ORIGINAL ARTICLE

Postural Control of the Lumbar Spine in Unstable Sitting

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ABSTRACT. Preuss RA, Grenier SG, McGill SM. Postural control of the lumbar spine in unstable sitting. Arch Phys Med Rehabil 2005;86:2309-15.

Objective: To evaluate the neuromuscular strategy adopted during sitting balance on an unstable surface in the frontal plane.

Design: Electromyographic evaluation of trunk muscles.

Setting: University spine biomechanics laboratory.

Participants: Seventy asymptomatic men (mean age, 34.5y).

Interventions: Not applicable.

Main Outcome Measures: "Balancers" and "nonbalancers" were identified by principal component analysis of their lumbar spine side flexion angle during sitting balance. Average electromyographic levels were used as a measure of muscle activation. Pearson correlations were used to identify coactivation versus asymmetrical muscle activation of opposite muscle groups.

Results: External oblique, internal oblique, and thoracic erector spinae (TES) were most active, and most likely to be used asymmetrically, with other muscles showing low levels of coactivation. Between groups, the average electromyographic levels in the balancers was lower than in the nonbalancers ($P<.05$), with further differences in the symmetry of external oblique, internal oblique, and TES activation between groups.

Conclusions: Sitting balance in the frontal plane appears to involve a combined feedforward-feedback strategy of muscle activation. Successful balance was characterized by low levels of muscle coactivity, along with higher levels of asymmetric activation in the global trunk muscles, specifically external oblique, internal oblique, and TES.

Key Words: Electromyography; Equilibrium; Lumbosacral region; Motor activity; Rehabilitation.

THE STABILIZING SYSTEM of the spine, as described by Panjabi,[1] is divided into 3 subsystems: a passive (osteoligamentous) system, an active (musculotendinous) system, and a neural control system. In the absence of the latter 2 systems, the lumbar spine will buckle at compressive loads of less than 100N.[2-4] The addition of neuromuscular activity (representing the combined action of the neural and musculotendinous systems), however, allows the spine to remain stable under loads of several thousand newtons.[4,5] Two potential neuromuscular strategies have been suggested to achieve this goal.[6] The first involves the use of feedforward levels of muscle stiffness, adequate to maintain the stability of the spine like a Euler column (a spine-stiffening strategy). The second involves a combined feedforward-feedback approach in which low levels of spine stiffness, sufficient only to thwart immediate spine buckling, are maintained by feedforward commands (aided by the mechanical properties of the muscles themselves) but with feedback control required to maintain spine stability in the event of a perturbation.

In standing, with a stable base of support (BOS), a strategy of spine-stiffening through muscle coactivation has been observed in response to changes in static spine stability.[7] Under dynamic conditions, however, such as when the subject's BOS is not stable, such a strategy of spine stiffening is unlikely to be effective if the trunk musculature is to be involved in the maintenance of postural control and balance as well as spine stability. Under such conditions, the combined feedforward-feedback strategy would appear to be more effective. One means to test this would be through an evaluation of sitting balance, which can challenge both the postural control and stability of the trunk and spine, and in particular of the lumbar spine, by limiting the role of the lower extremities in the maintenance of postural control.[8] In a recent study, Radebold et al[9] found that poor trunk postural control while sitting on an unstable surface correlated with delayed muscle response to sudden loading of the trunk. These authors hypothesized that these deficits may be the result of decreased spine proprioception, potentially impairing the dynamic postural control and stability of the spine and placing these subjects at greater risk for injury. This previous study did not, however, address any differences in the neuromuscular strategies adopted during the balance task itself by those subjects who were more and less adept at the task.

The purpose of this study, therefore, was to evaluate the neuromuscular strategy adopted in the lumbar spine during sitting balance on an unstable surface and to compare the strategies used by subjects who were more or less adept at maintaining balance and trunk postural control under this dynamically unstable condition. We hypothesized that, during successful sitting balance, a combined feedforward-feedback strategy would be observed, characterized by low-level muscle cocontraction, and higher asymmetric activations in response to the postural challenges of the task.

METHODS

Participants

Sitting balance was tested in 70 men (mean age, 34.5y; range, 20–51y) employed in manual labor positions at the time of testing. Subjects were included if they had no low back pain at the time of testing and had no contraindications to participation in a variety of physical fitness and back fitness tests that were run in conjunction with this study.[10,11] The study received ethics approval from the Office of Research Ethics at the University of Waterloo. All subjects gave informed consent before testing.

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Table 1: Electrode Placement for Electromyographic Recording of the Trunk Musculature

Muscle	Surface Electrode Placement
Rectus abdominis	3cm lateral to the umbilicus (caudal bead of rectus abdominis)
External oblique	15cm lateral to the umbilicus
Internal oblique	1cm medial and caudal to the ASIS
Latissimus dorsi	Lateral to T9, slightly medial to the axillary line
TES	3cm lateral to T9
LES	3cm lateral to L3
Lumbar multifidus	Immediately lateral to L5

Abbreviation: ASIS, anterior superior iliac spine.

Data Collection

Sitting balance was tested in high sitting on a 1 degree of freedom (*df*) rocker board,[a] with the subject's feet unsupported. The board was aligned with its axis in the anteroposterior direction, free to rotate in the frontal plane. Lumbar spine orientation was measured by using a 3SPACE electromagnetic, 6 *df* tracking instrument,[b] sampled at 30Hz. Two sensors were fixed to the subject's skin over the S1 and T12 vertebrae by using SkinBond adhesive.[c] The transmitter was fixed to a stationary wooden mount nearby. The orientation of the T12 sensor relative to the S1 sensor was determined, following orthopedic convention (flexion-extension, lateral bending or side flexion, and axial rotation calculated in this order[12]), by using a matrix rotation algorithm based on the orientation of each sensor relative to the transmitter.

Surface electromyographic recordings were taken bilaterally from 7 trunk muscles: the rectus abdominis, external oblique, internal oblique, latissimus dorsi, thoracic (TES) and lumbar erector spinae (LES), and lumbar multifidus. Surface electromyographic data were acquired by using Ag-AgCl electrodes in a bipolar configuration. Electrodes were positioned less than 4cm apart and parallel to the muscle fibers after careful skin preparation. Electrode placement for each channel is given in table 1. Electromyographic data were gathered and amplified by using a 10 to 500Hz bandwidth electromyographic amplifier with common mode rejection ratio of 80dB at 60Hz, digitally sampled at 1024Hz, and stored along with the 3SPACE recordings, for further analysis.

Subjects underwent 2 balance trials, beginning with them resting on one side of the rocker board, with their arms crossed on their shoulders. At the "go" command, the subjects were instructed to bring the board to the upright position, so that they were sitting as straight as possible, and to maintain this position for 30 seconds.

Data Processing and Analysis

Histograms of the individual subject lumbar spine side flexion angles were generated over the 30-second trial duration, with 41 bins ranging from $-20°$ to $20°$ of side flexion. Principal component analysis (PCA) was performed on the resulting histograms, by using singular value decomposition,[13] to identify common balance patterns over the sample population. Principal components were selected for each trial from the resulting factor loading matrices, explaining a cumulative percentage total variation within the data of 80% or more (factors below this cutoff each explained $<2\%$ of the overall data variance). Sitting balance, for each trial, was also quantified by using 2 other measures: the mean absolute lumbar spine side flexion angle over the 30-second duration of the trial (MSF) and the standard deviation (SD) of the lumbar spine side flexion angle over the 30-second balance trial (SDSF).

To compare those subjects who were more or less adept with the balance task, subjects were classified as "balancers" or "nonbalancers" based on their factor loadings from the first, "novel" trial PCA. A comparison of MSF between the 2 groups, performed by using a paired sample *t* test, was then used as a means to validate the subject groupings.

Raw electromyographic data were full wave rectified and low-pass filtered at a cutoff frequency of 2.5Hz by using a second-order digital Butterworth filter. The resulting linear-enveloped electromyographic (LEMG) data for each muscle was then normalized to a similarly processed maximum voluntary contraction (MVC) for each muscle taken before testing (this procedure has been described in detail by McGill[14]). The normalized LEMG was used for all subsequent analysis. The average level of muscle activation (AEMG=integrated LEMG divided by the duration of the trial) for each trial was taken to be representative of the level of muscle activity during that trial. The SD of the LEMG (SDEMG) was used as a measure of the steadiness of the muscle contraction throughout the balance trial.

A between-group comparison of the balancers and nonbalancers was conducted for each muscle (both AEMG and SDEMG) by using paired samples *t* tests. Further, a comparison between the 2 balance trials, for the entire subject population, was conducted. The quantitative measures of sitting balance (MSF, SDSF) were compared by using paired samples *t* tests. Pearson correlations were conducted on the MSF and SDSF values from trials 1 and 2 for the entire subject population to determine if any changes between trials were consistent across the population. Analysis of variance (ANOVA), with Tukey post hoc analysis, was used to compare the AEMG of the trunk muscles tested during balance to determine which muscles were most active during the balance trials. Pearson correlations of the LEMG of opposite muscle groups were performed to determine if the right and left trunk muscles were coactivated or asymmetrically activated during balance. Paired sample *t* tests were used to compare AEMG and SDEMG for individual muscles between the first and second balance trial. Before the parametric analyses of the AEMG described previously (*t* tests, ANOVA), a log transformation of these data was conducted; a Kolmogorov-Smirnov test indicated that these data were not normally distributed. All other data were found to satisfy the criterion of normality for parametric statistical analysis.

Finally, ANOVA, with Tukey post hoc analysis, was used to compare the age, height, and weight of the balancers, the nonbalancers, and the unclassified subjects. For all statistical analyses, an α level of .05 was used.

RESULTS

PCA revealed 4 common balance patterns for the first balance trial. Three of these patterns were identified as unimodal, based on the presence of a single major peak. The remaining pattern was identified as bimodal, based on the presence of 2 distinct peaks. Subjects with 60% or more of their factor loading falling into 1 of the 3 patterns classified as unimodal were grouped as balancers (n=37), whereas subjects with 60% or more of their factor loading falling into the pattern classified bimodal were grouped as nonbalancers (n=20). Comparison between groups, for the first trial, revealed a significantly larger MSF for the nonbalancers than for the balancers (7.9° vs 4.3°, $P<.001$). For the second balance trial, although no distinct bimodal pattern of balance was identified, the MSF for the nonbalancers identified in the first trial remained significantly

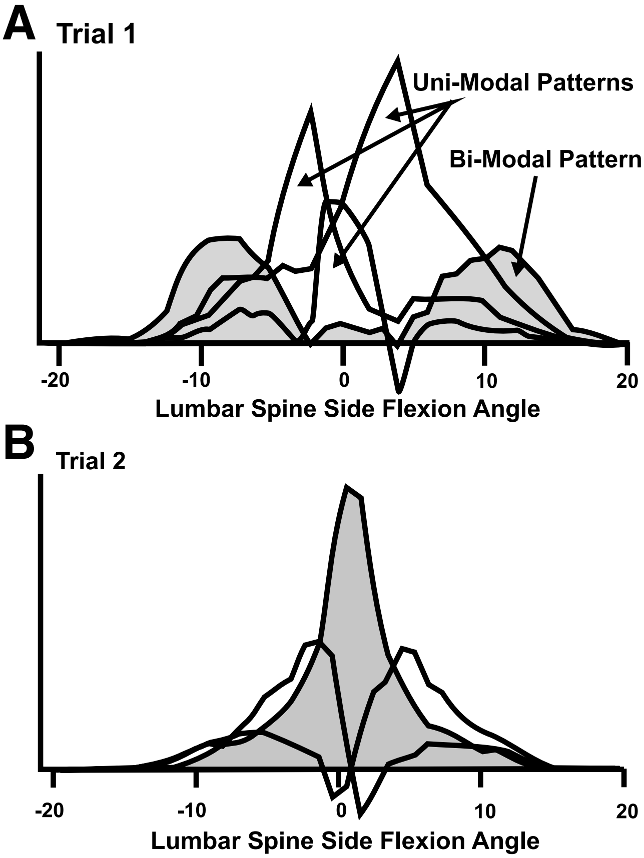

Fig 1. Common balance patterns for (A) trial 1 and (B) trial 2 derived from PCA.

larger than that of the balancers (6.7° vs 3.3°, $P<.001$). The remaining subjects (n=13) could not be clearly grouped based on their factor loadings (no single factor loading ≥60%) and were not considered in the between-group comparison. The common balance patterns for both trials, derived from PCA, are presented in figure 1. The results of 2 individual balance trials, representing examples of unimodal and bimodal balance patterns, are shown in figure 2.

Comparison of individual muscle activity between balancers and nonbalancers revealed significantly higher ($P<.05$) levels of muscle activation (AEMG) for the nonbalancers in 12 of 14 muscles tested for trial 1 and in 11 of 14 for trial 2. A similar trend was observed for the measure of muscle steadiness (SDEMG), with the nonbalancers tending to have a greater SDEMG than the balancers. This difference proved significant, however, for only 4 of 14 muscles in the first trial and 8 of 14 in the second. The results of these analyses are presented in table 2.

The between-trial comparison for the entire subject population revealed a significant improvement ($P<.001$) in both quantitative measures of balance (MSF, SDSF) from the first to the second trial, supported by the absence of a bimodal balance pattern in the PCA of trial 2. In addition, a relatively good correlation was found between the balance scores from the 2 trials (MSF $r=.805$, SDSF $r=.823$), indicating a general trend toward improvement in balance across the entire sample population. The distribution of sitting balance scores (MSF, SDSF) for both trials is shown in figure 3. Further, a significant decrease in AEMG ($P<.05$) was found bilaterally in all muscles tested, with a similar decrease in SDEMG ($P<.05$) observed bilaterally in the external oblique, internal oblique, TES, lumbar multifidus, and the right LES.

Tukey post hoc analysis of the ANOVA results revealed the TES, external oblique, and internal oblique bilaterally to have the highest activation levels (AEMG) during both trials (fig 4). Pearson correlations between LEMG of opposite muscle groups (right vs left) also revealed the largest number of negative correlations in these 3 muscles (as well as in the LES), whereas the rectus abdominis, latissimus dorsi, and lumbar multifidus had both low AEMG and positive correlations between right and left sides (table 3). Balancers, however, showed a tendency toward greater asymmetric activation of the external oblique, whereas nonbalancers had greater asymmetric activation of the internal oblique and TES. This trend was present in both trials, becoming somewhat more pronounced in trial 2 (see table 3).

No significant differences in height or weight ($P=.26$, $P=.45$, respectively) were found between the balancers (mean, 1.78m; mean, 83.9kg), the nonbalancers (mean, 1.82m; mean, 88.4kg), and the unclassified subjects (mean, 1.81m; mean, 86.9kg). The ANOVA did, however, reveal a significant main effect of age between the 3 groups ($P=.03$), with the Tukey post hoc analysis revealing the largest difference between the balancers (mean, 32.7y; range, 20–49y) and the nonbalancers (mean, 39.3y; range 21–51y). The mean age of the unclassified subjects was the same as the mean age of the entire subject population (mean, 34.5y; range, 23–46y).

DISCUSSION

The purpose of our study was to evaluate the neuromuscular strategy used during sitting balance on an unstable surface: a task that challenges both the postural control and stability of the trunk and lumbar spine. We had hypothesized that, although a spine-stiffening strategy might be used in response to challenges to static spine stability, a combined feedforward-feedback strategy in the lumbar musculature would be required in response to the dynamic challenge of sitting balance.

Our results appear to support this hypothesis. Three muscles, the external oblique, internal oblique, and TES, were identified as having the highest levels of activation during the balance task. These 3 muscles were also most likely to be active in an asymmetric manner, suggesting a degree of feedback control in response to the postural challenges of the task. In contrast, the rectus abdominis, latissimus dorsi, and lumbar multifidus showed lower levels of overall activity, with an activation

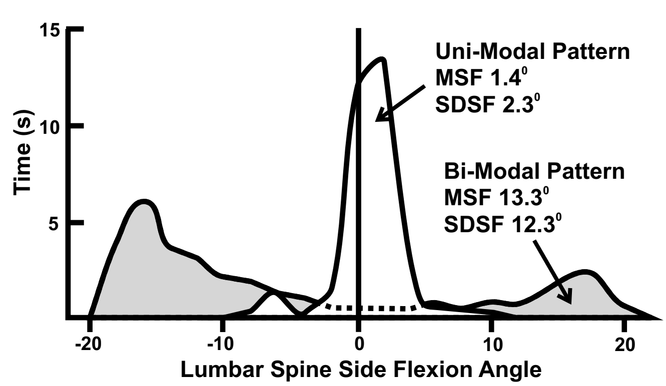

Fig 2. Distributions of lumbar spine side-flexion angle during sitting balance for 2 subjects, representing unimodal and bimodal balance patterns.

Table 2: Comparison of Individual Muscle Activity Between Balancers and Nonbalancers

	Trial 1				Trial 2			
	Mean AEMG		Mean SDEMG		Mean AEMG		Mean SDEMG	
Muscle	Balancers	Nonbalancers	Balancers	Nonbalancers	Balancers	Nonbalancers	Balancers	Nonbalancers
Right								
RA	3.05	3.22	1.22	0.92	3.24	2.82	1.28	0.90
EO	5.66*	8.80*	3.15*	3.99*	5.05*	7.95*	2.63*	3.60*
IO	6.93*	9.29*	4.65	5.13	5.81*	7.90*	3.53	4.17
LT	3.06*	4.49*	1.26	1.53	2.88*	4.79*	1.01*	1.57*
TES	6.39*	8.10*	2.29*	2.88*	5.78*	8.18*	1.93*	2.67*
LES	2.68*	4.39*	1.42*	2.23*	2.46*	4.59*	1.15*	1.87*
MF	3.21*	5.59*	1.91	2.35	2.66*	5.41*	1.46	2.20
Left								
RA	2.88*	3.81*	1.23	1.07	2.89	3.37	1.13	1.04
EO	5.74*	9.01*	3.21	3.72	5.04*	8.20*	2.68*	3.54*
IO	7.26*	9.20*	4.72	5.84	6.33*	8.38*	3.84	5.05
LT	3.80*	4.90*	1.69	1.98	3.67	4.20	1.63	1.72
TES	7.22	8.17	2.68	3.31	6.38*	8.37*	2.15*	2.99*
LES	2.95*	5.56*	1.81	2.35	2.55*	4.90*	1.36*	2.17*
MF	4.09*	5.62*	1.73*	3.15*	3.16*	5.72*	1.26*	2.11*

Abbreviations: EO, external oblique; IO, internal oblique; LT, latissimus dorsi; MF, lumbar multifidus; RA, rectus abdominis.
*Indicates a significant between-group difference ($P<.05$).

pattern indicative of low-level cocontraction, likely modulated by feedforward commands. LES, on the other hand, tended toward lower levels of activation but was still found to be active in an asymmetric manner in several subjects. These findings are in keeping with the architecture of the lumbar spine, given the balance task in the frontal plane.

Thirty-seven of the 70 subjects were classified as balancers, while 20 were classified as nonbalancers, based on PCA analysis of spine kinematics during the first balance trial (the remaining 13 subjects did not fall clearly into 1 group or the other). A comparison of the neuromuscular activity during the balance task revealed important between-group differences. The balancers tended to have lower levels of muscle activation (AEMG) during the balance task, including the 3 muscles identified as being most active in the task (external oblique, internal oblique, TES), with an overall trend toward more stable muscle activation (SDEMG), particularly in the second trial (see table 2). Further, a general trend toward lower levels of trunk muscle activation (AEMG) was noted for the entire sample population, from the first to the second trial, which coincided with a general improvement in balance as the subjects became more familiar with the task. This suggests that higher levels of muscle activation, as have been observed in response to challenges to static spine stability,[7] do not occur under dynamically unstable conditions. On the contrary, increased success during the sitting balance task was accompanied by lower levels of muscle activation, suggesting an increased reliance on feedback control to maintain spine stability and postural equilibrium.

Differences were also noted in the patterns of activation of the external oblique, internal oblique, and TES between the balancers and the nonbalancers, with the former tending toward more asymmetric activation of the external oblique and less of the internal oblique and TES, and the latter showing the opposite tendency (see table 3). Because all 3 of these muscle groups are important stabilizers, and movers, of the lumbar spine, a conclusive explanation for this difference in neuromuscular strategy between the 2 groups is not immediately apparent. Our recent work has suggested that additional stiffness and stability is achieved through a binding effect when the

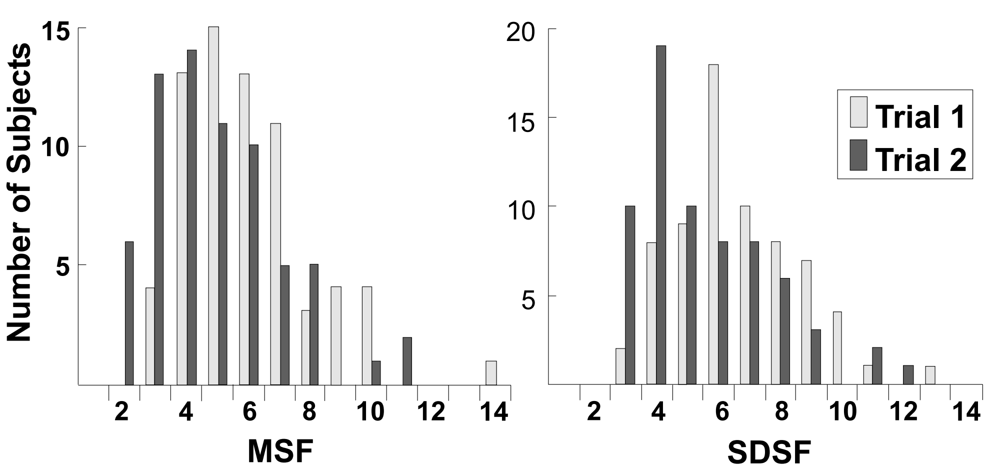

Fig 3. Binned histograms of balance data showing the distribution of quantitative sitting balance scores for the 2 trials.

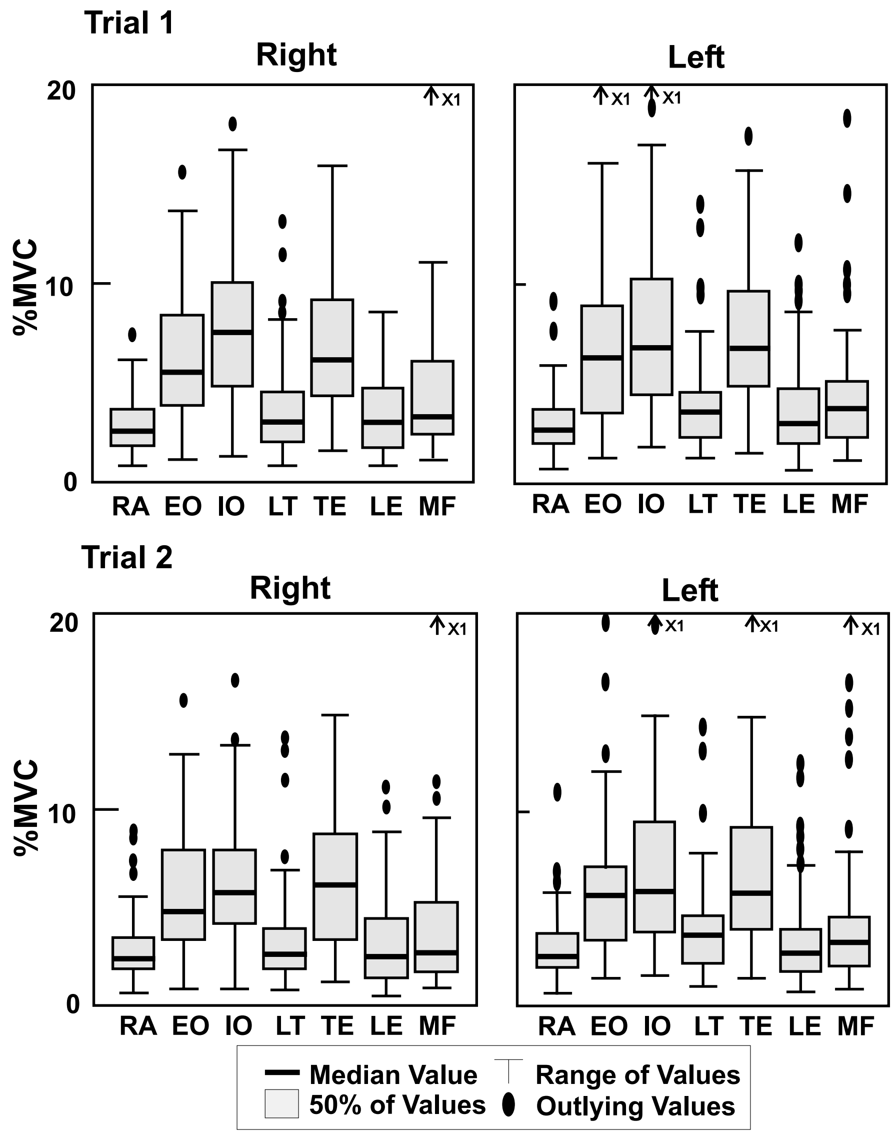

Fig 4. Average levels of electromyographic activity during the 2 balance trials, in the 14 trunk muscles tested. Abbreviations: EO, external oblique; IO, internal oblique; LE, lumbar erector; LT, latissimus dorsi; MF, lumbar multifidus; RA, rectus abdominis; TE, thoracic erector.

3 layers of the abdominal wall are activated together.[15] The neuromuscular strategy used by the balancers may therefore reflect a similar synergistic pattern of muscle activation, providing a more efficient mix between postural adjustments and the maintenance of spine stability. It has also been suggested that the internal oblique may assist in the stabilizing action of the transversus abdominis (tensioning the thoracolumbar fascia), particularly at the lower lumbar levels.[16] Similarly, the TES has a very close relation with the LES and to a lesser extent the lumbar multifidus, both of which can act as local stabilizers of the lumbar spine[6] because of their direct attachments to the lumbar vertebrae. Given the potential synergic actions of the internal oblique and TES with local stabilizers of the lumbar spine, the neuromuscular strategy preferred by the bal-

Table 3: Asymmetric Activation of Muscles as Indicated by Negative Correlation for Bilateral Muscle Activation

		Muscle						
Trial		RA	EO	IO	LT	TES	LES	MF
1	All Subjects	0.0 (.883)	51.4 (.036)	40.0 (.134)	2.9 (.641)	50.0 (−.056)	47.1 (−.016)	27.1 (.390)
	Balancers	0.0 (.893)	59.5 (−.069)	35.1 (.238)	0.0 (.726)	43.2 (−.014)	43.2 (−.018)	21.6 (.505)
	Nonbalancers	0.0 (.862)	35.0 (.264)	50.0 (−.027)	0.0 (.577)	50.0 (−.017)	40.0 (.036)	30.0 (.285)
2	All Subjects	0.0 (.897)	59.4 (−.055)	39.1 (.179)	4.3 (.630)	40.6 (.038)	37.7 (.076)	15.9 (.525)
	Balancers	0.0 (.912)	70.3 (−.132)	24.3 (.333)	2.7 (.717)	29.7 (.153)	37.8 (.097)	5.4 (.671)
	Nonbalancers	0.0 (.850)	35.0 (.150)	55.0 (−.019)	0.0 (.537)	55.0 (−.078)	30.0 (.112)	25.0 (.384)

NOTE. Values are percentage of subjects with negative correlations (mean *r* value).

ancers—that of asymmetric activation of the external oblique more than the internal oblique and TES—may be more beneficial for maintaining both spine stability and postural equilibrium in unstable sitting.

Our findings support a strategy in which a certain level of spine stiffness is maintained to prevent outright and immediate spine buckling but with feedback control responsible for the maintenance of global postural control and spine stability. The presence of asymmetrical activation patterns in the external oblique, internal oblique, and TES, the 3 most active muscles during sitting balance, indicates that these muscles must be frequently used to meet the postural perturbations that occur in this unstable sitting condition and must therefore be largely modulated by feedback control. The more stable, symmetric activation patterns of the other trunk muscles recorded, however, indicate that a certain level of trunk stiffness is likely maintained throughout the balance task. This is further apparent in the decrease in SDEMG between trials, implying a more stable level of muscle activation (along with the general decrease in AEMG) as the subjects became more familiar with the task.

These data also indicate that, although a combination of spine stiffness and feedback modulated postural responses are used during unstable sitting, certain muscle activation patterns may be more appropriate or successful than others. In particular, the balancers were more likely to use the external oblique, rather than the internal oblique or TES, in an asymmetric manner, likely to meet the perturbations to posture and balance that occurred during the trials. The nonbalancers, on the other hand, were more prone to using the internal oblique and TES in an asymmetrical manner for balance, limiting the possible synergic spine stabilizing action at the local level of these muscles.

Unlike previous studies,[8] the present study found no significant link between sitting balance and subject height or weight, although a statistically significant difference was found between the age of the balancers and nonbalancers. It is possible that the nonbalancers, who were on average slightly older, developed less efficient balance strategies over time, possibly as a result of age-related decreases in spine proprioception, as have been shown in the peripheral joints.[17] It is questionable, however, whether the small difference in age (6.6y) between the balancers and nonbalancers should have any important biologic significance. As such, this difference may simply represent an anomaly resulting from the study design, which was not intended to test for the effects of aging on sitting balance. This difference in age between the 2 groups does, however, warrant further study.[18]

As with all studies, certain limitations must be addressed. Previous studies of sitting balance[8,9] used the path of the center of pressure (COP) to analyze the success of the balance task, as is typical of studies of standing balance. We chose to use the kinematics of the lumbar spine as our measure of task success because we were interested primarily in sitting balance as a measure of postural control in the lumbar spine. As such, we believed it to be more relevant to these data to monitor the kinematics of the spine itself, rather than the movement of the COP, even though this latter measure may provide a more accurate representation of balance. Second, these previous studies have evaluated sitting balance in 2 *df*, rather than limiting the balance task to the frontal plane, as was done in this study. Although the spine is free to rotate with 3 *df*, Gardner-Morse et al[5] have identified "lateral bending, with a small amount of torsional twisting" as a primary buckling mode of the lumbar spine. This suggests that sitting balance in the frontal plane provides a reasonable challenge to the postural control and stability of the lumbar spine. Further, limiting the balance task to the frontal plane has the advantage that, in upright sitting, with the thighs supported, the subject's weight is relatively evenly distributed in this plane, eliminating the need for significant isometric contraction to maintain a static position, which may have complicated analysis of these findings if movement had also been allowed in the sagittal plane, in which weight distribution is uneven, relative to the spine, in the sitting position. This study also made use of surface electrodes to acquire electromyographic data, providing a "global" view of muscle activation. Because of the proximity and overlap of many trunk muscles, electrode sites were named for convenience and cannot be said to preclude crosstalk from adjacent muscles. For example, the signal collected at the lumbar multifidus site, medial at the level of the L5 vertebra, may include some background signal from muscles such as the longissimus. Further, the use of electromyographic data normalized to MVC has several assets and liabilities—one of the liabilities being the motivation to achieve an MVC. It is possible, although unlikely, that the nonbalancers possessed different motivational factors. If this was the case, it would introduce a potential confounding factor in the quantitative comparison of AEMG between groups. Finally, it should be noted that those subjects classified as nonbalancers were more likely than the balancers to contact the sides of the rocker board with the support surface. It is possible that any external contacts may have had some influence on the neuromuscular strategy used by the subjects in the time around the contact and may therefore be reflected in our global measures of neuromuscular activity.

CONCLUSIONS

Our findings support the hypothesis that a strategy of feedforward spine stiffening does not accompany successful postural control in unstable sitting—a dynamic challenge to sitting balance. Rather, a combined feedforward-feedback strategy appears to be used. This implies that dynamic spine stability must rely on feedback control of the trunk musculature, from proprioceptive and other sources of afferent feedback, rather than exclusively relying on feedforward mechanisms to maintain spine stiffness. As such, it may be inadvisable for clinicians to limit treatment of lumbar spine instabilities to stabilization exercises designed to maintain isometric muscle contractions in a static, neutral spine posture. We recommend that treatment programs be progressed to include dynamic exercises in which muscles are trained to respond to perturbations to spine posture and stability, rather than simply trying to anticipate them.

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Suppliers

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