Posteroanterior stiffness of the lumbar spine is influenced by factors, including trunk muscle activity and intra-abdominal pressure (IAP). Because these factors vary with breathing, this study investigated whether stiffness is modulated in a cyclical manner with respiration. A further aim was to investigate the relationship between stiffness and IAP or abdominal and paraspinal muscle activity.

Stiffness was measured from force-displacement resistance and IAP or abdominal and paraspinal muscle activity and intra-abdominal pressure (IAP). Because these factors vary with breathing, this study investigated whether stiffness is modulated in a cyclical manner with respiration. A further aim was to investigate the relationship between stiffness and IAP or abdominal and paraspinal muscle activity.

Stiffness was measured from force-displacement responses of a posteroanterior force applied over the spinous process of L2 and L4. Recordings were made of IAP and electromyographic activity from L2/L4 erector spinae, abdominal muscles, and chest wall. Stiffness was measured with the lung volume held at the extremes of tidal volume and at greater and lesser volumes. Stiffness at L4 and L2 increased above base-level values at functional residual capacity (L2 14.9 N/mm and L4 15.3 N/mm) with both inspiratory and expiratory efforts. The increase was related to the respiratory effort and was greatest during maximum expiration (L2 24.9 N/mm and L4 23.9 N/mm). The results indicate that changes in trunk muscle activity and IAP with respiratory efforts modulate spinal stiffness. In addition, the diaphragm may augment spinal stiffness via attachment of its crural fibers to the lumbar vertebrae.

Spinal stiffness changes throughout the respiratory cycle. J Appl Physiol 95: 1467–1475, 2003; 10.1152/japplphysiol.00939.2002.—Posteroanterior stiffness of the lumbar spine is influenced by factors, including trunk muscle activity and intra-abdominal pressure (IAP). Because these factors vary with breathing, this study investigated whether stiffness is modulated in a cyclical manner with respiration. A further aim was to investigate the relationship between stiffness and IAP or abdominal and paraspinal muscle activity. Stiffness was measured from force-displacement responses of a posteroanterior force applied over the spinous process of L2 and L4. Recordings were made of IAP and electromyographic activity from L2/L4 erector spinae, abdominal muscles, and chest wall. Stiffness was measured with the lung volume held at the extremes of tidal volume and at greater and lesser volumes. Stiffness at L4 and L2 increased above base-level values at functional residual capacity (L2 14.9 N/mm and L4 15.3 N/mm) with both inspiratory and expiratory efforts. The increase was related to the respiratory effort and was greatest during maximum expiration (L2 24.9 N/mm and L4 23.9 N/mm). The results indicate that changes in trunk muscle activity and IAP with respiratory efforts modulate spinal stiffness. In addition, the diaphragm may augment spinal stiffness via attachment of its crural fibers to the lumbar vertebrae.

It has been argued variously that manual handling tasks are better performed during a Valsalva maneuver, during expiration, or with the breath held to optimize the stability of the spine (12, 22, 23, 28). This proposal is based on the assumption that the increased muscle activity and intra-abdominal pressure (IAP) during these tasks is more ideal for control of the spine. However, few studies have investigated whether stability or stiffness of the spine changes during the respiratory cycle or during respiratory tasks that increase inspiratory or expiratory activity of the trunk muscles.

Stiffness of the spine is a function of many factors, including muscle activity and associated stiffness of the muscles and other surrounding soft tissues (e.g., ligament) (21). The effect of paraspinal muscle activity on stiffness has been investigated extensively (6, 24), and activity of erector spinae (ES) of 10% of a maximal voluntary contraction increases stiffness of the spine to a posteroanterior (PA) force by 12% (24). Thus the cyclical variation of paraspinal activity that occurs during respiration (16) may modulate stiffness throughout the respiratory cycle, although this requires clarification.

In addition to the effects of activity of the paraspinal muscles, activity of the muscles that surround the abdominal (AB) cavity, such as the AB muscles and diaphragm, may modulate spinal stiffness, either directly as a result of muscle contraction or via increased IAP. Numerous studies indicate that activity of the AB muscles increases stiffness of the spine (5, 6). However, in vivo experiments, it has been difficult to determine whether this effect is due to the muscle tension directly or the associated increase in IAP (6). Recent data from human (14) and porcine (17) studies confirm that spinal stiffness can be increased when IAP is elevated without concomitant activity of the AB muscles. It has been suggested that elevated IAP may increase spinal stiffness as a result of tensioning the lumbar spine (9), generation of a posterior shear force against the lumbar spine (1), decreasing the compliance of AB contents (23), or indirectly by increasing the tension in the thoracolumbar fascia (27). However, it is important to consider that the effects of IAP and activity of the muscles that surround the abdominal wall are interdependent and cannot occur in isolation. In addition to the effects of AB muscle activity and IAP, spinal stiffness may also be influenced by contraction of the crural diaphragm due to its attachment to the lumbar vertebrae. The crural diaphragm usually extends to L3 on the right and L4 on the left (29). Thus diaphragm contraction may have a direct effect on stiffness at the upper lumbar levels.

Activity of the diaphragm, paraspinal, and AB muscles and IAP are modulated differently across the respiratory cycle. During quiet breathing diaphragm activity is greatest in inspiration and is associated with increased IAP (4), and activity of the paraspinal and AB muscles varies between individuals and postures (10, 15, 16). When expiratory volume or flow is in-
increased, activity of the AB and paraspinal muscles is increased and is associated with increased IAP (4). As a result of this complex interplay between muscle activity and IAP with respiration, it is uncertain whether or how stiffness of the spine varies across the respiratory cycle, although data of respiration-related changes in tissue forces have been reported (22).

Thus the aims of the present study were 1) to determine whether stiffness of the spine is modulated during quiet respiration, 2) to compare the effect of inspiration and expiration above and below tidal volume on spinal stiffness, 3) to investigate the relationship between changes in spinal stiffness, muscle activity, and IAP, and 4) to investigate whether any respiration-related changes in spinal stiffness differed between L2 and L4 (i.e., above and below the crural diaphragm attachment).

METHODS

Subjects. Eight subjects of mean (±SD) age, height, and weight of 39 ± 9 yr, 1.78 ± 0.06 m, and 76 ± 12 kg, respectively, volunteered for this study. Subjects were excluded if they had a history of low back pain or any significant neurological, respiratory, or cardiovascular disease. The study was approved by the institutional Human Research Ethics Committee, and informed, written consent was obtained.

Measurement of stiffness of the trunk (response of the spine to PA force). PA stiffness was measured at L4 and L2 using a device that recorded the force required to achieve a set PA displacement of an indenter applied over the spinous process. The device consists of a servomotor, which drives an indenter to a set distance with variable force. The indenter is positioned in contact with the skin over the spinous process of the vertebrae and force is applied in a cyclical manner (1 Hz) for five repetitions in the PA direction. Force and displacement are measured with a strain gauge and linear potentiometer, respectively. Force was applied at an angle of 4.5° in a caudal direction at L4 and at 11.5° in a cephalad direction at L2 (26). The range of displacement was selected to obtain a linear relationship between force and displacement. The maximal applied force was set at 150 N (20). Measurement of PA stiffness by this device has good test-retest reliability and is highly accurate (20). The measure obtained with this device is a composite measure of shear and bending of the torso and not simply intersegmental stiffness at a specific intervertebral level.

IAP. IAP and intrathoracic pressures were measured via a pair of transducers inserted into the stomach via the nose (Gaeltec). One transducer was positioned in the stomach to record gastric pressure (Pga) and the other above the diaphragm to record esophageal pressure (Pes). The optimal position of the tube was confirmed by opposite changes in Pga and Pes with a sniff and a Mueller maneuver. Once the tube was located in the desired position, it was taped to the nose.

Respiratory measurements. Airflow was measured with a pneumotachograph (Hans Rudolf) and integrated on-line to be recorded as volume. Feedback of volume was displayed on an oscilloscope for tasks in which subjects were required to match specific breath volumes. Respiratory movement of the rib cage was measured with an inductance plethysmograph (Respirac 5, Ambulatory Monitoring) placed around the chest.

Electromyography. Electromyographic (EMG) recordings of the trunk muscles were made using Ag-AgCl surface electrodes (5-mm disks, Conmed). Pairs of electrodes (interelectrode distance: 20 mm) were placed over the ES muscles ~4 cm lateral to the spinous processes of L2 and L4 in parallel with the muscle fascicles and over the lateral abdomen midway between the rib cage and iliac crest in an oblique direction to record activity of the AB muscles. This electrode site was selected to provide a general recording of the AB muscles and would include contribution from the obliquus externus and internus abdominis and transversus abdominis. An additional pair of electrodes was placed over the 7th and 8th intercostal spaces in the midclavicular line to record EMG activity from over the diaphragm. Because this electrode records activity from muscles other than the diaphragm, (e.g., intercostal and AB muscles), it was referred to as “chest wall” EMG. EMG data were band-pass filtered between 53 Hz and 1 kHz (Digitimer) and sampled at 2 kHz using a Power1401 and Spike2 software (Cambridge Electronic Design). Data were filtered below 53 Hz to remove movement and electrical artifact.

Experimental procedure. Subjects were positioned face down on a rigid plinth with their arms by their sides and neck supported in a neutral posture (i.e., without flexion or extension) (Fig. 1A). The pelvis and rib cage were supported on blocks so that there was no external pressure on the abdomen, and a belt was placed firmly around the rib cage to minimize differences in rib position between lung volumes. The spinous processes of the L2 and L4 vertebral levels were identified by palpation and marked with a pen. The indenter was placed over the spinous process of the target vertebra (L4 or L2 for separate trials), and five cycles of force were applied at 1 Hz (Fig. 1A).

The response of the lumbar spine (at L4 and L2) to PA force was assessed while subjects performed a series of respiratory tasks (Fig. 1B) in pseudorandom order. In all tasks, lung volume was held with the glottis open. The tasks were as follows: lung volume held at functional residual capacity (FRC), lung volume held at end-tidal inspiratory volume, lung volume held at 50% of maximal inspiratory volume, lung volume held at total lung capacity (TLC), lung volume held at 50% of the volume of expiration from FRC to maximal expiration, and lung volume held at residual volume (RV).

In an additional trial, the measure at TLC was repeated with the glottis held closed. This was included to confirm that changes in stiffness that occurred with inspiratory efforts were due to the associated increase in muscle activity and Pga and not the effects of increased lung and rib cage volume. Closure of the glottis maintains lung volume, but allows inspiratory muscles to relax. Furthermore, Pga is decreased when the glottis is closed as a result of equalization of the pressure in the AB and thoracic cavities. Identification of small fluctuations in the airflow (due to cardiac movements) confirmed that the glottis had remained open during the procedure. Subjects practiced each task while maintaining an open glottis. Feedback of the required lung volume and targets was provided for each task, and subjects were instructed to maintain the volume during the stiffness measurement.

Data analysis. Force-displacement plots were generated from the output of the stiffness device. Stiffness was calculated as the slope of a regression line fitted to the force-displacement curve between 50 and 110 N (Fig. 1C). This range of force was chosen because the force-displacement curves were linear in this range. Other studies that have measured stiffness with PA force applied to the lumbar spine have shown good reliability when stiffness is calculated in
Stiffness values were averaged over the last three consecutive cycles in each test. The first cycle was not used because it is more variable and inconsistent with stiffness recorded during subsequent cycles. Stiffness values were expressed as a proportion of the stiffness at FRC.

Root-mean-square EMG amplitude was measured for a 1-s epoch (i.e., an entire loading cycle) during the application of the PA force and normalized as a proportion of the value at FRC. The mean amplitude of Pga and Pes was recorded during the same 1-s epoch as the EMG data and were normalized as a proportion of the value at FRC. Transdiaphragmatic pressure (Pdi) was calculated as the difference between Pga and Pes.

Statistical analysis. Stiffness at L4 was compared between respiratory tasks and vertebral levels by using a one-way ANOVA and Duncan’s multiple-range test. Pearson’s r was calculated to determine the correlation between mean stiffness at L4 and the mean values for EMG and pressure. The amplitude of each parameter between respiratory conditions was compared with separate ANOVAs and Duncan’s multiple-range test. The α-level was set at 0.05.

RESULTS

Changes in stiffness at L4. Stiffness was measured at L4 during respiratory maneuvers representing different efforts across the respiratory cycle. Stiffness for L4 at FRC was 15.3 ± 3.2 (SD) N/mm and increased to 18.5 ± 5 N/mm at TLC and 23.9 ± 7.3 N/mm at RV (Fig. 2). There was no significant change in the stiffness at L4 between FRC and the end of a tidal inspiration (with the glottis open) (P = 0.111). However, there was a trend for the stiffness to increase above FRC. When measurements were made during tasks in which lung volume was held above (inspiratory) and below (expiratory) the normal tidal volume, the stiffness at L4 was increased above the values recorded at FRC (Table 1) (P < 0.05). Stiffness at L4 was increased from the FRC value by a factor of 1.22 for 50% inspiration, 1.24 for TLC, and 1.53 for RV (Table 1). The increase in stiffness was greater at RV than TLC (P < 0.005).

Relationship between changes in stiffness, EMG, and pressure. To investigate the mechanism for the change in stiffness with increased respiratory efforts, we evaluated the relationship between stiffness and changes in EMG and pressure. Raw data for a representative subject are shown for each condition in Figs. 3, 4, and 5. All EMG and pressure measurements were positively correlated with stiffness across the range of tasks (Fig. 6). Consistent with these data, when the amplitude of each parameter was compared between respiratory tasks, the general trend was similar to that identified for changes in L4 stiffness; i.e., the values were increased above those recorded at FRC for the tasks in which lung volume was held above or below the normal tidal volume (Fig. 7). There were several exceptions to this trend. EMG recorded with electrodes over L4 ES, AB, and the chest wall was greater than that recorded at FRC for all lung volumes (P < 0.05). In contrast, the EMG amplitude recorded with electrodes over ES at the L2 level was only increased above that recorded at FRC during the expiratory tasks (P < 0.001). Pdi and AB pressures increased above FRC values with all tasks, but the amplitude of the increase was greater with inspiratory compared with expiratory tasks (P < 0.05).

We compared the stiffness at L4 at TLC with the glottis open and closed to confirm that the changes in stiffness were due to the elevated EMG and pressure.
and not simply the change in lung volume. Chest wall EMG (P < 0.001) and Pdi (P < 0.001) decreased with closure of the glottis, but there was no change in Pga (P = 0.48) (Fig. 4, Table 2). Correspondingly, when stiffness was compared between glottis conditions at TLC, the stiffness at L4 was greater with the glottis open (18.5 N/mm) than with the glottis closed (15.6 N/mm) (P < 0.01), and the values recorded with the closed glottis were not different from the values at FRC (15.3 N/mm) (Table 2) (P = 0.704). There was no change in AB (P = 0.51) or ES EMG (L2: P = 0.36, L4: P = 0.25) between conditions (Fig. 4, Table 2). This suggests that changes in chest wall EMG and Pdi, which are both indicators of activity of the diaphragm (in the absence of change in ES and AB EMG), are sufficient to influence L4 stiffness.

Comparison of stiffness changes at L2 and L4. Because of the direct attachment of the crural diaphragm to the upper lumbar vertebrae, we tested whether the stiffness at L2 was increased to a greater extent than that at L4. When subjects held lung volume at TLC, stiffness at L2 was greater than at L4 (P = 0.038). However, there was no difference between stiffness at these two lumbar levels during other conditions (Fig. 8, Table 1) (FRC: P = 1.0, inspiratory volume: P = 0.14, 50% of maximal inspiratory volume: P = 0.66, 50% of

Table 1. Mean values for lumbar stiffness at L4 and L2 during all respiratory tasks

<table>
<thead>
<tr>
<th></th>
<th>FRC</th>
<th>VT</th>
<th>50% Insp</th>
<th>TLC</th>
<th>TLCGC</th>
<th>50% Exp</th>
<th>RV</th>
</tr>
</thead>
<tbody>
<tr>
<td>K L4</td>
<td>15.3 ± 3.2</td>
<td>18.4 ± 3.6</td>
<td>18.6 ± 4.1*</td>
<td>18.5 ± 5.0**</td>
<td>15.6 ± 3.7</td>
<td>19.4 ± 6.2</td>
<td>23.9 ± 7.3*</td>
</tr>
<tr>
<td>K L2</td>
<td>14.9 ± 1.8</td>
<td>16.1 ± 2.5</td>
<td>18.3 ± 3.2</td>
<td>19.9 ± 2.1</td>
<td>15.1 ± 3.4</td>
<td>17.0 ± 3.9</td>
<td>24.9 ± 9.0*</td>
</tr>
</tbody>
</table>

Values are means ± SD in N/mm. K, lumbar stiffness; FRC, functional residual capacity; VT, end-tidal inspiration; 50% Insp, 50% of maximal inspiration; TLC, total lung capacity; TLCGC, TLC with glottis closed; 50% Exp, 50% of maximal expiration; RV, residual volume.
*Significantly different from FRC, P < 0.05.
the volume of expiration from FRC to maximal expiration: $P = 0.59$, RV: $P = 0.23$). Group data show that the increase in stiffness at L4 reaches a plateau at the lung volumes above normal tidal volume (Fig. 8). In contrast, L2 stiffness increases with chest wall EMG for all increments in lung volume.

**DISCUSSION**

The present study demonstrates the effect of respiration on stiffness measured in response to force applied to the lumbar spine during mechanical loading. Although stiffness at L4 did not change with lung volumes within the normal tidal range, when lung volume increased above or below FRC, stiffness was increased. Across tasks, changes in stiffness were positively correlated with changes in trunk muscle EMG and both Pdi and AB pressure. Furthermore, increased stiffness associated with increased pressure and chest wall EMG, but with ES and AB muscle activity held constant, supports the proposal that activity of the diaphragm and the associated increase in IAP contribute to the stiffness modulation. However, contribution of the diaphragm to spinal stiffness is likely to be dependent on activity of the other muscles.

The results of the present study are consistent with a brief report that demonstrated no change in stiffness with quiet respiration during breath holding or tidal breathing (2) and data that indicate changes in tissue loading with respiration (22). More recently, stiffness has been observed to increase during a Valsalva maneuver, although when breath was held at full inspiration, stiffness did not increase in the majority of subjects (19). However, it is unclear whether the glottis was maintained open or closed. Although there has been some investigation of the effects of respiration on spinal stiffness, our study is the first to compare the stiffness response of a variety of respiratory efforts, including both inspiration and expiration.

Stiffness of the spine is a composite measure of the response of the spine that not only does measure intersegmental stiffness at a specific intervertebral level, but also is influenced by stiffness of the entire spine and its supporting structures. For instance in the lumbar spine, measurement of the response of the spine to
PA force reflects a complex movement of the spine, which involves soft tissue compression, extension of the spine, anterior rotation of the pelvis, deformation and rigid-body displacement of the rib cage, and a small amount of anterior shear of the target vertebra (21). Accurate measurement of intervertebral stiffness requires fixation of adjacent segments of the spine. However, the stiffness measured at the segments at which the force is applied is likely to have a larger contribution from the mechanical properties of that segment than adjacent segments. In the present study, motion of the rib cage was restricted by application of a firm belt to minimize further compression and ensure that rib position was consistent between test conditions, and the abdomen was left unsupported to reduce the effect of the compliance of the AB contents on the

![Figure 5](image5.png) Representative data for the 50% expiration (A) and RV (B) conditions. Increases in stiffness were greatest during testing at 50% expiration and RV. Increases in muscle activity were greater at RV, which involved greater respiratory effort. There were also corresponding increases in pressures.

![Figure 6](image6.png) Relationship between L4 stiffness and EMG and pressure. EMG and pressure recordings were correlated with stiffness. A: L4 ES EMG, B: abdominal EMG, C: Pdi. D: chest wall EMG. E: L2 ES EMG, F: Pga. All values (stiffness, EMG, and pressure) are the mean values expressed as a proportion of the value at FRC.
stiffness measure. Despite the limitations of this measure of spinal stiffness, it does provide a noninvasive technique to evaluate spinal stiffness, at least in one direction.

This study indicates that the behavior of muscle activity and pressures was consistent with the change in stiffness, in general it was not possible to distinguish between them, and it is unlikely that these factors act independently on spinal stiffness. During increased respiratory effort, there are increases in activity of the AB and ES muscles and chest wall EMG (i.e., diaphragm and intercostal muscles), as well as increases in pressures (Pga and Pdi), and these increases were associated with increased spinal stiffness. Although it is not possible to confirm a causal relationship, the data suggest that this is likely. The high correlation between ES EMG activity and stiffness suggests that this muscle contributes significantly to stiffness (24). The EMG recordings of ES and AB muscles in this study are most likely to represent activity of the superficial muscles; however, activity of the deeper trunk muscles, such as the multifidus and transversus abdominis, is also likely during some of the respiratory tasks and will contribute to the net change in stiffness. Many muscles affect spinal stiffness (3, 7). Notably, of the AB muscles, transversus abdominis has the lowest threshold for respiratory activity (10) and is thought to be important for control of intersegmental stiffness of the spine (8, 13, 18) via increased IAP (11, 13) or tension in the thoracolumbar fascia (27). However, other muscles also have an important contribution to modulation of spinal stiffness.

Stiffness was unchanged over tidal volume, and the associated changes in Pga and Pdi were small. Both Pga and Pdi were greater during inspiratory effort than expiratory effort. Insp, inspiration; Exp, expiration; VT, end-tidal inspiration.

Table 2. Mean values during testing at L4 for all parameters when the lung volume was held at FRC, TLC, and TLCGC

<table>
<thead>
<tr>
<th>Parameter</th>
<th>FRC</th>
<th>TLC</th>
<th>TLCGC</th>
</tr>
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<tbody>
<tr>
<td>L4</td>
<td>15.3 ± 3.2</td>
<td>18.5 ± 5.0*</td>
<td>15.6 ± 3.7†</td>
</tr>
<tr>
<td>L4 ES EMG</td>
<td>1</td>
<td>3.3 ± 2.8*</td>
<td>2.5 ± 3.7*</td>
</tr>
<tr>
<td>L2 ES EMG</td>
<td>1</td>
<td>2.1 ± 1.2</td>
<td>1.5 ± 0.9</td>
</tr>
<tr>
<td>AB EMG</td>
<td>1</td>
<td>3.2 ± 2.9*</td>
<td>1.9 ± 1.2*</td>
</tr>
<tr>
<td>Chest wall EMG</td>
<td>1</td>
<td>5.1 ± 3.1*</td>
<td>2.6 ± 2.0**</td>
</tr>
<tr>
<td>Pga</td>
<td>24.8 ± 4.6</td>
<td>59.7 ± 24.7*</td>
<td>45.1 ± 8.1*</td>
</tr>
<tr>
<td>Pdi</td>
<td>16.1 ± 5.8</td>
<td>69.5 ± 34.5*</td>
<td>21.9 ± 14.3**</td>
</tr>
</tbody>
</table>

Values are means ± SD. Stiffness and pressure data are presented as absolute values, and electromyogram (EMG) is normalized as a proportion of the values recorded at FRC. ES, erector spinae; AB, abdominal; Pga, gastric pressure; Pdi, transdiaphragmatic pressure.

*Significantly different from FRC, P < 0.05. †Significant difference between TLC and TLCGC, P < 0.05.
above and below normal tidal volume with the greatest increase in these pressures measured during the maximal inspiratory effort. In contrast, the greatest increase in stiffness was recorded with maximal expiration. This finding does not indicate that changes in pressure (Pga and Pdi) and stiffness were not related, but it highlights the complex manner by which pressures and muscle activity contribute to the stiffness increase. That is, the measured stiffness represents the net effect of all factors that are independently changed by respiratory efforts. Although other studies have identified increased spinal stiffness during tasks that increase Pga, such as a Valsalva maneuver (19), it is difficult to confirm to what extent the pressure increase affects the stiffness measurement in these studies as activity of the AB and back extensor muscles increase to produce the pressure change. One recent study in which Pga was increased by electrical stimulation of the phrenic nerves has confirmed that pressure alone can increase spinal stiffness (14). However, in function the net stiffness of the spine will be the product of all interrelated components of muscle activity and pressure.

EMG activity recorded with the electrodes over the chest wall was also correlated with stiffness. Stiffness at TLC with the glottis closed was the same as at FRC but was increased with the glottis open. Lung volume was similar in both conditions, and, therefore, it is unlikely that passive changes in lung volume alone have an important influence on stiffness. Thus it is likely that the sustained activity of the diaphragm to maintain lung volume in the open-glottis condition contributed to the increase in stiffness.

Contraction of the diaphragm may also increase spinal stiffness via the attachment of the crural diaphragm to the upper lumbar vertebrae. We compared changes in stiffness at L2 and L4 with inspiratory efforts because the crural diaphragm attaches to the lumbar vertebrae as caudal as L2 or L3 (29). Therefore, if the crural diaphragm contributes to lumbar stiffness, it should be greater at L2 than at L4, where there is no direct attachment. The greater increase in stiffness at L2 reported here is consistent with this proposal. This is also consistent with recent data from porcine studies that indicate that the effect of diaphragm activity on spinal stiffness is reduced when the crural attachments are cut (17). These data suggest that the crural fibers may modulate stiffness due to compressive forces. However, it is important to consider that the present study cannot rule out contribution from other factors, such as nonuniformity of the paraspinal muscles. As mentioned above, noninvasive measurement of spinal stiffness cannot provide an ideal measure of intersegmental stiffness and is affected by the stiffness at segments distant from the site of application of the PA force. However, the finding that the change in stiffness was greater at L2 compared with L4 during similar tasks suggests that there was independence between the two measures and that the measures are likely to reflect changes at the specific level, in addition to an influence from other distant sites. The proposal that stiffness is increased by contraction of the crural fibers is further strengthened by comparison of the pattern of the increase in stiffness at L2 and L4 (see Fig. 8). The stiffness increase with increasing inspiratory efforts at the L2 level corresponds to an incremental increase in chest wall EMG. In contrast, the increase in stiffness at L4 reached a relative plateau at higher lung volumes, independent of further increases in chest wall EMG.

The results of this study have several functional and clinical implications. First, numerous studies have investigated the relationship between breathing and lifting. Generally, these studies argue that breath holding and expiration are normal behavior (12, 23). The present data suggest that stiffness of the spine is increased with both inspiratory and expiratory efforts but more so with expiration, at least in the direction tested in the present study. This is consistent with the natural behavior to expire when performing demanding tasks such as lifting. Furthermore, the present data argue that stiffness is greater with the glottis open and trunk muscle contraction maintained; therefore, it might be beneficial to recommend this action when attempting procedures requiring greater spinal stability. Second, clinical application of PA force to the spine is commonly used in the assessment and treatment of acute spinal pain. The present data suggest that it may be necessary to standardize the point in the respiratory cycle to make accurate judgments of spinal stiffness.

We thank Liset Pengel, Inger Heijnen, and Lorimer Moseley for assistance with data collection and Dr. Elizabeth Ellis for comments on the manuscript.

DISCLOSURES

Financial support for this study was provided by the National and Medical Research Council of Australia and Physiotherapy Research Foundation of Australia.

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