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Effects of different levels of torso coactivation on trunk muscular and kinematic responses to posteriorly applied sudden loads

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Abstract

Background. Studies examining rapid spine loading have documented the influence of steady-state trunk preloads, and the resulting levels of trunk muscle preactivation, on the control of spine stability. However, the effects of different levels of muscle coactivation, and resulting spine loads, on the response to a perturbation of the externally unloaded trunk are unclear.

Methods. Fourteen male subjects coactivated the abdominal muscles at four different levels (approximately 0%, 10%, 20% and 30% of the maximal voluntary contraction) monitored by an electromyography biofeedback system while semi-seated in a neutral lumbar spine position. They were loaded posteriorly in two directions (0° and 30° from the sagittal plane) and with two different loads (6.80 and 9.07 kg). Force perturbation, spine displacement and electromyography activity were measured, and torso compression and stability were modeled.

Findings. Abdominal coactivation significantly increased spine stability and reduced the movement of the lumbar spine after perturbation, but at the cost of increasing spinal compression. Preactivation also reduced the frequency and magnitude, and delayed the onset of muscle reactions, mainly for the back muscles and the internal oblique. The higher magnitude load and the load applied in an oblique direction both showed more potentially hazardous effects on the trunk.

Interpretation. Torso coactivation increases spinal stiffness and stability and reduces the necessity for sophisticated muscle responses to perturbation. Although further investigation is needed, it appears there is an asymptotic function between coactivation and both stiffness and stability. There also appears to be more hazard when buttressing twisting components of a sudden load compared to sagittal components. Patients with trunk instability and intolerance to spine compression may benefit from low to moderate levels of coactivation.

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Keywords: Cocontraction; Sudden loading; Spine stability; Electromyography; Spine compression

1. Introduction

The ligamentous spine is an unstable structure that buckles under small compressive loads (Crisco and Panjabi, 1992; Lucas and Bresler, 1961). Sudden external forces that occur during daily activities (slipping, falling, etc.) can compromise stability and alter the potential

* Corresponding author. *E-mail address:* mcgill@healthy.uwaterloo.ca (S.M. McGill). risk of injury to the spinal structures. Fortunately, active contractile properties of the torso muscles can mechanically stabilize the spine (Panjabi, 1992). The mechanical contribution of muscle stiffness, which increases through modulation of muscle activation and trunk muscle coactivation, to spine stability has been quantitatively documented (Cholewicki and McGill, 1996; Cholewicki et al., 1999; Gardner-Morse and Stokes, 1998, 2001). However, quantification of various levels of trunk muscle coactivation, stability and the muscle response to rapid perturbations is needed.

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In several sudden loading studies, the influence of different levels of trunk muscle coactivation resulting from isometric preloading on trunk stiffness, kinematics, and muscle responses have been observed. Generally, the results of these studies indicate that torso muscle preactivation increases trunk stiffness (Andersen et al., 2004; Essendrop et al., 2002; Gardner-Morse and Stokes, 2001) and reduces the subsequent torso displacement (Essendrop et al., 2002; Krajcarski et al., 1999; Stokes et al., 2000) and the reflex muscle response to rapid perturbation (Andersen et al., 2004; Granata et al., 2004; Krajcarski et al., 1999; Stokes et al., 2000). Moreover, anticipatory trunk muscle activation has been shown when rapid perturbations are expected (Brown et al., 2003; Thomas et al., 1998). A general conclusion is that the coactivation of the trunk muscles appears to be a good strategy to increase spine stiffness and stability before rapid spine loading.

In addition to increasing trunk stiffness and stability, cocontraction imposes increased spinal load which, at high levels, can be considered a risk factor for low back disorders (Gardner-Morse and Stokes, 1998; Granata and Marras, 2000; NIOSH, 1981) and may compromise a back pain patient who is intolerant of compression. If stability is achieved by means of muscular cocontraction but at the cost of increased spinal loads, the question arises as to what levels of muscle coactivation are required to achieve sufficient spine stability with the minimum compressive penalty? Cholewicki and McGill (1996) have shown that modest levels of torso coactivation in the neutral posture can be sufficient for ensuring spinal stability under pure compressive loading. Hence, patients with low back disorders could benefit from low to moderate levels of trunk coactivation without imposing great loads on the spinal column. On the other hand, higher levels of preactivation may be necessary to effectively stiffen the trunk to withstand higher loads, and in highly demanding plyometric sport activities to better store elastic energy. Thus, many variables need to be considered to understand the relationship between stability and issues of safety, performance, sensitivity to specific intolerances and injury mechanisms. We were motivated to obtain more insight into the optimal muscular preactivation mechanism as it pertains to the spine stabilization system to provide useful information to ultimately assist in prescribing trunk rehabilitation and training programs.

The purpose of this study was to document the effect of four levels of torso muscle coactivation on the trunk response to posterior sudden loading in a neutral lumbar spine position (neutral lordosis). An electromyography (EMG) biofeedback system was used to achieve the different initial levels of trunk muscle preactivation. Specifically the muscular preactivation and applied loads were quantified, the muscular responses and torso kinematic responses were measured, and torso compression and stability were modeled, to enable a discussion around the objective of better understanding links to spine stability. Also investigated was whether the responses depended on the direction and amplitude of the perturbation.

2. Methods

2.1. Participants

Fourteen male volunteers were recruited from the university population. None of the participants had experienced back pain in the past year. Subjects had a mean (SD) age of 28.14 (8.33) years, height of 1.78 (0.05) m, and mass of 77.78 (10.41) kg. Participants completed an informed consent form approved by the University Office for Research Ethics.

2.2. Instrumentation and data collection

2.2.1. EMG biofeedback

While maintaining the lumbar spine in a neutral position, participants were instructed to isometrically coactivate the abdominal muscles ("abdominal bracing") (Kavcic et al., 2004b) at four different levels. The Myo-Trac[™] (Thought Technology Ltd., Montreal, Canada) EMG Biofeedback System was used to control and monitor the intensity of the abdominal brace. An EMG sensor (MyoScan[™]) was placed over the lower region of the right external oblique using a disposable triode electrode (Ag–AgCl). The MyoTrac[™] offers an expanded range of visual feedback by means of a 13-segment LED bargraph. The central LED segment was calibrated as the desired target, shining when the EMG amplitude matched the pre-established value. The intensity and color of the light changed when it moved away from the target. Participants were instructed "to try to attain the EMG activation target and to maintain it". The target was programmed at 0%, 10%, 20% and 30% of the maximal voluntary isometric contraction (MVC) amplitude at the right external oblique site. The MVC amplitude was obtained in resisted maximal twist and bend efforts while restrained in a sit up posture.

2.2.2. Sudden loading

Participants were placed in a semi-seated position in a wooden apparatus that restricted hip motion while leaving the trunk free to move in all directions (Fig. 1). This has been shown to foster a neutral spine posture and elastic equilibrium for the hips and spine (Sutarno and McGill, 1995). Then, while resting (no brace) or bracing at 10%, 20% or 30% MVC, rapidly applied sudden loads were administered posteriorly to the participant via a steel cable attached to a harness (Fig. 1). The cable



Fig. 1. Experimental set-up for generating sudden posterior loading. The above right view shows two loading directions: 0° and 30° from sagittal plane.

was aligned approximately with the T7 level, and directed horizontally through a pulley and attached to the load, which was dropped from a height of 20 cm to load the cable. The cable load was delivered either in the sagittal direction (0°-sagittal condition) or in an oblique direction from the sagittal plane (30° -sagittal condition). At each direction, two different loads (6.80 and 9.07 kg) were applied by the investigators without warning, within a 15 s window. There were 16 test conditions (four preactivation levels, two loading directions and two loading amplitudes), all of which were presented randomly.

2.2.3. External force measures

The magnitude and timing of the force perturbation produced by dropping the load was measured using a load-cell force transducer located in-series between the cable and the harness. The force signals were amplified, and A/D converted (12 bit resolution over ± 10 V) at 1024 Hz.

2.2.4. Trunk kinematics

Lumbar spine kinematics were measured about three orthogonal axes (flexion–extension, lateral bend, and twist) using an electromagnetic tracking instrument (3Space ISOTRAK, Polhemus Inc., Colchester, VT, USA), sampled at a frequency of 32 Hz. This instrument consists of an electromagnetic transmitter and a small receiver. The transmitter was strapped to the pelvis over the sacrum and the receiver on the ribcage, over the T12 spinous process. Thus, the three-dimensional angular displacements of the ribcage relative to the sacrum were measured.

2.2.5. EMG recording

Surface electromyographic signals were collected bilaterally from the following trunk muscles and

locations: rectus abdominis (RA), approximately 3 cm lateral to the umbilicus; external oblique (EO), approximately 15 cm lateral to the umbilicus and just superior to the biofeedback sensor site; internal oblique (IO), halfway between the anterior superior iliac spine of the pelvis and the midline; latissimus dorsi (LD), lateral to T9 over the muscle belly; and erector spinae at T9, L3 and L5 (ET9, EL3, EL5, respectively), located approximately 5, 3 and 1 cm lateral to each spinous process. EMG from the sternal segment of right pectoralis major (RPM) was also recorded. Ag-AgCl surface electrodes were positioned with an interelectrode distance of 3 cm. The electromyographic recording was synchronized to the ISOTRAK and load-cell data with a common trigger. The EMG signals were amplified $(\pm 2.5 \text{ V})$, A/D converted (12 bit resolution) at 1024 Hz and full wave rectified.

For the modeling purposes to be described later, EMG was low pass filtered (second order single pass Butterworth) at 2.5 Hz, and normalized to MVC amplitudes. This process has been shown to link activation with force (Brereton and McGill, 1998). The MVCs were obtained in isometric maximal exertion tasks carried out prior to the sudden load trials. For the abdominal muscles, each participant was placed in a sit up position and manually braced by a research assistant. The participant produced a sequence of maximal isometric efforts in trunk flexion, right lateral bend, left lateral bend, right twist and left twist; little motion took place. For the extensor muscles, an isometric trunk extension was performed against resistance in the Biering-Sorensen position. The MVC for RPM was measured while participants were positioned supine on a slightly padded bench with the right shoulder in a flexed, abducted and externally rotated position with the elbow slightly bent. A research assistant resisted shoulder adduction, extension and internal rotation.

2.3. Data reduction

The onset of the force perturbation was detected from the load-cell signal by visually identifying when the force-time slope changed significantly; these load impacts were immediate and very clear (Fig. 2). Time windows of 200 ms before and 250 ms after the force perturbation were selected for subsequent analyses. EMG, force and kinematic signals were visually inspected. Trials with artifacts due to the contact of pads and harness with electrodes (mainly latissimus dorsi and erector spinae at L5 and L3 sites) and data resulting from other technical problems were excluded from further analyses. As a result, 11.1% of the EMG channels were eliminated. Similar problems were found by Radebold et al. (2000), Reeves et al. (2005) and Stokes et al. (2000).

2.3.1. External force and kinematics

The peak of the load-cell signal and the peak angular displacement of the lumbar spine (extension, bend and twist) in the 250 ms after sudden loading were recorded in every trial.

2.3.2. Preactivation and response magnitudes

For each muscle site, the average normalized EMG of the 50 ms before the perturbation was used to evaluate the amplitude of the muscle preactivation at each of the four preactivation levels (no brace, 10%, 20% and 30% MVC). Further, the peak EMG level achieved in the 250 ms post-loading was recorded. The ratio between the peak EMG response and average EMG preactivation (response/preactivation) was calculated in order to evaluate the relative magnitude of the muscle responses.

2.3.3. Frequency and timing of muscle response

A computer algorithm established by Radebold et al. (2000) was used to facilitate the detection of the onset of

EMG activity. According to this algorithm, a muscle response was considered to occur if, for at least a 25 ms duration, the rectified (un-filtered) EMG signal post-loading exceeded the sum of the mean plus 1.4 standard deviations of the EMG signal calculated over the 80 ms before the perturbation (threshold). Each muscle was processed separately and checked visually. The response latency was determined as the time between the start of the perturbation and the onset of muscle activation. The response frequency of each muscle for each preactivation level was calculated as the average of the dichotomous response scores (no response = 0; response = 1) from the trials of different loading directions and amplitudes (four trials).

2.3.4. Stability and compression

First, static whole-body postures were hand digitized from a single digital video image and entered into a fullbody linked segment model to determine the 3-D reaction forces and moments at the L4–L5 joint. Next, 14 channels of EMG and three-dimensional lumbar spine angles acquired from the 3Space were entered into an anatomically detailed spine model representing 118 muscle elements as well as lumped passive tissues, spanning the 6 lumbar joints (T12–L1 through L5–S1). Muscle stiffness and force were calculated as the first and second moments respectively of a Distribution Moment Model (as per Ma and Zahalak, 1991) representing the instantaneous number of attached cross-bridges in a given muscle, dependent on muscle cross-sectional area, activation, length and velocity.

To quantify spine stability, an 18×18 (six joints by three anatomical axes) Hessian matrix of the second partial derivatives of the potential energy of the entire lumbar spine system was calculated, and diagonalized to obtain its eigenvalues. The potential energy theory states that each eigenvalue of the matrix must be posi-



Fig. 2. Example of force signal (thick grey line) and full wave rectified EMG from right rectus abdominis (thin black line) for a sudden loading trial without preactivation. The horizontal dashed line represents the threshold (algorithm = mean + 1.4 SD of the 80 ms before loading) used for the detection of EMG onset.

tive definite in order for the system to be stable. Both the lowest eigenvalue and the stability index (an average of the 18 eigenvalues, Howarth et al., 2004) were therefore utilized as measures of spine stability.

L4–L5 compressive force and spine stability were analyzed at two time periods during each trial: (i) an average over the 50 ms prior to the sudden load; (ii) the peak compressive force and minimum stability in the 250 ms post-load.

Because of the difficulties in maintaining sufficiently clean data in all the instruments and EMG channels necessary to drive the current model, only 10 of the 14 subjects were utilized for the modeling analyses.

2.4. Statistical analysis

A two-way repeated measures analysis of variance was used to compare the EMG preload amplitudes between preactivation levels for each muscle and between muscles for each preactivation level. Differences between preactivation conditions for the lowest eigenvalue, stability index and compressive force variables before loading were assessed using one-way repeated measures analyses of variance.

Three-way repeated measures analyses of variance were performed to evaluate the influence of preactivation levels, load direction and load magnitude on the following dependent variables: peak force magnitude, peak angular displacement of the trunk (extension, twist and lateral bend), minimum lowest eigenvalue and stability index, and maximum compressive force after loading. Four-way repeated measures analyses of variance were performed to evaluate the influence of preactivation levels, load direction, load magnitude, and muscle on the response/preactivation ratios and onset latencies of muscle responses. Where applicable, post hoc analyses were carried out using Tukey's honestly significant difference test. A Friedman repeated measures analysis of variance was used to compare the frequency of response (averaged response scores). The significance for these contrasts was based on the Wilcoxon signed ranks test. Analyses were performed using a significance level of $\alpha = 0.05$ for all tests.

3. Results

3.1. Muscle preactivation

The four experimental preactivation levels, monitored by EMG biofeedback, resulted in the following actual EMG amplitudes (averaged for all the abdominal muscles): "no brace" = 1.9% MVC (SD 1.4%); "10% MVC" = 12.9% MVC (SD 7.1%); "20% MVC" = 21.4% MVC (SD 11.9%); "30% MVC" = 27.2% MVC (SD 15.0%). As shown in Fig. 3, greater preactivation levels resulted in greater EMG amplitudes for all muscles. Differences between the four preactivation levels were statistically significant only for external oblique which was the muscle monitored by biofeedback. While bracing, internal oblique reached the highest preactivation levels (approximately 25–50% MVC) followed by latissimus dorsi (10–35% MVC) and erector spinae at T9 (8–25% MVC).

3.2. Stability and compression

Preactivation of the trunk muscles significantly increased the stability and the compression of the spine before and after loading ($P \le 0.001$). For compression the differences between preactivation levels were always statistically significant. For the stability index, the differences were not significant between 20% and 30% MVC levels after loading. For the lowest eigenvalue, differences were significant between no brace and the bracing levels as well as between 10% and 30% MVC levels. Also note that potential unstable behavior (lowest eigenvalue ≤ zero) occurred post-perturbation in nine subjects for the no brace condition, and in four subjects in each of the 10%, 20% and 30% MVC conditions. The smaller applied load (6.80 vs. 9.07 kg) resulted in higher lowest eigenvalues (higher stability) (P = 0.021) and lower peak compressive forces (P = 0.031) postloading. The interaction between load, direction and preactivation was not significant. As a result, in Fig. 4 we present the data averaged across conditions.

3.3. External force

When averaged for all conditions, the peak magnitude of the applied force recorded in the load-cell was 257.5 N (SD 50.1 N). The force magnitude was higher in the 0°-sagittal condition than the 30°-sagittal condition [321.7 N (SD 55.3 N) vs. 193.2 N (SD 44.9 N) respectively; P < 0.001]. The heavier load resulted in greater peak forces as compared to the lighter load [9.07 kg = 277.3 N (SD 60.3 N) vs. 6.80 kg = 237.7 N (SD 40.0 N); P < 0.001]. Preactivation significantly augmented the peak force magnitude (P < 0.001) (Fig. 5). From no preactivation to 10% MVC, the peak force increased 14.0% (P < 0.001) while from 10% to 20% MVC it increased 6.6% (P = 0.018). However, differences between 20% and 30% MVC were not significant (1.1%) (Fig. 5).

3.4. Trunk kinematics

Preactivation and direction significantly affected the spine movement (P < 0.001) in response to the sudden load. More bracing stiffened the spine resulting in less post-perturbation motion. Their interaction was also



Fig. 3. Averages and standard deviations of the normalized EMG amplitudes before the perturbation for the right side muscles and four preactivation levels (no brace, 10%, 20% and 30% MVC). While the external oblique muscle was monitored for subject feedback, the internal oblique channel responded with higher activation levels and rectus abdominis with lower than target levels of activation. Results of the multiple comparison (P < 0.05): *significantly higher than no brace; **significantly higher than no brace and 10% MVC; ***significantly higher than no brace and 10% and 20% MVC.

significant (P < 0.001). However, the magnitude of the load (6.80 vs. 9.07 kg) did not significantly influence lumbar spine movement. Thus, in Fig. 6, we present the kinematics data averaged over both loading amplitudes.

Sudden loading in the 0°-sagittal condition resulted primarily in trunk extension. Increasing the preactivation level reduced lumbar spine extension with decreases being significant between each of 0%, 10% and 20% MVC (reduction from no brace to 20% MVC: 41.1%, P < 0.001). Differences between the 20% and 30% MVC preactivation levels did not reach statistical significance. Sudden loading in the 30°-sagittal condition resulted in a combination of extension, twist and lateral bend. Preactivation significantly reduced the lumbar extension and lateral bend movements from no brace to 20% MVC (50.9% and 54.3% respectively, P < 0.001); differences between levels 20% and 30% MVC failed to achieve statistical significance. For twisting, the differences between preactivation levels were always significant (reduction from no brace to 30% MVC: 38.8%, P < 0.01).

3.5. Muscular response

Posterior loading while semi-seated with a neutral spine position activated mainly the anterior musculature of the trunk. For the no brace condition, the mean fre-

BEFORE LOADING (50 ms)

AFTER LOADING (250 ms)



Fig. 4. Averages and standard deviations of the lowest eigenvalue, the stability index and the compression force for four preactivation levels (no brace, 10%, 20% and 30% MVC). This was examined both as the average over the 50 ms preload, and as the peak compressive force and minimum lowest eigenvalue and stability index post-load. More preactivation stiffens and stabilizes the spine at the cost of higher spine compressive load. Results of the multiple comparison (P < 0.05): *significantly higher than no brace; **significantly higher than no brace and 10% MVC; ***significantly higher than no brace and 10% MVC; Note that possible instability occurred after loading with no brace as indicated with the negative value.



Fig. 5. Averages and standard deviations of the peak force magnitudes during the perturbation for two loading directions, two loading amplitudes, and four preactivation levels. The increasing coactivation levels stiffened the torso, reducing the trunk "shock absorption", and caused higher peak cable tensions to develop.

quency of response for abdominals (RA = 100%, EO = 100%, IO = 98.1%) and pectoralis major (100%)

was significantly higher than for erector spinae sites (ET9 = 79.5%, EL3 = 78.1%, EL5 = 63.0%); the onset



Fig. 6. Averages and standard deviations of the peak amplitudes of the lumbar spine extension, lateral bend and twist displacements after the perturbation for both loading directions and four preactivation levels. A similar phenomenon is observed here as was observed in Fig. 4, the stiffened torso caused less spinal motion upon perturbation.

of the response latencies of pectoralis major (45–55 ms), abdominal muscles (RA = 55–68 ms, EO = 55–62 ms, IO = 55–76 ms), and latissimus dorsi (45–70 ms), were shorter than the latencies of the erector spinae muscles (ET9 = 98–104 ms, EL3 = 81–111 ms, EL5 = 94– 110 ms) (Fig. 7); and the abdominal response/preactivation ratios were higher than those of the back muscles and pectoralis major (Fig. 8).

When preactivated, a high reduction in the frequency of muscle responses was observed for the back muscles and internal oblique (reduction from no brace to 30% MVC: LD = 65.3%, ET9 = 57.2%, EL3 = 71.3%, EL5 = 52.3%, IO = 65.6%). It should be noted that the decrease in the frequency of responses between 20% and 30% MVC conditions was not significant for the majority of the back muscles. On the other hand, rectus abdominis demonstrated a non-significant reduction in the response frequency (10.1%), and external oblique and pectoralis major a moderate reduction (EO = 24.0\%, PM = 22.5\%), significant only for the 30% MVC condition.

The response latencies were higher in the 30°-sagittal condition than the 0°-sagittal condition [93.3 ms (SD 35.8 ms) vs. 81.7 ms (SD 33.5 ms) respectively; P = 0.041]. The load amplitude did not affect the onset latencies. The effect of preactivation was different depending on the direction of the load application and the muscle (P = 0.022) (Fig. 7). In the 30°-sagittal condition, onset latencies of internal oblique, external oblique, latissimus dorsi and left erector spinae at T9 were in general greater for the bracing levels when compared to no brace. A similar trend was found for the 0°-sagittal condition; however, increases were statistically significant only for left latissimus dorsi and left erector spinae at T9.

For the response/preactivation ratios, the interactions between preactivation, direction and type of muscle were significant (P < 0.01). On the contrary, the load magnitude did not have any influence on the ratios. As shown in Fig. 8, the preactivation significantly reduced the response/preactivation ratios when comparing no brace with the bracing levels, but it did not when comparing between the bracing levels (with the exception of right erector spinae at L3 in the 30°-sagittal condition).

4. Discussion

Sudden load paradigms are usually designed to examine the influence of preparatory muscle recruitment on the control of spine stability. Generally, several steadystate preloads are used to achieve different initial levels of trunk muscle activity prior to the superimposed force perturbation. Nevertheless, to our knowledge, the effects of several levels of muscle coactivation on the response of the externally unloaded trunk had not been previously investigated. Using EMG biofeedback participants were able to differentiate four levels of abdominal coactivation prior to the sudden application of the posteriorly directed perturbation. In order to keep the spine in a neutral lumbar position while activating the abdominal muscles, the flexion torque was counteracted by the activation of the back muscles (see Fig. 3). Because of the high level of trunk cocontraction, it was a challenge for some subjects to reach the maximum amount of abdominal preactivation used in this study (EMG-target of 30% MVC). Since the coactivation of all trunk muscles increased the stability of the spine and reduced lumbar displacement after loading (see Figs. 4 and 6), all the torso muscles seem play an important role in securing spinal stability, and must work harmoniously to reach this goal. This is consistent with other datasets which demonstrate that all muscles are



Fig. 7. Averages and standard deviations of the onset latencies of muscle responses after sudden loading. (A) 0°-sagittal condition; (B) 30°-sagittal condition. The preactivation affects the timing of the muscle responses, mainly in the 30°-sagittal condition. For most muscles, onset latencies were longer for preactivation levels of 10%, 20% and 30% MVC than no brace. Results of the multiple comparison ($P \le 0.05$): *significantly higher than no brace; [†]significantly higher than 10% and 20% MVC; [‡]significantly higher than 30% MVC.

essential for maintaining spine stability (Kavcic et al., 2004a). Interestingly enough, although the investigators placed the biofeedback sensor on the external oblique, the muscle that reached the greatest level of relative activity (% MVC) in this particular task was the internal oblique. Further, when abdominal coactivation patterns were individually examined, all subjects but two activated primarily the internal oblique, followed by the external oblique and rectus abdominis. This may indicate that internal oblique plays a vital role in generating abdominal bracing maneuvers. However, cross-talk in the biofeedback signal from the internal oblique activation or an underestimation of the amplitudes used to normalize the EMG signal of this muscle could influence these results. Finally, in our other studies we have observed that once moments in the twist or lateral bend axes are introduced, the external oblique becomes the most responsive muscle (Axler and McGill, 1997; McGill, 1991).

Muscle cocontraction, monitored through the EMG biofeedback, increased the stability of the trunk and reduced the movement of the lumbar spine, but at the cost of increasing spinal compression (see Fig. 4). The results of this study are in general agreement with the literature. Previous sudden load studies found that trunk preloading, and the resulting muscle preactivation, increased the torso stiffness (Andersen et al., 2004; Cholewicki et al., 2000; Gardner-Morse and Stokes, 2001), and reduced the trunk displacement after loading (Krajcarski et al., 1999; Stokes et al., 2000). The effect of cocontraction on compressive spine forces has also been documented (Gardner-Morse and Stokes, 1998; Granata and Marras, 2000). Granata and Marras (2000) evaluated the cost-benefit relationship of muscle



Fig. 8. Averages and standard deviations of the EMG response/preactivation ratios for four preactivation levels. (A) 0°-sagittal condition; (B) 30°-sagittal condition. The trunk preactivation increased the torso stiffness, thereby reducing the need for high muscle responses in order to stabilize the spine after sudden loading. Results of the multiple comparison (P < 0.05): *significantly higher than 10%, 20% and 30% MVC; [†]significantly higher than 20% and 30% MVC; [†]significantly higher than 30% MVC.

cocontraction in protecting against spinal instability during lifting activities. They found that the margin between stability and spinal compression increased significantly with cocontraction, mainly at low trunk moments. However, increasing stability through modulation of muscle coactivation may aggravate the low back pain of patients who are intolerant of compression and is a factor of risk for spine injuries (NIOSH, 1981). Therefore, a balance between stability and compression is needed. Theoretically, the optimal level of trunk stiffness would be the minimal level that assures spinal stability without imposing unnecessary loads on the tissues (Cholewicki and McGill, 1996; McGill et al., 2003). On the basis of the results in this investigation, we cannot determine an optimal level of stiffness, however, there appears to be an asymptotic function of stiffness and stability, where these measures level off with continuously increasing muscular activity.

As determined by the load-cell values, from 0% to 20% MVC bracing levels, tension of the cable pulley

during the load application increased in conditions with higher preactivation (see Fig. 5). Most likely, the increase of trunk muscle cocontraction enhanced the torso stiffness and rigidity, and provided the cable with a more stable and rigid surface of anchorage. Consequently the applied cable forces were higher with higher cocontraction levels. In contrast, increasing the abdominal preactivation from 20% to 30% MVC did not further increase the tension of the cable. Furthermore, although preactivation reduced the extension, bend and twist displacement of the lumbar spine after loading, the differences between 20% and 30% MVC conditions did not reach statistical significance for extension and lateral bend. These results could suggest that these preactivation levels generated similar amounts of trunk stiffness and stability. In a similar fashion, Sinkjaer et al. (1988) examined the muscle stiffness of the ankle dorsiflexors at different levels of voluntary contraction, and they found that the stiffness remained approximately constant from 30% to 80% of MVC. The results

of the current study indicate that a comparable trunk stiffness asymptote could occur at approximately 20-30% of MVC for the abdominal muscles.

As we have shown, preparatory trunk muscle recruitment is a fundamental mechanism of spinal stabilization. However, the stability of the spine also relies on the neuromuscular response to loading (Granata et al., 2001). Previous studies have shown that when the level of torso stability is not sufficient for a given external perturbation, additional muscular reflex adjustment may be necessary (Andersen et al., 2004; Granata et al., 2004; Krajcarski et al., 1999; Stokes et al., 2000). In the current study, all the trunk muscles reacted to the perturbation. For the no brace condition, the onset response latencies of more than 99% of the detected responses happened within the 150 ms following the perturbation. We believe that these latencies are indicative of reflex or automatic behavior rather than voluntary response. The neural pathways for controlling a trunk muscle's response to loading are not clearly understood. In this study, the perturbation posteriorly displaced the trunk and shoulder, and most likely triggered polysynaptic spinal stretch reflexes or automatic postural adjustments mediated via brainstem pathways. The onset latencies observed in abdominal muscles and pectoralis major (about 45–80 ms) are similar to those found in the erector muscles after anterior loading (Andersen et al., 2004; Cholewicki et al., 2000; Radebold et al., 2000). On the contrary, Granata et al. (2004) reported shorter latencies (about 30 ms) after impulse loading. The origin of this difference could be, in part, the computer-based method used for latency determination (Hodges and Bui, 1996). Granata et al. (2004) utilized filtered and normalized EMG and a threshold value of 2 SD beyond the mean of baseline activity. In contrast, this study utilized full wave rectified EMG and a threshold of 1.4 SD beyond the mean of baseline activity.

The frequency, magnitude and timing of the response of each muscle depended on the level of preactivation. For the no brace condition, the abdominal muscles and pectoralis major showed higher response frequencies, greater response/preactivation ratios and shorter onset latencies than the erector muscles. When the trunk muscles were preactivated, a reduction of the frequency of detected responses and the response/preactivation EMG ratios, and an increase of the response latencies (mainly in the 30°-sagittal condition) were observed. Likely, muscular preactivation stabilized the spine, reducing the necessity for a sophisticated muscle response to the rapidly applied posterior loads. Low back pain patients have been shown to have delayed response latencies in flexor and extensor trunk muscles in comparison with healthy controls (Radebold et al., 2000, 2001; Reeves et al., 2005), and these delays can occur in a variety of muscles. Thus, patients with poor muscular responses to perturbations could obtain some additional stiffness from a robust brace involving all trunk muscles during loading situations for safety. Interestingly, although a large decrease in the frequency of reflex responses was observed for the internal oblique, a small to moderate reduction was found in rectus abdominis, external oblique and pectoralis major. The differences between these muscles may be explained by the levels of muscle preactivation, significantly higher for internal oblique.

The outcomes of the studies examining the effects of preactivation on the onset of muscular response to perturbation are not consistent. Granata et al. (2004) found that preactivation lead to an increase in onset latencies, supporting the results of our study. However, Andersen et al. (2004) and Stokes et al. (2000) did not report any preactivation affect on the timing of muscle responses. The reason for this lack of consistency between the different studies may be the method used to evaluate the timing of the muscle response, as previously noted. On the other hand, a high variability of the onset latencies was observed. The reasons for latency variability could be diverse: (1) different upper body weights, resulting in inertial differences between subjects (Reeves et al., 2005); (2) variety of recruitment patterns to achieve the same kinematic response (Reeves et al., 2005); (3) differences in existing EMG "backgrounds" between subjects and preactivation levels (more "background" leading to longer latencies) (Hodges and Bui, 1996); and (4) different number of detected responses between preactivation levels. Unlike our study, three trials of every condition have often been used in previous investigations. However, it was determined that repeated trials were not possible in the current study because such an addition to the current number of trials would most likely have induced participant fatigue and/ or educated the participant to "expect" the load. It is quite probable that if we had been able to perform repeated trials and take the average of multiple repetitions for data analysis, the variability would have been smaller.

Finally, in order to gain a wider understanding of the trunk response to sudden loading, the characteristics of the load must be taken into consideration. In this study, the magnitude (6.80 vs. 9.07 kg) and direction $(0^{\circ} \text{ vs. } 30^{\circ} \text{ from the sagittal plane})$ of the applied perturbation had a strong influence on the results. Assuming that the stiffness of the harness acted in the same way in both loading directions, these results would indicate that the stiffness produced by trunk muscle preactivation and the muscular responses to the perturbation may be less effective for spine stabilization when loads were applied in an oblique direction. If this interpretation is correct, patients with low back instability may be more sensitive to loading in a twisting direction, or at least find it more difficult to maintain stability under twisting torque.

5. Conclusions

The current study investigated the effect of four levels of muscle coactivation, monitored through an EMG biofeedback system, on the trunk responses to posteriorly applied sudden loads in a neutral lumbar spine position. The following conclusions were reached: (1) Trunk muscle coactivation increased the stability of the trunk and reduced the movement of the lumbar spine, but at the cost of increasing spinal compression. Although further investigation is needed, the function linking coactivation with stiffness and stability appears to be asymptotic. (2) All muscles reacted to the rapid posterior loading, but differently. The anterior muscles of the trunk, principally rectus abdominis and pectoralis major, showed higher response frequencies and amplitudes, and shorter onset latencies. (3) Preactivation reduced the frequency and magnitude, and increased the onset latency of muscle reactions, mainly for the back muscles and the internal oblique. (4) The highest loads and the loads applied in an oblique direction demonstrated more potentially hazardous effects on the trunk (i.e. the twist axis seems to be the least buttressed against sudden loading). Overall, this study provides insight into the relationship between muscle coactivation, stability and muscular responses to rapid perturbations.

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