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Recovery of Force Output and Electromyography from Trunk Muscles After Cyclic Passive Loading.

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RECOVERY OF FORCE OUTPUT AND ELECTROMYOGRAPHY FROM TRUNK MUSCLES AFTER CYCLIC PASSIVE LOADING

by

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Continuous loading of the low back tissues results in modified neuromuscular and kinetic output during trunk extension efforts. It is believed that the viscoelastic behavior of these low back tissues is modified, but the ramifications of these loading schemes needs further study for longer durations. The purpose of this research project was to observe force output and muscle activation pattern changes during trunk extension efforts before and up to 60 minutes after passive cyclic loading of the lumbar spine during trunk flexion-extension exercise. Sixteen healthy male and female volunteers (20.3 ± 2.1 yrs, 1.63 ± 0.04 m, 50.2 ± 9.3 kg) participated in the study. An isokinetic dynamometer was used in performing a 10 min set of cyclic trunk flexion-extension at a preset velocity of 0.17rad/s through each participant's range of trunk flexion from seated upright position. Participants performed maximum voluntary isometric contraction (MVIC) trunk extension efforts before, immediately after, and at 15 minute intervals ($T_{15} - T_{60}$) for 60 min after cessation of the passive loading scheme. Maximum and average torque output, as well as surface electromyography (EMG) from thoracic (TP) lumbar paraspinal (LP), rectus abdominis (RA) and external oblique (EO) muscles (bilaterally), were recorded. One way ANOVAs were used to identify changes at each time period of testing compared to baseline values. Alpha was set at < 0.05. Maximum and average torque measures did not change over time ($p > 0.05$). Rate of force development did not change over time ($p > 0.05$). Average EMG did not change over time ($p>0.05$) in TP,LP and RA muscles. There was significant difference

across time for the LEO muscle ($p < 0.04$). Average EMG at T_{15} and T_{30} were significantly lower than the initial pre-loading values. Similarly, a significant difference was present in the right EO over time ($p < 0.01$). Average EMG values at T_{15}, T_{30} and T_{45} were significantly lower than at the initial pre-loading values. All peak EMG data during recovery were significantly reduced compared to the initial value all ($p<0.01$) however, no changes in EMG measures per muscle group were indicated between recovery times ($p > 0.05$) Range of motion did not significantly change from pre to post trials. These data provide inconclusive results as to the force output and EMG modifications when the passive viscoelastic tissues are cyclically loaded in flexion-extension. There are indications of modifications in the EMG detected from EO, providing further potential evidence of neuromuscular modification to potentially compensate for increased compliance of the viscoelastic tissues.

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CHAPTER 1

INTRODUCTION

Low back disorders (LBDs) are a significant condition in industrialized countries even with the advancement of modern technologies used in the work place. Many professions as well as activities of daily living, require continuous bending and lifting heavy weights, which may lead to lower back pain (Granata & Sanford, 2000). The risk of a LBD is closely associated with the magnitude of mechanical loading on the spine (Bakker, Verhagen, Lucas, Koning, de Haan & Koes, 2007) and these disorders are likely to occur when spinal loading exceeds tissue tolerance limits (McGill, 1997). Therefore, an understanding of recovery after spinal loading during task performance is vital for aiding in prevention of this common condition.

During task performance, lumbar soft tissues passively generate forces and moments to initiate, maintain, or terminate trunk motions by repetitive muscle contraction and relaxation (Ning & Nussbaum, 2015). In repetitive lifting movements the posterior muscles of the trunk provide the primary resistance to the gravitational vector acting upon the center of mass COM of the head-arms-trunk segment until the trunk attains a full flexion position (McGill & Kippers 2004). At this portion of the movement the connective supporting tissues are believed to generate sufficient force to allow for myoelectric silence of the paraspinal muscles (Olson,Li & Solomonow, 2004; Dickey, McNortan & Potvin, 2003). It is interesting to note that these connective tissues provide muscle force transmission between the muscle, bone, and other surrounding tissues (Yucesoy, Baan, Koopman, Grootenboer, & Huijing 2005; Yucesoy, Baan & Huijing, 2010). The myoelectric silence of the erector spinae muscles in the trunk flexion posture is suggestive of increased load sharing on passive structures, which causes tissues to succumb under excessive loading conditions and this may be a source of low back pain (Colloca &

Henrich, 2005). Over prolonged periods of time the neuromuscular reflexes are reduced, indicating the reduced sensitization of mechanoreceptors to cyclic and static loading as the mechanical behavior of the tissue is modified either due to tension-relaxation or creep (Claude, Solomonow, Zhou, Baratta & Zhu, 2003; Solomonow, Baratta, Banks, Freudenberger & Zhou, 1999). The muscular activity pattern of the flexion relaxation phenomenon shows significant changes, with the development of creep during static lumbar flexion (Solomonow et al 1999). Prolonged static (Granta, Slota &Bennett., Shin, Shu,Li, Jiang & Mirka., Solomonow et al. 2003) and cyclic (Dickey et al 2003, Olson et al 2004; Olson, Li & Solomonow, 2009; Olson 2011) trunk flexion protocols have been used in humans, to observe the response of the neuromuscular system overtime (Olson et al., 2009)

Previously, viscoelastic properties of lumbar posterior tissues, in-vivo, have been described (Olson et al., 2009; Toosizadeh, Nussbaum, Bazrgari, and Madigan, 2012) and a number of in-vitro studies have investigated viscoelastic properties in individual lumbar posterior ligaments (Ambrosetti-Giudici, Gedet, Ferguson, Chegini, Burger, 2009; Lucas, Bass, Crandall, Kent, Shen & Salzar. 2009; Provenzano, Lakes, Keenan & Vanderby. 2001; Yahia, Audet & Drouin, 1991) the results of which have shown that the tissue strain and strain rate affect stress levels on those tissues (Ning & Nussbaum, 2015). McGill and Brown (1992) imply that prolonged static flexion of the trunk drastically alters the viscoelastic properties of the tissues which may lead to a potential compromise in the stability of the spine. Thus, the connective tissues are shown to provide a significant role in load transfer from the muscle to the bone, and between muscles (Andersson, Oddson, Grundstrom, Nilsson, Thorstensson, 1996; Toussaint, Winter, Haas, Looze, Dieën, & Kingma, 1995). The load transfer abilities of the connective tissues may be the determining factor in the ability of the neuromuscular system to

generate sufficient force during maximum or sub-maximum effort after prolonged stretch (Olson, 2011). Repetitive cyclic stretch has been tested in human models previously during trunk flexion–extension as the posterior lumbar tissues were loaded passively (Olson et al. 2009).

Based upon the previous studies looking into the modifications of viscoelastic soft tissue and the ensuing neuromuscular responses, the goal of this study was to observe the force output and muscle activation pattern changes during trunk extension efforts before and up to 60 minutes after passive cyclic loading. It was hypothesized that the passive cyclic loading of the posterior viscoelastic tissues of the low back will elicit a neuromuscular compensation with the increase in time. It was also hypothesized that the range of motion of the lumbar spine is modified due to the passive loading of the low back tissues.

CHAPTER 2

METHODS

Participants

Sixteen healthy individuals (10 male, 6 females, age 21.6± 2.9 years, height 1.69 ± 0.10 m, mass 69.5± 12.3 kg) with no history of lower back pain/injury were recruited for this study. Participants were excluded if they had any current or previous incidents of back pain/dysfunction within the past 12 months. The procedures were reviewed and approved by the Human Subjects Committee of Southern Illinois University Carbondale (SIUC, protocol #14059). All participants agreed to perform the procedures and signed a written informed consent form prior to participation. After agreeing and signing each participant warmed up by walking for 10 minutes on a motorized treadmill at a comfortable pace to each individual.

Instrumentation

Isokinetic dynamometer.

A Biodex System 3 dynamometer (Biodex Medical Systems, Inc., Shirley, NY, USA) was used to collect reaction moment data and control the passive movement of the trunk, while the participants were secured to an attachment bar fixed across the scapulae of each participant. The attachment bar was fixed to the dynamometer for the passive sessions and maximum voluntary isometric contractions (MVIC) efforts. Isometric mode was used for the MVIC efforts, while the passive mode was used to control the trunk movement during the passive loading session. The MVICs for trunk flexion and extension movements were performed three times in each direction. The dynamometer was calibrated before and after data collection and was within the manufacture's specifications. Data were collected at a rate of 100 Hz and saved for future processing.

Electromyography (EMG).

Surface EMG were collected bilaterally from thoracic paraspinal (TP), lumbar paraspinal (LP), rectus abdominis (RA), external oblique (EO) muscles using a MA-300 system (Motion Lab Systems, Baton Rouge, LA, USA). The skin was abraded and cleaned with alcohol pads prior to electrode placement. Pre-gelled Ag–AgCl electrodes (Biopac Systems, Inc., Goleta, CA, USA) were positioned at a distance of 2.0 cm center to center from the 1.0 cm^2 collection area of each electrode and aligned parallel along the length of the respective muscle. Each electrode pair was positioned with a bipolar configuration over the TP (4.0 cm lateral from the body midline at T11), LP (3.0 cm lateral from the body midline at L3), RA (3.0 cm lateral from the umbilicus), and EO muscles (15 cm lateral from the umbilicus, midway between the 12th rib and iliac crest). A ground electrode was positioned on the skin over the left iliac crest. Surface EMG signals were band-pass filtered 20-500 Hz with a common mode rejection ratio of >100dB at a frequency of 60 Hz, an input impedance of >100 MΩ, and amplified up to 1000 times. Data were collected at a rate of 1200 Hz using a 12 bit A/D board and saved for future processing.

Procedures

Each participant was placed in an upright-seated position and securely fastened to the dynamometer using shoulder harnesses and a lap belt. Each participant performed three MVIC efforts in flexion and extension for 5s with 60 s rest between each exertion in the upright position. The arms were positioned across the chest to prevent any assistance from other body segments during the MVICs. The MVIC efforts were staggered so that flexion and extension efforts were alternated. Subjects were instructed to begin their maximal efforts immediately at the conclusion of a 3-second countdown. After a 10 min rest period an initial passive range of motion was performed to determine the movement parameters for the computer controls. After

the initial passive range of motion was assessed the 10 min passive session (PS) was initiated. This PS was implemented to isolate cyclic sagittal plane loading of the lumbar tissues. This PS consisted of moving each participant passively through his/her full range of trunk flexion, from the upright seated position, while moving within the anatomical limits of the individual. Participants were told to relax and move very little during the session. The angular velocity for each phase of the movement (flexion/extension) was set at 0.17 rad s^{-1}. Once the PS had been concluded, two ROM trial and two flexion/extension MVIC efforts were tested immediately after the PS and 15 min thereafter for up to an hour. After each set of ROM and MVICs, participant were released from the sitting position and were asked to lie supine on an athletic training table and rest for fifteen minutes, until the next set.

Data analysis.

Surface EMG signals collected during the MVIC trials and passive sessions were rectified and smoothed with a low pass fourth-order, zero-lag Butterworth filter with a cut off frequency of 4 Hz. The maximum values from the initial MVIC efforts were calculated and used for the normalization of the EMG of the respective muscle groups for the subsequent EMG peaks and averages during efforts in the post-PS (T_0-T_{60}) MVIC efforts.

Torque data were filtered with a low pass fourth-order Butterworth filter with a cut off frequency of 2 Hz. The rate of force development was calculated by difference between the maximum torque value and the initial torque value over the time period between these two points. Subsequently, the average of the two extension efforts for each time period and the greatest value during MVIC efforts were calculated. During the pre PS and post PS ROM, maximum flexion torques were averaged between two efforts.

Statistical analysis

One-way analysis of variance (ANOVA) with repeated measures was performed over time (pre, T_0, T_{15}, T_{30}, T_{45}, T_{60}) on the dependent variables (force, range of motion and rate of force development) Simple contrast were used to determine differences between time periods when a significant difference was indicated. Alpha was set at < 0.05.

CHAPTER 3

RESULTS

Electromyography

Average EMG values did not change over time ($p>0.05$) in TP,LP and RA muscle groups (Table 1). There was a significant difference across time for the left EO muscle group ($F_{5,148}$= 2.52, $p < 0.04$). Simple contrasts indicated average EMG at T_{15} and T_{30} were significantly lower than the initial pre-loading values. Similarly, a significant difference was present in the right EO muscle group over time ($F_{5,141}$=4.368,$p < 0.01$). Simple contrasts were used to determine that the average EMG values at T_{15}, T_{30} and T_{45} were significantly lower than at the initial pre-loading values. All peak EMG data were significantly different than the initial value: LTP ($F_{5,147}$= 10.134, $p>0.001$), RTP ($F_{5,144}$=21.313, $p>0.001$), LLP ($F_{5,143}$=10.658, $p>0.001$), RLP ($F_{5,142}$= 11.125, $p>0.001$), LEO ($F_{5,144}$=57.37, $p>0.001$), REO (F_{5},137=37.95,$p>0.001$), LRA ($F_{5,137}$=31.92, $p>0.001$), RRA ($F_{5,142}$=20.594, $p>0.001$) (Table 2).

Table 1 Means (±sd) of average EMG for pre and post sessions.

Time (min)	LTP	RTP	LLP	RLP	LEO	REO	LRA	RRA
Pre	0.34 (0.19)	0.42 (0.22)	0.46 (0.27)	0.47 (0.35)	0.36 (0.34)	0.43 (0.26)	0.36 (0.29)	0.27 (0.17)
T_0	0.40 (0.39)	0.36 (0.29)	0.45 (0.42)	0.61 (0.35)	0.25 (0.20)	0.25* (0.24)	0.28 (0.35)	0.25 (0.25)
T_{15}	0.37 (0.41)	0.34 (0.28)	0.42 (0.51)	0.41 (0.38)	0.16* (0.16)	0.14* (0.10)	0.16 (0.17)	0.20 (.23)
T_{30}	0.40 (0.41)	0.36 (0.29)	0.40 (0.38)	0.43 (.36)	0.19* (0.17)	0.34 (0.38)	0.23 (0.30)	0.21 (0.19)
T_{45}	0.48 (0.51)	0.39 (0.30)	0.46 (0.45)	0.52 (0.37)	0.27 (0.20)	0.22* (0.19)	0.17 (0.29)	0.30 (0.29)
T_{60}	0.45 (0.46)	0.44 (0.31)	0.32 (0.32)	0.44 (0.38)	0.28 (0.21)	0.34 (0.26)	0.20 (0.25)	0.32 (0.32)

*Indicates significant difference ($p<0.05$) between other time periods.

TP= thoracic paraspinal, LP= lumbar paraspinal, RA= rectus abdominis, EO= external oblique, L= left side, R= right side.

Table 2 Means (±sd) of peak EMG for pre and post sessions.

Time (min)	LT	RT	LL	RL	LEO	REO	LRA	RRA
Pre	1 (0)	1 (0)	1 (0)	1 (0)	1 (0)	1 (0)	1 (0)	1 (0)
T_0	0.37 (0.38)	0.34 (0.29)	0.44 (0.43)	0.58 (0.37)	0.24 (0.21)	0.24 (0.25)	0.27 (0.31)	0.33 (0.27)
T_{15}	0.33 (0.39)	0.31 (0.29)	0.39 (0.50)	0.52 (0.37)	0.19 (0.21)	0.16 (0.17)	0.21 (0.25)	0.27 (0.28)
T_{30}	0.33 (0.40)	0.33 (0.28)	0.33 (0.36)	0.43 (0.36)	0.22 (0.19)	0.34 (0.36)	0.28 (0.31)	0.31 (0.25)
T_{45}	0.43 (0.51)	0.39 (0.33)	0.41 (0.44)	0.52 (0.37)	0.29 (0.24)	0.25 (0.24)	0.25 (0.28)	0.39 (0.35)
T_{60}	0.42 (0.47)	0.35 (0.34)	0.24 (0.28)	0.44 (0.38)	0.27 (0.23)	0.29 (0.26)	0.25 (0.28)	0.51 (0.37)

Indicates significant difference ($p<0.05$) between other time periods.
TP= thoracic paraspinal, LP= lumbar paraspinal, RA= rectus abdominis, EO= external oblique, L= left side, R= right side.

Force data and RFD

Normalized torque of the passive phase before and after 10 minute PS did not show a statistically significant change from pre to post trials. No significant differences were found in forces during the trials from $T_{0\text{-}60}$ (Table3). RFD for pre and post trials was not statistically significant ($p > 0.05$) in $T_{0\text{-}60}$ (Table 3).

Table 3- Mean (sd) of normalized peak flexion torque values during passive range of motion testing and rate of torque development during the MVIC tests.

Time	Normalized torque	Rate of torque development.(Nm/s)
Pre	65.1 (43.1)	62.3 (34.9)
T_0	63.0 (47.4)	59.6 (38.0)
T_{15}	68.6 (51.9)	67.4 (38.5)
T_{30}	72.7 (46.9)	67.9 (40.1)
T_{45}	91.7 (63.1)	69.8 (49.7)
T_{60}	76.4 (53.2)	69.9 (43.4)

Range of motion (ROM)

Range of motion did not significantly change from pre to post trials. Table 4 provides the data to illustrate the range of motion.

Table 4. Range of motion of the trunk during passive range of motion testing in preloading and recovery periods.

Time	Range of Motion (degrees)
Pre	81.0 (8.5)
T_0	85.4 (6.7)
T_{15}	86.4 (8.1)
T_{30}	85.8 (4.9)
T_{45}	86.2 (4.9)
T_{60}	86.3 (5.2)

CHAPTER 4

DISCUSSION

The purpose of this study was to observe force output and muscle activation pattern changes during trunk extension efforts before and up to 60 minutes after passive cyclic loading of the lumbar spine during trunk flexion-extension exercise. The results show that muscle activity was reduced during recovery, in comparison to the initial measures, it was also observed that the torque output and the rate of force development did not change significantly. The range of motion did not change with the assumed increased compliance of the viscoelastic tissues.

The data from the study did not show any significant changes in the average EMG activity except for the EO activities, However, the peak EMG values for each muscle group (i.e., TP, LP, EO and RA) were significantly different from the initial values, suggesting a reduced ability of the muscles to be maximally activated during the recovery period. Solomonow D, Davidson, Zhou, Lu, Patel, and Solomonow (2008) suggest that cyclic loading decreases the peak muscular activity after work while significantly enhancing the magnitude and timing of the muscular contributions. Olson (2011) states that the lumbar paraspinal muscle activation is regulated by the inhibitory reflex response when the mechanical behavior of the passive connective tissues are modified, thereby indicating the presence of neuromuscular modifications independent of neuromuscular fatigue. Neuromuscular modifications are seen after prolonged loading to maintain joint stability during movement (Olson et al., 2009). Sanchez-Zuriaga., Adams and Dolan (2010), suggests that the delay in the activation of the muscles is due to stress in the soft tissues and not fatigue. Sanchez-Zuriaga et al. (2010) went on to report that fatigue had no effect on the onset of muscle activation but the reflex activation of the back muscles in vivo can be impaired by creep.

The average EMG values were reduced during early recovery compared to the initial pre-loading values for left and right EO muscles, but not for any other muscle group. Similar results were also found by Olson (2011). This could be due to the minimal neuromuscular activation during passive sessions thereby showing that the muscles were not actively involved in resisting or assisting the passive movement as proposed by Olson. As the load on posterior lumbar tissues is increased the angle of trunk flexion also increases, whether statically or in repetitive cyclic movements (Dickey et al. 2003; McGill & Brown1992). The increase in trunk flexion causes the lumbar tissues to become the dominant load bearers (Ning & Mirka, 2012). These repetitive loading schemes reduce the posterior viscoelastic tissue tension independent of muscular fatigue or external load applications (Olson).

It was also observed that the torque output and the rate of force development did not change significantly. Some studies have reported that the acute and prolonged stretching decreases human performance through decrease in force and power and this decrease has been attributed to neural output as well as changes in musculotendinous unit mechanical behavior (Behm, Button & Butt, 2001., Cornwell, Nelson & Sidaway, 2002., Fowles & Sale, 1997). The force output reduction could be due to impaired force transfer from muscle fibers to tendons and maybe due to damage in myotendinous junction (Liber, Woodburn and Friden, 1991). The ability of the musculotendinous structures and the surrounding connective tissues to transfer forces is affected due to changes in mechanical behavior of viscoelastic tissues after loading (Olson 2011). Olson also suggests that EMG peak and average amplitudes are invariant in recovery during flexion and extension phases even though EMG amplitude of the paraspinal muscles is reduced after passive loading. Toussaint et al. (1995) state that when localized muscles are unable to generate sufficient force during repetitive movement, the surrounding

musculature is engaged to compensate and the load is transferred between spinal levels. The thoracic part of the erector spinae has greater mechanical advantage due to its moment arm (Potvin, McGill, Norman, 1991) and the direction in which it exerts force (Macintosh & Bogduck, 1991) could cause the reduction in compression of lumbar spine.

This study also showed that that the range of motion was not significantly influenced by the increased compliance of the soft viscoelastic tissues of the lumbar spine. Previous studies show that the viscoelastic connective tissues become more compliant after continuous stretching and are expected to contribute less to trunk load until greater trunk flexion angles are attained during lifting efforts (Olson, 2011; Olson et al., 2009). Dickey et al (2003) observed that both the flexion angle and flexion-relaxation increased due to repeated spinal flexion. Adam and Dolan (1996) suggest that the changes in flexion angle are due to the increase in spinal creep which is caused by repeated loading. Toosizadeh et al. (2012) report that elastic and viscoelastic properties change during loading but the overall viscoelastic state of the trunk is independent of lumbar flexion angle. It has also been noted that the procedures that require constant torque are more effective in increasing range of motion. Solomonow et al (2003) argue that the cyclic loading of viscoelastic tissues may cause micro-damage in the collagen structure with increases in the muscular force which is applied to the spine in order to limit the range of motion and unload the viscoelastic tissues in order prevent further damage to the soft tissues

CHAPTER 5

CONCLUSION

In summary, the present study reported the inability of the muscles to be fully activated when the passive viscoelastic tissues are cyclically loaded in flexion-extension. The range of motion was not significantly influenced by the proposed increased compliance of the viscoelastic tissues. There are indications of modifications in the EMG detected from EO muscle groups, providing further potential evidence of neuromuscular modification to potentially compensate for increased compliance of the viscoelastic tissues. Further studies are required to evaluate the recovery pattern of the trunk muscles after cyclic passive loading.

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