straight leg raising test and mobility of the pelvic joints. Eur Spine J. 1999; 8: 468-473.
38. Snijders C, Vleeming A, Stoeckart R. Transfer of lumbosacral load to iliac bones and legs. Part 1. Biomechanics of self-bracing of the sacroiliac joints and its significance for treatment and exercise. Clin Biomech. 1993; 8: 285-194.
39. Liebenson C, Karpowicz A, Brown S, Howarth S, McGill S. The active straight leg raise test and lumbar spine stability. PM&R. 2010; 1: 530-535.
40. Roussel N, Nijs J, Truijen S, Smeuninx L, Stassijns G. Low back pain: clinimetric properties of the Trendelenburg test, active straight leg raise test, and breathing pattern during active straight leg raising. J Manipulative Physiol Ther. 2007; 30: 270-278.
41. Mens J, Vleeming A, Snijders C, Koes B, Stam H. Reliability and validity of the active straight leg raise test in posterior pelvic pain since pregnancy. Spine. 2001; 26: 1167-1171.
42. Rabin A, Shashua A, Pizem K, Dar G. The interrater reliability of physical examination tests that may predict the outcome or suggest the need for lumbar stabilization exercises. J Orthop Sports Phys Ther. 2013; 43: 83-90.
43. Bruno P, Millar D, Goertzen D. Inter-examiner agreement, sensitivity, and specificity of the prone hip extension test and active straight leg raise test. Chiropr Man Therap. 2014; 22: 23.
44. Bruno P, Barden J. Comparison of two alternative technical marker sets for measuring 3D pelvic motion during gait. J Biomech. 2015; 48; 3876-3882.
45. Ryan N, Bruno P. Analysis of 3D multi-segment lumbar spine motion during gait and prone hip extension. J Electromyogr Kinesiol. 2017; 33: 111-117.
46. Murphy D, Hurwitz E, Hart B. Comparison of findings of active straight leg raise test in patients with lumbar versus sacroiliac pain. J Chiropr Educ. 2012; 26: 100.
47. Hausdorff J. Gait variability: methods, modeling and meaning. J Neuroeng Rehabil. 2005; 2: 19.
48. Stergiou N, Harbourne R, Cavanaugh J. Optimal movement variability: a new theoretical perspective for neurologic physical therapy. J Neurol Phys Ther. 2006; 30: 120-129.
49. Goldberger A, Amaral L, Hausdorff J, Ivanov P, Peng C-K, Stanley H. Fractal dynamics in physiology: alterations with disease and aging. Proc Natl Acad Sci. 2002; 99: 2466-2472.
50. Hausdorff J. Gait dynamics, fractals and falls: finding meaning in the stride-to-stride flucutations of human walking. Hum Move Sci. 2007; 26: 555-589.
51. Moon Y, Sung J, An R, Hernandez M, Sosnoff J. Gait variability in people with neurological disorders: a systematic review and meta-analysis. Hum Move Sci. 2016; 47: 197-208.
52. Hausdorff J, Rios D, Edelberg H. Gait variability and fall risk in community-living older adults: a 1-year prospective study. Arch Phys Med Rehabil. 2001; 82: 1050-1056.
53. Kikkert L, de Groot M, van Campen J, Beijnen J, Hortobágyi T, Vuillerme N, Lamoth C. Gait dynamics to optimize fall risk assessment in geriatric patients admitted to an outpatient diagnostic clinic. PLoS One. 2017; 12: e0178615.
54. König N, Taylor W, Armrecht T, Dietzel R, Singh N. Identification of functional parameters for the classification of older female fallers and prediction of 'first-time' fallers. J R Soc Interface. 2014; 11: 20140353.
55. Clermont C, Barden J. Accelerometer-based determination of gait variability in older adults with knee osteoarthritis. Gait Posture. 2016; 50: 126-130.
56. Kiss R. Effect of severity of knee osteoarthritis on the variability of gait parameters. J Electromyogr Kinesiol. 2011; 21: 695-703.
57. Hamacher D, Hamacher D, Herold F, Schega L. Are there differences in the dual-task walking variability of minimum toe clearance in chronic low back pain patients and healthy controls? Gait Posture. 2016; 49: 97-101.
58. Lamoth C, Stins J, Pont M, Kerckhoff F, Beek P. Effects of attention on the control of locomotion in individuals with chronic low back pain. J Neuroeng Rehabil. 2008; 5: 13.
59. Kobsar D, Olson C, Paranjape R, Barden J. The validity of gait variability and fractal dynamics obtained from a single, body-fixed triaxial accelerometer. J Appl Biomech. 2014; 30: 343-347.