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The intrinsic stiffness of the in vivo lumbar spine in response to quick releases: Implications for reflexive requirements

Stephen H.M. Brown *, Stuart M. McGill

Department of Kinesiology, University of Waterloo, 200 University Ave West, Waterloo, ON, Canada N2L 3G1

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Abstract

Torso muscles contribute both intrinsic and reflexive stiffness to the spine; recent modeling studies indicate that intrinsic stiffness alone is sometimes insufficient to maintain stability in dynamic situations. The purpose of this study was to experimentally test this idea by limiting muscular reflexive responses to sudden trunk perturbations. Nine healthy males lay on a near-frictionless apparatus and were subjected to quick trunk releases from the neutral position into flexion or right-side lateral bend. Different magnitudes of moment release were accomplished by having participants contract their musculature to create a range of moment levels. EMG was recorded from 12 torso muscles and three-dimensional lumbar spine rotations were monitored. A second-order linear model of the trunk was employed to estimate trunk stiffness and damping during each quick release. Participants displayed very limited reflex responses to the quick load release paradigms, and consequently underwent substantial trunk displacements (>50% flexion range of motion and >70% lateral bend range of motion in the maximum moment trials). Trunk stiffness increased significantly with significant increases in muscle activation, but was still unable to prevent the largest trunk displacements in the absence of reflexes. Thus, it was concluded that the intrinsic stiffness of the trunk was insufficient to adequately prevent the spine from undergoing potentially harmful rotational displacements. Voluntary muscular responses were more apparent than reflexive responses, but occurred too late and of too low magnitude to sufficiently make up for the limited reflexes.

Keywords: Spine; Reflex; Muscle; Abdominals; Trunk stiffness

1. Introduction

The study of spine stability has advanced from limitations of static analyses of the instantaneous potential energy state of the muscularly supported vertebral column, to more thoughtful and probing analyses of the continually changing trade-off between loading states and stored energy, or compliancy and stiffness. A stiff system will usually be quite stable, with the trade-off of high joint compressive loads, whereas a compliant system will present an inherently greater opportunity for instability but experience less load. Performing dynamic activities requires consideration of both mobility and stability, and often requires individuals to adopt muscular patterns that may not, in themselves, lend much of a margin of safety in terms of preventing "spine buckling" type injuries. In these instances, the ability for reflexes to respond appropriately appears essential to adapt to unexpected changes in the environment.

Muscular reflexes are thought to be modulated or gained to pre-existing levels of activation, so as activation increases, the reflexive response increases to maintain a fairly consistent relation (Matthews, 1986). This can be confounded, however, by the current state of the system, presumably to optimally select the strategy to best serve the needs of the system. For example, reflexes can be either inhibited (Gottlieb and Agarwal, 1979; Stein et al., 2007) or facilitated (Nielsen et al., 1994; Akazawa et al., 1983) by the presence of activity in antagonist muscle groups. In addition, reflex

* Corresponding author. Tel.: +1 519 888 4567x36791; fax: +1 519 746 6776.
E-mail address: shmbrown@uwaterloo.ca (S.H.M. Brown).

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contribution to joint stiffness has been documented by different research groups to either increase (Carter et al., 1990; Zhang and Rymer, 1997) or decrease (Toft et al., 1991; Mirbagheri et al., 2000) with increasing moment demands on the system. The contradictory findings of these studies highlight the potential dependence of reflexes on the ability of the intrinsic stiffness alone to adequately respond to situational perturbations, as well as limiting large reflex gains from preventing oscillations in the system. Further, research has demonstrated situations in which torso muscles, opposing the recovery from a perturbation, actually reflexively increase activation in response to the perturbation (Krajcarski et al., 1999; Gregory et al., 2008; Thomas et al., 1998; Stokes et al., 2000), presumably to rapidly increase stiffness of the spine. It has also been recently hypothesized that the motor control system will sometimes reflexively respond to exacerbate a perturbation, providing that it assists an already planned voluntary movement response (Hasan, 2005). All of this research suggests that muscular reflexes play a role in stabilizing and stiffening the spine, but to what extent these are essential, or potentially complimentary, to limiting trunk displacements, and how an inhibition to isolate inherent stiffness can affect system stability, still requires further experimental research.

Properly functioning reflexes play a fundamental role in maintaining the integrity of spinal tissues in a dynamically changing environment. Repeated links have been made to delayed reflexes in numerous muscles in individuals experiencing back pain or disorders (e.g. Hodges and Richardson, 1998; Radebold et al., 2000; Reeves et al., 2005; Thomas et al., 2007), or, potentially more importantly, those at an increased risk of developing back injury (Cholewicki et al., 2005). More recently, muscular reflexes were suggested to account for approximately 42% of trunk stiffness necessary to stabilize the spine in a dynamically loaded state (Moorhouse and Granata, 2007), and it has been predicted that the spine, even supported by substantial levels of muscular activation and corresponding stiffness, could not be adequately stiffened to resist small ($\pm$2 mm translation and 1° rotation) externally applied torso perturbations in the absence of reflex responses (Franklin and Granata, 2007). Therefore, based on modeling analyses, it appears that intrinsic trunk stiffness cannot adequately stiffen the spine to prevent substantial trunk displacements in response to dynamic perturbations; however, this needs to be further tested experimentally. Thus, the current study was designed to examine the effect of increasing co-activation of the trunk on its dynamic stiffness response to perturbation, while limiting both reflexive responses of the musculature and the inherent passive stiffness of the spinal joints. This was accomplished by applying trunk perturbations to participants lying both on their backs and right sides; pilot work indicated that participants would be much less likely to reflexively respond to the perturbations in these positions. Further, the removal of the gravity vector acted to minimize the inherent spinal joint compressive stiffness. By generating trunk perturbations similar to those in the previously mentioned studies, with the added effects of inhibiting reflexive responses and innate joint passive stiffness, the role of both intrinsic and reflex muscular stiffness components on overall trunk stiffness was elucidated.

2. Methods

2.1. Participants

Nine healthy male individuals volunteered from the University population (mean/SD: age 23.9/2.8 years; height 1.81/0.05 m; 79.0/7.1 kg). All signed consent forms approved by the University Office of Research Ethics.

2.2. Data collection

Participants were secured at the hips, knees and ankles on a solid lower body platform. Each participant's upper body was secured to a cradle with a plexi-glass bottom surface, about their upper arms, torso and shoulders. The upper body cradle was free to glide overtop of a similar plexi-glass surface with precision nylon balls between the two structures. This jig eliminates measurable friction and allows lumbar trunk movement about either the flexion–extension or lateral bend axis, depending upon how the participant is secured. Participants lay on their right-side for the flexion trials and on their back for the lateral bend trials. Each participant's torso was supported in both positions to ensure that his adopted and maintained spine posture did not deviate throughout the testing.

Participants began each trial in their position of neutral elastic equilibrium (no applied external forces acting about the axis of interest). They were then instructed to generate either a flexor or right-side lateral bend moment to one of three distinct target activation levels as monitored from biofeedback of their right external oblique muscle site. The target levels were set at 5%, 10%, and 15% of maximum isometric activation (termed light, moderate, and heavy, respectively, for the remainder of the paper). The 15% level corresponded to the maximum activation that they were able to achieve during an isometric abdominal brace contraction (producing no external moment) in the test position. Six of the nine participants also performed a fourth target activation of the maximum flexor or lateral bend moment that they could produce in the test position (termed maximum). The internally generated moments were resisted (so as to keep the participant in their neutral position) by the experimenter via a cable instrumented with a force transducer. The line of pull of the cable was maintained perpendicular to the upper body cradle at all times, necessary to maintain the consistency of the resistive moment that opposed torso motion. Once the target activation was achieved and held steadily for a period ranging between one to three seconds, the cable was rapidly released via a latch mechanism, thus causing a rotational perturbation of the participants' trunk in either the flexion or right-side lateral bend direction (Fig. 1). Participants were instructed to react in a natural manner (i.e. without any conscious or pre-conceived plan) to the perturbation. Participants performed two trials of each condition.

2.3. Instrumentation

Twelve channels of EMG were collected from the following muscles bilaterally: rectus abdominis (RA), external oblique (EO), internal oblique (IO), latissimus dorsi (LD), and two levels of the

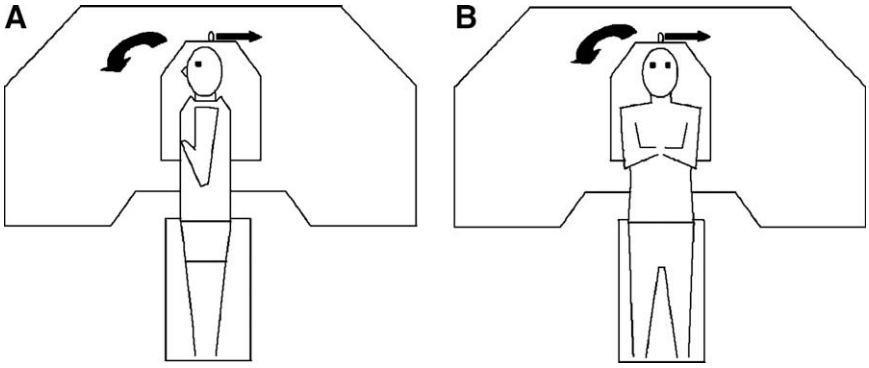

Fig. 1. Experimental set-up for the conditions in flexion (A) and lateral bend (B). Bold straight arrow indicate the direction of the applied force, bold curved arrows indicated the direction of rotational trunk displacement post-release.

erector spinae (EST9, ESL3). Blue Sensor bi-polar Ag–AgCl electrodes (Ambu A/S, Denmark, inter-electrode distance of 2.5 cm) were placed over the muscle belly of each muscle in line with the direction of muscle fibres. Signals were amplified (±2.5 V; AMT-8, Bortec, Calgary, Canada; bandwidth 10–1000 Hz, CMRR = 115 db at 60 Hz, input impedance = 10 GΩ) captured digitally at 2048 Hz, low-pass filtered at 500 Hz, rectified and low-pass filtered at 2.5 Hz (single pass 2nd order) and normalized to the maximum voltage produced during isometric maximum voluntary contraction (MVC) trials to produce a linear envelope.

An EMG biofeedback device (MyoTrac, Thought Technology Ltd., Montreal, Canada) was placed in line with the right EO electrode site to allow participants to visually monitor muscle activity at this level.

Three-dimensional trunk motion was recorded using an electromagnetic tracking system (Isotrak, Polhemus, Colchester, VT, USA) with the source secured over the sacrum and the sensor over T12 for the flexion/extension trials, and the source over the lower abdomen at a level slightly below the ASIS and the sensor over the xiphoid process for the lateral bend trials. The trunk motion data was sampled digitally at 32 Hz and dual-pass filtered (effective 4th order 3 Hz low-pass Butterworth).

The torso moments were calculated by the product of the force applied perpendicular to the distal end of the upper body cradle and the moment arm from the location of the applied force to the level of L4/L5. Force was recorded with a force transducer (Transducer Techniques Inc., Temecula, CA, USA) and digitally sampled at 2048 Hz. Force signals were dual-pass filtered (effective 4th order 3 Hz low-pass Butterworth). Both the linear enveloped EMG and force signals were downsampled to 32 Hz to match the trunk motion data.

2.4. Model description

A second-order linear model of the trunk was used to model the rotational motion of the trunk post-perturbation. The form of the model was as follows:

$$I\ddot{\theta} + B\dot{\theta} + K(\theta - \theta_0) = 0, \quad (1)$$

where I = moment of inertia of the upper body and cradle (kg m^2),
B = trunk rotational damping (Nm s/rad),
K = trunk rotational stiffness (Nm/rad),
Θ_0 = trunk angle offset (release angle of the trunk in the plane of interest),
Θ = trunk rotational displacement.

The length of post-perturbation data analyzed in order to obtain trunk characteristics of K, B, and θ_0 was taken from the time of quick release to the time of maximum trunk deflection (Cholewicki et al., 2000). In the current study this time averaged 1150 ms (range 780–1700 ms). Pilot work found this model to produce very good matches to the experimentally measured trunk deflections, thereby indicating that for the conditions studied here the assumption of a linear time invariant model was adequate.

The upper body moment of inertia was calculated for each participant via anthropometrics (Winter, 2004). The moment of inertia of the upper body cradle was calculated via the pendulum method (Dowling et al., 2007). An optimization algorithm was utilized to solve for the three equation unknowns by minimizing the root-mean-square difference between the measured and modeled trunk angular displacements.

2.5. EMG onset and offset latencies

Muscle latencies were calculated by rectifying and low-pass filtering (dual-pass effective 4th order 50 Hz Butterworth) each individual EMG channel. A muscle was considered to respond with an onset at the time when the signal crossed the threshold of the mean plus three standard deviations of the signal pre-perturbation baseline (calculated over the 50 ms prior to the perturbation) and was maintained for at least 20 ms (Hodges and Bui, 1996; Gregory et al., 2008). A muscle offset was determined by analyzing the signal in reverse time order (from time 1 s to time zero), and was considered to occur if the signal crossed the threshold of the mean plus three standard deviations of the signal post-perturbation baseline (calculated over the 50 ms from 950 to 1000 ms post-perturbation) and maintained for at least 20 ms.

Muscle latencies were analyzed between 20 and 1000 ms post-release. If a latency occurred between 20 and 150 ms it was considered reflexive in nature (Cholewicki et al., 2005) and between 150 and 1000 ms voluntary in nature.

Probability of onset was calculated as the percentage of muscles acting in opposition to the originally generated internal moment that turned on in response to the perturbation; probability of offset was calculated as the percentage of muscles acting to generate the original internal moment that turned off in response to the perturbation. For example, in the flexor moment

trials, the six back muscles opposed the generated internal moment and thus would be expected to turn on in response to the trunk flexion displacement while the six abdominal muscles generated the original internal moment and thus would be expected to turn off in response to the perturbation.

2.6. Statistics

Repeated Measures 2-way (movement direction and contraction level) ANOVAs were performed on both the rotational trunk stiffness and damping. Repeated Measures 1-way (contraction level) ANOVAs were performed on the applied moments, as well as the pre-perturbation activation levels and EMG latency probabilities for all muscles in each movement direction. Finally, Repeated Measures 1-way (on versus off) ANOVAs were run on the likelihood of muscle onset and offset for time periods of 150 ms and 1000 ms post-perturbation for each movement direction. Tukey's HSD post-hoc analyses were utilized to test for differences when alpha levels were determined significant ($p < 0.05$).

3. Results

3.1. Stiffness and damping

Table 1 displays the average and standard deviations of the root-mean-square differences between the model predicted and experimentally determined trunk rotational displacements, calculated as a percentage of the actual experimental displacements. The modeling analysis fit the experimental data quite well, with average model predicted trunk rotational displacements never exceeding an error of 4.2% of the true experimentally calculated displacements (Table 1; Fig. 2).

There was a significant effect of movement direction on rotational trunk stiffness ($p < 0.0001$; LB > Flex) (Fig. 3). Also, for both flexion and lateral bend trials stiffness was higher in the maximum contraction condition as compared to each of the light, moderate and heavy contraction conditions ($p < 0.0001$) (see Fig. 3).

There was also a significant effect of movement direction on rotational trunk damping ($p = 0.0019$; Flex > LB) (Fig. 4).

3.2. Applied moment and EMG pre-perturbation activation levels

In flexion, the applied moments were significantly different ($p < 0.0001$) between the maximum (mean/SD = 46.9/21.1 Nm) and each of the light (14.4/11.3 Nm), moderate (19.6/16.1) and heavy (22.5/19.5 Nm) contraction levels. Similarly, the pre-perturbation activation level was significantly different in the maximum as compared to each of the light, moderate, and heavy contractions for all muscles except the ESL3 where the maximum was different from only each of the light and moderate contractions. In addition, the IO muscle activation was significantly different in the light as compared to the heavy contraction condition (Fig. 5).

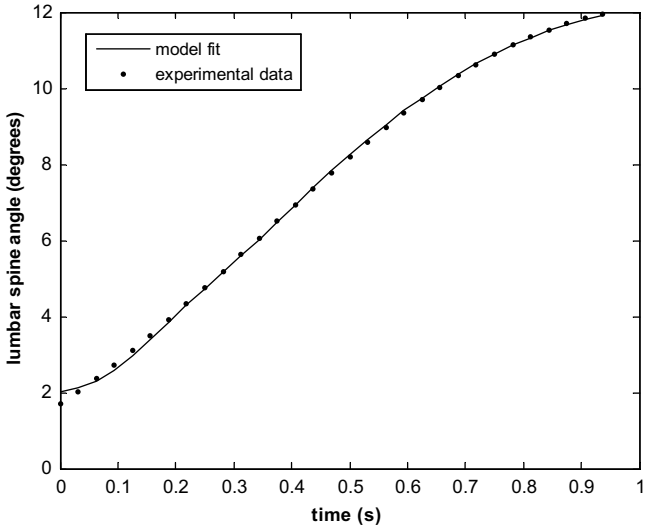

Fig. 2. Example of the model predicted and experimentally determined lumbar spine rotational displacement for a heavy flexor moment contraction trial. Model parameters for this trial: stiffness = 141 Nm/rad; damping = 11 Nm s/rad; percent RMS error = 0.78%.

In lateral bend, the applied moments were significantly different ($p < 0.0001$) between the maximum (mean/SD = 53.0/10.7 Nm) and each of the light (19.0/13.6 Nm), moderate (22.2/13.8) and heavy (27.9/18.7 Nm) contraction levels. Again similarly, pre-perturbation activation level was significantly different in the maximum as compared to each of the light, moderate, and heavy contractions for all left side muscles, as well as for the right RA, right EO and right IO muscles (Fig. 5).

3.3. EMG latency probabilities

In the flexion trials, the only muscle to display differences in the probability of post-perturbation onset was the right EST9 muscle ($p = 0.0388$), which displayed an increased likelihood of onset in the light (27.8%) as compared to the heavy (0%) contraction condition. EMG

Table 1
Mean and standard deviation (SD) of the percent root-mean-square error between the model predicted and the experimentally calculated trunk rotational displacements for each moment magnitude

	Flexion				Lateral bend			
	Light	Moderate	Heavy	Maximum	Light	Moderate	Heavy	Maximum
Mean (%)	2.91	2.67	3.08	3.76	3.06	3.49	3.49	4.24
SD	1.59	0.95	2.24	1.36	1.40	1.25	1.43	1.31

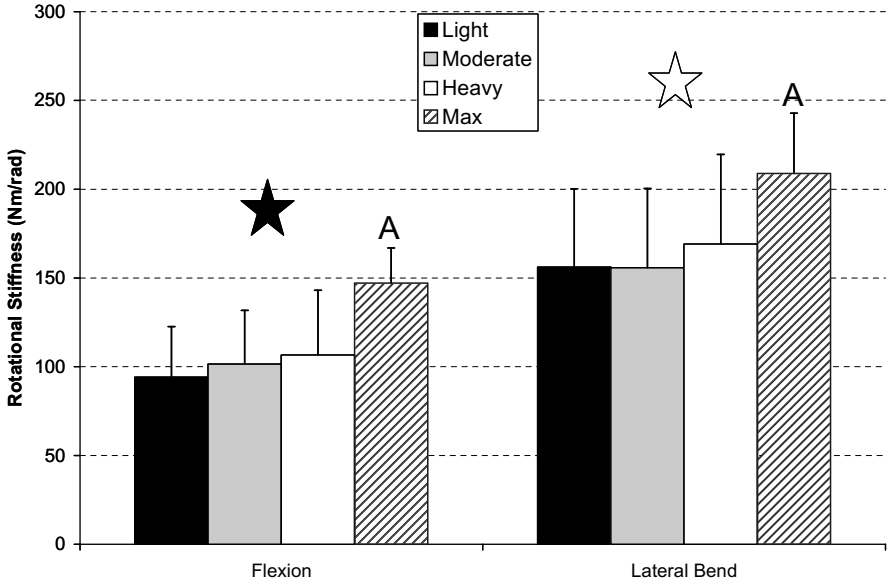

Fig. 3. Average rotational stiffness values calculated for each of the four muscle activation levels in the flexion and lateral bend directions. Directions highlighted by stars of different colour indicate significant differences between one another ($p < 0.05$). A = heavy contraction level significantly different from each of relaxed, light and moderate contraction levels ($p < 0.05$). Error bars denote standard deviations.

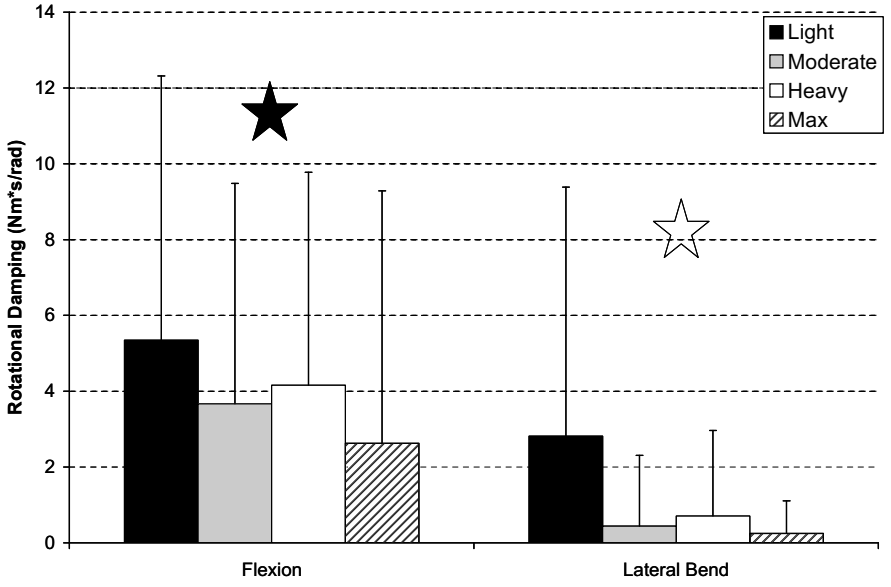

Fig. 4. Average rotational damping values calculated for each of the four muscle activation levels in the flexion and lateral bend directions. Directions highlighted by stars of different colour indicate significant differences between one another ($p < 0.05$). Error bars denote standard deviations.

traces for two muscles (one shutting off and one turning on) in an example flexion trial are shown in Fig. 6.

Also in the flexion trials, there was a significant difference ($p < 0.0001$) in the likelihood of muscle reflex (within 150 ms post-perturbation) onset (11.4%) as compared to offset (1.5%). However, when allowing for voluntary reactions within 1-second post-perturbation, the significant difference ($p < 0.0001$) became opposite (offset 79.8% as compared to onset 42.4%).

In the lateral bend trials, no differences in individual muscle latency probabilities were detected between any of the different muscle contraction levels. Similar to the flexion trials, in lateral bend the likelihood of muscle reflex onset was greater than offset ($p = 0.0172$; onset 5.6% versus offset 2.3%), while in terms of voluntary reaction the likelihoods were reversed ($p < 0.0001$; offset 70.5% versus onset 35.6%).

4. Discussion

The primary result of this study is that despite voluntary muscular responses that acted to influence the quantified

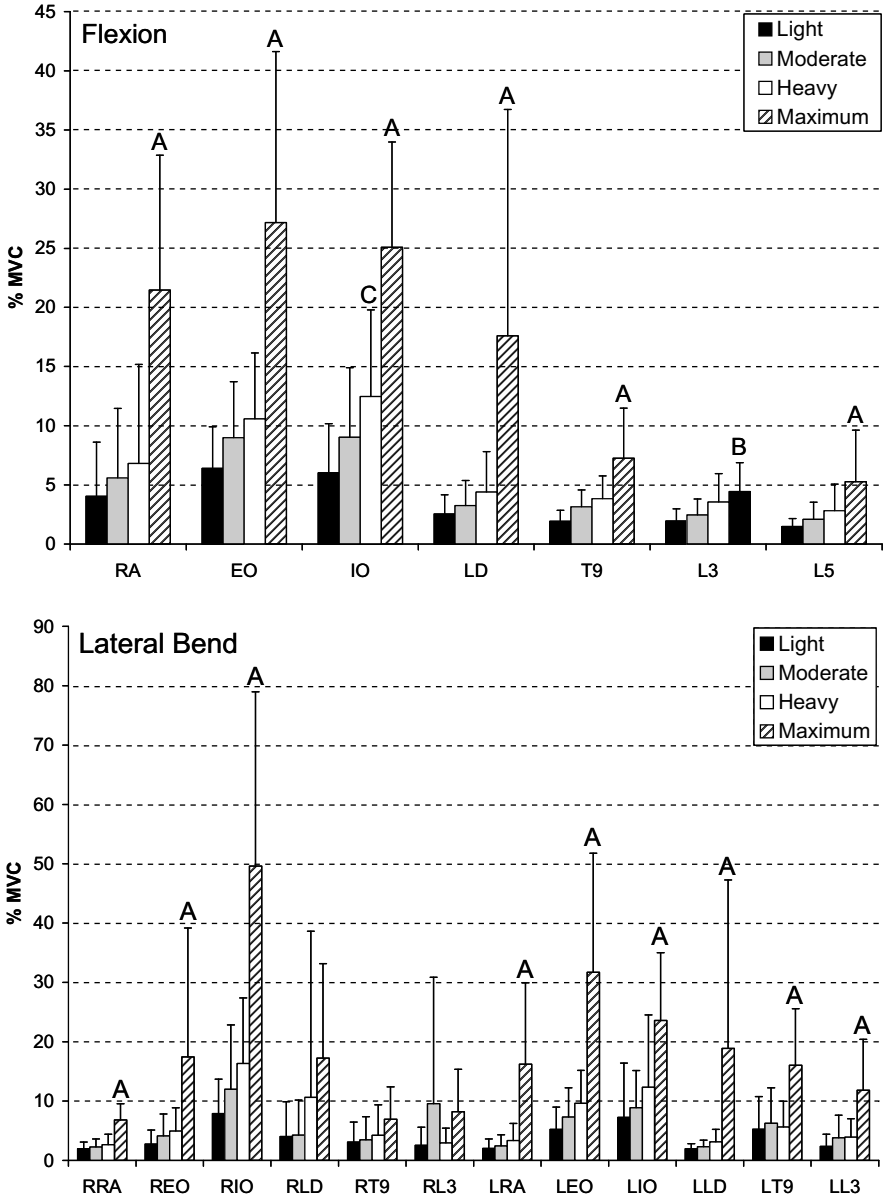

Fig. 5. EMG averages for the 50 ms prior to quick release, across all trials for flexion (averaged bilaterally) and lateral bend (both right and left-side muscles) directions. Significance ($p < 0.05$): A = different from all other levels; B = different from light and moderate levels; C = different from moderate level. Error bars denote standard deviations.

trunk motion, rotational joint stiffness was much lower than would be expected in the presence of reflexive responses. Average trunk deflections during maximum moment trials exceeded 50% of the trunk's passive limit in flexion and 70% in lateral bend, far greater than what has been shown to occur when reflex responses are fully active (e.g. Krajcarski et al., 1999; Cholewicki et al., 2000; Chiang and Potvin, 2001; Vera-Garcia et al., 2007). This indicates that reflexes play an essential role in stiffening the trunk to dynamic perturbations, and that voluntary responses are unable to make up for any neural deficits in reflexive ability within these shortened time periods. In addition, it was found that rotational trunk stiffness increased significantly in conjunction with significant increases in trunk activation that were generated to produce external trunk moments. Smaller, non-significant increases in trunk muscle activation did not result in significant increases in trunk stiffness in these trials.

It has previously been estimated that reflexes can account for levels approaching 50% of the rotational stiffness about a joint during dynamic motions (Sinkjaer et al., 1998; Bennett et al., 1994; Kearney and Stein, 1997; Moorhouse and Granata, 2007; Mirbagheri et al., 2000). The total rotational stiffness is a combination of intrinsic passive tissue, intrinsic muscle, and reflexive muscle contributions. The current study confirms that, of the total muscular contribution to spine rotational joint stiffness, reflexive components contribute the majority of the

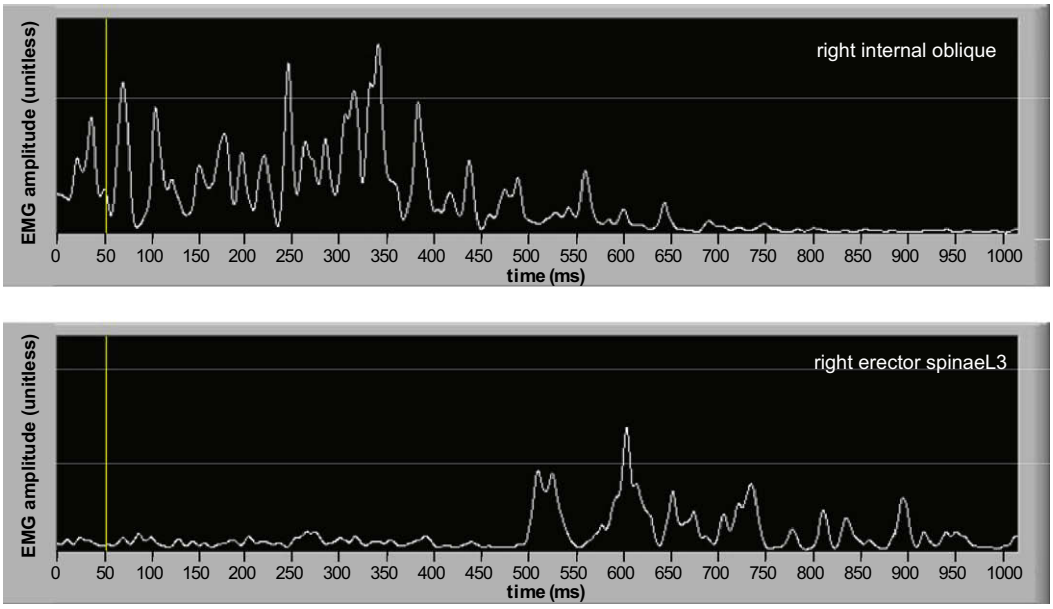

Fig. 6. EMG traces from an example maximum flexion trial. Top: right internal oblique (RIO); Bottom: right erector spinae at the level of L3 (RES-L3). Vertical line on each plot indicates the time of release. EMG signals have been rectified and dual low-passed filtered at 50 Hz (4th order Butterworth). Note that the RIO responded by turning off and the RES-L3 responded by turning on; however, both responses were voluntary in nature, as evidenced by their relatively long latencies.

stiffness. In the current protocol, the effects of intrinsic stiffness due to passive joint structures were somewhat minimized, in particular in the conditions with relaxed musculature, due to the removal of the gravity vector acting to compress the spinal joints. A great deal of stiffening of the intervertebral joints occurs as a result of compressive loading (e.g. Edwards et al., 1987; Janevic et al., 1991; Gardner-Morse and Stokes, 2003). The average rotational joint stiffness values calculated here were 109 Nm/rad for the flexion perturbations, which corresponds to approximately 9–11% of the average values calculated by Cholewicki et al. (2000) in the presence of full reflexes using similar modeling approaches. Assuming, based on documented research, that approximately 40% of trunk stiffness in the Cholewicki et al. study resulted from reflex responses, yields reflexive stiffness values nearly four to five times greater than our intrinsic muscle stiffness values. This is slightly higher than Hoffer and Andreassen (1981) who showed a nearly threefold increase in the stiffness of cat muscle when allowing for reflexes at moderate force levels; the higher stiffness theoretically created by the reflexes in Cholewicki et al. (2000) is most likely due to the additional intrinsic compressive effects of muscular responses on the human trunk.

As participants increased moment levels through the flexor contraction of their abdominal muscles, the only significant differences in EMG level occurred in the maximum as compared to each of the light, moderate and heavy contractions. Fittingly, rotational joint stiffness was significantly higher in the maximum as compared each of the other conditions, but not between any of the other conditions. Despite the increase in EMG activity and stiffness, the likelihood or latency of reflexes did not change, thereby indicating that stiffness was due primarily to changes in the intrinsic stiffness of the muscle. Previous work has shown that reflexes are gained to match background muscle activation levels (Neilson and McCaughey, 1981; Matthews, 1986; Slot and Sinkjaer, 1994), at least from low to mid-range activation; however, the current work has detailed a situation whereby reflexes were inhibited by the experimental protocol (discussed in detail later), thereby nullifying this normal gain adjustment.

In all cases in the current study, lateral bend was shown to be stiffer than flexion. This is most likely due to characteristics of the trunk musculature. In fact, if only considering the stiffness of the passive osteo-ligamentous spine, it has been shown that the lumbar spine is least stiff about the lateral bend axis (Crisco et al., 1992; Gardner-Morse and Stokes, 2003). However, the geometry of the trunk musculature, at least around the neutral posture, is such that potential for rotational joint stiffness is greatest about the lateral bend axis (Brown and Potvin, 2007). It is thought here that, even in the relaxed muscle condition, the passive stiffness of the musculature provided a great deal of stiffness to the lumbar spine, resulting in lateral bend being the stiffest of the two axes. This agrees with previous experimental work examining the passive stiffness of the in vivo human trunk (McGill et al., 1994).

Rotational trunk damping levels were quite low across all trials, and were in fact predicted to be zero in 56% and 85% of flexion and lateral bend trials, respectively. The effect of damping on the stiffness results was probed by conducting simulations on the data of two participants in which damping was held to zero, and the remaining two

coefficients (stiffness and angle offset) were re-calculated as in Eq. (1). Results indicated that rotational stiffness and angle offset would be reduced by an average of approximately 3.4% and 10%, respectively, from the values estimated in the presence of damping. When these simulations were repeated with the additional constraint of holding the angle offset to its originally determined value in the presence of zero damping, rotational stiffness increased by an average of approximately 5.5%. Thus, it can be concluded that rotational damping had little effect on the results, and that rotational stiffness of the trunk dominated the perturbation response for the conditions assessed in the current study.

A number of factors potentially contributed to the lack of reflexive responses during the perturbation trials in the current study. First, the mechanical set-up of the experimental protocol acted to remove the force of gravity that would serve to carry the trunk away from its position post-release were an upright posture initially adopted. Participants lay either on their right-side or back on a near-frictionless apparatus, and were perturbed only by their own internal moment generation. Thus, the peak rotational trunk velocities in response to the perturbations were relatively low (avg/SD 26.3/16.7 deg/s), thus resulting in longer times to maximum trunk deflection than in previous studies (1150/200 ms, as compared to 250/112 ms in Cholewicki et al., 2000). Furthermore, maximum trunk deflections were likely not limited by muscle responses, as in the maximum moment generation trials, maximum deflections approached the trunk's elastic limit (54.3/13.3% or 29.0/7.8° of flexion; 74.0/14.0% or 24.7/6.8° lateral bend). These rotational displacements are far greater than those documented previously in our laboratory for quick releases from upright positions (average 4.9°; Brown et al. (2006) unpublished portion of study) despite similar ranges of EMG activity. Further, the large displacements combined with the relatively slow velocities may not be conducive to eliciting muscle spindle responses (Hunt and Ottoson, 1976; Houk et al., 1981; Proske et al., 2000). Finally, in the lying down position, the threat to trunk stability was most likely perceived by the participants as relatively low, which may have influenced the reflexive responses. It has previously been shown that reflex magnitudes are reduced when threat is minimized in postural control and gait (Cordo and Nashner, 1982; Rietdyk and Patla, 1998), and also when muscles no longer act in their normal postural sense (Marsden et al., 1981; van der Fits et al., 1998), indicating that reflexes likely have a cortical pre-setting of gain and therefore may be context dependent (Matthews, 1991). Finally, it must be considered that the method of reflex detection utilized in the current study was not sensitive to all of the true muscle reflexes. Lee et al. (2007) have recently demonstrated that background muscle activity can hinder the ability to detect muscle responses using automated techniques. The automated detection method used here was checked during pilot testing by visually inspecting and selecting reflex latencies for each muscle over a number of trials; these visually selected latencies matched quite well with those detected using the automated method. Thus, while it is possible that some muscle reflexes may have been missed during the detection analysis, it is believed that the vast majority were at least correctly identified as being present or absent. Further, any potential missed reflex responses do not alter the conclusion that such reflexes were insufficient to limit the substantial (>50% ROM) trunk displacements that were measured in response to the trunk perturbations.

The trunk perturbations examined in the current study do not specifically mimic those naturally occurring in common life. However, this type of perturbation and participant positioning was necessary to limit the reflexive responses that are present when similar perturbation paradigms are administered in the upright posture. In addition, the study was focused on a relatively small, young healthy male population, and thus further work will be necessary to determine if the findings can be generalized to a broader range of individuals.

In conclusion, it has been demonstrated that intrinsic muscle stiffness does not provide adequate stiffening of the spinal joints to prevent excessive rotations upon rapid perturbation. Torso muscle activation levels were similar in the current report as in previous quick release studies, yet spine displacements, and thus the potential for injury, were far greater in the current work, thereby highlighting the lack of adequate stiffening present due to intrinsic muscle properties. This experimental finding substantiates previous model based predictions (Moorhouse and Granata, 2007; Franklin and Granata, 2007) that intrinsic stiffness alone is inadequate to stabilize the human spine, at least in the positions tested in the current study. It is clear that reflexive pathways serve to provide the bulk of the muscular contribution to torso stiffness, and thus continues to shed light on mechanisms necessary for the optimal maintenance of spine control and stability during dynamic activities. Future work will be dedicated to further understand the role of both reflex and intrinsic muscle stiffness, and related muscle activation patterns, designed to reduce the likelihood of acute and chronic spinal injury.

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Stephen Brown received his Ph.D. in April 2008 from the Department of Kinesiology at the University of Waterloo. Previously he received his Bachelors (2000) and Masters (2003) degrees in Human Kinetics from the University of Windsor. His Ph.D. thesis focused on the anatomical and mechanical function of the torso muscles, in particular the abdominals, and their role in the stiffening of the lumbar spine. His main research objectives are to investigate EMG-based modeling, imaging, and muscle/tissue-based techniques, to examine spine function and dysfunction, with an ultimate goal to understand and reduce the likelihood of low back injury.

Stuart McGill is a Professor of Spinal Biomechanics and an author of many scientific publications that address the issues of lumbar function, low back injury mechanisms, development of evidence-based rehabilitation and performance exercise, and the formulation of injury avoidance strategies.