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Modification of Paraspinal Muscle Activation Patterns during Walking Gait after Prolonged Cyclic Flexion-Extension of the Trunk

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Abstract

The purpose of this study was to assess the temporal and spatial Electromyography (EMG) parameters from Lumbar paraspinal (LP) muscles during walking gait after induced tension-relaxation of the lumbar spinal tissues. Ten healthy participants (22.4 ± 3.9 yrs, 1.80 ± 0.09 m, 83.3 ± 11.2 kg) volunteered for this experiment. A passive cyclic trunk flexion-extension loading protocol was performed for 10 mins. Prior to (pre-loading) and immediately after (post-loading) this loading protocol, each participant performed five walking trials across a walkway. Repeated measures ANOVAs were used to compare LP EMG and trunk and lower extremity kinematics between conditions. LP EMG timing significantly shifted from pre (13.0 ± 24.7 ms after heel contact) to post (70.0 ± 23.9 ms prior to heel contact) loading walking conditions ($p<0.05$). There was no significant EMG amplitude modifications observed. Transverse trunk angle at heel contact was the only significant kinematics difference between walking conditions ($p<0.05$). Passive loading of the viscoelastic passive lumbar tissues significantly modified LP muscle activity and may contribute to a neuromuscular compensatory mechanism to attenuate shock at foot contact when the low back tissues stiffness is reduced. The results from this work may assist in progressing the understanding of mechanisms of low back pain.

Keywords: Electromyography; Locomotion; Gait kinematics

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Introduction

Neuromuscular control of the trunk is vital to the maintenance of posture and attenuation of loads applied to the body. External perturbations to the trunk significantly influence the neuromuscular and mechanical responses by the tissues ensuring static or dynamic equilibrium [1,2]. During walking gait the trunk extensor muscles activate synchronously with the foot contact [3]. This neuromuscular response is believed to contribute to spinal attenuation of the transient vertical ground reaction forces [4].

Individuals with Low Back Pain (LBP) present with greater paraspinal muscles activation, particularly in the lumbar region, compared with healthy controls [5,6]. The higher muscle activity is believed to increase both stiffness of the spine and pain avoidance strategies [7,8]. Artificially simulating trunk stiffness is reported to modify the thoracic-pelvis coordinative patterns of the gait cycle, similar to individuals with LBP [9]. Saline injections to the paraspinal muscles of healthy individuals induced pain symptoms and result in greater Electromyography (EMG) variability during walking [10,11]. Nevertheless, increased muscle activity induces greater trunk stiffness which has been reported to coincide with pain intensity [8].

Performing prolonged durations of activities of daily living increase the potential for neuromuscular fatigue. It is unclear whether fatigue directly or indirectly contributes to the incidents to pain and injury to the low back [12]. Increased neuromuscular fatigue of the lumbar paraspinal muscles in healthy individuals does not change walking gait kinematics, but could provide a compensatory mechanism to attenuate ground reaction forces [13]. Further, delayed activation of the lumbar paraspinal muscles relative to foot contact occurs in healthy individuals after low back fatigue is induced [4]. These examples suggest that the neuromuscular system must be flexible in adapting to the changes either within the constraints of the task or the environment. However, neuromuscular fatigue may disguise the influence of external loading on the passive structures of the system.

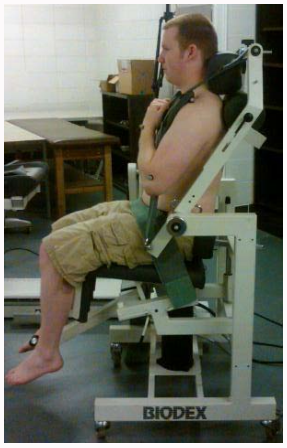

Figure 1: Participant set-up in the Isokinetic Dynamometer.

Simulated loading schemes evoke tension-relaxation and mechanical creep to the lumbar passive viscoelastic tissues (ligaments, fascia, tendons) in humans [1,14]. It is theorized that these loading schemes reduce the stability of the trunk by increasing tissue compliance (reduced stiffness) leading to the manifestation of musculoskeletal disorders [15,16]. In addition, active loading of the lumbar tissues for prolonged durations results in modified paraspinal muscles activation patterns, with either increased temporal activity [17] or reduced temporal activity [18]. However, passive loading schemes also show modified neuromuscular activation in temporal and spatial/amplitude parameters of the EMG signals [19,20]. These loading schemes lead to modified trunk muscle activation patterns during walking gait in individuals possessing low back pain [21]. The relationship between the neural, muscular, and passive connective tissues is complex when linking these with injury and pain. Although previous research has explored the mechanical behavior changes of the passive viscoelastic tissues the direct application of these tissue behavior modifications to activities of daily living is limited.

It is believe that passive loading schemes provide further insight to the neuromotor control of the trunk during gait. Therefore, the purpose of this study was to examine the temporal and spatial parameters of the surface EMG from the lumbar paraspinal muscles prior to and after passive cyclic loading of the passive lumbar tissues. It is hypothesized that peak activation timing will occur prior to foot contact and amplitude parameters of the EMG from the LP muscles will be increase after passive loading during the foot contact in walking gait.

Methods

Participants

Ten healthy volunteers recruited from kinesiology courses (8 males, 2 females, age: 22.4 ± 3.9 yrs, height: 1.80 ± 0.09 m, mass: 83.3 ± 11.2 kg) participated in this experiment. Approval for this study was provided by the Institutional Human Subjects Committee prior to any data collection. All participants gave informed written consent after verbal and written instructions were provided. Participants completed a health questionnaire to verify that that they were free of any neurological disorders, low back pain and lower extremity dysfunction within the last 12 months, cardiovascular disorders, and, if female, were not pregnant.

Instrumentation

Isokinetic dynamometer: A Biodexsystem 3 Isokinetic Dynamometer (Shirley, NY, USA) was used to control the movement of the trunk from an upright sitting position to full trunk flexion while seated and secured in the chair attachment (Figure 1). The dynamometer axis was aligned with the L4-L5 interspace while the pelvis was fixed in the seated position. The trunk was passively loaded in flexion-extension through each participant's range of trunk motion from the erect seated position for 10 mins at a rate of 10°/s [19]. The dynamometer was calibrated before and after data collection and was within the manufacture's specifications. Data were collected at 100 Hz and saved for future processing.

Electromyography: Surface Electromyography (EMG) was collected using a MA-300 system (Motion Lab Systems., Baton Rouge, LA, USA). The skin was abraded and cleaned with alcohol prep 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. EMG were collected bilaterally from the Lumbar Paraspinal (LP) muscles 3.0 cm lateral form the spinous process at the L3 level. A ground electrode was positioned on the skin over the left iliac crest. Surface EMG signals were band-pass filtered 20 Hz to 500 Hz with a common mode rejection ratio of >100 dB at a frequency of 60 Hz, an input impedance of >100MΩ, and amplified up to 1000 times. Data were collected at 1200 Hz using a 12 bit A/D board and saved for future processing.

Kinematics: An infrared motion capture system (Qualisys AB, Gothenburg, Sweden) was used to collect kinematics data (100 Hz) from 14 mm diameter retro-reflective markers adhered to the landmarks on each subject. Reflective spheres were affixed to the skin over boney landmarks bilaterally at the acromion process, posterior superior iliac spine (PSIS), iliac crest, greater trochanter, lateral femoral epicondyle, lateral malleolus, 5^{th} metatarsophalangeal joint, and heel. Markers were also adhered to the skin at the L1 and S1 spinous processes. Residuals were calculated to be less than 0.99 mm during calibration trials. Kinematics data were synchronizing with EMG recordings using the Qualisys Track Manager (QTM) software interfaced with a USB 2533 A/D board (Measurement Computing, Inc, Norton, MA, USA).

Procedures

Participants first warmed up while walking on a treadmill at their preferred walking pace for 10 mins. After the warm-up, their footwear was discarded and the electrodes and markers were placed on the anatomical landmarks, as mentioned previously. Participants performed walking trials barefoot at their preferred gait velocity across the 10 m laboratory walkway 5 times before (pre-loading walking condition) and 5 times immediately after (post-loading walking condition) passive cyclic loading of the trunk for a total of 10 trials.

Following the pre-loading walking trials, participants were secured to the chair attachment of the isokinetic dynamometer. One strap secured the pelvis, and another strap secured the thighs to the chair to ensure minimal movement of these segments. The trunk was then harnessed onto the back of the chair once the dynamometer axis had been aligned with the L4-L5 interspinous space, and after palpation of the lower back to assure proper alignment. The arms

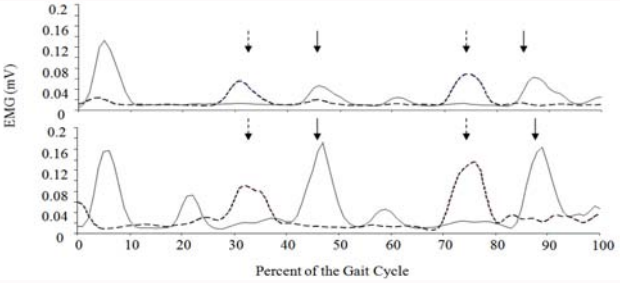

Figure 2: Electromyography profiles of the left (top lines) and right (bottom lines) LP muscles during a single gait cycle of the right leg from one subject. Solid lines represent the pre-loading condition, while the dashed lines represent the post-loading walking condition. Changes in the timing are denoted with the downward arrows.

were held across the chest to minimize arm movements (Figure 1). The range of motion in trunk flexion from erect seated trunk positioning was performed by each individual. Once the range of motion was determined, the passive mode on the dynamometer was engaged to control the passive motion of the trunk at $10°/s^{-1}$ through each participant's range of motion.

Data analysis

Kinematics data from walking trials were low pass filtered at 5 Hz using a fourth-order zero-lag Butterworth filter. A static standing trial was used to reference the segmental position changes performed during walking trials. Custom designed software was used to calculate segment angles of the trunk, thigh, and leg of the right side. The right-hand coordinate system (X-anterior, Y-lateral, Z-vertical) was used to establish the segments coordinates relative the laboratory (X,Y,Z -right hand) coordinate system. Sagittal trunk movement was determined from the markers at the right acromion process and the right PSIS and referenced from the vertical axis of the room coordinate system. Transverse trunk movement was calculated as the position of the left acromion process marker with respect to the right acromion process marker with respect to the laboratory coordinate system. Sagittal trunk velocity was calculated as the first derivative of trunk displacement. Hip and knee joint angles were calculated as the relative angles between the trunk and right thigh, and the thigh and leg, respectively, with respect to anatomical position. Heel contact was determined when anterior displacement of the right heel marker was calculated to be zero. Stride length was determined as the horizontal displacement of the heel marker at consecutive ipsilateral right foot contacts in the anteroposterior direction. Walking velocity was determined as the average anterior/posterior velocity of the PSIS markers over a complete gait cycle. Five complete gait cycles were identified for pre- and post-loading walking trials, respectively.

The EMG signals from the walking trials were rectified and low pass filtered at 3 Hz using a fourth-order zero-lag Butterworth filter. The absolute timing of the LP EMG amplitudes during the gait cycles were initially calculated and compared to the start of each gait cycle. Timing of the LP EMG signals were then determined within the gait cycle and compared between pre- and post-loading walking conditions. The EMG amplitude of the LP muscles during walking conditions was normalized to the largest EMG peak which occurred during the pre-loading walk trials [4].

The EMG signals collected during the passive loading session were rectified and low pass filtered at 2 Hz using a fourth-order Butterworth filter. These data were analyzed to ensure minimal muscle activity during the loading session was present. A baseline signal collection was performed during 5 sec quiet relaxed sitting. This was used as a reference for the EMG signals during the passive loading session. EMG data were collected every other cycle due to memory constraints of the data acquisition system.

Torque data collected during the passive loading session were low pass filtered at 2 Hz with a fourth-order Butterworth filter. These data were collected continuously and used to quantify the tension-relaxation in the posterior lumbar tissues during the loading cycles [19]. The peak torque values during the flexion phase of the passive loading from the first 5 and the last 5 cycles were analyzed to determine the trend of the data.

Statistical analysis

Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS, v16, Chicago, IL, USA). Wilks-Shapirio tests of normality were performed on all data to ensure a normal distribution. One-way repeated measures ANOVAs were performed on the EMG peak timing, normalized EMG amplitude, as well as the walking velocity. The timing of the EMG peaks from each LP muscle at right heel contact was initially compared using paired T-tests. Based upon these results, there were no differences in timing between the muscles, and peak timing data are presented to represent both left and right LP muscles. LP EMG amplitudes, relative to the baseline values during quiet sitting, during the passive loading session were monitored to ensure reduced influence of neuromuscular fatigue. A repeated measures ANOVA was performed on the LP EMG during the passive session. The peak flexion torques from the first 5 cycles and the last 5 cycles were compared using paired T-tests. Sagittal and transverse trunk angles at right heel contact were compared using one-way repeated measures ANOVA. Maximum, minimum, and total ranges of motion for the trunk, right hip, and right knee in the sagittal plane, as well as sagittal trunk velocities and stride length were compared between pre and post loading conditions using one-way repeated measures ANOVA. Alpha set at <0.05. Data are presented as means ± SE, unless otherwise denoted.

Results

Passive loading

Relative LP EMG amplitudes were not observed to increase above base-line levels during the passive loading and did not significantly change over cycles (72.6 ± 3.0% for the left LP, and 66.6 ± 3.0% for the right LP, $p>0.05$). Peak flexion torque values increased from the initial 5 loading cycles (30.8 ± 4.2 Nm) to the last 5 loading cycles (35.5 ± 4.8 Nm) ($p<0.05$), indicating that tension relaxation was present in the low back tissues.

Gait electromyography

During the pre-loading walking condition, LP EMG peaks from both left and right sides occurred 13.0 (± 24.7) ms after heel contact. The timing of these peaks significantly shifted in the time domain to 70.0 (± 23.9) ms prior to heel contact during the post-loading walking condition ($p<0.05$) (Figure 2).

The peak normalized EMG amplitude between sides was not significantly different between conditions (Pre: 0.538 ± 0.02, post: 0.552 ± 0.04, $p>0.05$) or between sides (left: 0.540 ± 0.04, right: 0.549 ± 0.02, $p>0.05$), with no interaction effects present ($p>0.05$). No other amplitude parameters were indicated to change.

Table 1: Mean (SE) trunk angle measures (°) of maximum angle, minimum angle, and range of motion kinematics and mean (SE) angular velocities at these respective measures within the sagittal plane.

Condition	Angle (°)	Angular velocity (°/s)
Range of Motion		
Pre-loading	8.2 (8.7)	8.7 (2.7)
Post-loading	10.7 (13.0)	8.6 (2.6)
Maximum		
Pre-loading	-0.4 (8.6)	46.4 (13.1)
Post-loading	0.9 (6.7)	46.9 (12.0)
Minimum		
Pre-loading	-8.5 (2.6)	-27.0 (20.4)
Post-loading	-9.8 (7.9)	-39.8 (55.6)

Gait kinematics

The walking velocities between pre- (1.05 ± 0.02 $m \cdot s^{-1}$) and post-loading (1.05 ± 0.02 $m \cdot s^{-1}$) conditions were not significantly different ($p>0.31$), indicating that walking velocity was not influenced by the loading scheme. Stride length did not change between pre (1.31 ± 0.06 m) and post (1.36 ± 0.06 m) loading walking conditions ($p>0.8$).

A significant difference between transverse trunk angles at heel contact was present between conditions (pre: -0.37 ± 4.6, post: 1.66 ± 4.0°, $p<0.05$). No significant differences between conditions in sagittal trunk position at heel contact (pre: -6.12 ± 0.03°, post: -7.20 ± 0.7°, $p>0.1$) were observed.

There were no differences in sagittal trunk velocities between conditions. Similarly, sagittal plane range of motion was not different for the trunk segments between pre and post-walking loading conditions (Table 1).

In the lower extremities, there were no changes in the ranges of motion for hip or knee (all $p>0.05$) angles (Table 2). Similarly, no significant differences were present for the maximum and minimum hip and knee angles, respectively (all $p>0.05$) (Table 2).

Table 2: Mean (SE) sagittal of range of motion, maximum, and minimum angle for the hip and knee joints of the right limb.

Condition	Angle		
Pre-loading	ROM	Maximum	Minimum
Hip	42.3 (12.6)	21.9 (4.9)	- 20.4 (8.6)
Knee	71.2 (17.5)	70.0 (7.8)	- 1.2 (11.2)
Post-loading			
Hip	36.8 (3.6)	21.0 (2.4)	- 15.5 (4.1)
Knee	67.1 (11.9)	69.9 (9.0)	2.8 (4.6)

Discussion

The purpose of this study was to measure temporal and spatial parameters of the LP muscle activation during walking gait prior to and after passive cyclic flexion-extension loading of the lumbar viscoelastic tissues. During repetitive loading of the trunk the soft viscoelastic passive tissues of the lumbar spine are exposed to external forces which modify the mechanical behaviors of these tissues. This, in turn, directly modifies the activation of the paraspinal muscles. From this information, it was hypothesized that activation of the paraspinal muscles with respect to the heel contact phase of the gait cycle would be modified both temporally and in amplitude. In agreement with this hypothesis, there was a modified temporal activation centered about the right heel-contact event of the walking gait cycle. However, there was no indication that amplitude modifications were present in the data. A significant transverse trunk rotation was observed between the conditions and may indicate compensation by the trunk during walking to the increased lumbar tissue compliance.

As the low back tissues are cyclically loaded the ability of the viscoelastic passive lumbar tissues (tendonous connections, fascia surrounding the muscles, as well as ligamentous tissues spanning the various levels of the lumbar spine) to sustain the external loads applied to the trunk is modified. Tension-relaxation, or an inability of the tissues to maintain a specified force at a determined tissue length, influences the mechanical behavior of the tissues [17,20]. This loading results in tension developed by the musculotendonous units and lumbodorsal fascia of the paraspinal tissues to occur at greater tissue deformation [12,22]. This tension-relaxation was denoted in the passive loading session. Further, the rate of force development decreases in the LP muscles and lumbar tissues as a result of passive loading [19], while muscle activation has been shown previously to change [23]. The significance of this passive loading scheme indicates an increased viscoelastic tissue compliance with a reduced ability to transmit active force generated from the paraspinal muscles. Accordingly, increased viscoelastic tissue compliance increases the time to respond to a perturbation [1,24]. Evidence of impaired trunk postural control due to increased lumbar viscoelastic tissue compliance has been reported previously [2] and indicates modifications to the control of the trunk segment. Postural control was observed to change in the transverse plane as the trunk was rotating further anterior at heel contact. However, there were no kinematics indications of modified postural control within the sagittal plane during the gait cycle after the passive loading session. Increasing the loading duration may be required to facilitate measurable postural control adaptations within the sagittal plane. Sanchez-Zuriaga et al. [24] reported 60 min duration of static loading significantly modified reflex-response timing of the paraspinal muscles, while a 10 min passive-loading protocol may not significantly contribute to modified reflex responses [25]. Similarly, the specific loading scheme may indicate the response of the spatial and temporal parameters of muscle activation. Cyclic trunk flexion-extension in the sagittal plane primarily emphasizes stress and strain to the posterior viscoelastic tissues (ligaments, fascia, disc), but axial loading during the gait cycle emphasizes compressive loading of the spinal tissues in series with the vertebrae (the discs), as well as transverse rotation [26]. In addition, engagement of the spinal musculature increased the loading on the disc and vertebrae [27]. The surrounding viscoelastic tissues about the vertebrae and discs serve to maintain the integrity of the spine [28], thus assisting in spinal stability. It is possible that the coupled dynamic movements of the trunk in the three cardinal planes serve to assist in the activation of the paraspinal muscles when viscoelastic soft tissue behaviors are mechanically altered.

The temporal shift of the LP muscle activation after passive loading may indicate a compensatory mechanism used by the neuromuscular system. During the gait cycle, the musculoskeletal system must attenuate the forces/shock attained during foot contact [29]. Desensitization of the mechanoreceptors in the soft viscoelastic tissues has been suspected and determined in animal models [15,16]. If these sensory receptors were incapable of responding appropriately due to modifications in the tissue behavior, then even greater delays in the EMG peak would be expected. When the paraspinal muscles

are fatigued there is delayed response of paraspinal muscles activation at heel contact during walking [4]. Contrary to the previous report, the results of the current study indicate EMG peak of the paraspinal muscles occurred prior to the heel contact after the passive loading session. These results are in agreement with previous literature observing trunk extensor activation in individuals with chronic low back pain [6]. It can be speculated that neuromotor input from additional sensory information is utilized to compensate for the impaired afferent responses within the viscoelastic passive lumbar tissues. Additionally, these data indicate the mechanisms of neuromuscular control of the trunk during dynamic activities are different when fatigue and passive loading methods are utilized. An overriding feed forward mechanism may be present to ensure the trunk is properly adjusted to attenuate the shock of the ground reaction forces acting upon the system during gait. Although the passive viscoelastic tissues were the focus of the cyclic loading scheme, the musculotendonous units of the low back were also loaded. In addition, tuning of muscle spindle threshold levels to lengthening of neighboring synergist muscles and connective tissues can modify sensory information sent to the central nervous system [30].

The alignment of the trunk at heel contact is an important indicator of the system's ability to prepare for collisions with the external environment. Specifically, the stride length influences the shock attenuation of the musculoskeletal system [31]. As a result of modified stride length, the position of the trunk at foot contact will also be altered. If stiffness of the tissues is a factor in force attenuation, then the system provides both active and passive mechanisms to increase stiffness:

1. Pre-contact muscle activation

2. Additional lengthening of the ligamentous, tendonous and fascial tissues.

However, there were no measureable changes in the stride length due to the passive-loading scheme. The pre-contact muscle activation may be a viable explanation to compensate for the laxity within the lumbar soft viscoelastic tissues, as previously stated, where the stiffness of the posterior lumbar tissues must be sufficient to allow for greater attenuation of the ground reaction forces. Although not analyzed in the current results, frontal plane kinematics is reported to differ in individuals with current and previous low back pain compared to pain-free individuals [33-35]. Similarly, transverse plane movements of the trunk and pelvis have been assessed during walking gait analyses between pain-free and individuals with low back pain [35]. Crosbie et al. [35] indicated that adaptive changes and not pain per se, were driving factors in the differing axial rotation patterns in individuals with recurrent low back pain. In pain-free individuals, antiphase rotation of the trunk and pelvis is reported, however, in individuals with low back pain this movement is more in-phase [33]. Further studies of the kinematics coupling of the trunk and pelvis are warranted to provide additional understanding of these motor control strategies.

Kinematics changes of the lower extremities, although not present in the current data, may indicate additional compensation by the system to attenuate the forces at foot collision. Stride length has been reported to influence the loading of the body during running activities [36]. It is possible that a reduction in stride length during the walking gait cycle would reduce loads acting upon the system and modify the activation of the trunk musculature. In the current study, the hip and knee joint ranges of motion were used to indicate any changes in the gait kinematics. There were no changes in these values, thus any modifications to the viscoelastic tissue behavior in the low back region may not have influenced lower extremity function. Similarly, muscle activation patterns of the trunk and lower extremities has been modified in individuals with and without low back pain when traveling at different gait velocities [11], while spatial and temporal EMG parameters have been modified when gait velocity is increased or decreased from a preferred pace [37]. However, in the current study the gait velocity did not change between pre and post-loading walking conditions. Any trunk muscle activation pattern modifications due to the passive loading scheme applied cannot be linked with alterations to the lower extremity kinematics parameters of the walking gait cycle as these did not change.

Conclusion

The influence of low back loading on the parameters controlling the gait cycle are still not clear. Although most kinematics variables were not significantly altered as a result of the cyclic passive-loading scheme, there were definite neuromuscular alterations which could potentially lead to adaptations if the loading persists. Prolonged loading of the low back tissues modifies the mechanical behavior of the viscoelastic passive tissues which leads to neuromuscular adaptations in order to control trunk movements. A better understanding of the interaction between loading and neuromuscular responses can provide additional information regarding the susceptibility of the low back to pain and injury. Further research is warranted to explore the potential hazards of these loading schemes to the manifestations of low back pain and injury.

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