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## EMG-Based Classification of Low Back Pain

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# **EMG-Based Classification of Low Back Pain**

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REPORT SUBMITTED IN FULFILLMENT OF  
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## **EXECUTIVE SUMMARY**

To make an impact in the rising number and associated cost with LBP, an accurate, objective and safe method is needed to assess lumbar impairment. To maintain a healthy spine, trunk muscles must function properly to stabilize the spinal column. With LBP, the ability of paraspinal muscles to resist fatigue is reduced, and the reactive response of trunk muscles is delayed. Two methods for classifying LBP based on these impairments were developed and assessed for accuracy in a preliminary study. Results indicate that assessment of reactive responses produced the most accurate method for classifying LBP. Based on this study, a more comprehensive study was undertaken to further develop and then validate a reaction classification model. Once again, clear differences in reaction times were observed between patients and healthy controls. In particular, patients had longer latencies and more variability in their responses to sudden unloading than healthy controls. Using the average reaction time, a model was developed that was able to accurately classify people with and without LBP. Because the reaction times measured during the study indicate that the responses were reflexive in nature and not voluntary, it appears that the protocol is objective and cannot be affected by motivational issues or fear of injury. Due to the small magnitude of perturbation force and displacement generated during testing, the protocol also appears to be a safe method for classifying LBP.

There are a number of implications for the work presented in this report. Clearly, there is a need to assess lumbar impairment and classify LBP. With the reaction classification model presented, better decisions regarding disability and work readiness can be made

based on objective assessment of impairment relevant to lumbar mechanics and injury mechanisms. In addition, with a tool to assess impairment, it will also be possible now to evaluate various LBP treatment options to help establish evidence-based approaches. Accomplishing this may help to reduce significant and possibly unnecessary costs to employers as a result of lengthy disability and reduce unnecessary suffering for workers who experience LBP.

## RESEARCH PROBLEM

### Rationale

Low Back Pain (LBP) is a common condition affecting a large percentage of the population. It is estimated that between 70 to 85 % of the population will experience LBP at some point in their lives<sup>5;8;29;65</sup>. The majority of these cases resolve without medical intervention within the first six weeks<sup>65</sup>; however, the minority of cases that progress to become chronic bear a significant cost burden<sup>1;23;61</sup>. Not surprisingly, LBP is one of the most prevalent and costly health problems in Western Society<sup>4</sup>, since it comprises 25 % of all workplace injuries<sup>68</sup> and accounts for approximately 40 % of compensation costs<sup>61</sup>.

Injury trends have shown that disability from LBP dramatically increased between 1950 and 1980<sup>19;65</sup> with disability rates increasing by 14 times the rate of population growth over this time period<sup>19</sup>. Improper medical management is believed to be one of the primary causes of this rise in number and associated cost<sup>65</sup>. Not having a valid and reliable method for assessing and classifying LBP may have contributed to this problem through misdiagnosis and inappropriate prescription of treatment.

## **Lumbar Mechanics**

LBP is a multi-factorial problem with exposure to physical loading being one of the most significant risk factors<sup>18;30;37</sup>. Occupations with manual material handling, and in particular lifting, have been strongly correlated with LBP accounting for 50-75% of all back disorders<sup>9</sup>. However, the spinal load is not always as indicative of level of risk as one would expect. Epidemiological evidence suggests that high levels of spinal loading, exceeding the NIOSH maximum permissible limit of 6400 N, are not always prevalent in occupations where the risk for LBP is high<sup>24</sup>. Furthermore, workers in hazardous occupations who survive without developing LBP may generate significantly more spinal loading than their injured counterparts<sup>20</sup>. This suggests that physical exposure to loading is associated with LBP but not necessarily the degree of loading. How people cope with the loading plays a key role in determining injury.

A novel injury model was proposed by Panjabi<sup>44;46</sup> based on the concept of system stability of the spine. The model identifies three subsystems: the passive subsystem consisting of osteoligamentous structures (bones, ligaments, and intervertebral discs); the active subsystem consisting of muscles; and the motor control subsystem which is primarily responsible for controlling muscle activation in response to changing demands for stability. A dysfunction in any of these subsystems may result in or lead to mechanical instability of the spine and injury. Given the structural characteristics of the spine, it is not surprising that it can be injured at relatively low loads. Suppose each vertebral body represents a small block. It is easy to imagine, as the stack of blocks gets higher, it also becomes progressively unstable. Considering that the spine is comprised

of 24 separate units stacked on top of one another, it is not surprising that it is mechanically unstable and incapable of bearing much load. This has been verified through in vitro experiments. For the lumbar portion of the spine, Crisco et al.<sup>15</sup> demonstrated that the structural tolerance of the osteoligamentous spine is approximately 90 N or 20 lbs. This is interesting since the upper body mass which it supports is significantly higher (8 times as much!)<sup>39</sup>. Clearly, trunk muscles provide the necessary support, much like guide wires, in order to provide stability. Consequently, the control of these trunk muscles must be optimal to maintain structural integrity of the spine and avoid injury.

Stability can be quantified by measuring the potential energy of the system. Mechanical systems typically adopt a minimum energy configuration. The energy state can be represented as a ball in a bowl with the minimum energy configuration for the system occurring when the ball finds its resting position at the bottom<sup>14</sup>. If the ball is perturbed so that it moves up the side of the bowl, it will increase its potential energy. Predictably, the ball should reduce its potential energy by returning to its stable position at the bottom. As long as the perturbation does not cause the ball to leave the bowl, the system is stable. Obviously, as the bowl becomes deeper and the sides steeper the system becomes more stable and resistant to perturbation (meaning the ball will be displaced less with the same amount of force). Muscles acting as variable stiffness springs effectively increase the steepness of the sides in our bowl example. This muscle stiffness is proportional to activation level<sup>14</sup> or expressed differently, force output.

Antagonistic muscle groups function as parallel springs to increase joint stiffness and stability<sup>40;41</sup>. For the trunk, antagonist coactivation of muscles has been shown to increase spine stability {Cholewicki, Panjabi, et al. 1997 #1980}. Predictably, exposure to more unstable environments increases trunk muscle coactivation<sup>12;21</sup>. Granata<sup>21</sup> observed this phenomenon when subjects lifted objects to different heights while maintaining constant load moments. These heights represented varying levels of instability with the higher held loads producing more unstable conditions (similar to the block analogy). The results of this study showed that antagonist muscle activity increased proportionally with the increase in potential energy of the load. Cholewicki et al.<sup>12</sup> also observed more antagonistic coactivation when extra mass was added to the trunk increasing the potential energy in the system. It appears the neuromuscular system will impose additional loading on the spine to maintain stability even at the cost of this compressive penalty<sup>26</sup>.

Bergmark<sup>6;7</sup>, it is believed, was the first to study spinal mechanical stability. He defined muscles crossing a single intervertebral joint as the local muscular system and muscles spanning all joints from the ribcage to pelvis as the global muscular system. Analysis of stability demonstrated that for a given activation level in the local muscular system, there exists a maximum limit of activation for the global muscular system, above which the spine will buckle.

Later, Crisco and Panjabi<sup>14;16</sup> demonstrated that the overall stability of the five-vertebrae spinal column depends on muscle architecture. Empirically, they determined that the

spine was unstable if any vertebral level was devoid of muscle. Therefore, stability of the *in vivo* human spine must depend on the relative activation of all muscles crossing the lumbar spine and requires an orchestrated effort to function properly. With such a complex mechanical system, it is plausible that the potential for error exists in neuromuscular control, and if it were to occur, would likely result in an injury. During an experiment investigating spine loading during power lifting, an injury was observed in real-time that appears to be attributed to such an error<sup>11</sup>. Using video fluoroscopy, investigators witnessed the rotation of a single vertebral unit past its physiological limits. Investigators believe that the lifter was distracted during the lift and may have deactivated a local stabilizer causing a brief period of instability at that spinal level.

There is some evidence suggesting fatigue may compromise spinal stability.

Epidemiological prospective studies have shown that poor trunk muscular endurance is associated with an increased risk of developing first-time LBP<sup>2;3</sup>. Fatigue appears to affect the ability to modulate force<sup>60</sup>, as well as the timing of activation<sup>67</sup>, and reduces proprioceptive awareness<sup>62</sup>. As a result of this, the control of movement is impaired<sup>17;47</sup>. This impaired neuromuscular control may be compensated by antagonist activity<sup>50</sup>; however, increasing levels of coactivation is physiologically costly. With fatigue, the ability for muscles to generate force, and consequently, stiffness is diminished<sup>34</sup>. This, in conjunction with fatigue induced-control deficits, could possibly progress to the point where the paraspinal muscles can no longer stabilize the spine.

During *in vitro* experiments where spinal buckling was observed, vertebral displacement was large enough to strain the spinal column to the point of catastrophic failure<sup>15;32</sup>. *In vivo*, the neuromuscular system would most likely react prior to any major vertebral displacement to avoid severe damage; however, compromised neuromuscular control would allow for some excess motion<sup>47</sup> and strain resulting in micro trauma to connective tissue. This accumulated wear may result in joint laxity thus reducing the passive stiffness of osteoligamentous structures. Damage to the osteoligamentous structures has been shown to reduce passive stiffness of the intervertebral joint<sup>42;43</sup>. It has been proposed that muscles could compensate for this loss of passive stiffness by increasing their activation levels<sup>12;45</sup>. This has been shown in LBP patients who elicit higher levels of antagonistic activity than healthy controls<sup>36;63</sup>. Compensatory recruitment has been estimated to increase muscle activation from 3.4 to 5.5 % of maximal effort<sup>13</sup>. This slight increase in activation levels may be detrimental over prolonged periods. Activation levels of 5% for an eight hour period have been shown to be correlated with the manifestation of fatigue related pain<sup>27</sup>. Coupled with this, muscle biopsies revealed that people with LBP have less fatigue resistant muscle fibres than healthy controls<sup>35</sup> making it even more difficult for them to cope with the added demands associated with joint instability.

To summarize, the mechanical characteristics of the spine suggests that muscle recruitment is important for providing stability to prevent injury. Unfortunately, control of muscles appears to be compromised in people with LBP. This may be attributed to a number of causes. In particular, less fatigue resistant muscle fibres and a loss of passive

stiffness in the osteoligamentous spine may significantly tax the endurance capabilities of the paraspinal muscles. With fatigue, the ability to generate and modulate force is diminished which in turn may increase joint wear and laxity. Chronic LBP conditions may simply result from the progression of a vicious cycle in which added demands are placed on paraspinal muscles while concurrently their abilities to protect the spine are compromised.

### **LBP Classification**

In the past and currently, patients with LBP have been classified on the basis of a pathoanatomic approach to identify damaged spinal tissue. The literature reports that fewer than 15% of LBP cases are confirmed by medical imaging techniques<sup>49</sup>.

Furthermore, there is a high incidence of false-positives from spinal imaging, which can lead to misdiagnosis, and consequently, inappropriate prescription of treatment.

In an attempt to overcome limitations with the pathoanatomic approach, clinical assessment procedures were developed that focused on mechanical outputs like force production (strength) and kinematic information (range of motion); however, these tests do not appear to be able to reliably discriminate people with LBP from those without LBP. When tested for accuracy, these conventional clinical parameters correctly identified only 57 % of subjects with LBP and 67 % without<sup>58</sup>. There are a number of reasons why these tests may not be highly predictive. For force parameters, maximal performance on these tests could often lead to variable results from inconsistent levels of

effort. For the range of motion parameter, there is some evidence to suggest that ROM is not highly correlated with spinal instability<sup>43</sup>. Because of the poor predictive power of these tests, researchers have begun to look for alternative methods to discriminate LBP.

Since spinal instability results in increased challenges for paraspinal muscles, and people with LBP have less fatigue resistant muscles, a logical approach to assessing LBP would be to investigate the fatigue characteristics of the paraspinal muscles. Starting in the late 80's, a number of investigators have used spectral electromyography (EMG) to assess fatigue in the paraspinal muscles, and classify LBP<sup>22;31;48;56;57;59</sup>. Typically, mean or median frequency is used as a fatigue marker during sustained isometric contraction at a specified effort level. It has been claimed that these tests are objective due to the fact that it is not possible to volitionally alter the action potential signal registered with EMG<sup>48</sup>. However, with these protocols, subjects are required to perform maximum voluntary efforts to set the resistance level during the test. Unfortunately, people with LBP are unable or reluctant to perform maximum exertion due to fear of injury<sup>28;38;64</sup>. Because of inaccuracy in predicting MVC, the power of these tests may be diminished and may lead to erroneous results. It could be argued that during MVC tests, motivational factors and/or fear of re-injury could have a significant effect, which in turn could alter muscle activation patterns during the fatigue trials. However, even with these shortcomings, these tests appear to be able to classify individuals with LBP.

It has been well documented in the literature that recruitment patterns of trunk muscles are altered following an injury. Specifically, it has been reported in a number of studies

that the reaction time of trunk muscles is delayed in LBP patients<sup>25;33;51;52;66</sup>. Radebold et al.<sup>51</sup> used a quick release protocol to measure reaction times for trunk muscles and found muscles typically responded within 100 ms following the load-release. This quick response is indicative of a reflex and not voluntary movement which suggests that this measure may serve as the basis for an object classification tool. Although differences have been observed between LBP patients and healthy controls, the use of EMG reaction parameters has not been used to classify LBP.

A preliminary study was conducted to test which parameters (fatigue versus reaction) were best for classifying LBP. Results from this study indicated that reaction parameters out-performed fatigue parameters when discriminating people with LBP from those without. Fatigue parameters were shown not to be statistically different between patients and healthy controls; whereas, a number of reaction parameters reached significant levels ( $p < .05$ ). In addition, reaction based classification also showed that it was categorically distinct with scores falling at either end of the range while fatigue based classification was more ambiguous. Unfortunately, these findings were based on a small sample size of 15 subjects due to some difficulty in recruitment of subjects. Some subjects withdrew from the study and stated they had concerns regarding the fatigue protocol which required exertions of 60% of maximum. Consequently, a second study was conducted using a larger sample with matched controls to develop and validate a reaction classification model. The details of this study will be presented in the following sections. It should be noted that reaction time data was collected at two locations: Simon

Fraser University and Yale University using similar experimental protocol. Differences in experimental protocol are identified in the methodology section.

## **METHODOLOGY**

### **Ethics Approval**

Before testing, all subjects read and signed a consent form which outlined the testing protocol. The protocol was approved by the Ethics Review Committee at Simon Fraser University (Appendix A) and the Human Investigation Committee at Yale University (Appendix B).

### **Subjects**

Sixty people in total participated in the study of which 31 were LBP patients and 29 were healthy controls. Healthy control subjects were defined as persons who had never experienced back pain lasting longer than 3 consecutive days. LBP patients were defined as persons past the acute phase of their condition with no neurological deficits and no structural deformities, genetic spinal disorders or previous spinal surgery. The subject pool was divided into two groups with 40 being assigned to a learning group to develop the classification model, and 20 to a holdout group to test the accuracy of the classification model. For the learning group, healthy controls were matched as closely as possible for gender, age, weight, and height.

Table 1. Number, mean age, weight, and height (SD) of LBP patients and matched controls for the learning group.

	Females		Males	
	Controls	Patients	Controls	Patients
Number	6	6	13	15
Age (years)	41.33 (12.90)	40.00 (10.97)	40.23 (11.76)	37.073 (11.60)
Weight (kg)	60.43 (12.25)	67.42 (11.17)	81.40 (17.60)	83.48 (13.33)
Height (m)	1.69 (0.08)	1.62 (0.08)	1.76 (0.09)	1.80 (0.05)

Analysis of variance revealed that there were no significance differences between controls' and patients' age, weight, and height ( $p=0.640, 0.372, 0.428$  respectively).

Table 2. Number, mean age, weight, and height (SD) of LBP patients and healthy controls for the holdout group.

	Females		Males	
	Controls	Patients	Controls	Patients
Number	4	2	7	7
Age (years)	29.00 (10.61)	35.00 (15.56)	41.00 (10.63)	34.86 (11.16)
Weight (kg)	61.38 (4.11)	63.50 (10.61)	81.19 (11.40)	79.57 (13.10)
Height (m)	163.88 (7.88)	160.00 (0.00)	177.86 (6.01)	177.29 (8.84)

### **Data Collection**

Subjects were placed in a semi-seated position in the testing apparatus for exerting isometric contraction in trunk flexion, extension and lateral bending to the left and right (Figure 1). This apparatus was designed to restrain hip motion while allowing the upper body to move freely in any direction. Consequently, response patterns of trunk muscles were responsible for all postural readjustments. Isometric force was applied through a cable attached to a chest harness at approximately T9 and was held with an electromagnet. The quick release of this electromagnet produced the sudden unloading on the trunk resulting in displacement of the trunk, and initiating reactive responses in the trunk muscles.

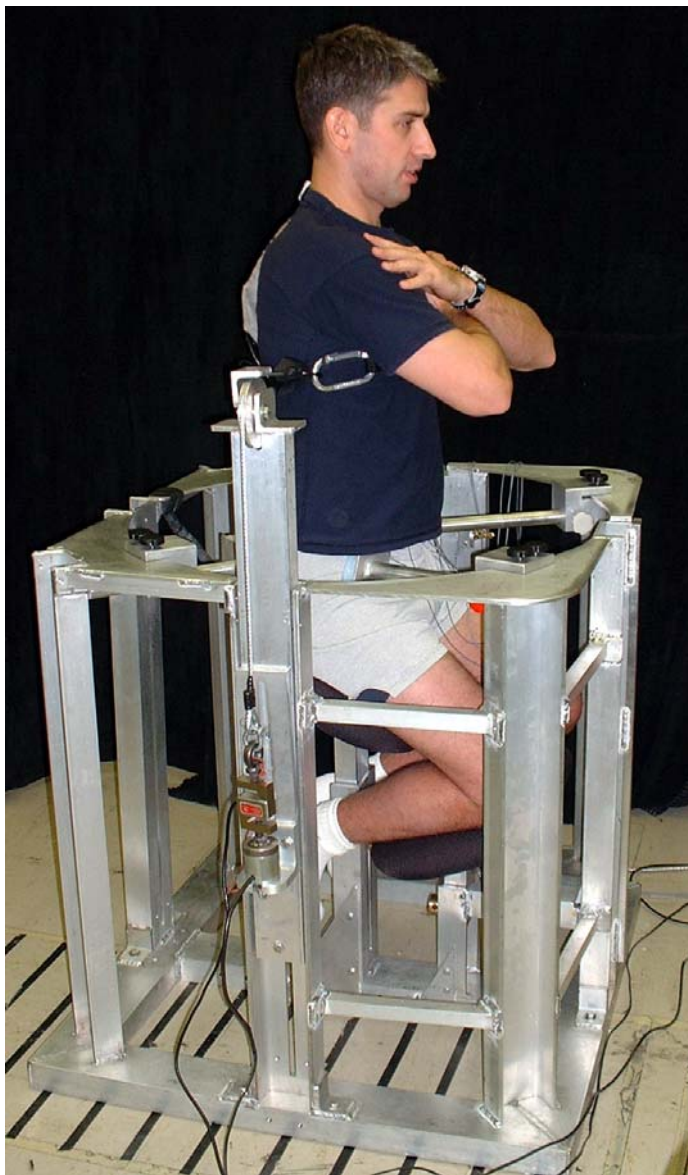


Figure 1: Subject position in testing apparatus. Tension through a cable connected to a chest harness produces an isometric moment.

Each subject performed three trials at a predetermined force level of 65 N for males and 40 N for females. These force magnitudes were established in a preliminary study, and are approximately 20 % of the maximal isometric exertion averaged for flexion, extension, and lateral bending. An oscilloscope was used to provide visual feedback of force to the subject. The force was displayed as a line which moved up and down while the target was displayed as a stationary line. Subjects were asked to keep the force output line steady at the target and were asked not to try to anticipate the release. The time of release was randomly varied after the target force level was reached.

Eight channels of EMG were recorded using bipolar, Ag-AgCl, disposable electrodes with a centre-to-centre spacing of 30 mm (at Yale), and bipolar, stainless steel electrodes with an active circuit and centre-to-centre spacing of 13 mm (at SFU). The electrodes were positioned over the following muscles on each side of the body: rectus abdominis (3 cm lateral to the umbilicus), external oblique (approximately 15 cm lateral to the umbilicus), thoracic erector spinae (5 cm lateral to T9 spinous process), and lumbar erector spinae (3 cm lateral to L5 spinous process). In previous studies, this electrode placement had been shown to maximize signal-to-noise ratio while minimizing channel cross-talk<sup>10:12</sup>. All EMG signals were band-pass filtered between 20 and 250 Hz (at Yale) and 20-500 Hz (at SFU), differentially amplified between 25X to 2000X, then A/D converted at a sample rate of 1600 Hz (at Yale) and 2000 Hz (at SFU). Initially, 12 channels of EMG were collected. The four additional channels recorded the activity of latissimus dorsi, and internal oblique muscles bilaterally for the Yale subject pool. These muscles were later removed from the Yale data and were not measured for the SFU

subject pool. Due to electrode position for these muscles, there were some concerns with artefact entering the signal from contact with the pads and harness. Analysis of the Yale data indicated that the latissimus dorsi muscle group played a minor role in postural adjustments following load-release as indicated by lower EMG activity. Similarly, discriminant analysis on the Yale data indicated that the internal oblique muscle group activity was not significantly different between healthy controls and patients ( $p=.158$ ).

To facilitate the detection of switching-on and shutting-off of muscle activities from EMG signals, a computer algorithm was developed (Figure 2). When rectified mean EMG increased by 1.5 standard deviations, muscle onset was detected. When rectified mean EMG decreased by 1.5 standard deviations, muscle offset was detected. When the muscle activity did not change after the load-release, the computer did not mark any shut-off or switch-on time. Time between this detection and a force drop represented the reaction time. A decrease of 50 % (at Yale) and 5 % (at SFU) in mean force prior to the release was used as the threshold. At SFU, the increased strength of the electromagnet resulted in a slower rate of force decline; consequently, the force threshold appeared to be consistent between data sets. Statistical analysis revealed that there were no significant differences between mean reaction times at Yale and SFU ( $p=.793$ ).

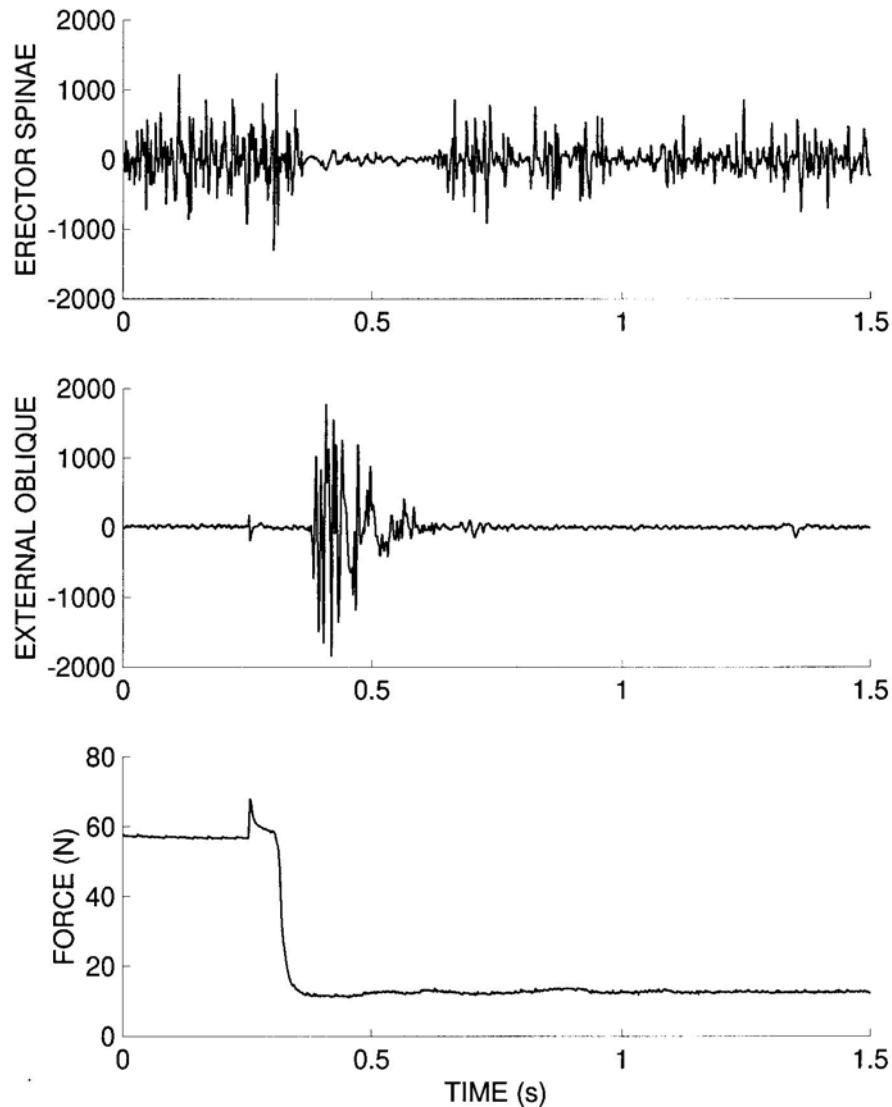


Figure 2. Examples of muscles switching-on and shutting-off following isometric back extension. The magnet release was followed by a sudden drop in tension in the cable seen in the force record (bottom trace). The reaction times were calculated from a 5% drop in force to an increase or decrease of 1.5 standard deviations in the EMG signal. Top, Detection of lumbar erector spinae muscle switching-on. Center, Detection of external oblique muscle shutting-off.

To clarify, agonist muscles were defined as muscles that were active before the load-release and were expected to shut-off. Antagonist muscles were quiet before the load-release and were expected to switch-on. In flexion, flexors acted as agonists and extensors as antagonists. In extension, extensors acted as agonists and flexors as antagonists. In lateral bending to the left, ipsilateral muscles (left side) acted as agonists and contralateral muscles (right side) acted as antagonists, and vice versa for lateral bending to the right.

### **Data Analysis**

Individual muscle reaction times were recorded and grouped into onsets and offsets. The average of each muscle for the three trials was determined for each of the four directions. The average reaction time for all onsets and offsets were determined.

For the learning group, a Kolmogorov-Smirnov test was used to determine if the EMG reaction parameters were normally distributed. This was done by comparing the reaction parameters distributions to a normal distribution to determine if they were significantly different ( $p < .05$ ). To determine if reaction times were statistically different ( $p < .05$ ) between patients and healthy controls, an independent t-test was used for parameters that were normally distributed, and a Mann-Whitney test was used for parameters that were not normally distributed. Parameters reaching significance were then entered into logistical regression analysis to determine which parameters should be included in the

classification model. From the regression analysis, a prediction equation was formulated and applied to the holdout group to validate the model.

## RESEARCH FINDINGS

### Results

In general, patients tended to have longer reaction times than healthy controls and showed more variability as indicated by mean reaction times and standard deviations between groups (Table 3).

Table 3. Learning group mean reaction time (standard deviation) and independent t-test scores.

	Off (ms) Agonist		On (ms) Antagonist	
	Patients	Healthy Controls	Patients	Healthy Controls
Extension (E)	70 (42)	44 (10)	90 (34)	73 (20)
	p=.004		p=.071	
Flexion (F)	49 (29)	67 (58)	83 (14)	65 (11)
	p=.268		p<.000	
Lateral Bending Left (L)	74 (57)	46 (20)	96 (21)	84 (34)
	p=.233		p=.01	
Lateral Bending Right (R)	43 (29)	45 (23)	97 (31)	80 (15)
	p=.673		p=.051	

Results from the parametric and nonparametric independent t-tests identified 3 parameters that were significantly different between the patients and controls (Table 3). Correlational analysis of the 3 parameters indicated that none were significantly related. It was decided that inclusion of all 3 parameters would account for greater variance in the classification model. Although RON was close-to-significant, to minimize over fitting

the data, this parameter was not entered into regression analysis. Binary logistical regression analysis yielded the following prediction equation incorporating 3 parameters:

$$y = e^{(28.274 - 231.927 * FON - 99.034 * EOFF - 66.835 * RON)} \quad \text{Eq. 5}$$

$$P = \frac{y}{(y + 1)} \quad \text{Eq. 6}$$

Re-entering the learning group into the classification model resulted in 90% of patients and 84.2% of healthy controls being correctly classified with the overall model accuracy of 87.2% (1 missing data point). For the holdout group, the model accurately classified 100 % of patients and 89 % of healthy controls with the overall model accuracy being 94.4 % (2 missing data points). As depicted by the classification plots, the majority of the cases scored at either end of the range as opposed to the midrange making the model more categorically distinct (Figure 3 and 4).

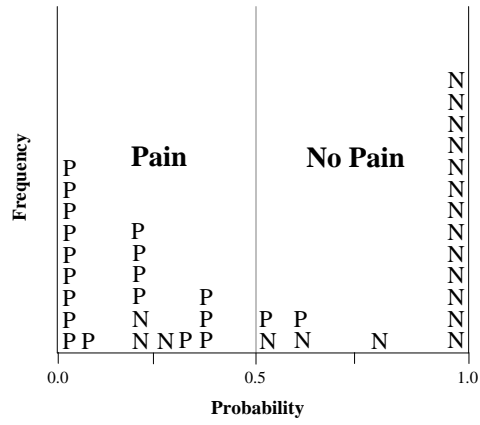


Figure 3. Classification plot for learning group.

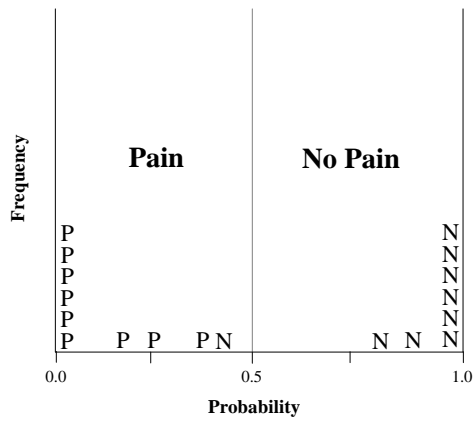


Figure 4. Classification plot for holdout group.

## Discussion

The reaction times found in the present study matched closely with those found in earlier work<sup>33;51;52;66</sup>, and are indicative of a reflex response rather than voluntary movement.

Clear group differences were observed in reflex responses to load-release: patients showed longer latencies and more variability than healthy controls. This was particularly true for parameters dominated by responses of the erector spine muscle group (i.e., FON and EOFF).

The primary goal of the study was to determine if reaction parameters could accurately classify people with and without LBP. From the results of this study, measuring reaction time following load-release appears to be an accurate method for classifying LBP.

Correct classification of males was higher than females (93% versus 75%) which suggests there are gender differences that may need to be accounted for in future iteration of this type of model. Unfortunately, the small sample of female participants in this study was not adequate to evaluate if gender differences exist, or whether a gender specific model would be more accurate.

From a safety perspective, only low-level exertion (20% MVC) is required for this type of model which is substantially lower than that of fatigue based protocols (60% MVC).

No subjects experienced any discomfort during testing or had their injury aggravated suggesting that the test is safe. In addition, it also appears to be less susceptible to the

inaccuracies of predicting effort level than fatigue-based protocols. However, a model that normalizes effort to acceleration following release instead of setting effort to a predetermined force level may produce better classification. During testing, subjects with less upper body mass tended to be displaced more than heavier subjects as result of inertial differences. If the sensory receptors mediating the reflex responses are affected by length and velocity changes in muscle this difference in inertial mass may result in a decrease in model accuracy.

To test if the protocol was sensitive to changes in trunk stiffness that could be modulated volitionally, reaction times were compared between coactivation and no coactivation conditions in preliminary tests on control subjects. The hypothesis was that increased coactivation would increase the stiffness of the spine, reducing displacement of trunk. With less displacement, receptor-evoked contributions in EMG would be diminished or delayed resulting in longer reaction times. Interestingly, this appeared to be the case for agonists ( $p=.003$ ) but not antagonist muscle groups ( $p=.570$ ). It should also be noted that agonist reaction times in extension appeared not to be affected by coactivation (mean agonist reaction times in extension were 51 ms for no coactivation and 48 ms for coactivation). This is important for the classification model since agonist reaction time in extension is one of the parameters used for classification. Although trunk position was not measured during testing, it would be expected that increased trunk stiffness would reduce trunk displacement which appeared to be the case from visual observation. From this, it appears that the model is not significantly affected by changes in trunk stiffness.

Finally, it should be mentioned that the algorithm for detecting muscle onsets and offsets sometimes required manual correction. A number of different protocols were tested to improve accuracy as well as reduce the time spent processing data. However, because of signal artefacts, it is difficult to completely automate this process. Consequently, there is some subjectivity in determining whether a change in the signal was a result of a myoelectric event or from an external source. This is particularly true for the abdominal muscles which tend to have lower signal-to-noise ratio due to increased subcutaneous fat. However, signals generated from the erector spinae muscle group appeared to be free of artefacts making the process of determining onsets and offsets more objective for parameters derived from these muscle groups.

## **IMPLICATIONS FOR FUTURE RESEARCH ON OCCUPATIONAL HEALTH**

The next iteration of the reaction classification model should investigate differences in gender to determine if a gender-specific model needs to be developed.

In order to compensate for differences in inertial mass, it may be better to choose force levels based on trunk displacement instead of a fixed percentage of maximum voluntary force.

With the use of the reaction classification model, the effectiveness of stabilization exercise in rehabilitating LBP patients could be evaluated.

Also, it might be possible to use the classification model to conduct a prospective study to determine if people are predisposed to injury or if injury causes motor control deficits that exacerbate LBP.

## **POLICY AND PREVENTION**

In terms of prevention, this research provides further evidence to suggest that people with LBP have altered recruitment of trunk muscles. Clearly with compromised neuromuscular control, the potential for further injury may be increased. This is particularly true under conditions of mechanical instability (i.e., where load shifts occur and the trunk muscles must respond rapidly to stabilize the spine). With the ability to assess neuromuscular control using the reaction protocol, it might be possible to assess individuals prior to starting work or following an injury to determine if they are at risk for injury when working in such hazardous conditions. It might also be possible to monitor individuals in high-risk occupations (i.e., nursing, homecare workers, etc...) and where necessary introduce early intervention to prevent LBP when alterations in neuromuscular control are identified.

Decision-makers at WCB who manage injury claims may find the classification tool useful in providing additional information regarding a claimant's impairment. This objective assessment of impairment could help determine, in conjunction with input from other sources, work readiness. Following return-to-work, it might be valuable to retest the claimant to determine if work reintegration is having a positive or negative effect on their condition. In some conditions, early reactivation of the injured worker could have a positive outcome on reducing disability. It has been shown that pain avoidance behaviour can account for a large part of the disability associated with chronic LBP and can be successfully treated with reactivation<sup>53-55</sup>.

## **DISSEMINATION/KNOWLEDGE TRANSFER**

The first draft of a paper entitled, *Muscle Reaction Classification of Low Back Pain*, has been completed and will soon be submitted for publication in the journal, *Spine*. In addition, results from the research will be presented in the upcoming year at international biomechanics conferences. Acknowledgement of WCB financial contributions has been noted in the paper and will also be highlighted in the presentation.

In addition, this information will also be made available to occupational health professionals, physicians, and other interested healthcare providers through public presentation at the request of WCB.

In terms of applying the technology for practical purposes, the most obvious use would be assessing impairment and aiding in work readiness decisions. We would be happy to discuss with the Board an implementation strategy for this application.

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## APPENDIX A - SFU CONSENT

### SIMON FRASER UNIVERSITY

OFFICE OF VICE-PRESIDENT, RESEARCH



BURNABY, BRITISH COLUMBIA  
CANADA V5A 1S6  
Telephone: (604) 291-4370  
FAX: (604) 291-4860

April 6, 2001

Dr. Theodore Milner  
School of Kinesiology  
Simon Fraser University

Dear Dr Milner:

**Re: EMG-based Assessment and Classification of Low Back Pain**  
W.C.B.

I am pleased to inform you that the above referenced Request for Ethical Approval of Research has been approved on behalf of the University Research Ethics Review Committee. This approval is in effect for a period of three years from the start of the research project or for the term of your faculty appointment at SFU. Any changes in the procedures affecting interaction with human subjects should be reported to the University Research Ethics Review Committee. Significant changes will require the submission of a revised Request for Ethical Approval of Research.

Best wishes for success in this research.

Sincerely,

A handwritten signature in black ink, appearing to read 'J. Ogloff', written over a white background.

Dr. James R.P. Ogloff, Chair  
University Research Ethics Review Committee

c: R. M Marteniuk, Dean  
/bjr

## APPENDIX B – YALE CONSENT



### Yale University

School of Medicine  
Human Investigation Committee  
47 College Street, Suite 204  
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New Haven, Connecticut 06520-8010

Telephone: 203/785-4688  
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**DATE:** February 14, 2001  
**TO:** Jacek Cholewicki, Ph.D.  
**FROM:** Sarah B. Putney, J.D.  
HIC Coordinator *Sarah B. Putney, J.D.*  
**RE:** HIC #8590  
**TITLE:** *Motor Control of the Lumbar Spine in Healthy and Low Back Pain Populations*

Approval of this protocol was renewed by the HIC on February 14, 2001.

**Reapproval:** It is the investigator's responsibility to apply for reapproval of ongoing research at least one year from the date this protocol was reviewed by the full HIC. Therefore this protocol must be reapproved before February 9, 2002. Please allow two months for reapproval.

As a result of recent federal action to tighten controls over research using human subjects, the HIC has had to re-evaluate and revise some of its policies and practices. In an attempt to ensure that all HIC protocols meet current applicable standards, we have instituted a new policy that requires all active protocols to be re-written after five years. **This letter shall serve as notice that you will need to re-write this protocol and submit it for full committee review before the next anniversary date of February 9, 2002.** Please refer to current HIC Guidelines as you prepare your protocol and be sure to secure a primary reviewer. You can find a list of all HIC members at the HIC website (<http://info.med.yale.edu/hic/welcome.html>) or you may call the HIC office (785-4688) for information.

In compliance with federal regulations and current guidelines, the Human Investigations Committee requires that the Principal Investigator provide a copy of each grant application, for federal or other funding that is associated with the above-listed protocol. Please forward a copy of the grant or other funding document as soon as possible.

Please feel free to call me if you have any questions about your protocol or this letter. I would be happy to help you in any way that I can.

SBP/nt  
Enclosure: Signed HIC Form #5

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