**Title:** Neuromuscular responses to combined neuromuscular electrical stimulation and motor control exercises in a patient with recurrent low back pain: A case report

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## ABSTRACT

*Objective:* To present the effects of combined neuromuscular electrical stimulation (NMES) and motor control exercises (MCE) on the lumbar multifidus muscle (LM) activation, motor unit firing rate, and movement control in a patient with recurrent low back pain (rLBP).

*Methods:* An Inertial Measurement Unit (IMU) system was used to measure quality of movement during an active forward bend, while ultrasound imaging and decomposition electromyography were used to measure the LM activation and motor unit firing rates during the Sorensen test. These data were collected pre and post intervention (15 minutes of NMES followed by MCE). Overall perceived improvement was also recorded.

*Results:* Thoracic and lumbar angular velocities showed changes post intervention, with increases in LM activation from 68.1% to 97.7%, and 74.2% to 86.7% on the right and left sides, respectively. The motor unit firing rate changed from a range of 7-15 pulses/second (PPS) pre intervention to 4-13 PPS after the intervention, indicating a change in motor unit recruitment, with a perceived improvement of +2 on the Global Rate of Change (GROC) post intervention.

*Conclusion:* These findings indicate improvements in movement control, LM activation, and changes in motor unit behavior suggesting the clinical utility of combined NMES and MCE in the treatment of patients with rLBP.



**Keywords:** *Neuromuscular electrical stimulation, Motor control exercise, Recurrent low back pain, Multifidus muscle*

# INTRODUCTION

Movement coordination impairment (MCI) is one subgroup in people with low back pain (Fritz et al., 2007; Hebert et al., 2011; Hides et al., 2019; Sahrmann et al., 2017). This subgroup has underlying muscle activation deficits, particularly in the lumbar multifidus (LM) muscle (Hides et al., 2019; Hodges & Danneels, 2019; MacDonald et al., 2009). Suboptimal LM activation can compromise lumbar stability, which in turn may increase the risk of injury and pain in the lumbar area (Hodges & Danneels, 2019; MacDonald et al., 2009; Russo et al., 2018). Altered movement control and LM activation deficits exist in people with recurrent low back pain (rLBP) (Hodges & Danneels, 2019; MacDonald et al., 2009; Russo et al., 2018). Many studies have also shown that the LM did not spontaneously recover and further interventions are needed to try and restore the LM function (Hides et al., 1996).

According to the clinical practice guideline for low back pain, motor control exercises (MCE) have been recommended for patients in the MCI subgroup to reduce pain and disability (Delitto et al., 2012). However, one intervention study using MCE demonstrated a non-significant change in the LM cross-sectional area using ultrasound imaging (Hicks et al., 2016). This finding implies a persistence in LM activation deficit, which has been reported to be responsible for rLBP (Hides et al., 1996).

Neuromuscular electrical stimulation (NMES) has been proposed to facilitate motor unit recruitment in patients with LM activation deficit (Baek et al., 2016; Kim et al., 2016). Accordingly, adding NMES to the MCE should improve LM activation along with pain reduction and improvement in disability. However, we need to explore the potential effect of combined NMES and MCE on the LM activation prior to the full implementation of our proposed intervention. Therefore, this treatment report aimed to describe the immediate effect of combined

NMES and MCE on the movement control, LM activation, motor unit behavior, and overall perceived improvement in a patient with rLBP.

## CASE DESCRIPTION

### Patient

The patient with rLBP in this case report is a computer programmer (male, age 34 years and BMI 26.28 kg/m$^2$) working 8 hours per day, 5 days per week. The patient was recruited on an ongoing research study approved by the university institutional review board (MU-CIRB 2018/215.0712), in which inclusion criteria were 1) age between 20 and 40 years old, 2) having more than two episodes of low back pain that interfere with activities of daily living, 3) during remission phase of rLBP, 4) presence of aberrant movement patterns during active forward bend, and 5) an average right and left straight leg raise angle of greater than 91 degrees. An age of less than 40 years, presence of aberrant movements, and an average straight leg raise angle of greater than 91 degrees are clinical criteria in the identification of individuals within the MCI subgroup (Delitto et al., 2012; Fritz et al., 2007; Hebert et al., 2011). Exclusion criteria included 1) presenting a red flag (e.g., spinal infection, tumor, or fracture), 2) previous spinal surgery, 3) diagnosed osteoporosis, severe spinal stenosis, and/or inflammatory joint disease, and 4) definitive neurological signs including weakness or numbness in the lower extremity.

### History

The patient reported the first episode of low back pain five years previously which manifested itself after prolonged sitting. After the first episode, he had rLBP on average of 3 episodes per year, with each episode lasting approximately 3 days. Overall pain (0 to 10 scale) during the last active episode (7 days ago) was 7 on the right side and 6 on the left side, while the level of disability was 5 out of 10. The patient described the pain as dull pain in the lower back

area without any radiating pain after sitting for approximately 1 hour. The pain dissipated after the patient changed from a sitting position to standing or walking for 5 minutes. The patient did not report any numbness or weakness in his lower extremities.

Physical examination

A clinical observation of standing posture demonstrated reduced lumbar lordosis and slight anterior pelvic tilt. The patient had limited trunk range of motion during active forward flexion. Instability catch sign was also observed during the first half of the forward flexion movement (Fritz et al., 2007; Hebert et al., 2011). The patient had no limited hip range of motion during the passive movement test; however, hip muscle length tests showed hip abductor, hamstring, and quadriceps muscle tightness.

Outcome measures

1) Four Trigno™ Avanti sensors (Delsys Inc., MA, USA) were used to collect kinematic data during active forward bend. The sensors were attached over T3, L1, and S2 spinous processes, and 5 cm proximal to the lateral epicondyle of the right femur (Wattananon et al., 2017). The patient was asked to perform 3 consecutive repetitions of active forward flexion while kinematic data were concurrently recorded at 148 Hz using EMGworks 4.7.3 (Delsys Inc., MA, USA). Thoracic, lumbar, and pelvic sagittal plane segment angular velocities were filtered with a $4^{th}$ order low pass filter with a cut off frequency of 10 Hz, and these data were used to explore the smoothness of movement and control during the flexion phase of the active forward bend.

2) Ultrasound imaging (USI) (model CX50, Philips, NV, USA) in conjunction with NMES (Sonopuls 490 combination therapy, Enraf-Nonius BV, Netherlands) was used to measure LM activation. The USI transducer was placed over the L4-5 facet joint. Resting LM thickness was recorded. Then, the patient was asked to perform a maximum voluntary isometric contraction

(MVIC) against a mobilization belt positioned at T3 level, while LM muscle thickness was simultaneously captured. Two pairs of self-adhesive NMES electrodes were placed on the lumbopelvic region. The NMES was set using a scanning interferential mode at 6000 Hz, and amplitude modulated with a low frequency (20-50 Hz) (Fuentes et al., 2010), and the LM thickness at rest was measured again. Then, the NMES intensity was set at maximal pain tolerance, and the patient was asked to perform the MVIC again during this maximal stimulation, in which the LM thickness was again recorded. Theoretically, the LM thickness during combined NMES and MVIC would represent activation of all motor units available in the LM. The percentage of LM activation was calculated by the following formula:

$$\%LM\ activation = \frac{LM_{MVIC} - LM_{REST\_1}}{LM_{NMES+MVIC} - LM_{REST\_2}} X\ 100$$

Where, REST_1 = resting before performing MVIC; REST_2 = resting before combined NMES and MVIC (NMES+MVIC).

3) Surface EMG signals were recorded at a sampling frequency of 2222 Hz using a four-channel decomposition EMG (dEMG) Trigno Galileo wireless sensor (Delsys Inc., Boston, USA). The skin was cleaned using a 70% alcohol swab and then the sensor was attached over LM at the same location as the USI transducer. Baseline noise was assessed and values under 10 microvolts were deemed acceptable. Data were collected from a 30 second MVIC back extension. The dEMG data were then decomposed to individual motor unit action potential trains using the Neuromap software (Delsys Inc., Boston, USA), and individual motor unit firing rates were extracted (Nawab et al., 2010).

4) The overall perceived change was recorded after the intervention using an 11 point Global Rate of Change (GROC) score, which rated the change from +5 (much better) to -5 (much worse). Improvements above +2 were considered to be clinically important (Kamper et al., 2009).

Intervention

The intensity of the NMES was gradually increased to maximal pain tolerance to facilitate motor unit recruitment (Fuentes et al., 2010; Kim et al., 2016). After the NMES was applied to the LM for 15 minutes, the patient then received MCE for 30 minutes using a quadruped rocking backward exercise (Sahrmann et al., 2017). The patient was initially in quadruped position with bilateral shoulder, hip, and knee at 90 degrees. The patient was asked to perform maximal anterior pelvic tilt and gently rock back toward the heels without pelvic movement. When the physical therapist noticed that the pelvis started to move posteriorly, the patient was asked to stop and resume the starting position. The patient performed 15 repetitions of the MCE for 15 sets, with a one-minute resting period at the end of each set. Rating of perceived exertion (RPE) and muscle fatigue were monitored throughout the study protocol. The patient was asked to report any delayed onset muscle soreness within one week follow up.

## RESULTS

The thoracic angular velocity demonstrated fluctuations during late trunk flexion before receiving the intervention. However, the velocity appeared to become smoother during the same period after receiving combined NMES and MCE (Figure 1A). Lumbar angular velocity showed disruption in angular velocity during the initial phase of the movement, while the angular velocity gradually increased in the initial phase post intervention, (Figure 1B).

The USI data demonstrated increases in the LM activation from 68.13% to 97.75% on the right side and 74.23% to 86.73% on the left (Table 1). The motor unit behavior (firing rate) ranged between 7 and 15 pulses/second (PPS) pre intervention (Figure 1C), this showed a shift to lower firing rates between 4 and 13 PPS after the intervention (Figure 1D). Overall perceived

improvement was rated as +2 at post intervention. The averaged RPE throughout the study was 4.7 out of 10. No muscle fatigue was reported during the intervention, and the patient reported no muscle soreness one week after the intervention.

## DISCUSSION

The results demonstrated improvement in movement control, increase in the LM activation, and reduction in the LM firing rate suggesting an immediate effect of the combined NMES and MCE on the neuromuscular system. These changes could have primarily resulted from neuromuscular adaptation (Gabriel et al., 2006), in which the electrical current from the NMES enhance motor unit recruitment in the LM (Baek et al., 2016; Kim et al., 2016). Theoretically, the muscle utilizes two concurrent strategies when requiring a greater muscle contraction (De Luca, 1985; De Luca & Contessa, 2015). The first strategy is spatial recruitment, or increase in the number of active motor units in the muscle (De Luca, 1985; De Luca & Contessa, 2015). Another strategy is temporal recruitment, or increasing the firing rate of individual motor units (De Luca, 1985; De Luca & Contessa, 2015). The findings show a trend towards a lower firing rate which can be related to the recruitment of the slower larger motor units, or a slowing down of the recorded motor unit pool (Nawab et al., 2010), both of these could indicate an improved efficiency in motor unit recruitment after the intervention.

A previous study has suggested that an activation level below 80% may be considered as a muscle activation deficit (Hart et al., 2010). The ultrasound imaging data at pre intervention demonstrated the patient with rLBP had LM activation below 80% indicating persisting muscle activation deficit. However, combined NMES and MCE increased the LM activation to 97.75%

and 86.73% suggesting that the level of muscle activation has been restored after intervention (Hart et al., 2010).

Reduction in the LM firing rate could indirectly suggest that increased LM thickness resulted from the voluntary recruitment of the larger later recruited motor units being activated, which supports the concept of a spatial strategy rather than a temporal strategy (Gabriel et al., 2006). However, it might be argued that any decrease in the LM firing rate may be due to muscle fatigue (De Luca, 1985). Patient reported the RPE as 4.7 out of 10 during the study protocol, and no muscle soreness had been reported within one week follow up. Therefore, the change in the LM firing rate is unlikely to be due to muscle fatigue. In addition, the LM is primarily responsible for postural control and has a greater proportion of low twitch muscle fiber type (Russo et al., 2018). The restoration of the contraction of low twitch muscle fibers may also be related to a reduced motor unit firing rate; however, further work is required to explore this interrelationship.

The smoothness and gradual increase and decrease in thoracic and lumbar angular velocity after intervention indicate improvements in movement control (Wattananon et al., 2017), which could be as a result of greater LM activation. In addition to change in kinematic data, this change was noticed by the patient with an overall perceived improvement after the intervention with a score of +2 in the GROC. Therefore, the changes in angular velocity seen could be considered as a clinical meaningful change.

Some limitations should be addressed in this case report. First, this case report was a representation of one patient, which would limit the generalizability of our findings. Replication with an adequate sample size should be performed to further explore the changes reported. However, the data presented could be used to inform a future study to better investigate the role of the LM activation in patients with rLBP. Another limitation is hip muscle tightness, which

potentially altered lumbopelvic movement control. We did not control muscle tightness in this case report as hip muscle tightness is commonly found in patients with low back pain. Further comprehensive intervention study should take this into consideration.

## CONCLUSION

Altered movement control and LM activation deficits exist in patients with rLBP. This study was to present the effects of combined NMES and MCE on the LM activation, motor unit firing rate, and movement control in a patient with rLBP. Results demonstrate improvements in movement control, LM activation, and changes in motor unit behavior. These findings suggest the clinical utility of combined NMES and MCE in the treatment of patients with rLBP.

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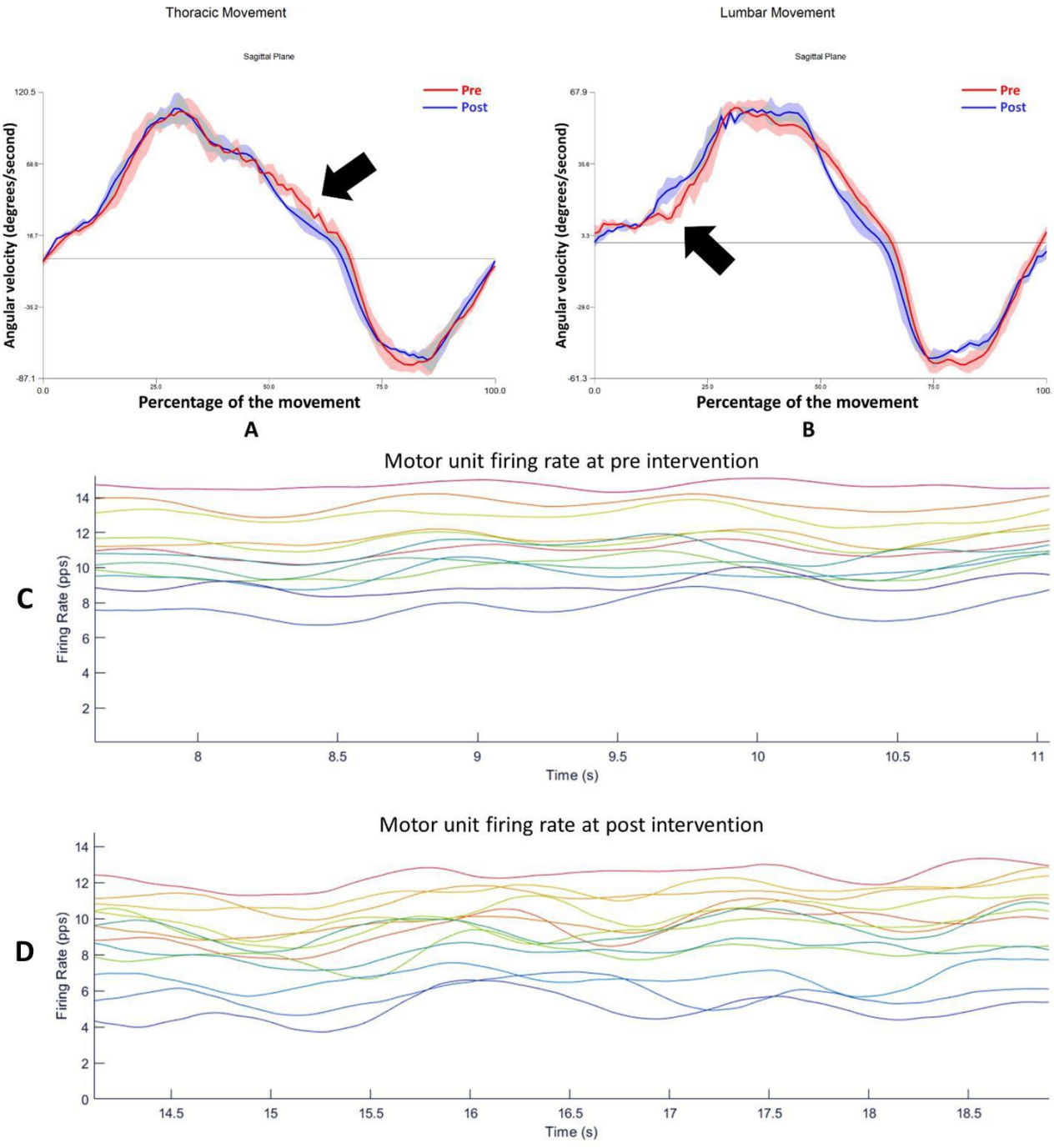


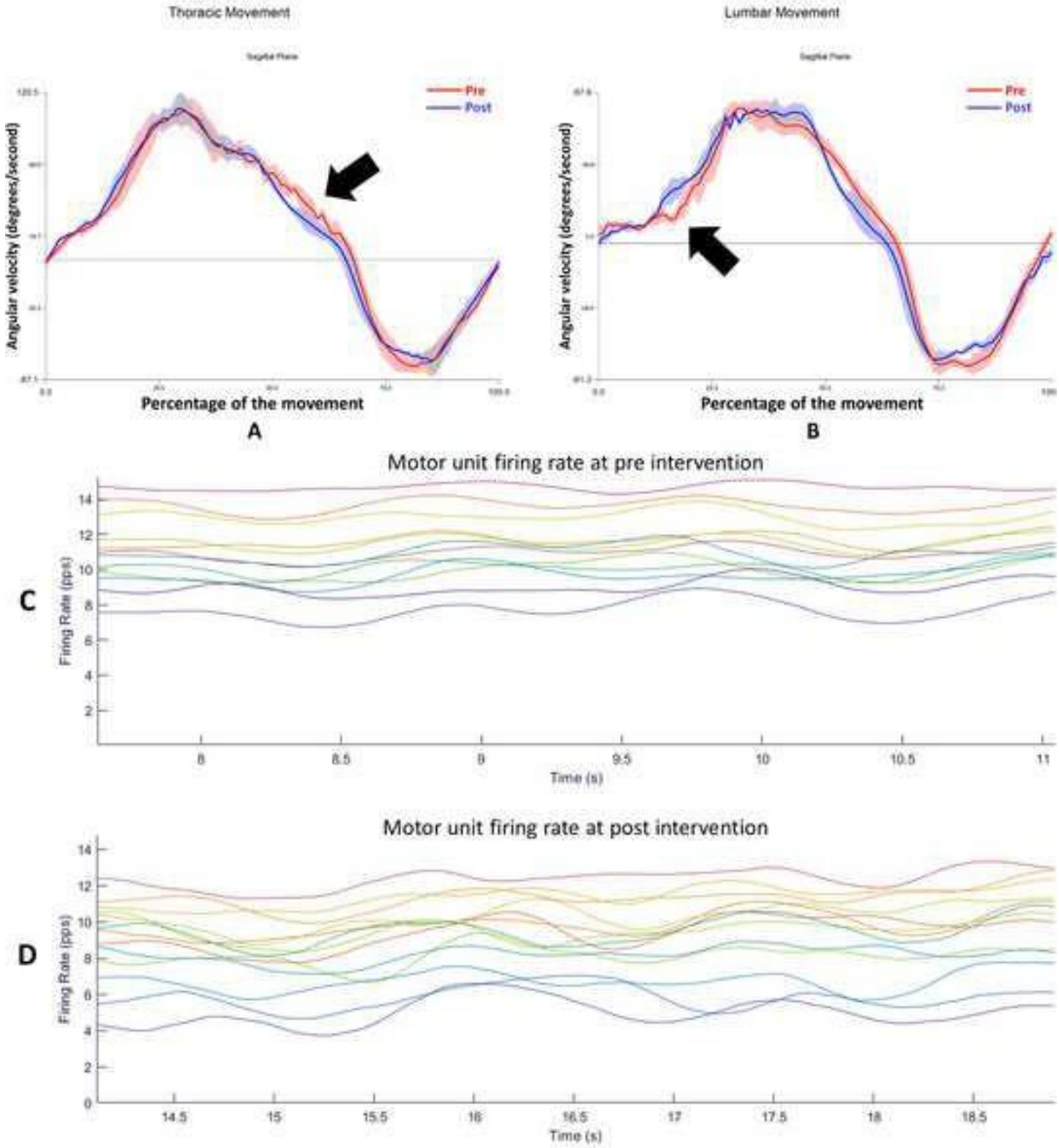

**Figure 1.** Mean (solid line) and standard deviation (shade) of thoracic (A) and lumbar (B) angular velocities during active forward bend. Fluctuation in thoracic angular velocity is detected during late forward bend before intervention (red). The velocity appears to become smoother during the same period after intervention (blue). Similarly, disruption in lumbar angular velocity is found during the initial phase of the movement at pre intervention (red), while the angular velocity gradually increases in the initial phase post intervention (blue). The motor unit behavior (frequency) during maximum voluntary isometric contraction of left lumbar multifidus muscle at pre (C) and post (D) intervention. Noted range of firing rate is shifted to lower range after intervention.

**Table 1.** Lumbar multifidus muscle thickness during different conditions and percentage of lumbar multifidus muscle activation at pre and post intervention

| Side | Pre-intervention | | | | | Post-intervention | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | $LM_{REST\_1}$ | $LM_{MVIC}$ | $LM_{REST\_2}$ | $LM_{NMES+MVIC}$ | %LM | $LM_{REST\_1}$ | $LM_{MVIC}$ | $LM_{REST\_2}$ | $LM_{NMES+MVIC}$ | %LM |
| Right | 3.32 | 3.94 | 3.31 | 4.22 | 68.13 | 3.35 | 4.22 | 3.34 | 4.23 | 97.75 |
| Left | 3.36 | 4.08 | 3.31 | 4.28 | 74.23 | 3.36 | 4.21 | 3.34 | 4.32 | 86.73 |

$LM_{REST\_1}$ = lumbar multifidus muscle thickness at rest for maximum voluntary isometric contraction; $LM_{REST\_2}$ = lumbar multifidus muscle thickness at rest for combined neuromuscular electrical stimulation and maximum voluntary isometric contraction; $LM_{MVIC}$ = lumbar multifidus muscle thickness during maximum voluntary isometric contraction; $LM_{NMES+MVIC}$ = lumbar multifidus muscle thickness during combined neuromuscular electrical stimulation and maximum voluntary isometric contraction; %LM = percentage lumbar multifidus muscle activation

Figure_1


Thoracic Movement
Lumbar Movement
Pre
Post
Angular velocity (degrees/second)
Percentage of the movement
A
B
Motor unit firing rate at pre intervention
Motor unit firing rate at post intervention
Firing Rate (pps)
Time (s)
C
D

Figure_caption

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| Right | 3.32 | 3.94 | 3.31 | 4.22 | 68.13 | 3.35 | 4.22 | 3.34 | 4.23 | 97.75 |
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