The Magnitude of Pre-Programmed Reaction Dysfunction in Back Pain Patients - Experimental Pilot Electromyography Study


Abstract

Preprogrammed Reactions (PPR) appear at a latency of higher than 40 ms, but before the voluntary muscle responds (~120 ms) to postural perturbations. We examined the difference in magnitude of preprogrammed reactions in patients with chronic low back pain (CLBP) and without low back pain. We analyzed electromyographic Root Mean Square (RMS) amplitudes of asymptomatic \(n = 25\) and CLBP patients \(n = 25\) on stable & unstable surfaces during expected and unexpected perturbations for rectus abdominus and erector spinae muscles. The mean PPR and PPR-combined voluntary response RMS amplitudes (VRPPR) were compared between the two groups. To find the presence of PPR in LBP patients, a criteria was set that the obtained PPR RMS amplitude value should exceed 60% mean reflex RMS amplitude that occur within 50 ms after perturbation. Fleiss’ kappa revealed a good agreement \(\kappa = 0.7\) to 0.9) among raters for absence of PPR in patients with CLBP and presence of PPR in asymptomatic population. The two way ANOVA revealed significantly different mean PPR and VRPPR RMS amplitudes between asymptomatic and LBP population for rectus abdominus and erector spinae muscles \(p<0.05\). PPR responses were found absent (< 60% of Mean Reflex RMS) in patients with CLBP. Further, patients with CLBP demonstrated lower PPR amplitudes with higher peak voluntary responses compared to asymptomatic population, indicating difficulties in presetting of voluntary responses for regaining postural stability after perturbation.

Keywords: Back Pain, Electromyography, Spine, Muscle, Reflex.

2.1 Introduction

The postural and neuromuscular control responses during perturbation differ between patients with low back ache and healthy control subjects. (Hodges and Bui 1996; Luoto et al. 1996; Newcomer et al. 2002; Paquet et al. 1994; Radebold et al. 2001; Richardson et al. 1999; Volpe et al. 2006; Zeevi et al. 1991).
Postural adjustments for voluntary movements in humans are premeditated in the central nervous system and constantly modified based on multi-sensory feedback. Before the muscle activation or voluntary response occurs, the preparedness of the muscle recorded in the EMG can be categorized in terms of short latency (<40ms, M1, Stretch reflex) and longer latency (>40ms to <120ms, M2-M3) response (MacKinnon et al. 2000; Hammond 1955). The long latency responses are otherwise called pre-programmed reactions (M2-M3 response, functional stretch reflex) and most of their processes are controlled by higher supra-spinal centers (Matthews 1991).

Since these reactions occur after a form of challenge or perturbation to the posture, they are relatively distinct from anticipatory postural adjustments (Latash 2008). But, anticipatory postural adjustments tend to defend postural equilibrium before the imposed perturbations. Hence preprogrammed reactions are classified as compensatory type of reactions that balance adjustments induced by the intended movement (Latash 2008; Matthews 1991). Thus, pre-programmed reactions are critically important in fine-tuning of any voluntary movement and play a role in coordinated activation of muscles across segments, which is necessary for an adequate response in the context of changing postural equilibrium (Gielen et al. 1988).

Notably, PPRs are continuously cortically modulated for task demands by increasing the activity in a graded fashion and exhibit smooth tuning, which is similar to that observed in voluntary response (Pruszynski et al. 2008; Shemmell et al. 2009; Lewis 2004).

The magnitude of the PPR response varies with task requirements, instructions, previous experience of the subjects, and cannot be correlated with strength of perturbation and muscle length (Beckley et al. 1991; Colebatch et al. 1979; Lewis et al. 2004; Kurtzer et al. 2008; Pedersen et al. 2004). Any attempt to compensate the perturbation with prior instructions (Beckley et al. 1991), higher trials of reproduction of perturbation amplitude and unstable mechanical loads (De Serres and Milner 1991) may lead to progression in PPR amplitude (De Serres and Milner 1991; Pedersen et al. 2004; Skotte et al. 2004).

Previous studies reported (Granata et al. 2004; Moorhouse and Granata 2007; Newcomer et al. 2002; Radebold et al. 2001) temporal discrepancies in spinal stretch reflexes and changes in feed forward postural adjustments in back pain population compared to healthy controls. However, the magnitude and extent of these preprogrammed
reactions and their relation to voluntary responses in global trunk muscles of CLBP patients were largely overlooked.

On the other hand, studies reported shortening of the stopping time (measured between sudden loading and stopping) with training focused on reactions to a variety of expected and unexpected sudden trunk loadings, including balance and coordination exercises (Skotte et al. 2004; Pedersen et al. 2007; Pedersen et al. 2004). Pedersen et al. (2004; 2007) reported that this improvement was due to a more efficient pre-programmed response via increased EMG amplitudes during the period of 50-250ms and not due to improved reaction time (EMG onset latency).

Hence, the purpose of this study was to quantify and study the gain in amplitude of PPR and PPR combined with voluntary responses (VRPPR) as two separate entities in order to portion the role of PPR commonly associated with voluntary responses. Hence, EMG responses were partitioned into two distinct temporal intervals: 40ms to ~120ms for PPR and 40ms to 250ms for VRPPR (Latash 2008). Quantifying proportions of VRPPR in CLBP and normal healthy population could prove useful in clinical assessment, addressing a fundamental component of postural control and may provide an insight into the extent to which these compensatory types of reactions behave during sudden trunk perturbation that may act as a potential risk for low back injuries.

Further in this study, stable and unstable platforms were used to find out the nature of PPR amplitudes on various surfaces. Anticipatory and non-anticipatory perturbations were added to find out the extent of gain or fine-tuning in PPR for voluntary muscle response to adjust postural threat. It was hypothesized that expected perturbation and unstable surface generate higher PPR gain in asymptomatic subjects than unexpected perturbation and stable surface. This may be vice versa in CLBP patients.

2.2 Methods

2.2.1 Patients

Patients with CLBP (n=25, 18-Male, 7-Female) were selected prospectively, conveniently from the physiotherapy Out Patients’ Department clinics of hospitals around Mangalore affiliated to Srinivas College of Physiotherapy and Research Center. Patients with chronic CLBP or non-specific mechanical recurrent low back pain having a pain history of three months and without radiating pain below buttocks were included in the study. A medical screening was performed by a physician, fourth author (S.N) to exclude individuals with central nervous system impairment, fractures, spondylolisthesis, prolapsed
vertebral disc, history of any lumbar, abdominal or limb surgery, spinal tumors, infections, systemic illness and pregnant women. The inclusion criterion for the control group was that they should be free from LBP for atleast one year and should have never had an episode of LBP that lasted more than 3 months.

Asymptomatic individuals (n=25, 15-Male 10-Female) were selected from a pool of 2100 normal individuals, who were taken from institute student and staff population. All participants provided well-informed, written consent prior to participation and they were extensively briefed about the testing methods of perturbation and reporting procedures. The ethical committee of Department of Sports Medicine and Physiotherapy, Guru Nanak Dev University approved the study procedures. The baseline characteristics of the population participated in the study is reported in table 1.

2.2.2 Instruments

EMG equipment (Neurocare TM – advanced 2000 Surface EMG) manufactured by Bio-tech TM, India and Bertec force plate, Columbus, U.S.A was used to collect the data.

2.2.3 Surface Electromyography Recordings

The area for the placement of the electrodes was well rubbed and cleaned with sprit using swab and for Rectus Abdominis (RA), a pair of recording electrodes were placed on the right and left side of umbilicus, oriented parallel with the muscle fibers (3 cm lateral to the midline at the level of umbilicus). For lumbar Erector Spinae (ES), a pair of recording electrodes were kept 3cm lateral to the midline on the right side and left side between L3-L4. The inter electrode distance was kept at 2 cm. The reference electrode was kept approximately 5 cm laterally left side over the lumbar spine (Soderberg and Knutson 2000). Ag-AgCl surface, non-disposable electrodes (area: 4mm) were used in this study.

Each bipolar electrode pair was connected to a differential amplifier having an input impedance of $10^8$ ohms, a gain of 1650 and a common mode rejection ratio of 70 decibels and A/D converted at a sample rate of 1,000 Hz (Kamibayashi and Muro 2006). The EMG machine was set with the following parameters suitable for reflex amplitude, PPR and voluntary response measurements i.e., sensitivity: 100µv/div, filter setting: 20 Hz- 3KHz and sweep speed: 50 ms/div. The extreme low pass filtering with 2.5 or 3 Hz was avoided as it causes distortion of EMG content.

2.2.4 Protocol for Perturbation Tasks

Perturbations were given by release of three kilograms (kgs) cushioned steel cylinders onto the outstretched hand, anteriorly from a height of approximately 8 cms to
initiate sudden forward flexion movement on spine while the subjects were standing on the forceplate (Krajcarski et al. 1999; Mawston et al. 2007).

Perturbations were provided unexpectedly and expectedly while standing on a stable platform i.e., force plate surface and unstable surface i.e., foam surface on force plate (density 4×16). All the participants were instructed to straighten the arms to a maximum extent to catch the released weight. The time of release was manipulated to set the perturbation anticipatory or otherwise. During expected perturbations, prior instruction for time of weight release, i.e., the precise time, exact second of weight release was informed to participants and in case of unexpected perturbations the time of weight release was randomly chosen and given without prior knowledge to the participants.

A number of trials were made to standardize the weight to bring adequate perturbation on trunk by releasing weights on the outstretched hand. Weights more than 3kg tended to produce higher voluntary displacements, i.e., higher EMG voluntary responses in trials; hence it was decided to use 3kg as the reference weight. However, the weights were adjusted lower or higher to achieve clear reflex as well as PPR responses based on the participant’s body weight (Range 3.5% to 5%). Granata et al (2004) observed sensitive reflex responses for smaller movement disturbance than larger disturbances. Further, the pelvis segment was immobilized by solid support posteriorly and strapped anteriorly to restrain any pelvic movements and stepping forward from the forceplate (Alexiev 1994; Granata et al. 2004; Horak and Moore 1993), (Appendix E, Figure I to IV).

Participants were instructed not to resist before the impact or let go along with the perturbation after the impact. However, to gain maximum reflex and PPR amplitude, participants were instructed to return to their previous position as quickly as possible (Bonnet 1983).

Two operators were used to collect the data. The first operator was asked to monitor the 5 seconds of EMG data on real time with the beep sound by metronome for every second and to store the data. The second operator was asked to release the weights coordinately for the beeps, using randomly assigned second for weight release for each subject trial.

Using the known second of COP shift on forceplate, 250 ms prior to weight release (baseline data) and after the weight release were stored for data collection.

2.2.5 EMG and Forceplate Settings
The following methods were used to differentiate the PPR amplitudes and voluntary contraction. The guidelines were adapted from previous studies (Hodges and Bui 1996; Noth et al.1991).

1) Pre-weight release base line EMG reading was calculated. 2) Minimum two standard deviations from the “pre-weight release base line EMG” after perturbation was accepted for reflex amplitude i.e., M1, present within 40 ms after perturbation. 3) The perturbation was identified by defined millisecond of COP shift from y-axis of COP displacement by considering PA direction of perturbation on forceplate (Minimal 2SD from baseline on force plate data for minimal 50ms). 4) For PPR amplitude, the EMG signal present within 120ms corresponding to forceplate COP shift (center of pressure shift time on Forceplate) was considered for data collection. 5) Along with the above method, visual observation method was also used to identify the onset of well-defined PPR amplitudes i.e., m2-m3 polyphasic waves (Latash 2008) [23] or voluntary responses. 6) To identify the onset of PPR, the size of the M2 response should exceed the size of the M1 response by at least 60% (Noth et al.1991). 7) In cases of generalized delayed responses, particularly in CLBP population, the initial voluntary responses were identified after perturbation and a window length of 80ms was analyzed from 40ms to 120ms for PPR amplitude. PPR was also identified and measured between 40ms to the voluntary response peak of window length. Each trial data was coded by the identification process used i.e., either PPR or voluntary response as the first value. 8) Peak values of voluntary responses from m2 (rise time) were calculated as RMS PPR combined voluntary response amplitude value.

2.2.6 PPR and VRPPR RMS Data

 Cursors introduced at the beginning of m2 to beginning of voluntary contractions were traced as the mean RMS amplitude of preprogrammed reactions (40ms to 120ms). The window length of the same was noted. Further, cursors were introduced at the beginning of m2 to peak of voluntary response (peak rise time indicated by EMG during 40ms to 250ms) was traced as mean PPR combined voluntary response amplitude and considered as RMS amplitude of preprogrammed reactions along with Peak Voluntary Response (VRPPR). The window length was also noted.

Mean RMS amplitude of PPR and PPR to voluntary response peak amplitude was calculated for each muscle pertaining to anticipatory/non-anticipatory weight release and stable/unstable surface. A minimum baseline value of 2 μv was accepted and subtracted from the PPR amplitude measurement for three trials of each perturbation and averaged as
mean PPR RMS amplitude for all participants. Keeping in view the reflex nature of data collected, 30 minutes rest was given between each trial for recuperation. Up to six trials for each task were repeated to get well-defined data of M1, M2-M3 and voluntary responses for clear differentiation.

The mean EMG PPR amplitude was calculated for RA and ES pertaining to each trial. 4% of trial EMG readings of task tested, indistinguishable for above differentiation (m1, m2-m3 and voluntary response and even after six trial attempts with adequate day in-between) were considered as missing value during statistical analysis.

2.2.7 Inter-Rater Reliability Testing

After masking participants’ names, time of weight release and serial number details, the raters (3-Neurologist, 4- orthopedic surgeons & 2-physiatrists) were asked to analyze the EMG patterns and rate ‘yes’ or ‘no’ on the basis of presence or absence of preprogrammed reactions. The criteria set were as follows: the presence of M2-M3 polyphasic waves in the time window of 40ms to 120ms the size of M2 should exceed atleast 60% of M1, i.e., short-loop, mono-synaptic reflex (Hodges and Bui 1996; Noth et al. 1991).

Random values of CLBP and asymptomatic subject’s EMG patterns were given to the raters to avoid the potential bias of rating for the given group. If raters failed to identify the presence or absence of PPR, it was recorded as missing value for statistical analysis (5% of ratings in this study).

In our study, 91% (58% data obtained by identification of PPR first/ 33% data obtained by identification of voluntary responses first) of the trials observed with PPR amplitudes were used for statistical analysis Inter rater’s reliability and agreement among raters were arrived at by using a minimum of 100 EMG patterns for each group and 50 EMG patterns for each muscle.

The mean RMS EMG recordings of either left or right muscles were randomly chosen from CLBP as well as asymptomatic population to avoid potential bias of taking one side of EMG recording. 200 out of 364 total EMG patterns of right and left muscles were taken form both groups and pooled separately for data analysis.

2.2.8 Statistical Analysis

SPSS 12.0 Version with the significance level set at 0.05 was used for statistical analysis and post-hoc testing was performed using Duncan’s multiple range test. Two way mixed model ANOVA was used to analyze results by keeping two muscles as independent factors, whereas RMS PPR and VRPPR values were considered as dependent variables.
Fleiss’ kappa statistical measure was used to examine the reliability of agreement between raters to identify the absence and presence of PPR in CLBP and asymptomatic population. This was done to internally validate the findings of the study.

2.2.9 Normalization of EMG Data

Mean PPR and VRPPR RMS amplitudes of erector spinae and rectus abdominus muscles were normalized to stable-unexpected perturbation of asymptomatic participants. Reference and task specific normalization was prepared to evidently analyze and compare the nature of PPR behavior between asymptomatic population and CLBP patients for perturbation tasks.

2.3. Results

2.3.1 Inter-Rater Reliability

Fleiss’ Kappa analysis revealed a good agreement among the nine raters for the presence of pre-programmed reactions in asymptomatic individuals and absence of pre-programmed reactions in CLBP subjects. The agreement values ranged between 0.7 to 1 and it signifies that there was a good to very good agreement between the raters (Table 2).

As hypothesized earlier, all the raters identified the absence of preprogrammed reactions in CLBP population and identified the presence of M2-M3 responses (PPR) in healthy individuals in randomly provided EMG patterns pertaining to various perturbations on stable and unstable surfaces.

2.3.2 ANOVA Effect on EMG PPR and VRPPR RMS of EA & RA Muscle

The main ANOVA effect of RA and ES muscle for PPR amplitudes between asymptomatic and LBP population was significantly different at \( p<0.05 \). Further, VRPPR amplitudes \( (p<0.02(RA) \text{ and } p<0.05(ES)) \) were also significantly different in both the groups (Table 3).

No significant differences were found in PPR or VRPPR values of asymptomatic or CLBP group alone. No interaction effect was observed between stable unexpected perturbation, stable expected perturbation, unstable unexpected perturbation and unstable expected perturbation between LBP group and asymptomatic group. It was evident that PPR preparation dysfunction in CLBP population was mainly associated with voluntary movements.
2.3.3 Mean Window Length Data

The mean window length of PPR RMS amplitude at all testing conditions was measured for a pair of electrodes. For ES of asymptomatic subjects, the MWL was (mean ± SD) 53±12 ms and for LBP the MWL was 59±22 ms. For RA; 53±19 ms (asymptomatic) and 52±27 ms (LBP) was observed. The mean window length for mean VRPPR RMS amplitude for a pair of electrodes were reported cumulatively (ES (Asymptomatic 112±15 ms; LBP 140±28 ms)), (RA (Asymptomatic 100±14 ms; LBP 104±29 ms)) for all testing conditions.

2.4. Discussion

2.4.1 PPR

We found a decrease in the PPR RMS amplitude in global muscles of spine in the CLBP population compared to asymptomatic participants. The PPR amplitude of ES in CLBP population was decreased up to 22% compared to asymptomatic participants on UXST perturbation task (Fig 1,3,4). On the other hand, the PPR amplitude of RA was also reduced up to 41%. Similar results were reported in several previous studies. Patients with CLBP were found to demonstrate abnormal reflex response, including reduced reflex gain, slowed latency and anomalous large reflex delays (Hodges 2001; Krajcarski et al. 1999; Radebold et al. 2001; Volpe et al. 2006).

In expected perturbations on stable/unstable surfaces, the CLBP population was unable to gain PPR amplitude as asymptomatic subjects achieved. This finding reflects the poor nature of PPR responses in CLBP subjects during perturbation related motor loading tasks on upper extremities.

Effective neuromuscular control of spinal stability or effective Trunk stiffness is attributed to the role of both intrinsic-innate joint passive stiffness and reflex muscular contributions (Cholewicki et al. 2000). Moreover, Kurtzer et al (2008) emphasized flexible PPRs for multi-joint systems “to account limb-geometry changes that affect the transformation between joint torque and joint motion”.

Franklin & Granata (2007) found that spinal reflexes modulate the spine and help to stabilize with less antagonistic co-contraction, which leads to energy efficient control for stability, than intrinsic stiffness. They also found that increased reflex delay and reduced reflex gain requires greater antagonistic co-contraction to maintain spinal stability. Higher percentages of VRPPR values found in ES and RA muscles of our study may be attributed
to greater antagonistic co-contraction to perturbation tasks in CLBP population (Fig 1 & 2).

The nature of data obtained and plotted indicates very explicitly that the CLBP population has poor PPR modulation during perturbations. The contribution of reflexes to spinal stability accounted for 42% of the total stabilizing trunk stiffness (Moorhouse and Granata 2007). It was assumed from our results that poor PPR activation and modulation along with higher voluntary responses could cause repetitive micro tissue trauma around the neutral zone by abnormal vertebral translations. This notion was supported by many studies. Any reflex modulation increases the stiffness of the joint (De Serres and Milner 1991), which, in turn, prevents too large an unwanted displacement to take place. It was found that large unexpected displacements were nearly halved when reflexes were present compared with no reflex feedback at the wrist and elbow (Sinkjaer and Hayashi 1989; Bennett et al. 1994; De Serres and Milner 1991).

2.4.2 VRPPR

The PPR that was traced along with voluntary response peak RMS amplitudes of ES muscle in CLBP population largely increased from 28% to 49% compared to asymptomatic population on perturbation tasks. The VRPPR RMS amplitude of the RA muscle in LBP population was found to increase marginally (7% to 14%) compared to asymptomatic population on stable/unstable surfaces and expected/unexpected perturbations (Fig 2,3,4).

Similar results were found in studies evaluating muscular activation patterns during voluntary movements in control subjects and persons with CLBP. They have shown that during lumbar flexion, persons with LBP have increased electromyographic activity of the ES muscle and decreased activity of the abdominal muscles (Alexiev 1994; Newcomer et al. 2002; Nouwen et al. 1987; Paquet et al.1994). These higher ES and RA EMG activations can be attributed to spinal reflex delays and reduced reflex gains.

Another possible reason for arise in disproportionate EMG amplitude between ES and RA in CLBP may be due to dysfunction in a reciprocal pattern of preprogramming (Bonnet 1983; Latash 2008) expected to occur with known direction in advance, i.e., moment of trunk flexion initiated by sudden release of load on the outstretched arm in the anterior direction. During the sudden trunk flexion movement, i.e., during the acceleration phase, trunk flexors acted as agonists and extensors as the antagonists and vise versa in deceleration phase, i.e., returning to neutral. As mentioned earlier greater voluntary antagonist co-contraction in ES and RA in respective phases produced inappropriate rise in
EMG during perturbation tasks reflecting disturbances in balancing PPR gain and consequently voluntary responses via reciprocal preprogramming on both directions compared to healthy subjects.

2.4.3 PPR and VRPPR

Another interesting trend was varying RA and ES VRPPR RMS amplitudes on EXUN perturbations. CLBP subjects were found to have an exorbitant difference up to 47.49% compared to 12% difference in asymptomatic population for various perturbations.

In a nutshell, the asymptomatic population tends to balance the agonist and antagonist voluntary responses to create a balanced level of EMG activity and hence improve the performance with learning to different level of perturbations. However, the CLBP group is not able to equalize global trunk agonist and antagonist responses on perturbations and exhibit poor learning across tasks (Dugas and Marteniuk 1989).

With inadequately prepared PPR, the CLBP group further exhibited larger peak voluntary responses, which may place the spine at a risk for instability injury. This may be due to a change in centrally mediated task-dependent presynaptic modulation of the stretch reflex gain which commonly occurs during a movement along with peripheral feedback (Capaday and Stein 1986; Doemges and Rack 1992; Shemmell et al. 2009) or change in muscle activation pattern. Additionally, it could also be a sign of the favored, misrepresented central nervous system strategy used by CLBP subjects.

This absence of PPR in combined VRPPR amplitudes in the CLBP group could have deflated the mean VRPPR RMS amplitude in low back pain patients. In contrast, with this absence, the data was found inflated. This contrasting finding may be an indicator of inadequate preparation of muscles in terms of time (refer to mean window length of LBP), change in property of spinal muscles and inability to modulate the voluntary response to perturbations. Parkinson (Beckley et al. 1993) and cerebellar patients (Nashner 1976) also reported inability to modulate PPR during standing changes akin to CLBP population found in this study. This implies changes in the task dependent modulation action of the downstream inputs and the primary motor cortex to regulate PPR response in CLBP population (Mackinnon et al. 2000).

2.4.4 Study Limitations and Recommendations

The speed of the weight release was not controlled and done by manual process. This could have affected the magnitude of the EMG signal. However, participants were instructed to return to their natural speed as quickly as possible in order to produce an appropriate situation for PPR measurements.
Caution is warranted in interpreting results as mean window length was averaged between pairs of muscles, and randomly chosen EMG readings were used for statistical analysis. Nevertheless, the good Interrater reliability obtained by experienced physicians in our study suggests homogenous outcome measurements and thus indicate no threat to internal validity. Subjects with back pain and higher kinesiophobia scores (Elfving et al. 2007) need to be studied in future to evaluate pain behavior and its relation to trunk muscle reflexes, PPR, and voluntary responses.

However, these results emphasize the importance of PPR and voluntary responses while evaluating and training back dysfunction. This study also emphasizes the need of training for PPR that is mediated by central nervous system to tune the voluntary responses to perturbation along with training short latency reflex components mediated by the peripheral feedback for comprehensive motor control rehabilitation.

2.5 Conclusion

LBP group exhibited poor modulation of highly flexible preprogrammed reactions during perturbation tasks compared to asymptomatic population. A disproportional increase in EMG amplitudes of voluntary responses of global trunk muscles to perturbation was associated with poor PPR modulation in the CLBP group compared to asymptomatic participants.
2.6 References


Table Captions

**Table 1:** Demographics for experimental and control subjects.

**Table 2:** Fliess Kappa (κ) analysis of PPR RMS values as analyzed by set of nine raters for patients with CLBP and normal population.

**Table 3:** Summary of F values obtained in normal and CLBP population for PPR and VR-PPR responses on stable/unstable surfaces for unexpected/expected perturbations.
Table 1: Demographics for Experimental and Control Subjects

<table>
<thead>
<tr>
<th>Characteristics (Mean ± SD)</th>
<th>Controls (n=25)</th>
<th>CLBP (n = 25)</th>
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<tr>
<td>Age (years)</td>
<td>32.16 ± 9.621</td>
<td>40.68 ± 10.606</td>
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<tr>
<td>Height (cm)</td>
<td>171.52 ± 12.018</td>
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<tr>
<td>Weight (kg)</td>
<td>71.76 ± 8.642</td>
<td>73.12 ± 8.308</td>
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<tr>
<td>VAS* Back pain(mm)</td>
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<td>2.7 ± 2.4</td>
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<tr>
<td>RMDQ†</td>
<td>NR</td>
<td>6.2 ± 3.6</td>
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<tr>
<td>Kinesiophobia Score‡</td>
<td>NR</td>
<td>20 ± 0.7</td>
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<tr>
<td>Lifetime highest VAS* pain</td>
<td>ND</td>
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<tr>
<td>Annual LBP duration</td>
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<td>(Days,range)</td>
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<td>Medication taken (Past 12 months, %)</td>
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<td>Activity reduction (Past 12 months, %)</td>
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<td>Past previous LBP episode experience</td>
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*Visual Analog Scale, †Roland Morris Disability Scale, ‡Tampa scale for kinesiophobia – adjusted version-13, NR- Normal, NA- Not Done
Table 2: Fliess Kappa (κ) analysis of PPR RMS Values as Analyzed by set of Nine Raters for Patients with CLBP and Normal Population

<table>
<thead>
<tr>
<th>Surfaces/Perturbations/ Muscles</th>
<th>Groups</th>
<th>(κ) Kappa value</th>
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<tr>
<td>Stable/Unexpected/ES</td>
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<td>CLBP</td>
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<td>Stable/Unexpected/RA</td>
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<td>CLBP</td>
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<td></td>
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ES- Erector spinae, RA- Rectus abdominus
Table 3: Summary of F Values Obtained in Normal and CLBP Population for PPR and VR-PPR Responses on Stable/Unstable Surfaces for Unexpected/Expected Perturbations

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Variable</th>
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<th>Simple Main Effect</th>
<th>Interaction Effect</th>
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<tr>
<td></td>
<td></td>
<td>F</td>
<td>p</td>
<td>F</td>
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<tr>
<td>RA</td>
<td>PPR</td>
<td>48.63</td>
<td>0.05*</td>
<td>2.199</td>
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<td></td>
<td>VRPPR</td>
<td>5.463</td>
<td>0.020*</td>
<td>2.64</td>
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<td>ES</td>
<td>PPR</td>
<td>15.96</td>
<td>0.05*</td>
<td>1.028</td>
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<td></td>
<td>VRPPR</td>
<td>84.20</td>
<td>0.05*</td>
<td>2.391</td>
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</table>

*Significant at $p < 0.005$
Figure Captions

**Figure 1:** Analysis of mean PPR RMS percentages of rectus abdominus & erector spinae muscle (Normalized to stable-unexpected perturbation on normal group) on normal and CLBP group on various perturbation tasks.

**Figure 2:** Analysis of mean VRPPR RMS percentages of rectus abdominus & erector spinae muscle (Normalized to stable-unexpected perturbation on normal group) on normal and CLBP groups on various perturbation tasks.

**Figure 3:** Estimated Marginal RMS mean of PPR & VRPPR values of Erector Spinae in asymptomatic and CLBP population during various perturbation tasks.

**Figure 4:** Estimated Marginal RMS mean of PPR & VRPPR values of Rectus abdominis in asymptomatic and CLBP population during various perturbation tasks.
Figure 1: Analysis of mean PPR RMS percentages of rectus abdominus & erector spinae muscle (Normalized to stable-unexpected perturbation on normal) on normal and CLBP group on various perturbation tasks

Perturbation tasks: UX-Unexpected, EX-Expected, ST-Stable, UN-Unstable
Figure 2: Analysis of mean VRPPR RMS percentages of rectus abdominus & erector spinae muscle (Normalized to stable-unexpected perturbation on normal) on normal and CLBP groups on various perturbation tasks.

Figure 1 and 2: Perturbation tasks: - UX-Unexpected, EX-Expected, ST-Stable, UN-Unstable
Figure 3: Estimated Marginal RMS mean of PPR & VRPPR values of Erector Spinae in asymptomatic and CLBP population during various perturbation tasks.

Figure 4: Estimated Marginal RMS mean of PPR & VRPPR values of Rectus abdominis in asymptomatic and CLBP population during various perturbation tasks.

Figure 3 and 4: Perturbation Tasks: - Measured in Mean EMG RMS (uv), SD-Standard Deviation, Preprogrammed reactions tasks: 1 to 4, 9 to 12, Voluntary response with preprogrammed reactions tasks: 5 to 8, 13 to 16 Surfaces and Nature of Perturbations: Stable unexpected: Tasks 1, 5, 9, 13, Stable expected: Tasks 2, 6, 10, 14, Unstable unexpected: Tasks 3, 7, 11, 15, Unstable expected: Tasks 4, 8, 12, 16.