Alterations of static and dynamic balance in patients with lumbar radiculopathy

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Background
Lumbosacral radiculopathy (LR) is strongly associated with delayed recovery and persistent disability. Chronic LR may lead to somatosensory system impairment, resulting in decline of postural balance.
Purpose
The aim of the study was to investigate static and dynamic postural balance alterations in individuals with LR owing to lumbar disc herniation.
Participants and methods
In this case–control study design, 12 patients presenting with unilateral LR were included, whereas 12 normal individuals were randomly selected for control. Static balance was assessed functionally using Functional Reach Test. Dynamic balance was assessed via Biodex Balance System, where postural stability indices and the dynamic limits of stability were evaluated. Dynamic limits of stability parameters were expressed as direction control and time required to complete the test.
Results
There was significant reduction of mean values of Functional Reach Test in LR group ($P<0.0001$) when compared with the control. In addition, there was a significant increase of the mean values of overall stability index ($P<0.0001$) and postural stability indices ($P<0.0002$) and a significant decrease of the mean values of direction control ($P<0.0001$) in the LR group.
Conclusion
Patients with chronic LR have shown to have limited functional abilities and decreased postural balance both statically and dynamically when compared with normal individuals.

Keywords:
balance, Biodex Balance System, lumbosacral radiculopathy

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Introduction

Low back pain (LBP) combined with leg pain is a common complaint, although the pain duration is usually self-limited, with a favorable prognosis up to 90% of LBP cases within 6 weeks [1]. The prevalence of lumbosacral radiculopathy (LR) is roughly 3–5%; however, the association of adjacent lumbosacral nerve roots producing neural dysfunction and pain is more resistant to conservative treatment than LBP alone [2,3]. The most common cause for LR is a herniated disc impinging or irritating a nerve root [4]. The most frequently affected intervertebral discs are L4–L5 and L5–S1, leading to L5 or S1 radiculopathies, also referred to as sciatica [5]. The clinical presentation of LR is described by most patients as sharp, dull, piercing, throbbing, stabbing, shooting, or burning pain and paresthesias in the involved dermatome [6,7]. Neurological findings of nerve root entrapment include sensory deficits, reflex changes, and/or muscle weakness. Radicular pain may last for more than 3 months in 25% of patients [8]; however, the consequences are disability, reduced quality of health, and reduced working capability [9].

Postural control and balance are essential attributes in activates of daily living. Visual, vestibular, and somatosensory systems transmit their input to the central nervous system (CNS), resulting in the most optimal muscle forces and body reactions to maintain the center of mass (COM) within the support base, hence executing adequate postural balance [10]. Numerous uncontrollable factors may promote to the decline of postural balance such as reduced sensory-motor system performance with aging and neurological or musculoskeletal disorders [11]. Chronic impairment of proprioceptors in the lumbar spine, trunk, or lower extremities may affect postural balance [12]. Deterioration of this proprioceptive information from these areas may be the influential factor in reducing the precision in the sensory integration process [13].

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Recent consistent evidence suggests that LBP accounts for the increased postural sway amplitude and/or sway velocity [14]. Few studies exist that describe the characteristics and clinical course of long-term LR, i.e., for more than 3 months, on postural control [15,16]. Most studies emphasize on patients with back pain alone, mixed populations with back and leg pain (without differentiating between them), or are involved with describing the characteristics of highly selected populations including postoperative candidates [10,13–16]. As postural balance is controlled by sensory information, central processing, and neuromuscular responses, any alterations in proprioception, asymmetrical load of lower extremities, distorted muscle activation timing, sequencing, and asymmetry in foot pressure owing to long-term radicular pain may alter postural balance in individuals with LR [12,13,15,16]. Robust evidence concerning the long-term effects of LR on postural balance is lacking and needs to be addressed in individuals with lumbar disc herniation (LDH). Hence, the aim of this study was to investigate if chronic LR (>3 months) is associated with an altered performance in static and dynamic postural balance.

Participants and methods

Trial design and sample

A case–control trial design was carried out at the Balance Laboratory of the School of Physical Therapy, Cairo University, Giza, Egypt, from July 2017 to October 2017. A convenient sample of 24 participants, with age ranging from 35 to 55 years, was included in this study. Investigative group consisted of 12 participants with LR, with six men and six women, who were selected from the outpatient clinic of the School of Physical Therapy at Cairo University. Participants were about to undergo physical therapy sessions for their condition. Inclusion criteria consisted of the following: (i) LDH confirmed by a lumbosacral MRI at L4–L5 and/or L5–S1 levels; (ii) experienced LR that lasted more than 3 months; (iii) a positive straight leg raising with induced symptoms; (iv) a score of 5 or more on the visual analog scale; and (v) a BMI ranging from 18.5 to less than 30. Participants were excluded if they had the following: (i) history of cerebral concussions and orthopedic or vestibular disorders; (ii) any neurological deficit affecting balance; (iii) history of spine surgery; (iv) pregnancy; (v) alcoholics or the consumption of alcohol 24 h before the evaluation; (vi) visual acuity impairment; and (vii) physical therapy interventions in the past 3 months. The control group consisted of 12 normal individuals (have not experienced LBP for >3 months before the study). They were selected from the employees working at the School of Physical Therapy, Cairo University.

All participants, in both groups, underwent an evaluative procedure to test static [maximum anterior distance (MAD)] and dynamic [postural stability indices (PSIs) and dynamic limits of stability (DLOS)] balance control. They provided written informed consent to participate in the study. The Board Council of Higher Education of the School of Physical Therapy, the Institutional Review Board of Higher Education and Research of Cairo University, and the Supreme Council of Universities at Egypt approved the study.

Test methods and measurement outcome

Functional static balance control assessment

Functional Reach Test was carried out for all participants. It has demonstrated high intrarater reliability of 0.97 and an inter-rater reliability of 0.99 in various adult populations [17–19]. With a yardstick mounted on a wall at shoulder height, and the participant in standing position next to the yardstick, but not touching it, the participant was instructed to flex their shoulder to 90° and fist their hand. This starting position was documented by determining which metacarpophalangeal joint lined up with on the yardstick. Afterward, the participant was instructed to reach as far forward as possible in a plane parallel with the yardstick, without taking a step nor touching the wall. This was considered the end position with the metacarpophalangeal joint against the ruler. The difference between the starting and ending position was documented and was considered the MAD (inches). A score less than 6 inches showed limited functional static balance. Three successive measurements were recorded, and the mean was used in the analysis.

Dynamic balance control assessment

Biodex Balance System (Biodex Medical Systems Inc., Shirley, New York, USA) was used to assess both PSIs and DLOS. Biodex Balance System has demonstrated high reliability for evaluating dynamic postural balance in healthy people [20–24], in blind people [25], as well as rheumatoid arthritis [26] and ankle instability [27]. The system comprises eight stability levels, with level 8 the most stable and 1 the least stable [28,29]. It also consists of a movable balance foot platform providing up to 20° of surface tilt in a 360° range of motion. The platform includes a foot grid illustration to determine the optimal foot position, allowing consistency in each trial in positioning the vertical ground reaction forces as well as the centre of gravity in each test trial. The platform is

connected to computer software that automatically calculates the measurement outcomes [29].

The PSI measurement outcome consisted of anterior–posterior stability index (APSI), mediolateral stability index (MLSI), and the overall stability index (OSI). These measures calculate the amount of deviation and displacement (°) of the platform from the baseline position [26]. The higher the scores, the increased motion from baseline level, the higher the sway, and the poorer the balance [29]. With the participant standing barefoot on the platform holding onto the support handle, its height was adjusted accordingly. With eyes open, the participant was instructed to maintain his/her foot in a centered position on the platform by using the foot angles and coordinates on the platform grid. The participant's weight, height, and age were then logged into the device. The platform stability level was set at five (moderate) [30], and test duration was set for 30 s [29]. As the test proceeded, the participant was instructed to release the device handle and maintain a levelled platform by means of sustaining a cursor centered on a bull's eye located on the screen grid through visual feedback. The start key was then engaged in the control panel to unlock the platform (which took five seconds to actually start), and an auditory alarm beeped just before the test proceeded. Two test trials were executed before the specific trial outcome was recorded for the purpose of instrumentation familiarity before data collection. At the end of each test trial, a printout report was obtained documenting OSI, APSI, and MLSI.

The DLOS measurement outcome consisted of direction of control (DC) and the time required for completing the test. This represented the motor control skills, where the lower DC scores and prolonged time to complete the test indicated impaired dynamic balance [24,28,30]. The participants were once again centered on the platform as the pervious test; however, the stability level of the platform was set to level seven [31]. Here the participant was instructed to shift and move the cursor over a target box located on the screen. This cursor was sustained over the target box for a minimum of 0.5 s and then returned back to the center target. Little deviation and quick movement were needed before the next target box emerged. This was achieved by un-leveling the platform to reach the target box. The test ended when eight target boxes were completed, and the cursor was repositioned in the central box. Touching the device handle was permitted to avoid falling but grasping it was not allowed. When the test was completed, the DC (%) and time (s) were recorded and printed out. To minimize errors from adaptation, a two-minute rest period was taken between PSIs and DLOS.

Sample size

The sample size was calculated using the G*Power software, Heinrich Heine University Düsseldorf, Düsseldorf, Germany (version 3.0.10). Independent *t*-test was selected. A pilot study was conducted on 16 participants: eight with LR and eight normal individuals. Standardized mean difference effect size (d) of the difference in MAD was calculated (d=1.6). Considering a power of 0.95, an α level of 0.05, two groups, and response variables of six, a generated sample size of at least 12 participants per group was required.

Data analysis

Statistical analysis was computed using SPSS for Windows, version 22 (SPSS Inc., Chicago, Illinois, USA). χ^2 and independent *t*-tests were used to describe the means, SD, and percentages of the participants' characteristics. Before data analysis, Shapiro–Wilk test was used to test data normality. A one-way multivariate analysis of variance was used to compare between LR group versus the control group. Bonferroni correction was used to account for multiple analyses of variance. Thus, level of significance was accepted at P value less than 0.008 (α/6).

Results

Table 1 lists the general physical characteristics of the 24 participants in this study. There was no significant difference in the mean values of age, sex, weight, height, or BMI between both groups (P>0.05). There was a statistically significant difference in measures of stability between groups (overall effect with values: F=22.059 and P<0.0001). The mean (SD) value of VAS for participant with LR was 7.4 (1.4). Table 2 represents the mean values as it was revealed that there was a significant decrease in the mean values of MAD in the LR group. In addition, OSI, APSI, and MLSI had significant increase in the mean values in the LR group. Furthermore, DC mean value had a significant decrease, whereas the mean value of the total time to complete the test had a significant increase in the LR group. Regarding between-group comparison, it was revealed that there was a significant difference between both groups, with P value less than 0.0001 for all measurement outcome.

Discussion

The purpose of this study was to investigate the effect of chronic LR on functional static and dynamic balance

Table 1 General characteristics of the participants

Items	LR group (mean±SD)	Control group (mean±SD)	Comparison		Significance
			t-value	*P* value	
Age (years)	47.25±5.61	41.41±6.8	2.292	0.05	NS
Weight (kg)	73.58±5.29	74.5±12.3	–0.237	0.815	NS
Height (cm)	166.75±4.47	168.41±8.06	–0.626	0.538	NS
BMI (kg/m^2)	26.44±2.27	26.13±3.65	0.248	0.806	NS
Sex distribution [*n* (%)]					
Female	7 (58.3)	5 (41.7)	χ^2=0.667	0.684	NS
Male	5 (41.7)	7 (58.3)			

LR, lumbar radiculopathy.

Table 2 Comparison between groups regarding all test parameters

Measurement outcome	Mean±SD		Univariate test	
	LR group	Control group	*F*-value	*P* value
MAD	16.91±4.54	37.25±4.28	127.164	0.0001*
PSIs				
OSI	5.15±2.2	1.64±0.66	27.849	0.0001*
APSI	4.28±1.88	1.38±0.49	26.461	0.0001*
MLSI	2.85±1.66	1.05±0.57	12.476	0.002*
DLOS				
DC	22±11.34	50.75±17.5	22.796	0.0001*
Test time	2.79±1.2	1.25±0.28	17.754	0.0001*

APSI, anterior–posterior stability index; DC, direction control; DLOS, dynamic limits of stability; MAD, maximum anterior distance; MLSI, mediolateral stability index; OASI, overall stability index; PSI, postural stability index. *α<0.05, significant.

in patients having LDH. When comparing both groups, a significant decrease in MAD in the LR group was found when compared with that of the control indicating limited functional static balance when trying to reach forward. Postural control and balance robustness require sensory and motor-processing strategies along with learned responses from previous experience and the anticipation of change [32]. In addition, proprioception has a very important neurophysiological role in motor control of postural balance [33].

Static balance was found affected, both in standing [33–35], as well as in sitting postural conditions in chronic LBP population [36]. One possible mechanism is that chronic deterioration and reduced proprioceptive afference in the lumbar spine, trunk [37]. or lower extremities [12] may have affected postural balance. In addition, altered proprioceptive reweighting owing to chronic LR combined with inconsistent postural strategies [38] delayed onsets of both abdominal and back muscles [36], which may have contributed to the impaired robustness in static postural tasks. Moreover, greater repositioning errors in isolated spinal movements were found [37,39], and also less capacity to upweight proprioceptive feedback from paraspinal muscles to provide optimal standing postural control in people with LBP is seen [40,41]. These findings suggest that proprioceptive impairments at the lumbar spine and lower extremities may have played an important role in the deterioration of static postural balance as observed in our findings.

Frost *et al.* [15] found a reduction in somatosensory information from the sole of the foot that may have contributed to deficits in quiet standing balance control in individuals having LBP with associated radiculopathy. These results come in line with the findings of this study, where MAD was found to be decreased in individuals with LR. Another possible mechanism behind static balance alterations in patients with LR is 'pain inhibition' [42]. High-threshold nociceptive afferent discharge owing to nerve root compression may interfere with spinal motor pathways [43] as well as the motor cortex [44]. In addition, exaggerated pain may cause an increased presynaptic inhibition of muscle afferents [45] leading to the central modulation of muscle spindle proprioception [46], resulting in extended latencies owing to the reduction in muscle spindle feedback. These alterations may have led to the decreased muscle control that resulted in a decreased MAD as found in our results.

Besides postural balance in static postures (e.g. standing and sitting), it was also found that performance of a dynamic task such as the sit-to-

stance-to-sit movement was affected in patients with LBP [47]. In this study, when comparing between both groups, our results revealed OSI, APSI, and MLSI had significant increases in mean values in the LR group, indicating poor dynamic postural balance. This can be explained by the two stages classified during complex sagittal movements (sit-to-stand) in patients with LBP: a preliminary phase and a movement phase. According to Cordo and Gurfinkel [48] during the preliminary phase, the CNS coordinates the body to perform movement optimally with the least energy demands in the next phase [49,50]. It was found that pelvic movement was essential to transfer the COM in the preliminary phase [48]. It was also suggested that patients with LDH may sometimes present with a forward-bending posture while walking, owing to radiculopathy that affected the sagittal balance resulting in tonic contraction of the surrounding lumbopelvic muscles [51]. This might explain the increases of mean values of APSI and OSI found in LR group indicating an increased motion from baseline level, hence a higher sway and poorer dynamic balance. Our results come in line with multiple studies that observed a pronounced anteroposterior sway with higher ankle stiffness in patients with LBP [52–54]. They suggested it may be seen as a compensatory mechanism to enhance sensory discrimination and thereby compensate for diminished proprioceptive input from the lumbar spine and trunk muscles owing to long-term neurological adaptations and the deterioration of the feedback loop [54].

Claeys *et al.* [47] found that individuals with LBP demonstrated a decreased use of lumbar proprioceptive inputs and performed the sit-to-stance-to-sit movement significantly slower than healthy individuals. They suggested that this slower performance of the total task was the result of a decrease in speed during the preliminary (transition) stage and not during the focal movement stage, owing to the switch of direction of the COM of the body in the opposite direction [47]. These results come in line with our study, where it was found that individuals with LR took more time to complete the DLOS test. In addition, DC mean value had lower scores, indicating poor dynamic stability. Our results seem to fit the pain adaptation model well, postulating that pain owing to radiculopathy reduces activation of agonist muscles and increases activation of antagonist's muscles [55]. Such muscle control change would decrease movement velocity and range of motion, to prevent mechanical provocation of pain [55–57].Moreover, our results come in line with Kuai *et al.* [58] where they found that individuals with LDH displayed more muscle activities and larger intradiscal forces during trunk flexion and two types of picking up. They concluded that these changes might be a compensatory response to relieve pain and improve spinal stability. However, these responses further burdened the trunk musculature, passive soft tissue, and spinal structure during functional tasks [58]. Another argument has proposed that the endpoint of chronic pain consists of structural remodeling processes in the CNS that open new pathways for nociceptive information and cause pain to persist over the long term [59]. Taken together, these data postulate that adaptive changes of the sensorimotor system [60] and alteration in brain function may be the mechanisms that underpin the problem of LR [61,62]. Thus, there are reasons to believe that these adaptations may lead to the deficits found in both static and dynamic postural balance in LR persisting for more than 3 months.

The results of this study may have some clinical significance. First, the rehabilitation of the proprioceptive deficiencies in the lumbosacral region in individuals with LDH and LR needs further attention. Exercises must depend more on back and lower extremity muscle proprioceptive inputs in static and dynamic postural conditions. In addition, the relationship between lumbopelvic muscles and complex movements in the cardinal planes should be addressed. Further work needs to focus on the possible mechanisms behind postural balance deficiencies in LR.

Conclusion

Patients with LDH associated with radiculopathy demonstrated some significant differences from control participants in terms of time to complete a test, sequencing, and overall static and dynamic balance control.

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Conflicts of interest

There are no conflicts of interest.

References

1 Konstantinou K, Dunn K, Ogollah R, Vogel S, Hay E. Characteristics of patients with low back and leg pain seeking treatment in primary care:

baseline results from the ATLAS cohort study. BMC Musculoskelet Disord 2015; 16:332.

2 Kelsey J, White A. Epidemiology of low back pain. Spine 1980; 6:133–142.

3 Frymoyer J. Back pain and sciatica. N Engl J Med 1988; 318:291–300.

4 Arts M, Peul W. Timing and minimal access surgery for sciatica: A summary of two randomized trials. Acta Neurochir 2011; 153:967–974.

5 Frymoyer J, Moskowitz R. Spinal degeneration. Pathogenesis and medical management. In: Frymoyer JW, editor. The adult spine: principles and practice. New York, NY: Raven 1991. pp. 611–634.

6 Kennedy D, Noh M. The role of core stabilization in lumbosacral radiculopathy. Phys Med Rehabil Clin N Am 2011; 22:91–103.

7 Van Boxem K, Cheng J., Patijn J, Kleef V, Lstaster A, Mekhail N, Zundert V. Lumbosacral radicular pain evidence-based medicine. Pain Pract 2010; 10:339–258.

8 Mens J, Chavannes A, Koes B, Lubbers W, Ostelo R, Spinnewijn W, Kolnaar B. NHG standard lumbosacral radicular syndrome. NHG Stand 2009; 4:436–448.

9 Manchikanti L, Singh V, Falco J, Benyamin M, Hirsch A. Epidemiology of low back pain in adults. Neuromodulation 2014; 17;3–10.

10 Carver S, Kiemel T, Jeka J. Modeling the dynamics of sensory reweighting. Biol Cybern 2006; 95:123–134.

11 Madeleine P, Prietzel H, Svarrer H, Arendt-Nielsen L. Quantitative posturography in altered sensory conditions: a way to assess balance instability in patients with chronic whiplash injury. Arch Phys Med Rehabil 2004; 85:432–438.

12 Mazzocchio R, Scarfo G, Mariottini A, Muzii V, Palma L. Recruitment curve of the soleus H-reflex in chronic back pain and lumbosacral radiculopathy. BMC Musculoskelet Disord 2001; 2:4.

13 Sipko T, Kuczynski M. The effect of chronic pain intensity on the stability limits in patients with low back pain. J Manipulative Physiol Ther 2013; 36:612–618.

14 Ruhe A, Fejer R, Walker B. Center of pressure excursion as a measure of balance performance in patients with non-specific low back pain compared to healthy controls: a systematic review of the literature. Eur Spine J 2011; 20:358–368.

15 Frost L, Bijman M, Strzalkowski J, Bent R, Brown M. Deficits in foot skin sensation are related to alterations in balance control in chronic low back patients experiencing clinical signs of lumbar nerve root impingement. Gait Posture 2015; 41:923–928.

16 Frost L, Stephen M. Muscle activation timing and balance response in chronic lower back pain patients with associated radiculopathy. Clin Biomech 2016; 32:124–130.

17 Duncan P, Weiner D, Chandler J, Studenski S. Functional reach: a new clinical measure of balance. J Gerontol 1990; 45:192–197.

18 Uchiyama M, Demura S, Shin S. Is there a relationship between the functional reach test and flexibility? Adv Phys Educ 2011; 11:11–15.

19 Joshi R, Rathi M, Palekar S, Sadhale S. Prediction of risk of fall in community-dwelling elderly population-a survey. Int J Pharm Bio Sci 2016; 7:1154–1157.

20 Karimi N, Ebrahim I, Kahrizi S, Torkaman G. Evaluation of postural balance using the biodex balance system in subjects with and without low back pain. Pak J Med Sci 2008; 24:372–377.

21 Cachupe W, Shifflett B, Kahanov L, Wughalter E. Reliability of Biodex Balance System measures. Meas Phys Edu and Exerc Sci. 2001; 5: 97–108.

22 Schmitz R, Arnold B. Intertester and intratester reliability of a dynamic balance protocol using the Biodex Stability System. J Sport Rehabil 1998; 7:95–102.

23 Baldwin S, vanArnam T, Ploutz-Snyder L. Reliability of dynamic bilateral postural stability on the Biodex Stability System in older adults. In: MARC Annual Meeting. Mid-atlantic chapter of the American College of Sports Medicine. Indianapolis, USA: Symposium Book; 2003. 44–52.

24 Pincivero DM, Lephart SM, Henry T. Learning effects and reliability of the Biodex Stability System. J Athl Train 1995; 30:S35.

25 Aydog ST, Aydog E, Çakci A, Doral MN. Reproducibility of postural stability score in blind athletes. Isokinet Exerc Sci 2004; 12:229–232.

26 Aydo E, Bal A, Aydo S, Çakci A. Evaluation of dynamic postural balance using the Biodex Stability System in rheumatoid arthritis patients. Clin Rheumatol 2006; 25:462–467.

27 Testerman C, Griend RV. Evaluation of ankle instability using the Biodex Stability Systems. Foot Ankle Int 1999; 20:317–321.

28 Hinman M. Factors affecting reliability of the Biodex Balance System: a summary of four studies. J Sport Rehabil 2000; 9:240–252.

29 Pereira H, Campos T, Santos M, Cardoso J, Garcia M, Cohen M. Influence of knee position on the postural stability index registered by the Biodex Stability System. Gait Posture 2008; 28:668–672.

30 Sherafat S, Salavati M, Ebrahimi Takamjani I, Akhbari B, Mohammadirad S, Mazaheri M, Negahban H. Intrasession and intersession reliability of postural control in participants with and without nonspecific low back pain using the Biodex Balance System. J Manipulative Physiol Ther 2013; 36:111–118.

31 Bisson E, Ewen D, Lajoie Y, Bilodeau M. Effects of ankle and hip muscle fatigue on postural sway and attentional demands during unipedal stance. Gait Posture 2011; 33:83–87.

32 Liu-Ambrose T, Eng J, Khan K, Mallinson A, Carter N, McKay H. The influence of back pain on balance and functional mobility in 65-75-year-old women with osteoporosis. Osteoporos Int 2002; 13:868–873.

33 Mok N, Brauer S, Hodges P. Hip strategy for balance control in quiet standing is reduced in people with low back pain. Spine 2004; 29:107–112.

34 Mok N, Brauer S, Hodges P. Failure to use movement in postural strategies leads to increased spinal displacement in low back pain. Spine 2007; 32:537–543.

35 Henry S, Hitt J, Jones S, Bunn J. Decreased limits of stability in response to postural perturbations in subjects with low back pain. Clin Biomech (Bristol, Avon) 2006; 21:881–892.

36 Radebold A, Cholewicki J, Polzhofer G, Greene H. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. Spine 2001; 26:724–730.

37 Brumagne S, Cordo P, Lysens R, Verschueren S, Swinnen S. The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. Spine 2000; 25:989–994.

38 Claeys K, Brumagne S, Dankaerts W, Kiers H, Janssens L. Decreased variability in postural control strategies in young people with non-specific low back pain is associated with altered proprioceptive reweighting. Eur J Appl Physiol 2011; 111:15–123.

39 Descarreaux M, Blouin JS, Teasdale N. Repositioning accuracy and movement parameters in low back pain subjects and healthy control subjects. Eur Spine J 2005; 14:185–191.

40 Brumagne S, Cordo P, Verschueren S. Proprioceptive weighting changes in persons with low back pain and elderly persons during upright standing. Neurosci Lett 2004; 366:63–66.

41 Brumagne S, Janssens L, Janssens E, Goddyn L. Altered postural control in anticipation of postural instability in persons with recurrent low back pain. Gait Posture 2008; 28:657–662.

42 Moseley G, Hodges P. Are the changes in postural control associated with low back pain caused by pain interference? Clin J Pain 2005; 21:323–329.

43 Rossi A, Decchi B, Ginanneschi F. Presynaptic excitability changes of group Iafibres to muscle nociceptive stimulation in humans. Brain Res 1999; 818:12–22.

44 Rossi S, della Volpe R, Ginanneschi F, Ulivelli M, Bartalini S, Spidalieri R, Rossi A. Early somatosensory processing during tonic muscle pain in humans: relation to loss of proprioception and motor 'defensive' strategies. Clin Neurophysiol 2003; 114:1351–1358.

45 Sibley K, Carpenter M, Perry J, Frank J. Effects of postural anxiety on the soleus H-reflex. Hum Mov Sci 2007; 26:103–112.

46 Capra N, Ro J. Experimental muscle pain produces central modulation of proprioceptive signals arising from jaw muscle spindles. Pain 2000; 86:151–162.

47 Claeys K, Dankaerts W, Janssens L, Brumagne S. Altered preparatory pelvic control during the sit-to-stance-to-sit movement in people with nonspecific low back pain, J Electromyogr Kinesiol 2012; 22:821–828.

48 Cordo P, Gurfinkel V. Motor coordination can be fully understood only by studying complex movements. Prog Brain Res 2004; 143:29–38.

49 Cordo P, Hodges P, Smith T, Brumagne S, Gurfinkel V. Scaling and non-scaling of muscle activity, kinematics, and dynamics in sit-ups with different degrees of difficulty. J Electromyogr Kinesiol 2006; 16:506–521.

50 Shum G, Crosbie J, Lee R. Energy transfer across the lumbosacral and lower-extremity joints in patients with low back pain during sit-to-stand. Arch Phys Med Rehabil 2009; 90:127–135.

51 Endo K, Suzuki H, Tanaka H, Kang Y, Yamamoto K. Sagittal spinal alignment in patients with lumbar disc herniation. Eur Spine J 2010; 19:435–438.

52 Hamaoui A, Mc Do, Poupard L, Bouisset S. Does respiration perturb body balance more in chronic low back pain subjects than in healthy subjects? Clin Biomech (Bristol, Avon) 2002; 17:548–550.

53 Hamaoui A, Do MC, Bouisset S. Postural sway increase in low back pain subjects is not related to reduced spine range of motion. Neurosci Lett 2004; 357:135–138.

54 Popa T, Bonifazi M, Della Volpe R, Rossi A, Mazzocchio R. Adaptive changes in postural strategy selection in chronic low back pain. Exp Brain Res 2007; 177:411–418.

55 Lund J, Donga R, Widmer C, Stohler C. The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. Can J Physiol Pharmacol 1991; 69:683–694.

56 Van Dieën H, Selen P, Cholewicki J. Trunk muscle activation in low-back pain patients, an analysis of the literature. J Electromyogr Kinesiol 2003; 13:333–351.

57 Hodges W, Moseley L. Pain and motor control of the lumbopelvic region: effect and possible mechanisms. J Electromyogr Kinesiol 2003; 13:361–370.

58 Kuai S, Zhou W, Liao Z, Ji R, Guo D, Zhang R, Liu W. Influences of lumbar disc herniation on the kinematics in multi-segmental spine, pelvis, and lower extremities during five activities of daily living. BMC Musculoskelet Disord 2017; 18:216.

59 Mense S. Muscle pain: mechanisms and clinical significance. Dtsch Arztebl Int 2008; 105:214–219.

60 Tsao H, Galea P, Hodges P. Driving plasticity in the motor cortex in recurrent low back pain. Eur J Pain 2010; 14:832–839.

61 Schmidt-Wilcke T, Leinisch E, Gänssbauer S, Draganski B, Bogdahn U, Altmeppen J, May A. Affective components and intensity of pain correlate with structural differences in gray matter in chronic back pain patients. Pain 2006; 125:89–97.

62 Apkarian V, Sosa Y, Sonty S, Levy M, Harden N, Parrish B, Gitelman R. Chronic back pain is associated with decreased prefrontal and thalamic gray matter density. J Neurosci 2004; 24:10410–10415.