The Biomechanical and Clinical Significance of the Lumbar Erector Spinae Flexion-Relaxation Phenomenon: A Review of Literature

Christopher J. Colloca, DC,[a] and Richard N. Hinrichs, PhD[b]

Abstract

Objectives: The aim of this study was to review the biomedical literature to ascertain the biomechanical and clinical significance of the lumbar erector spinae flexion-relaxation phenomenon (FRP).

Data Sources: *Index Medicus* via PubMed, the Noble Science Library's e-journal archives, and the Manual Alternative and Natural Therapy Index System databases were searched using the same search terms.

Discussion: The presence of the FRP during trunk flexion represents myoelectric silence consistent with increased load sharing of the posterior discoligamentous passive structures. Passive contributions from erector spinae stretching during the flexion posture and active contributions from other muscles (quadratus lumborum and deep erector spinae among others) further assist in load sharing in the trunk flexion posture. A number of studies have shown differences in the FRP between patients with chronic low back pain and healthy individuals, and the reliability of the assessment. Persistent activation of the lumbar erector spinae musculature among patients with back pain may represent the body's attempt to stabilize injured or diseased spinal structures via reflexogenic ligamentomuscular activation thereby protecting them from further injury and avoiding pain.

Conclusions: The myoelectric silencing of the erector spinae muscles in the trunk flexion posture is indicative of increased load sharing on passive structures, which tissues have been found to fail under excessive loading conditions and shown to be a source of low back pain. The studies that show differences in the presence of the FRP among patients and control subjects are encouraging for this type of clinical assessment and suggest that assessment of the FRP is a valuable objective clinical tool to aid in the diagnosis and treatment of patients with low back pain. (J Manipulative Physiol Ther 2005;28:623-631)

Key Indexing Terms: *Biomechanics; Electromyography; Low Back Pain; Lumbar Vertebrae; Flexion-Relaxation Phenomenon; Trunk Flexion*

Movements in the lumbar spine, including flexion and extension, are governed by a complex neuromuscular system involving both active (muscle) and passive (vertebral bones, intervertebral disks, ligaments, tendons, and fascia) components.[1] Common among spinal disorders are disruption to the neuromuscular balance and load sharing of the spinal tissues, ultimately resulting in pain and disability, and an enormous economic burden to society.[2] In the assessment of patients with lumbar complaints, measuring the electromyographic (EMG) activity of the trunk musculature is one objective means used by biomechanists and clinicians to assess the function of the lumbar spine. The clinical utility of the use of electromyography, however, is controversial in the diagnosis of patients with low back pain without lower extremity symptoms.[3]

There is evidence to suggest that EMG differences exist between patients with back pain and healthy subjects during dynamic flexion tasks performed at peak flexion.[4,5] To this extent, several studies have examined the apparent myoelectric silencing of the low back extensor musculature during a standing to full trunk flexion maneuver or the flexion-relaxation phenomenon (FRP). The electrical signal reduction or silence that occurs in healthy subjects during lumbar spine flexion has been hypothesized to represent

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Sources of support: None declared.

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Paper submitted February 13, 2004; in revised form July 14, 2004.

0161-4754/$30.00

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doi:10.1016/j.jmpt.2005.08.005

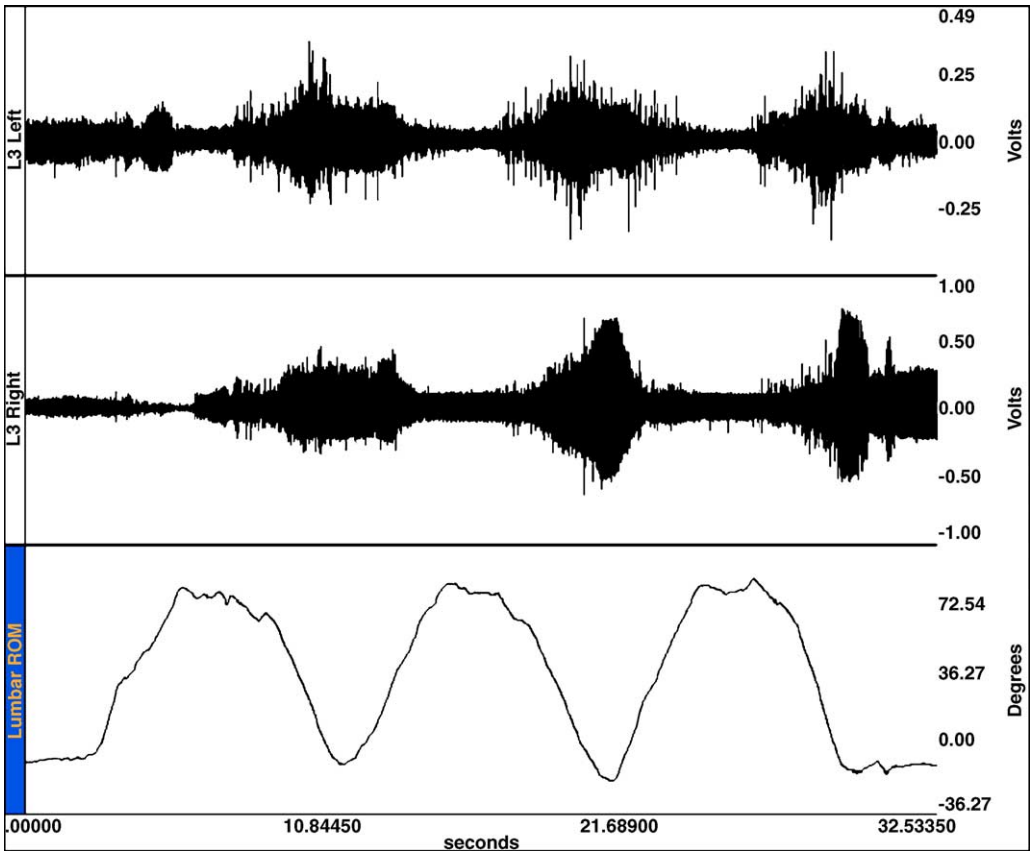

Fig 1. *Flexion-relaxation phenomenon. Original raw surface electromyographic recordings for the right (top) and left (center) erector spinae muscles at the level of L3 are depicted for an asymptomatic subject during 3 consecutive lumbar flexion tasks (data acquisition at 5000 Hz by means of Biopac MP150; Biopac Systems, Inc, Goleta, Calif). Lumbar range of motion (bottom) was measured simultaneously during lumbar flexion efforts by means of an electrogoniometer (Biopac Systems, Inc). Silencing of the erector spinae muscles are observed during the 3 repeated flexion efforts consistent with the FRP in this graph.*

the extensor musculature being relieved of its moment-supporting role by the passive tissues, particularly the posterior spinal ligaments.[6] Likewise, a failure of the muscles to relax in patients with back problems is indicative of heightened erector spinae resting potentials or underlying back muscle spasticity.

The FRP is modulated by a number of factors including the magnitude of applied load,[7] loading rate,[8] and patient clinical status.[9] Creep developed during a short static lumbar flexion has also been found to elicit significant changes in the muscular activity pattern of the FRP.[1] Although there are many factors to consider, understanding the biomechanical importance of the load sharing between the erector spinae musculature and passive lumbar spinal tissues will aid in the understanding of the FRP from both a biomechanical and clinical standpoint. Moreover, reviewing the biomechanical implications of the FRP and reported differences among patients with back pain and healthy subjects assists in understanding the benefits, limitations, and clinical utility of this type of EMG assessment. Thus, the purpose of this study is to review the biomedical literature to ascertain the biomechanical and clinical significance of the lumbar erector spinae FRP.

Methods

A review of the biomedical literature on the lumbar erector spinae FRP was conducted by searching the National Library of Medicine's *Index Medicus* via their PubMed database, the Noble Science Library's e-journal archives, and the Manual Alternative and Natural Therapy Index System using the following search terms: *biomechanics*, *electromyography*, *erector spinae muscle*, *flexion-relaxation*, *lumbar spine*, and *trunk flexion*. Keywords were input in various combinations, always including the terms flexion-relaxation or trunk flexion, and relevant citations were noted. Collectively, these searches revealed 36 articles generally related to the research question in the current study, ultimately resulting in 16 specific articles on the FRP. These 16 articles among

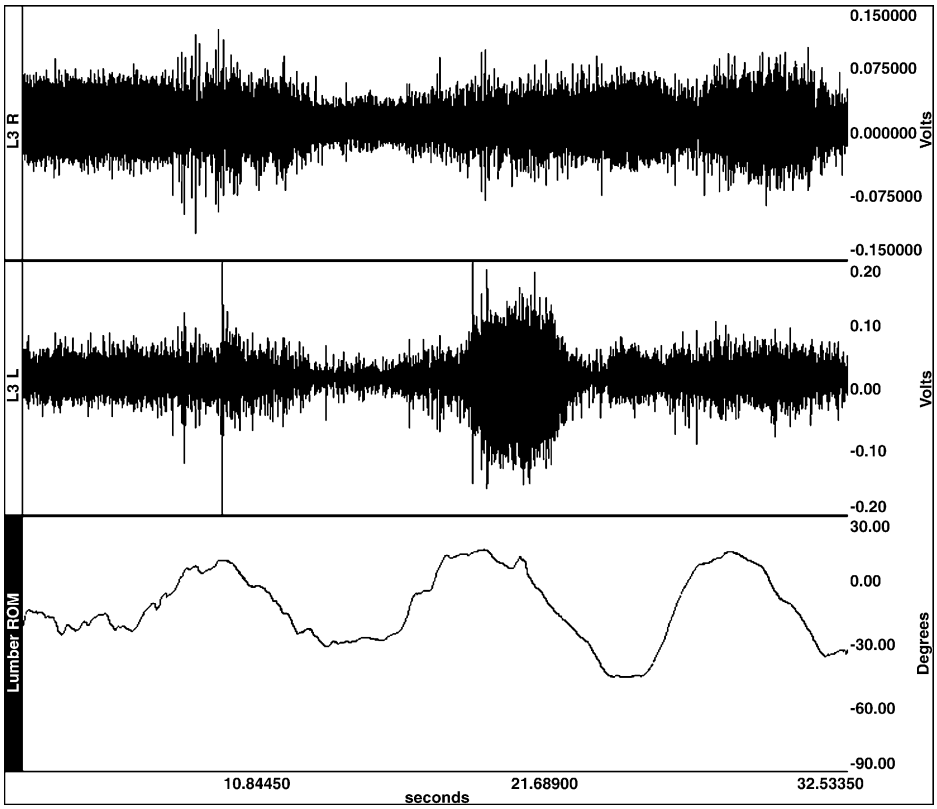

Fig 2. *Absence of FRP. Original raw surface electromyographic recordings for the right (top) and left (center) erector spinae muscles at the level of L3 are depicted for a patient with CLBP during 3 consecutive lumbar flexion tasks (data acquisition at 5000 Hz by means of Biopac MP150; Biopac Systems, Inc). Lumbar range of motion (bottom) was measured simultaneously during lumbar flexion efforts by means of an electrogoniometer (Biopac Systems, Inc). A lack of the FRP is observed in the top graph for the right electrode (L3 right) during the 3 flexion tasks and for the third flexion task on the left (L3 left) in this patient with chronic back pain.*

others were obtained via interlibrary loan. Upon review of relevant citations, secondary references were further identified and subsequently similarly retrieved.

Discussion

Physical activities involving full trunk flexion are common in activities of daily living, occupational demands, and sport. Thus, knowledge of the biomechanics and clinical implications of trunk flexion is important. Lumbar spinal tissue sprain and strain have been reported in lumbar flexion postures,[10] and patients with low back disorders often avoid such positions. Understanding the transfer of tissue loads in the trunk flexion posture thus assists in the understanding of normal trunk biomechanics, mechanisms of injury, and the consequential pathogenesis of low back pain.

Several biomechanical and clinical studies have examined the apparent myoelectric silence of the lumbar spine extensor musculature during lumbar flexion from a neutral upright standing posture. The FRP refers to a pattern of muscle activity during trunk flexion in which the lumbar muscles ultimately relax at what appears to be a distinct point in the lumbar flexion range of motion (Fig 1). Floyd and Silver[11] first described the term *flexion-relaxation* of the lumbar extensor musculature using EMG and suggested that the passive lumbar posterior elements, namely, the posterior spinal ligaments and intervertebral disks, supplied the needed moment during full flexion in the absence of erector spinae muscle activity. The mechanism for the silencing of the erector spinae muscles during trunk flexion has been proposed to result from stimulation of stretch receptors in the posterior discoligamentous tissues during the flexed posture, acting to reflexogenically inhibit erector spinae activity.[1,7,12-14] A number of biomechanical studies have since examined the transfer of loads among tissues during lumbar flexion, and several clinical studies have further investigated the significance of the presence or absence of the FRP in patients with lower back pain (Fig 2). Such research is not only important to understand the biomechanical consequences of the trunk flexion posture but to further understand the clinical utility of using the FRP as an objective outcome measure to discriminate patients with low back pain. The FRP is an appealing quantitative test for adding objectivity to a movement in which pain inhibition and

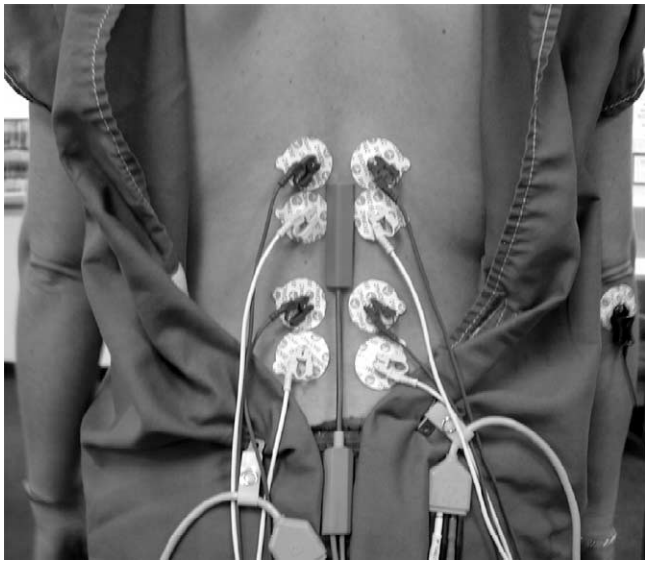

Fig 3. *Typical electrode and electrogoniometer placement for dynamic surface EMG assessment. Surface EMG electrodes are placed overlying the erector spinae musculature at the levels of T12 and L3, and an electrogoniometer (paddles) is placed at the level of T12 and S1. A reference ground electrode is shown on the right olecranon. Simultaneous erector spinae muscle EMG activity can be measured as a function of lumbar range of motion with this experimental setup.*

voluntary effort limitations may confound the examiner's ability to assess actual lumbar flexibility.[15]

In normal trunk flexion with the knees straight, the 5 lumbar vertebral segments flex forward during the first 50° to 60°, followed by the pelvis rotating between the hips.[16] At 75% to 85% of trunk flexion, the lumbar spine reaches its maximum range of motion, whereas the pelvis is providing terminal flexion to achieve the final stages of total trunk flexion.[7] It has been postulated that, at this point, the passive noncontractile soft tissues (intervertebral disks, ligaments, fascia) are providing most of the spinal support, and little erector spinae activity is required to maintain this posture.[6,7] Interestingly, lumbar flexion is the usual posture for stoop labor, a position selectively chosen by workers in many types of field labor for its efficiency (lack of voluntary muscle effort).[17] From a clinical standpoint, patients with chronic low back pain (CLBP) have been reported not to achieve flexion-relaxation because of an abnormal neuromuscular coordination between the trunk and hip movements.[9]

Task Performance

To assess the EMG activity of the lumbar trunk muscles during trunk flexion, use of a data acquisition system is necessary. Surface electromyographic electrodes are attached to the skin at the levels of T12 and L3 over the belly of the erector spinae muscles, and a trunk measurement device (electrogoniometer) is attached at the levels of T12 and S1 to quantify the lumbar motion during the test (Fig 3). Alternatively, a video motion analysis system can be used and synchronized with the EMG signals obtained to assess the

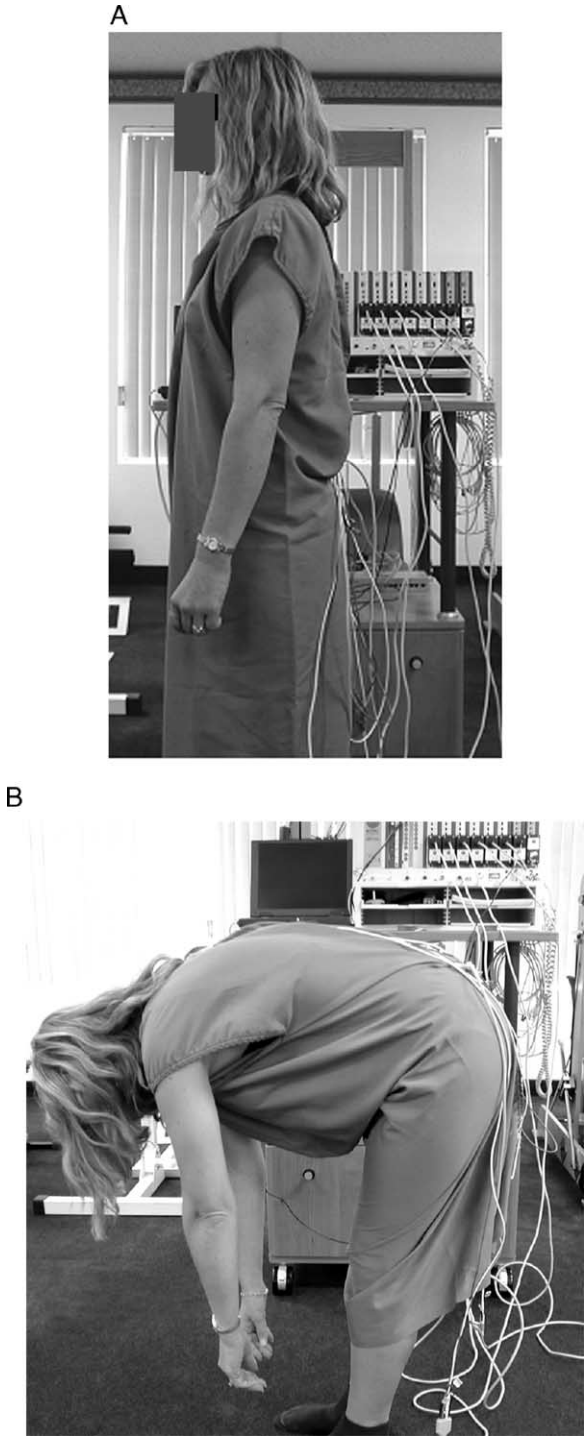

Fig 4. *Lumbar flexion test performance. (A) The standing patient flexes forward (B) and returns to the standing position (A) during the flexion range of motion test. Surface electromyographic recordings are obtained from the erector spinae muscles, and simultaneous range of motion is monitored by means of a dual paddle electrogoniometer.*

EMG response in relation to the trunk motion. In the standing position, the patient is then asked to stand quietly in their neutral upright posture and bend forward to the point of full flexion at a speed that they feel comfortable with as EMG signals are recorded (Fig 4). Generally, 3 repetitions are performed to allow the examiner to observe any discontinuity of the task effort and to obtain mean values of range of motion and EMG activity. During the test, surface EMG activity is recorded in relation to the spinal motion during the trials and peak-peak amplitude of the EMG signals can be assessed. Several variables such as patient population, recording technique, data analysis and interpretation (defining *on* and *off*), normalization techniques, and task performance (including controlling for velocity of motion) among others are responsible for differences in results among studies investigating the FRP. In addition, the onset and cessation of the myoelectric silence of the FRP can be influenced by several factors including lumbar lordosis, general laxity of the joints, strength and relative length of the muscles of the trunk and hip, coordination of trunk and hip movements, and the velocity of the flexion extension movements.[18]

The notion that the FRP represents a dichotomous pattern of muscles being either on or off likely is misleading. Physiologically, lumbar muscles show electrical activity because they are active and the degree of electrical activity is roughly proportional to the rate and amplitude of muscle fiber firing. Nevertheless, defining EMG activity to denote when muscles are active is necessary to proportion and interpret EMG amplitude results from raw data that have been collected. Exploring the different assessment and analytical techniques presented in the literature serves to point up differences in results reported in the literature and subsequently understand subsequent limitations.

Quantifying the FRP and Load Transfer During Trunk Flexion

A number of studies have shown the presence of the FRP during upright trunk flexion in asymptomatic subjects.[1,7,12,15,19-22] Consistent with these studies, recently, Solomonow and coworkers[1] recorded EMG activity at the L3 through L4 level over the erector spinae musculature during upright trunk flexion tasks and quantified trunk joint angles using a two-dimensional video–based motion analysis system in 49 asymptomatic subjects. To nullify time shifts of the EMG data, a digital algorithm to yield a mean absolute value with a time shift of 100 milliseconds was performed and *EMG-on* and *EMG-off* were defined, with the latter representing EMG silence. Flexion-relaxation was observed in all subjects during trunk flexion, with the mean EMG-off lumbar flexion angle for men and women ranging between 46° to 50° of trunk flexion. Studies showing the FRP in asymptomatic subjects will be further reviewed hereinafter, in context with their biomechanical and clinical significance.

In any static position of trunk flexion, the hip flexion is maintained by balance of the torque of the upper body weight resisted by a combination of the tension and mass of the structures posterior to its axis, and vertebral flexion is maintained by the torque of the upper body weight resisted by combination of tension and compression of the vertebral structures.[7] Thus, it is rationalized that the FRP of the erector spinae musculature in the trunk flexion posture represents a "switching off" of electrical activity due to equilibrium being achieved between the torques because of gravity and the extension torque provided by the stretched posterior vertebral elements.[18] The erector spinae thus plays the key role of balancing the two passive forces and providing controlled movement of vertebral flexion through its eccentric action.

To understand the biomechanical consequences of the FRP, other research has been conducted to determine the load sharing among lumbar spinal tissues in the flexed posture. Schultz et al[7] measured trunk muscle EMG in 8 healthy subjects during upright lumbar flexion with various loading conditions, and biomechanical analyses were used to estimate the subsequent loads imposed on the lumbar trunk structures. In these analyses, the net support reaction needed for equilibrium across a transverse section at the level of L3 was computed followed by analysis of muscle action that could supply the net reaction derived using an optimization technique. The resulting spine compression and shear loads were then estimated as well. The authors found that, during lumbar flexion, myoelectric signals of the erector spinae muscles were substantially smaller compared with quiet upright standing despite the need to develop posterior tissue tensions equivalent to erector spinae contractions exceeding 700 N on each side at 40° or more of flexion to maintain equilibrium. Based upon the data obtained and the biomechanical analyses performed in this study, it was clear that passive tissue forces are required to sustain the load requirements in the trunk flexion posture.

The resistance to the large flexion moments generated at the trunk has been further investigated by McGill and Kippers[6] who examined the loads on individual tissues during the performance of the FRP using an anatomically detailed model of the lumbar spine using vertebral displacement and myoelectric signals to estimate individual muscle and passive tissue force-time histories. The authors reported that, in full trunk flexion, loading of the interspinous and supraspinous ligaments and posterior annulus of the intervertebral disk in particular were found to be high relative to their failure tolerances. Flexion of the trunk places the posterior passive elements of the spine at risk for failure, which is consistent with injury mechanisms in many patients with low back pain. McGill and Kippers[6] further noted that although many muscles show force generation consistent with electrical activity from neural activation, forces are still predicted in the lumbar extensor muscles because of passive stretching far beyond their resting length in the trunk-flexed posture, enabling the muscles to generate forces in the absence of neural activation. Thus, this was the first study to propose that although the erector spinae muscles become

electrically silent during trunk flexion, they still appear to be generating force elastically through passive stretching.

Dolan et al[23] further investigated the passive tissue contributions of the spine during lifting in the trunk flexion posture in 149 healthy subjects. In their study, subjects in the trunk-flexed posture pulled upward with steadily increasing force on a floor-mounted load cell, whereas EMG activity was recorded from the erector spinae muscles at L3 and T10. Extensor moment was calculated from the load cell data and was plotted against the averaged and full-wave rectified EMG data. A linear relationship was observed with an intercept on the extensor moment axis (M_a) that indicated that flexion moment was resisted by passive tissues. The dependence of M_a was further studied by having the subjects repeat the isometric pulls at varying amounts of trunk flexion. The isometric pulls showed that, on aver age, 3- to 4-fold increases in M_a occurred for the full flexion posture from the lordotic posture, corroborating the findings of McGill and Kippers[6] for risks for injury in the flexed posture.

Others too have contributed to the understanding of the biomechanical consequences of the FRP during upright trunk flexion. Gupta[18] measured differences in myoelectric signals by experimentally inducing abnormal combinations of hip and trunk movements by having subjects bend forward with the pelvis held against a wall, which prevented posterior migration of the pelvis and limited the pelvic component of trunk flexion. In addition, subjects bent forward with weights tied posteriorly around the iliac crest, restricting pelvic movement, and with weights in their hands. During these tasks, EMG signals were obtained from surface electrodes placed on the erector spinae (at L3), rectus abdominis, hamstrings, and gluteus maximus muscles. A video camera captured the motion of the trunk flexion via markers placed on each subject and video was synchronized with the EMG data. The FRP was observed in all 25 subjects examined during quiet forward bending at 57% and 84% of mean maximum hip and vertebral flexion, respectively. Strong electrical activity was observed in the hamstring muscles of all subjects during the flexion task, and 18 of 25 subjects showed mild activity of the gluteus maximus muscles during trunk flexion. The FRP was seen to come much earlier ($P < .001$) at an average hip and vertebral flexion of 28% and 75%, respectively, of the maximum flexion value when forward bending was performed with the buttocks held against the wall. This finding is consistent with the additional stability provided to the pelvis, thereby creating greater passive forces earlier in the flexion range of motion, further supporting the rationale for myoelectric silencing of the erector spinae muscles. The addition of weights placed either anteriorly or posteriorly was found to increase the tensile torque about the spine, requiring the erector spinae to remain active through a longer range of motion until the extension torque by the posterior vertebral elements is increased proportionally enough to reach equilibrium.

In another work, to understand the contributions of other muscles during trunk flexion, Andersson et al[19] used fine wire electrodes to measure the electrical activity of the quadratus lumborum (QL), the deep lateral and superficial medical lumbar erector spinae, the psoas, and the iliacus muscles during the FRP. The QL showed an increased involvement from erect standing to full forward flexion of the trunk. In the latter part of the trunk flexion, a cessation of activity was observed for the superficial medial portion of the lumbar erector spinae but not in the deep lateral lumbar erector spinae muscles. Based upon the results of this study, the common interpretation that extensor torque required at the lumbar spine to balance gravity being accomplished only by the passive tissues (ie, disks and ligaments) was found not to be entirely true. Muscles such as the QL and deep lateral erector spinae indeed assist in the load sharing for spinal stability in the flexed trunk posture. Knowledge of muscle activation patterns during trunk motions assists biomechanists and clinicians in understanding mechanisms of injury and providing direction in training and treatment regimens.

Clinical Studies on the FRP

Bridging the gap between the biomechanical research and clinical studies of the FRP was perhaps best accomplished by Kaigle et al[24] who simultaneously quantified the muscle activation patterns, the kinematic behavior of the lumbar motion segment, and the overall trunk flexion during dynamic flexion-extension in 7 patients with CLBP and 6 asymptomatic control subjects. Kaigle and coworkers[24] performed invasive measurements in vivo by attaching interosseous pins into the spinous processes of lumbar motion segments. To the pins, a linkage transducer system allowed for dynamic measurement of intersegmental lumbar motion in the midsagittal plane with accuracies of 0.4° and 0.14 mm for rotatory and translatory movements, respectively.[25] Electromyographic activity of the erector spinae muscles was simultaneously recorded from surface electrodes placed bilaterally at the level of L3 through L4.

With this experimental setup, subjects were asked to stand erect in quiet stance followed by bending forward in the sagittal plane to maximum trunk flexion, followed by extension to the upright neutral posture. Significant differences between the patients and asymptomatic subjects were observed throughout the flexion tasks. In the asymptomatic group, the authors found a 78% decrease in the root-mean-square EMG activity at full flexion, indicating the FRP. Conversely, in the patient group, 4 of the 7 patients showed no decline in erector spinae muscular activity at full flexion, and in the remaining 3 patients, two showed a less than 18% reduction in activity, whereas the third patient experienced a 60% reduction in activity at full flexion. Overall, the mean reduction for the patient group was 13%. Kaigle et al[24] further reported that the patients with CLBP

were significantly limited in their ability to flex and extend the trunk compared with the asymptomatic group.

Data obtained from the spinal linkage system revealed that sagittal rotation and axial shear translation were significantly less in the patients than in the control subjects. The authors reported that the restricted intervertebral motion in the patient group as well as trunk flexion may have been due to the persistent activation of the musculature. In this manner, the muscles would behave more as stabilizers to compensate for joint laxity in an injured or diseased motion segment to protect these spinal structures from movements that may cause pain and/or further tissue damage.[26] Because subjects in both groups were able to flex greater than 70° and previous study has shown that the FRP is seen approximately at this trunk angle in asymptomatic subjects,[9,12] it is likely that the lack of the FRP in the patient group was not due to insufficient trunk flexion.

It is likely that the muscular alterations responsible for the absence of FRP in persons with back pain is related to their clinical status, namely, pain and dysfunction. The underlying rationale lies in the sensory innervation of spinal ligaments and their direct interactions with the surrounding spinal musculature. Although ligament has been traditionally categorized as a mechanical structure responsible for joint stability, they have equally important sensory functions. Neuroanatomic and neurophysiologic studies show that spinal ligaments and intervertebral disks are endowed with mechanoreceptors and nociceptive afferents[27-31] to signal joint loads, motion, and the presence of inflammation in the latter. Ligamentomuscular reflexes have subsequently been established between the spinal ligaments and disks, and surrounding muscles, which act to directly or indirectly modify the load imposed on spinal ligaments through muscles.[32-35] Such reflexes may be inhibitory or excitatory as may be required to preserve joint stability: inhibiting muscles that destabilize the joint or increasing antagonist coactivation to stabilize the joint.[36] In fact, Solomonow et al[14] recently described a neuromuscular disorder composed of 5 distinct components associated with static loads imposed to the spine. This model sheds light on theoretical mechanisms supporting the lack of FRP in patients with low back pain. The first component consists of a gradual decrease in reflexive muscular activity directly related to the creep developed in the ligaments, eliciting a shift in the sensory trigger threshold of the reflex.[37] The second component consists of spasms observed during the static loading (lumbar flexion) period, caused by microdamage in the ligamentous collagen fibers and subsequently relayed nociceptively. The third component was observed in the first hour of rest after a static loading period, expressed as a transient hyperexcitability of reflexive muscular activity. Fourth, a relatively prolonged reflex muscular hyperexcitability (2- to 3-fold) that gradually increased from the second to sixth hour of rest after static loading has been observed correlated to the consequent ligamentous inflammation.[37] The fifth component is the slow exponential recovery of the EMG to its normal (initial) level as rest time progresses.

The neuromuscular model as described by Solomonow et al[14] may indeed represent the musculoskeletal dysfunction experienced by those with back pain and absent FRP. More recently, the effects of static flexion-relaxation on paraspinal reflex behavior have been reported.[38] Reflexes showed a trend toward increased gain after a period of flexion-relaxation and were increased with trunk extension exertion.

Other studies have investigated the reliability of detecting the FRP and its ability to discriminate between subjects with back pain and asymptomatic control subjects. Watson et al[39] assessed the test-retest reliability of the FRP measure in a group of patients with CLBP (n = 11) and further compared the results between a group of healthy control subjects (n = 20) and a group of patients with CLBP (n = 70). In their study, Watson and coworkers[39] developed a flexion-relaxation ratio of the activity during the neutral and fully flexed positions by taking the mean rectified mean square (RMS) EMG values during a 15-second resting standing period, RMS of the maximal activity during 1 second of activity during forward flexion, RMS activity for 1 second in the fully flexed position, and the RMS for the maximum activity during the reextension movement. Using a repeated measures design of 2 testing sessions 4 weeks apart, intersession reliability was found to range between 0.81 and 0.98 for EMG activity assessments. In the second experiment, the flexion-relaxation ratio was compared among the patients with CLBP and control subjects. The researchers reported that the flexion-relaxation ratio was significantly greater in the fully flexed trunk position in the CLBP group than in the control subjects, clearly discriminating the patients from the healthy control subjects in this study.

In related work, Shirado and coworkers[9] also found that the FRP could discriminate between patients with chronic back pain and healthy subjects in their study of 25 healthy subjects and 20 patients with CLBP. Full wave rectified and averaged values of the EMG signal amplitudes were derived for the neutral upright and trunk-flexed postures, and based upon the averaged values of EMG activity during the relaxed erect position, changes (percentage) of EMG activities were calculated and compared between the groups. The authors found that all 25 healthy control subjects exhibited the FRP. Conversely, no patients with CLBP revealed the FRP during the flexed posture. Because of the significant difference in erector spinae EMG activity observed between the groups and a time lag between trunk and hip motion being greater in patients than in control subjects, Shirado et al[9] concluded that neuromuscular coordination between trunk and hip function is altered among patients with CLBP.

Another study examining 40 patients with chronic back pain and 40 control subjects also showed a lack of FRP in patients with low back pain.[40] In this work, Ahern et al[41] noted that pain behavior, more specifically, guarded movements, was significantly correlated to the FRP. Adding to this

line of investigation of assessing the FRP, Mannion et al[5] investigated 148 patients with CLBP and found that 55% showed no relaxation of the erector spinae muscles at L5 in the fully flexed position. Admittedly, the variance between the results of Mannion et al[5] and those of studies previously cited indeed may lie in their choice of electrode location and methodology of EMG analysis. Nevertheless, this study corroborates the findings of others revealing an absence of the FRP among patients with CLBP.

Recently, Neblett and coinvestigators[15] performed a two-part investigation of 12 asymptomatic subjects in an intra- and interrater repeated-measures protocol to examine the reliability of EMG readings of the FRP and further compared 54 patients with CLBP for differences in FRP responses before and after a spinal rehabilitation program. The authors reported that the ability of clinicians to measure range of motion and EMG reliably during the flexion task was high ($r > 0.92$, $P < .001$). Of further interest, all asymptomatic subjects achieved the FRP at mean EMG signals of 2.3 μV. Only 30% of the patients with CLBP achieved the FRP pretreatment; however, after a 7-week rehabilitation program, 94% of the patients with CLBP were able to achieve the FRP. This study not only showed the reliability of the FRP assessment procedure but also was the first study to systematically show that an absence of the FRP in patients with CLBP could be corrected with treatment.

Conclusions

The biomechanical consequence of the FRP is to accommodate the transfer of loads to the passive elements of the spine to achieve equilibrium. From a review of the biomechanical literature relevant to the FRP, the presence of the FRP during trunk flexion represents myoelectric silence consistent with increased load sharing of the posterior discoligamentous passive structures.[7] The myoelectric silence of the erector spinae muscles per se, however, may not mean that these muscles are not providing forces themselves from passive stretching as previously thought[6] nor that other lumbar spinal muscles do not assist in load sharing, namely, the QL and deep erector spinae muscles.[19] The myoelectric silencing of the erector spinae muscles in the trunk flexion posture, although not exclusive, may be indicative of increased load sharing on passive structures,[6,7,18,23] which tissues have been found to fail under excessive loading conditions,[42] and showed to be a source of low back pain.[43]

From the research reviewed, there is clinical significance to the presence or absence of the FRP. A number of studies have shown differences in the FRP between patients with CLBP and healthy individuals, and the reliability of the assessment. Persistent activation of the lumbar erector spinae musculature among patients with back pain represents the body's attempt to stabilize injured or diseased spinal structures thereby protecting them from further injury and avoiding pain. Noteworthy is that few clinical tests show 100% correlation to the disease or disorder. The studies that show differences in the presence of the FRP among patients and control subjects are encouraging for this type of clinical assessment and suggest that assessment of the FRP is a valuable clinical tool to aid in the diagnosis and treatment of patients with CLBP. Further study into the response of different patient populations, including those patients with acute low back pain and sciatica, and the effects of different rehabilitation strategies will serve to improve the body of knowledge relevant to the clinical utility of the FRP.

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