Pain-Related Fear, Lumbar Flexion, and Dynamic EMG Among Persons With Chronic Musculoskeletal Low Back Pain

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Objectives: The purpose of this study was to examine the relationship between pain-related fear, lumbar flexion, and dynamic EMG activity among persons with chronic musculoskeletal low back pain. It was hypothesized that pain-related fear would be significantly related to decreased lumbar flexion and specific patterns of EMG activity during flexion and extension.

Study Design: Data was obtained from subjects who, on a single day, completed self-report measures of pain and pain-related fear, and were interviewed to determine demographic and pain information. Subjects then underwent a dynamic EMG evaluation for which they were asked to stand, then bend forward as far as possible, stay fully flexed, and return to standing. Lumbar EMG and angle of flexion were recorded during this time. A flexion-relaxation ratio (FRR) was computed by comparing maximal EMG while flexing to the average EMG in full flexion.

Subjects: Seventy-six persons with chronic musculoskeletal low back pain.

Results: Zero-order correlations indicated that pain-related fear was significantly related to reduced lumber flexion ($r = -0.55$), maximum EMG during flexion ($r = -0.38$) and extension ($r = -0.51$), and the FRR ($r = -0.40$). When controlling for pain and demographic factors, pain-related fear continued to be related to reduced lumbar flexion. Using a path-analytic model to examine whether angle of flexion mediated the relationship between fear and EMG activity, the models examining maximal EMG during flexion and extension supported the notion that pain-related fear influences these measures indirectly through its association with decreased range of motion. Conversely, pain-related fear was independently related to higher average EMG in full flexion, while angle of flexion was not significantly related. Pain-related fear was directly related to a smaller FRR, as well as indirectly through angle of flexion.

Conclusions: Pain-related fear is significantly associated with reduced lumbar flexion, greater EMG in full flexion, and a smaller FRR. The relationship between pain-related fear and EMG during flexion and extension appears to be mediated by reduced lumbar flexion. These results suggest that pain-related fear is directly associated with musculoskeletal abnormalities observed among persons with chronic low back pain, as well as indirectly through limited lumbar flexion. These musculoskeletal abnormalities as well as limited movement may be involved in the development and maintenance of chronic low back pain. In addition, changes in musculoskeletal functioning and flexion associated with pain-related fear may warrant greater attention as part of treatment.

Key Words: pain-related fear, flexion-relaxation phenomenon, EMG, lumbar flexion, chronic low back pain

(*Clin J Pain* 2004;20:61–69)

Chronic low back pain (CLBP), particularly of musculoskeletal origin, has become a major focus of concern for both employers and health care professionals. Although several factors have been proposed to be responsible for or to perpetuate CLBP, little is known about the disorder. Several theories have been advanced to explain the disorder, and research addressing the tenability of each one has been mixed. One major theory, the biomechanical model, proposes that CLBP may be the result of muscle asymmetries or abnormally low levels of muscle activity.[1] This theory suggests that abnormal electromyographic (EMG) activity is a result of poor posture and guarding that develops in response to an original injury.[2] Several investigators proposed that this irregular muscle activity contributes to spinal instability, which in turn enhances the potential of infringement upon nerve endings and thereby produces pain.[1,3,4] It has also been suggested that pain may also originate from muscle secondary to microcontractures as in myofascial pain,[5] as well as other structures such as the disks, joints, and ligaments.[6–8]

Another major theory of chronic back pain is the reflex-spasm model. While proponents of this theory debate the relative importance of physical or psychosocial stressors in the

Received for publication April 14, 2002; first revised August 25, 2002; second revised October 2, 2002; accepted October 5, 2002.

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This research was supported by grant HD35893 from the National Center for Medical Rehabilitation Research, National Institute of Child and Human Development, National Institutes of Health.

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development of CLBP, one or both of these factors are believed to contribute to an ongoing cycle of reflex-spasm.[1,9–11] The model that emphasizes the contribution of psychosocial stress to reflex-spasm has been referred to as the diathesis-stress[12] or stress-hyperactivity model of chronic pain.[13]

Evidence for these various theories is mixed. Studies supporting the biomechanical model have found that persons with CLBP display lower EMG levels compared with controls while sitting and during movement,[3,13–15] as well as abnormal muscle patterns or asymmetries compared with controls.[2,16,17] In support of the reflex-spasm model, studies have found increased EMG in response to painful stimulation, and greater EMG in static postures.[9,17,18] Research has also demonstrated that persons with CBP have elevated EMG during dynamic activity, greater EMG in response to stress, and higher left-right asymmetries.[19–22] Additional studies have failed to support the reflex-spasm model.[10,23]

Several methodological issues have been raised to explain these discrepancies, including heterogeneous groups of patients with different pathology and pain in different sites.[24] Many authors encourage the evaluation of dynamic EMG activity, as EMG in static positions may not be representative of what occurs during activities of daily living.[25] Two studies have examined continuous ambulatory monitoring of EMG activity in persons with CBP. One found that paraspinal muscle activity did not differ between persons with CBP and controls, nor was it related to self-reported pain intensity.[26] Geisser et al[27] found a significant relationship between physical activity and pain among persons with CLBP, as well as an association between perceived stress and pain. No relationship between EMG activity and pain was observed.

One pattern of abnormal muscle activity that has consistently been shown to be associated with CLBP is an absence of relaxation of the lumbar paraspinal muscles in full-flexion. In healthy persons who are asked to stand quietly, the surface EMG signals from the paraspinal muscles are generally low. As a person bends forward, EMG activity increases as the muscles support the trunk at a greater angle to gravity. Near full flexion, ligamentous support becomes more important and lumbar EMG activity drops, often to a level less than the activity recorded when standing upright. This paraspinal relaxation on terminal flexion, first reported in the late 1940s by Allen,[27] is known as the flexion-relaxation phenomenon (FRP). Many studies conducted among persons with CLBP indicate that the FRP is absent in this population.[3,28–30] Ahern et al[3] reported that the presence or absence of the FRP correctly identified 86% of persons with CLBP and healthy controls, and Watson et al[8] reported a similar rate of 89%. This response has been shown to be significantly related to disability ratings and pain behavior among persons with CLBP,[31–32] and has been shown to be sensitive to spinal pathology such as disk herniation.[33] Studies indicate that the FRP has good test-retest reliability, even in clinical populations.[8,34]

Little is known about the factors that contribute to the absence of the FRP in persons with CLBP. Ahern et al suggested that the absence of the FRP in persons with CLBP may be a result of limited lumbar flexion.[3] Most argue that limited flexion is not responsible for this finding, as persons with CLBP can generally reach 40 degrees of flexion, where the FRP is believed to begin.[31] However, some observe that there is substantial variability in the angle where the FRP starts.[3,31] In addition, due to altered body mechanics, persons with CLBP have been observed to display only 27 degrees of true lumbar flexion.[3] Alternatively, it has been proposed that muscle EMG abnormalities may be a response to pain, as pain has been shown to have some influence on muscle firing.[35] However, two studies failed to find a relationship between pain state and EMG among persons with CLBP.[3,36] Finally, it has been suggested that changes in paraspinal activity may be a compensatory response to improve spinal stability and decrease pain during movement.[8,35]

Several authors have suggested that EMG abnormalities in persons with back pain may be due to pain-related fear or volitional guarding during movement.[3,8] It has been demonstrated that persons with CLBP display abnormal muscle activity while watching a videotape of persons performing strenuous activity.[37] The authors predicted that muscular reactivity would be greater for persons high in pain-related fear, and that this effect would be moderated by negative affect. Unexpectedly, the authors found that subjects responded to the videotape with decreased muscle tension, although subjects high in pain-related fear tended to display less of a decrement compared with persons with CLBP who reported low fear. Vlaeyen and Linton[38] propose that pain-related fear and resulting muscular reactivity may be related to the development and maintenance of CLBP.[38] This assertion seems reasonable given that pain-related fear has been shown to be consistently related to disability among persons with CLBP, and has also been found to be a potent predictor of chronicity among persons with acute low back pain.[39]

Watson et al[40] examined the relationship between pain-related fear and the flexion-relaxation ratio (FRR), defined as the ratio of maximal muscle activity in flexion to the average EMG activity in full flexion, among persons with CLBP. The authors found a significant, inverse relationship between pain-related fear and the FRR, whereby persons with higher pain-related fear tended to display a lower ratio. In addition, the authors found a significant increase in the FRR following a pain management program, which was significantly associated with decreased pain-related fear. Changes in range of motion or self-report of pain as a function of treatment were not related to changes in the FRR. The authors concluded that pain-related fear promotes muscle guarding during flexion, which in turn contributes to the development and maintenance of chronic pain. However, the authors did not examine how fear was related to each component of the FRR, as previous studies have

suggested that persons with CLBP demonstrate reduced EMG during flexion as well as higher EMG activity in full flexion.[3,8]

In the present study, we wished to directly examine the relationship between pain-related fear, as measured by the Tampa Scale of Kinesiophobia-2 (TSK-2),[41,42] and EMG activity and angle of flexion among persons with CLBP. Extending the findings of Watson et al,[40] we wished to determine how pain-related fear influences the components of the FRR (maximum EMG in flexion and average EMG in full flexion) as well as the FRR itself. We hypothesized that persons with CLBP who demonstrated higher pain-related fear would display decreased EMG levels in flexion and extension, greater EMG levels in full flexion, and a lower FRR compared with persons with CLBP who exhibited low pain-related fear. In addition, we hypothesized that pain-related fear would be significantly associated with decreased flexion. Lastly, we wished to examine whether angle of flexion might account for any observed relationship between pain-related fear and lumbar EMG by statistically examining the independent influence of pain-related fear and angle of flexion on EMG activity. As pain-related fear has been shown to be associated with restricted movement,[43] it is possible that fear may be related to abnormal EMG activity through limited movement or flexion.

MATERIALS AND METHODS

Subjects

Participants were 76 persons with chronic (3 or more months) musculoskeletal low back pain (as diagnosed by their treating physiatrist) who presented to the University of Michigan Spine Program for evaluation and treatment. Subjects were recruited to participate in a larger study examining the impact of manual therapy and specific adjuvant exercise on CLBP. Informed consent was obtained from all subjects, and the study methods were approved by the Institutional Review Board. Participants had a mean age of 40.6 years (SD = 11.9), and a mean duration of pain of 85.5 months (SD = 103.8). Thirty-four were male, and 42 were female. Twenty percent of patients were receiving some type of compensation related to their pain, and 10.5% were involved in litigation. Thirteen persons had previous lumbar surgery (laminectomy or discectomy). Forty persons were working full or part-time, while 29 were not working due to pain. Two persons were full-time students, 1 was a homemaker, and 4 were retired for reasons other than their pain. On the average, subjects in the sample completed 14 years of formal education (SD = 2.6). Sixty-six persons were Caucasian, 7 were African-American, 2 were Asian-American, and 1 was Hispanic.

Measures

Pain and Demographic Information

Participants were asked about their duration of pain, education, race, and other sociodemographic information such as litigation and compensation status after informed consent was obtained. For the correlational analyses, categorical variables were dummy coded. Compensation and litigation status, and surgical history were coded 0 for no, 1 for yes. For sex, males were coded as 0, and females as 1.

Pain

Subjects completed the McGill Pain Questionnaire (MPQ)[44] on the first day of assessment. The MPQ measures subjective pain experience in a quantitative form, and consists of 20 groups of single word pain descriptors with the words in each group increasing in rank order intensity. The Total PRI was used in the present study as the measure of self-reported pain intensity. Repeat administration of the MPQ has revealed a 70.3% rate of consistency in the PRI score.[44]

Self-report of clinical pain intensity was also obtained from subjects by having patients rate the highest, lowest, and average or usual pain during the past week. Participants were asked to rate the highest, lowest, and average level of pain on three separate 10 cm Visual Analog Scales (VASs) anchored by the statement "no pain" on the left and "the most intense pain imaginable" on the right. VAS pain ratings have demonstrated good reliability[45,46] and concurrent validity when compared with other methods of pain measurement.[47,48]

Pain-Related Fear

Beliefs that pain is a signal of serious damage to the body, and that activities that cause pain should be avoided were assessed using the TSK-2.[42] The measure is a 13-item version of the original scale[41] where persons are asked to rate their level of disagreement or agreement with each item on a 1 (strongly disagree) to 4 (strongly agree) scale. A total score for the scale is obtained by summing the items. Internal consistency (Cronbach's alpha) for the scale has been reported to be high (0.86).[42] Factor analysis of the scale suggests that items measure 2 constructs, reflecting activity avoidance and a pathologic somatic focus or fear regarding the meaning of pain. The total score was used in the present study.[42,49]

Dynamic EMG–Flexion-Relaxation Ratio

Subjects underwent a dynamic EMG evaluation and calculation of the FRR.[3,8] EMG activity was measured using the Back Flexion Monitor (Measurement Systems Inc., Ann Arbor, MI). The device measures two channels of EMG that record the rectified mean square (RMS) activity with a time constant of 55 milliseconds. The signals are band pass filtered from 30 to 1000 Hz prior to RMS conversion. The device has an adjustable gain and signals the examiner to adjust it if the peak signal output from a channel is above 5 volts, or if the signal drops 1% (0.05 volts) below the scale. Thirty-two by 22 mm pre-gelled silver/silver chloride electrodes were placed on both the right and left sides of the spine approximately 3 cm from the midline. One electrode was centered at L3 vertebral body, and the other at the L5 vertebral body. To measure flex-

ion, a goniometer was placed on a Velcro® strap that was wrapped around the torso just below the axillae.

Once the equipment was placed on the subjects, they were asked to go through a series of movements verbally led by the examiner. Subjects were asked to stand upright for 2 seconds (Phase 1), then bend as far forward as they could to the count of two (Phase 2). As head position has been suggested to influence EMG activity during the task,[8] subjects were asked to tuck their chin into their chest as they bent forward. During Phase 3, subjects were asked to stay in full flexion for 2 seconds, then return to standing over a period of 2 seconds (Phase 4). Data collection was performed over a period of 10 seconds to assure that all the data was captured. Subjects performed 5 trials, and data from the first trial was discarded as it was treated as a practice trial. The gain was adjusted during this trial to produce a signal within allowable limits. Additional practice trials were performed as needed to obtain an adequate signal. The gain was kept constant for remaining trials.

The data for each subject were then divided into the 4 phases described above. The data was visually inspected for integrity, and interference (spikes) were removed. EMG signals from the left and right sides were averaged. Each phase was defined based on the goniometer reading. Any change or stabilization in the angle by two or more degrees constituted a change in the phase. To control for differences in amplification, the influence of static EMG on dynamic values, and other individual differences such as obesity that might influence the surface EMG signal, the data were normalized by dividing the data for a phase by the average EMG during Phase 1. A similar method is reported by Shirado et al.[29] Maximum (MAX) EMG during flexion was calculated by dividing the MAX EMG in Phase 2 by the average EMG from Phase 1. The average (AVE) EMG in full flexion was calculated by dividing the AVE EMG during Phase 3 by the AVE EMG in Phase 1. Finally, the MAX EMG in extension was calculated by dividing the MAX EMG in Phase 4 by the AVE EMG in Phase 1. The FRR was computed by dividing the normalized maximum EMG during flexion by the normalized average EMG in full flexion. This is the ratio typically reported in previous studies that, however, did not employ normalization of EMG values.[3,8,40]

Procedure

Data for the current experiment were obtained on the first day of testing from the larger study. Informed consent was obtained from each subject, and each subject was then interviewed to obtain demographic and pain information. Subjects were told that the purpose of the testing was to examine changes in pain and other self-report measures, as well as changes in muscle function while bending as a function of treatment. Subjects then underwent dynamic EMG testing, and completed self-report measures of pain, pain-related fear, disability, and other instruments used in the larger study. Questionnaires were checked for thoroughness of completion.

RESULTS

Prior to examining the relationships between the EMG, flexion data, and the variables of interest, these data were checked for normalcy. Visual inspection of the EMG data suggested that distribution of scores were positively skewed. A Kolmogorov-Smirnov Goodness of Fit Test was conducted on the normalized EMG Data. The Kolmogorov-Smirnov Z statistics for the MAX EMG in flexion ($Z = 2.3$, $P < 0.001$), AVE EMG in full flexion ($Z = 2.3$, $P < 0.001$), MAX EMG in extension ($Z = 1.6$, $P < 0.05$), and the FRR ($Z = 2.3$, $P < 0.01$) were all statistically significant, suggesting the distributions were not normal. The Z statistic for MAX angle of flexion suggested that this distribution was normal. The normalized EMG data were then subjected to a logarithmic transformation to attempt to normalize the distributions, and repeat Kolmogorov-Smirnov Goodness of Fit Tests indicated that the data transformations were successful. The sample means for the normalized, log transformed EMG data, and maximum angle of flexion are presented in Table 1.

Zero-order correlations between the pain and demographic information, TSK-2 scores, and EMG and flexion data are presented in Table 2. As the associations between MPQ scores and VAS scores were similar, analysis of MPQ scores was dropped. We were particularly interested in examining the relationship between fear and EMG and angle of flexion, in addition to pain and demographic variables that might be related to EMG activity, as these would need to be controlled for in later analyses. Age was negatively associated with MAX EMG during extension ($r = -0.26$, $P < 0.05$) as well as a lower FRR ($r = -0.23$, $P < 0.05$). Gender was also associated with

TABLE 1. Sample Means and Standard Deviations for Pain Intensity Measures, Pain-Related Fear, Angle of Flexion, and Log Transformed EMG Data

Variable	Mean	Standard Deviation
Average VAS	4.9	2.3
MPQ total PRI	26.7	12.4
TSK-2 total score	31.3	7.6
MAX angle of flexion	100.3	29.1
MAX EMG flexion	2.16	0.9
AVE EMG full flexion	0.84	0.8
MAX EMG extension	2.34	0.9
FRR	1.31	0.8

MAX = maximum; AVE = average; FRR = flexion-relaxation ratio; TSK-2 = tampa scale of kinesiophobia; MPQ = McGill pain questionnaire; PRI = pain rating index.

TABLE 2. Correlations Between Demographic and Pain Variables, Pain-Related Fear, and Maximum Flexion, and EMG Activity

Variable	2	3	4	5	6	7	8	9	10	11	12	13
1. Age	−.11	.34†	.07	−.05	−.11	−.16	.00	−.13	−.16	.06	−.26*	−.23*
2. Sex		.07	−.15	.02	.00	−.12	−.14	−.36†	−.33†	.18	.35†	.14
3. Pain duration			−.10	−.17	.09	−.18	−.29*	.15	−.06	−.07	−.09	.03
4. Surgery				.01	.13	.07	.20	−.14	−.22	−.11	−.29*	−.14
5. VAS					.39†	.32†	.53†	−.45†	−.23*	−.16	−.30†	−.15
6. Compensation						.09	.13	−.31†	.00	.10	−.03	−.13
7. Litigation							.36†	−.40†	−.27*	−.19	−.30†	−.06
8. TSK-2								−.55†	−.38†	.02	−.40†	−.45†
9. Angle flexion									.54†	.10	.67†	.51†
10. MAX flexion										.56†	.87†	.59†
11. MAX full flexion											.57†	−.34†
12. MAX extension												.43†
13. FRR												

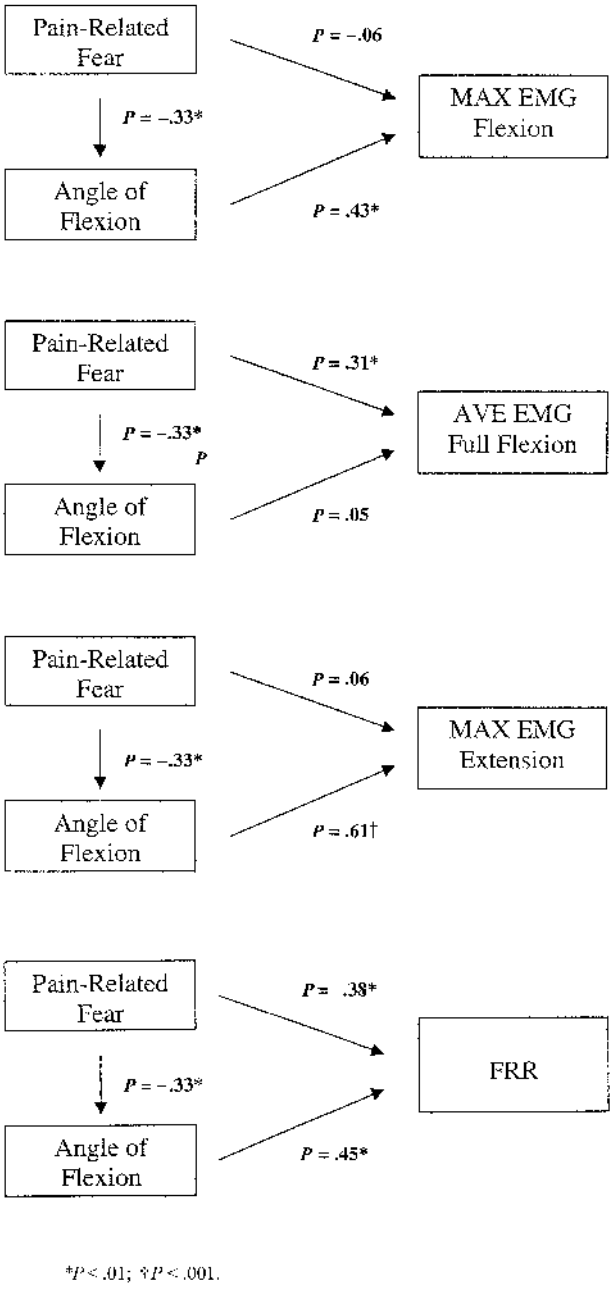
*$P < 0.05$; †$P < 0.01$; VAS, visual analog scale; TSK-2, Tampa scale of kinesiophobia; FRR, flexion relaxation ratio.

EMG activity and flexion, as males tended to display greater flexion (r = 0.36, $P < 0.01$) and greater MAX EMG during flexion (r = 0.33, $P < 0.01$) and extension (r = 0.35, $P < 0.01$). Pain duration was not significantly associated with EMG or flexion. Persons with previous lumbar surgery displayed significantly lower MAX EMG during flexion (r = −0.29, $P < 0.05$). Average VAS ratings were significantly and negatively associated with maximum angle of flexion (r = −0.45, $P < 0.01$), and MAX EMG during flexion (r = −0.23, $P < 0.05$) and extension (r = −0.30, $P < 0.05$). Persons receiving compensation were more likely to display lower angles of flexion while bending (r = −0.31, $P < 0.01$), as were persons involved in litigation (r = −0.40, $P < 0.01$). Persons in litigation were also significantly more likely to display lower MAX EMG in both flexion (r = −0.27, $P < 0.05$) as well as extension (r = −0.30, $P < 0.01$).

The TSK-2 total score was significantly and inversely related to MAX angle during flexion (r = −0.55, $P < 0.01$), MAX EMG during flexion (r = −0.38, $P < 0.01$), MAX EMG during extension (r = −0.40, $P < 0.01$), and the FRR (r = −0.45, $P < 0.01$).

To further explore the relationship between pain-related fear, angle of flexion, and EMG activity, a series of path models were constructed to examine whether pain-related fear influences EMG activity directly, or indirectly through its influence on range of motion. As several demographic and pain variables displayed significant zero-order relationships to angle of flexion and the various EMG measures, they were treated as exogenous variables in the path models and were controlled for statistically. Exogenous variables included age, sex, prior surgery, average VAS pain ratings, and compensation and litigation status. Path coefficients were calculated using multiple regression analysis.[50] The path coefficient examining the direct influence of pain-related fear on EMG activity was calculated as the regression beta weight controlling for the variables listed above in addition to maximum angle of flexion. The indirect pathway examining the mediating effect of angle of flexion was constructed by calculating the beta weight for the relationship between pain-related fear and angle of flexion controlling for the exogenous variables. In addition, a beta weight was computed for the pathway between maximum angle of flexion and EMG activity controlling for TSK-2 scores and the exogenous variables. Four separate models were constructed with MAX EMG in extension, AVE EMG in full flexion, MAX EMG in flexion, and the FRR as dependent measures, as pain-related fear demonstrated significant zero-order relationships with each EMG measure. The significance of the path coefficients was based on the *t* test of the significance of the beta weight in the regression.

The path models are presented in Figure 1. The models that examined maximum EMG during flexion and extension as the dependent variables suggested that the relationship between pain-related fear and EMG is mediated by angle of flexion. The direct path coefficient between TSK-2 scores and MAX EMG was nonsignificant for both maximum EMG during flexion ($\beta = -0.06$) and extension ($\beta = 0.06$). The indirect path coefficient between pain-related fear and angle of flexion was significant in each model ($\beta = -0.33$, $P < 0.01$), as were the paths between MAX EMG in flexion ($\beta = 0.43$, $P < 0.01$) and MAX EMG in extension ($\beta = 0.61$, $P < 0.001$). These results suggest that pain-related fear influences EMG during flexion and extension through its association with decreased range of motion. None of the exogenous variables were significantly related to maximum EMG during flexion in these



*P < .01; †P < .001.

FIGURE 1. Path analyses examining the relationships between pain-related fear, angle of flexion, and EMG activity.

analyses, while surgical status ($\beta = -0.18$, $P < 0.05$) and age ($\beta = -0.18$, $P < 0.05$) were significantly related to maximum EMG in extension while simultaneously controlling for all other variables in the model.

In the path model examining the relationship between pain-related fear and AVE EMG in full flexion, pain-related fear demonstrated a direct relationship to increased average EMG activity in full flexion ($\beta = 0.31$, $P < 0.05$). Although pain-related fear is significantly related to decreased angle of flexion ($\beta = -0.33$, $P < 0.01$), the path between angle of flexion and average EMG in full flexion was not significant. Examining the exogenous variables in the model, only pain intensity was significantly and negatively associated with average EMG in full flexion ($\beta = -0.34$, $P < 0.05$).

Examining the FRR as the dependent variable, the path model suggested that pain-related fear has both a direct and indirect influence on the ratio of maximum EMG during flexion to the average EMG in full flexion. The direct path coefficient is significant ($\beta = -0.38$, $P < 0.01$), indicating that pain-related fear is associated with a smaller FRR independent of angle of flexion. In addition, the model indicated that pain-related fear also has an indirect relationship to the FRR as TSK-2 scores were again significantly related to angle of flexion ($\beta = -0.33$, $P < 0.01$), and angle of flexion was also significantly and positively related to the FRR ($\beta = 0.45$, $P < 0.01$). Age was the only exogenous variable significantly related to the FRR, as older age was significantly associated with a lower ratio ($\beta = -0.25$, $P < 0.01$).

DISCUSSION

The findings of the present study indicate that pain-related fear is significantly associated with decreased lumbar flexion in persons with CLBP. Zero-order correlations indicated that TSK-2 scores were significantly associated with lower MAX EMG during flexion and extension, and a smaller FRR. Path analyses conducted to examine whether range of motion mediates the relationship between pain-related fear and EMG activity suggested that pain-related fear indirectly influences maximum EMG in flexion and extension through limited flexion. In addition, the path models suggested that pain-related fear was directly related to increased maximal EMG activity in full flexion, while angle of flexion was unrelated to this variable. When the FRR was examined as the dependent variable, pain-related fear displayed both a direct and indirect relationship to this variable. As clinical pain was controlled for in the path analyses, the results suggest that pain-related fear is associated with range of motion and EMG independent of pain intensity.

There are limitations to the present study. First, we only examined muscle activity among persons with CLBP and not normal healthy persons. Thus, it is difficult to determine whether the data reflect "abnormal" muscle activity. Second, pain during actual bending was not assessed. This may have been more highly associated with muscle activity compared with self-report of clinical pain intensity obtained before the dynamic EMG evaluation. Third, the study is cross-sectional and therefore does not allow for examination of cause-effect relationships.

Despite not assessing pain while bending, the findings are consistent with previous investigations that have not observed a significant relationship between pain and muscle ac-

tivity.[3,36] In one path model, average EMG in full flexion was inversely related to pain intensity, suggesting that clinical pain may inhibit muscle activity when in full flexion. In addition, Watson et al[40] found no relationship between present pain intensity just prior to bending and the FRR. Regardless, future studies should address whether relationships between pain-related fear, lumbar range of motion, and EMG are independent of pain experienced during the functional activity performed. In addition, it would be beneficial to examine how perceptions of muscle activity and range of motion also influence the data.

The present findings replicate the findings of Watson et al[40] in that a significant relationship was observed between pain-related fear and the FRR. Expanding on the findings of Watson et al,[40] the present results suggest that pain-related fear influences the FRR both through its association with maximal muscle activity during flexion, as well as increased muscle activity in full flexion. The relationship between maximal muscle activity and pain-related fear is mediated by decreased range of motion, while the relationship between fear and average EMG in full flexion was independent of range of motion. Thus, pain-related fear has both a direct and indirect influence on the FRR, and the path analysis examining the FRR as the dependent variable supported this conclusion. Watson et al[40] concluded that the relationship between pain-related fear and the FRR did not appear to be due to limited range of motion, as no significant changes in range of motion were observed before and after treatment in a pain management program despite significant increases in the FRR. However, the FRR was significantly associated with flexion at 2 sites measured prior to treatment, but not at two others. The data from the present study suggest that limited flexion among persons with CLBP may be responsible for some of the previous findings related to an abnormal FRP among persons with CLBP. Future studies examining dynamic EMG among persons with CLBP should consider the potential influence of angle of flexion on muscle activity, particularly during flexion and extension.

The present findings are consistent with the biomechanical model of chronic pain as well as previous studies that have demonstrated decreased paraspinal muscle activity during flexion among persons with CLBP, as well as increased EMG in full flexion.[3,8,29–31] The results suggest that persons with CLBP who are highly fearful not only guard when in full flexion, but also significantly limit movement. These data suggest that it may be important to address both EMG abnormalities and limited lumbar flexion as part of the treatment of CLBP.

It is possible that high levels of pain-related fear may lead to restricted range of motion and abnormal muscle utilization patterns that may increase the likelihood of developing CLBP. Indeed, it has been proposed that activity avoidance may lead to muscle atrophy, which in turn promotes pain.[51] These dysfunctional muscles may become inhibited over time, causing other muscles to compensate by becoming overactive.[52] Vlaeyen et al[37] found that muscles such as the anterior tibialis were overactive during exposure to stress among persons with CLBP. While this muscle is far removed from the paraspinals to compensate during flexion, it might be beneficial to examine other nearby muscles such as the iliosoas. In addition, Haig et al suggested that decreased lumbar paraspinal activity during flexion might be due to co-contraction of the abdominal muscles.[33] Monitoring of other muscles during movement might be beneficial in examining the types of muscle activity patterns that occur among persons with CLBP.

While previous studies have debated the influence of angle of flexion on EMG activity when examining the FRP, the results of the present study provide some direct examination of this issue. It appears that angle of flexion impacts maximal EMG during flexion and extension, but not EMG in full flexion. The finding that maximal EMG during flexion and extension is dependent on angle of flexion is consistent with research indicating a positive, linear relationship between peak torque and angle of extension on a task of isometric lumbar extension.[53] Angle of flexion may be unrelated to EMG in full flexion as most authors argue that myoelectric silence during flexion begins around 40 degrees of flexion, and most persons with CLBP are able to achieve this degree of flexion.

The conclusions regarding the direct influence of pain-related fear and EMG in full flexion are based on the results of the path analyses. The zero-order correlation between pain-related fear and EMG activity in full flexion was not statistically significant. The discrepancy between the zero-order correlation and the path analysis suggests that one of the variables in the path model acted as a suppressor variable. While a relationship between fear and EMG activity in full flexion was not specifically examined in the Watson et al[40] study, most studies examining the FRP in persons with CLBP and controls have consistently found elevated EMG in full flexion among persons with CLBP. It is possible that the normalization used in the present study may have contributed to this suppression, as normalization has not been typically employed in previous studies. It is also possible that the observed suppression effect is specific to this sample.

Most prior studies examining the FRP in relation to CLBP have not normalized EMG data. Whether or not EMG data should be normalized is debatable, although normalizing EMG, or controlling for factors that may systematically influence surface EMG, should be considered. As deconditioning and obesity have been reported to play a role in chronic pain and are known to influence surface EMG signals,[54] group differences in these variables may contribute to observed differences in EMG activity. In addition, within chronic pain populations, factors such as pain-related fear may systematically vary with variables that influence surface EMG. For example, it has been proposed that pain-related fear and activity avoidance lead to greater deconditioning among persons with CBP.[55] Methods used to normalize EMG in some studies, such

as examining EMG as a percentage of maximum voluntary contraction, may potentially be invalid in pain populations as pain and lack of effort may influence ability to give a maximum voluntary muscle contraction.[8,56,57] We chose to normalize the data relative to baseline or standing EMG to avoid problems that might arise with assessment of maximum voluntary contraction among persons with CLBP. In addition, this method potentially allows for control of any subtle or significant differences in static EMG activity on the dynamic EMG data.

It would be interesting to examine the impact of anti-spasm medications on muscle activity and movement among persons with CLBP in relation to pain-related fear. There is much debate regarding the physiological basis of muscle spasm, and anti-spasm medications have not been shown to have a direct impact on muscle activity.[58] It is possible that their clinical effectiveness may be related more to their psychoactive properties, particularly among patients high in pain-related fear.

In summary, pain-related fear appears to be highly associated with restricted movement and muscle activity among persons with CLBP. Future studies should examine this relationship not only in persons with CLBP but also in normal, healthy controls. The results of this study suggest that angle of flexion should be considered as a possible confound when examining EMG activity among clinical and normal populations. Future studies might benefit from monitoring other muscles during flexion to examine how altered lumbar EMG impacts firing in other muscles. Cause-effect relationships between pain-related fear, flexion, and muscle activity need to be established. Altering pain-related fear, restrictions in movement, and accompanying musculoskeletal changes may be beneficial in the treatment of CLBP.

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