

SPECIAL ISSUE

Surface Electromyography and Low Back Pain

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Objective assessment of low back pain (LBP), particularly chronic LBP, is difficult as objective pathology in the majority of cases is lacking, or pain is not highly associated observable pathology when it is present. This article describes a recent review of the ability of different surface electromyography (SEMG) measures to distinguish between persons with and without LBP. One SEMG measure, the flexion-relaxation ratio (FRR), appears to hold promise of being an objective maker of LBP. Possible factors that underlie the FRR in persons with LBP are discussed, and suggestions for future research are presented.

Low back pain (LBP) is highly prevalent and costly to society. It is estimated that 80% of all adults will experience at least 1 episode of LBP that impacts their ability to perform daily activities. In the United States, back pain is the second leading cause of work absenteeism and leads to more productivity loss than any other medical condition (Glazer & Glazer, 2002). In 1990, population surveys indicated that 9.4% of the population sought out health care for back pain, and back pain health care expenditures exceed \$33 billion each year. Total annual costs of low back disability in the United States have been estimated to be approximately \$50 billion, with the average cost of a single case of work-related back pain exceeding \$8,000 (Hazard, Haugh, Reid, Preble, & MacDonald, 1996). It has been estimated that 70%–80% of the costs for work-related low back claims are accounted for by 7%–10% of patients who develop chronic (persistent pain lasting 3 or more months) LBP (Fast, 1977; Spengler et al., 1986).

Diagnosis and treatment of LBP is extremely difficult as the majority of persons with the disorder lack objective indicators of pathology detected by tests such as magnetic resonance imaging (MRI). It has been estimated that as many as 85% of persons with LBP have no identifiable organic pathology (White & Gordon, 1982), and such persons are often given nonspecific diagnoses such as “nonspecific pain” or “chronic intractable benign pain.” Some authors suggest that many of these persons have evidence for musculoskeletal abnormalities that may contribute to their pain. For example, Lewit (1990) reported that over 90% of patients with “nonspecific pain”

have evidence for myofascial restrictions and dysfunctional (e.g., misaligned) joints. In a large open-ended clinical study, Rosomoff, Fishbain, Goldberg, Santana, and Rosomoff (1989) found that 43% of the patients evaluated in a 1-year period fulfilled the diagnostic criteria for chronic intractable benign pain. Of these patients, all had at least 1 physical finding suggestive of musculoskeletal dysfunctions, defined by the authors as tender/trigger points, decreased range of motion in the back or neck, nonanatomical sensory loss, rigid musculature, decreased range of hip motion, gait disturbance, and miscellaneous nonneurological signs. Despite this, musculoskeletal pain syndromes such as myofascial pain are somewhat controversial, and some dispute their existence (Thompson, 1996). Factors that contribute to the lack of acceptance of musculoskeletal causes of LBP include the lack of objective tests for many of these disorders, the absence of an accepted pathophysiological model of chronic muscle pain, and poor interrater reliability across clinicians in identifying specific musculoskeletal dysfunctions.

Several models of LBP have been proposed that focus on biomechanical and musculoskeletal factors in the etiology and maintenance of back pain. Such research often employs surface electromyography (SEMG) to examine the tenability of these models which propose that abnormalities in muscle activity contribute to development and maintenance of LBP. Despite the fact that SEMG assessment is simple and noninvasive, several concerns have been raised about its use in LBP populations. First, there are an infinite number of conditions under which SEMG can be studied in relation to LBP. SEMG can be collected during static postures or different dynamic movements, from multiple sites on the back, and from each side of the back. To date, there has been little study of the types of SEMG measurements and experimental conditions that are most characteristic of persons with LBP. Second, previous literature reviews have suggested that SEMG has little utility in diagnosing and treating back pain, as the technique is deemed to be inferior to other methods such as needle electromyography (Haig, Gelblum, Rechten, & Gitter, 1996; Pullman, Goodin, Marquinez, Tabbal, & Rubin, 2000). Finally, SEMG is prone to measurement artifacts such as impedance produced by

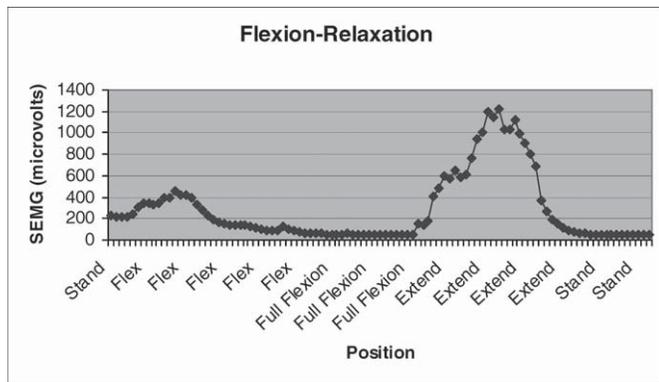


Figure 1. Lumbar surface electromyography (SEMG) in a person without low back pain during flexion and extension. During flexion, SEMG rises and falls to a low level when the person reaches full flexion.

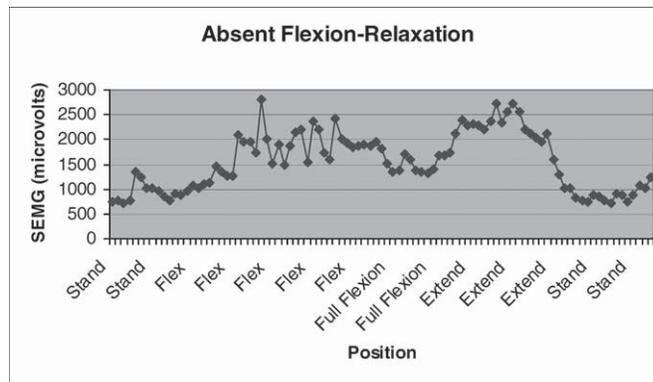


Figure 2. Lumbar surface electromyography (SEMG) in a person with low back pain during flexion and extension. During flexion, SEMG rises but does not significantly decrease when a person reaches full flexion.

the skin. Because of this, some recommend “normalizing” SEMG signals to a reference measurement taken from the same individual to reduce these artifacts. One common normalization method is to take an SEMG measurement during a maximal contraction as a reference and then calculate the SEMG levels during other target movements as a percentage of the maximal contraction. Such a reference in LBP populations may be biased, as persons with LBP often have difficulty putting forth maximal effort during tasks.

Recently, we conducted a meta-analytic review of studies examining the utility of SEMG to distinguish between persons with and without LBP (Geisser et al., 2005). Of the various methods and measures reviewed, several studies reported a consistently large effect size for an SEMG measure termed the flexion-relaxation ratio (FRR). The average standardized mean difference (*d*; the difference between the means divided by the pooled standard deviation) between persons with and without LBP was -1.71 . Assuming that the distribution of FRR scores is normal in both populations, this single measure alone could correctly identify 76% of the persons with and without back pain, with some studies reporting higher accuracy. Based on this literature review, this single measure holds promise as an objective marker of LBP. Combining this measure with other factors, such as lumbar range of motion, produced even greater sensitivity and specificity in distinguishing between persons with and without LBP.

The FRR is a quantitative measure of something known in the literature as the flexion-relaxation phenomenon (FRP). To assess the FRR, SEMG is measured in the lumbar paraspinal muscles dynamically, with the person first standing upright, then flexing fully forward, then holding a fully flexed position, and finally returning to a standing position. When healthy persons are asked to stand quietly, SEMG signals from the lumbar paraspinal muscles are generally low. As a

person bends forward, SEMG activity increases as the lumbar paraspinal muscles support the trunk at a greater angle to gravity. Near full flexion, ligaments take over the burden of supporting the trunk, and lumbar paraspinal SEMG activity drops, often to a level less than the activity recorded when standing upright. This paraspinal relaxation on terminal flexion was first reported in the late 1940s by Allen (1948). There is good evidence to suggest that the FRP in persons with LBP is absent. Instead, SEMG activity remains high when a person with LBP is in full flexion.

SEMG activity in healthy persons during the movements described above is presented in Figure 1. The methods used to assess the FRR in the graphs are described in detail elsewhere (Geisser, Haig, Wallbom, & Wiggert, 2004). Briefly, SEMG activity was measured using the Back Flexion Monitor (Measurement Systems Inc., Ann Arbor, MI). The device measures 2 channels of SEMG that record the rectified mean square activity with a time constant of 55 milliseconds. The signals were band pass filtered from 30 to 1000 Hz prior to rectified mean square 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% (.05 volts) below the scale. Thirty-two by 22 mm pregelled 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 flexion, a goniometer was placed on a Velcro strap that was wrapped around the torso just below the axillae.

As can be seen in the figure, SEMG activity increases as the person bends forward, and then drops. When the person is in full flexion, SEMG is low. SEMG increases as the person extends to stand upright, and then decreases when the person reaches an upright posture. The FRR is calculated as the ratio of maximum SEMG during flexion to the average SEMG in

full flexion. A high ratio is reflective of paraspinal relaxation at terminal flexion. For Figure 1, the maximum SEMG during flexion is 457 mV, and the average SEMG in full flexion is 54.6 mV. The FRR for this person is $457 \div 54.6$, or 8.4.

Figure 2 is characteristic of a person with an absent FRP or a person with LBP. During flexion, SEMG declines very little and remains high even when the person is in terminal flexion. This produces a lower FRR. The maximum SEMG for this person in flexion is 2802 mV, and the average SEMG in full flexion is 1457mV. The FRR for this person is then $2802 \div 1457$, or 1.9.

Researchers have begun to investigate the mechanisms that underlie the absence of an FRP in persons with LBP. Initially, it was suggested that the lack of FRP in persons with LBP was due to the fact that these persons have restricted range of motion. Thus, persons with LBP may not bend far enough to obtain paraspinal muscle relaxation. However, researchers suggested that paraspinal muscle relaxation generally begins when one obtains 40° of flexion, and that most persons with LBP generally have trunk flexion greater than 40°. For this reason, many dismiss this as the primary explanation for why the FRP is absent among persons with LBP.

Haig, Weismann, Haugh, Pope, and Grobler (1993) reported an interesting case study of a person who underwent SEMG assessment before and after an accident in which the subject suffered a herniated nucleus pulposus. Following injury, this subject had an absent FRP response that gradually returned after the subject's clinical symptoms subsided. The authors proposed that the decrease in range of motion, and loss of the FRP, was due to the subject having increased paraspinal muscle activity while standing. They indicated that this response may have been voluntary or involuntary, perhaps due to the subject "splinting" his back, or experiencing muscle spasms.

Other authors have suggested that the lack of FRP in persons with LBP is part of a volitional guarding response to limit movement of the back (pain-related fear) or a result of muscle adaptation to pain. Watson, Booker, Main, and Chen (1997) were the first to report a relationship between pain-related fear, or kinesiophobia, and the FRR among persons with chronic LBP. The authors reported a significant, inverse relationship between pain-related fear and the FRR, whereby persons with higher pain-related fear displayed a lower ratio (or less paraspinal muscle relaxation in full flexion). 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. Change in range of motion or self-report of pain as a function of treatment was 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.

Geisser et al. (2004) expanded on the Watson et al. (1997) study by examining the influence of pain-related fear on range of motion and SEMG activity during different phases of movement in persons with chronic LBP. We found that pain-related fear was significantly related to reduced lumbar flexion, maximum SEMG during flexion and extension, and the FRR. These relationships persisted even when controlling for clinical pain and demographic factors. Using statistics to model causal relationships, pain-related fear reduced SEMG during flexion and extension through its influence on decreasing range of motion. Pain-related fear was also associated with increased SEMG in full flexion, independent of range of motion. Finally, pain-related fear was associated with a lower FRR dependent on, and independent of its relationship with range of motion. These findings suggest persons with LBP who are higher in pain-related fear tend to restrict their range of motion and produce greater muscle activity when in full flexion. The research also indicated that clinical pain intensity is not related to the FRR, suggesting that the FRP is not solely a response to pain.

Objective indicators of LBP, such as the FRR, may serve as useful adjuncts in the study of musculoskeletal LBP and as objective markers of treatment outcome in this population. To the extent that abnormally elevated muscle tension in flexion serves as a marker for LBP, it would be interesting to examine whether training persons with LBP to reduce their paraspinal muscle activity in full flexion using methods such as SEMG biofeedback might help to remediate pain and functional limitations associated with LBP. However, it is unclear whether this can be done using such simple methods. If the FRR is associated with pain-related fear in patients with chronic LBP, interventions specifically designed to reduce pain-related fear may be more effective. Vlaeyen, DeJong, Sieben, and Crombez (2002) have recently outlined and tested an exposure intervention for reducing pain-related fear among persons with chronic LBP. Perhaps patients with LBP who are low in fear may benefit from minimal intervention, while patients who are high in fear, and/or who are more chronic, may require more intensive intervention.

Given that SEMG equipment and monitoring of lumbar range of motion is relatively simple and inexpensive, widespread assessment of the FRR in research and clinical practice seems feasible. At the present time, more research on the validity/utility of the FRR as an objective marker for LBP appears to be warranted.

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