



ELSEVIER  
SAUNDERS

Clin Sports Med 27 (2008) 449–462

# CLINICS IN SPORTS MEDICINE

## Neuromuscular Consequences of Low Back Pain and Core Dysfunction

Robert R. Hammill, MA, ATC<sup>a</sup>,  
James R. Beazell, PT, DPT, OCS, FAAOMPT, ATC<sup>b</sup>,  
Joseph M. Hart, PhD, ATC<sup>c,\*</sup>

<sup>a</sup>Health and Exercise Science, Bridgewater College, 402 East College Street,  
Box 166, Bridgewater, VA 22812, USA

<sup>b</sup>University of Virginia-Healthsouth, 545 Ray C. Hunt Drive, Suite 210, PO Box 800105,  
Charlottesville, VA 22903, USA

<sup>c</sup>Department of Orthopaedic Surgery, University of Virginia, 400 Ray C. Hunt Drive,  
Suite 330, PO Box 800159, Charlottesville, VA 22908, USA

Low back pain (LBP) remains a significant health care issue with reportedly over 50% prevalence in the general population and over 70% likely to experience at least one episode of LBP during in their lifetime [1,2]. The annual incidence has been estimated to involve almost half of the population, resulting in over \$40 billion in economic costs in the form of medical treatment and lost wages [3,4]. The social and economic impact of LBP is evident in the literature and in practice. Considering the high prevalence of LBP, it is not surprising that 2% of the United States workforce are compensated for back injuries each year [4], suggesting an enormous economic burden reaching \$25 billion annually [5].

The prevalence of nonspecific LBP may vary because of vague diagnostic and research-based definitions of nonspecific LBP and the myriad of possible etiologies and associated comorbidities [6,7]. Regardless of these issues, nonspecific LBP presents a major clinical problem because of the likelihood of high cost [5], limited activity levels [8], and recurrence [9]. Recurrent episodes, particularly those that cause modified or limited activity levels in people who choose to or who must remain active.

LBP that does not require surgical intervention presents a clinical dilemma for the treating physicians and therapists. Nonspecific LBP has been defined as a recurring, benign, and self-limiting condition [10], but it causes considerable pain for patients and reduced quality of life, demanding a multidisciplinary treatment strategy. Although many treatment strategies for nonspecific LBP have been studied at length, the recurrent nature of nonspecific LBP suggests that optimal and most effective treatment strategies have yet to be defined

\*Corresponding author. *E-mail address:* joehart@virginia.edu (J.M. Hart).

clearly. It is important for clinicians to progress through a careful process of evaluation and ruling out to reach the appropriate diagnosis and make appropriate treatment recommendations. An understanding of underlying structural and neuromuscular components contributing to the development of LBP and the consequences of such injury in active individuals warrants consideration when developing treatment strategies.

This article discusses neuromuscular deficits that are present in people who have LBP. Additionally, it discusses how stability of the lumbar, pelvic, and hip regions (collectively called the core) contributes to neuromuscular deficits in people who have LBP.

### **SUBJECTIVE MEASURES OF LOW BACK PAIN SEVERITY**

The Oswestry Disability Index (ODI) originally was presented in the literature in 1980 in a small sample [11] and later modified [12]. Its current form functions to identify disturbances in activities of daily living caused by LBP. The ODI was developed to define low back disability and thus assess changes associated with treatment of an LBP population. It is the most commonly used functional outcome questionnaire, representing an estimated 59% of all randomized control trials that use similar tools [13]. The original study used only 25 participants, and each was retested within 24 hours. Despite the confounding changes that can occur in a symptomatic population over time, excellent reliability has been reported for its one day test–retest reliability [14], with naturally reduced reliability for retest after 1 week [15].

To complete the ODI, subjects rate their disability caused by LBP in 10 categories (pain intensity, personal care, lifting, walking, sitting, standing, sleeping, social life, traveling, and homemaking/employment). Some versions include a question about sex life instead of homemaking/employment. Numeric values are assigned to the disability description that is selected by the subject/patient, where low scores indicate less perceived disability, and high scores indicate greater disability. The total score is tallied and normalized to a percentage, where smaller percentages indicate less disability. Scores under 20% indicate minimal functional disability; 20% to 40% scores indicate moderate disability, and 40%–60% scores indicate a severe disability [11]. People who have chronic LBP, and people without low back pain have been compared using the ODI. A weighted mean of 43.3 for chronic LBP sufferers and 10.2 for people without low back pain has been reported previously [16]. Therefore it is possible for a person to perceive disability according to the ODI without having a history.

The Roland-Morris Disability Questionnaire (RDQ) is a measure of health status relative to disability caused by LBP [17]. The RDQ lists 24 statements regarding disability caused by LBP. Subjects/patients select which statements pertain to them (ie, which statements they perceive to be true for them at the time they are completing the questionnaire). The range of scores is 0 to 24, with 0 indicating no disability. The test–retest reliability originally was reported to be very good ( $r = .91$ ) if administered on the same day, with reduced reliability over time [18], and it is sensitive to changes in patients' LBP [19].

A complete side-by-side comparison of the ODI and RDQ has been presented previously [20].

Both the ODI and RDQ are short, simple surveys that are effective in describing current state and changes in disability caused by LBP. In previous research, subject/patient interview/history-taking has been used to identify patients who have recurring LBP. For example, people reporting more than three episodes of LBP in the past year and/or more than five lifetime episodes have been identified as a group with a history of recurring episodes of LBP, where an episode of LBP was defined as that which causes a limitation or modification to daily, routine activities [21–23]. People who have recurrent LBP according to these criteria and who report to be recreationally active have been found to respond differently following fatiguing lumbar paraspinal exercise [21–24]. Proper identification of LBP for research purposes is of utmost importance and may differ based on the research question and study design. For example, the subjective rating scale may need to match the primary outcome of the study. Although the ODI may work well for a young and active population, quantifying the number of previous episodes of LBP has successfully identified neuromuscular differences compared with healthy control subjects (average age of low back pain subjects was approximately 22 years) [21,23]. No specific subjective rating scale, however, has been developed and validated for a young and athletic population.

### **RISK FOR DEVELOPING LOW BACK PAIN**

The etiology of chronic low back dysfunction has been described as 70% from internal disc disruption, zygapophyseal (facet) joint pain, and sacroiliac joint pain [25]. LBP has been attributed to sacroiliac joint dysfunction, accounting for up to 15% of LBP [25]. Ten percent to 40% of LBP cases arise from lumbar facet joints [26]; however, LBP arising from a combination of disc and facet joint is rare [27]. The relative contribution of the sacrococcygeal joint is not well-reported in the literature; however, it has been proposed that dysfunction of the coccyx also can contribute to LBP [28].

The likelihood of experiencing LBP increases with age, where the peak prevalence of LBP occurs in people aged 55 to 64 years in the United States but seems to cause the greatest level of activity level limitations in persons younger than 45 years [4,29]. The likelihood of experiencing LBP also increases with time, where lifetime prevalence is highest, indicating 85% to 90% of the population are likely to experience an episode of LBP in their lifetime, and about 2% to 5% of the population report experiencing an episode of back pain at least once every year [30–32]. A person who has an episode of recurrent LBP experiences a 60% to 70% recovery rate by 6 weeks and a 80% to 90% recovery rate by 12 weeks following onset [4]. Residual LBP symptoms and recurrence, however, are common. Longer-duration LBP may reduce the likelihood of returning to work or activity considerably [4]. Lengthy durations of LBP symptoms also reduce the likelihood of recovery. Patients reporting symptoms for more than 6 months have 50% chance of returning to work and may never

experience pain-free periods of uninterrupted work greater than 2 years [33]. This suggests that recurrent LBP and chronic LBP considerably limit functional ability and may have a profound effect on quality of life.

Previous LBP seems to be the best predictor for the likelihood of experiencing a future episode of LBP [34,35]. Physical and biological measures, however, also have proven valuable tools for predicting LBP. Weakness and imbalances in the muscles that surround the hips, spine, and pelvis [36–38]; poor abdominal muscle endurance [39]; hamstring tightness [39–41]; poor spinal flexibility [39,41]; and reduced lumbar lordosis [41] are associated with risk for developing episodes of LBP. Although it is likely that a different combination of factors influences the development of each episode of LBP, recurring episodes pose a major clinical dilemma because of the risk for chronic disability. Therefore, recurrent, nonspecific and nonsurgical LBP presents a challenge to the surgical or rehabilitation team, because pinpointing an underlying cause is extremely difficult.

Panjabi [42] has put forth the concept of clinical instability. This is not instability that an orthopedist would have to treat surgically but a lack of segmental control of the lumbar spine. An intricate interplay between the osseoligamentous system, the myofascial system, and the neural control system works together to provide stability to the lumbar spine and surrounding areas such as the hips and pelvis. Disruption in any of these systems will lead to a lack of segmental control. The ability to maintain the spinal segment in a neutral zone is important for maintaining overall stability and preventing spinal pain. Stability of most joints in the body is maintained by a combination of the different tissues including the osseoligamentous system, muscular system, and the neuromuscular system [42–46]. The ability of the patient to maintain an efficient coordination between these systems will allow him or her to function without undue stress on other systems.

## LOW BACK PAIN AND CORE STABILITY

Practitioners have been inundated for some time now regarding the importance of the core. The core of the body has been identified as the structures about the lumbo-pelvic-hip complex [47]. Active and passive structures in this area provide dynamic and static stability to this area of the body, which provides an essential base for appendicular movement. Core stability is instantaneous and relies heavily on muscular capacity (ie, endurance) and neuromuscular control [48]. An intricate relationship between these factors provides core stability and allows for appropriate control of movement and positioning of the trunk over the pelvis and legs [49] during activity, which, in turn, provides a stable base for extremity movement and efficient absorption of forces transmitted through the extremities during complex multijoint activities.

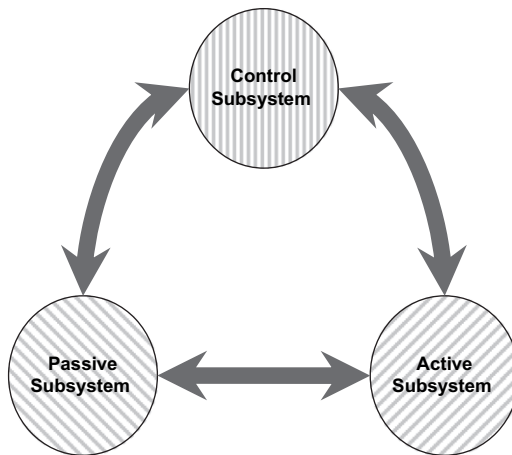
There is a distinction between muscles that provide gross movement and those that provide intrinsic stability of the various segments of the spine. Bergmark [50] suggested that most muscles that act on the trunk can be categorized into local or global divisions. The local division can be defined as those muscles that act

locally, typically across just one or two joints, and function to provide segmental stability [50]. Conversely, the global division may be defined as muscles that act to provide gross motion and contribute very little to segmental stability [50].

Recent literature supports the theory that recurrent, nonspecific LBP is the result of inefficient neuromuscular control of the transverse abdominus muscle [51–53]. The integrity of the muscle tissue is not faulty, but the way people who have LBP plan movements is not effective. The transverse abdominus muscle should become more active before the prime mover of any limb. In people who have LBP, this is not the case. Essentially, the way a person with LBP prepares for movement is at fault [51–53]. In the presence of inappropriate and/or aberrant muscle activity caused by poor core stability, potentially detrimental body positions and neuromuscular adaptations may prevail as the person who has LBP continues with activities of daily living because of necessity or persistence. These adaptations may explain the recurrent nature of LBP and may be discouraging to the LBP sufferer, resulting in modified or limited activity levels. Inappropriate neuromuscular or positional adaptations also may expose joint surfaces to excessive and unusual forces, thereby exposing to further injury local to the site of dysfunction (ie, the core) and ultimately reduce quality of life.

### NEUROMUSCULAR CHARACTERISTICS OF PERSONS WITH LOW BACK PAIN

Panjabi [54] presented three subsystems for spinal stability. Ligaments and bones comprise the passive subsystem; muscle makes up the active subsystem, and the nervous tissue serves as the control subsystem (Fig. 1). Function of the control subsystem in the neutral zone of the spine's range of motion [43,44] is



**Fig. 1.** Panjabi's theoretic model of the three subsystems associated with control of the spine. (Data from Panjabi MM. The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. *J Spinal Disord* 1992;5(4):390–6. Panjabi MM.) The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *J Spinal Disord* 1992;5(4):383–9.

integral to preventing LBP. It is at this neutral zone in the range of motion where the passive structures (passive subsystem) of the spine contribute little to each segment's stability [55]. Faulty control of the deep local muscles may lead to a back injury or recurrence [55,56].

Nonspecific LBP often is attributed to weak abdominal musculature. Although this may be true, a mass of evidence points toward a faulty motor program. The literature indicates that a preactivation of the transverse abdominus before peripheral movements of the upper [52,57] or lower extremity [52] occurs in healthy people (without low back pain). The fact that some of the stabilizing muscles of the trunk are activated in a feed-forward fashion suggests they play an integral role in the performance of peripheral movements and protecting the spine in preparation for movement. In the case of subjects who have LBP, the transverse abdominus muscle does not activate in advance [43]. This delay may put stress on the passive structures of the spine and pelvis at the initiation of movement, as the spinal segments and pelvis are not being influenced by dynamic or static stabilizers.

The intervertebral joints of the lumbar spine require a cocontraction for stability [58]. In normal stance, the lumbar segments are in an open-packed position because of the lack of passive restraint at this point in space. This point in space where lumbar segments have no passive restraints acting on them has been termed the neutral zone [59] and requires muscle contraction to stabilize. The lumbar vertebrae and pelvic girdle can be stabilized effectively by cocontracting the transverse abdominus and multifidus muscles [60–63]. This technique may be valuable for two reasons. One, it will increase intra-abdominal pressure, thereby stabilizing the lumbar segments [60] and, two, it may compress the sacroiliac joints [64]. Other muscles may contribute to sacroiliac joint stability. These muscles include the gluteus maximus, erector spinae, biceps femoris, and latissimus dorsi [58], but the extent to which each of these contributes the stability of the sacroiliac joint remains speculative.

Conversely, cocontractions of the superficial abdominal musculature (ie, the rectus abdominus and external obliques) also may provide significant stability to lumbar segments, but it is probably not as efficient as cocontractions of the transverse abdominus and multifidus muscles [58]. This hypothesis was generated by investigating the intra-abdominal pressure change associated with each contraction type [65]. Regardless, cocontractions that recruit abdominal muscles may generate intra-abdominal pressure to stabilize the lumbar segments during most movements [66–68].

## **LOW BACK PAIN, CORE STABILITY, AND EXERCISE**

Persons who have LBP tend to have weakness and imbalances in the muscles that surround the hips and pelvis [36–38] which may absorb impact forces of running and walking less efficiently. The failure to absorb the forces of gait with weak lower extremity muscles may impose high demand on lumbar spine-stabilizing muscles during gait and exercise [36]. It is concerning that people who have LBP exhibit increased lumbar paraspinal muscle activity

during gait [69] and lifting exertions [70]. Higher demand placed on weaker, fatigable, unbalanced, and possibly inhibited muscles may prevent patients from being active or result in harmful neuromuscular or postural adaptations during exercise. During prolonged exercise, neuromuscular adaptations in people who have LBP may alter gait mechanics placing lower extremity joints, including the sacral and lumbar spine, at risk for injury or degeneration.

Patients who have LBP commonly exhibit fear of pain or reinjury because of movement [71,72] possibly resulting in gait adaptations including slower walking speeds [67]. Accordingly, LBP sufferers perceive they are less physically active compared with their preinjury condition [8] and may continue to perceive pain and disability related to back pain a year after onset despite the lack of physician consultation after 3 months [73]. This is of concern for LBP sufferers who want, or need, to maintain an active lifestyle. LBP sufferers may adapt to their pain and disability through coping mechanisms to continue their normal activities possibly through self-treating/medicating or through modification/limitation of activities. Formal patient education and guided exercise intervention can reduce frequency of LBP recurrence and severity of individual episodes of LBP [74] and help chronic LBP sufferers to return to work or normal activities [75–77].

There is an abundance of information in the medical literature about kinematic variables and specific temporal patterns of gait in people who have LBP, including stride length, cadence and speed. People who have LBP tend to walk at slower speeds [68,78] and tend to take shorter strides during gait [79]. Kinematic differences during activity in people who have LBP exist, including differences in hip range of motion and increased stride–stride variability [78,80]. People who have LBP also may exhibit lateral trunk list (deviation of the trunk over the pelvis) [79] and reduced lumbar lordosis [41] that likely will cause a more anterior position of the trunk. Changes in trunk positioning during standing or during gait is of concern for people who have LBP because of the possibility of altered spatial location of the body's center of gravity, which may result in a reorganization of lower extremity movements and forces during gait. For example, LBP sufferers walk with reduced hip extension range of motion [81] and may use different adaptive mechanisms while walking at more challenging, faster speeds [69], including increased lumbar muscle activity [82,83]. This increase in lumbar paraspinal muscle activity may be an adaptive mechanism for more efficient center of gravity location and avoid anterior and/or lateral deviations of the trunk.

Surface electromyography has been used to measure paraspinal muscle endurance during lumbar extension exercise as a shift in the median frequency (MedF) of the EMG power spectrum [84]. MedF shifts provide an “appropriate representation of biochemical events” during sustained, fatiguing contractions and describe a change in recruitment from high frequency to low frequency motor units [85]. High fatigability (quicker rates of MedF shift during fatiguing exercise) of the lumbar paraspinal muscles is associated with the presence of and risk for developing LBP [85]. Therefore it is likely that people who have a history

of LBP will exhibit different rates of fatigue in the paraspinal muscles during prolonged exercise. Because the force producing capacity of a muscle is reduced as it fatigues, people who have LBP may require a compensatory strategy from surrounding muscles to maintain the desired level of activity or function.

## MODELS OF CORE INSTABILITY

Poor lumbar extension endurance, measured as the duration of sustained isometric contraction of the lumbar paraspinals (ie, the Biering-Sorensen test), has been identified as a risk factor for developing LBP [86–88]. Likewise, people who have current LBP [89] or a history of LBP [90] exhibit poor lumbar extension endurance compared with controls. Therefore, people who have poor lumbar spinal endurance may progress quickly to a state of core instability during exercise. These models may contribute to the understanding of the recurrent nature of LBP. Research using isolated lumbar paraspinal fatigue [91] creates a simulated condition of poor core stability to study potential adaptations during exercise in controlled settings.

In healthy individuals, localized lumbar paraspinal fatigue caused deteriorated standing postural control [92] and produced a forward-leaned position of the body, defined as an anterior excursion of the center of pressure and center of mass during static stance [91,92]. In addition, the variability in anterior–posterior and medial–lateral trunk displacement following lumbar paraspinal fatigue was increased significantly during static stance [92]. This finding supports previous findings that lumbar paraspinal fatigue causes deteriorated trunk proprioception [93], which also has been observed in persons with chronic low back pain [94]. These neuromuscular changes suggest that fatigue in muscles that stabilize the core is accompanied by a coping mechanism that may increase risk for falls, thereby increasing the risk for injury and reducing quality of life. In people who have recurrent LBP and core instability, the lumbar spine musculature is likely to fatigue quicker, relative to surrounding muscles, during exercise or activity. Therefore, the body may adapt, through positional and neuromuscular changes during gait, to maintain symmetry and balance while preserving function.

There are also neuromuscular adaptations in response to local lumbar paraspinal fatigue. Immediately following lumbar paraspinal fatigue, healthy subjects experienced a reduction in quadriceps neural activation (measured with the superimposed burst technique) [21,22,24], suggesting a reduced ability to voluntarily recruit quadriceps motor units, despite the fact that subjects did not experience quadriceps fatigue, and had healthy lower extremity joints. In addition, during jogging gait, healthy people experienced reduced knee joint torques during the loading phase of jogging gait immediately following lumbar paraspinal fatigue [24]. Reduced knee joint torques, suggesting a quadriceps avoidance strategy, has been observed in people who have knee joint injury or degeneration. A similar pattern, however, was observed in healthy people without knee injury. Finally, fatigue in the hamstring muscles explained variance in increased quadriceps inhibition following fatiguing isometric lumbar

extension exercise in people who had recurrent LBP, not controls [23]. This suggests that people who have LBP may be using their hamstring muscles more as an adaptive mechanism following lumbar fatigue, possibly because of existing weakness in those muscles.

### **CORE INSTABILITY AND LOWER EXTREMITY INJURY RISK**

The relationship between the lumbar/pelvic and hip regions with the remainder of the lower extremity is intuitive. Clinicians routinely evaluate knee, leg, ankle, and foot mechanics in people who have LBP. Likewise, it is common to evaluate for pelvic obliquities in the presence of lower extremity overuse syndromes and other injuries. In a recent review article, Wilson and colleagues [47] wrote, “core stability may provide several benefits to the musculoskeletal system, from maintaining low back health to preventing knee injury.” This statement is supported by recent knee injury epidemiologic studies suggesting that lower extremity injury risk may be explained partially by neuromuscular function of the core. Zazulak and colleagues [95] measured transverse plane trunk proprioception in athletes and followed them prospectively for 3 years. They reported a mean, statistically significant difference of  $0.7^\circ$  in transverse plane trunk active reposition error in females who experienced knee joint injuries compared with females who did not sustain knee injury during the follow-up period. This small difference resulted in an 2.9-fold increased odds ratio for experiencing a knee injury for every degree in increased transverse plane active position error. In addition, small differences in maximum flexion, extension, and lateral trunk displacement in response to a sudden perturbation was a significant predictor for knee ligament injury, with 91% sensitivity and 68% specificity, indicating that greater trunk displacements predict knee ligament injury [96]. Therefore, small differences in core proprioception and neuromuscular control may have profound effects on lower extremity injury risk in active populations.

Changes in posture in the presence of core instability may exist during exercise. It has been proposed that people who have chronic LBP exhibit more forward-leaned postures, which may be because of the loss of lumbar lordosis commonly seen in people who have LBP. In theory, a forward-leaned posture or a reduction in trunk proprioception would cause a shift the center of mass, resulting in a reorganization of lower extremity moments during activity. For example, in the sagittal plane, a more anteriorly displaced center of mass would cause ground reaction forces to pass closer to the knee joint in the sagittal plane, resulting in altered knee joint mechanics. This is concerning since poor ground reaction force attenuation by eccentric quadriceps activity may lead to proximal transmission of forces through the knee, hip, and lumbar spine joints. This combined with the possibility of reduced quadriceps muscle activation (increased inhibition) in the presence of lumbar paraspinal fatigue leaving the musculature of the lumbar spine responsible to absorb an excessive amount of force. In the presence of core instability, the muscles surrounding the spine may be fatigued and/or unconditioned and unable to efficiently

provide stability. In essence, this creates an environment for the lumbar spine where excessive demand is placed on weak and inhibited muscles, placing the surrounding static stabilizing tissues of the lumbar spine, pelvis, and hips at risk for injury or degenerative processes. This cycle of neuromuscular, kinetic, and kinematic events may explain the recurrent nature of nonspecific low back pain in the active individual and is an area of future investigation involving models of core instability and exercise models with people who have LBP.

## SUMMARY

Recurrent and nonspecific LBP presents a clinical dilemma for the treating clinician, because it is a major source of pain, limited activity levels, and reduced quality of life in patients. Weakness, imbalance, poor endurance, and motor control in the muscles that stabilize the core are a likely culprit that can be addressed easily with a multidisciplinary strategy including rehabilitation and exercise prescription. Neuromuscular and postural adaptations occurring as a result of poor core stability may explain the recurrent nature of low back pain and may place lower extremity joints at risk for injury or degenerative processes.

## References

- [1] Lawrence JP, Greene HS, Grauer JN. Back pain in athletes. *J Am Acad Orthop Surg* 2006;14(13):726–35.
- [2] Papageorgiou AC, Croft PR, Ferry S, et al. Estimating the prevalence of low back pain in the general population. Evidence from the South Manchester Back Pain Survey. *Spine* 1995;20(17):1889–94.
- [3] Andersson G. The epidemiology of spinal disorders. In: Frymoyer J, editor. *The adult spine*. New York: Raven Press; 1997. p. 93–141.
- [4] Andersson GB. Epidemiological features of chronic low back pain. *Lancet* 1999;354(9178):581–5.
- [5] Frymoyer JW, Cats-Baril WL. An overview of the incidences and costs of low back pain. *Orthop Clin North Am* 1991;22(2):263–71.
- [6] Coste J, Spira A, Ducimetiere P, et al. Clinical and psychological diversity of nonspecific low back pain. A new approach towards the classification of clinical subgroups. *J Clin Epidemiol* 1991;44(11):1233–45.
- [7] Ozguler A, Leclerc A, Landre MF, et al. Individual and occupational determinants of low back pain according to various definitions of low back pain. *J Epidemiol Community Health* 2000;54(3):215–20.
- [8] Verbunt JA, Sieben JM, Seelen HA, et al. Decline in physical activity, disability, and pain-related fear in subacute low back pain. *Eur J Pain* 2005;9(4):417–25.
- [9] MacDonald MJ, Sorock GS, Volinn E, et al. A descriptive study of recurrent low back pain claims. *J Occup Environ Med* 1997;39(1):35–43.
- [10] Keller A, Hayden J, Bombardier C, et al. Effect sizes of nonsurgical treatments of nonspecific low back pain. *Eur Spine J* 2007;16(11):1776–88.
- [11] Fairbank JC, Pynsent PB. The Oswestry Disability Index. *Spine* 2000;25(22):2940–52 [discussion: 2952].
- [12] Meade T, Browne W, Mellows S, et al. Comparison of chiropractic and hospital outpatient management of low back pain: a feasibility study. *J Epidemiol Community Health* 1986;40:12–7.
- [13] Fairbank JC. The use of revised Oswestry Disability Questionnaire. *Spine* 2000;25(21):2846–7.

- [14] Gronblad M, Hupli M, Wennerstrand P, et al. Intercorrelation and test–retest reliability of the Pain Disability Index (PDI) and the Oswestry Disability Questionnaire (ODQ) and their correlation with pain intensity in low back pain patients. *Clin J Pain* 1993;9(3): 189–95.
- [15] Fairbank JC, Couper J, Davies JB, et al. The Oswestry Low Back Pain Disability Questionnaire. *Physiotherapy* 1980;66(8):271–3.
- [16] Roland M, Morris R. A study of the natural history of back pain. Part I: development of a reliable and sensitive measure of disability in low back pain. *Spine* 1983;8(2):141–4.
- [17] Roland M, Morris R. A study of the natural history of low back pain. Part II: development of guidelines for trials of treatment in primary care. *Spine* 1983;8(2):145–50.
- [18] Jensen MP, Strom SE, Turner JA, et al. Validity of the Sickness Impact Profile Roland scale as a measure of dysfunction in chronic pain patients. *Pain* 1992;50(2):157–62.
- [19] Beurskens AJ, de Vet HC, Koke AJ. Responsiveness of functional status in low back pain: a comparison of different instruments. *Pain* 1996;65(1):71–6.
- [20] Roland M, Fairbank J. The Roland-Morris Disability Questionnaire and the Oswestry Disability Questionnaire. *Spine* 2000;25(24):3115–24.
- [21] Hart JM, Fritz JM, Kerrigan DC, et al. Reduced quadriceps activation after lumbar paraspinal fatiguing exercise. *J Athl Train* 2006;41(1):79–86.
- [22] Hart JM, Fritz JM, Kerrigan DC, et al. Quadriceps inhibition after repetitive lumbar extension exercise in persons with a history of low back pain. *J Athl Train* 2006;41(3):264–9.
- [23] Hart JM, Kerrigan DC, Fritz JM, et al. Contribution of hamstring fatigue to quadriceps inhibition following lumbar extension exercise. *Journal of Sports Science and Medicine* 2006;2006(5):70–9.
- [24] Hart JM. Quadriceps inhibition and gait kinetics following fatiguing isometric lumbar paraspinal exercise [dissertation]. Charlottesville (VA): Sports Medicine/Athletic Training, University of Virginia; 2005.
- [25] Bogduk N. The anatomical basis for spinal pain syndromes. *J Manipulative Physiol Ther* 1995;18(9):603–5.
- [26] Dreyer SJ, Dreyfuss PH. Low back pain and the zygapophysial (facet) joints. *Arch Phys Med Rehabil* 1996;77(3):290–300.
- [27] Schwarzer AC, Aprill CN, Derby R, et al. The relative contributions of the disc and zygapophysial joint in chronic low back pain. *Spine* 1994;19(7):801–6.
- [28] Maigne JY, Lagache D, Doursounian L. Instability of the coccyx in coccydynia. *J Bone Joint Surg Br* 2000;82(7):1038–41.
- [29] Kent PM, Keating JL. The epidemiology of low back pain in primary care. *Chiropr Osteopat* 2005;13:13.
- [30] Trainor TJ, Wiesel SW. Epidemiology of back pain in the athlete. *Clin Sports Med* 2002;21(1):93–103.
- [31] Trainor TJ, Trainor MA. Etiology of low back pain in athletes. *Curr Sports Med Rep* 2004;3(1):41–6.
- [32] Bono CM. Low back pain in athletes. *J Bone Joint Surg Am* 2004;86-A(2):382–96.
- [33] Waddell G. Low back pain: a twentieth century health care enigma. *Spine* 1996;21(24): 2820–5.
- [34] Biering-Sorensen F. A prospective study of low back pain in a general population. I. Occurrence, recurrence, and aetiology. *Scand J Rehabil Med* 1983;15(2):71–9.
- [35] Wasiak R, Pransky G, Verma S, et al. Recurrence of low back pain: definition–sensitivity analysis using administrative data. *Spine* 2003;28(19):2283–91.
- [36] Nadler SF, Malanga GA, DePrince M, et al. The relationship between lower extremity injury, low back pain, and hip muscle strength in male and female collegiate athletes. *Clin J Sport Med* 2000;10(2):89–97.
- [37] Nadler SF, Malanga GA, Feinberg JH, et al. Relationship between hip muscle imbalance and occurrence of low back pain in collegiate athletes: a prospective study. *Am J Phys Med Rehabil* 2001;80(8):572–7.

- [38] Nadler SF, Malanga GA, Bartoli LA, et al. Hip muscle imbalance and low back pain in athletes: influence of core strengthening. *Med Sci Sports Exerc* 2002;34(1):9–16.
- [39] Jones MA, Stratton G, Reilly T, et al. Biological risk indicators for recurrent nonspecific low back pain in adolescents. *Br J Sports Med* 2005;39(3):137–40.
- [40] McClure PW, Esola M, Schreiber R, et al. Kinematic analysis of lumbar and hip motion while rising from a forward, flexed position in patients with and without a history of low back pain. *Spine* 1997;22(5):552–8.
- [41] Hultman G, Saraste H, Ohlsen H. Anthropometry, spinal canal width, and flexibility of the spine and hamstring muscles in 45–55-year-old men with and without low back pain. *J Spinal Disord* 1992;5(3):245–53.
- [42] Panjabi MM. Clinical spinal instability and low back pain. *J Electromyogr Kinesiol* 2003;13(4):371–9.
- [43] Panjabi MM. The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. *J Spinal Disord* 1992;5(4):390–6 [discussion: 397].
- [44] Panjabi MM. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *J Spinal Disord* 1992;5(4):383–9 [discussion: 397].
- [45] Comerford MJ, Mottram SL. Movement and stability dysfunction—contemporary developments. *Man Ther* 2001;6(1):15–26.
- [46] Comerford MJ, Mottram SL. Functional stability retraining: principles and strategies for managing mechanical dysfunction. *Man Ther* 2001;6(1):3–14.
- [47] Willson JD, Dougherty CP, Ireland ML, et al. Core stability and its relationship to lower extremity function and injury. *J Am Acad Orthop Surg* 2005;13(5):316–25.
- [48] Leetun DT, Ireland ML, Willson JD, et al. Core stability measures as risk factors for lower extremity injury in athletes. *Med Sci Sports Exerc* 2004;36(6):926–34.
- [49] Kibler WB, Press J, Sciascia A. The role of core stability in athletic function. *Sports Med* 2006;36(3):189–98.
- [50] Bergmark A. Stability of the lumbar spine. A study in mechanical engineering. *Acta Orthop Scand Suppl* 1989;230:1–54.
- [51] Hodges PW. Changes in motor planning of feedforward postural responses of the trunk muscles in low back pain. *Exp Brain Res* 2001;141(2):261–6.
- [52] Hodges PW, Richardson CA. Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb. *J Spinal Disord* 1998;11(1):46–56.
- [53] Hodges PW, Richardson CA. Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. *Arch Phys Med Rehabil* 1999;80(9):1005–12.
- [54] Panjabi MM. Lumbar spine instability: a biomechanical challenge. *Curr Orthop* 1994;8(2):100–5.
- [55] Cholewicki J, McGill SM. Lumbar posterior ligament involvement during extremely heavy lifts estimated from fluoroscopic measurements. *J Biomech* 1992;25(1):17–28.
- [56] Panjabi M, Abumi K, Duranceau J, et al. Spinal stability and intersegmental muscle forces. A biomechanical model. *Spine* 1989;14(2):194–200.
- [57] Hodges PW, Richardson CA. Contraction of the abdominal muscles associated with movement of the lower limb. *Phys Ther* 1997;77(2):132–42 [discussion: 142–14].
- [58] Cholewicki J, Juluru K, McGill SM. Intra-abdominal pressure mechanism for stabilizing the lumbar spine. *J Biomech* 1999;32(1):13–7.
- [59] Richardson CA, Jull GA. Muscle control—pain control. What exercises would you prescribe? *Man Ther* 1995;1(1):2–10.
- [60] Richardson CA, Snijders CJ, Hides JA, et al. The relation between the transversus abdominis muscles, sacroiliac joint mechanics, and low back pain. *Spine* 2002;27(4):399–405.
- [61] Richardson C, Jull G, Hodges P, et al. *Therapeutic exercise for spinal segmental stabilization in low back pain*. London: Churchill Livingstone; 1999.
- [62] Sahrman S. *Diagnosis and treatment of movement impairment syndromes*. St. Louis (MO): Mosby, Inc.; 2002.

- [63] Kennedy B. A muscle-bracing technique utilizing intra-abdominal pressure to stabilize the lumbar spine. *Aust J Physiother* 1965;11(3):102–6.
- [64] van Wingerden JP, Vleeming A, Buyruk HM, et al. Stabilization of the sacroiliac joint in vivo: verification of muscular contribution to force closure of the pelvis. *Eur Spine J* 2004;13(3):199–205.
- [65] Essendrop M, Schibye B, Hye-Knudsen C. Intra-abdominal pressure increases during exhausting back extension in humans. *Eur J Appl Physiol* 2002;87(2):167–73.
- [66] Lamoth CJ, Meijer OG, Daffertshofer A, et al. Effects of chronic low back pain on trunk coordination and back muscle activity during walking: changes in motor control. *Eur Spine J* 2006;15(1):23–40.
- [67] Al-Obaidi SM, Al-Zoabi B, Al-Shuwaie N, et al. The influence of pain and pain-related fear and disability beliefs on walking velocity in chronic low back pain. *Int J Rehabil Res* 2003;26(2):101–8.
- [68] Khodadadeh S, Eisenstein SM. Gait analysis of patients with low back pain before and after surgery. *Spine* 1993;18(11):1451–5.
- [69] Arendt-Nielsen L, Graven-Nielsen T, Sværre H, et al. The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. *Pain* 1996;64(2):231–40.
- [70] Ferguson SA, Marras WS, Burr DL, et al. Differences in motor recruitment and resulting kinematics between low back pain patients and asymptomatic participants during lifting exertions. *Clin Biomech (Bristol, Avon)* 2004;19(10):992–9.
- [71] Sieben JM, Vlaeyen JW, Tuerlinckx S, et al. Pain-related fear in acute low back pain: the first two weeks of a new episode. *Eur J Pain* 2002;6(3):229–37.
- [72] Sieben JM, Portegijs PJ, Vlaeyen JW, et al. Pain-related fear at the start of a new low back pain episode. *Eur J Pain* 2005;9(6):635–41.
- [73] Croft PR, Macfarlane GJ, Papageorgiou AC, et al. Outcome of low back pain in general practice: a prospective study. *BMJ* 1998;316(7141):1356–9.
- [74] Lonn JH, Glomsrod B, Soukup MG, et al. Active back school: prophylactic management for low back pain. A randomized, controlled, 1-year follow-up study. *Spine* 1999;24(9):865–71.
- [75] van Tulder MW, Malmivaara A, Esmail R, et al. Exercise therapy for low back pain. *Cochrane Database Syst Rev* 2000;(2):CD000335.
- [76] Casazza BA, Young JL, Herring SA. The role of exercise in the prevention and management of acute low back pain. *Occup Med* 1998;13(1):47–60.
- [77] van Tulder MW, Koes BW, Bouter LM. Conservative treatment of acute and chronic nonspecific low back pain. A systematic review of randomized controlled trials of the most common interventions. *Spine* 1997;22(18):2128–56.
- [78] Vogt L, Pfeifer K, Portscher M, et al. Influences of nonspecific low back pain on three-dimensional lumbar spine kinematics in locomotion. *Spine* 2001;26(17):1910–9.
- [79] Gillan MG, Ross JC, McLean IP, et al. The natural history of trunk list, its associated disability, and the influence of McKenzie management. *Eur Spine J* 1998;7(6):480–3.
- [80] Vogt L, Pfeifer K, Banzer W. Neuromuscular control of walking with chronic low back pain. *Man Ther* 2003;8(1):21–8.
- [81] Lamoth CJ, Daffertshofer A, Meijer OG, et al. How do persons with chronic low back pain speed up and slow down? Trunk–pelvis coordination and lumbar erector spinae activity during gait. *Gait Posture* 2006;23(2):230–9.
- [82] Dederling A, Nemeth G, Harms-Ringdahl K. Correlation between electromyographic spectral changes and subjective assessment of lumbar muscle fatigue in subjects without pain from the lower back. *Clin Biomech (Bristol, Avon)* 1999;14(2):103–11.
- [83] Kramer M, Ebert V, Kinzli L, et al. Surface electromyography of the paravertebral muscles in patients with chronic low back pain. *Arch Phys Med Rehabil* 2005;86(1):31–6.
- [84] Basmajian JV, De Luca CJ. *Muscles alive: their functions revealed by electromyography*. Baltimore (MD): Williams & Wilkins; 1985.

- [85] Mannion AF, Connolly B, Wood K, et al. The use of surface EMG power spectral analysis in the evaluation of back muscle function. *J Rehabil Res Dev* 1997;34(4):427–39.
- [86] Biering-Sorensen F. Physical measurements as risk indicators for low-back trouble over a one-year period. *Spine* 1984;9(2):106–19.
- [87] Biering-Sorensen F. A one-year prospective study of low back trouble in a general population. The prognostic value of low back history and physical measurements. *Dan Med Bull* 1984;31(5):362–75.
- [88] Biering-Sorensen F, Thomsen CE, Hilden J. Risk indicators for low back trouble. *Scand J Rehabil Med* 1989;21(3):151–7.
- [89] Latimer J, Maher CG, Refshauge K, et al. The reliability and validity of the Biering-Sorensen test in asymptomatic subjects and subjects reporting current or previous nonspecific low back pain. *Spine* 1999;24(20):2085–9 [discussion 2090].
- [90] Simmonds MJ, Olson SL, Jones S, et al. Psychometric characteristics and clinical usefulness of physical performance tests in patients with low back pain. *Spine* 1998;23(22):2412–21.
- [91] Davidson BS, Madigan ML, Nussbaum MA. Effects of lumbar extensor fatigue and fatigue rate on postural sway. *Eur J Appl Physiol* 2004;93(1-2):183–9.
- [92] Madigan ML, Davidson BS, Nussbaum MA. Postural sway and joint kinematics during quiet standing are affected by lumbar extensor fatigue. *Hum Mov Sci* 2006;25(6):788–99.
- [93] Taimela S, Kankaanpaa M, Luoto S. The effect of lumbar fatigue on the ability to sense a change in lumbar position. A controlled study. *Spine* 1999;24(13):1322–7.
- [94] O’Sullivan P, Mitchell BT, Bulich P, et al. The relationship between posture and back muscle endurance in industrial workers with flexion-related low back pain. *Man Ther* 2006;11(4):264–71.
- [95] Zazulak BT, Hewett TE, Reeves NP, et al. The effects of core proprioception on knee injury: a prospective biomechanical-epidemiological study. *Am J Sports Med* 2007;35(3):368–73.
- [96] Zazulak BT, Hewett TE, Reeves NP, et al. Deficits in Neuromuscular Control of the Trunk Predict Knee Injury Risk: A Prospective Biomechanical-Epidemiologic Study. *Am J Sports Med* 2007;35(7):1123–30.