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Evaluation of the Flexion Relaxation Phenomenon of the Trunk Muscles in Sitting

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Study Design. A normative, single-group study was conducted.

Objective. To investigate the flexion relaxation phenomenon in the thoraco-lumbopelvic muscles among a pain-free population when moving from an upright to a slump sitting posture.

Summary of Background Data. The presence of the flexion relaxation phenomenon (FRP) of the back muscles is well documented at end-range spinal flexion when standing. This phenomenon is commonly found disrupted in low back subjects. However, whether FRP occurs in sitting remains controversial.

Methods. The sample consisted of 24 healthy pain-free adults. Surface electromyography was used to measure activity in the superficial lumbar multifidus (SLM), the thoracic erector spinae (TES), and the transverse fibers of the internal oblique (IO) muscles while subjects moved from an erect to a slump sitting posture. An electromagnetic motion-tracking device simultaneously measured thoracolumbar kinematics during this task.

Results. There was a significant decrease in both the SLM and the IO activity when moving from an erect to a slump sitting posture ($P = 0.001$ and $P = 0.004$, respectively), indicating the presence of FRP. TES activity was highly variable. While 13 subjects exhibited an increase in activity ($P = 0.001$), 11 demonstrated a decrease in activity ($P = 0.001$), indicating the presence of FRP. FRP occurred in the mid-range of spinal flexion for the SLM, IO and TES when present.

Conclusion. The findings show that the SLM and the IO are facilitated in neutral lordotic sitting postures and exhibit FRP at mid range flexion while moving from upright sitting to slump sitting. These findings show that FRP in sitting differs from that in standing. Variable motor patterns (activation or FRP) of the TES were observed. These findings suggest that sustaining mid to end-range flexed sitting spinal postures result in relaxation of the spinal stabilizing muscles.

Key words: lumbar spine, flexion relaxation phenomenon, electromyography, posture, trunk muscles, sitting. **Spine 2006;31:2009–2016**

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Prolonged sitting is frequently associated with the aggravation of low back pain.[1–3] However, little research has investigated the motor control of the lumbar spine in relation to sitting. During upright sitting cocontraction of spinal stabilizing muscles such as the superficial lumbar multifidus (SLM), erector spinae and transverse abdominal wall muscles is observed.[4,5] In contrast, passive postures such as sway standing and slump sitting have been shown to result in decreased activation of these same muscles.[4] End-range flexion in standing has also been associated with a decrease in back muscle activity.[6] During forward bending in standing, this inhibitory response of the back muscles is commonly referred to as the flexion relaxation phenomenon (FRP), where the transition of load moves toward other active and/or passive structures close to the end range of flexion.[7–10]

The FRP has not been widely investigated in sitting, and the studies performed in sitting lack consensus as to whether FRP occurs in the lumbar paraspinal muscles.[4] Callaghan and Dunk[11] reported that when moving from an upright to a slump sitting posture, FRP occurred in the thoracic erector spinae (TES) muscles at the end range of movement, while the lumbar erector spinae muscles did not exhibit FRP. This is in contrast to a study by O'Sullivan *et al*[4] who demonstrated a clear reduction in muscle activity of the SLM, the TES and the transverse fibers of internal oblique (IO) muscles during slump sitting, when compared with upright sitting, thus suggesting the occurrence of the FRP. If FRP of the lumbopelvic muscles does occur in sitting, uncertainty remains as to where in the range of spinal flexion this phenomenon occurs.

In light of these inconsistencies, the objective of the present study was to determine whether FRP occurs in the SLM muscles, the TES muscles and the transverse fibers of IO muscles when moving from a lumbopelvic upright sitting to a slump sitting posture, and if so, where in the range of spinal flexion the reduction in surface electromyography (sEMG) activity occurs. Understanding these factors will provide greater insight into the lumbopelvic postures that result in the activation and relaxation of the spinal stabilizing muscles in sitting.

Materials and Methods

Subjects. Twenty-four healthy adult subjects, including 14 men and 10 women without LBP (mean age, 32 ± 13 years; mean height, 172 ± 10 cm; mean weight, 71 ± 11 kg) were recruited for this study from the Perth metropolitan region. Ethical approval for the study was obtained from the Curtin University Ethics committee and informed consent was obtained from all subjects before testing. Subjects were excluded from the study if they were pregnant, had a body mass index greater than 28 kg/m^2, had any reports of LBP within the last 2 years (and/or required medication, consultation with a health professional, or days off work for LBP), or had any known spinal disorders.

Experimental Protocol. Synchronized recordings of the activation of selected trunk muscles (sEMG) and the range of motion measurements (3Space Fastrak) of the thoracolumbar spine were obtained for each subject when moving from an upright to a slump sitting posture. Three trials of 15 seconds duration each were conducted, with a 30-second rest period given between trials. The protocol was developed in this manner, as the maneuvers were functional, nonfatiguing and highly repeatable.

Subjects were instructed to sit on a stool that was adjustable for height, but with no back support. The subjects were instructed to sit with their hips and knees at 90° and with feet positioned at shoulder width apart, and arms relaxed at the side of their body. They were instructed to focus straight ahead at a designated point while assuming an upright sitting posture. The upright position was defined as an anterior rotation of the pelvis to achieve a neutral lordosis of the lumbar spine and relaxation of the thorax, as described by O'Sullivan *et al.*[4] Subjects were asked to maintain the upright position for 5 seconds and then move from the erect sitting posture to a slump sitting posture by relaxing the thoracolumbar spine and rotating their pelvis into posterior tilt. This slump sitting position was then maintained for 5 seconds as they continued to look straight ahead (Figure 1). This time period is similar to that used by previous studies,[4] allowing for the comparison of the results. Timing was controlled using a metronome and standardized instructions were given to position the participants for each trial.[4] Before data collection, a trial period was undertaken to familiarize the subject with the protocol.

Data Collection Equipment. Surface EMG signals from three bilateral trunk muscles were recorded at a sampling frequency of 1,000 Hz using two Octopus Cable Telemetric systems (Bortec Electronics Inc., Calgary, Alberta, Canada). The sEMG system bandwidth was 10 to 500 Hz and the common mode rejection ratio was greater than 115 dB at 60 Hz. All raw myoelectric signals were amplified with a gain of 2000. Data were collected and processed with a customized software program employing LabVIEW V6.1 (National Instruments).

For the purposes of sEMG measurement, the skin was prepared to reduce skin impedance to below 5 kΩ by cleaning the site with alcohol, shaving the electrode site and lightly abrading the skin with fine sandpaper.[12] Pairs of circular self-adhesive disposable Ag/AgCl disc surface electrodes (3 M Red Dot, 3 M Health Care Products, London, Ontario, Canada) with an electrical contact surface of 1 cm^2 were placed unilaterally, with a 2.5-cm interelectrode distance and parallel to the muscle fibers of the following muscles: transverse fibers of the IO with the potential to pick up cross talk from the underlying transverses abdominis (1 cm medial to the anterior superior iliac spine beneath a line joining both anterior superior iliac spines),[13] superficial fibers of the SLM (L5 and aligned parallel to the line between the posterior superior iliac spine and the L1–L2 interspace),[14,15] and the TES (5 cm lateral to the T9 spinous process).[11] Two common earth electrodes (one for each EMG unit) were placed over the iliac crest. Snap leads were used to connect the surface electrodes to the amplifiers, and electrodes were taped securely to avoid excessive movement of the leads.

Before testing, a series of maximum voluntary isometric contractions (MVIC) of the trunk muscles were performed using standardized procedures for the purpose of sEMG data normalization in order to allow the findings to be compared with other comparable research.[11] To generate MVIC for IO, the subject was positioned supine with the legs straight and strapped with a belt. A resisted crossed curl-up, with the right shoulder moving towards the left and maximal manual isometric resistance applied through the right shoulder by the investigator (standing at the left side) for left IO muscle. For the right IO, the same procedure was repeated to the right with the investigator standing at the right side applying resistance to the left shoulder.[16] For the back muscles, the subject was positioned prone with the legs straight and strapped with a belt. One normalization technique was used for the SLM and TES. The subject was in prone position, hands on the neck, and asked to lift the head, shoulders, and elbows just off the examination table. Symmetric manual resistance was provided to the scapular region by the investigator (standing at the head of the subject).[16] Clear instructions were given to the subject to perform a maximum contraction.

These procedures have been shown to demonstrate high levels of reliability.[16,17] Normalization measurements for each subject were obtained by conducting three MVIC trials of three seconds duration each,[18] with a 3-minute rest period given between trials so as to avoid the cumulative effect of fatigue.[19] The mean MVIC value from the three trials was used as the measurement for each subject.

In order to be able determine where the FRP occurred in the range of thoracolumbar motion, the 3Space Fastrak motion tracking system (model 3SF0002, Polhemus Navigation Science Division, Kaiser Aerospace, VT) was used. Both the reliability and validity of this three-dimensional measurement system have been demonstrated for the measurement of lumbar spine movement, with a recorded accuracy of 0.2°.[20] For the purposes of this study, we used the electromagnetic source and three sensors, and data were sampled at a frequency of 25 Hz. Sensors were placed over the spinous processes of S2, T12, and T6 to allow for calculation of the lower thoracic and lumbar spine curvatures. In order to maintain the integrity of the sensor positioning throughout testing, subjects were asked to bend forward slightly while the three sensors were taped securely in place.

Data Management. The raw sEMG data were visually checked for electro-cardiac artifacts. Where it was observed

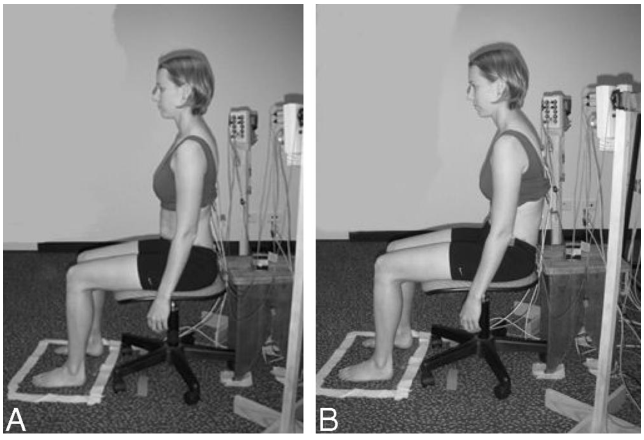

Figure 1. (**A**) Erect sitting posture. (**B**) Slump sitting posture.

that electro-cardiac signal contaminated the EMG signal, the artifact was manually removed and replaced with adjacent unaffected data of the same duration, using a customized program in Labview. The sEMG data were then normalized to MVIC, demeaned, full wave-rectified, and filtered using a fourth order zero lag Butterworth filter[21] with a cutoff frequency of 4 Hz to yield linear envelopes from each channel.

A number of methods have previously been described to determine the presence or absence of FRP. These include either visual inspection of muscle activity of the back extensors,[9,22]or a definition based on a reduction in MVIC by either less than 1% of the sEMG levels during upright sitting[11] or 3% of MVIC.[10] FRP has also been expressed as a statistical reduction of muscle activity,[4] as a ratio of the sEMG activity during the forward flexion and fully flexed positions or as a ratio of the sEMG activity during extension and flexion movements.[23–25]

In the present study, the FRP was analyzed statistically, visually, and on the basis of a change in MVIC of greater than 3% in order to compensate for the inconsistencies in defining FRP in the literature and to allow comparison of the present study results with other research on FRP in sitting.[11] Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) statistical analysis software, version 10.0 (SPSS, Chicago, IL). An average of three seconds of amplitude normalized data were calculated for the static phases of the procedure, in both the upright sitting position (between the first and the fourth seconds) and the slump sitting position (between the 11th and the 14th seconds). A paired *t* test was then used in order to determine whether a significant difference in muscle activity existed between these two postures for each muscle. Furthermore, as the data were normalized to MVIC, the presence of FRP could be determined based on reductions in the percentage of MVIC as reported in the literature.[4,11]

Visual identification of the point at which FRP occurred was determined using the raw sEMG traces. If there was a clear sudden reduction in motor activity, this was defined as the point at which FRP occurred. At the point of FRP, the angles of the lower thoracic and lumbar spine curvatures were calculated and expressed as a percentage of the total range of motion between the upright and the slump sitting postures. The reliability of visually detecting the point of FRP was determined using the intraclass correlation coefficient (ICC) and standard error of measurement (SEM). Results for all three muscles showed excellent reliability; SLM (ICC = 0.90, SEM = 0.33 seconds), transverse fibers of the IO (ICC = 0.93, SEM = 0.46 seconds), and TES (ICC = 0.99, SEM = 0.05 seconds).

Before data processing, the spinal kinematics data were visually inspected and any trials with irregular artifact were discarded. Data were transformed *via* the matrix algebra procedures outlined by Burnett *et al*.[26] Two kinematic variables were then calculated from the converted spinal kinematics data. First, the lumbar spine curvature was defined as the angle between the tangent to the skin surface at T12 and the tangent at S2. Second, the lower thoracic curvature was defined as the angle between the skin surface at T6 and the tangent at T12.[27] The lower thoracic and the lumbar spine curvature were calculated for upright sitting and for slump sitting postures and the average value of this point for the three trials was used.

■ Results

Preliminary analysis revealed that there was no significant channel (side) effect for the trunk muscles; therefore, the left and right channels were averaged for analysis. Furthermore, the point of FRP was consistent for the left and the right trunk muscles. Thus, the average of the 3 tests of the spinal posture data for each muscle (left and right averaged) was used to calculate the values for the lower thoracic and lumbar spine curvatures.

FRP Statistically Analyzed

The FRP phenomenon was shown to consistently occur in both the SLM and the transverse fibers of the IO, as demonstrated by a significant reduction in muscle activation between the upright and slump sitting postures. For the SLM, there was a reduction of 17% (SD ± 10%) MVIC (t [23]= 8.40; $P \leq 0.001$), and for the transverse fibers of the IO there was a reduction of 11% (SD ± 16%) MVIC (t [23] = 3.20; $P = 0.004$) (Figure 2A). Conversely, the TES showed a nonstatistically significant increase in muscle activity of 2% (SD ± 6%) MVIC (t [23] = −1.25; $P = 0.221$) (Figure 2A).

Further visual inspection of the normalized TES data revealed two distinct groups within the subjects based on changes in muscle activity pattern between the upright and the slump sitting postures. Thirteen subjects demonstrated an overall increase in muscle activity of 7% (SD ± 5%) MVIC, whereas 11 subjects demonstrated a decrease of 4% (SD ± 2%) MVIC in the slump sitting position. Based on these observations, *post hoc* analysis using a two-way repeated measures ANOVA revealed a significant interaction between the two groups (onset and offset) in the upright and the slump sitting postures ($F_{1,22} = 38.65$, $P = 0.000$) (Figure 2B).

FRP Visually Analyzed

Visual inspection of the sEMG traces revealed consistent patterns of motor activity in the SLM and the transverse

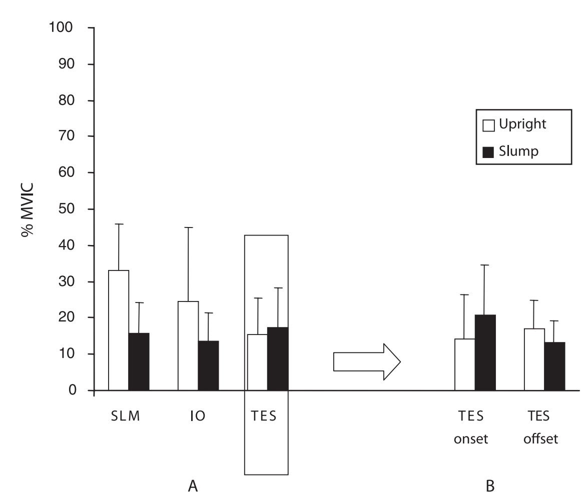

Figure 2. (**A**) Difference in sEMG activity expressed as a percentage of maximal voluntary isometric contraction between upright and slump sitting postures. Error bars indicate standard deviations. SLM = superficial lumbar multifidus; IO = transverse fibers of the internal oblique; TES = thoracic erector spinae. (**B**) *Post hoc* analysis of the difference in sEMG activity expressed as a percentage of maximal voluntary isometric contraction between upright and slump sitting postures for the thoracic erector spinae (TES).

fibers of the IO, with a reduction in baseline activity observed in both of the muscles when moving from an upright to a slump sitting posture. As for motor activity in the TES, however, four distinct patterns were found. In 7 subjects, the TES showed a clear reduction in motor activity, similar to the pattern observed with the SLM and the transverse fibers of the IO. However, the opposite was the case in 8 subjects, who demonstrated a lower level of base line in upright sitting followed by a clear increase in TES activity when moving from an upright to a slump sitting posture. Another 7 subjects exhibited a sustained discharge of motor activity above baseline when moving from an upright to a slump sitting posture, followed by either an overall increase (n = 5) or a reduction in activity (n = 2) when compared with the upright position. Two subjects had no visually detectable change in sEMG activity.

FRP and Range of Spinal Flexion

For purposes of calculating the lower thoracic spine and lumbar curvatures, the data of two subjects had to be excluded due to an artifact in the signal detected from the sensor at T12. The total range of motion was 31.5° (SD ± 9°) for the lumbar spine and 6° (SD ± 3.5°) for the lower thoracic spine. The FRP occurred in the SLM and the transverse fibers of the IO at 50% (SD ± 5%) and 65.0% (SD ± 16.5%) of the total range of lumbar spine flexion in sitting, respectively, and at 32.0% (SD ± 15.5%) and 44.0% (SD ± 14.5%) of the total range of lower thoracic spine flexion, respectively. In the subgroup of subjects with an onset in TES activity, this occurred at 31.1% (SD ± 19.9%) of total lumbar spine flexion and preceded the FRP of the SLM and transverse fibers of the IO (Figure 3). In the subgroup with FRP in the TES, this phenomenon occurred at 59.4% (SD ± 8.7%) of total lumbar spine flexion at a point within the range of the FPR of the SLM and the IO (Figure 4). In the additional two subgroups of subjects (n = 7) that displayed the sustained discharge of TES during the dynamic phase of movement the motor activity, the onset of this activity occurred at 13.0% (SD ± 2.0%) of total lumbar spine flexion and before the FRP within the SLM and the IO. This was followed by a sudden reduction in motor activity at 88.7% (SD ± 6%) close to the end of the range (Figures 5, 6)

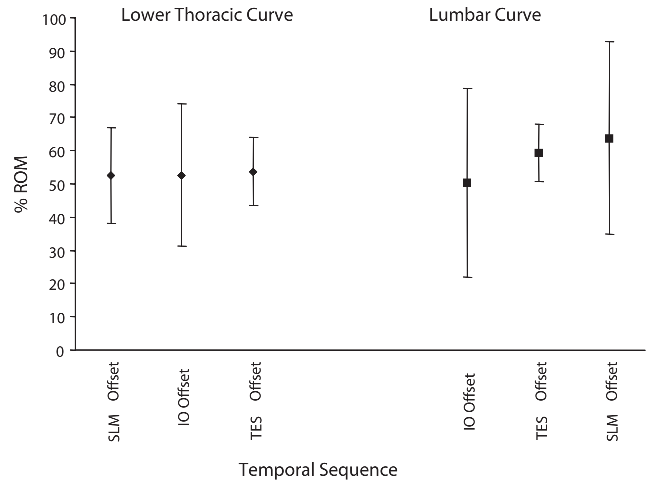

Figure 4. Temporal sequencing of muscle firing patterns of the thoracic erector spinae offset (FRP) group (n = 7) for both the lower thoracic and lumbar curves. Error bars indicate standard deviations. SLM = superficial lumbar multifidus; IO = transverse fibers of the internal oblique; TES = thoracic erector spinae.

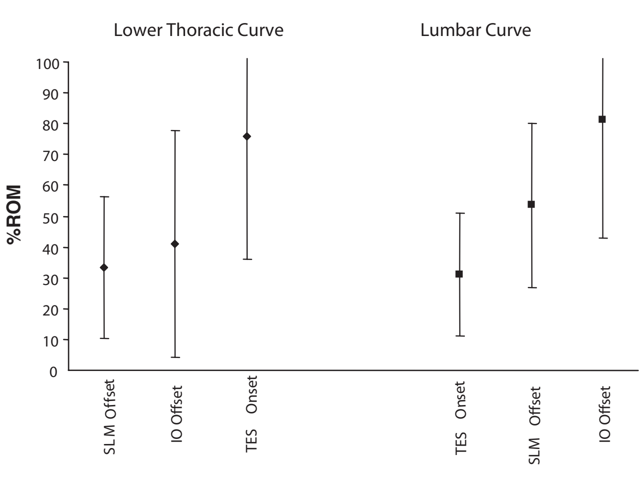

Figure 3. Temporal sequencing of muscle firing patterns of the thoracic erector spinae onset group (n = 8) for both the lower thoracic and lumbar curves. Error bars indicate standard deviations. SLM = superficial lumbar multifidus; IO = transverse fibers of the internal oblique; TES = thoracic erector spinae.

Discussion

Presence of FRP

The results of this study show that FRP occurred consistently in the SLM and transverse fibers of the IO when moving from upright to slump sitting. The presence of

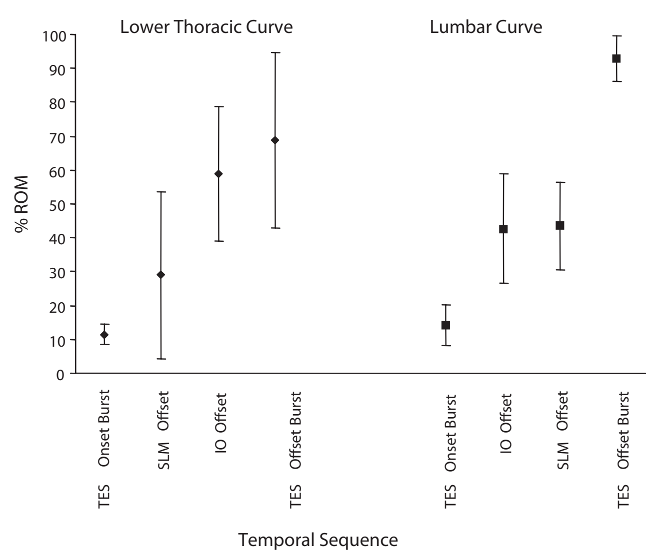

Figure 5. Temporal sequencing of muscle firing patterns of the thoracic erector spinae onset burst group (n = 5) for both the lower thoracic and lumbar curves. Error bars indicate standard deviations. SLM = superficial lumbar multifidus; IO = transverse fibers of the internal oblique; TES = thoracic erector spinae.

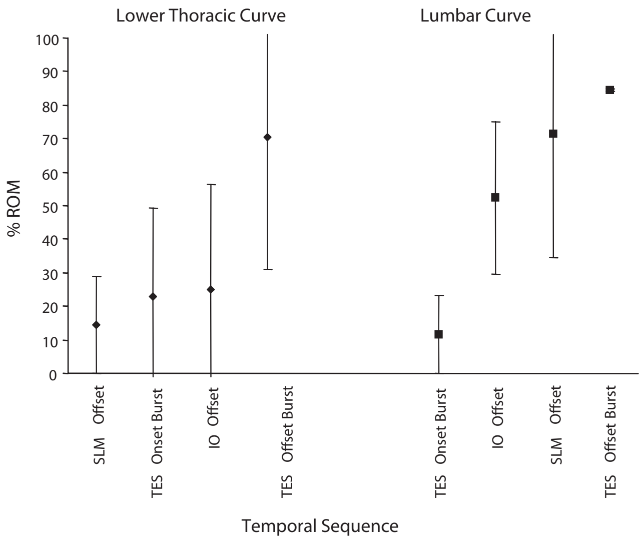

Figure 6. Temporal sequencing of muscle firing patterns of the thoracic erector spinae offset burst group (n = 2) for both the lower thoracic and lumbar curves. Error bars indicate standard deviations. SLM = superficial lumbar multifidus; IO = transverse fibers of the internal oblique; TES = thoracic erector spinae.

FRP in these muscles was confirmed by statistical analysis, visual inspection, and a reduction greater than 3% of MVIC as defined by Callaghan and Dunk[11] and McGill and Kippers.[10] This is consistent with the findings of O'Sullivan *et al*[4] who reported that the SLM and transverse fibers of the IO displayed a clear reduction in sEMG in slump compared with upright sitting. However, these findings are in contrast to Callaghan and Dunk[11] who found that FRP occurred very rarely in the lumbar erector spinae in sitting.

The apparent contrast between the findings of the current study and Callaghan and Dunk[11] may be due to differences in methodology and definition of posture between the two studies. In the current study, measuring activity from the SLM (just lateral to L5) was chosen over lumbar erector spinae (3 cm lateral to the L3 spinous process) as was carried out by Callaghan and Dunk.[11] As lumbar multifidus is considered to be an important segmental stabilizer of the lumbar spine,[28] this was deemed to be of greater clinical significance. It may be that the contrast between the two studies reflects different motor responses in the different lumbar muscles during slump sitting, where FRP of the SLM occurs in the absence of FRP in the lumbar erector spinae. This concept is supported by Danneels *et al*,[29] who showed differences in muscle activation patterns between SLM and the iliocostalis lumborum during asymmetric lifting activities. Further studies investigating the FRP responses of both muscles are needed to clarify this issue.

Another possibility is that the two studies used different methodologies for establishing upright and slump sitting positions. The present study followed the procedure described by O'Sullivan *et al*,[4] whereby achieving the upright and the slump sitting positions was largely controlled *via* the pelvis with relative relaxation of the thorax. Callaghan and Dunk,[11] on the other hand, defined slump sitting only as "rounding of the lumbar spine" with no specific definition given for "upright" sitting posture. These methodologic differences may have resulted in different activation patterns in the upright and the slump sitting postures. This concept is supported when considering the levels of muscle activity observed in the upright condition for the SLM in the current study (33% MVIC), as compared with the minimal activation of the lumbar erector (4% MVIC) and the lack of reduction in muscle activity in the slump posture in Callaghan and Dunk's study.[11] These findings may highlight the critical role of the pelvis in facilitating the lumbopelvic muscles and should the focus of further investigation.

The current study demonstrated high TES variability across all subjects. This is again in contrast with Callaghan and Dunk[11] who reported that FRP occurred in the TES of 95% of subjects at the end range of slump sitting. Analysis of the TES data in the present study, based on a change in MVIC when moving from an upright to a slump sitting posture, revealed that only 58% of the subjects had an overall FRP in the TES. The findings of an onset in TES activation among many of the subjects are similar to those of Toussaint *et al*[30] who reported that lumbar erector spinae relaxation at the end of the range of spinal flexion in standing was associated with reciprocal TES activation.

There are a number of possible reasons for these different motor patterns observed in the TES. TES is a large torque producer with its origin on the pelvis and insertion on the thorax, allowing it to act on the pelvis, lumbar, and thoracic spine.[31] This provides the TES with a greater potential for variability in motor patterning[30] when compared with muscles such as the transverse fibers of IO and the SLM, which have a more local action on the lumbopelvic region.[28,32] Another possibility is that the different patterns of TES activation reflect differences in anthropometric factors such as relative spinal length, although *post hoc* analysis of the TES onset and offset groups revealed no difference in their height which tends to discount this. The five different patterns observed in the TES when moving from an upright to a slump sitting position in the current study may reflect different inherent motor control strategies adopted by the subjects tested. The differences in TES activity between the current study and that of Callaghan and Dunk[11] are difficult to interpret. This may again relate to the methodologic differences between the studies outlined previously. The interaction effect observed between TES offset and onset groups suggests different strategies of TES muscle activation between the groups in the upright and slump conditions. In the offset group, a higher mean level of TES activity was observed in the upright condition with a FRP in slump sitting, with the opposite finding observed in the TES onset group.

The sustained discharge of TES muscle activity observed in 7 of the subjects during the movement phase from upright to slump sitting is similar to that previously described in studies investigating flexion in standing.[33] However, no random spasms or spikes of EMG of the TES, SLM, or TES were observed during the end-range flexion phase of the procedure as previously reported by Olson *et al*[33] and Solomonow *et al*.[34] The reason for this appears to also relate to methodologic factors. Olson *et al*[33] reported "spasms" of EMG activity in the lumbar paraspinal muscles at the end-range flexion phase of flexion in standing following deep cyclical loading of the lumbar spine into flexion every 10 seconds over a 9-minute period.[33] Solomonow *et al*[34] reported similar findings following a 10-minute period of sustained end-range flexion in sitting. These bursts of motor activity were considered to be an indication of microdamage to the viscoelastic tissue.[33,34] In the current study, subjects were only moved from upright to slump sitting on three occasions with a 30-second rest between each test session; a procedure unlikely to stress the spine at end-range flexion.

The transverse fibers of IO have been previously reported to provide a stabilizing role to the sacroiliac joint.[32] The consistent FRP exhibited in the transverse fibers of IO when moving from upright to slump sitting posture may reflect a reduction of motor activity as result of load transfer to the passive system of the sacroiliac joints as previously proposed by Snijders *et al*.[35]

FRP and Range of Spinal Flexion

It was our initial hypothesis that FRP would occur toward the end range of the available spinal flexion in sitting, as previously demonstrated in standing.[6,10,11,22] However, the present study found that FRP occurred in the SLM and the transverse fibers of the IO at mid-range of spinal movement from an upright to a slump sitting posture. This finding suggests a different underlying motor control mechanism for FRP in sitting as compared with standing. It appears that in sitting, rather than it being a mechanism of load transfer to the passive system or other active structures at the end of range, there is a transfer of load to other muscles controlling the thoracolumbar spine at mid-range. This concept is supported by the data demonstrating either an increase in or a sustained discharge of motor activity in the TES ($n = 13$) that precedes the offset of the SLM and the transverse fibers of the IO at mid-range of flexion during the movement phase towards the slump position. It thus appears that the control over the extensor moment for the lumbar spine is transferred from the local muscle system (represented by the SLM and the IO) to the global muscle system (represented by the TES) at the mid-range of spinal flexion. In the other cases ($n = 7$) where FRP of the SLM and the transverse fibers of the IO occur in conjunction with FRP of the TES, this also occurred at mid-range. In these subjects, it is possible that the control of the thoracolumbar spine at this point in range was maintained by other muscles not measured in this study such as deep fibers of LM, and/or iliopsoas and/or quadratus lumborum).

These observed findings could represent a "switching" phenomenon described previously whereby the extension torque usually provided by the lumbar muscles is transferred to the TES[30] or to other trunk muscles during the movement phase of the spine towards flexion. The findings suggest that the TES functions either independently or synergistically with the SLM and the transverse fibers of the IO in controlling lumbar flexion in sitting. These findings highlight the variable and complex nature of motor control over the thoracolumbar spine and pelvic region and the close interrelationship between the different spinal muscles in controlling the extensor moments of the spine.[36] It is important to note that as this study used sEMG it only provided an insight into the superficial trunk muscles. These results cannot be extrapolated to deep spinal muscles such as the deep fibers of LM, transverses abdominus (although cross talk form these muscles may have occurred), and psoas but should provide the basis for further research.

These findings clearly show that it is not possible to extrapolate the motor control strategies known to control the thoracolumbar and pelvic region during spinal flexion in standing to those used in sitting. This may reflect a different motor control mechanism for FRP in the thoracolumbar and pelvic region in sitting *versus* in standing reflected in the different loading demands on the spine in a slump sitting compared with flexion in standing. These differences may also reflect the altered orientation of the hips and pelvis and the potential changes in the length tension association of the trunk muscles and their active/passive load contributions at different lengths in sitting when compared with standing.

Clinical Implications

The present study supports previous research suggesting that sitting with a neutral lordosis results in activation of the SLM and the transverse fibers of the IO.[4] Conversely, semiflexed and slump sitting postures result in FRP of the transverse fibers of the IO and the SLM with variable patterns of activation in the TES. These findings may have clinical significance given that maintenance of the lumbar lordosis has been reported to reduce low back and leg pain.[37] Recent studies have demonstrated an association between flexion-related low back pain and habitually assuming end-range flexed spinal postures.[38–41] One of those studies showed a correlation between sustaining end range of slump sitting postures and reductions in back muscle endurance.[38] Recent research has documented an absence of FRP in sitting, in the LM in specific chronic low back pain populations,[42] Furthermore dysfunction of the spinal stabilizing muscles such as the transverse fibers of the IO[43] and the SLM[29,43] is commonly reported in CLBP patients. The findings of the current study might suggest that habitually sustaining not only fully flexed but also semiflexed sitting spinal postures (that result in relaxation of important spinal stabilizing muscles) for long periods may result in deconditioning of these muscles, leaving individuals at greater risk of back injury or provocation of existing back pain. On the other hand, neutral lordotic postures facilitate tonic activation of the spinal stabilizing muscles, which supports the concept of postural retraining in the management of specific low back pain disorders.[44] Further research is required to further explore these issues.

Conclusion

The results of this study suggest that activation of key lumbopelvic stabilizing muscles is linked closely to posturing of the lumbopelvic region in upright sitting. Both the SLM and the transverse fibers of the IO were shown to exhibit FRP from mid- to end-range flexion in sitting. Alternatively, when moving in to a slump sitting posture, there is frequent activation of the torque producing TES, which would result in a large compressive penalty on the lumbar spine.[31] These findings support the importance of specific sitting postures to facilitate reflex activation of the spinal stabilizing muscles of the lumbar spine, which may bear clinical significance.

Key Points

- FRP occurred consistently in the SLM and the transverse fibers of the IO when moving from an upright to a slump sitting position.
- TES motor activity is highly variable between individuals, with several different patterns of activity displayed.
- FRP occurred from mid-range until end-range spinal flexion in sitting, which suggests the transfer of load to other active structures such as the TES.
- Motor control strategies of spinal flexion previously reported during standing are different from those used during sitting.

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