Journal of Sport Rehabilitation, 1996, 5, 275-286

Strength and Flexibility Characteristics of Athletes With Chronic Low-Back Pain

Kathleen J. Ashmen, C. Buz Swanik, and Scott M. Lephart

The purpose of this study was to identify strength and flexibility deficits in subjects with chronic low-back pain (CLBP). Subjects were 16 female Division I athletes: 8 athletes who had experienced CLBP for at least 6 months prior to testing and a control group of 8 matched subjects. Athletes with neurological symptoms, previous back operations, and leg length discrepancies and those who were diagnosed with scoliosis, spondylolisthesis, or spondylolysis were excluded from this study. Variables assessed included abdominal strength, erector spinae endurance, hip flexion and extension endurance, torso lateral flexibility, and low-back flexibility. Strength and endurance were calculated as a function of time in seconds. Goniometric measurements were used to determine flexibility. Significant mean differences were found by using dependent *t* tests for abdominal strength, erector spinae endurance, hip extension, and right lateral flexion of the torso. The results validate the necessity for pelvic stabilization and indicate that strength and flexibility deficits vary among populations.

Chronic low-back pain (CLBP) of musculoskeletal origin appears to be increasingly common in competitive athletes whose sports are characterized by repetitive flexion, extension, and torsional maneuvers of the lumbar spine (e.g., gymnastics, swimming, and basketball) (1, 14, 17, 25, 33). Often these individuals continue to participate without intervention, until the CLBP is severe enough to eliminate the athletes from competition or restrict their activities of daily living. These symptoms usually subside 10–20 days after the initial onset, at which time some athletes resume competition and disregard their rehabilitation. However, several studies have reported that a large percentage (30–70%) of those who experience an acute bout of low-back pain will have recurrent episodes, creating this chronic condition (9, 20, 22, 29).

Clinical evidence supporting the rehabilitation protocols used for CLBP is contradictory or incomplete (9). These protocols are frequently based on theoretical models of mechanical dysfunction and elucidating symptoms (7, 9, 20, 28, 32). The factors responsible for impairment in patients with CLBP, whether physiological, psychological, or structural, have yet to be determined. Studies assessing treatment protocols for CLBP emphasize variables such as hamstring flexibility, low back flexibility, hip flexibility, lateral flexibility of the torso, abdominal strength, and erector spinae strength. These factors all contribute to pelvic stability through a complex interrelationship of muscle length and tension properties. Although successful management protocols have been reported, the reports have not included the experimental evidence necessary to support their claims (11, 17, 22, 23). These reports suggest that there is no single underlying dimension to the rehabilitative treatment prescribed, casting doubt on whether the treatment relieved CLBP or whether other nonorganic variables were involved (11, 22, 33–35).

Kathleen J. Ashmen is with the Neuromuscular Research Laboratory, Sports Medicine Program, 169 Trees Hall, University of Pittsburgh, Pittsburgh, PA 15261. C. Buz Swanik is a doctoral student in the Neuromuscular Research Laboratory, Sports Medicine Program, University of Pittsburgh. Scott M. Lephart is with the Neuromuscular Research Laboratory, Sports Medicine Program, University of Pittsburgh.

The purpose of this study was to identify strength and flexibility characteristics in patients with CLBP. The goal was to demonstrate clinically that deficits are present, thus validating the rationale given for the flexibility and strengthening exercises that clinicians utilize. It was hypothesized that bilateral inequalities would be noted in subjects with CLBP when compared to matched, healthy athletes.

Methods

Subjects

The subjects were 16 female Division I athletes (mean age = 19 years, mean weight = 146.5 lb), all of whom were fully participating in practice and competition in gymnastics, swimming, and basketball. The experimental group consisted of 8 of these athletes who were identified and evaluated by a certified athletic trainer as having a primary complaint of CLBP for at least 6 months prior to enrollment. These subjects were matched by position and sport to a healthy control group without incidence of back pain. Subjects with previous back operations or with evidence of scoliosis, spondylolisthesis, spondylolysis, neurological disorders, or leg length discrepancies were excluded from this study.

Prior to testing, all subjects were asked to read and sign a consent form approved by the University of Pittsburgh Biomedical Institutional Review Board. Athletic status was determined for matching the control group, and both groups completed the McGill Pain Questionnaire (short form) to assess low back pain (24). The dependent variables assessed in this study were erector spinae performance, abdominal performance, low back flexibility, lateral flexibility of the torso, and hip flexibility. To familiarize the subject, each test position was practiced three times. A single test trial was then recorded. All tests were conducted by the same examiner, prior to the team's practice or individual's rehabilitation.

Testing Procedure

Erector spinae performance was assessed with the subjects lying prone on a table, hands crossed behind their heads. The axilla was used as a reference for the axis of a goniometer. The adjustable arm was aligned with the lateral side of the body and chin while the stationary arm was parallel to the table. Subjects were asked to extend their spines to the designated angle of 30° and hold this position for as long as possible. Moving above or below the designated angle marked the end of the test. Strength was measured as a function of time in seconds that the subjects could hold the designated angle (4, 14, 16) (Figure 1).

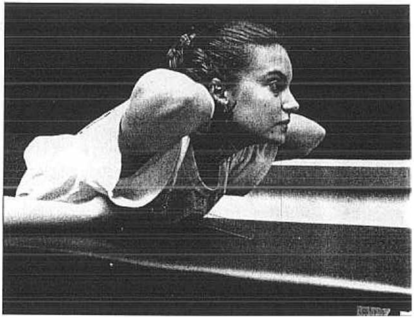

Figure 1 — Erector spinae endurance was assessed at 30° of back hyperextensi Subjects were prone, using the axilla as the reference point. This was a static t beginning the moment the athlete reached the designated angle until she could maintain the hyperextended position of 30°.

Abdominal muscle performance was assessed with the straight leg lower test (20, 33). Subjects were positioned supine on a plinth with their hips in 90 flexion and knees fully extended. The investigator's hand was placed between table and the subject's low back, at the L4–L5 interspace. Subjects were then structed to lower their legs to the table in time with a metronome, keeping th low backs pressed into the investigator's hand. The rate of leg lowering was proximately 9°/s such that it took 10 s to complete the test. To assist the exami in monitoring this task, lines were placed on the wall behind the subjects' le each corresponding to 10° increments of hip flexion. Abdominal performance recorded as the angle of the subjects' legs when their low backs began to raise the investigator's hand (14, 15). A lower angle corresponded with increased p formance (Figure 2).

Low-back flexibility was tested with the subjects sitting cross-legged on floor, hands placed behind their heads, and backs parallel to a plumb line. Th position stabilized the pelvis and thoracic spine. The stationary arm of a gonio eter was placed on the plumb line while the L4–L5 interspace was used as a ref ence for the movable arm. Forward flexion was measured from the plumb line the furthest degree of lumbar flexion while the subject maintained the specifi body position (Figure 3).

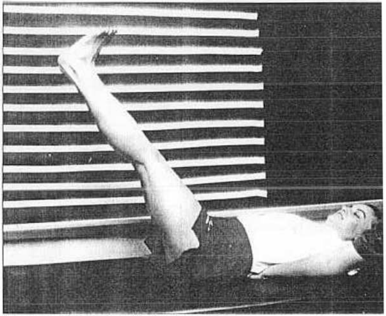

Figure 2 — Abdominal performance was assessed using the straight leg lowering test. Subjects were instructed to lower their legs in time with a metronome to each increment marking on the wall.

Lateral flexibility of the torso was assessed with subjects standing, th hands against the lateral aspect of their thighs. After the positions of both mid fingers on the thighs were marked, the subjects moved into lateral flexion on both sides, and the lowest position of the middle finger on each side was marked again. The distance between the two corresponding marks represented lateral flexibility (19, 22) (Figure 4).

Bilateral hip flexibility was assessed with the subjects both prone and supine on a table, to measure hip extension and flexion, respectively (26). The greater trochanter acted as a reference for the fulcrum of a goniometer. The stationary arm of the goniometer was parallel with the lateral midline of the pelvis, while the adjustable arm was aligned with the femur. Subjects were asked to actively flex and extend their hips while maintaining full knee extension. Measurements were recorded at the furthest points of hip flexion and extension (31, 35) (Figures 5 and 6).

Results

Dependent t tests were used to determine mean differences between the experimental and control groups. Significant differences were noted in abdominal performance ($t = 3.50$, $p < .01$) and erector spinae performance ($t = 2.69$, $p < .03$). Flexibility measurements also demonstrated significant differences between subjects with CLBP and the control group for left hip extension ($t = 4.01$, $p < .005$) and right lateral flexion of the torso ($t = 2.54$, $p < .04$) (Table 1). Results of the

Figure 3 — Low-back flexibility was assessed while the subject performed forward flexion in the seated position. The furthest degree of lumbar flexion was measured with a goniometer.

McGill Pain Questionnaire confirmed that the CLBP group experienced mild to moderate pain (mean = 1.57 ± 0.36) while the control group reported no discomfort.

Discussion

The purpose of this study was to determine any significant differences between female varsity athletes with CLBP and those with healthy low backs. Several studies have revealed a relationship between spinal and/or pelvic mobility and CLBP (1, 3, 8, 25, 30). The dependent variables assessed in this study and others are believed to play a role in pelvic stabilization and therefore are believed to be related to CLBP (8, 32, 35). Previous studies have claimed that controlled exercises involving these particular muscles groups can alleviate the symptoms of CLBP (11, 15, 20). Although most of the research focused on alleviation of back pain, none of the studies noted deficiencies in the muscle groups of interest prior to initiation of the studies. Other researchers have introduced possible rationales for implementing specific stretching and strengthening exercises but neglected to incorporate clinical evidence to support their beliefs (10 ,11, 15, 20, 22).

Figure 4 — Lateral flexibility of the torso was assessed bilaterally using the position of the third phalanx on the lateral aspect of the thigh. Measurements were taken between the initial standing position and the furthest position of lateral flexion.

The back extensor muscles, predominantly the erector spinae group, provide posterior stability for the vertebral column. Previous studies have found that erector spinae endurance and strength play a significant role in CLBP (2, 9, 11, 27, 31, 32). Calliet (3) found that subjects with a high degree of erector spinae endurance experienced back pain less often than those with poor muscular endurance. The erector spinae works against the forces of gravity to maintain erect posture and to control forward flexion. Weakness in the erector spinae muscles can lead to vertebral malalignment, ultimately resulting in abnormal loading on the spine. There is some debate whether this weakness can be attributed to muscle fatigue or to absolute force production (i.e., strength). Qualitative studies of back muscle fatigue have been conducted with CLBP patients and comparison groups of normal sub-

Table 1 Results of Strength/Endurance and Flexibility Assessments for the Experimental Group Versus the Control Group

	Experimental		Control			
	M	*SD*	*M*	*SD*		*p*
Abdominal performance	5.37	2.91	9.19	5.07	3.50	.010*
Erector spinae performance	44.72	31.75	123.27	80.89	2.69	.031*
Lumbar flexion	48.94	18.89	55.31	18.21	2.54	.442
Left lateral flexion	8.06	2.92	17.22	7.60	1.23	.259
Right lateral flexion	6.90	1.46	11.38	5.52	2.54	.039*
Left hip extension	15.94	5.82	23.75	4.60	4.01	.005*
Right hip extension	18.00	6.57	23.22	3.53	2.03	.082
Left hip flexion	107.25	24.61	110.50	15.98	0.54	.605
Right hip flexion	113.38	21.15	110.81	15.89	−0.56	.594

*Significant difference ($p < .05$)

jects. De Vries (6) found that subjects who exhibited CLBP displayed an increase in electromyographic activity, but this is not synonymous with force production. Roy et al. (30) concluded that spectral EMG shifts indicated specific fatigue patterns in low-back pain subjects and could be used to evaluate muscle function. Altered EMG activity may indicate inefficient firing patterns and accelerated fatigue of the extensor muscles in subjects with CLBP. Low-back endurance in our study was determined by monitoring the time to exhaustion during sustained isometric back extension (13, 22, 30, 35). Our results were consistent with previous observations in that subjects with CLBP had less endurance capacity than control subjects (3, 9, 11, 27, 30–32). Plowman (27) attributed this to a greater proportion of Type II (fast twitch) muscle fibers, whereas the demands for postural control are better managed by Type I (slow twitch) fibers. This scenario would result in the accumulation of metabolites in the fatiguing muscles (30). In fact, Delitto and Rose (5) theorized that high precontraction metabolite levels from persistent muscle spasm and prolonged muscle tension are associated with excessive back fatigue.

As with the erector spinae group, the role of abdominal strength and/or endurance in patients with CLBP has been subject to debate. Sward et al. (32) stated that the abdominal cavity encloses the contents of the thorax, forming an "air bag." Contraction of the abdominal musculature increases intra-abdominal pressure within this air bag, theoretically decreasing compressive forces on the spine (9, 12, 15, 16, 31). Activity in the oblique and transverse abdominal musculature also contributes to tensing of the thoracolumbar fascia (12). Gracovetsky and Farfan (12) stated that the primary purpose of increased abdominal pressure is to maximize the mechanism between the abdominals and thoracolumbar fascia by maintaining the proper geometry of the spine. The rectus abdominus not only increases intra-abdominal pressure but helps maintain the pelvis in a neutral position by counteracting pull from the extensor muscles. Unfortunately, this neutral position is largely dependent on the degree of lordosis for each individual (3, 16, 30). Strength and/or endurance deficits of the rectus abdominus muscle allow for an exaggerated anterior tilt of the pelvis, altering the distribution of compressive forces on the lumbar spine (15, 24, 30). Walker et al., however, found little correlation between abdominal strength and pelvic tilt (35).

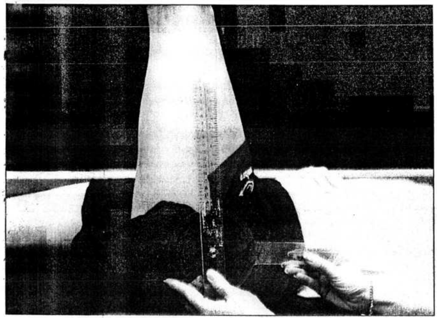

Figure 5 — Hip flexibility was assessed using traditional goniometric methods. Subjects were instructed to raise the testing leg while keeping the opposite leg flat on the table. The greater trochanter was the reference point.

Our research is consistent with the work of Smidt et al., who discovered strength deficits in subjects with low-back pain; curiously, these subjects were able to work for longer periods within the constraints of their endurance test (31). These subjects produced less force during the endurance test, which may have prolonged the time to reach the 25% decrement level used to terminate the test. This is a perfect example of the disparity in testing strength versus endurance, which is why we prefer the term "abdominal performance" when referring to the results obtained from the leg-lowering test (18). EMG studies show that activity in the abdominal and hip extensor musculature occurs simultaneously. Weakness in one or both of these areas disrupts pelvic stabilization, supporting its involvement in CLBP subjects.

Lack of lumbar flexion in the low back has also been associated with CLBP (9). Inflexibility will decrease the lordotic curve that naturally exists in the lumbar spine, causing an exaggerated posterior tilt of the pelvis. This diminishes the shock-absorbing capacity of the lumber segments and increases tension on the surrounding ligaments and musculature. The ballistic movements performed during athletics greatly magnify these forces when the lordotic curve is reduced and posterior pelvic tilt is increased. Previous studies have suggested that measurements of lumbar flexion may provide the most specific and objective data for basis of impairment (9, 30, 34). Despite common clinical beliefs, this study found that lumbar flexion

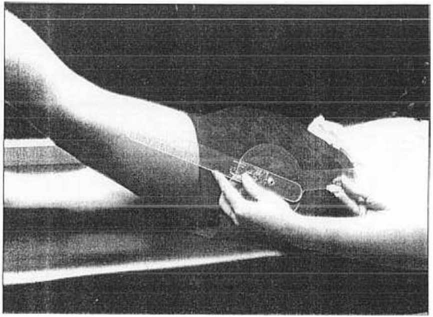

Figure 6 — Hip extension was assessed bilaterally in the prone position. Subjects were instructed to raise the involved leg while keeping the involved anterior superior iliac spine pressed to the table.

was not significantly different in subjects with CLBP. Waddell (33) found similar results in patients with CLBP. The inclusion of athletes was cited by Waddell et al. (34) as a confounding variable in lumbar flexion assessment. However, our subject population consisted exclusively of athletes and confirmed the results that lumbar flexion is not impaired with CLBP. The absence of flexion deficits implies that no true anatomical or structural impairment exists in the CLBP subjects (34).

Recent studies have also shown a relationship between lateral flexibility of the torso and CLBP. The oblique musculature, which helps control lateral flexion, is believed to be a key trunk stabilizer (10, 12, 14, 16, 22, 31). The primary role of the oblique complex is to reinforce the erector spinae fascia by pulling it laterally (12). This widened, reinforced fascia is a more efficient support and decreases strain on the lumbar vertebra. A unilateral reduction in oblique flexibility could result in asymmetrical forces on the lumbar fascia and pelvic girdle. Mellin (22) found that lateral flexibility of the torso has a direct correlation to CLBP. Likewise, our results showed that subjects with CLBP had significant deficits in right lateral flexibility. These findings do not account for the effects of anterior or superior tilting of the pelvis; nor do they exclude rotational stability of the vertebral segments as a cause for bilateral asymmetry. These differences may also be explained by hand dominance or side-dominated sports. To what degree the oblique muscles support the lumbar spine and CLBP has yet to be determined.

Inflexibility in hip flexion and extension has been identified as a factor altering pelvic stabilization (1, 22). For example, tight hip extensors will "flatten out" the lordotic curve in the lumbar spine or increase posterior pelvic tilt. This mechanism diminishes the shock-absorbing capacity of the normal vertebral alignment while increasing compressive forces on the lumber spine. For the purpose of this study we did not specifically test the hamstring muscle group, although clinically significant deficits have been demonstrated in these muscles when CLBP is present (11, 20, 23, 27). The test used to assess the degree of hip flexion in our study did not reveal significant differences; however, a more sensitive test to isolate hamstring flexibility may have been appropriate. The opposing hip flexors may be responsible for an exaggerated anterior pelvic tilt, if inflexibility exists. Anterior rotation of the pelvis could limit pelvic mobility, resulting in an excessive strain to the lumbar spine. In this study we found a decrease in left hip extension, suggesting tight hip flexors, which has the potential to limit pelvic mobility. Restrictions in hip extension could decrease the lumbar lordotic curve, making the spine less resilient to axial loading (11). In addition, Gracovetsky and Farfan(12) stated that the psoas muscles are essential for controlling lordosis and spinal torque during flexion and extension.

The findings in this study suggest that CLBP can result from three types of deficits attributed to muscle tissue: strength, flexibility, and endurance. Careful investigation of the pathomechanics may reveal that a combination of these deficits are specific to the individual or demands of the sport. Fortunately, the plasticity of muscle tissue permits acute and chronic adaptations with proper rehabilitation. Information gained from the physical examination and special tests will assist in the design of a rehabilitation program best suited for the athlete. The special tests in this study focused on identifying common mechanisms for pelvic instability, which is associated with CLBP. If pelvic instability is suspected, reeducating and/or reconditioning the involved muscles may decrease the likelihood of recurring low-back pain.

Conclusion

The tests utilized in this study are easy, cost-effective measurement tools that clinicians can use to screen athletes for strength, endurance, and flexibility deficits and to document the efficacy of rehabilitation protocols. In this study, strength and flexibility deficits were identified in those subjects with CLBP. Significant deficits were noted in erector spinae performance and abdominal musculature performance. It has been suggested that these muscle groups produce force couples that stabilize the pelvis. These results validate the necessity to focus on pelvic stabilization through strength and flexibility when treating CLBP patients. The goal of rehabilitation should be to increase pelvic stability by using specific strength and flexibility exercises. Competitive athletes should be screened for strength and flexibility deficiencies to reduce their risk of CLBP. Such screening will not only allow us to assess the efficacy of rehabilitation protocols but will also provide us with a better understanding of this idiopathic injury.

References

Biering-Sorensen, F. Physical measurements as risk indicators for low-back trouble over a 1 year period. *Spine* 14:123-125, 1989.

2. Bogduk, N., J. Macintosh, and T. Pearcy. A universal model of the lumbar back muscles in the upright position. *Spine* 17:897-913, 1992.
3. Calliet, R. *Low Back Pain Syndrome* (4th ed.). Philadelphia: Davis, 1988, pp. 13-18.
4. Cassisi, J.E., M.E. Robinson, P. O'Connor, and M. MacMillan. Trunk strength and lumbar paraspinal muscle activity during isometric exercise in chronic low back pain patients and controls. *Spine* 18:245-448, 1993.
5. Delitto, R.S., and S.J. Rose. An electromyographic analysis of two techniques for squat lifting and lowering. *Phys. Ther.* 72:438-448, 1992.
6. De Vries, H.A. EMG fatigue curves in postural muscles. A possible etiology for idiopathic low back pain. *Am. J. Phys. Med.* 47:175-181, 1968.
7. Dillard, D. Motion of the lumbar spine: Reliability of two measurement techniques. *Spine* 6:321-323, 1991.
8. DonTigny, R.L. Anterior dysfunction of the sacroiliac joint as a major factor in the etiology of idiopathic low back pain syndrome. *Phys. Ther.* 70:250-262, 1990.
9. Einagger, I.M., M. Nordin, and A. Sheikh. Effects of spinal flexion and extension exercises in low back pain and spinal mobility in chronic mechanical low back pain patients. *Spine* 16:967-971, 1991.
10. Ellison, J.B., S.J. Rose, and S.A. Sahrmann. Patterns of hip rotation range of motion: A comparison between healthy subjects and patients with low back pain. *Phys. Ther.* 70:537-541, 1992.
11. Foster, D., and M.N. Fulton. Back pain and the exercise prescription. *Clin. Sports Med.* 10:197-209, 1991.
12. Gracovetsky, S., and H. Farfan. The optimum spine. *Spine* 11:543-572, 1986.
13. Gracovetsky, S., M. Kary, and S. Levy. The importance of pelvic tilt in reducing compressive stress in the spine during flexion and extension exercises. *Spine* 14:412-416, 1989.
14. Haas, M., J. Nyiendo, C. Peterson, H. Thiel, T. Sellers, E. Dal Mas, C. Kirton, and D. Cassidy. Lumbar motion trends and correlation with low back pain: Part I. Roentgenological evaluation of coupled lumbar motion in lateral side bending. *J. Manipulative Physiol. Ther.* 15:145-158, 1992.
15. Hanson, F. Intensive, dynamic back muscle, conventional physiotherapy, or placebo control: Treatment of low back pain. *Spine* 18:98-113, 1993.
16. Hanson, T., S. Bigus, P. Beechee, and M. Wortley. The lumbar lordosis in acute and chronic low back pain patients. *Spine* 10:154-155, 1994.
17. Kellgreen, J.H. The anatomical source of low back pain. *Rheumatol. Rehabil.* 16:3-12, 1977.
18. Kendall, F.P., and E.K. McCreary. *Muscles: Testing and Function* (3rd ed.). Baltimore: Williams & Wilkins, 1983, pp. 100-107.
19. Khalil, T.M., S.S. Asfour, L.M. Martinez, S.M. Wally, R.S. Rosomoff, and H.L. Rosomoff. Stretching in the rehabilitation of low back pain patients. *Spine* 17:311-317, 1992.
20. Lidstrom, I., C. Ohlund, C. Eek, L. Wallin, L. Peterson, and A. Nachemson. Mobility, strength, and fitness after a graded activity program for patients with subacute low back pain. *Spine* 17:641-652, 1992.
21. Mayhew, T., B. Norton, and S.A. Sahrmann. Electromyographic study of the relationship between hamstring and abdominal muscles during unilateral straight leg raise. *Phys. Ther.* 63:1769-1774, 1983.
22. Mellin, G. Correlations of spinal mobility with a degree of low back pain after correction for age and anthropomorphic factors. *Spine* 12:464-468, 1987.
23. Mellin, G. Chronic low back pain in men 54-63 years of age. *Spine* 11:421-426, 1986.
24. Melzack, R. The McGill Pain Questionnaire: Major properties and scoring methods. *Pain* 1:277-299, 1975.
25. Nachemson, A.L. Advances in low back pain. *Clin. Orthop.* 11:266-273, 1985.
26. Norkin, C., and J. White. *Measurement of Joint Motion: A Guide to Goniometry*. Philadelphia: Davis, 1985, pp. 76-78.
27. Plowman, S.A. Physical activity, physical fitness, and low back pain. *Exerc. Sports Sci. Rev.* 20:221-242, 1992.
28. Risch, S.V., N.K. Norvell, M.L. Pollock, E.D. Risch, H. Langer, M. Fulton, J.E. Graves, and S.H. Leggett. Lumbar strengthening in chronic low back pain patients. *Spine* 18:232-238, 1993.
29. Roland, M., and R. Morris. A study of the natural history of back pain. *Spine* 8:141-145, 1993.
30. Roy, S.H., C. De Luca, and D. Casavant. Lumbar muscle fatigue and chronic low back pain. *Spine* 14:992-1001, 1989.
31. Smidt, G., T. Herring, L. Amundsen, M. Rogers, A. Russell, and T. Lehmann. Assessment of abdominal and extensor function. *Spine* 8:211-219, 1983.
32. Sward, L., B. Eriksson, and L. Peterson. Anthropometric characteristics, passive hip flexion and spinal mobility in relation to back pain in athletes. *Spine* 15:376-382, 1990.
33. Waddell, G. A new clinical model for the treatment of low back pain. *Spine* 12:632-641, 1987.
34. Waddell, G., D. Somerville, I. Henderson, and M. Newton. Objective clinical evaluation of physical impairment in chronic low back pain. *Spine* 17:617-627, 1992.
35. Walker, M.L., J.M. Rothstein, S.D. Finucane, and R.L. Lamb. Relationship between lumbar lordosis, pelvic tilt, and abdominal muscle performance. *Phys. Ther.* 67:512-515, 1987.