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Timing of Activation of the Erector Spinae and Hamstrings During a Trunk Flexion and Extension Task

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Study Design: Timing of activation of the hamstrings and erector spinae was assessed using surface electromyography.

Objectives: To investigate the influence of posture and movement speed during trunk flexion–extension on the flexion–relaxation response and trunk muscle activation patterns.

Summary of Background Data: The literature contains numerous reports on coactivity and synergistic behavior of major muscle groups during trunk flexion–extension. There are few reports on the timing of muscle activation.

Methods: Six subjects were recruited for a training session and six biweekly test sessions. Ten surface electromyogram electrodes and a lordosimeter were used to record timing of lumbar motion and muscle recruitment in the hamstrings and at four sites in the thoracolumbar region. A 3×2 within-subject factorial design was used to test the effects of posture and speed on activation patterns.

Results: Patterns of muscle activation were found to be dependent on posture and the direction of movement. The flexion–relaxation response was pervasive in the lumbar region but was less consistent at the T9 and hamstring sites. Significant differences in the delay between electromyogram activation and lumbar motion were found for the standing postures at initiation of extension, in which activation progressed in the caudad-to-cephalad direction.

Conclusions: The flexion–relaxation response is ubiquitous in the lumbar erector spinae and is present in the hamstrings and lower thoracic erector spinae, although not consistently in all subjects. In standing, timing of activation differed significantly by site in extension but not in flexion. Muscle activation patterns and flexion–relaxation were consistent over six biweekly test sessions. [Key Words: coordination, electromyogram, extension, flexion, flexion–relaxation, lumbar] **Spine 2001;26:418–425**

Functional human activity often involves repeated motions. Skills such as walking, eating, bending, and reaching evolve during development until the fundamental patterns can be evoked with minimal cognitive oversight and can be performed with a minimum of variation.[18] Control of functional movement patterns may involve synergies of muscle activity that are rooted in primitive reflexive patterns.[1] Reaching for an object may necessitate synergistic activity of muscles as remote as the trunk, pelvis, and lower extremities to provide postural adjustments and proximal stabilization. Basic synergy patterns can be modulated by higher neurologic control strategies when necessitated by a new or unique task or changing environmental conditions.

Coordination of the movement of the trunk around the pelvis and of the pelvis around the hips during trunk flexion and extension, often referred to as the lumbopelvic rhythm, may vary under conditions such as lifting a load,[16] low back pain,[9] or back belt use.[12] Other groups have reported on interjoint coordination of movement patterns and electromyogram activity involving multiple segments of the kinetic chain.[3] Thorstensson et al[26] described the temporal association and coactivity of the abdominal and trunk extensor electromyogram activity during several trunk motions. Oddson and Thorstensson,[17] and Sommerich and Marras,[21] described the underlying synergy patterns of the trunk electromyogram and the variation in those patterns under different conditions.

During trunk flexion from erect standing, the eccentric activity of the erector spinae typically increases as the moment arm of the torso increases, until a point at which it abruptly decreases as full lumbar flexion is approached. This phenomena, referred to as the flexion–relaxation response (FRR) by Floyd and Silver,[6] occurs in the lumbar region in 92% to 100% of the normal population.[6,23,25] The FRR is thought to be invoked by a stretch inhibition reflex, allowing the erector spinae to relax while the passive elements provide the necessary extension moment.[6,8,27] Andersson et al[2] reported that during trunk flexion, electromyogram activity of the quadratus lumborum and lateral erector spinae increases rapidly as the medial erector spinae muscles undergo flexion–relaxation and may assist the passive tissues in providing an extension moment. Variation in the FRR has been reported with changes in cadence, with the application of an external load, and with compound movements.[19,22] FRR has also been observed in the hamstrings and the cervical paraspinal muscles.[14,24] Although it is believed to be reflexive in nature, the FRR may be overridden by volitional or protective responses.[7] Research has consistently indicated that the FRR may be

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absent or significantly impaired (electromyogram activity persists) in cases of both acute and chronic low back pain.[19,23,25]

The findings in these studies have provided insight into the interplay of the trunk and extremity muscles during trunk flexion and extension, but there is little in the literature about timing of activation within the erector spinae. The pattern of activation during extension and deactivation during trunk flexion has not been elucidated. The range of influence of the FRR within the erector spinae has also not been well described. Although the literature indicates that muscle recruitment can be affected by initial posture and the velocity of movement, these factors have not been evaluated specifically regarding the erector spinae. In this study, the timing of activation of the erector spinae were examined at multiple sites in subjects approaching and extending from a posture of full lumbar flexion. The specific goals of this study were to observe the patterns of activation and FRR in the erector spinae and hamstrings, to investigate the effect of movement speed and posture on activation patterns, and to establish the stability and repeatability of erector spinae and hamstring activation patterns over time.

■ Methods

Subjects. Six male subjects in good health and with no significant history of musculoskeletal disease were recruited. The subjects' mean age was 41.9 ± 11.0 years (SD), mean stature was 176.7 ± 6.1 cm (SD), and mean weight was 87.6 ± 8.7 kg (SD). The experimental protocol was demonstrated and explained to the subjects, and all gave written informed consent.

Experimental Design. A 3 × 2 (posture by speed) within-subject factorial design was used. The three levels of the posture condition were free-standing, restrained-standing, and sitting. The two levels of the movement speed condition were fast and slow. The dependent variables were lumbar arc length and electromyogram activity.

Equipment.

Lumbar Arc Length Measurement. Lumbar spine kinematics were evaluated using a device called a lordosimeter (US patent 5772610), developed in the authors' laboratory, which provides an unobtrusive and direct dynamic measurement of change in lumbar arc length. This device uses a flexible rod that moves in guides attached to the skin overlying the lumbar spine. Flexion or extension of the lumbar spine results in a change in the lumbar arc length, which has been demonstrated to be a good surrogate of lumbar curvature. Further description and characterization of the device can be found elsewhere.[13]

Surface Electromyogram. Ten bipolar surface electromyogram electrodes (model MYO115; Liberty Technology, Hopkinton, MA), were used to measure the electrical activity of the trunk extensor and hamstring muscles. The electrodes had 4-mm diameter stainless steel pickups located 1.6 cm apart, and on-board differential amplification with a 63-dB gain, an input impedance of more than 10^{14} Ω and a common mode rejection ratio more than 90 dB, at 60 Hz. Second-order high- and low-pass filters provided a bandwidth of approximately 40 to 400 Hz.

Experimental Protocol. During the orientation session, the electromyogram sites were located 3 cm bilateral to the T9, T12, L2, and L5 spinous processes. The hamstring electromyogram sites were located on the posterior thigh midway between the ischial tuberosity and the head of the fibula, bilaterally. The upper and lower lordosimeter mounting sites were located and marked. The location of the electromyogram sites, lordosimeter mounting sites, and any distinguishing skin features were transferred to a transparent vinyl film, which was retained to permit precise repositioning during subsequent sessions. The 10 electrode sites were prepared, the electrodes gelled and then attached using skin tape, and the lordosimeter was donned. The two contacts of each electrode were oriented along the long axis of the muscle. The reference electrode was placed over the C7 spinous process.

The subjects were then trained in the experimental procedure—smoothly flexing the trunk from erect standing to full flexion, pausing, and then returning to the starting position, with the arms hanging freely by the sides. The experimenter paced the motion by counting aloud and announcing the flexion phase, the rest phase, and the extension phase. For the slow-speed trials, a 3-second pace for trunk flexion was followed by a 1-second rest and then a 3-second pace during extension. For the fast-speed trials, the same procedure was followed, except that a 2-second pace was used during flexion and extension.

In the protocol, three postures were evaluated. In free-standing, the feet were located approximately shoulder-width apart, and a slight knee bend was maintained. In restrained standing, the subjects stood in an apparatus designed to decrease pelvic rotation and stabilize the lower extremities as seen in Figure 1. In sitting, a chair and footrest were adjusted so that the subject's thighs were horizontal to the ground and the knees flexed to 90°.

This protocol was followed during the six biweekly experimental sessions. After briefly practicing the motions, 12 trials (3 postures × 2 speeds × 2 trials) were acquired. The raw electromyogram and lordosimeter signals were passed to an analog–digital converter, sampled at 1080 Hz, and stored in computer memory.

Data Analysis.

Determination of Timing of Muscle Activation. Data collected in the six experimental sessions after the initial training visit were imported into computer software (MATLAB; The MathWorks, Natick, MA). The electromyogram waveforms were rectified, then smoothed using a noncausal, midpoint, moving-average filter of 92.6 msec. The lordosimeter data needed no smoothing. A custom program was created to display the data on the computer screen. A training program was conducted for five raters, during which operational definitions were explained, and instruction in marking of the electromyogram waveforms was given. $TEMG_{FLEX}$ was defined as the inflection point of the negative slope of the rectified, smoothed waveform, and the flat (zero) slope occurring at full flexion (flexion–relaxation). $TEMG_{EXT}$ was defined as the inflection point of the positive slope of the waveform and the flat slope at flexion–relaxation (Figure 2). Trials in which there was no clear quiescent phase in a waveform (FRR was absent) were flagged

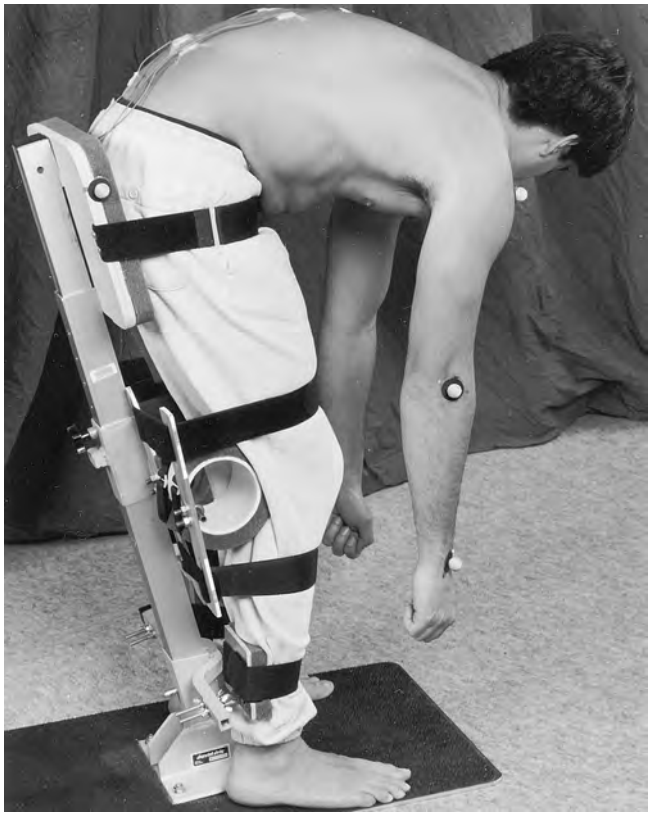

Figure 1. Subject at a fully flexed posture in the restraint stand, secured by thigh and calf straps.

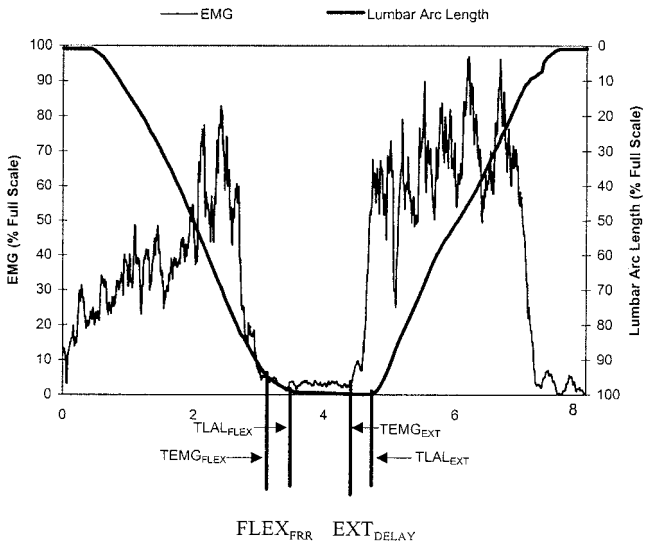

Figure 2. Plot of lumbar arc length and L5 electromyogram during a slow-speed, free-standing movement from erect standing to full lumbar flexion and return to erect standing. $FLEX_{DELAY}$The time from end of motion to the flexion–relaxation response ($TLAL_{FLEX} - TEMG_{FLEX}$) and EXT_{DELAY}, the time lag of the extension motion from initiation of electromyogram activity ($TLAL_{EXT} - TEMG_{EXT}$), are indicated.

for later analysis. Occasionally, small-magnitude bursts of electromyogram activity were observed before initiation of trunk extension. If the burst was completely separated in time from the main extension waveform, $TEMG_{EXT}$ was to be marked at the inflection point of the main waveform. If there was incomplete separation in time, $TEMG_{EXT}$ was to be marked at the beginning of the small burst. If a rater thought that any event could not be properly identified, that event was not marked. Approximately 40 waveforms were randomly selected, and marked by all five raters, as a group, to ensure consistency in marking technique. During the marking procedure the raters were blinded as to subject, posture, and trial. The speed of trial was obvious. In all, each of the five raters evaluated 432 experimental trials marking 4310 out of 4320 possible electromyogram waveforms (6 subjects × 6 sessions × 12 trials × 10 muscles). One trial was corrupted during computer storage.

An *a priori* criterion was established for evaluating lordosimeter tracings. The time of maximum lumbar arc length (LAL) in flexion was first achieved ($TLAL_{FLEX}$) and the time when extension motion was initiated ($TLAL_{EXT}$) were marked on each lordosimeter tracing (Figure 2). If either $TLAL_{EXT}$ or $TLAL_{FLEX}$ was not ratable by two or more of the raters, that trial was rejected. In all, 11 of the 431 lordosimeter tracings (and the associated electromyogram data) were rejected, because transitions were unclear, or resting phases were not stable. The *x*-axis values (time) for $TEMG_{FLEX}$, $TEMG_{EXT}$, $TLAL_{EXT}$, and $TLAL_{FLEX}$ for each trial were saved to a computer spreadsheet.

Calculation of Flexion–Relaxation Time and Electromechanical Delay. Flexion–relaxation time ($FLEX_{DELAY}$) was defined as the time difference between the end of trunk flexion motion and the beginning of the FRR. The electromechanical delay in extension (EXT_{DELAY}) was defined as the time from initiation of electromyogram activity during the extension phase to the start of trunk extension motion (Figure 2). These relationships are expressed in the following equations:

$$FLEX_{DELAY} = TLAL_{FLEX} - TEMG_{FLEX}$$

$$EXT_{DELAY} = TLAL_{EXT} - TEMG_{EXT}$$

Comparisons of $FLEX_{DELAY}$ and EXT_{DELAY} from different electromyogram sites can be used to investigate the activation sequence of muscles, because the value for each muscle is referenced to a common lumbar arc length value. Thus, differences in $FLEX_{DELAY}$ and EXT_{DELAY} are mathematically equivalent to the differences in $TEMG_{FLEX}$ and $TEMG_{EXT}$ between electromyogram sites.

Statistical Analysis. The mean of $FLEX_{DELAY}$ and EXT_{DELAY} values for the five raters was calculated for each muscle site for each trial. Analysis of variance (ANOVA) was used to independently evaluate effects of direction of movement (flexion *vs.* extension) and electrode side (left *vs.* right). Differences in the timing of activation due to the effects of speed of movement, posture, trial, and visit on activation time during the 420 trials was evaluated using the general linear model (GLM) procedure. The criterion selected for statistical significance was $P < 0.05$.

■ Results

A clearly defined quiet phase occurred in all but 5.5% of the waveforms. Most of the waveforms in which FRR was absent were recorded at T9, and most of these cases involved three of the six subjects. The muscle group with the next largest proportion of absent FRR waveforms

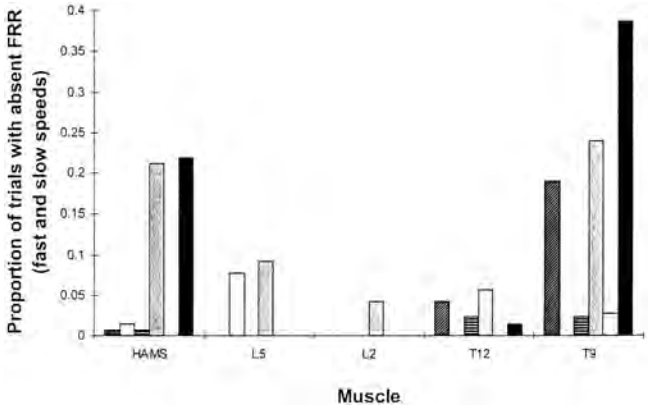

Figure 3. Proportion of all trials in which no flexion–relaxation response was observed, for each of the five electromyogram sites. Each bar represents one of the six subjects.

was the hamstrings, most of which were from two of these three subjects. The FRR was rarely absent at L5, L2, and T12. The distribution of trials with absent FRR by electromyogram site is presented in Figure 3.

The results of the ANOVA of the effect of movement direction (flexion *vs.* extension) on timing of activation was significant at all muscle levels. The result of the ANOVA of the effect of side (left *vs.* right) indicated that no significant differences were observed within paired electrodes for any combination of muscle level or movement direction, except for the hamstrings in the flexion direction. On review of the data, it was apparent that the left- to right-side difference in hamstring activation was due to the electromyogram data from one subject. The subject consistently had excess activity at the right hamstring site, even during quiet standing, during the six sessions. The ANOVA was repeated with the data for the right hamstring in the one subject removed, and the effect of side was no longer significant for the hamstrings during flexion. The paired electrode data were then collapsed, reducing the data to five muscle levels.

The results of the GLM of the five muscle levels in the flexion and extension directions for the main effects of speed, trial, visit, posture, and the second- and third-order interaction terms are presented in Table 1. Speed of movement was a significant factor at all muscle levels. The effect of posture was significant only in extension at T9. No significant effect of trial or visit was observed. Significant differences for the second- and third-order interaction terms were sporadic and exhibited no apparent pattern.

The effect of speed of movement (fast *vs.* slow) on activation time normalized by movement period ($FLEX_{DELAY}$/period and EXT_{DELAY}/period) for each trial was evaluated using a single-factor ANOVA. No significant differences were observed for any muscle level or direction of movement.

Because the effects of visit and trial were not significant, data were collapsed across these variables. Single-factor ANOVA of the data grouped by speed and posture levels indicated there were significant differences in timing of activation of the five electromyogram sites during the extension phase (EXT_{DELAY}) in the free- and restrained-standing postures at both the slow and fast speeds. No significant differences were found during the flexion phase ($FLEX_{DELAY}$) under any condition or in any sitting posture condition (Table 2). Histograms of the data grouped by the two speed and the three posture conditions are presented in Figure 4.

■ Discussion

The results showed that the effect of visit on the dependent variables $FLEX_{DELAY}$ and EXT_{DELAY} was not significant, indicating that the timing of deactivation at full lumbar flexion and activation at the initiation of exten-

Table 1. Results of ANOVA for Muscle Group and Direction of Motion, by Posture, Speed, Visit, Trial, and Interactions

	Muscle	HAMS		L5		L2		T12		T9	
	Direction	Flex	Ext	Flex	Ext	Flex	Ext	Flex	Ext	Flex	Ext
1st Order											
	(P)osture										*
	(S)peed	*		*		*		*		*	
	(V)isit										
	(T)rial										
2nd Order											
	P*S										
	P*V					*					
	P*T										
	S*V										
	S*T										
	V*T			*							
3rd Order											
	P*S*V										
	P*S*T										
	P*V*T									*	
	S*V*T										

* Indicates statistical significance at $P < 0.05$.

Table 2. Results of Single Factor ANOVA for the Five Muscle Groups by Direction of Motion, Posture, and Speed

	Slow		Fast	
	$FLEX_{DELAY}$	EXT_{DELAY}	$FLEX_{DELAY}$	EXT_{DELAY}
Free-Standing	.90	**.004**	.83	**.001**
Restrained-Standing	.91	**.014**	.89	**.003**
Sitting	.99	.86	.97	.93

Boldface indicates statistical significance at $P < 0.05$.

sion was consistent throughout the study period. This finding shows that the experimental protocol for locating, mapping, and relocating electrode positions during subsequent visits produced consistently repeatable measures of the timing of muscle activation.

The T9 electromyogram site had the highest proportion (14.4%) of waveforms with absent FRR. Most of these waveforms were from three of the six subjects. The reason for the variability in activity at the T9 site is not obvious. The increased activity may have been due to crosstalk from superficial muscles, individual differences in development and conditioning, or anthropometric variation in the trunk center of mass. Another possible explanation is that the FRR does not consistently dominate the motor control schema at this T9 site. Absence of FRR at or around the T9 site was also reported by Dolan and Adams,[5] and Toussaint et al.[27] McGill and Kippers[10] found true quiescence of the electromyogram signal at T9 in only one of eight subjects, using a criteria based on electromyogram amplitude.

The two speed levels yielded very consistent results across posture conditions. The $FLEX_{DELAY}$ and EXT_{DELAY} periods were significantly shorter during the faster speed trials. The differences between speeds were greatest as full flexion was approached, when the magnitude of $FLEX_{DELAY}$ was greatest. The difference between speeds for EXT_{DELAY} was less pronounced, particularly at the thoracic levels. The differences in activation timing due to movement speed found between the fast- and slow-movement conditions were not unexpected, considering that velocity and acceleration would vary with movement period. Because movement speed was controlled by the experimental protocol, to properly evaluate the effect of speed on the coordination of muscular activation, it was necessary to control for movement period. Normalization of the time of muscular activation by trial period allowed for comparison across test speeds. The absence of significant differences in the normalized data indicates a consistency in the coordination of the muscular recruitment across speeds for a fluid trunk flexion and extension motion. More disparate speeds or ballistic motions may present quite different activation patterns.

A significant difference in timing between the muscle sites was observed in the free- and restrained-standing postures during extension, with activation occurring first in the hamstrings and last at the T9 site. The observed segmental recruitment pattern is consistent with reports of kinematic studies with findings indicating that pelvic motion leads trunk motion during extension in standing postures.[9,16,19] Several possible mechanisms may be responsible for the observed segmental activation patterns. The need for proximal stabilization could explain the earlier activation of the hamstrings and the more proximal erector spinae segments. There may also be a slight advantage in lever arm length at the lower lumbar level of the erector spinae,[11,15] which could favor early recruitment.

Differences in activation timing were not significant in sitting during extension, indicating a more simultaneous pattern of recruitment. The more kyphotic posture of the spine during sitting may also offer some explanation for the significantly earlier activation at the T9 site than in standing. Daggfeldt et al[4] noted that the lever arm of the lumbar erector spinae decreases with increasing kyphosis. The thoracic erector spinae, acting through the aponeurosis can produce an extension moment at the lower lumbar region. Postural pretensioning of the muscles and ligaments of posterior trunk, due to a more kyphotic thoracic and lumbar spine and a more posteriorly tilted pelvis, may favor a concurrent recruitment pattern. Another possible mechanism is ligament creep under cyclic loading. Solomonow et al[20] reported a decrease in erector spinae activity under creep conditions in a cat model and speculated that it may be due to mechanoreceptor desensitization. Although ligament creep could occur in the sitting posture, this explanation seems less likely, because no significant effect of trial sequence on timing of activation was observed.

The delay between muscle activation and initiation of extension motion (EXT_{DELAY}) was greatest at the hamstrings and least at T9 for both standing postures. Dolan and Adams[5] reported an electromechanical delay of approximately 70 msec between initiation of myoelectric activity and tension generation during isometric extension of the erector spinae. The EXT_{DELAY} observed in this experiment were much longer. Delays at the hamstrings ranged from 326 to 704 msec, depending on posture and speed. Lumbar delays ranged from 123 to 360 msec. Delays in the thoracic region were the most variable, ranging from −43 to 361 msec. The large differ-

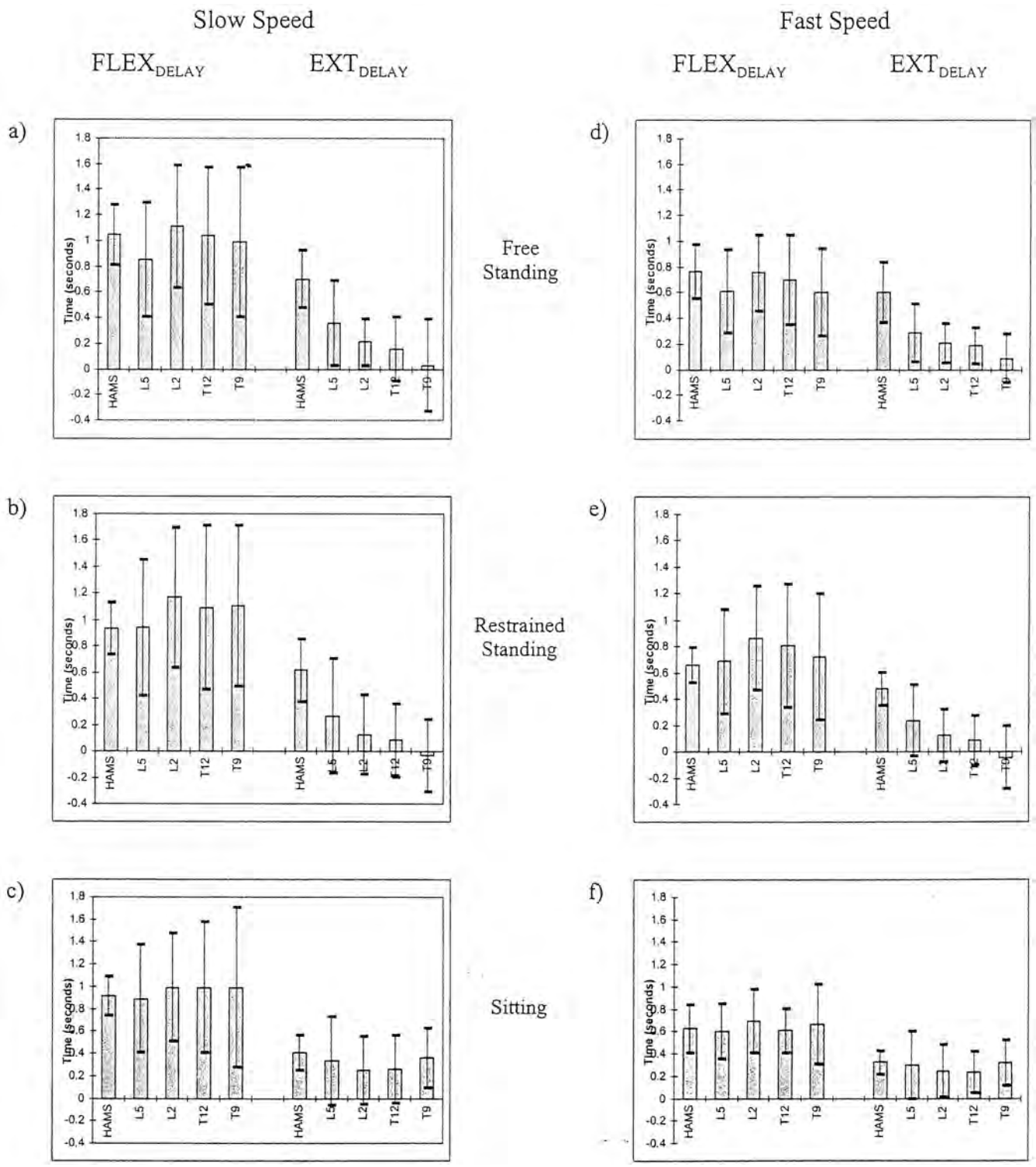

Figure 4. Mean $FLEX_{DELAY}$ and EXT_{DELAY} (±SD) for the slow-speed condition in (**A**) free-standing, (**B**) restrained-standing, and (**C**) sitting postures. Fast-speed condition: (**D**), (**E**), and (**F**), respectively.

ences between the EXT_{DELAY} observed in this study and the electromechanical delay reported by Dolan and Adams,[5] may be a matter of definition and protocol. Electromechanical delay, a measure of neuromuscular function, involved a rapid isometric contraction for the purpose of estimating the time from signal propagation to torque generation by the contractile elements. In this study, EXT_{DELAY} is a measure of the time from signal propagation to lumbar motion production during a task involving a slow extension motion. EXT_{DELAY} may include the time required to develop tension to stabilize the spine before motion and perhaps delays due to the effects of cocontraction. The initiation of lumbar extension is an event that serves as an index for muscle activation

timing and is not a true measure of neuromuscular function.

During the flexion phase, $FLEX_{DELAY}$ was fairly consistent, with no significant differences in activation pattern noted for the five electromyogram sites across the three postures and two speeds. Activation was much more concurrent among the electromyogram sites at the approach to full flexion than at the initiation of extension. This recruitment pattern is consistent with the results of kinematic studies reported in the literature.[9,16] Typically, the electromyogram activity at the upper lumbar and thoracic sites ended earlier than at L5. Generally, for all postures, activity at the L5 site ended (the FRR began) later than at the L2 and T12 sites. The timing of hamstring activity in flexion showed the greatest variation with posture. In the free-standing posture, hamstring activity ended before L5 activity, but cessation of activity was nearly simultaneous in the restrained-standing and sitting postures. The observation of FRR in the hamstrings tends to support the finding of Sihvonen.[24] However, as noted previously, this behavior was not universal among all subjects.

A particular strength of this study was the ability to observe trunk extensor muscle activation patterns in the healthy subjects with time. Consistency in the timing of muscle activation was observed for the thoracic and lumbar erector spinae and hamstrings during six biweekly test sessions, a protocol necessitating repeated removal and application of surface electromyogram electrodes. This result should be useful in the investigation of temporal changes in electromyogram activity in low back pain.

A weakness of this study was sample size. Inclusion of a greater number of subjects would have provided greater statistical power for identification of the main effects and potential interactions.

■ Conclusion

The FRR was observed at the end of lumbar flexion at all test sites of the erector spinae and hamstrings. The response was most ubiquitous in the lumbar region. Timing of muscular activation at each muscle site varied with movement speed, these differences were not significant when normalized by movement period. Patterns of activation of the thoracolumbar erector spinae and hamstrings were dependent on the direction of movement. The coordination of the trunk extensor mechanism was dominated by the FRR during flexion, during which the five muscle test sites exhibited a fairly concurrent deactivation across speeds and postures. The extension motion exhibited a unique pattern of segmental activation progressed in the caudad-to-cephalad direction in the standing postures. The absence of a segmental activation pattern in sitting indicates that postural effects such as pelvic inclination, ligament, or muscular tension, or changes in mechanical advantage may modulate coordination.

Knowledge of the patterns of erector spinae and hamstring activation provide data for more detailed modeling of trunk extensors during trunk flexion and extension. Knowledge of the pervasiveness of FRR in the lumbar erector spinae, and the presence of the response, although less consistent in the thoracic region and hamstrings (in the normal population), should be useful to those investigating FRR changes in subjects with low back pain. Investigations of changes in FRR over time in the low back pain population will be strengthened by the finding of stability of the response over time in a healthy population.

■ Key Points

- The FRR was observed in the hamstrings and at T9, T12, L2, and L5 of the erector spinae during trunk flexion, although the response was less pervasive at T9 and the hamstrings.
- Coordination of trunk extensor recruitment was independent of speed, when trunk flexion and trunk extension movement period were normalized.
- Segmental muscle recruitment progressed in the caudad-to-cephalad direction during trunk extension from full flexion in standing.
- The onset of FRR occurred relatively concurrently throughout the erector spinae and hamstrings during trunk flexion in standing and sitting.

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