SPINE Volume 26, Number 23, pp 2566–2574


# Spine Loading Characteristics of Patients With Low Back Pain Compared With Asymptomatic Individuals

William S. Marras, PhD,* Kermit G. Davis, PhD,* Sue A. Ferguson, PhD,*
Benjamin R. Lucas,* and Purnendu Gupta, MD†

**Study Design.** Patients with low back pain and asymptomatic individuals were evaluated while performing controlled and free-dynamic lifting tasks in a laboratory setting.

**Objective.** To evaluate how low back pain influences spine loading during lifting tasks.

**Summary of Background Data.** An important, yet unresolved, issue associated with low back pain is whether patients with low back pain experience spine loading that differs from that of individuals who are asymptomatic for low back pain. This is important to understand because excessive spine loading is suspected of accelerating disc degeneration in those whose spines are damaged already.

**Methods.** In this study, 22 patients with low back pain and 22 asymptomatic individuals performed controlled and free-dynamic exertions. Trunk muscle activity, trunk kinematics, and trunk kinetics were used to evaluate three-dimensional spine loading using an electromyography-assisted model in conjunction with a new electromyographic calibration procedure.

**Results.** Patients with low back pain experienced 26% greater spine compression and 75% greater lateral shear (normalized to moment) than the asymptomatic group during the controlled exertions. The increased spine loading resulted from muscle coactivation. When permitted to move freely, the patients with low back pain compensated kinematically in an attempt to minimize external moment exposure. Increased muscle coactivation and greater body mass resulted in significantly increased absolute spine loading for the patients with low back pain, especially when lifting from low vertical heights.

**Conclusions.** The findings suggest a significant mechanical spine loading cost is associated with low back pain resulting from trunk muscle coactivation. This loading is further exacerbated by the increases in body weight that often accompany low back pain. Patient weight control and proper workplace design can minimize the additional spine loading associated with low back pain. [Key words: electromyography, lifting, low back pain, musculoskeletal, spinal loads] **Spine 2001;26:2566–2574**

An important, yet unresolved, issue associated with low back pain (LBP) is whether the spine loadings of patients with LBP differ from those of individuals who are asymptomatic for LBP. Recent studies[1,3] have suggested that excessive mechanical loading on spinal structures that already are compromised can progressively affect disc degeneration, possibly resulting in chronic LBP. Therefore, it is important to understand the mechanisms by which spine loading occurs in patients with LBP to allow identification of situations that might lead to further spine damage.

There have been several attempts toward a better understanding of the biomechanical differences between patients with LBP and asymptomatic individuals. Studies have explored the strength differences between patients with LBP and asymptomatic individuals[22,24,30,48,49,51,58]. Although most of these studies have identified differences in strength production capacity, they do not offer much insight into the nature of the loading imposed on the spine. Other studies[6,9,25,26,54,55,57,58] have attempted to document the back muscle electromyographic (EMG) activities. Although increased coactivity (guarding) in patients with LBP have been documented by several researchers,[6,9,26–28] none have determined whether the increased coactivity translated into higher spine loading.

Deterministic biomechanical models have been developed that estimate spine loads in uninjured individuals by assuming that specific internal structures must support an external load moment and contribute to internal spine loading.[7,8,14,53,59] However, it is well known that patients with LBP have greater levels of guarding, recruit their muscles in a significantly different manner, move slower, and have altered flexion–relaxation responses, as compared with asymptomatic patients[4–6,28,34,42,46,60]. These facts suggest that patients with LBP may use very different muscle activation patterns to generate internal load support, making deterministic models inappropriate for patients with LBP.

Clearly, to predict spine loads in patients with LBP, the clinician must be able account for atypical recruitment patterns of the trunk muscles. Although EMG-assisted biomechanical models could be well suited to such a task, these models need to assess the relative contribution of multiple muscles accurately, which usually requires an EMG calibration (*e.g.*, typically normalized to maximum exertion). Unfortunately, patients with LBP are unable or unwilling to produce such exertions.[21,39,47] Recently, studies in the authors' laboratory have developed an EMG calibration procedure based on submaximal exertions that nevertheless can estimate maximal EMG.[31,32]

Given this development in EMG calibration, a study intended to compare spine loads of patients with LBP with those of asymptomatic individuals was designed.The study consisted of two phases: one interpreting spine loads in both groups of subjects exposed to external loads of the same magnitude while positioned in

From the *Biodynamics Laboratory, Ohio State University, Columbus, Ohio, and the †Section of Orthopaedic Surgery and Rehabilitation Medicine, University of Chicago, Chicago, Illinois.
Partial funding for this study was supplied by a grant from the Ohio Bureau of Workers' Compensation.
Device status category: 1.
Conflict of interest category 12.

**Table 1. Anthropometry for Individuals With Low Back Pain (LBP) and Asymptomatic Individuals**

| | Asymptomatic | | | Low Back Pain | | |
|---|---|---|---|---|---|---|
| | Males | Females | Overall | Males | Females | Overall |
| Age (years) | 34.3 ± 10.4 | 38.8 ± 12.0 | 36.4 ± 11.1 | 41.6 ± 9.5 | 35.9 ± 10.4 | 39.0 ± 10.1 |
| Weight (kg)* | 87.5 ± 14.8 | 62.1 ± 7.0 | 76.0 ± 17.1 | 101.6 ± 14.1 | 84.6 ± 12.7 | 93.8 ± 15.7 |
| Stature (cm) | 180.4 ± 6.7 | 165.7 ± 4.6 | 173.7 ± 9.4 | 180.0 ± 4.9 | 166.0 ± 7.1 | 173.6 ± 9.2 |
| Trunk depth (cm)* | 23.5 ± 3.3 | 20.0 ± 2.4 | 21.9 ± 3.4 | 27.0 ± 2.0 | 25.3 ± 4.1 | 26.2 ± 3.1 |
| Trunk breadth (cm)† | 32.8 ± 2.8 | 27.3 ± 1.8 | 30.3 ± 3.7 | 34.6 ± 3.2 | 30.7 ± 2.7 | 32.8 ± 3.5 |
| Trunk Circumference (cm)* | 92.8 ± 15.0 | 75.8 ± 7.5 | 85.1 ± 14.7 | 108.9 ± 11.8 | 100.5 ± 10.7 | 105.1 ± 11.8 |
| Percentage of normal* | 79.6 ± 25.2 | 66.7 ± 32.7 | 73.8 ± 28.9 | 5.9 ± 6.3 | 17.5 ± 24.4 | 11.2 ± 17.6 |

* Significant differences between individuals with LBP and asymptomatic individuals for females, males, and overall.
† Significant differences between individuals with LBP and asymptomatic individuals for females and overall.

identical postures, and another permitting subjects to adjust their postures so the researchers could gain insight into how patients with LBP compensate for their pain limitations under realistic conditions.

## ■ Methods

**Participants.** Of the 44 participants in this study, 22 (12 males and 10 females) had LBP at the time of the testing and were recruited from the orthopedic practice of one of the authors (P.G.). In addition, 22 age- and gender-matched individuals who had been asymptomatic during the previous year were recruited. Gross anthropometric characteristics are reported in Table 1. Table 2 summarizes the LBP characteristics of these individuals with LBP.

**Study Design.** All the participants were involved in both phases of the testing. Phase 1 testing was intended to control torso posture precisely at 20° of torso flexion and force exertion level. During this exertion, the participant's pelvis was fixed in a structure that measured external trunk moment *via* a force plate (Figure 1). While in this posture, the participants were asked to control sagittal extension exertion by ramping up to a predetermined force exertion level for 2 seconds. Three exertion targets (40, 60, and 80 Nm) and injury group membership (LBP *vs* asymptomatic) served as the independent variables for this phase of the study. The dependent variables for this phase consisted of trunk muscle EMG activity, trunk kinetic information, and the resulting spine loads.

Phase 2 testing was intended to evaluate whether kinematic compensation occurred in patients with LBP, as compared with asymptomatic participants. This phase used a repeated-measures within-subject design. The independent variables in this study were group membership (LBP *vs* asymptomatic), weight lifted, and lift origin. Four weights weighing 4.5, 6.8, 9.1, and 11.4 kg, respectively, were lifted under free-dynamic sagittally symmetric conditions, each starting from six lift ori-

**Table 2. Description of the Group of Patients With Low Back Pain (LBP)**

| | Pain Distribution | | | | | | |
|---|---|---|---|---|---|---|---|
| LBP Participant | 100% Back | 75% Back 25% Leg | 50% Back 50% Leg | Muscular (M) *vs* Structural (S) | Pain Level at Time of Testing* | History of Back Pain | Duration of Current Episode (weeks) |
| 1 | | X | | M | 7 | Yes | 6 |
| 2 | X | | | M | 4 | No | 6 |
| 3 | X | | | M | 6 | No | 8 |
| 4 | X | | | M | 6 | No | 24 |
| 5 | | X | | M | 3 | Yes | 5 |
| 6 | X | | | M | 6 | No | 6 |
| 7 | | X | | M | 3 | Yes | 4 |
| 8 | X | | | M | 7 | Yes | 4 |
| 9 | | X | | M | 5 | No | 11 |
| 10 | | | X | S | 5 | Yes | 3 |
| 11 | | X | | M | 6 | Yes | 3 |
| 12 | X | | | M | 3 | No | 24 |
| 13 | X | | | M | 2 | No | 8 |
| 14 | | X | | S | 5 | No | 24 |
| 15 | | X | | M | 5 | No | 240 |
| 16 | | | X | M | 6 | Yes | 7 |
| 17 | | X | | S | 5 | Yes | 144 |
| 18 | | X | | M | 5 | Yes | 5 |
| 19 | | X | | M | 5 | No | 72 |
| 20 | X | | | M | 0 | Yes | 4 |
| 21 | | X | | M | 5 | Yes | 168 |
| 22 | | X | | S | 7 | No | 6 |
| Summary | 36% | 54% | 10% | | Average 4.8 | 50% Yes | Mean = 35.5<br>Median = 6.5 |

* Scale is from 0 (no pain) to 10 (worse pain).

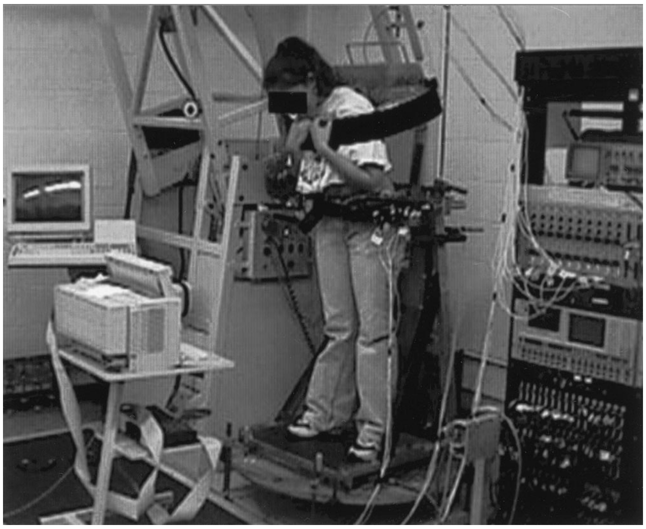


Figure 1. Subject performing static exertions while positioned in the pelvic support structure and the asymmetric reference frame during the electromyographic calibration procedure and Phase 1 controlled exertions.

gins varying in vertical height and horizontal distance from the spine: shoulder height at a moment arm of 30.5 cm, waist height at 30.5 cm, knee height at 30.5 cm, mid-shin height at 30.5 cm, waist height at 61 cm, and knee height at 61 cm (Figure 2). The lifts ended with the body in an upright position and the weight located at elbow height. The dependent variables for this phase comprised the EMG activity of 10 trunk muscles, trunk and hip kinetic and kinematic information, and the resulting spine loads.

**Apparatus.** Electromyographic activity was collected with the use of bipolar silver–silver chloride electrodes. The electrodes recorded activity at the 10 major trunk muscle sites consisting of right and left muscle pairs of erector spinae, latissimus dorsi, rectus abdominis, external oblique muscles, and internal oblique muscles. Electromyographic preparations and electrode placements have been described previously.[41] The raw EMG signals were preamplified, high-pass filtered at 30 Hz, low-pass filtered at 1000 Hz, rectified, and smoothed with a 20-ms sliding window filter. Skin impedances were maintained below 100 KΩ.

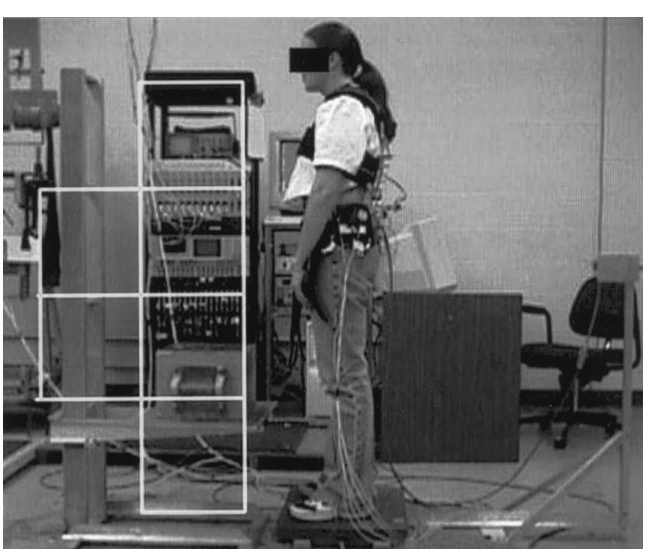


Figure 2. Subject performing the free-dynamic lifts during Phase 2. The overlay (boxes) indicates the lift origins.

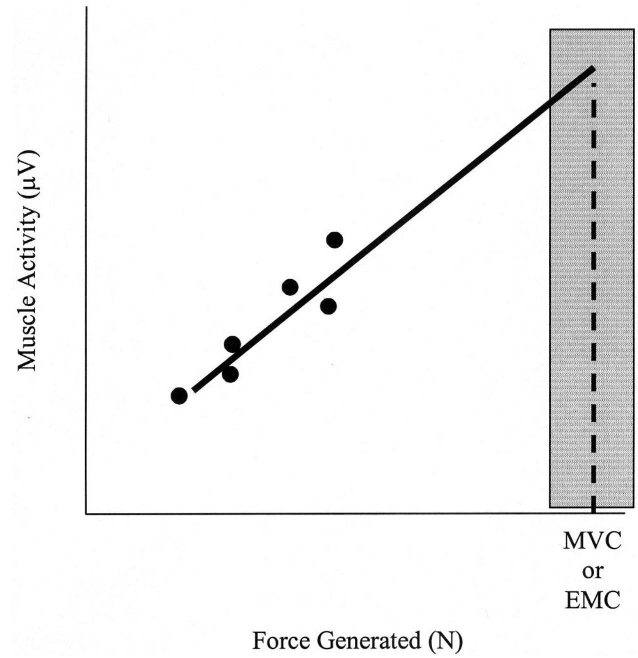



Figure 3. Graph showing the linear slope relation between forces generated and muscle electromyographic activity with a reference point, either maximum voluntary contraction (MVC) or expected maximum contraction (EMC). The dots represent muscle activity at a given exerted moment, and the shaded area shows typical variability in the MVC because of inaccuracies. EMC defines the midpoint of this region. Adapted from Marras and Davis.[31,32]

Electromyographic calibration normalization testing and Phase 1 testing were performed using an asymmetric reference frame.[40] Pelvic and leg positions also were controlled using a pelvic support structure[19] (Figure 1). The asymmetric reference frame provided static resistance against the upper body and monitored torque production about L5–S1,[19] which was displayed on a computer so participants could control the magnitude.

During Phase 2 testing, trunk kinematics were documented with a lumbar motion monitor. The device design, accuracy, and application have been reported previously.[33] Ground reaction forces were monitored *via* a force plate in conjunction with a set of electrogoniometers[13] (Figure 2). All signals were collected simultaneously at 100 Hz using customized Windows-based software developed in the Biodynamics Laboratory (Columbus, OH).

**EMG Calibration.** A new EMG calibration procedure reported recently does not require maximal exertion for calibration of the EMG signal.[31,32] This new technique estimates the slope of the EMG–force relation and predicts an expected maximum contraction for "anchoring" the maximum value (Figure 3). The EMG–force relation (slope) is established by a series of low-level exertions performed in flexion, extension, and axial twisting. Subjects are asked to produce three subjectively determined exertion levels (*e.g.*, ⅓ or ½ of their available strength) and three set exertion levels (*e.g.*, 10, 20, and 30 Nm).

The expected maximum contraction, as a substitute for maximum voluntary contraction, was derived from regression equations describing the relations between maximum exerted trunk moments and anthropometric measurements for 120 subjects.

**Functional Assessment.** To quantify the extent of low back impairment associated with the experimental subjects, a dynamic functional assessment was performed using the lumbar motion monitor. This method has been thoroughly reported and validated.[34,39,42,46] The motion characteristics of the participants with LBP were significantly lower than those of asymptomatic individuals ($P < 0.01$), with the asymptomatic group within normal range.

**Spine Load Assessment.** Over the past 18 years, the authors' laboratory has developed a three-dimensional dynamic biomechanical model that can determine how the vertebral joint at L5–S1 is loaded during a dynamic motion[10,15–18,35–37,43–45,50,56]. The model has been validated for forward bending,[16,45] lateral bending,[36] and twisting[35] exertions and adjusted for anatomic gender differences.[20,38] For this study, adjustments to muscle location and size relative to each subject's body mass index also were made[61] because the LBP group was considerably heavier than the asymptomatic group, yet similar in stature.

Two additional biomechanical measures were defined in this study. The *coactivity index* was defined as the activity of the antagonist muscles relative to agonist muscle activities. Next, *cumulative compression* was defined as the spine load summed continuously over the entire lift period (*i.e.*, integration of instantaneous loads).

**Procedure.** On arriving at the Biodynamics Laboratory, the subjects first were informed about the study procedures, their prerogative to refuse performance of a particular lift, and their need to inform experimenters about any further discomfort. Consent to participate was acquired *via* a document approved by the University Institutional Review Board. Second, anthropometric measurements were collected, and surface electrodes then were applied using standard placement procedures.[41]

Next, the participant was positioned in the pelvic support structure in the asymmetric reference frame, and the EMG calibration procedure was performed. This procedure was followed by the Phase 1 experiment, in which the subjects completed the three targeted extension exertions. After a rest period, Phase 2 test conditions were completed twice for each load weight condition. All six lift region conditions (Figure 2) were completed for each weight before the increase to the next weight. Hence, lifts were performed in the least taxing positions (*e.g.*, lowest expected lift moment) first before the participant progressed to more demanding lifts at each weight level. In this test, the participants were required to keep their feet stationary on the force plate, but were free to move the rest of the body as they wished.

**Statistical Analyses.** Repeated measures analyses of variance (ANOVA) were performed for the spine load variables to explore whether differences exist between individuals with LBP and asymptomatic individuals. When statistically significant independent variables were identified, *post hoc* analyses (Tukey multiple pairwise comparisons) were performed to determine the source of the significant effects. Significant effects identified were considered statistically significant at a $P$ value less than 0.05.

**Table 3. Descriptive Statistics for the Low Back Pain (LBP) and Asymptomatic Injury Groups for the Dependent Variables in the Two Phases of the Study***

| | LBP | Asymptomatic |
|---|---|---|
| Static controlled (Phase 1) | | |
| Trunk kinetics | | |
| Sagittal trunk moment (Nm) | 89.4 ± 15.6 | 77.5 ± 15.5 |
| Absolute spinal loads† | | |
| Lateral shear (n) | 32.4 ± 35.3 | 15.9 ± 17.2 |
| Anteroposterior shear | 327.3 ± 99.3 | 293.2 ± 65.2 |
| Compression | 1395.8 ± 500.0 | 948.2 ± 331.5 |
| Normalized spinal loads‡ | | |
| Lateral shear (n/Nm) | 0.36 ± 0.34 | 0.20 ± 0.17 |
| Anteroposterior shear (n/Nm) | 3.71 ± 1.10 | 3.81 ± 0.50 |
| Compression (n/Nm) | 15.69 ± 5.82 | 12.42 ± 3.92 |
| Muscle coactivity§ | | |
| Coactivity index | 0.46 ± 0.26 | 0.31 ± 0.19 |
| Dynamic lifting (Phase 2)‖ | | |
| Trunk kinematics | | |
| Sagittal trunk position (°) | 22.56 ± 17.87 | 27.31 ± 20.84 |
| Sagittal trunk velocity (°) | 21.30 ± 18.57 | 36.54 ± 28.03 |
| Trunk kinetics | | |
| Sagittal trunk moment (Nm) | 108.5 ± 71.5 | 90.5 ± 61.0 |
| Absolute spinal loads† | | |
| Lateral shear (n) | 110.7 ± 142.9 | 81.4 ± 100.0 |
| Anteroposterior shear (n) | 609.6 ± 367.3 | 585.3 ± 555.6 |
| Compression (n) | 2825.5 ± 2021.4 | 2340.5 ± 1633.1 |
| Normalized spinal loads‡ | | |
| Lateral shear (n/Nm) | 1.00 ± 1.34 | 0.86 ± 0.83 |
| Anteroposterior shear (n/Nm) | 7.72 ± 8.61 | 8.20 ± 6.39 |
| Compression (n/Nm) | 26.95 ± 20.24 | 26.90 ± 10.36 |
| Cumulative spinal loads¶ | | |
| Lateral shear ($\times 10^4$ n) | 0.94 ± 1.14 | 0.52 ± 0.66 |
| Anteroposterior shear ($\times 10^4$ n) | 6.93 ± 4.68 | 4.18 ± 3.06 |
| Compression ($\times 10^4$ n) | 32.76 ± 25.97 | 18.25 ± 11.29 |
| Muscle coactivity§ | | |
| Coactivity index | 0.32 ± 0.19 | 0.20 ± 0.16 |

* Bold values indicate significant differences between LBP and asymptomatic groups ($P < 0.05$).
† Peak spinal loads predicted by the electromyogram-assisted model.
‡ Peak spinal loads normalized to the sagittal trunk moment.
§ The relative muscle activity corresponding to the sum of the antagonistic muscle activity (restuc abdominus, external oblique) divided by the agonistic activity (latissimus dorsi, erector spinae, internal obliques).
‖ Significant interactions between injury group and the other independent variables (weight lifted or region of lift origin) are shown in Figures 6 through 8.
¶ The summed value of the instantaneous spinal load during the entire trial.

## Results

### *Phase 1*

Evaluation of the controlled exertions indicated that the LBP group experienced significantly greater (47%) absolute compression and lateral shear (103%) than the asymptomatic group (Table 3). When the spine loadings were adjusted for the greater body mass of the LBP group (*via* moment normalization), significant increases in compression (26.3%) and lateral shear (75.5%) were still present for the LBP group (Figure 4). In addition, the LBP group exhibited statistically significant increases in muscle activities for all 10 muscles, averaging 123% of the asymptomatic group values (Figure 5). The coactivity index for the LBP group was significantly larger (0.46) than the index for the asymptomatic group (0.31) (Table 3). These analyses indicate that when subjects with LBP

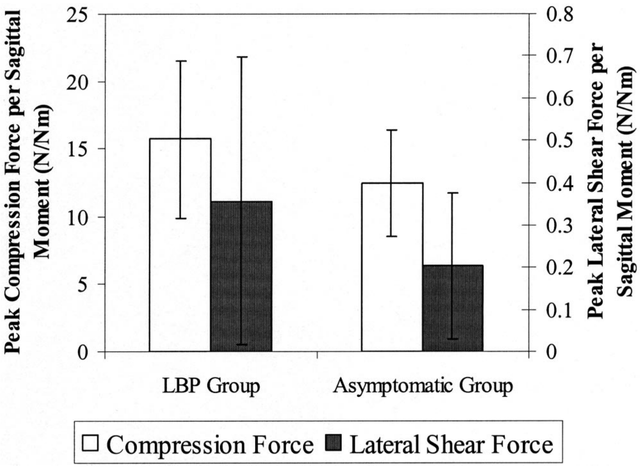



Figure 4. Normalized lateral shear and compression force for individuals with low back pain and asymptomatic individuals during controlled static exertions (Phase 1).

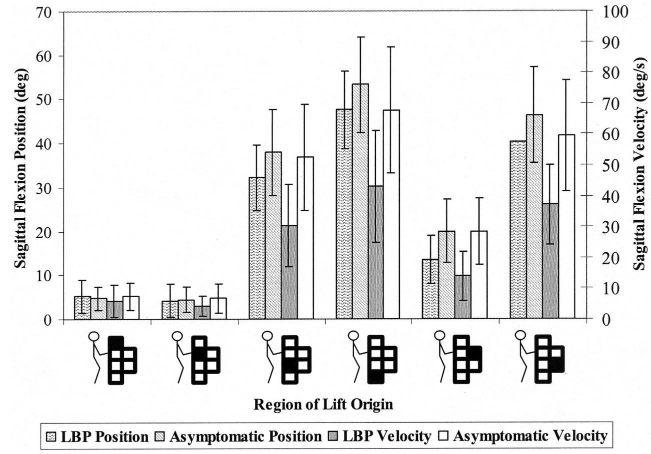



Figure 6. Sagittal trunk position and velocity for individuals with low back pain and asymptomatic individuals as a function of lift origin as identified by shaded box (Phase 2).

and asymptomatic subjects perform the same exact exertion, the absolute and relative biomechanical costs to the patients with LBP are much greater.

As expected (but not shown), increases in exertion levels also resulted in significant linear increases in both absolute and normalized spine loads. No interactions between group membership and exertion levels were significant.

### *Phase 2*

Under the free-dynamic lifting conditions, the patients with LBP experienced approximately 20% greater magnitude of external moment and about 21% greater absolute spine compression (Table 3). They also experienced significantly larger cumulative compression, lateral shear, and anteroposterior shear. As expected, region and weight lifted significantly influenced many of the spine loading measures. Kinematic analyses indicated that the participants with LBP significantly reduced their trunk and hip kinematics in terms of sagittal position and velocity, as compared with the asymptomatic group, when lifting from origins farther away from the spine (greater moment arms), and when lifting from locations with a lower vertical origin (Figure 6). They therefore did not bend as far forward as the asymptomatic group, yet their increased torso mass caused a net increase in external moment.

Cumulative loading was particularly large (average, >70%) in the LBP group when the participants lifted from lower origins (Figure 7), and when they lifted the heavier box weights (Figure 8). The kinematic compensations were effective at reducing the relative effect of spine loading, as evidenced by a lack of significance for any spine loading variables normalized per unit of moment exposure. However, this was accomplished at the cost of greatly restricted movement.

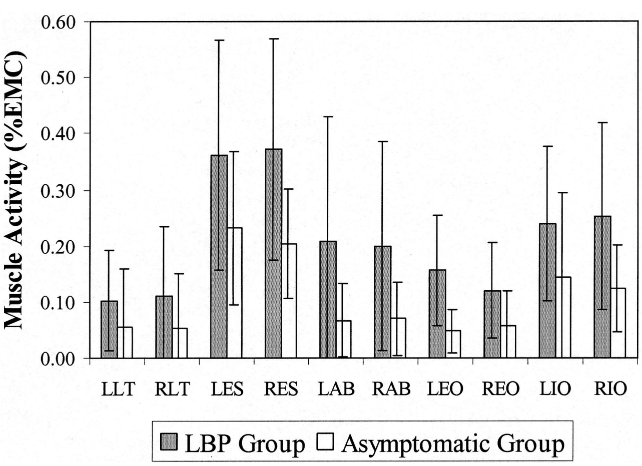



Figure 5. Muscle activities for the 10 trunk muscles (LLT = left latissimus dorsi; RLT = right latissimus dorsi; LES = left erector spinae; RES = right erector spinae; LRA = left rectus abdominus; RRA = right rectus abdominus; LEO = left external oblique; REO = right external oblique; LIO = left internal oblique; RIO = right internal oblique) for individuals with low back pain and asymptomatic individuals during controlled static exertions (Phase 1).

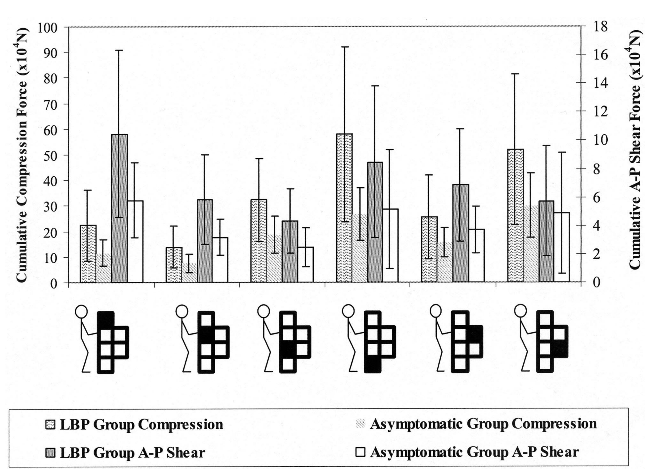



Figure 7. Cumulative compression and anteroposterior shear forces for individuals with low back pain and asymptomatic individuals as a function of lift origin as identified by shaded box (Phase 2).

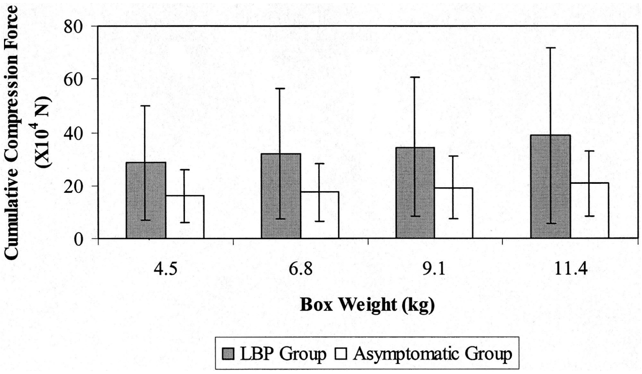



Figure 8. Cumulative compression forces for individuals with low back pain and asymptomatic individuals as a function of box weight (Phase 2).

As in Phase 1, the LBP group displayed significantly more muscle activity in all 10 trunk muscles (Figure 9). The LBP group also displayed greater muscle antagonistic coactivity (index = 0.32) than the asymptomatic group (index = 0.2).

## ■ Discussion

This study represents the first quantitative EMG-assisted biomechanical evaluation of spine loading differences between individuals with and those without LBP. The use of a new EMG calibration procedure in conjunction with a well-developed EMG-assisted model allowed the authors to show that LBP subjects experience significantly greater spine loads than asymptomatic individuals when subjected to the same postural loading conditions. After normalization for differences in body mass, patients with LBP experienced 26% more compression and 75% more lateral shear than their asymptomatic counterparts. The increased spine loads were related to increased trunk muscle coactivity. These loads can further increase when the impact from the additional body weight of patients with LBP is considered. In the current study, when the body mass of the patients with LBP also was considered, absolute compression values represented almost a 50% increase over those for the asymptomatic group and more than a 100% increase in lateral shear over that for the asymptomatic group under all exertion conditions.

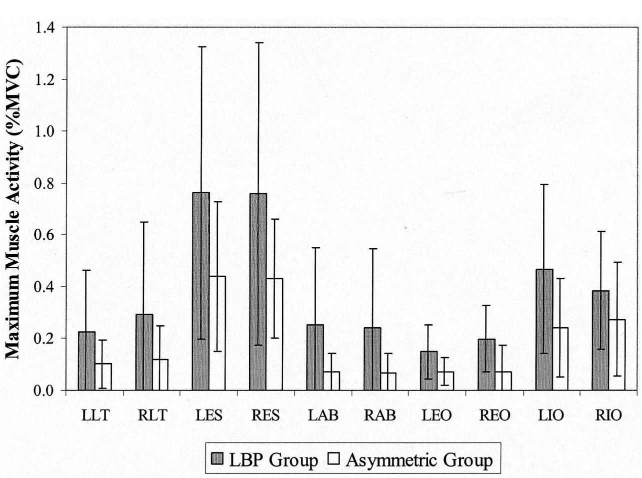



Figure 9. Peak muscle activities for the ten trunk muscles (LLT = left latissimus dorsi; RLT = right latissimus dorsi; LES = left erector spinae; RES = right erector spinae; LRA = left rectus abdominus; RRA = right rectus abdominus; LEO = left external oblique; REO = right external oblique; LIO = left internal oblique; RIO = right internal oblique) for individuals with low back pain and asymptomatic individuals (Phase 2).

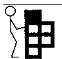
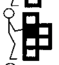
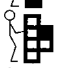
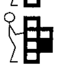
Under unrestricted lifting conditions, the participants with LBP kinematically compensated for their back pain, thereby minimizing external moment exposure. However, the LBP group's coactivity along with their increased body mass offset any spine loading benefits that might have been derived from these kinematic reductions in movements. Therefore, under realistic situations, patients with LBP typically experience significantly greater spine loads.

This logic was confirmed by comparing a subsample of five LBP-asymptomatic subject pairs matched for both height and weight. Under Phase 1 conditions, this LBP group subset experienced 28% and 38% greater normalized and absolute compression, respectively. However, under Phase 2 conditions, the LBD group reduced their flexion by 30% and their torso velocity by 60%. These changes reduced the external moment to levels that were less than those of their matched asymptomatic group counterparts, but resulted in virtually identical spine loads. Hence, although the LBP group compensated by reducing the moment imposed about the torso, their increased trunk muscle coactivaiton offset the gains derived through moment reduction. The larger body mass of the typical patient with LBP[11,12,29] would further increase spine loading.

The degree of spine loading experienced by the LBP group was strongly dependent on the lift origin height and the moment arm distance, with greater spine loading occurring at lower lift heights and farther from the spine. These also are the regions that would be expected to impose greater moments on the spines of the LBP group because of their greater torso mass. Furthermore, these same conditions were associated with greater differences in coactivations, and most likely were those related to guarding. These findings suggest a strong musculoskeletal system reaction to the patient's body weight during a lift when the moment is great or the lift must be performed at a low level.

These findings suggest two implications. First, body weight appears to play a major role in spine loading for the LBP group, imposing much greater spine loading than would be expected for asymptomatic subjects of equal size. The findings suggest that patients with LBP could benefit greatly from weight control during the recovery process. Second, when patients with LBP return to a workplace that requires materials handling, it is even more important that the workplace be designed to accommodate their capabilities than it would be for asymptomatic individuals. In the case of the patient with

**Table 4. Summary of Regression Relation Between the Percentage of Normal (Impairment Level) and the Spine Loads Within Each of the Regions of Lift Origin***

| | Absolute Lateral Shear Force | | Absolute Anteroposterior Shear Force | | Absolute Compression Force | | Cumulative Lateral Shear Force | | Cumulative Anteroposterior Shear Force | | Cumulative Compression Force | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Region of Lift | Model | $R^2$ | Model | $R^2$ | Model | $R^2$ | Model | $R^2$ | Model | $R^2$ | Model | $R^2$ |
|  | Cubic | 0.08 | Linear | 0.04 | Linear | 0.08 | Linear | 0.11 | Logarithmic | 0.34 | Logarithmic | 0.35 |
|  | Linear | 0.05 | Linear | 0.06 | Cubic | 0.12 | Linear | 0.09 | Logarithmic | 0.40 | Logarithmic | 0.35 |
|  | Quadratic | 0.06 | Linear | 0.03 | Linear | 0.06 | Linear | 0.11 | Logarithmic | 0.23 | Logarithmic | 0.38 |
|  | Quadratic | 0.05 | Quadratic | 0.05 | Quadratic | 0.18 | Cubic | 0.14 | Cubic | 0.18 | Logarithmic | 0.46 |
|  | Linear | 0.03 | Linear | 0.04 | Cubic | 0.07 | Cubic | 0.13 | Logarithmic | 0.38 | Logarithmic | 0.41 |
|  | Quadratic | 0.08 | Linear | 0.03 | Quadratic | 0.05 | Linear | 0.06 | Quadratic | 0.03 | Logarithmic | 0.40 |

* Model indicates the highest-order significant regression model. $R^2$ shows the percentage of variance explained.

LBP, it is important to arrange the workplace so that loads can be lifted at waist height with the lift origin close to the body.

Because guarding and trunk muscle coactivation appear to exert a strong influence on the magnitude of spine loading, it might be expected that patients with LBP and greater impairment might generate more coactivation, and therefore greater spine loading. In the current study, follow-up analyses identified many significant trends between the extent of low back disorder, as measured by the authors' functional analysis test, and both peak and cumulative spine loading. Table 4 summarizes these relations for various lift origins, showing that nonlinear models can explain up to 45% of the variance between impairment and spine compression. There is a monotonic, yet nonlinear, relation between spine loading and degree of impairment as measured in this study.[34,42] Because the functional analysis test is a measure of the musculoskeletal system's ability to recruit muscles synergistically in an efficient manner,[34] patients may derive more benefit from kinematic conditioning than from strength conditioning.

These increases in spine loading associated with LBP may accelerate degeneration of the spine. Adams et al[1–3] recently showed that even minor damage to a vertebral body endplate leads to progressive structural changes in the intervertebral discs. In addition, because the patients with LBP performed the lifting tasks over a longer period, there may also have been an additional risk because of cumulative spine loading. For the tasks described in our study, the cumulative lateral shear, anteroposterior, and compressive loads were 44%, 59%, and 57% higher, respectively, for patients with LBP. Recent studies have suggested that increased LBP risk can indeed be related to cumulative loading.[23,52] In addition, recent efforts[2] have found that sustained loading (suspected under slower movement conditions) result in higher concentrations of stress within the intervertebral discs. Therefore, given the current findings, it would be expected that the risk of further spine damage would be even greater when patients with LBP are exposed to greater levels of cumulative loading.

Finally, several potential limitations must be considered in a study such as this. First, the exposure to loading occurred over a short (2-hour) test period. Under realistic, longer exposure to lifting conditions, the detrimental effects of spine loading may be much greater. However, subjects may adapt better under these prolonged conditions. Further testing is needed to resolve this issue. Second, the current LBP population consisted primarily of patients with pain from suspected muscular origins. A similar study needs to be conducted in which patients with structural disorders are tested to determine whether they would respond in a similar fashion. Third, future studies may want to explore spine loading for patients with acute, as compared with chronic, LBP. The LBP population in the current study did not offer statistical power sufficient for exploring this issue. Fourth, the new normalization technique is based on the assumption that both LBP and asymptomatic individuals have the same muscle control strategies. Future research needs to establish whether a difference may exist. Finally, spine loading was evaluated in this study for a limited range of materials-handling tasks. Future studies could use similar spine-loading analysis techniques to investigate spine loads associated with other forms of work performed by patients with LBP. This might include more complex lifts, pushing–pulling, carrying, lifting while the feet are moved, or seated work tasks.

In conclusion, this is the first EMG-driven biomechanical evaluation of spine loading in patients with LBP, as compared with spine loading in asymptomatic individuals. It has shown that spine loading is significantly greater for patients with LBP than for asymptomatic subjects during materials-handling tasks. Spine loading increased with increasing impairment, primarily as result of increased trunk muscle coactivation. Although patients with LBP kinematically adjusted their postures to minimize external moment exposure, these compensations did not offset the increased loading resulting from coactivation (guarding) and from the large body mass typical of patients with LBP. The study suggests that most patients with LBP would benefit greatly from reduction in body weight, kinematic conditioning, and efforts to design materials-handling tasks so that lift origins are close to the body and at reasonable lift heights.

## ■ Key Points

- Patients with low back pain produced higher spine loads during highly controlled exertions than their asymptomatic counterparts.
- During free-dynamic lifting, patients with low back pain were found to compensate by reducing flexion and motion, thus reducing trunk moment.
- However, the impact of body mass and increased muscle coactivity was greater than the kinematic compensation because patients with low back pain had higher spinal compression, particularly when lift origins were below the waist.

## Acknowledgment

The authors acknowledge the contribution of Michael Jorgensen to certain parts of this study.

## References

1. Adams MA, Freeman BJ, Morrison HP, et al. Mechanical initiation of intervertebral disc degeneration. Spine 2000;25:1625–36.
2. Adams MA, McMillan DW, Green TP, et al. Sustained loading generates stress concentrations in lumbar intervertebral discs. Spine 1996;21:434–8.
3. Adams MA, McNally DS, Dolan P. "Stress" distributions inside intervertebral discs: the effects of age and degeneration. J Bone Joint Surg [Br] 1996; 78:965–72.
4. Ahern DK, Follick MJ, Council JR, et al. Comparison of lumbar paravertebral EMG patterns in chronic low back pain patients and nonpatient controls. Pain 1988;34:153–60.
5. Ahern DK, Hannon DJ, Goreczny AJ, et al. Correlation of chronic low back pain behavior and muscle function examination of the flexion–relaxation response. Spine 1990;15:92–5.
6. Arena JG, Sherman RA, Bruno GM, et al. Temporal stability of paraspinal electromyographic recordings in low back pain and nonpain subjects. Int J Psychophysiol 1990;9:31–7.
7. Bean JC, Chaffin DB, Schultz AB. Biomechanical model calculation of muscle contraction forces: a double linear programming method. J Biomech 1988; 21:59–66.
8. Chaffin DB. A computerized biomechanical model: development of and use in studying gross body actions. J Biomech 1975;2:429–41.
9. Chiou W-K, Lee Y-H, Chen W-J. Use of surface EMG coactivational pattern for functional evaluation of trunk muscles in subjects with and without low back pain. International Journal of Industrial Ergonomics 1999;23:51–60.
10. Davis KG, Marras WS, Waters TR. The evaluation of spinal loads during lowering and lifting. Clin Biomech 1998;13:141–52.
11. Dempsey PG, Burdorf A, Webster BS. The influence of personal variables on work-related low back disorders and implications for future research. J Occup Environ Med 1997;39:748–59.
12. Deyo RA, Bass JE. Lifestyle and low back pain: the influence of smoking and obesity. Spine 1989;14:501–6.
13. Fathallah FA, Marras WS, Parnianpour M, et al. A method for measuring external spinal loads during unconstrained free-dynamic lifting. J Biomech 1997;30:975–8.
14. Freivalds A, Chaffin DB, Garg A, et al. A dynamic biomechanical evaluation of lifting maximum acceptable loads. J Biomech 1984;17:251–62.
15. Granata KP, Marras WS. An EMG-assisted model of loads on the lumbar spine during asymmetric trunk extensions. J Biomech 1993;26:1429–38.
16. Granata KP, Marras WS. An EMG-assisted model of trunk loading during free-dynamic lifting. J Biomech 1995;28:1309–17.
17. Granata KP, Marras WS. The influence of trunk muscle coactivity on dynamic spinal loads. Spine 1995;20:913–9.
18. Granata KP, Marras WS, Davis KG. Variation in spinal load and trunk dynamics during repeated lifting exertions. Clin Biomech 1999;14:367–75.
19. Granata KP, Marras WS, Fathallah FA. A method for measuring external loads during dynamic lifting exertions. J Biomech 1996;29:1219–22.
20. Jorgensen MJ, Marras WS, Granata KP, et al. Moment arm distances of female and male trunk loading muscles. Clin Biomech 2001;16:182–93.
21. Keller A, Johansen JG, Hellesnes J, et al. Predictors of isokinetic back muscle strength in patients with low back pain. Spine 1999;24:275–80.
22. Kishino ND, Mayer TG, Gatchel RJ, et al. Quantification of lumbar function: Part 4. Isometric and isokinetic lifting simulation in normal subjects and low back dysfunction patients. Spine 1985;10:921–7.
23. Kumar S. Cumulative load as a risk factor for back pain. Spine 1990;15: 1311–6.
24. Kumar S, Dufresne RM, Van Schoor T. Human trunk strength profile in flexion and extension. Spine 1995;20:160–8.
25. Lariviere C, Gagnon D, Loisel P. An application of pattern recognition for the comparison of trunk muscles EMG waveforms between subjects with and without chronic low back pain during flexion–extension and lateral bending tasks. J Electromyogr Kinesiol 2000;10:261–73.
26. Lariviere C, Gagnon D, Loisel P. The comparison of trunk muscles EMG activation between subjects with and without chronic low back pain during flexion–extension and lateral bending tasks. J Electromyogr Kinesiol 2000; 10:79–91.
27. Lariviere C, Gagnon D, Loisel P. The effect of load on the coordination of the trunk for subjects with and without chronic low back pain during flexion–extension and lateral bending tasks. Clin Biomech 2000;15:407–16.
28. Leach RA, Owens EF Jr, Giesen JM. Correlates of myoelectric asymmetry detected in low back pain patients using hand-held post-style surface electromyography. J Manipulative Physiol Ther 1993;16:140–9.
29. Leboeuf-Yde C. Body weight and low back pain: a systematic literature review of 56 journal articles reporting on 65 epidemiologic studies. Spine 2000;25:226–37.
30. Lee JH, Ooi Y, Nakamura K. Measurement of muscle strength of the trunk and the lower extremities in subjects with history of low back pain. Spine 1995;20:1994–6.
31. Marras WS, Davis KG. A non-MVC EMG normalization techniques for the trunk musculature: Part 1. Method development. J Electromyogr Kinesiol 2001;11:1–9.
32. Marras WS, Davis KG, Maronities A. A non-MVC EMG normalization techniques for the trunk musculature: Part 2. Validation and use to predict spinal loads. J Electromyogr Kinesiol 2001;11:11–8.
33. Marras WS, Fathallah F, Miller RJ, et al. Accuracy of a three-dimensional lumbar motion monitor for recording dynamic trunk motion characteristics. International Journal of Industrial Ergonomics 1992;9:75–87.
34. Marras WS, Ferguson SA, Gupta P, et al. The quantification of low back disorder using motion measures: methodology and validation. Spine 1999; 24:2091–100.
35. Marras WS, Granata KP. A biomechanical assessment and model of axial twisting in the thoracolumbar spine. Spine 1995;20:1440–51.
36. Marras WS, Granata KP. Spine loading during trunk lateral bending motions. J Biomech 1997;30:697–703.
37. Marras WS, Granata KP. The development of an EMG-assisted model to assess spine loading during whole-body free-dynamic lifting. J Electromyogr Kinesiol 1997;7:259–68.
38. Marras WS, Jorgensen MJ, Granata KP, et al. Size and direction of female and male trunk loading muscles. Clin Biomech 2001;16:38–46.
39. Marras WS, Lewis KE, Ferguson SA, et al. Impairment magnification during dynamic trunk motions. Spine 2000;25:587–95.
40. Marras WS, Mirka GA. A comprehensive evaluation of trunk response to asymmetric trunk motion. Spine 1992;17:318–26.
41. Marras WS, Mirka GA. Electromyographic studies of the lumbar trunk musculature during the generation of low-level trunk acceleration. J Orthop Res 1993;11:811–7.
42. Marras WS, Parnianpour M, Ferguson SA, et al. The classification of anatomic- and symptom-based low back disorders using motion measure models. Spine 1995;20:2531–46.

43. Marras WS, Reilly CH. Networks of internal trunk-loading activities under controlled trunk-motion conditions. Spine 1988;13:661–7.
44. Marras WS, Sommerich CM. A three-dimensional motion model of loads on the lumbar spine: I. Model structure. Hum Factors 1991;33:123–37.
45. Marras WS, Sommerich CM. A three-dimensional motion model of loads on the lumbar spine: II. Model validation. Hum Factors 1991;33:139–49.
46. Marras WS, Wongsam PE. Flexibility and velocity of the normal and impaired lumbar spine. Arch Phys Med Rehabil 1986;67:213–7.
47. Mayer T, Gatchel R, Betancur J, et al. Trunk muscle endurance measurement: isometric contrasted to isokinetic testing in normal subjects. Spine 1995;20:920–7.
48. Mayer TG, Smith SS, Keeley J, et al. Quantification of lumbar function: Part 2. Sagittal plane trunk strength in chronic low back pain patients. Spine 1985;10:765–72.
49. Mayer TG, Smith SS, Kondraske G, et al. Quantification of lumbar function: Part 3. Preliminary data on isokinetic torso rotation testing with myoelectric spectral analysis in normal and low back pain subjects. Spine 1985;10:912–20.
50. Mirka GA, Marras WS. A stochastic model of trunk muscle coactivation during trunk bending. Spine 1993;18:1396–409.
51. Newton M, Thow M, Somerville D, et al. Trunk strength testing with iso-machines: Part 2. Experimental evaluation of the Cybex II Back Testing System in normal subjects and patients with chronic low back pain. Spine 1993;18:812–24.
52. Norman R, Wells R, Neumann J, et al. A comparison of peak *vs* cumulative physical work exposure risk factors for the reporting of low back pain in the automotive industry. Clin Biomech 1998;13:561–73.
53. Nussbaum MA, Chaffin DB, Rechtien CJ. Muscle lines-of-action affect predicted forces in optimization-based spine muscle modeling. J Biomech 1995; 28:401–9.
54. Peach JP, Sutarno CG, McGill SM. Three-dimensional kinematics and trunk muscle myoelectric activity in the young lumbar spine: a database. Arch Phys Med Rehabil 1998;79:663–9.
55. Radebold A, Cholewicki J, Panjabi MM, et al. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. Spine 2000;25:947–54.
56. Reilly CH, Marras WS. Simulift: a simulation model of human trunk motion. Spine 1989;14:5–11.
57. Roy SH, De Luca CJ, Casavant DA. Lumbar muscle fatigue and chronic lower back pain. Spine 1989;14:992–1001.
58. Roy SH, De Luca CJ, Emley M, et al. Spectral electromyographic assessment of back muscles in patients with low back pain undergoing rehabilitation. Spine 1995;20:38–48.
59. Schultz AB, Andersson GB. Analysis of loads on the lumbar spine. Spine 1981;6:76–82.
60. Triano JJ, Schultz AB. Correlation of objective measure of trunk motion and muscle function with low back disability ratings. Spine 1987;12:561–5.
61. Wood S, Pearsall DJ, Ross R, et al. Trunk muscle parameters determined from MRI for lean to obese males. Clin Biomech 1996;11:139–44.

*Address reprint requests to*

William S. Marras, PhD
*Biodynamics Laboratory*
*Ohio State University,*
*1971 Neil Avenue*
*Columbus, OH 43210*
*E-mail: marras.1@osu.edu*