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SPINE LOADING DURING TRUNK LATERAL BENDING MOTIONS

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Abstract—Increases in lateral trunk velocities have been identified as a mechanism for increasing the risk of low-back disorder. Previous studies have identified an increase in coactivation of the trunk musculature during lateral bends, but no studies have evaluated how spine loading changes as lateral trunk velocity increases. Twelve subjects were asked to lift loads laterally at one static and three dynamic velocities. Ten trunk muscle activities and trunk kinematics were documented and used as input parameters to an EMG-assisted model to evaluate spine loading. Muscle coactivation was observed in all lateral bends. Coactivation significantly increased during dynamic trials compared to the static trials. Coactivity increased spinal loads by as much as 25% compared to values predicted by models that did not consider coactivity. Movements to the right significantly increased spine loadings (252 N increase in compression) compared to movements to the left. Spine compression, A-P shear, and lateral shear all increased in the dynamic trials compared to the static conditions. Peak compression increased by an average of 525 N at 45° s⁻¹ compared to static loading. Compression and lateral shear increased monotonically as trunk velocity increased. It is expected that this combined (compression and lateral shear) loading is the mechanism for increased risk observed in industry. © 1997 Elsevier Science Ltd

Keywords: Spine; Model; Lateral bendig; EMG; Low-back disorder.

INTRODUCTION

Lateral bending of the trunk has been identified by several researchers as a risk factor for low-back disorders. Lateral spine loading increases the risk of scoliosis (Noone et al., 1993) and when coupled with movements in other trunk directions increases the risk of low-back pain (Fathallah, 1995; Haas and Nyiendo, 1992). Industry-based studies have shown that increases in lateral bending velocity during work-related tasks significantly increases the risk of occupationally-related low-back disorder (Marras et al., 1993, 1995). Thus, greater levels of lateral bending velocity magnitude have been identified as an important risk factor for increased risk of low-back disorder, however, it has not been determined how and to what extent velocity increases the mechanical load on the spine and the subsequent risk of low-back disorder.

We hypothesize that increases in lateral bending velocity would require increased antagonistic coactivity of the trunk musculature which would result in an increase in spinal loading. The complexity of trunk muscle coactivation has been documented under lateral bending conditions (Lavender et al., 1995; McGill, 1992). However, the issue of lateral bending velocity has not been addressed during these assessments. The degree of muscle coactivation in such an activity suggests that EMG-assisted models would be best suited to assess the loading of the spine. Several researchers have developed EMG-assisted models capable of assessing spinal loads during non-sagittally symmetric bending conditions. McGill (1992) developed an anatomically detailed EMG-assisted model of lateral bending motions containing 50 muscles

and 12 ligaments. Other models (Thelan et al., 1994) have demonstrated that it is possible to assess simultaneously spinal loads in three dimensions under static loading conditions. An EMG-assisted model developed in our laboratory has been specifically designed to evaluate the role of dynamic trunk motions under symmetric (Marras and Sommerich, 1991a, b), asymmetric (Granata and Marras, 1993, 1995) and twisting (Marras and Granata, 1995) trunk loading conditions.

The goal of this study was to determine how spine loading changes as a function of lateral trunk velocity in a load-supporting task. In order to achieve this goal it was necessary to document trunk musculature behavior and the subsequent influence on three-dimensional spine loading.

MATERIALS AND METHODS

An EMG-assisted model was used to assess spine compression, lateral shear, and anterior-posterior (A-P) shear under lateral trunk motion loading conditions. The model has been developed in our laboratory over the past decade (Granata and Marras, 1993, 1995b; Marras and Granata, 1995; Marras and Reilly, 1988; Marras and Sommerich, 1991a, b; Reilly and Marras, 1989). Generally, the model conditions (modulates) the EMG signals (to account for muscle velocity, length, gain, and crosssectional area) collected from ten trunk muscles so that a vector force contribution can be determined for each muscle. The vector components are then summed in each cardinal plane to determine compression and shear forces acting on the spine. The model used in this study employed the muscle insertion data described in Granata and Marras (1995b) and the muscle cross-sectional areas described in Marras and Granata (1995). While details of the most current version of the model have been reported extensively in these two papers, only modifications to the model will be reported here. The model required a slight

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Table 1. Modifications to the length and velocity modulation factors used in the biomechanical model

Muscle length modulation factor F (length)

$$F(\text{Length}) = -3.25 + 10.20 \times \text{Length} - 10.40 \times \text{Length}^2 + 4.59 \times \text{Length}^3$$

where

Length = f(Sagtl Ang, Latrl Ang/2, Twist Ang)

Muscle velocity modulation factor F(Vel)

$$F(\text{Vel}) = 0.65 + 0.35 \times \exp(-\text{Vel}/.025) \qquad \text{for Vel} \le 0$$

$$F(\text{Vel}) = 1.5 \qquad \text{for Vel} > 0$$

Note. The length modulation factors, F(Length), were necessary to account for lateral trunk angle as defined by the trunk angle goniometer. The velocity modulation factor was described as an exponential that closely matched the polynomial form in the appropriate region, but allowed a more realistic description of the force-velocity at greater muscle contraction velocities (|Vel| > 1.2 Rest Lengths/sec).

adjustment to the muscle length and velocity modulation components to account for lateral trunk angle as defined by our instrumentation (Table 1). A back monitor was used to measure trunk angle as an angle between L5/S1 and T10. Since the monitor did not take into account lateral spine curvature due to the changing angle of each vertebral segment a correction factor was needed to adjust for over-prediction of angle and velocity.

Model performance or validity among the trial runs was assessed via several measures. The trunk moment predicted by the model (via EMG processing) was compared with the measured moment. This comparison is made by assessing the R^2 statistic which assessed the trend in the changing applied moment. In addition, the average absolute error (AAE) between the predicted and measured moment was assessed for the magnitude of difference. The muscle gain value and consistency from trial to trial were also assessed as measures of model stability and to ensure that the gain value was physiologically reasonable.

Twelve male volunteers from 24 to 33 yr of age participated in this experiment. Average (S.D.) subject height was 180 cm (6.3 cm) and average subject weight was 78 kg (10.2 kg). Trunk anthropometry measures were also collected for modeling purposes. The protocol for human subjects was approved by the Human Subjects Committee at the Ohio State University.

Subjects performed static and isokinetic lateral exertions while standing on a force plate (Bertec 4060A) with their hips and legs strapped into a structure designed to isolate the lower body and transmit all forces and moments into the force plate. A light-weight freely rotating assembly was constructed to provide the subject with tactile (guidance) feedback so they could maintain the trunk motions in a purely lateral plane (Fig. 1). A lumbar motion monitor (LMM) was also worn by the subjects to record three-dimensional trunk motions. Subjects observed a real-time display of their lateral trunk angles and motions, permitting them to control their trunk position and isokinetic velocity within a displayed motion envelope. The static and dynamic exertions were performed while the subjects held weights in their right

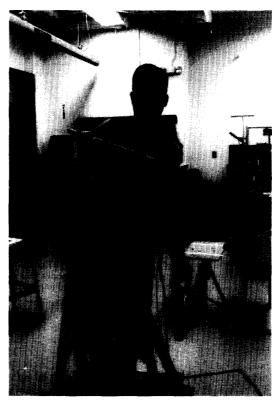


Fig. 1. Subjects performed static and isokinetic lateral exertions while standing on a force plate with their hips and legs strapped into a structure designed to isolate the lower body and transmit all forces and moments into the force plate. A light-weight freely-rotating assembly provide subjects with tactile (guidance) feedback so they could maintain trunk motions in a purely lateral plane. Trunk motion was recorded by a lumbar motion monitor (LMM) electrogoniometer and muscle activity was monitored from ten bipolar surface electrode pairs.

hand, i.e. left lateral exertion, or left hand, i.e. right lateral exertion. Subjects were required to hold the weights away from their bodies throughout the entire exertion so as to avoid dragging the weight along their side.

Independent variables consisted of three isometric lateral trunk angle conditions (15° left, 0° (upright), 15° right), three isokinetic lateral trunk velocity conditions (15, 30, and 45° s⁻¹), two weight levels (13.6, 27.3 kg), and two exertion directions (right, left). The lateral angles and velocities were chosen to represent the typical maximum lateral bend angles and range of average trunk velocities observed in low risk (static conditions) and high risk (dynamic conditions) of LBD among industrial jobs (Marras *et al.*, 1993, 1995). Each subject performed each experimental condition once. Dependent variables included the measured force plate data, three-dimensional trunk motions, ten channels of EMG activity, and the modeled three-dimensional spinal loads.

Ten channels of EMG data were collected from bipolar surface electrodes over the right and left sides of the erector spinae, rectus abdomini, latissimus dorsi, external abdominal obliques, and internal abdominal oblique muscles as described in Marras and Mirka (1992). The EMG signals were amplified initially using a remote

preamplifier at the muscle site in order to minimize noise. Further amplification was performed at the main amplifier where each channel's gain was individually adjusted to optimize the EMG recording. The raw EMG signal were high pass filtered at 30 Hz and low pass filtered at 1000 Hz. The signals were further processed using an RMS procedure. EMG data were normalized by myoelectric maxima recorded from each muscle during a series of six static calibration exertions. Data describing external kinetic loads about the L5/S1 spine [translated from force plate data via Granata et al. (1995)], trunk motions, and RMS processed EMG data were simultaneously smoothed using a 10 Hz Hanning filter.

Further sensitivity analyses were performed to examine the influence of muscle coactivity, upon spinal loads. This was accomplished by removing the antagonistic muscle activity from the data set. These adjusted data were re-modeled, and the output compared with the analysis of the fully coactive data set.

Quantitative analyses of both the muscle activities and model predictions were performed so that statistically significant differences could be identified. Analysis of variance (ANOVA) was used to determine whether the mean normalized electromyographic activity (among the 12 subjects) observed during peak moment generation differed for each of the ten muscles between the lateral velocity conditions. Statistical significance for this analysis was determined apriori at a level of p < 0.05. Model predictions were dependent upon the collective activity of the trunk muscles in that trade-offs in muscle activities influence the nature of spinal loading (compression vs shear forces). Therefore, multivariate analysis of variance (MANOVA) was used to determine whether the spinal load measures (collectively) changed as a function of the experimental conditions. ANOVA was employed to further assess whether the individual spine load measure was affected by the experimental conditions and interactions. Statistical significance for both the MANOVA and ANOVA were also established a priori at p < 0.05.

RESULTS

Muscle activities during lateral exertions were dominated by the agonist muscles which had horizontal muscle-orientation components (Table 2). The electromyographic activity of the ipsilateral latissimus dorsi, external oblique and internal oblique muscles were significantly greater than the activities of the erector spinae, rectus abdomini, and contralateral latissimus dorsi and oblique muscles. Changes in normalized EMG level as a function of lateral flexion angle were not statistically significant.

There was significant contralateral as well as ipsilateral equipoise muscle contraction during the lateral exertions. EMG activity of the rectus abdomini and erector spinae ranged from 13 to 39% of their MVC values on the ipsilateral side and 3-6% on the contralateral side (Table 2), and was influenced significantly by lateral velocity, exertion level, and direction. Antagonistic activity of the contralateral latissimus dorsi, internal, and external obliques was also statistically greater than zero (3-9% of MVC). Latissimus dorsi and external oblique antagonistic

Table 2. Summary of muscle activity as a function of trunk lateral velocity

Muscle	Lateral velocity (° s ⁻¹)								
	Right lateral exertions				Left lateral exertions				
	0	15	30	45	0	15	30	45	
Rt Lat	0.46	0.75	0.72	0.74	0.04	0.08	0.09	0.09	
Lt Lat	0.03	0.06	0.07	0.07	0.43	0.68	0.69	0.70	
Rt ErSp	0.23	0.36	0.38	0.39	0.03	0.05	0.05	0.06	
Lt ErSp	0.03	0.05	0.05	0.05	0.18	0.35	0.38	0.39	
Rt RAbd	0.17	0.24	0.27	0.28	0.04	0.06	0.06	0.06	
Lt RAbd	0.04	0.05	0.06	0.05	0.13	0.19	0.20	0.21	
Rt ExO	0.31	0.51	0.54	0.54	0.05	0.06	0.07	0.08	
Lt ExO	0.06	0.08	0.09	0.08	0.32	0.58	0.63	0.64	
Rt InO	0.40	0.70	0.73	0.75	0.05	0.06	0.07	0.08	
Lt InO	0.04	0.06	0.06	0.07	0.39	0.69	0.72	0.75	

Note. Mean activity level of all subjects during the generation of peak moment (in % MVC, where $\max=1.0$) of the prime movers were significantly greater during dynamic exertions than during static exertions, however there was no statistically significant difference in the normalized EMG levels between dynamic exertions.

*Bold entries indicate significantly (p < 0.05) different EMG levels than levels at other velocities.

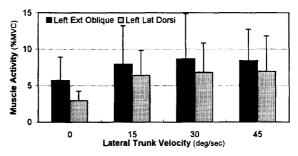


Fig. 2. Electromyographic activity (mean and standard deviation of 12 subjects) of the antagonistic musculature during generation of peak moment were statistically greater than zero and increased significantly with velocity and exertion level. The antagonistic activity is readily observed in the left latissimus dorsi and left external obliques during right lateral exertions.

activity increased significantly with velocity (Fig. 2), and exertion level.

Prime movers were significantly more active during dynamic exertions than during static exertions. However there was no statistically significant difference between the normalized EMG levels among dynamic exertions (Table 2).

The EMG-assisted model performed well under the experimental conditions and accurately predicted static and dynamic lateral trunk moments. Gain and AAE values were relatively stable and were not greatly influenced by experimental conditions. The average gain value predicted by the model was 64.9 N cm⁻² which is within the physiological range. Gain values were not affected significantly by trunk angle, velocity, or weight level. Greater gain values were predicted when modeling exertions toward the left than toward the right. However, subjects were able to generate on average 12% greater muscular force to the left. This agrees with anatomical CT surveys (McGill et al., 1988) indicating the left side internal and external oblique muscles were larger in

cross-sectional area by approximately 13% at L4/L5 level. The mean AAE between predicted and measured trunk moments was between 6 and 10% among the experimental conditions. The AAE statistic did not fluctuate significantly as a function of experimental weight level or lateral direction conditions. The AAE was slightly greater for laterally flexed postures (8.3% of the maximum lateral moment) than for upright postures (6.5%), and was significantly (p < 0.05) lower for static exertions (4.8%) than for the dynamic exertions (9.9% at 15° s⁻¹, 11.6% at 30° s⁻¹ and 12.6% at 45° s⁻¹). Average R^2 between measured and predicted moments was 0.91.

Maximum predicted spinal compression, lateral shear, and anterior-posterior shear forces were influenced primarily by the weight supported, velocity of the exertion, and direction of movement (Table 3). Compression was also influenced by an interaction between trunk angle and direction of movement.

As expected, increases in the lifted load significantly increased the magnitude of the spinal loads in all three dimensions. Typical lateral shear loads were between 9 and 34 N, A-P shear loads were between 566 and 869 N, and compressive loads between 1942 and 2764 N.

Spinal loads were significantly influenced by the rate of lateral bending (velocity). Post hoc analyses indicated that the maximum lateral shear, A-P shear, and compressive forces all increased significantly when movement was permitted in the exertion (Fig. 3). Average peak compressive loads were 525 N greater during lateral exertions performed at 45° s⁻¹ than during isometric exertions; this was equivalent to 23% of the static load. Although the magnitude of the difference was not large, average lateral shear load more than tripled in magnitude between the static and 45° s⁻¹ conditions. These trends agree with observations that the static lateral motion conditions are associated with low occupational LBD risk and the dynamic conditions associated with high risk (Marras et al., 1993, 1995).

A-P shear force and compression were significantly influenced by trunk direction, but the trend for A-P shear was opposite that of compression. Left lateral exertions were 15% greater than right lateral exertions and associated with increased compression during static and dynamic trials. The magnitude of this difference upon static compression was 252 N, equivalent to 13% of the

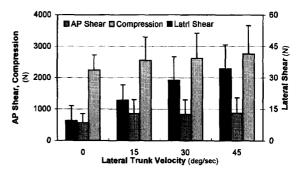


Fig. 3. Spinal loads (mean and standard deviation of 12 subjects) were significantly influenced by lateral velocity. Average peak compressive loads were 525 N greater during lateral exertions performed at $45^{\circ} \, \text{s}^{-1}$ than during isometric exertions, equivalent to 23% of the static load.

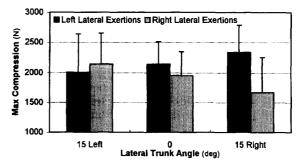


Fig. 4. Compressive load on the spine (mean and standard deviation of 12 subjects) was significantly influenced by lateral flexion angle and direction of motion. Load increased as the trunk was laterally flexed away from the direction of the applied lateral moment.

compression during right lateral exertions. A-P shear was less when the trunk was laterally flexed to the left than when flexed toward the right. The magnitude of the A-P shear force changed an average of 340 N equivalent to approximately 55% of the shear load during upright lateral exertions.

An interesting interaction between trunk angle and direction of motion influenced spinal compression force (Fig. 4). Compressive load on the spine increased as the

Table 3. Summary of statistical significance (p values) among experimental conditions

		ANOVA					
	MANOVA	Max Latrl shear	Max A-P shear	Max compression			
Angle	p < 0.144	p < 0.084	p < 0.118	p < 0.820			
Velocity	p < 0.001*	p < 0.001*	p < 0.001*	p < 0.001*			
Weight	p < 0.001*	p < 0.001*	p < 0.001*	p < 0.001*			
Direction	p < 0.001*	p < 0.175	p < 0.001*	p < 0.001*			
Angle × Weight	p < 0.090	p < 0.901	p < 0.001*	p < 0.494			
Angle × Dir	p < 0.001*	p < 0.473	p < 0.090	p < 0.001*			
Vel × Weight	p < 0.060	p < 0.712	p < 0.267	p < 0.003*			
Weight × Dir	p < 0.453	p < 0.031	p < 0.895	p < 0.756			

Maximum lateral shear, anterior-posterior (A-P) shear and compressive forces were significantly influenced by lateral velocity, lateral exertion level (Weight) and the direction of lateral exertion. Static lateral trunk angle significantly influenced spinal load through interactions with exertion level and direction.

^{*} Indicate statistical significance.

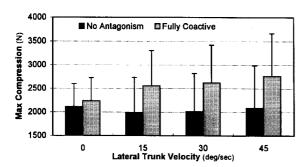


Fig. 5. Compression predictions (mean and standard deviation of 12 subjects) with and without antagonistic coactivation. Analyses which fail to include antagonistic muscle activity predicted static spinal compression values that were 25% lower than the values determined from the fully coactive analyses and failed to show any influence of velocity upon the spinal loads.

trunk was laterally flexed away from the direction of the applied lateral moment. During right lateral exertions, compression forces were greater when the trunk was laterally flexed 15° to the left compared to upright exertions, and much greater than when flexed 15° to the right. Left lateral exertions resulted in compressive loads which were greatest when flexed 15° to the right, however, compressive loads were nearly as large as those produced by right lateral exertions when flexed 15° to the left.

Changes in spinal loading as a function of velocity can be attributed to a large part to changes in antagonistic coactivation. Coactivation was observed to increase significantly as the lateral velocity increased among the experimental conditions. Failure to incorporate coactivity resulted in much smaller estimates of spinal compression (Fig. 5).

DISCUSSION

This study hypothesized that increased lateral trunk velocity would involve increased levels of antagonistic coactivation in the trunk muscles, and thus, higher loads on the spine. These results have shown that as lateral trunk velocity increases the lateral shear force increases proportionally and more intensely than do the other spinal forces. Compression also increased monotonically as velocity increased. A-P shear increased in the nonzero velocity conditions compared to the static conditions but remained at relatively constant levels once movement was permitted in the exertion. Thus, we can speculate that combinations of lateral shear and compression is the biomechanical mechanism responsible for the increased risk associated with more rapid lateral trunk movements as observed in industrial studies (Marras et al., 1993, 1995).

Our goal in this study (and throughout the development of this model) was to implement a model that could account for differences in spine loading under static and dynamic movement conditions without making the model unnecessarily complex. Following this logic, the model included the major muscle groups that can be directly monitored using EMG. Monitoring of deep muscle groups might have predicted even further in-

creases in spinal loading. Our model did not include ligaments. However, we do not believe this was a serious limitation because we did not test subjects in positions beyond the active range of the muscles. McGill (1992) found 'ligament contributions were very small' in the lateral positions we tested. McGill (1992) has produced the only other model that has assessed loading during lateral velocity but did not investigate changes in velocity magnitude.

Our results compared favorably with those from other studies. The lateral torques exerted by our subjects were similar to those reported by McGill (1992). His subjects lifted a 200 N load which imposed approximately a 90 Nm torque about the spine as the subjects passed though a 15° lateral bending angle. In our study the heaviest weight condition required subjects to lift a 267 N load which resulted in a lateral torque of 116 Nm under the 45° s⁻¹ condition. This torque appeared relatively large compared to the maxima of 157 and 140 Nm reported by Parnianpour et al. (1988) and Kumar et al. (1995), respectively. However, Parnianpour et al. (1988) required subjects to exert torque against a dynamometer and Kumar tested subjects while seated. Both of these conditions would be expected to reduce significantly available strength compared to an upper body free dynamic lift as performed in our study.

Maximum compressive loads during the lateral exertions, 1900-2800 N, were similar to values predicted from analyses of dynamic lifting and twisting exertions (Freivalds et al., 1984; Granata and Marras, 1993, 1995; Jager and Luttman, 1989; Marras and Granata, 1995; Marras and Sommerich, 1991b). The only other study that assessed dynamic lateral spinal loads was the study of McGill (1992). Our results in the 15° s⁻¹ velocity condition were similar to McGill's data reported under conditions at 15° of lateral bend. McGill's predicted compression was 2400 N compared to ours of 2500 N. A-P shear was different (that of McGill was approximately 200 N compared to our value of 700 N). This difference may be explained by different testing procedures. We ensured that subjects produced pure lateral motions by guiding their motion. McGill's subjects were not guided. A difference also appeared in lateral shear. McGill's lateral shear was estimated to be approximately 60 N where ours was less than 20 N for the 15° s⁻ condition. However, differences in testing procedures would also easily explain this difference. McGill asked his subjects to pull on a cable attached to the floor which introduced a horizontal component to the resisted force. Our subjects lifted a free weight that did not contact the body, and thus, did not introduce a horizontal component of load. Taking these protocol differences into account, our predictions compare reasonably well with the more anatomically detailed model of McGill.

This study emphasizes the importance of models and assessments that are capable of considering coactivation of the trunk musculature when considering dynamic trends in spinal loading. McGill (1992) found large amounts of coactivation in lateral bending as did we. Our findings indicated that analyses which neglected antagonistic muscle activity predicted static spinal compression values that were 25% lower than the values determined from the fully coactive analyses. Similar trends were

observed for predicted A-P shear values. When the data were modeled without coactive antagonism, the results failed to show any influence of velocity upon the spinal loads, whereas, results using a fully coactive model demonstrated increasing spinal load with lateral velocity.

Coactivity levels of the trunk musculature in this study were greater than the magnitude of coactivity observed during lifting exertions (Granata and Marras, 1995b), but less than twisting (Marras and Granata, 1995) (Fig. 6). Right and left lateral exertions resulted in coactivity levels of 30 and 28%, respectively, of the most active muscle.

These results may help to explain how rapid lateral exertions might increase the risk of back injury. The construction of the vertebrae with its neural arch adds considerable resistance to forward shear; however, lateral shear strength which relies more on disc strength, is probably less than 900 N (Farfan, 1988). The literature is rich with studies indicating that disc strain increases greatly with lateral loading and with increases in combined plane loading (Broberg, 1983; Lin et al., 1978; Schultz et al., 1979; Shirazi-Adl, 1989, 1991; Shirazi-Adl and Drouin, 1987; Shirazi-Adl et al., 1986). Thus, even though the magnitude of the lateral spinal shear was low in this study, the rapid increase in shear force and the added influence of combined loadings is expected to challenge easily the lateral shear tolerance of the disc.

Several experimental limitations were inherent in this study. First, the lateral exertions were performed with the hips secured in a harness attached to the forceplate. Under realistic lifting conditions, this may alter the mechanics of spinal loading since the hips could rotate and thereby change the trunk muscle orientation with respect to the spine. In addition, the effects of coupled motions or loadings on the spine were not evaluated. These may indeed play a major role in the definition of low-back disorder risk.

In conclusion, this study has demonstrated that spine loading significantly increases with increased lateral velocity. Specifically, the combination of lateral shear and compression increase most dramatically with increasing velocity indicating an increase in the 'coupled' loading pattern on the spine. This loading is greatly influenced by

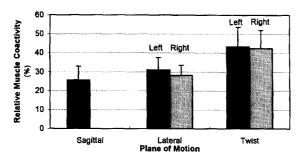


Fig. 6. Mean and standard deviation of coactivity (average activity relative to the prime mover) demonstrates measured coactivity during lateral exertions were slightly greater than the magnitude of observed during lifting exertions, but was less than twisting. The coactive value during right lateral exertions (30%) was similar to the magnitude during left lateral exertions (28%).

the coactivation of the trunk muscles. A 10 muscle EMG-assisted model was sufficient for evaluating these effects, yet emphasizes the need for a level of model complexity that considers the coactivation within the multiple muscle system in order to interpret complex trunk motions.

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