Quantification of spine stability: Assessing the role of muscles and their links to eigenvalues and stability

by

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AUTHOR'S DECLARATION

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

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Abstract

Approximately 50% - 80% of the population will experience disabling low back pain at some point in their life. Assessing and developing interventions based on “lumbar stability” and/or joint stiffness to reduce low back pain has been a common research focus. Specific focus has been on identifying which muscles influence lumbar stability/stiffness, with one argument being between focusing training on the transverse abdominis and lumbar multifidus muscles versus broader training approaches involving the entire abdominal wall and erector spinae muscles. However, there has not been research on whether pain reduction was due to increased stability/stiffness or another mechanism. The main goals of this thesis were to determine the effect of individual muscles on stability/stiffness through a two phase process. In the first phase, a model sensitivity analysis was performed to assess the interactions of variables that influence the quantification of stability. Stability was quantified via the eigenvalues (EV) of the Hessian matrix of potential energies at each lumbar level and axis of rotation, for a total of 15 EVs (3 axes of rotation x 5 joints). In phase 2, assessment of clinical interventions on patients with low back pain designed to alter biomechanics was conducted to assess factors in stability/stiffness quantification and mechanisms of action in pain modulation. More detail of the study phases are described below, in order to test the following hypotheses:

1) It was hypothesized that individual muscles affect specific EVs, but no one muscle can be associated with one EV level.
2) It was hypothesized that specific muscles do affect specific planes of stability/stiffness.
3) It was hypothesized that EVs are affected by posture.
4) It was hypothesized that overactivating muscles by increasing muscle activation to 100% MVC negatively affects the EVs.

5) It was hypothesized that the relationship between muscles and specific EVs obtained during simulation remains with real subjects performing loaded tasks.
6) It was hypothesized that coaching and cueing specific movement patterns and motor patterns would alter pain in low back pain patients.
7) If hypothesis 6 is true, then it was hypothesized that changes in pain would be reflected in changes in EVs.

Methods for Phase 1

The first phase involved a sensitivity analysis using an anatomically detailed spine model. Theoretical data including posture, motion and muscle activity were synthesized to include 23 static spine postures, including neutral, 0° - 50° flexion, 0° - 30° extension, 0° - 30° right and left lateral bend, and 0° - 40° right and left axial twist, all in increments of 10°. For each posture, all eleven muscles included in the model, some with several fascicles, were artificially activated to 50% MVC. A knockout approach ensued whereby activity in single muscles were systematically reduced to 0% MVC or increased to 100% MVC. The relationships between the 15 EVs and the changes in muscle activity and posture were assessed. This muscle knockout model was repeated with actual muscle activity values obtained from electromyographic (EMG) signals and postures obtained from four subjects who performed a walking task with a 15 kg load in each hand.

Results for Phase 1

The sensitivity analysis showed that the abdominal muscles contribute a greater stabilizing effect on the L4 and L5 EVs, while the multifidus and erector spinae muscles contribute a greater effect on the L1, L2 and L3 EVs. When examining the effect of muscles on a specific plane in terms of influencing stability/stiffness, it was found that the abdominal muscles contribute a greater effect on the bend axis and twist axis EVs than the flexion axis EVs, while the erector spinae muscles contribute the greatest effect on the flexion axis EVs. Posture was found to have a biologically significant effect on EVs, with the 50°

flexion and 30° extension postures having the most detrimental effect in terms of compromising stability/stiffness. In addition, when there was a 10° excursion in any axis, there was little change in the EVs, while postures at angles greater than this were often associated with decreases in stability/stiffness in some EVs. Increasing the muscle activation from 50% MVC to 100% MVC did not have a large effect on most EVs, but when there was a meaningful change, as defined by a change of 10% or greater in the EV, the 100% MVC activation level always resulted in more stability/stiffness at that particular EV. Finally, using actual EMG and lumbar angle patterns resulted in similar results as the theoretical data, as expected. Interpretation of these findings is limited by the following. Even though EVs changed, there is no guarantee that the magnitude of change in one EV could be interpreted to equal a similar magnitude of change in another EV, nor may it be assumed that EVs have a linear relationship with stability/stiffness. These results suggest that when the goal is to increase lumbar stability, a neutral spine should be maintained and activating the larger abdominal muscles is more important than activating the transverse abdominis or multifidus, as proposed by some clinical groups.

Methods for Phase 2

Four case studies of individuals with chronic low back pain were recruited from whom kinematic, kinetic and EMG data were collected in addition to a measure of pain intensity using an 11-point verbal numerical rating scale. Pain provocation tests were performed by a clinician (professor Stuart McGill) to identify the motions, postures and loads that exacerbated their pain. Then these tasks were repeated while the motion and EMG data was collected. This was followed by interventions coached by the clinician that could include the abdominal brace (stiffening the abdominal wall), latissimus dorsi stiffening, incorporating a hip-hinge motion rather than spine bending, or any combination of these. The intention of the intervention was to immediately reduce pain intensity. These tasks, arranged in a repeated measures design, were assessed with the anatomically detailed spine model to calculate stability/stiffness from evaluation of the 15 EVs, and lumbar compression and shear forces.

Results for Phase 2

The results from phase 2 suggest that pain was sometimes reduced by altering motions, postures and load, but the mechanism of what proved effective and the degree of success was variable from patient to patient. In most situations, the EVs, lumbar compression forces and lumbar shear forces increased due to the intervention that was chosen. In addition, the lumbar flexion angle typically trended to a more neutral posture and in tasks where spine motion occurred, there was less spine motion when using the suggested intervention. Further, the biomechanical variable that would be expected to change based on clinical assessment did not always react in the expected way (i.e. a compression intolerant individual would be expected to have decreased compression linked with decreased pain, but this did not occur). While the stability/stiffness increased, the associated compression was tolerated suggesting that the increase in concomitant stiffness enhanced the compression load bearing tolerance.

Overall Conclusions

This thesis showed that careful examination of the EVs did not offer substantial insight into links between changes in individual EVs and individual muscles, as muscle activity was not reflected in the EVs. Specifically, single muscles contributions were not reflected in specific EVs as was hypothesized. Further, it was difficult to interpret the EVs collectively because of the inherent non-linearity between EV magnitude and changes in muscle activation/stiffness; it can only be said that there was more or less stability/stiffness with each change in an EV, not how much. In addition, pain reduction appeared to be due to a combination of altered motions, postures and loads, but this did not result in systematic EV changes. Globally, the present work provides evidence supporting the idea that maintaining a neutral posture and activating the abdominal muscles results in less pain and larger EVs, suggesting an increase in stability/stiffness. This work has potential for informing clinicians on possible options for immediate reduction in low back pain.

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0/A	Comparison between 0% MVC and Actual EMG
AP	Anterior to Posterior
DM	Distribution Moment
DOF	Degree of Freedom
EMC	Expected Maximal Contraction
EMG	Electromyography
EO	External Oblique
EV	Eigenvalue
FAE	Flexion Angle Excursion
Ilio	Iliocostalis Lumborum
IO	Internal Oblique
L1B	L1 lumbar level, Bend axis
L1F	L1 lumbar level, Flexion axis
L1T	L1 lumbar level, Twist axis
L2B	L2 lumbar level, Bend axis
L2F	L2 lumbar level, Flexion axis
L2T	L2 lumbar level, Twist axis
L3B	L3 lumbar level, Bend axis
L3F	L3 lumbar level, Flexion axis
L3T	L3 lumbar level, Twist axis
L4B	L4 lumbar level, Bend axis
L4F	L4 lumbar level, Flexion axis
L4T	L4 lumbar level, Twist axis

L5B	L5 lumbar level, Bend axis
L5F	L5 lumbar level, Flexion axis
L5T	L5 lumbar level, Twist axis
LD	Latissimus Dorsi
LES	Lower Erector Spinae
Long	Longissimus
ML	Medial to Lateral
Mult	Multifidus
MVC	Maximum Voluntary Contraction
NRS	Numerical Rating pain Scale
A/100	Comparison between Actual EMG and 100% MVC
Pars	Pars Lumborum
QL	Quadratis Lumborum
RA	Rectus Abdominis
RVC	Reference Voluntary Contraction
TrA	Transverse Abdominis
UES	Upper Erector Spinae
VAS	Visual Analog Scale

Chapter 1
Introduction

One of the most prevalent ailments in the adult population is low back pain. Some clinicians appear to be able to alter the mechanics and pain in individuals with low back pain. The interventions used may include a variety of mechanical techniques to change posture and muscle activity, but the mechanisms for altering pain remain unknown. Investigating the mechanics of pain mechanisms and interventions may improve the management and identification of patients likely to respond to conservative management. Moreover, this type of investigation (understanding mechanical pain in many forms) may help improve the management and clinical decision making process with this heterogeneous group of patients. An underlying assumption is that specific motions, postures and external loads, cause tissue overload resulting from increased stress concentrations leading to tissue irritation and the development of pain. The corollary to this assumption is that altering motions, postures and loads can be used by skilled clinicians to reduce pain (McGill 2007). This thesis was designed in two phases to first, further understanding of quantitative stability analysis and second, to quantify the mechanisms incorporated into some clinical kinesiological approaches, such as altering movement and muscle recruitment patterns, in an attempt to modulate back pain.

A more complex concept of spine function includes that of stability. The flexible column does not have sufficient stiffness to support the weight of the upper body without buckling unless muscles are activated and stiffened around the column (Lucas & Bresler 1961). Perturbed muscle activation patterns leading to instability have been shown to be both a cause and consequence of low back pain. Addressing the perturbed patterns with corrective exercise appears, at least in some patients, to reduce or eliminate their pain immediately. Insufficient stability is thought to allow micro movements in the spine motion segments resulting in painful stress concentrations of innervated tissues (McGill 2007). For example, if it is found that increased torso stiffness and stability consistently results in decreased low back pain with

loading tasks, clinicians could focus on prescribing movements and postures that primarily increase stability/stiffness. This could also underpin techniques for patients to perform currently painful daily activities in such a way that increases the stability/stiffness in their low back to try to decrease their pain.

One method of attempting to quantify low back stability has been developed by McGill and colleagues (eg. Cholewicki & McGill 1996). The approach uses an anatomically detailed model of the lumbar spine that represents 118 muscle lines of action spanning six lumbar joints (L5-sacrum to T12-L1). A number of variables are calculated, including active muscle and passive tissue forces and joint compressive and shear forces and moments, and quantitative stability. It uses the idea of elastic potential energy to calculate an eigenvalue (EV) for each axis and joint, for a total of eighteen EVs (six joints, three axes). These EVs are arranged into an 18x18 Hessian matrix where the diagonal elements are used to calculate an overall spine stability index, or the EVs are examined individually with the assumption that the lowest EV will be the level of least stability/stiffness (Cholewicki and McGill 1996; Howarth et al. 2004).

This thesis was centred around the EVs of the spine model and what these actually represent in biomechanical terms. The first phase involved sensitivity tests of several variables to determine which variables are important to influence stability/stiffness and investigate the links between the individual muscles and the various EVs. Part two of phase one involved using actual data to address whether the conclusions from the sensitivity analysis still hold true using actual EMG patterns and spine angles. Five hypotheses emerged:

1) It was hypothesized that individual muscles affect specific EVs, but no one muscle can be associated with one EV level.
2) It was hypothesized that specific muscles do affect specific planes of stability/stiffness.
3) It was hypothesized that EVs are affected by posture.

4) It was hypothesized that overactivating muscles by increasing muscle activation to 100% MVC negatively affects the EVs.
5) It was hypothesized that the relationship between muscles and specific EVs obtained during simulation remains with real subjects performing loaded tasks.

Phase two of this thesis addressed the relationship between EVs, mechanisms and pain of individuals with low back pain. Several questions emerged; Can pain be altered by changes in movement and muscle activation patterns?; if so, do these alterations influence spine stability/stiffness? If the specific level and axis of instability can be determined for each patient, are the muscle activation patterns that reduce pain linked or associated with those predicted by the model? Two hypotheses emerged:

6) It was hypothesized that coaching and cueing specific movement patterns and motor patterns would alter pain in low back pain patients.
7) If hypothesis 6 is true, then it was hypothesized that changes in pain would be reflected in changes in EVs.

If hypothesis 7 was true, study of the EVs could guide clinical intervention in the future. Specifically, while not posed as a formal hypothesis, it was expected that the sensitivity testing of EVs (phase 1) would suggest which muscles were important to influence stability/stiffness and pain changes in phase 2. The muscle activation patterns applied through clinical intervention may or may not match predicted stability/stiffness variables, but may match other variables such as joint load or muscle activity.

Since all patients have different presentations in terms of painful exacerbating motions, postures and loads, and because each patient will have seen many other clinicians and tried different strategies to reduce their pain, phase 2 had no standard of base disability or interventions used. These issues presented a challenge to formulating an appropriate experimental design. Therefore, this phase of the thesis reports a series of case studies that parallels high level clinical practice. In summary, phase one establishes a theoretical framework for understanding the links between movement, posture and muscle activity, which

results in force and ultimately spine load and stability/stiffness. Phase two forms a “proof of principle” to link laboratory observations with theory and possible relevance to clinical practice.

Chapter 2
General Review of the Literature

2.1 Overview

Approximately 50% to 80% of the human population will experience back pain at some point in their life (Andersson 1998). Back pain is the most common ailment that limits activity in individuals younger than 45, and third most common in individuals ages 45-65, behind only arthritis (Frank et al. 1996). However, the source of pain is often unknown despite efforts to determine the pathophysiology. Studies have shown low back pain to be associated with many things including muscle fatigue (Takahashi et al. 2007), posture (Granata & Wilson 2001), and mechanical stability, as defined as the ability to withstand buckling (Cholewicki and McGill 1996; Panjabi 1992a; Panjabi 1992b).

Provocative testing is based on the notion that mechanical irritation of a sensitive tissue will provoke pain, thus identifying the motions, postures and external loads that cause pain. Once provoking motions, postures and loads are identified, which give some insight into injury mechanisms, clinical interventions alter them to reduce pain (McGill 2007). The mechanisms by which pain reduction occurs remains unknown and understanding them is a goal of this thesis.

Interventions are also thought to alter stiffness and stability. Movements aimed to increase spine stability/stiffness are commonly used as a treatment for low back pain (Kavcic, Grenier & McGill 2004b; McGill 2007). However, it is currently unknown whether the stability exercises decrease pain due to an increase in stability/stiffness or a change in some other factor, such as spine compression or shear loads. Therefore, this study will examine the relationship between pain reduction and spine stability/stiffness.

2.2 Anatomical Candidates of Pain

There are four conditions required for a structure to be a cause of back pain. First, the structure must be innervated. Second, there must be evidence that shows the structure is capable of producing the

pain seen clinically in normal individuals. Third, the structure must be susceptible to painful diseases or injuries, or loads in excess of the pain tolerance. Finally, reliable and valid diagnostic tests must indicate that the structure can be a pain source in individuals affected by low back pain (Bogduk 2005).

There are a number of different possible candidates for low back pain, with three leading contenders: muscle pain, zygapophyseal joint pain, and discogenic pain (Bogduk 1995). Of these three types of pain, the least is known about muscle pain. Although patients do experience muscle pain, there is no scientific evidence that allows clinicians to know exactly what the pain is (i.e. muscle spasm or something else) or how to diagnose it. No measurable entity has been scientifically proven to be indicative of a muscle spasm, including EMG activity (Bogduk 1995). Therefore, the prevalence of spinal muscle pain is unknown.

The most common type of low back pain is disc pain, with approximately 40% of pain being disc related (Bogduk 1995). Disc pain is diagnosed using discography. Discography is a procedure where a contrast agent is injected into a disc while the pain response is being measured. Following injection into the disc, a computed tomography scan is often taken for further evaluation. This procedure allows the clinician to diagnose disc degeneration and disc herniation (Walsh et al. 1990). It has been shown that discography will not provoke pain in healthy individuals, making this a good method to diagnose disc pain (Walsh et al. 1990). One condition that has been identified as causing disc pain is internal disc disruption. Internal disc disruption is diagnostically characterized by pain upon discography and radial fissures shown on computed tomography (Bogduk 1991).

Zygapophyseal joint pain is the second most common type of spinal pain, with a prevalence of approximately 15 – 40% depending on the country and type of study. This type of pain cannot be diagnosed by CT or clinical features; it can only be diagnosed by anaesthetizing the painful joint under radiological guidance (Bogduk 1995). However, there is a high placebo response rate in zygapophyseal joint blocks (Schwarzer et al. 1995), and a high false-positive rate (Schwarzer et al. 1994).

One way to differentiate between zygapophyseal pain and discogenic pain is determining whether the pain is midline or paramidline pain, with midline pain being in-line with the spinous process, and paramidline pain being lateral to the midline. Depalma et al. (2011) did a retrospective study to determine the prevalence of discogenic, facet joint, and sacroiliac joint pain based on the pain location. He found that if a patient has midline pain, it is much more likely that they have discogenic pain versus facet joint pain or sacroiliac pain. If the patient does not experience midline pain and experiences paramidline pain, there is a greater likelihood that the individual has either facet joint pain or sacroiliac joint pain. However, there is no way to differentiate between facet joint pain and sacroiliac joint pain if paramidline pain is present (Depalma et al. 2011).

2.3 Provocative Testing

Provocative testing is used to identify the postures, motions or loads that result in discomfort in individuals with low back pain. These tests can also help distinguish between joint, muscles or nerves that are causing the pain. Once the cause of pain has been determined with the provocative tests, exercises and avoidance strategies can be implemented to remove the cause, therefore remove the pain (McGill 2007).

Compression tests are used to help determine the compression tolerance or possible end plate and vertebral body damage in an individual. One such test is the heel drop test. This test involves the patient rising on the balls of their feet and dropping down to flat foot, imposing a compressive load on the spine. This load is typically 2.5 – 3 times body weight. A second compression test commonly used is the seated compression test to determine if compression intolerance is related to posture. This test involves the patient sitting on a stool and pulling up on the seat pan. This is performed for both an upright posture and a flexed posture, giving insight into the relationship between posture and compressive loads (McGill 2007).

Several tests are typically used to determine if the back pain is discogenic. For example, one is the McKenzie posture test that requires the patient to lie prone in one of three positions: arms relaxed, chin resting on the fists, or supported on the elbows. If the patient finds relief in these postures, it indicates that the pain is likely discogenic. When the patient returns to a standing posture, if they feel more stable or less pain than before assuming the McKenzie posture, and when reactions to leg raise and pelvic rock tests are administered, the patient can be further classified into the "posterior discogenic" category (McGill 2007).

Pain due to an aggravated, or sensitized, nerve is important to diagnose so that exercises can be prescribed that do not further irritate the affected nerve. The sensitivity or trapping of the sciatic nerve can be tested in a variety of ways, including the sitting slump test and the supine passive leg raise test. The sitting slump test requires the patient to slouch while sitting on a table or chair. The clinician elicits progressive nerve tension in three steps: 1) the leg is extended at the knee, 2) dorsi flexion is added, and 3) cervical spine flexion is added. If none of these steps cause symptoms, the sciatic nerve is not the cause of the pain. However, if pain does arise, further testing should be done for more insight using a test such as the supine passive leg raise test (McGill 2007).

The supine passive leg raise test can be misleading if not performed correctly because it imposes both increased nerve tension and increased muscle tension. Therefore, it is important that the clinician constantly palpate the hamstrings to monitor muscle tension. While palpating the hamstrings, the clinician raises the leg to the point where pain arises. The cervical spine is then flexed, suggesting neural tension as the cause of pain if pain increases in the back or along the sciatic tract. Finally, if pain is reduced while the leg is being lowered, it indicates that the nerve root is not adhered or tethered (McGill 2007).

Lumbar joint shear stability is tested manually by the clinician via the prone instability test. The patient is asked to lie prone with the body on a table, legs hanging off and feet on the ground. While the

patient relaxes the torso, the clinician applies a small force, no more than 1 kg, downward onto every spinous process beginning at the sacrum. If pain is present or the clinician feels shear displacement, that segment is considered unstable. The patient then raises their legs to activate the lumbar extensors, which should reduce shear instability and pain when the same force is applied to each segment. If pain increases with contraction, the individual may either be compression intolerant or extending their legs using lumbar extension instead of hip extension. If pain decreases, it indicates that exercises that enhance lumbar extensor contraction, increasing stability/stiffness, should be prescribed to help decrease pain (McGill 2007). This test was shown to be most predictive of those who would do well with a stabilization, corrective exercise approach (Hicks et al. 2005).

These provocative tests, among several others, help identify the cause of low back pain in each individual. Once this cause is established, the intervention suggested can be tailored to each patient to most effectively correct the cause, ultimately reducing pain.

2.4 Clinical Diagnosis and Interventions

Spinal dysfunction is often associated with alignment impairments, stabilization impairments and impairments in spinal movement patterns. These issues then contribute to painful diseases in the anatomical structures such as the vertebral disc and zygapophyseal joints. Correcting the abnormal stresses on the spinal column by training the trunk muscles to hold the vertebral column in the optimal alignment and reducing excess movement often will alleviate the patient's pain (Sahrmann 2002).

A common low back impairment is classified as extension-rotation syndrome. As the name suggests, symptoms are increased during extensile and axial rotational movements. Often the back extensor muscles are recruited more heavily than the abdominal muscles when the patient attempts to stand up. If the patient is bending forward, there is typically a more dominant recruitment of the back extensors than the hip extensors. The imbalance of the dominant back extensor muscle recruitment compared with the abdominal muscle recruitment must be corrected to alleviate pain. This is achieved

through instruction on the proper techniques for performing daily activities that contribute to the impairment. As indicated by the patient's specific needs, exercises are also used to decrease rotational alignment, increase abdominal muscle strength, or improve other impairments (Sahrmann 2002).

Another common low back impairment is termed extension syndrome. In this syndrome, the hip extensors are typically more active than the abdominal muscles, but the back extensor muscles are the most dominant. The abdominal muscles are not always weak, but there is a lack of control. Therefore, the patient must learn to effectively use the abdominal muscles to reduce excessive lumbar extension. Exercises used to correct extension syndrome most often aim to stiffen the abdominal muscles and stretch the hip flexors (Sahrmann 2002).

O'Sullivan (2000) used to the term 'extension pattern' to describe individuals who reported low back pain related to extension/rotation. This author found that these individuals stand in a more extended posture and often have increased muscle activity at the affected lumbar level. They also have limited co-contraction of deep abdominal muscles and have dominant activation of the erector spinae muscles. The individuals classified with extension syndrome often hold their lumbar spine in an extended posture, contributing to the pain.

Lumbar rotation syndrome is not common; rotation typically causes pain when associated with another movement such as extension. In this condition, pain usually arises with position changes. Often the spine rotates about only one or two segments instead of through the entire spine. Muscle patterns typically show dominant recruitment of the rectus abdominis and weak recruitment of the external oblique and contralateral internal oblique muscles that control rotation. A common treatment for lumbar rotation syndrome is identifying which daily activities involve lumbar rotation. The patient is then instructed on how to properly perform these activities in a way that reduces lumbar rotation. In addition, exercises are typically subscribed to improve the stability/stiffness, control and performance of the abdominal muscles (Sahrmann 2002).

Tall men are especially susceptible to lumbar rotation-flexion syndrome because they often sit in lumbar flexion. Rotation-flexion syndrome is caused by an individual rotating while in a flexed position, as would occur when tall men reach for something that requires rotation while sitting. Typically, abdominal muscles are more heavily recruited than the back extensors when standing in a sway back posture, but they have poor control of lumbopelvic rotation. Therefore, one of the primary objectives for treating rotation-flexion syndrome is to improve abdominal control. Other objectives include shortening and stiffening the back extensor muscles, and correcting the short and stiff muscles associated with rotation. As always, it is also important to correct daily activities that cause incorrect movement patterns (Sahrmann 2002).

A final lumbar impairment is flexion syndrome. People in this category often sit with lumbar spine flexion and the majority of their movements are in the spine instead of about the hips. According to Sahrmann (2002), when leaning forward while in a sitting position, the abdominal muscles typically have a more dominant recruitment than the hip flexors, but the abdominals may actually be weak. In addition, the hamstring the gluteal muscles may be short and stiff, and the back extensors may be long and weak. Exercises are often used to correct these muscle imbalances. However, the primary objective for individuals with flexion syndrome is to teach the patient to sit correctly and to move about the hips instead of the lumbar spine. McGill and colleagues have found that there are a finite number of bending cycles before the disc is sensitized, causing all motion to be painful. Reducing spine motion appears to address this concern (McGill 2007).

Individuals with pain associated with flexion/rotation can also be classified as patients with ‘flexion pattern’. These individuals feel pain when performing flexion movements and often cannot withstand flexed postures; however they tend to stand and sit with a lack of lumbar lordosis. When moving into forward bending, the individual typically segmentally flexes at the most unstable segment

and has the most flexion measured at this joint. Similar to the extension pattern, individuals tend to have difficulty co-contracting the deep muscles (O'Sullivan 2000).

Poor spinal stability is associated with spinal dysfunction and exercises are often prescribed to improve stability/stiffness. Kavcic, Grenier & McGill (2004b) examined eight commonly used stabilization exercises to determine their efficacy in stabilizing the spine. These exercises included the abdominal curl, right side bridge, sitting on a ball, kneeing with the left arm and right leg lifted, kneeling with the right leg lifted, a back bridge, a back bridge with right leg lift, and sitting on a chair. Each of the exercises was performed while using an abdominal bracing technique. From this work, it was found that sitting on a ball or a chair created the lowest spine stability levels, while kneeling with the contralateral arm and leg lifted resulted in the highest spine stability. However, the contralateral arm and leg lift exercise, the right side bridge and back bridge with the right leg lift exercises resulted in the highest L4-L5 compression values. The lowest compression values were found during kneeling with one leg lifted. Based on the results obtained for compression and stability for the exercises examined, the researchers created a graphical scale to aid clinicians in determining which exercises should be prescribed given the individual patient's needs regarding stability together with their particular tolerance for the associated compressive loading (Kavcic, Grenier & McGill 2004b).

Another method employed to increase spine stability/stiffness is to focus on multifidus and the deep abdominal muscles, especially transverse abdominis (TrA). The idea behind training TrA to increase stability/stiffness is primarily based on the observation that TrA has delayed activation in individuals with low back pain (Hodges and Richardson 1996), but also based on the continuous activation during trunk flexion and extension (Cresswell et al. 1992) and consistent activation of the TrA versus different activation of the abdominal and erector spinae muscles during various directions of shoulder movement (Hodges 1999). Abdominal hollowing is typically used to activate the TrA muscle, also called the abdominal drawing in maneuver. This technique involves co-contraction of the TrA and

multifidus muscles through drawing the lower abdominal wall up and inwards (Richardson and Jull 1995). One study found that using the abdominal hollowing technique resulted in a significant decrease in pain, while the pain for the control group did not change. In this case, the control group continued seeing their original medical professional and did not have a standardized treatment protocol (O'Sullivan et al. 1997). França et al. (2010) examined the effect on pain when using an TrA and multifidus strengthening protocol, through abdominal hollowing, and an abdominal wall strengthening protocol, through sit-ups, sit-ups with a twist, leg raise and the 'superman' exercise. These authors found a larger decrease in pain when using the TrA and multifidus protocol than the abdominal wall strengthening protocol.

2.5 Pain Scales

Pain is a difficult symptom to quantify. The visual analog scale (VAS) and the verbal numerical rating pain scale (NRS) are the most commonly used pain scales to quantify the amount of pain an individual feels. The VAS scale consists of a 100 mm line with one end labeled 'no pain' and the other end labeled 'worst pain imaginable'. The individual is instructed to make a vertical mark on the scale indicating their pain intensity, and then the distance is measured in mm to determine the level of pain (Jensen et al. 1986). This type of pain scale has been validated (Bijur et al. 2001; Gallagher et al. 2001; Kelly 2001) and it has been found that the minimum clinically significant differences ranges from 9 mm (Kelly, 1998) to 14 mm (Bijur, Latimer, & Gallagher, 2003).

The NRS scale is typically an 11-point (0 – 10) or 101-point (0 – 100) scale where 0 is defined as 'no pain' and the upper value is defined as 'worst pain imaginable'. With this scale, the individual is asked to verbally rate their pain intensity (Jensen et al. 1986). It is often assumed that the 101-point scale would be better than the 11-point scale due to the extra levels for individuals to choose from. However, (Jensen et al. 1994) found that 11-point and 21-point scales provide enough levels for chronic pain patients to describe their pain intensity.

Both the VAS and NRS scales have been found to be comparable, indicating that both types of scales would be appropriate to use to measure pain intensity (Bijur et al. 2003; Holdgate et al. 2003). One study found the minimum clinically significant difference for an 11-point NRS scale to be 1.39 (Kendrick & Strout 2005), while another found the minimum clinically significant difference to be 1.4 (Holdgate et al. 2003). Yet another study found the minimum clinically significant difference for an 11-point NRS scale to be 1.3 (Bijur et al. 2003). These authors also warned that a difference in the 11-point NRS scale of 2.0 should be interpreted with caution.

2.6 Reference Voluntary Contractions

To interpret and report muscle activity from a subject, some type of normalization is required. Typically researchers will ask the subject to perform a maximum voluntary contraction (MVC) for this purpose. The MVC is probably the best form of normalization since it allows physiologic interpretation and modelling of force output. This requires the subject to exert themselves to achieve the maximum possible muscle activity (Lehman & McGill 1999). However, for people in pain, using a MVC is not an appropriate method for EMG normalization because these individuals are often unwilling or unable to perform a maximal exertion (Marras & Davis 2001). A submaximal process is possible but requires several assumptions.

Attempts to predict MVC from a submaximal contraction suggests it is possible. Marras & Davis (2001) determined a regression equation to calculate the expected maximal contraction (EMC). They examined the erector spinae, latissimus dorsi, rectus abdominis, external obliques, and internal obliques bilaterally. This method required the subject to perform submaximal contractions, while the maximum torque was predicted using anthropometric measures. The submaximal exertions used were sagittal flexion, right and left lateral bend, clockwise and counterclockwise twist at 0° flexion, and sagittal extension at 20° flexion. The target moment exertions were 40, 60 and 80 Nm for flexion and extension, 30, 60 and 90 Nm for lateral flexion, 10, 20 and 30 Nm for twist, and one-third, one-half and two-thirds

the subjective maximal ability for each direction. Using the maximum and submaximal torque in conjunction with the EMG activity, the maximum possible muscle activity was predicted using a linear relationship.

Marras et al. (2001) attempted to validate this normalization method by comparing predicted spine loads using MVC for normalization and using the EMC technique. Subjects performed exertions necessary for both the MVC and EMC normalization techniques and then performed a number of lift tasks. Data was input into an EMG-assisted biomechanical model to determine the effect of the normalization technique on spinal loads. It was found that both normalization techniques resulted in approximately the same spinal loads, despite lower muscle activities and higher muscle gain for the EMC technique. The trends seen for the muscle activities were the same for both the EMC and MVC normalization methods. The authors concluded that the EMC procedure is an appropriate way to normalize EMG in individuals with low back pain.

Oddsson et al. (1997) also used a regression analysis on anthropometric measurements to estimate the MVC. The MVC for back extension was measured for a group of 17 male subjects with similar anthropometry. A regression analysis of twelve anthropometric circumferences showed that shoulder, hip and thigh circumference were the three best predictors of MVC. A high correlation between estimated and predicted measures was found for this homogenous population.

More recently, Cholewicki et al. (2011) described a gain method for normalizing trunk EMG. These authors used sub-maximal ramp exertions in trunk flexion, extension, left lateral bending and right lateral bending to drive the calculation of a muscle gain factor. The gain factor was calculated using an optimization approach, matching the three-dimensional external joint moments and the corresponding muscle moment calculated by a biomechanical model. An individual gain was calculated for each EMG electrode site. The authors determined that the gain normalization method resulted in an estimate of absolute muscle force while the more common MVC normalization method gives a relative measure of

muscle effort. It was concluded that the MVC method would be more appropriate if the researchers were interested in muscle recruitment patterns, while the gain method would be more appropriate if differences between individuals were of more interest (Cholewicki et al. 2011).

2.7 Modeling Approach

One must be able to estimate compressive and shear loads on the spine in order to examine the functionality of the low back. This requires knowledge of the tissue loads and forces. Early spine models developed for tissue load estimation were typically static models in the sagittal plane and used simplistic anatomy, assuming the erector spinae muscles work through a 5 cm moment arm (Bejjani et al. 1984; Chaffin 1969; Schultz & Andersson 1981). In the following years, spine models evolved to include inertial components, but still assumed a 5 cm moment arm for the erector spinae muscles (McGill & Norman 1985; Anderson et al. 1985). These models often predicted compression loads that exceeded the maximum tolerance levels at that time of 6000 N (McGill & Norman 1986) with no injury to the individual. With the improvement of computational ability of computers, three-dimensional dynamic models were developed (Marras & Sommerich 1991a; Marras & Sommerich 1991b; McGill & Norman 1987), which included a more detailed anatomical representation of the lumbar region.

The model developed by Marras & Sommerich (1991a) required the EMG activity of five bilateral muscles, trunk torque, trunk flexion angle and trunk angular velocity as input to the model. These variables were used to calculate lumbar spine compression, shear, torsional forces and trunk torque production throughout a dynamic movement. This model only included the L5 level, assuming the weight of the upper body above this level as a whole. This model accounts for individual muscle activity differences and allows for calculation of peak loading, giving an indication of the loading imposed on the lumbar spine at any given point in time during an exertion. However, this model was designed for laboratory use, primarily for lifting tasks. This implies that this model would not be appropriate for any other type of dynamic task.

The spine model developed by Professor McGill and his graduate student colleagues included a much more detailed representation of the lumbar structures in an attempt to reduce the predicted compression loads to a more reasonable level. This model includes a rigid ribcage, pelvis/sacrum, and five lumbar vertebrae. Each of the vertebrae are separated by a mathematically represented lumped parameter disc that includes rotational stiffness about three axes. It also includes over 100 muscle fascicles representing the various lines of action of the torso muscles (Kavcic, Grenier & McGill 2004a). The intra-abdominal pressure is modelled as a compression reducing mechanism, using an adapted version of the Chaffin (1969) equation (McGill & Norman 1986).

In the past, models have typically used either an EMG approach or an optimization approach to estimate the forces of each individual muscle fascicle. The optimization approach allows moment constraints to be satisfied in all axes, while the EMG approach predicts forces according to the activation patterns seen from EMG signals. When these two approaches were compared using the model described by McGill & Norman (1986), it was found that using the optimization approach resulted in the same predicted forces for each person, regardless of individual recruitment pattern differences, while the EMG approach did not always satisfy the moment constraints (Cholewicki et al. 1995).

The spine model developed by Professor McGill and colleagues uses a hybrid approach, termed the EMG-assisted optimization approach, to estimate the forces of each individual muscle fascicle. The combined approach offers the benefit of predicting muscle forces similar to those seen from a pure EMG approach, while also ensuring moment balance, effectively combining the EMG approach and the optimization approach (Cholewicki & McGill 1994). The EMG-assisted optimization approach initially uses EMG signals to predict forces, and then an objective function is used to balance the three moment constraints by applying the smallest possible adjustment to the individual muscle forces (Cholewicki & McGill 1994). Although this approach is good for surface muscles where EMG is easy to obtain, some assumptions need to be made regarding deeper muscles, such as which muscles can be assumed to be

functionally equivalent. These deep muscles from which EMG cannot be obtained are assumed to have the same EMG profile as another functionally equivalent muscle (Cholewicki & McGill 1994).

In the spine model developed by Professor McGill and colleagues, the upper erector spinae electrode site, located approximately 5 cm lateral to T9, was used to drive the longissimus thoracis (Long) and iliocostalis lumborum (Ilio) muscles. The lower erector spinae electrode site, located approximately 3 cm lateral to L3, was used to drive the multifidus (Mult), pars lumborum (Pars) and quadratus lumborum (QL) muscles in the model (McGill & Norman 1986). The internal oblique electrode site was used to drive the psoas muscles, based on the assumption it is a spinal stabilizer (Nachemson 1968). Finally, the internal oblique electrode site was also used to drive the transverse abdominis activity because the two muscles have been shown to have synergistic activity (Cresswell 1993). These assumptions were based on an indwelling EMG study that found there was little error between surface electrode sites and the deep muscles, indicating that surface electrodes are sufficient to measure the muscle activation of certain deep muscles (McGill et al. 1996).

2.8 Stability Calculation

In addition to lumbar loads and individual muscle force and stiffness, lumbar spine stability during various activities is commonly discussed. One of the early attempts at quantifying stability was by Professor Anders Bergmark (1989). Bergmark used a potential energy approach with joint stiffness and 40 muscles to mathematically calculate energy minima, stiffness, stability and instability (Bergmark 1989). The potential energy approach is most commonly described by using the analogy of a ball rolling on a surface (Bergmark 1989; Howarth et al. 2004; McGill 2007). If the ball is in a bowl shaped object, it is considered stable because if the ball is slightly perturbed, it will come to rest at the lowest point, or the point of lowest potential energy. If the sides of the bowl are steeper, the system is more stable since a larger perturbation could be applied and the ball would still return to the lowest point. These situations indicate that the energy required for the perturbation is always smaller than the inherent potential energy

of the system. The system becomes unstable if the energy for the perturbation exceeds the energy of the system, or if the perturbation is large enough to cause the ball to roll out of the bowl. In the case of the spinal system, the slopes of the bowl sides represent the joint stiffness, and the width at the bottom of the bowl represents the joint laxity (McGill 2007).

Quantifying stability using the idea of elastic potential energy is also used in the model developed by Professor McGill and colleagues. The potential energy of the system (V) is given by:

$$V = U_L + U_T - W$$

where U_L and U_T are the elastic energy stored in the linear and torsional springs, respectively, and W is work performed on the external load (Cholewicki & McGill 1996). Here, the linear springs represent muscles and tendons, and the torsional springs represent passive tissues, such as the intervertebral discs and ligaments. The second partial derivatives of V are calculated for each joint and axis combination and arranged into an 18 x 18 Hessian matrix.

There are many ways to calculate the stability index from the Hessian matrix, as described by Howarth et al. (2004). One way is to calculate the determinant of the Hessian matrix is by manipulating the Hessian matrix to the reduced row echelon form to create an upper triangular matrix. The product of the elements on the diagonal, or the pivot elements, is the determinant of the original Hessian matrix. The system is considered stable if the determinant is positive and unstable if the determinant is negative (Cholewicki & McGill 1996; Howarth et al. 2004). This method can result in a falsely stable spinal column if there is an even number of negative pivot elements, thus the determinant must be examined in conjunction with the pivot elements. The false positive issue has been addressed by declaring the system unstable if there are one or more negative pivot elements. The determinant of the Hessian matrix method gives a measure of the global spinal stability (Cholewicki & McGill 1996; Howarth et al. 2004).

A second way of calculating the stability index is to diagonalize the Hessian matrix to find the 18 EVs, using the lowest EV as the stability index. These EVs represent the degree of curvature at a critical

point of the potential energy surface. The joint/axis combination is less stable as the EV decreases. This method offers a measure of local spinal stability, as the spine is most likely to buckle at the joint/axis combination that has the lowest EV (Howarth et al. 2004).

Both methods show the same trends for indications of stable vs. unstable spines, but the magnitudes of the stability index are different (Howarth et al. 2004). In addition, the determinant method appears more sensitive to muscle activation changes than the smallest EV method. This suggests that the best approach for interpreting stability would be to examine the determinant and lowest EV methods together (Howarth et al. 2004).

2.9 Summary

Patients have variable presentations and different motions, postures and external loads that cause pain. However, there are categories of pain provocation and intolerance, such as flexion bending intolerance, that sub-classify patients to guide corrective interventions by these functional classifications. This implies that an assessment is required to determine the exacerbating variable. Treatment involved modifying movement patterns to eliminate those which exacerbate pain and enhance those which are tolerable and address the painful movement flaws.

Chapter 3
Methods

This study was composed of two phases. The first involved conducting a sensitivity analysis of the stability portion using an anatomically detailed spine model to address the first five hypotheses. The sensitivity analysis was performed in two parts: 1) using a theoretical set of data and 2) using an actual set of data. The second phase applied the knowledge gained from the sensitivity analysis to assess the "proof of principle" via testing patients referred to Professor McGill with low back pain to address hypothesis six and seven.

This methods section is organized to first introduce the subjects used for each phase, then followed with a description of the common methodology of both phase 1 and phase 2, and finally the specific details of the protocol employed for both phases.

3.1 Subjects

3.1.1 Phase 1 (Sensitivity Analysis): Subjects

Part 1 of the sensitivity analysis was performed using a theoretical set of data; therefore no subjects were used. For part 2 of phase 1, four healthy male subjects (average ± SD: age 27 ± 3.65 years, height 1.75 ± 0.06 m, weight 85.5 ± 13.0 kg) with no history of back pain volunteered to participate in the study. Participants were given a brief verbal explanation of the task, preparation and equipment being used. Once comfortable with this information, they read and signed the informed consent approved by the University of Waterloo Research of Ethics Board. Before testing, the participants' height, weight, chest depth, pelvis depth and trochanter width were measured.

3.1.2 Phase 2 (Case Studies): Subjects

For the case studies portion of the thesis, individuals with low back pain referred to Professor McGill from which subjects were selected who reported "catches" of pain. Specifically, subjects were

included if they responded “yes” to the following three questions: 1) Do you have pain rolling in bed? 2) Do you have good and bad days in terms of pain? 3) Do you have pain or “catches” when you are in the mid-range of motion? Four subjects were deemed appropriate for the study.

Subject 1 was a male aged 22 years, height 1.63 m and weight 81.5 kg. He was a competitive power lifter and had pain when he performed an arched bench press. His pain was exacerbated by squatting with a load, multiple bench presses or sitting for prolonged periods of time. The most pain was felt at the L5/S1 level. These reports and provocative tests performed in a clinical assessment prior to the collection period led to the conclusion that this subject was likely compression intolerant.

Subject 2 was a male aged 27 years, height 1.83 m and weight 97 kg. This subject reported that pain was exacerbated by sitting or standing for extended periods of time. It was also noted that the subject had noticeable spine flexion when sitting. The most pain for this subject was felt at the L4 level. These reports and provocative tests performed in a clinical assessment prior to the collection period led to the conclusion that this subject was flexion intolerant.

Subject 3 was a female aged 31 years, height 1.85 m and weight 65.8 kg. She was an Olympic level volleyball player who reported exacerbated pain during serves and spikes, associated with extension and axial rotation of the torso. The most pain for this subject was felt at the L4 and L5 levels. It was concluded that this subject was extension intolerant.

Subject 4 was a female aged 54 years, height 1.63 m and weight 81.6 kg. This subject had experienced a cervical trauma and also had a disc herniation at T7/T8. In addition, her pain had a fibromyalgic overlay. She reported that sitting slouched caused pain. Through provocative testing it was also found that there was an apex of instability at T12 and at L5, but there was some degree of instability throughout the lumbar spine. Through these results and other provocative tests it was concluded that this subject could be classified in the instability category.

In all situations the participant was given a brief verbal explanation of the preparation and equipment being used. Once comfortable with this information, they read and signed the informed consent approved by the University of Waterloo Research of Ethics Board. Before testing, the participants' height, weight, chest depth, pelvis depth and trochanter width were measured.

3.2 Instrumentation

The following instrumentation was used for both phase 1 part 2 and phase 2.

3.2.1 Kinematics

Full body kinematics were recorded using the VICON motion tracking system (Vicon Motion Systems, Oxford, UK) at a sample rate of 60 Hz. Sixteen individual, 10 mm diameter reflective markers were adhered to the skin using hypoallergenic tape over the following landmarks: bilateral medial malleolus, bilateral lateral malleolous, bilateral calcaneous, bilateral medial femoral condyle, bilateral lateral femoral condyle, bilateral greater trochanter, bilateral iliac crest and bilateral acromion. Eight rigid bodies moulded from splinting materials were also adhered to the skin with hypoallergenic tape over the upper back around T12, sacrum, each thigh, each shank, and each foot. Each rigid body had four 10 mm diameter reflective markers attached with tape. Eight VICON MX20 cameras tracked the three-dimensional location of the reflective markers. A calibration trial was collected to create an individual anatomical model so that it was only necessary to track the eight rigid body marker clusters for the remainder of the trials.

3.2.2 Force Plate

Force plate data was collected using four AMTI force plates. The signals were amplified to a range of 20 V (± 10 V) and A/D converted using a 16-bit, 64 channel A/D converter at a sample rate of 2160 Hz. This data was also collected using VICON Nexus software.

3.2.3 Electromyographic Activity

EMG was recorded using Ag-Ag/Cl (Meditrace™ 130 Ag/AgCl electrodes, Covidien, MA, USA) self-adhesive surface electrode pairs, spaced approximately 25 mm apart in a bipolar configuration. Care was taken to ensure the electrodes were aligned parallel to the muscle fibre direction. Before the electrodes were adhered to the skin, the skin was shaved and cleansed with Nuprep abrasive skin prepping gel. The activity of six muscles on each side of the body, for a total of twelve muscles, was recorded. These muscles included: 1) rectus abdominis (RA), 2 cm lateral to the umbilicus, 2) internal oblique (IO), caudal to the anterior superior iliac spine and medial to the inguinal ligament, 3) external oblique (EO), 15 cm lateral to the umbilicus, 4) latissimus dorsi (LD) over the muscle belly, 15 cm lateral to T9, 5) thoracic, or upper erector spinae (UES), 5 cm lateral to T9 over the muscle belly and 6) lumbar, or lower erector spinae (LES), 3 cm lateral to L3 (Grenier & McGill 2007). EMG signals were amplified using a Bortec amplifier (Bortec Biomedical, Calgary, AB, Canada) and A/D converted using a 16-bit, 64 channel A/D converter at a sample rate of 2160 Hz. This data was collected using VICON Nexus software.

Two resting trials were collected, one while lying on the stomach and one while lying on the back with the limbs in a self-selected position for comfort and relaxation. For the patient population, reference voluntary contractions (RVC) were performed for normalization (figure 1). This involved the patient holding a weight in two hands directly in front of the body. The shoulder and elbow angles were positioned at approximately 90°, but these angles were not controlled. The weight held was dependent on the ability of the patient.

Figure 1 - Reference voluntary contraction (RVC) involving holding a weight with shoulder and elbow angles of 90°. The weight held was dependent on the patient's ability.

For the healthy population (phase 1 part 2), an MVC for each muscle was performed for normalization. For the abdominal muscles (RA, EO, and IO), each participant adopted a sit up posture at approximately 45 degrees of hip flexion and was manually braced by a research assistant. The participant was instructed to produce a maximal isometric flexor moment followed sequentially by a right and left lateral bend moment and a right and left twist moment. For the spine extensors (LES and UES) and LD muscles, a resisted maximum extension in the Biering-Sorensen position was performed for normalization (Biering-Sørensen 1984). The LD muscles were cued by instructing the participants to pull their shoulder blades back and down during extension. These contractions were performed according to established lab protocol (Grenier & McGill 2007).

3.3 Data Processing

Following data collection, EMG data was band pass filtered to leave a signal between 30 and 500 Hz, full-wave rectified and low-pass filtered with a single-pass second order Butterworth filter at a cut-off frequency of 2.5 Hz, as this level mimics the frequency response of torso muscles (Brereton and McGill 1998). A filter of 30 to 500 Hz was chosen to maintain the biological signal while removing the electrocardiographic signal (Drake and Callaghan 2006). The zero bias from the resting trial was removed from all trials to account for bias. Finally, all trials were normalized to the maximal EMG amplitudes obtained during the RVC or MVC procedure and the signals were down sampled to 60 Hz to allow for syncing of the EMG and kinematic data. This was completed using custom LabView software (National Instruments Corporation, Austin, TX, USA).

The remaining kinematic and kinetic data was processed using an established model in Visual 3D (C-Motion Inc., Rockville, MD, USA) to obtain joint forces and moments. Kinematic data was filtered using a low-pass second order dual-pass Butterworth filter at a cut-off frequency of 6 Hz (Winter 2009). Force plate data was filtered using a low-pass second order dual-pass Butterworth filter at a cut-off frequency of 15 Hz, since 99% of the signal power for gait is seen below this level (Antonsson & Mann 1985). A segmental model was created, which included a pelvis, torso, right and left thigh, right and left shank, and right and left foot. These segments were based on joint centres, as calculated from the markers placed on the anatomical landmarks. Each segment was then tracked by the marker cluster placed on that segment. For example, the right thigh segment was tracked by the marker cluster on the right thigh, the pelvis segment was tracked by the marker cluster on the pelvis, etc.

The segmental model was used to calculate time-varying orthopedic spine angles about the L4-L5 joint. To calculate these angles, a second ‘virtual’ pelvis was created. This virtual pelvis was created using the same anatomical landmarks as the original pelvis, but it was tracked using the torso marker cluster instead of the pelvis marker cluster. The lumbar spine angles were calculated using an x-y-z, or

flexion/extension-lateral bend-axial twist, rotation sequence for the virtual pelvis segment with the reference segment as the original pelvis segment, allowing for calculation of the angle between the torso marker cluster and pelvis marker cluster.

Time-varying three-dimensional reaction forces and moments about the pelvis joint were calculated using a rigid linked-segment model. This was completed using a bottom-up approach using the ground reaction forces and moments measured by the force plate. The forces and moments calculated at the pelvis joint were assumed to equal those at the L4-L5 joint.

3.4 Spine Stability/Stiffness Calculation

Spine stability/stiffness was quantified using an anatomically detailed spine model, as described elsewhere (McGill & Norman 1987; McGill 1992; Cholewicki & McGill 1996). The model uses Visual Basic (Microsoft Corp., USA) and MATLAB (The MathWorks Inc., USA). A short description of the model is provided here, with a flow chart of the steps shown in figure 2.

This spine model requires muscle activity from seven bilateral muscles as input, including: (1) RA, (2) IO, (3) EO, (4) LD, (5) UES, (6) LES and (7) Mult. When using collected EMG activity, it is assumed that the LES and Mult muscles have the same activation profiles due to limitations in the ability to accurately collect a separate Mult muscle activation profile. The spine model also requires lumbar spine angles in three degrees of freedom (DOF): (1) flexion/extension, (2) lateral bend and (3) axial twist. These spine angles are calculated using an established model in Visual3D as described previously. The EMG-assisted optimization portion of the spine model, described in detail later in this section, uses the L4/L5 reaction forces and moments, calculated by the linked-segment model in Visual3D described previously.

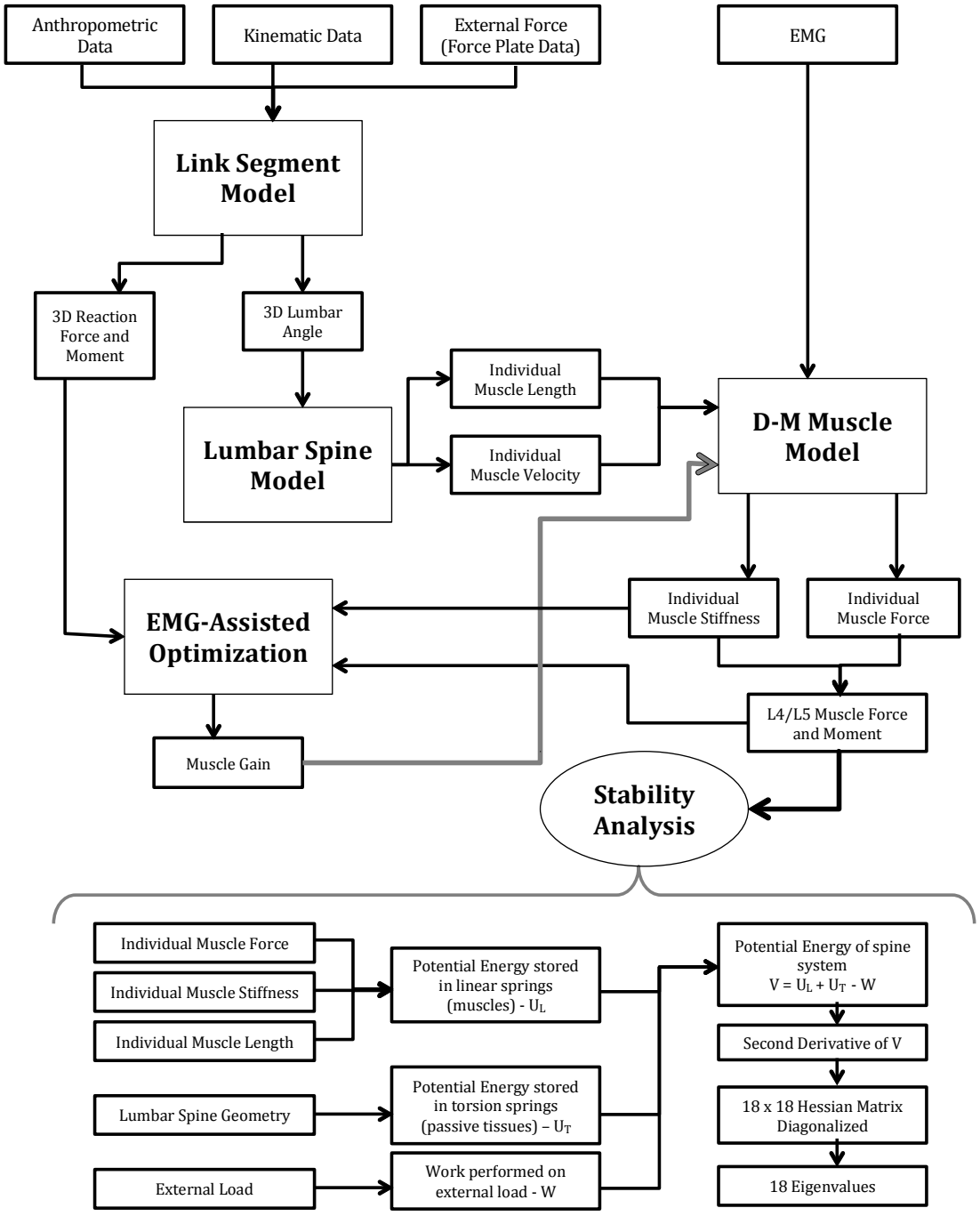

Figure 2 - Flow chart of the anatomically detailed spine model and steps required leading up to the stability analysis. Abbreviations: EMG - Electromyography, D-M - Distribution-moment

This model consists of two interdependent models: (1) a 'lumbar spine model', and (2) a 'distribution-moment muscle model' (D-M muscle model). The lumbar spine model describes the 3-dimensional anatomy of the lumbar spine. It consists of five lumbar vertebrae between a rigid pelvis/sacrum and a rigid ribcage. The vertebral discs connecting the vertebrae are modeled using torsional springs, while torso muscles and tendons are modeled with linear springs. Eleven muscles are divided into 59 muscle fascicles on each side, for a total of 118 muscle fascicles. This model uses the flexion/extension, lateral bend, and axial twist angles as input to calculate muscle lengths and velocities. The D-M muscle model then uses these muscle lengths and velocities as well as normalized EMG to calculate individual muscle force and stiffness profiles. These values are used to calculate the L4/L5 muscle forces and moments.

A separate EMG-assisted optimization routine executed in LabView was used to balance the L4/L5 reaction forces and moments, as calculated previously using the linked-segment model in Visual3D, and the L4/L5 muscle forces and moments, as calculated by the anatomically detailed spine model. The objective function for the optimization is to match the two moments with as little change to the EMG-driven stiffness profiles as possible using least squares difference, similar to that used by (Cholewicki & McGill 1994). The factor by which muscle forces are changed is called the "muscle gain". This gain factor is used to calibrate the model, which is based off a static 50^{th} percentile male, to a fit a broader range of individuals.

The EVs were evaluated as the measure of stability/stiffness at each of the 18 DOF (6 lumbar joints and 3 rotational axes). These were calculated by using the potential energy at each of the 18 degrees of freedom. The potential energy of the linear springs, or muscles (U_L), was calculated using the individual muscle force, stiffness and lengths, while the potential energy of the torsion springs, or passive tissues (U_T), was calculated using the lumbar spine geometry included in the spine model. These potential energies and the work performed on the external load (W) were used to calculate a total

potential energy of the spine system ($V = U_L + U_T - W$). The second derivative of V was arranged into an 18 x 18 Hessian matrix that was symmetrical about the main diagonal. The Hessian matrix was then diagonalized to determine the associated 18 EVs. These EVs were used as the measure of stability/stiffness at each of the lumbar joints in each degree of freedom. Gardner-Morse et al. (2006) have argued that the lowest EV forms the indicator of stability. However, it is not known if all EVs are comparable in terms of scale. Further mathematical detail on the EV calculation can be found in Howarth et al. (2004) for the interested reader.

3.5 Phase 1: Sensitivity Analysis

The stability portion of the spine model was evaluated using a muscle knockout approach. This was completed in two parts, first using theoretical data and then using actual data from healthy subjects.

For the theoretical data portion of the sensitivity analysis, muscle activity was set to always equal an arbitrary value of 50% MVC. This value was chosen so that a large change would be seen both when reducing the muscle activation to 0% MVC and increasing it to 100% MVC. A separate trial was then created for each lumbar spine posture. Each trial altered one lumbar spine angle DOF. The flexion axis ranged from -30° to 50°, the lateral bend axis ranged from -30° to 30°, and the axial twist axis ranged from -40° to 40°, all in increments of 10°, for a total of 23 postures (listed in table 1). These angles were chosen to represent the approximate full lumbar range of motion, as measured from subject 4 of phase 2. For example, trial 1 was a neutral lumbar spine posture with 0° flexion, 0° bend, and 0° twist. Trial 2 had 10° flexion, 0° bend, 0° twist, etc. The lumbar spine posture was then bent through the lateral bend axis, with 0° flexion, 10° bend, 0° twist, etc. Finally, the lumbar spine angles were set to 0° flexion, 0° bend, and 10° twist, etc. to account for twisting motions. These variable postures were used to test whether muscles affect the plane of stability/stiffness and whether muscles and EVs were affected by posture (hypotheses two and three).

Table 1 - Postures tested for the sensitivity analysis. Positive flexion/extension represents extension, positive lateral bend represents right bend and positive axial twist represents left twist

Posture	Flexion/Extension (°)	Lateral Bend (°)	Axial Twist (°)
Neutral Posture	0	0	0
10 Flexion	-10	0	0
20 Flexion	-20	0	0
30 Flexion	-30	0	0
40 Flexion	-40	0	0
50 Flexion	-50	0	0
10 Extension	10	0	0
20 Extension	20	0	0
30 Extension	30	0	0
10 Right Bend	0	10	0
20 Right Bend	0	20	0
30 Right Bend	0	30	0
10 Left Bend	0	-10	0
20 Left Bend	0	-20	0
30 Left Bend	0	-30	0
10 Left Twist	0	0	10
20 Left Twist	0	0	20
30 Left Twist	0	0	30
40 Left Twist	0	0	40
10 Right Twist	0	0	-10
20 Right Twist	0	0	-20
30 Right Twist	0	0	-30
40 Right Twist	0	0	-40

For the actual data portion (hypothesis five), data from a previous study performed by McGill and colleagues, consisting of a walking task, was used (unpublished data). In this collection, four healthy male participants performed a walking task while carrying a bucket with 15 kg in each hand. The only instructions the participants were given were to ensure their left foot made contact with the force plate. The participants performed two trials of the walking condition and the most complete trial was analyzed. Each trial was cut to include from right foot toe off to just prior to right foot contact, while the left foot was in contact with the force plate, visually determined by two separate examiners.

Both sets of data were input into the spine model with all 118 muscle fascicles activated to obtain time-varying EVs. Individual muscle activities were then systematically reduced to 0% MVC, one muscle at a time, in subsequent runs of the model. Twelve scenarios were executed to determine the effect of individual muscles on individual EVs (hypothesis one). These scenarios included: 1) All muscles active, 2) bilateral RA removed, 3) bilateral EO removed, 4) bilateral IO removed, 5) bilateral Pars removed, 6) bilateral Ilio removed, 7) bilateral Long removed, 8) bilateral QL removed, 9) bilateral LD removed, 10) bilateral Mult removed, 11) bilateral Psoas removed, and 12) bilateral TrA removed. These twelve situations were repeated, except the affected muscle was artificially activated to 100% MVC to determine the effect of muscle overactivation (hypothesis four).

The mean of each EV was calculated for each trial. For the theoretical part, the percent difference was calculated between individual EVs with the altered muscle activation and when all muscles were at 50% MVC (hypothesis 1 and hypothesis 4). These same percent differences were used for hypothesis 2, but the flexion, bend and twist axes were compared at each lumbar level. For hypothesis 3, percent difference was calculated between the neutral posture and the posture of interest.

For part 2, the actual data set (hypothesis five), the mean of each EV was calculated for each trial while the left foot was in contact with the force plate. Using SAS (SAS Institute, Cary, NC), a two factor repeated measures ANOVA was conducted on the EV with factors EV level and activation level. This

was repeated for each muscle. The effect of activation level was of primary interest. For each muscle, pairwise comparisons were used for the interaction effect to determine if there were significant differences ($p < 0.05$) between activation levels at each EV level.

3.6 Phase 2: Case Studies

For this final portion of the thesis, comprising of evaluating the "proof of principle", each subject was asked to perform different tests and activities as deemed appropriate for their pain presentation. In all situations, subjects were asked to perform tasks using the motion, posture or load technique that increased their low back pain, while EMG and kinematic data were simultaneously collected. Professor McGill then suggested a clinical intervention to alter motion and muscle activation patterns in attempt to immediately reduce or remove the pain. These techniques included bracing the abdominal wall and/or LD muscle, and using a hip hinge technique. When asked to use an abdominal brace, the subjects were asked to stiffen their abdominal muscles by "hardening" their abdominal wall out laterally, cued by Professor McGill's hands, without extending the stomach. For the LD intervention, subjects were instructed to stiffen the shoulders by depressing the scapulae by activating the pectoralis and LD muscles in a co-contracted state. The hip hinge movement technique, based on the principle of proximal stiffness and distal mobility, involved flexing through the hips instead of flexing the spine when performing tasks such as sit-to-stand or squat. This was coached using the short-stop squat technique (McGill 2007). Once the subject understood the new technique, the task was performed with this technique while EMG and kinematic data were collected.

Subjects were asked to rate the severity of their pain using integer values on an 11-point (0 – 10) verbal scale after each trial. This scale was chosen for ease of use during data collection, as it has been found that using a larger 101-point scale does not give any extra information than an 11-point scale, thus the 11-point scale is sufficient (Jensen et al. 1994). These data were used to determine the efficacy of each intervention for pain reduction. A change of 2 points in the pain scale was considered clinically

significant (Bijur et al. 2003). This allowed testing of hypothesis six, that coaching movement patterns would alter low back pain. The following is a description of the tasks and interventions used for each subject.

Subject 1 only performed a heel drop task. The heel drop test is a compression test to determine if pain occurs during compression with an upright and neutral spine. During this test, the individual is asked to rise onto the balls of their feet then drop to their flat foot. This causes a rapid compressive load on the spine up to approximately 2.5 times body weight (McGill 2007). For this subject, due to technical problems with the kinematic and force plate data, spine angles were assumed to be 0° flexion, 0° bend and 0° twist. When input into the spine model, it was assumed that the muscle gain was 1. This subject performed the task three times: 1) when instructed to have a "loose", or unbraced, core, 2) using an abdominal brace, and 3) using his LD muscles.

Subject 2 performed three tasks. These tasks included the heel drop test, as described previously, an unloaded squat and lifting a 45 lb bar from a height of 45 cm. For the heel drop test, the subject used 5 different strategies. These strategies were: 1) unbraced, 2) mild abdominal brace, 3) robust abdominal brace, 4) pull down with LD muscles, and 5) abdominal brace with the LD muscles. For the squat task, the subject used his own self-selected pattern (unbraced) and also with the LD intervention. Finally, for the lift bar task, the subject used his self-selected pattern (unbraced) and a hip hinge plus LD muscle intervention.

Subject 3 performed four tasks, including 1) heel drop test, 2) unloaded squat, 3) jump from a stool, and 4) one-step approach spike. For the heel drop test, the individual performed the task using an unbraced abdominal core and using an abdominal brace. She performed the squat task unbraced with a slouch and using a hip hinge plus LD activation intervention. Due to the subject reporting pain when landing after performing a volleyball spike or serve, the subject was also asked to jump from a stool and simulate a one-step approach spike. When the individual jumped from the stool, she did so 1) using a

self-selected pattern (unbraced), 2) with an abdominal brace and 3) pulling down with her LD muscles. For the spike, the subject performed the task using her self-selected pattern (unbraced) and using an abdominal brace.

Subject 4 performed three tasks: 1) sit-to-stand, 2) stand-to-sit, and 3) unloaded squat. For both the sit-to-stand and stand-to-sit tasks, the subject used the same three patterns. These patterns included 1) self-selected (unbraced), 2) abdominal brace with a hip hinge, and 3) abdominal brace with a hip hinge and the spread the floor technique (referred to as the spread floor intervention). When asked to spread the floor, the subject was asked to try to spread the floor apart while gripping her feet to the floor such that her feet did not move. Finally, for the squat task, the subject used a self-selected pattern (unbraced) and used a hip hinge intervention.

All data was input into the anatomically detailed spine model. Due to patients being unable to perform an MVC, making it necessary to use an RVC, the optimization portion of the spine model was not appropriate to use to calculate the muscle gain. Therefore, muscle gain was calculated using the RVC trial. The muscle gain is important because it matches the predicted moment from the spine model with the measured reaction moment so that the spine model is "tuned" and calibrated to the individual. For the patient population, the reaction flexion moment, as calculated from Visual 3D (described previously), was divided by the muscle flexion moment, as calculated from the spine model. The flexion moment was used because this is the axis that RVC was performed in. The muscle gain obtained from this calculation was averaged over the trial portion where the subject was holding the weight. This muscle gain value was manually input into the spine model and used for all subsequent runs of the model.

Fifteen EVs (not including those at the rib level), EMG amplitudes, spine compression, and spine shear were "rubberbanded" to normalize the task to 0 – 100% of the task with endpoints as distinctive points in the trial (i.e. time was expanded and compressed to 100% of the task). The endpoints used are

shown in table 2. These points were all determine visually while viewing the task in Visual 3D with the skeleton model applied.

Rubber banded time histories of the analyzed variables for before and after the interventions were plotted on separate graphs. For example, EMG amplitude for the right RA muscle was plotted on a separate graph as the EMG amplitude for the right EO muscle, which was on a separate graph as the L4F EV, etc. These graphs had the patterns obtained from all interventions on one plot, so that for example, for the heel drop task for subject 1, the unbraced, braced and LD interventions were all plotted on the same graph. These time histories were used to visually identify what variables appeared to change between movement and muscle activation strategies over the entire task.

Table 2 - Distinctive points for all tasks used for "rubberbanding" trials.

Task	**0% Movement**	**100% Movement**	**200% Movement**
Heel Drop	Begin rising from flatfoot	Top of the motion, fully on the toes	Heel impact
Squat	Begin descent	At bottom of movement	Upright standing position
Lifting	Begin descent	At bottom of movement	Upright standing position
Jump	Both feet not in contact with stool	Impact	
Spike	Right foot toe-off	Both feet not in contact with ground	Impact
Sit-to-Stand	First visual of hip and/or back movement	Upright standing	
Stand-to-Sit	Movement began to sit	Seated position	

The mean of each variable was calculated between the endpoints described in table 2. The percent difference between the original movement and each of the interventions was used to determine if there was a change in each of the variables between the original pain level and the pain reported with the intervention. The EV specifically allowed for testing of hypothesis seven, that changes in pain would be reflected in the EVs. Further insight was gained through analysis of the other variables calculated (lumbar compression and shear loads, EMG activation and lumbar angles).

Chapter 4
Results

This results section is organized to first report the results obtained from the sensitivity analysis as they relate to the first five hypotheses. This is followed by the results obtained from each of the four subjects involved in the case studies.

4.1 Phase 1: Sensitivity Analysis using Theoretical Data

The first objective was to assess the role of individual muscles on single EVs. Simulations were performed and sensitivity analysis conducted through a muscle knockout approach. The EVs representing the rib/L1 joint were not analyzed due to the lack of anatomical detail at this level. For the remaining five joints, the effect of a muscle activation level on an EV was assumed to be biologically significant, or important, when there was a 10% change or greater in an EV between when all muscles were present in the model versus when a certain muscle was adjusted. The value of 10% was arbitrary but based on the notion of biological significance, as a change smaller than 10% would probably not be clinically interpretable. If the percent change in EV was negative, it indicated that adjusting the muscle activity resulted in a decreased EV, and vice versa. Altering muscle activation bilaterally resulted in the same effect on EVs in the right and left bend postures as well as the right and left twist postures. In other words, symmetric muscle intervention resulted in symmetric EV change. For this reason, only the degree of bend and twist, not the direction, was examined. The TrA muscle was also found to never result in a biologically significant change, therefore it was not discussed here. Also notable is the fact that the greatest anatomical detail is at the L4/L5 level such that results at this level are probably the most robust.

4.1.1 Hypothesis 1: Effect of Muscles on Eigenvalues

Although posture had interactions with muscles in their effect on EVs, it was clear that single muscles did affect multiple levels of spine stability/stiffness and stability/stiffness at one segmental level

was affected by multiple muscles. Figures showing the effect of single muscles on individual EVs for all postures except neutral can be found in Appendix A.

For the L1F EV (neutral posture in figure 3), the Mult muscle had the largest effect followed by the Pars, Ilio and Long muscles in their contribution. On average across all postures, when removed, the Mult muscle resulted in a 67.3% ± 6.5% (mean ± SD) change, the Pars muscle resulted in a 53.2% ± 8.5% change, the Ilio muscle resulted in a 31.7% ± 6.8% change and the Long muscle resulted in a 24.0% ± 6.2% change for the L1F EV (average absolute values in table 3). The Psoas muscle also had a biologically significant change in the L1F EV in the end range postures (50° flexion, 30° extension and 30° bend), as well as in 10° and 20° flexion, with less than 20% change except in the 50º flexion posture where there was a 33.2% change.

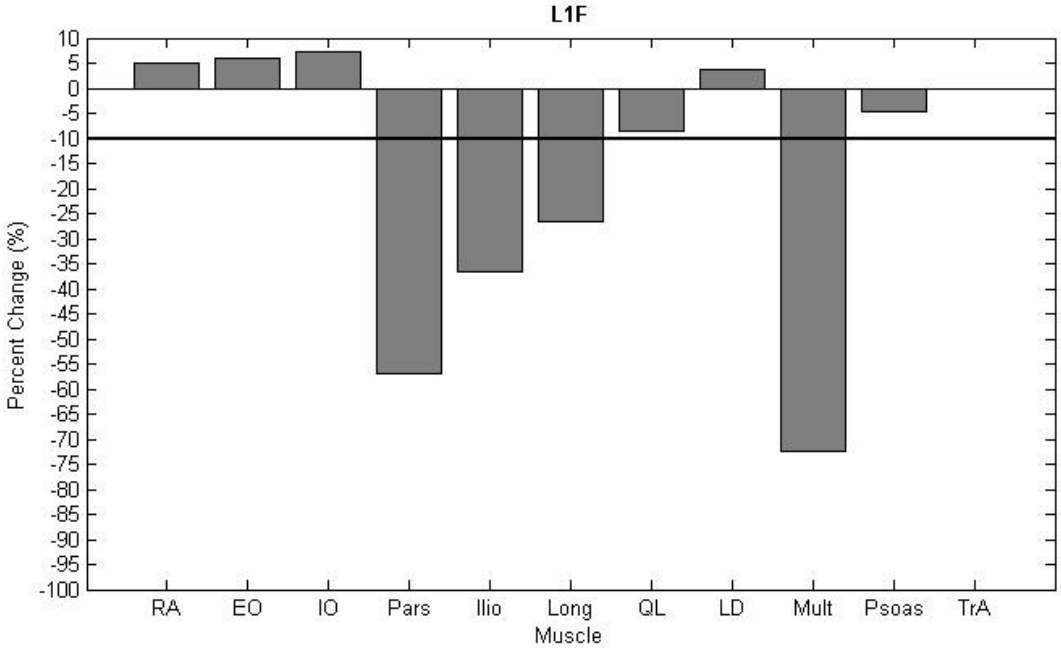

Figure 3 - Percent change in the L1F EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

Table 3 - EV magnitudes (J/rad^2) when averaged across all postures. (Mean ± SD)

	L1F *	L1B*	L1T*	L2F*	L2B*	L2T*	L3F*	L3B*	L3T*	L4F*	L4B*	L4T*	L5F*	L5B*	L5T*
All	**275.6**	**318.3**	**364.1**	**396.0**	**437.1**	**466.4**	**524.2**	**572.4**	**798.2**	**972.5**	**2149.1**	**2868.5**	**4002.0**	**6122.8**	**9658.1**
50%	(60.3)	(55.9)	(69.1)	(56.6)	(54.7)	(46.9)	(68.6)	(78.8)	(124.3)	(115.9)	(518.3)	(638.8)	(579.6)	(1252.0)	(1285.4)
RA	**289.2**	**322.9**	**371.3**	**398.5**	**434.9**	**464.3**	**498.0**	**564.6**	**791.5**	**965.8**	**2075.6**	**2746.2**	**3490.1**	**4757.6**	**8697.9**
0%	(61.5)	(54.6)	(65.0)	(56.8)	(52.1)	(47.6)	(45.6)	(72.2)	(135.7)	(121.2)	(575.1)	(553.5)	(488.3)	(616.1)	(1278.3)
EO	**288.7**	**320.5**	**371.6**	**398.1**	**436.1**	**462.4**	**498.0**	**543.1**	**756.4**	**864.4**	**1283.2**	**2395.0**	**3625.4**	**5298.8**	**7246.8**
0%	(67.2)	(55.8)	(63.8)	(60.6)	(54.2)	(46.5)	(51.1)	(69.4)	(118.3)	(110.5)	(241.9)	(514.9)	(822.2)	(1122.6)	(1231.8)
IO	**288.2**	**318.3**	**361.5**	**386.8**	**412.2**	**440.1**	**476.2**	**539.6**	**728.8**	**881.3**	**1128.7**	**2341.5**	**3177.3**	**4004.1**	**7288.3**
0%	(65.2)	(51.3)	(61.0)	(52.1)	(42.4)	(46.6)	(47.9)	(66.6)	(120.0)	(119.5)	(187.6)	(457.0)	(577.5)	(750.4)	(849.7)
Pars	**128.4**	**227.0**	**262.5**	**300.6**	**324.9**	**365.0**	**406.3**	**432.4**	**563.9**	**676.2**	**1898.6**	**2626.4**	**3435.2**	**6009.9**	**9631.8**
0%	(11.6)	(53.4)	(51.0)	(55.0)	(51.2)	(64.5)	(59.2)	(54.0)	(80.1)	(85.9)	(405.3)	(691.4)	(285.3)	(1345.2)	(1256.3)
Ilio	**189.3**	**222.3**	**263.5**	**300.8**	**350.2**	**399.8**	**458.5**	**536.9**	**585.5**	**872.6**	**1633.1**	**2186.2**	**2941.8**	**5990.1**	**9354.7**
0%	(31.8)	(35.7)	(47.4)	(43.9)	(58.0)	(43.0)	(47.4)	(69.5)	(69.8)	(140.9)	(288.3)	(531.1)	(301.7)	(1373.7)	(1340.6)
Long	**211.7**	**254.4**	**291.1**	**328.3**	**364.0**	**411.5**	**495.4**	**547.7**	**733.4**	**858.5**	**2024.7**	**2640.1**	**3361.9**	**5978.2**	**9582.9**
0%	(45.3)	(53.0)	(48.4)	(59.5)	(48.0)	(32.8)	(67.9)	(82.3)	(112.6)	(85.8)	(535.9)	(602.7)	(199.2)	(1353.3)	(1290.5)
QL	**257.4**	**308.8**	**347.7**	**383.7**	**416.7**	**443.9**	**509.1**	**549.8**	**686.5**	**888.7**	**1991.0**	**2770.3**	**3919.7**	**5982.8**	**9171.6**
0%	(54.9)	(57.8)	(68.6)	(58.0)	(55.9)	(44.1)	(67.8)	(71.9)	(92.5)	(127.0)	(473.4)	(646.2)	(605.0)	(1224.1)	(1321.4)
LD	**283.6**	**319.6**	**364.1**	**394.7**	**432.9**	**457.3**	**517.9**	**568.5**	**774.2**	**954.6**	**1878.5**	**2747.4**	**3926.4**	**5863.9**	**8955.0**
0%	(62.7)	(55.2)	(66.0)	(57.1)	(56.4)	(50.3)	(68.9)	(76.5)	(118.0)	(122.0)	(460.8)	(652.0)	(589.7)	(1249.7)	(1289.8)
Mult	**86.9**	**204.8**	**290.8**	**330.0**	**378.1**	**406.7**	**438.1**	**511.9**	**658.5**	**805.7**	**2101.5**	**2821.4**	**3804.4**	**6034.2**	**9643.1**
0%	(11.1)	(25.8)	(54.0)	(55.5)	(66.0)	(54.5)	(46.7)	(71.3)	(102.0)	(66.7)	(529.2)	(643.1)	(458.3)	(1269.6)	(1271.2)
Psoas	**250.3**	**284.1**	**334.2**	**367.6**	**394.5**	**424.0**	**486.7**	**543.7**	**724.6**	**956.5**	**1922.0**	**2724.4**	**3966.2**	**6121.4**	**9654.9**
0%	(61.9)	(51.7)	(63.8)	(63.6)	(62.8)	(51.6)	(65.8)	(78.4)	(101.7)	(140.1)	(434.3)	(695.1)	(619.2)	(1252.7)	(1286.7)
TrA	**275.1**	**315.1**	**362.8**	**393.2**	**433.5**	**464.1**	**522.2**	**570.4**	**792.5**	**971.4**	**2142.7**	**2863.9**	**3999.1**	**6122.5**	**9657.9**
0%	(60.6)	(56.4)	(68.8)	(57.2)	(54.6)	(46.4)	(68.9)	(79.5)	(122.4)	(116.8)	(515.1)	(640.0)	(581.7)	(1252.2)	(1285.3)

*** Units in J/rad^2**

The L1B EV (neutral posture in figure 4) had a similar trend to that of the L1F EV. Removing the Mult muscle had the largest effect at 36.3% ± 9.9%, followed by the Ilio muscle at 31.4% ± 5.9%. The Pars and Long muscles also had a biologically significant effect, with a change of 28.3% ± 5.7% and 21.0% ± 6.3%, respectively. However, no one muscle consistently had the largest effect on all postures. The Psoas muscle had a biologically significant effect on select postures, including 30° and 40° flexion, 20° and 30° bend, and all twist postures. Psoas always had the least effect of the muscles that influenced the EVs, with all postures having less than a 15% change, except for 40° flexion, which had a 22.5% change. Absolute magnitudes when averaged across all postures are shown in table 3.

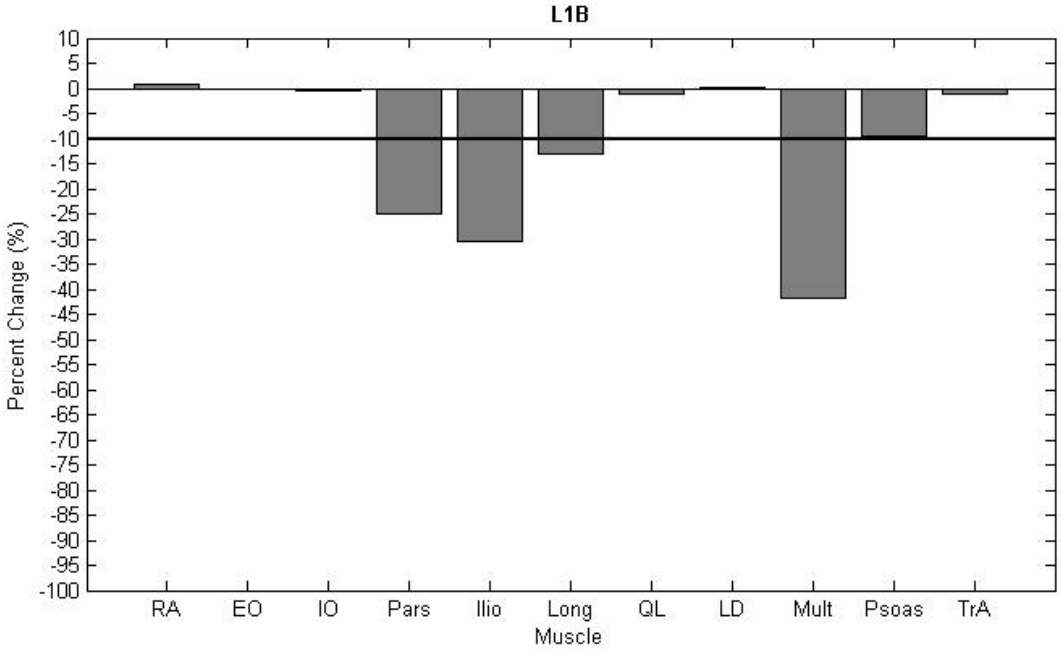

Figure 4 - Percent change in the L1B EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

Similar results were obtained when assessing the L1T EV (neutral posture in figure 5, absolute magnitudes in table 3) as the L1F and L1B EVs, but the Ilio and Pars muscles had the largest effect, with

averages of 28.1% ± 3.1% and 27.7% ± 7.8% change, respectively. Mult followed with an average of 21.1% ± 8.2%, and Long had an average of a 19.6% ± 3.3% change. As with the L1B EV, no single muscle consistently had the largest effect on the L1T EV as posture was varied. Again, the Psoas muscle was influenced by posture, resulting in a biologically significant effect for the neutral, 50° flexion, and all bend postures. For the L1T EV, Psoas always had the least effect of the muscles that had a biologically significant change, except in the 50° flexion posture where the Long muscle had the least effect. In all cases, the Psoas muscle caused less than a 22% change in the EV.

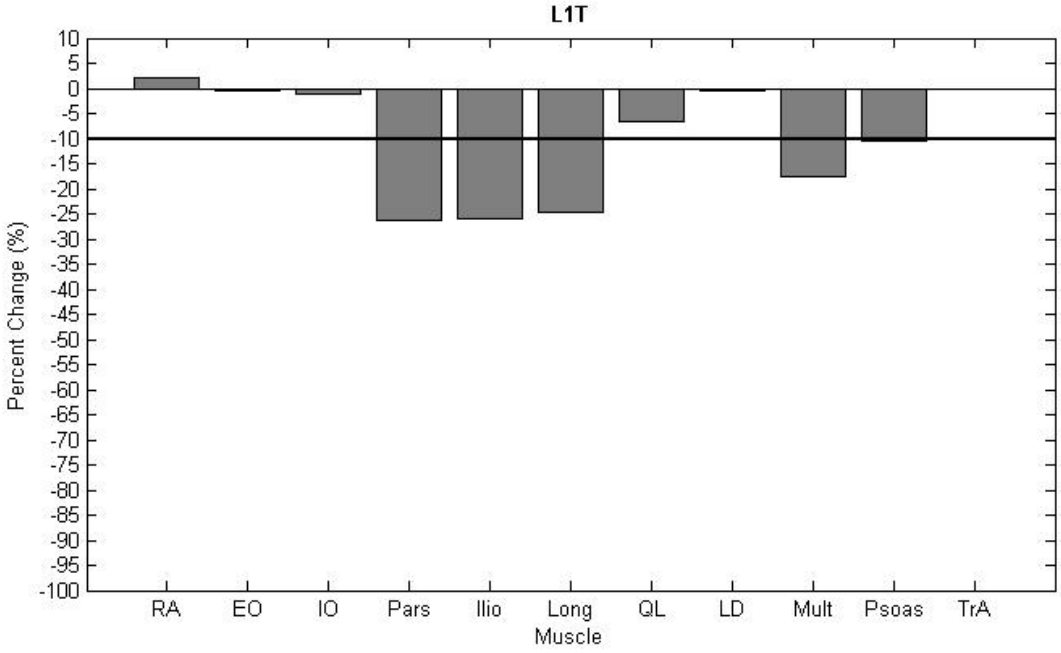

Figure 5 - Percent change in the L1T EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

The Mult, Pars, Ilio, Long and Psoas muscles were also the only muscles that affected the L2 level EVs. For the L2F EV (neutral posture in figure 6, average absolute values in table 3), the Mult muscle was more dependent on posture than at the L1 level, having a biologically significant effect on all

flexion postures, all bend postures, and the 10° and 20° twist postures (16.5% ± 10.1% change averaged across all postures). The Pars, Ilio and Long muscles caused a biologically significant change for all postures at the L2F level, except for the 30° and 40° twist postures where Long was not significant. When averaged across all postures, the Pars muscle resulted in a 23.5% ± 7.3% change, the Ilio muscle resulted in a 23.0% ± 4.6% change and the Long muscle resulted in a 16.0% ± 6.4% change in the L2F EV. The Psoas muscle was very dependent on posture, only resulting in a significant change for 40° and 50° flexion, 10° and 30° extension, and 30° bend. There was no muscle that resulted in the largest change in the L2F EV in all situations, but the Psoas muscle always had the smallest effect at less than 15% change.

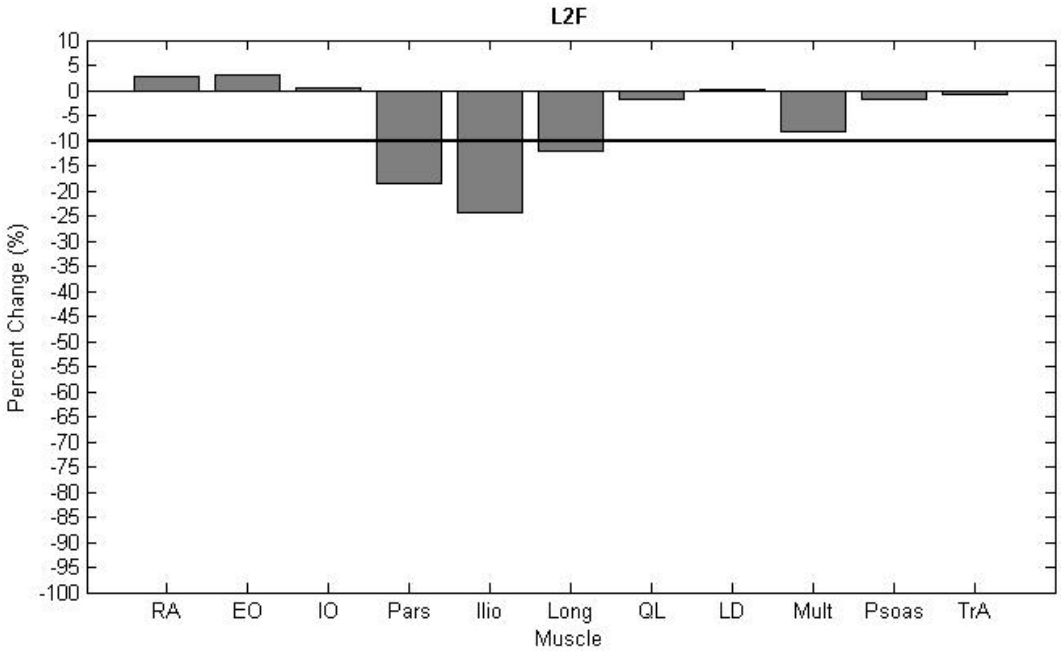

Figure 6 - Percent change in the L2F EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

For the L2B EV (neutral posture in figure 7, absolute values in table 3), the Pars muscle resulted in a biologically significant change for all postures, while the Ilio, Long and Mult muscles also caused a biologically significant change for most postures. When averaged across all postures, the Pars muscle resulted in a 25.0% ± 3.6% change, the Ilio muscle had a 19.3% ± 5.4% change, the Long muscle had a 15.7% ± 4.0% change and the Mult muscle resulted in a 13.9% ± 5.5% change in the L2B EV. The Psoas muscle always had less than a 22% change in the EV and was biologically significant for the two most extreme postures in flexion, extension and twist. The Pars muscle consistently had the largest influence across all postures for the L2B EV, except for in 20° and 30° extension, where the Ilio muscle had a slightly larger effect than the Pars muscle. Similar to the previous EVs, the Psoas muscle always had the smallest influence of the muscles that caused a biologically significant change except in 30° extension.

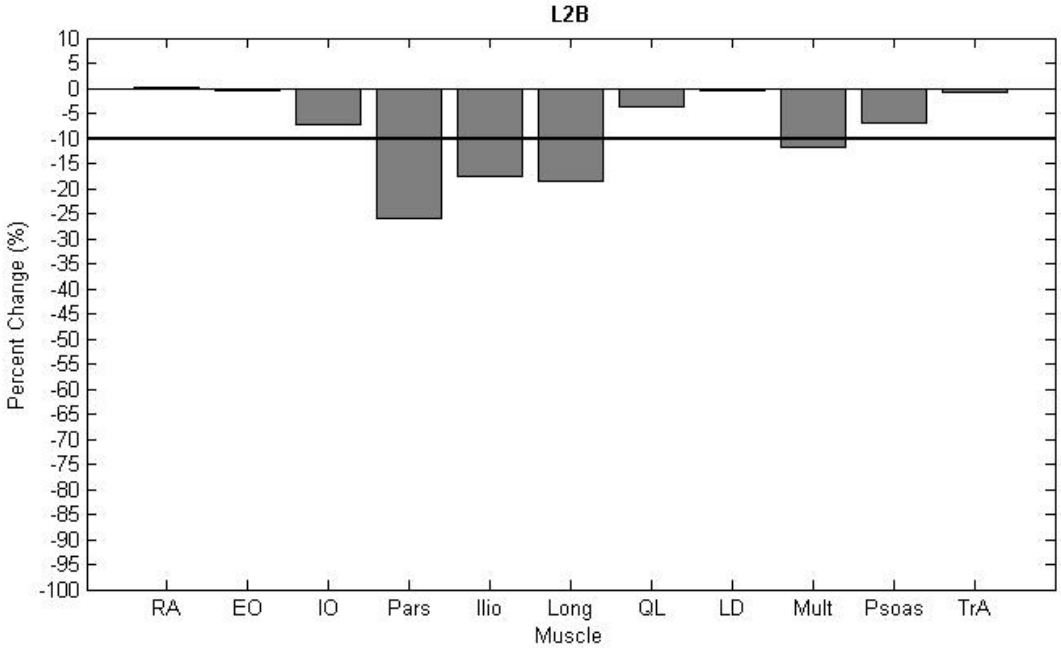

Figure 7 - Percent change in the L2B EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

The L2T EV (neutral posture in figure 8, average absolute values in table 3) showed similar trends as the L2B EV, with the Pars muscle causing a biologically significant change for all postures and the Ilio, Long and Mult muscles being dependent on posture. When averaged across all postures, the Pars muscle had a 21.4% ± 6.4% change, the Ilio muscle had a 13.4% ± 4.8% change, the Mult muscle had a 13.2% ± 4.6% change, and the Long muscle had an 11.8% ± 4.0% change in the EV. Similar to the L2B EV, the Pars muscle always had the largest influence on the L2T EV, except for 10° extension, where the Ilio muscle resulted in a slightly larger influence. The Psoas muscle resulted in a biologically significant change for the neutral posture and all extension postures, at less than a 21% change in the L2T EV. Unlike previous EVs, the Psoas muscle did not always have the smallest effect of the muscles that resulted in a biologically significant change.

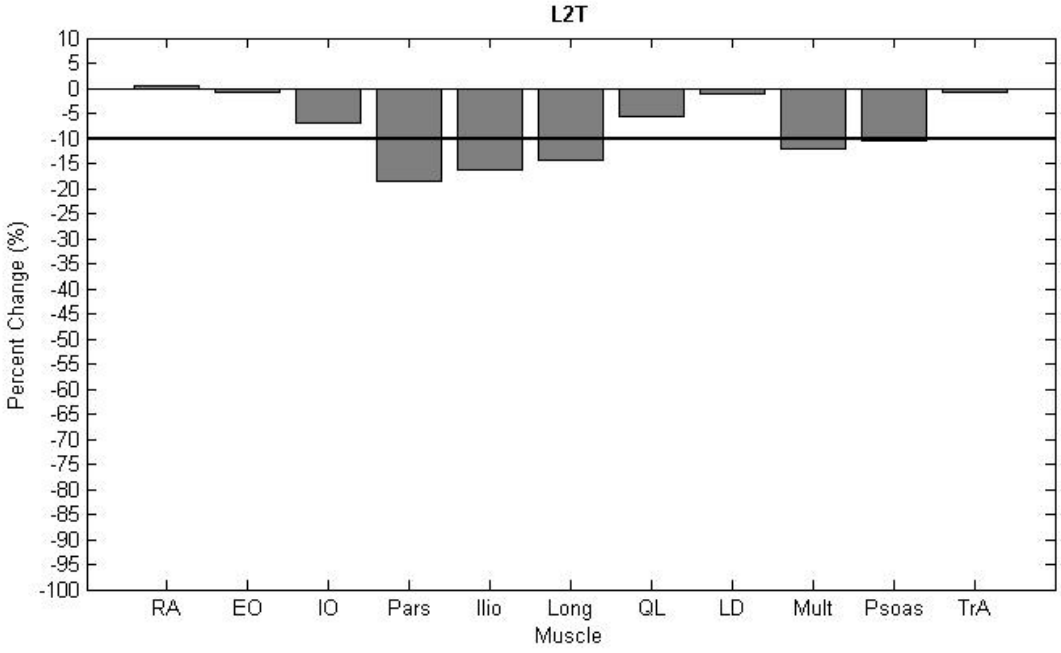

Figure 8 - Percent change in the L2T EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

The Pars muscle was the only muscle that resulted in a biologically significant change for all postures for the L3F EV (neutral posture in figure 9, average absolute values in table 3). The Mult, Ilio and IO muscles also resulted in biologically significant changes for most postures. The Pars muscle always resulted in the largest change, with a 22.5% ± 4.8% change in the L3F EV when averaged across all postures. The second largest change was typically seen with the Mult muscle, with an average of 16.5% ± 5.2% change across all postures. When the Ilio muscle was considered biologically significant, it had the third largest effect, at 11.8% ± 5.7% change in the L3F EV when averaged across all postures. The IO muscle always had a small effect, with a change of less than 15% in the EV in all postures. The Psoas muscle was biologically significant for the extension postures, with less than a 15% change in the L3F EV.

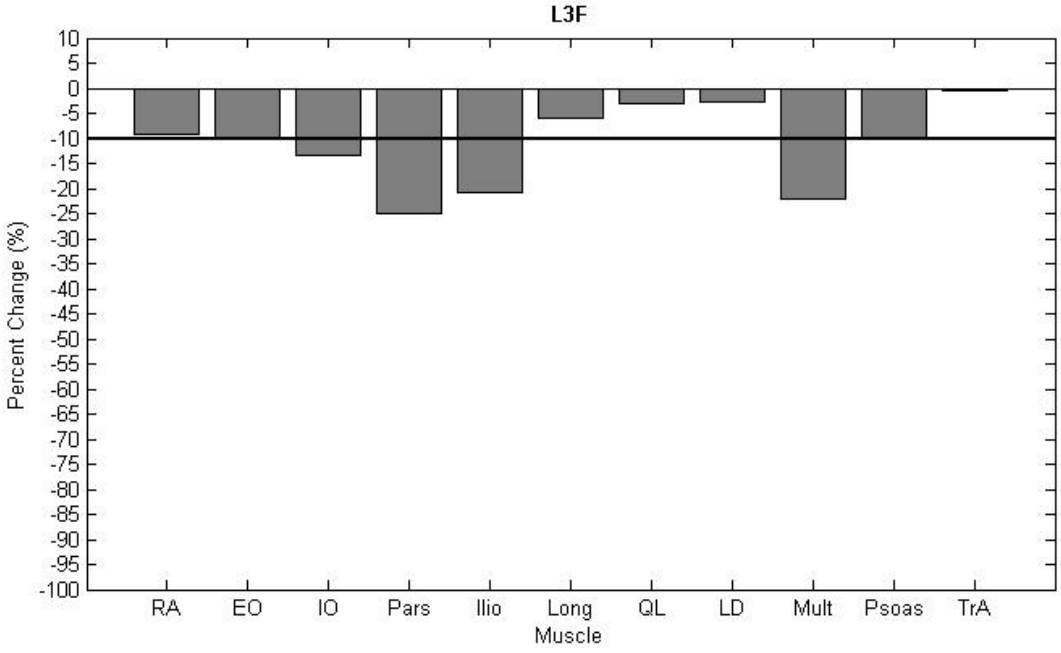

Figure 9 - Percent change in the L3F EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

The L3B EV (neutral posture in figure 10, average absolute values in table 3) was not affected by many muscles. In all postures, the largest change in the EV was seen with the Pars muscle, with an average of 25.1% ± 3.9% across all postures. The Mult and Ilio muscles also caused a biologically significant effect for multiple postures. When averaged across all postures, the Mult muscle resulted in an 11.5% ± 6.1% change in the L3B EV, while the Ilio muscle always had less than a 15% change in the EV.

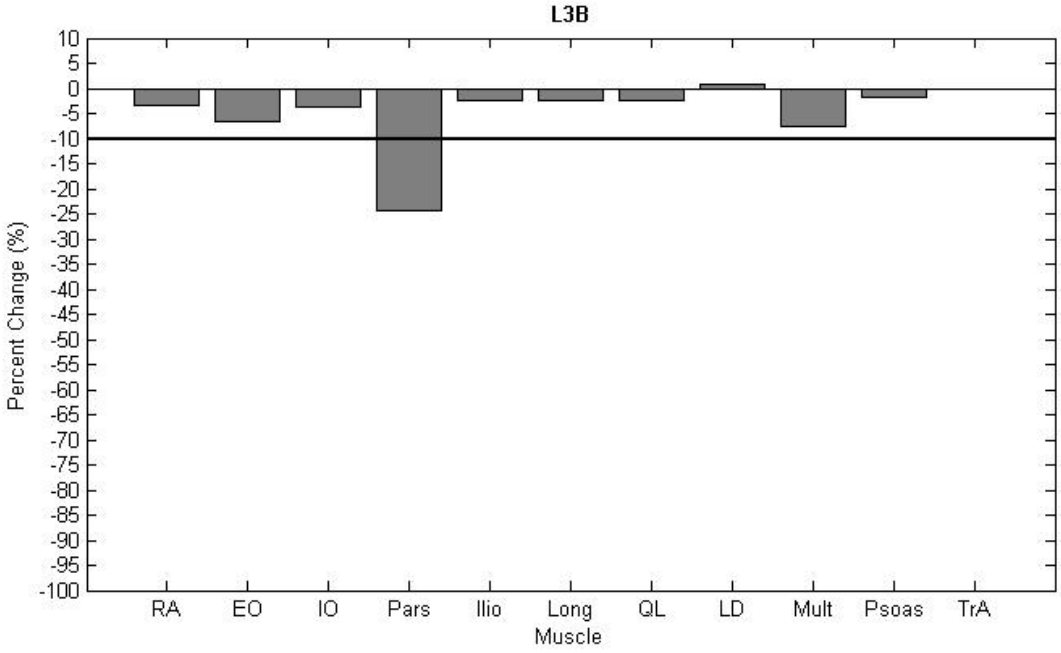

Figure 10 - Percent change in the L3B EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

No individual muscle consistently resulted in the largest change for the L3T EV (neutral posture in figure 11, average absolute values in table 3). The Pars and Ilio muscles always resulted in a biologically significant change except for the 50° flexion posture when the Ilio muscle did not have a biologically significant change. When averaged across all postures, the Pars muscle resulted in a 29.7% ±

6.3% change, while the Ilio muscle resulted in a 26.0% ± 5.6% change in the L3T EV. The Mult and QL muscles also caused biologically significant changes for most postures, with changes of 17.6% ± 7.7% and 13.9% ± 4.2% in the L3T EV, respectively, when averaged across all postures. The Psoas muscle was biologically significant for most postures, but had a change of less than 12% in all situations. The IO and EO muscles were also biologically significant for select postures, with changes in the L3T EV of less than 17% for the IO muscle and less than 19% for the EO muscle.

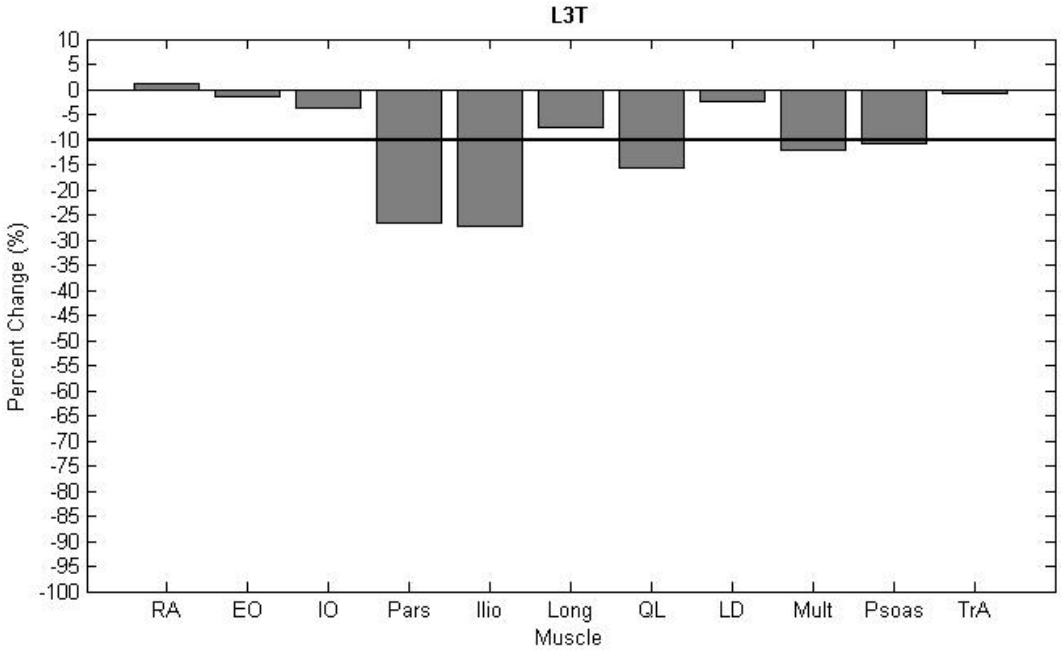

Figure 11 - Percent change in the L3T EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

The Pars muscle was the only muscle that resulted in a biologically significant change for all postures when examining the L4F EV (neutral posture in figure 12, average absolute values in table 3), with a change in the EV of 30.9% ± 5.0% when averaged across all postures. The Mult and Long

muscles were also biologically significant for most postures. When averaged across all postures, the Mult muscle had a change of 17.1% ± 6.9% and the Long muscle had a change of 12.9% ± 2.4% in the L4F EV. The IO and Ilio muscles also resulted in a biologically significant change for multiple postures. The highest percent change in the L4F EV for the IO muscle was 21.6%, seen in the 30° extension posture. For the Ilio muscle, all postures resulted in changes of less than 11%, except for the 40° and 50° flexion postures, which had 21.6% and 30.3% changes in the EV, respectively. The EO and QL muscles also resulted in a biologically significant change for select postures. The EO and QL muscles always had less than a 20% change in the L4F EV, except for the EO muscle in the 20° and 30° extension postures, which resulted in changes of 21.0% and 27.0%, respectively.

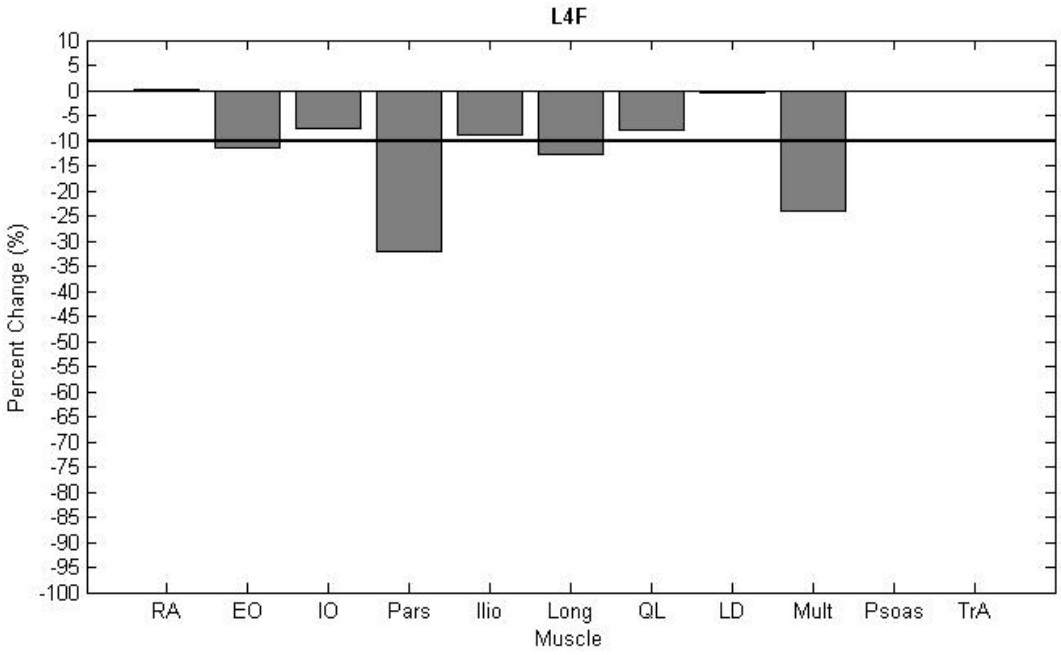

Figure 12 - Percent change in the L4F EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

The IO and EO muscles became the most influential muscles for the L4B EV (neutral posture in figure 13, average absolute values in table 3), while the Mult muscle no longer had a biologically significant effect. The EO muscle resulted in a biologically significant change for all postures, while the IO muscle was biologically significant for all postures except 40° twist. When averaged across all postures, the EO muscle resulted in a 38.8% ± 10.6% change, while the IO muscle resulted in a 40.3% ± 18.4% change in the L4B EV. The IO muscle had the largest influence on the neutral, flexion and extension postures, as well as the 10° bend and 10° twist postures, while the EO muscle had the largest influence on the remaining postures. The Ilio muscle caused a biologically significant effect for most postures, resulting in a 20.3% ± 10.2% change in the L4B EV when averaged across all postures. Although the RA muscle only caused biological significance for the 20° and 30° bend postures, it was responsible for the third largest change with these postures, at 29.6% change and 13.2% change, respectively, in the L4B EV. The Pars muscle resulted in a small biologically significant change for most postures, resulting in less than a 16% change in the EV for all postures. The LD muscle always had less than a 17% change in the L4B EV, except for the 50° flexion posture, which had a 23.6% change. The Psoas muscle was the only other muscle that showed a biologically significant change for more than two postures. This muscle always resulted in less than a 20% change in the L4B EV, except for the 30° extension posture where it had a 27.4% change. It was also noted for the L4B EV that the Mult muscle resulted in a 2.6% ± 2.0% change when averaged across all postures.

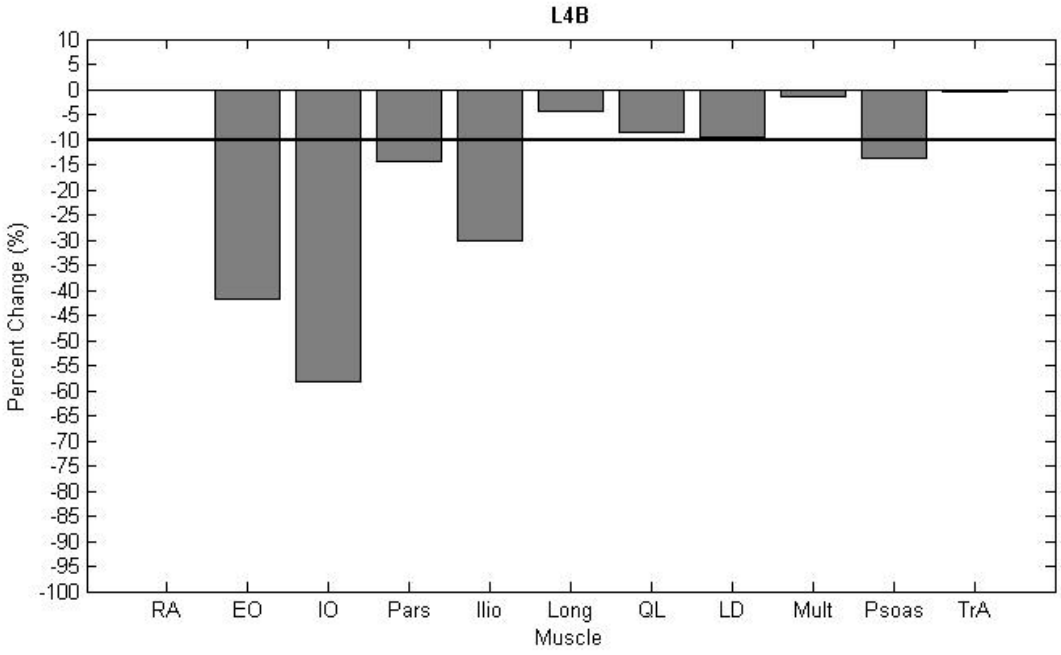

Figure 13 - Percent in the L4B EV change when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

For the L4T EV (neutral posture in figure 14, average absolute values in table 3), the muscle that had the largest biological significant effect varied between the EO, IO and Ilio muscles. The IO muscle had the greatest influence for the postures closer to neutral, while the EO muscle had the largest influence for the end range flexion postures. The Ilio muscle had the greatest influence for the remaining postures. The Ilio muscle had a biologically significant effect across all postures, with an average of a 23.6% ± 8.1% change in the L4T EV. The EO and IO muscles were also biologically significant for most postures. When averaged across all postures, the EO muscle caused a 12.9% ± 11.7% change and the IO muscle had a 15.5% ± 8.7% change in the L4T EV. The Pars muscle resulted in a biologically significant change for the end range of motion postures, always causing less than an 18% change in the EV. The

Psoas muscle also resulted in a biologically significant change for the end range of motion postures, with less than a 14% change, except for in 30° bend where there was an 18.1% change in the L4T EV. Finally, the Long muscle was only important for the 30° flexion, 10° extension and 20° extension postures, with a change of 17.0%, 15.2% and 25.7%, respectively, in the EV. It was also noted for the L4T EV that the Mult muscle resulted in a 1.8% ± 1.6% change when averaged across all postures.

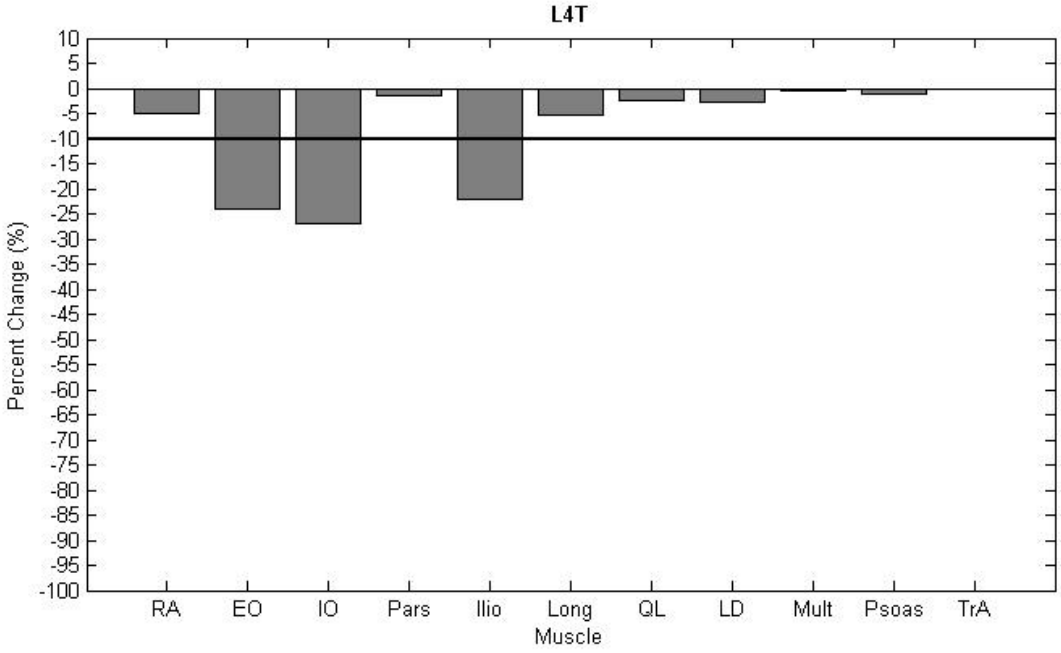

Figure 14 - Percent change in the L4T EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

No individual muscle resulted in a biologically significant change for every posture for the L5F EV (neutral posture in figure 15, average absolute values in table 3). However, the IO, Ilio, Pars and Long muscles all caused biological significance for the majority of postures. The Ilio muscle had the greatest influence for all postures except the extension postures where the IO muscle had the greatest

influence, and the 30° bend posture where the RA muscle had a slightly greater influence over the Ilio muscle. When averaged across all postures, the Ilio muscle had a 28.2% ± 11.2% change, the IO muscle caused a 20.8% ± 7.0% change, the Long muscle had a 16.6% ± 7.7% change and the Pars muscle resulted in a 14.6% ± 6.0% change in the L5F EV. The EO and RA muscles were the only other muscles that resulted in a biologically significant change. The EO muscle caused less than a 20% change in the EV, except for in the 30° extension and 30° bend postures, where it had changes of 37.6% and 28.0%, respectively. The RA muscle had less than a 17% change in the L5F EV, except for in the 30° bend, 30° twist and 40° twist postures, where there was a change of 32.1%, 24.5% and 32.5%, respectively. It was also noted for the L5F EV that the Mult muscle resulted in a 5.1% ± 2.5% change when averaged across all postures.

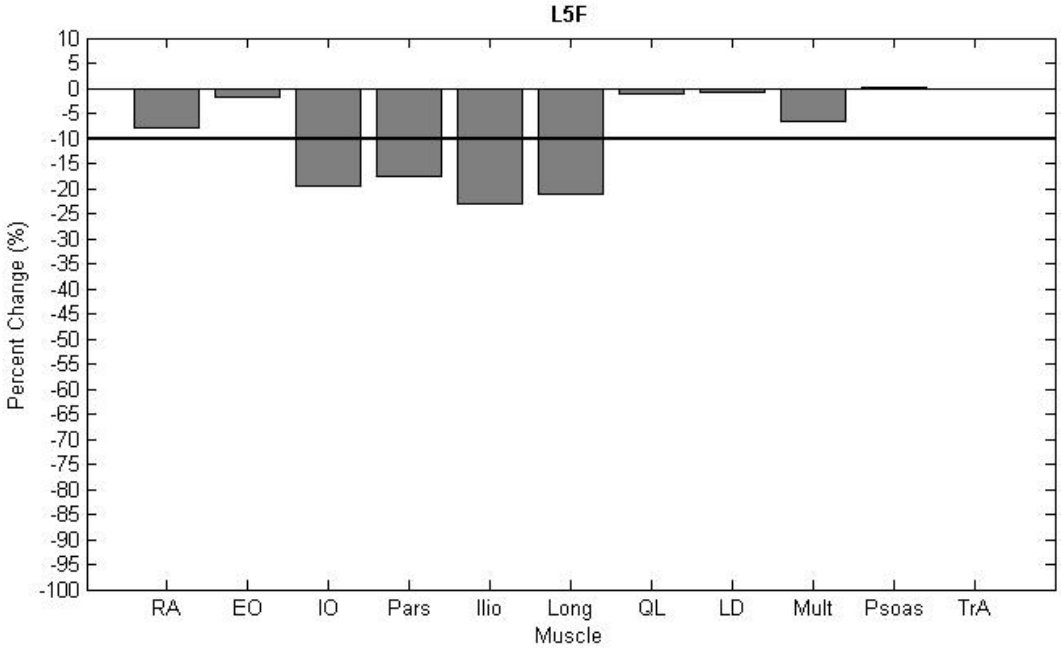

Figure 15 - Percent change for the L5F EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

The abdominal muscles were the only muscles that had a biologically significant effect on multiple postures for the L5B EV (neutral posture in figure 16, average absolute values in table 3), but no muscle had a biologically significant effect for all postures. For this EV, the EO muscle had the greatest influence for the 50° flexion posture and the RA muscle had the greatest influence for the 20° bend, 30° bend, and 20°, 30° and 40° twist postures, while the IO muscle had the greatest influence on the remaining postures. When averaged across all postures, the IO muscle resulted in a 30.2% ± 14.1% change, the RA muscle resulted in a 21.8% ± 9.2% change and the EO muscle caused a 13.8% ± 6.7% change in the L5B EV. It was also noted for the L5B EV that the Mult muscle resulted in a 1.4% ± 0.9% change when averaged across all postures.

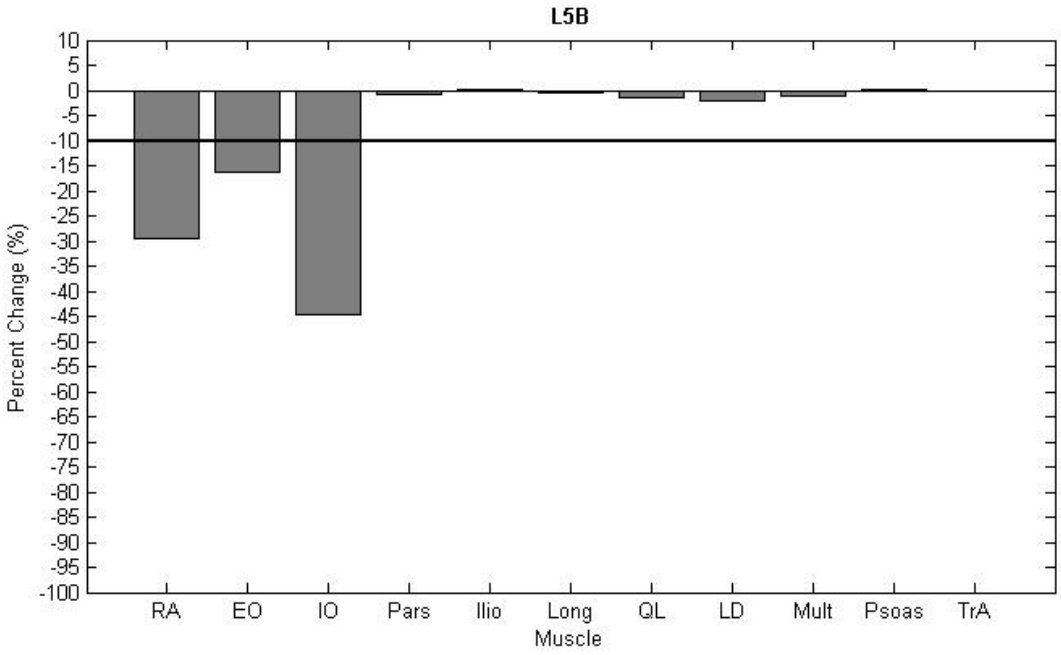

Figure 16 - Percent change in the L5B EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

Finally, for the L5T EV (neutral posture in figure 17, average absolute values in table 3), the abdominal muscles caused the greatest influence on the EV, but the LD muscle had a small influence resulting in less than a 13% change in the EV for the neutral and extension postures. The effect of the RA muscle was also small, with a change in the L5T EV of less than 15%. The IO muscle had the largest influence on the L5T EV for the neutral, extension, bend, and 10° and 20° twist postures, while the EO muscle had the greatest influence for the remaining postures. The EO muscle resulted in a biologically significant change for all postures, with an average of a 23.9% ± 10.3% change in the EV. The IO muscle resulted in a biologically significant change for all postures except 50° flexion and when averaged across all postures, there was a 26.4% ± 9.4% change in the L5T EV. It was also noted for the L5T EV that the Mult muscle resulted in a 0.2% ± 0.2% change when averaged across all postures.

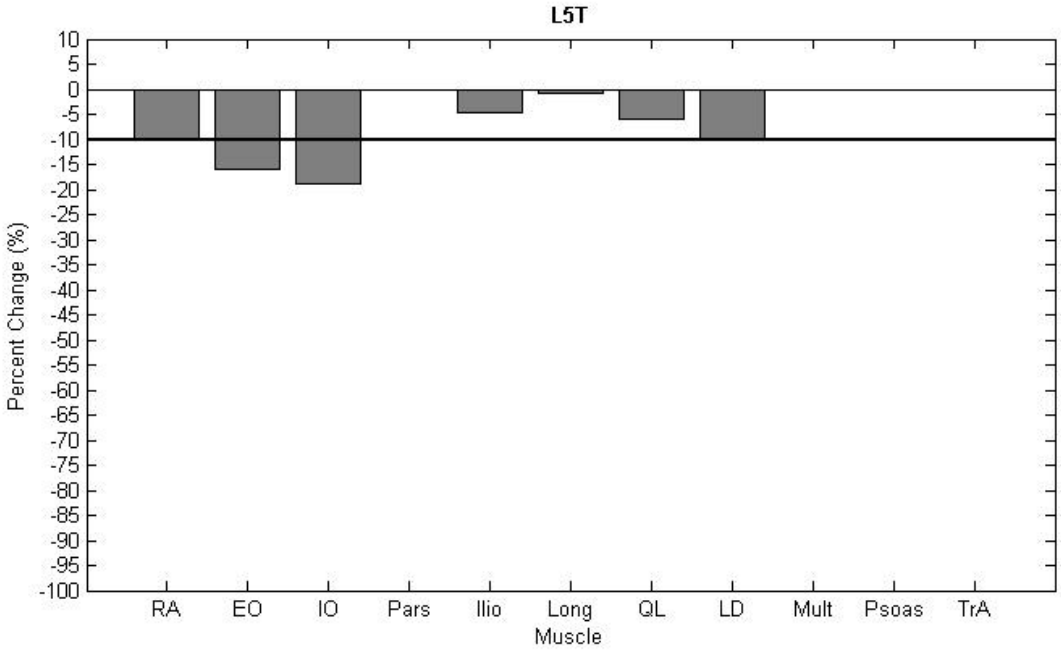

Figure 17 - Percent change in the L5T EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for the neutral posture. Negative change represents a lower EV when the single muscle's activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

In summary, the erector spinae muscles influenced the upper lumbar level EVs (L1, L2 and L3 levels), more than the abdominal muscles, while the opposite occurred for the lower lumbar level EVs (L4 and L5 levels), than the erector spinae muscles. In addition, single muscles did affect multiple levels of spine stability/stiffness and stability/stiffness at one segmental level was influenced by multiple muscles.

4.1.2 Hypothesis 2: Effect of Specific Muscles on Plane of Stability/Stiffness

Muscles do appear to be related to specific planes of stability/stiffness. The L4 lumbar level will be discussed in detail because the L4 lumbar level contains the most anatomical robustness. The trends that occur at the other levels are shown in Appendix B.

At the L4 level, the RA muscle did not result in a biologically significant change in any EV for majority of postures (figure 18, average absolute values in table 3).

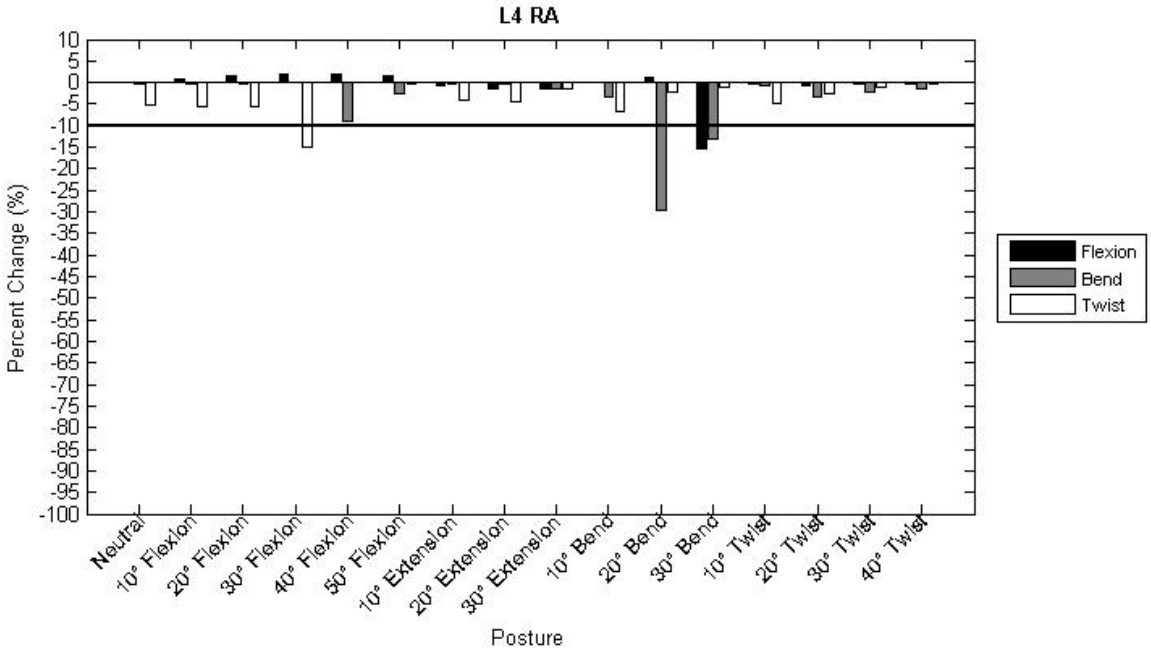

Figure 18 - Percent change in the L4 EVs when reducing the RA muscle activation to 0% MVC while all other muscles remained active to 50% MVC for all postures. Negative change represents a lower EV when the RA muscle activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

For the EO muscle (figure 19, average absolute values in table 3), when averaged across all postures, there was a 10.9% ± 6.0% change in the flexion axis L4 EV, 38.8% ± 10.6% change in the bend axis L4 EV, and 12.9% ± 11.7% change in the twist axis L4 EV. No individual plane of stability/stiffness was influenced the most by the EO muscle in all situations, but typically, the bend axis was most influenced followed by the twist axis.

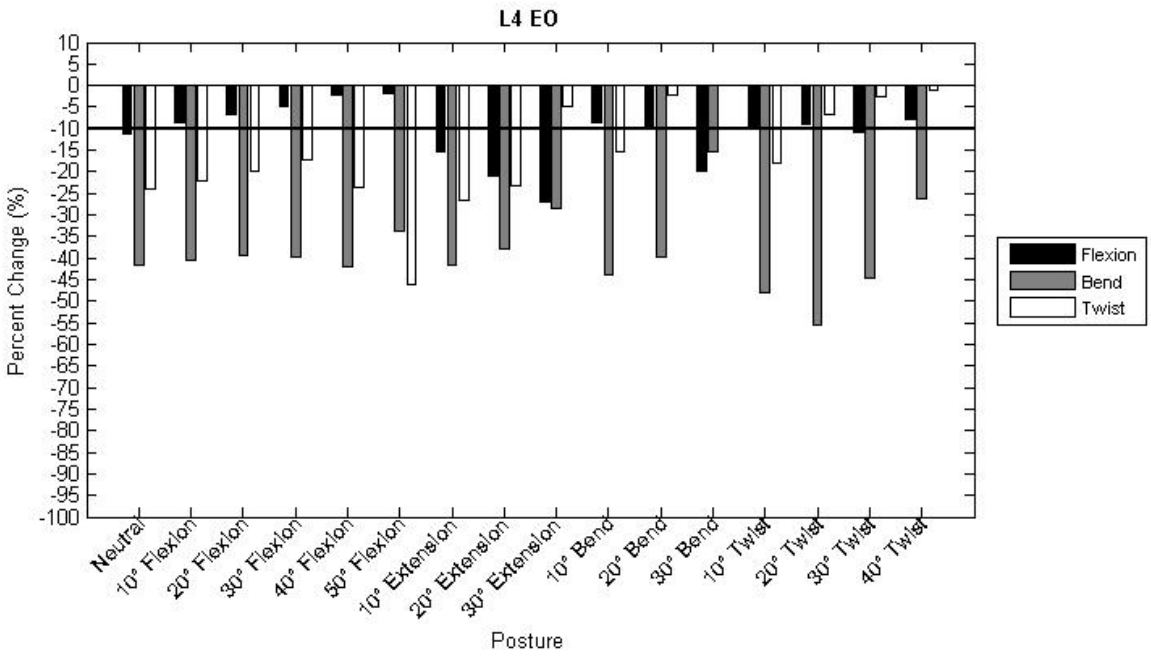

Figure 19 - Percent change in the L4 EVs when reducing the EO muscle activation to 0% MVC while all other muscles remained active to 50% MVC for all postures. Negative change represents a lower EV when the EO muscle activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

A similar trend was seen with the IO muscle (figure 20, average absolute values in table 3) as the EO muscle, with the bend axis L4 EV typically being the most influenced by removing the IO muscle followed by the twist axis L4 EV. At the L4 level, there was a change of 9.3% ± 5.2% in the flexion axis EV, 40.3% ± 18.4% in the bend axis EV and 15.5% ± 8.7% in the twist axis EV when averaged across all postures.

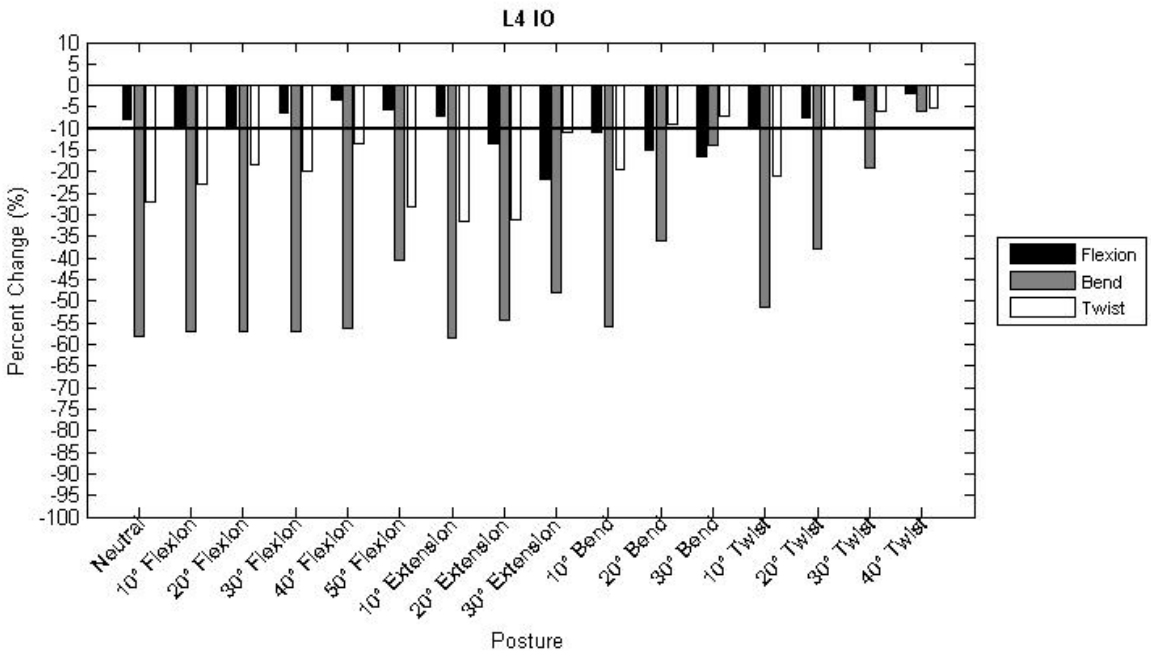

Figure 20 - Percent change in the L4 EVs when reducing the IO muscle activation to 0% MVC while all other muscles remained active to 50% MVC for all postures. Negative change represents a lower EV when the IO muscle activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

When the Pars muscle was removed (figure 21), the flexion axis always had a biologically significant change and was the most influenced plane of stability/stiffness at the L4 level. In the neutral and flexion postures, the bend axis was the second most influenced by the Pars muscle except for in the 30° flexion posture. With the bend and twist postures at the L4 level, there was no consistent pattern on whether the bend axis or twist axis was influenced the second most by the Pars muscle. When averaged across all postures, the flexion axis resulted in a 30.9% ± 5.0% change, the bend axis had a 10.2% ± 3.9% change and the twist axis caused a 9.6% ± 5.2% change in the L4 EVs.

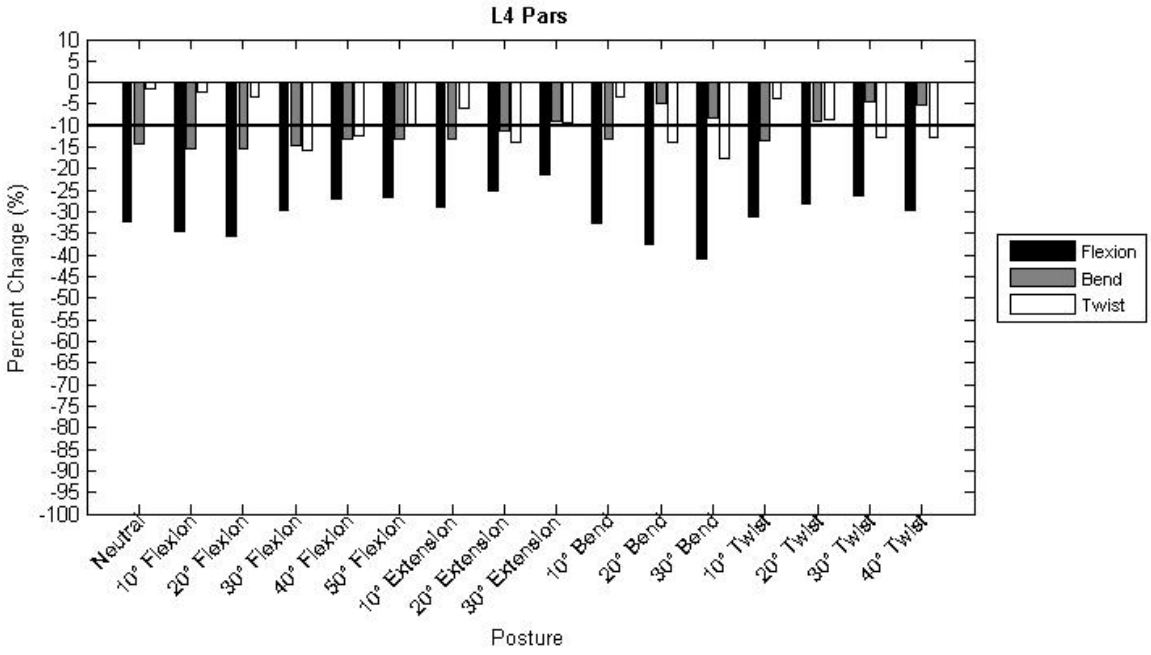

Figure 21 - Percent change in the L4 EVs when reducing the Pars muscle activation to 0% MVC while all other muscles remained active to 50% MVC for all postures. Negative change represents a lower EV when the Pars muscle activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

For the Ilio muscle at the L4 level (figure 22), no plane of stability/stiffness was consistently most affected across all postures. In the neutral, 10°, 20° and 40° flexion, 30° extension, 10°bend, and 10° and 20° twist postures the bend axis was the most influenced when Ilio was removed followed by the twist axis. The twist axis was the most influenced by the Ilio muscle in the remaining postures except the 50º flexion posture where the flexion axis was most affected. When averaged across all postures, the flexion axis had a 10.3% ± 5.4% change, the bend axis made a 20.3% ± 10.2% change and the twist axis caused a 23.6% ± 8.1% change in the L4 EVs.

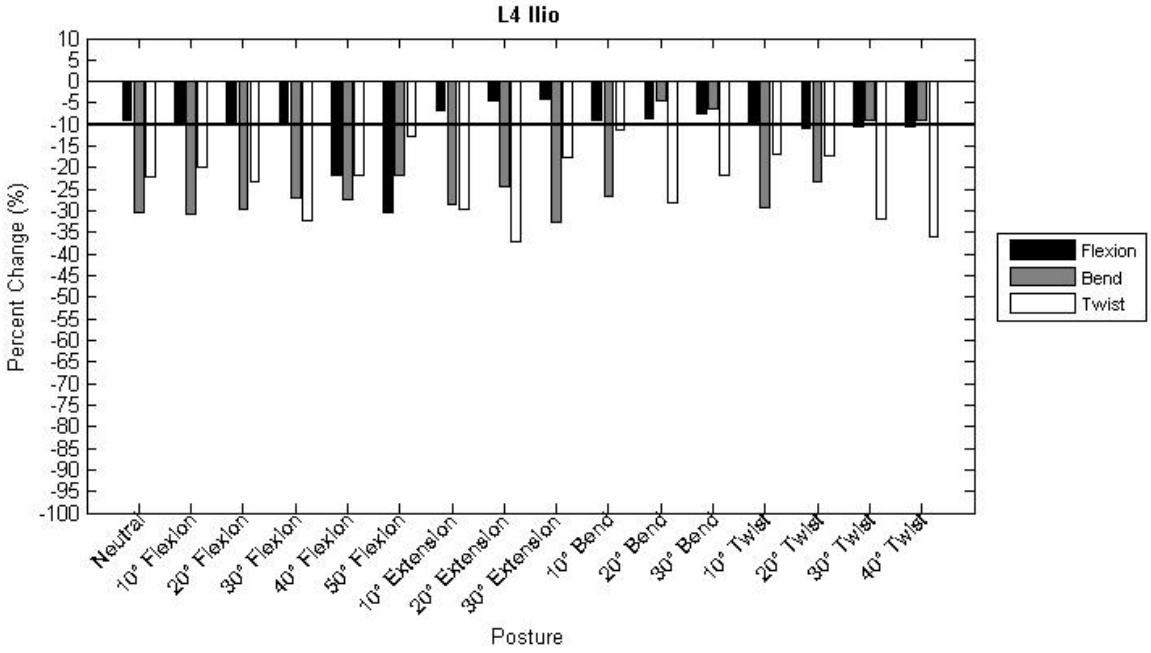

Figure 22 - Percent change in the L4 EVs when reducing the Ilio muscle activation to 0% MVC while all other muscles remained active to 50% MVC for all postures. Negative change represents a lower EV when the Ilio muscle activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

At the L4 level, the Long muscle (figure 23) had the greatest influence on the flexion axis of stability in all postures except the 30º, 40º and 50º flexion postures and extension postures. In the 40º flexion, 50º flexion and 30º extension postures, the Long muscle had the greatest influence on the bend axis, while for the 30º flexion, 10º extension and 20º extension postures, the twist axis was most affected. When averaged across all postures, the flexion axis resulted in an 11.9% ± 2.4% change, the bend axis resulted in a 5.7% ± 4.8% change and the twist axis resulted in a 7.4% ± 5.5% change in the L4 level EVs when the Long muscle was removed.

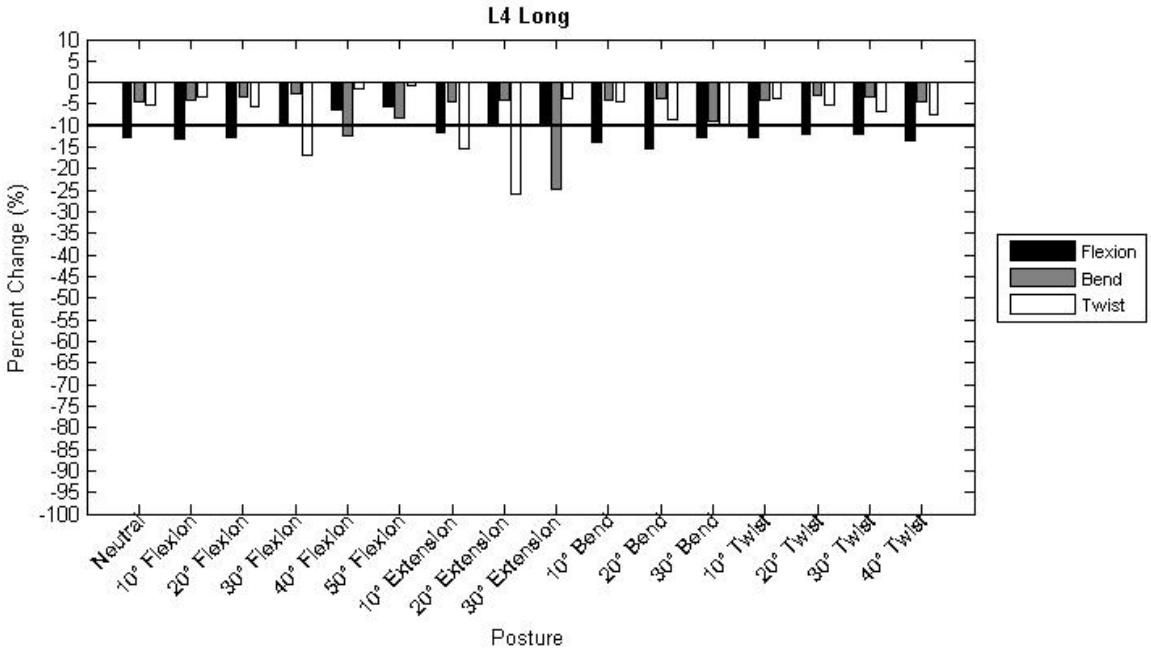

Figure 23 - Percent change in the L4 EVs when reducing the Long muscle activation to 0% MVC while all other muscles remained active to 50% MVC for all postures. Negative change represents a lower EV when the Long muscle activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

The Mult muscle only caused a biologically significant change in the flexion axis of stability/stiffness at the L4 level (figure 24). It was biologically significant for all postures except 30°, 40° and 50° flexion, with a 17.1% ± 6.9% change in the EV when averaged across all postures. In the majority of the postures, the bend and twist planes of stability/stiffness resulted in less than a 5% change in the L4 EV.

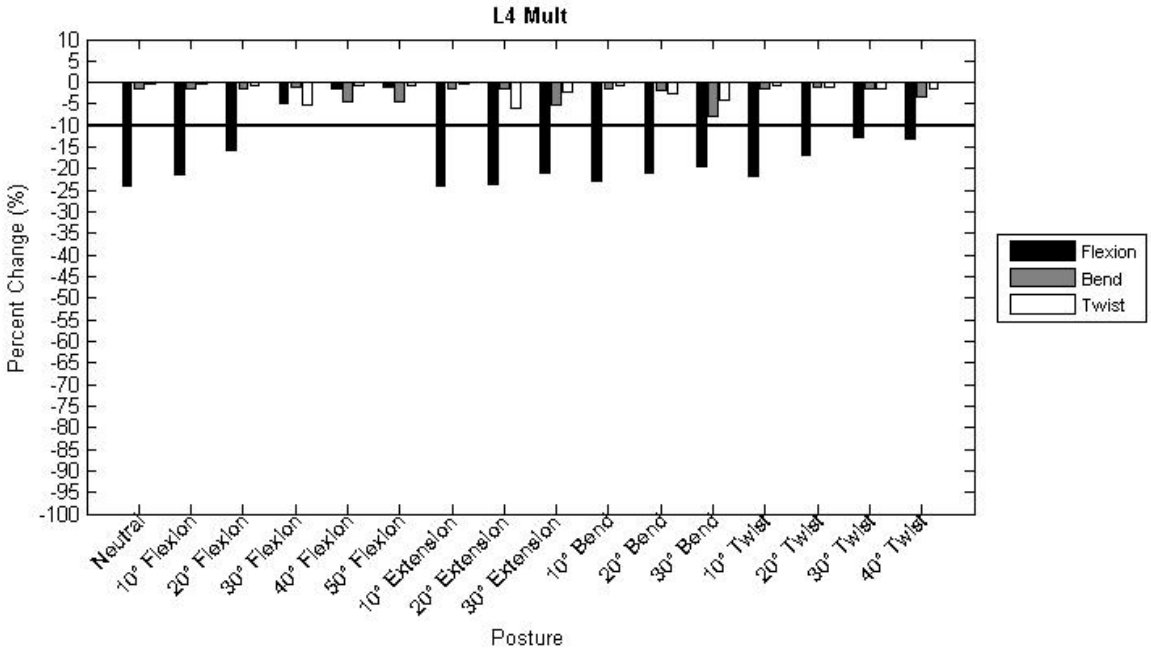

Figure 24 - Percent change in the L4 EVs when reducing the Mult muscle activation to 0% MVC while all other muscles remained active to 50% MVC for all postures. Negative change represents a lower EV when the Mult muscle activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

The QL muscle (figure 25) had biologically significant changes for the 40° and 50° flexion postures and all extension postures at the L4 level. In these postures, the flexion axis was influenced the most, but it was always less than a 20% change in the L4F EV.

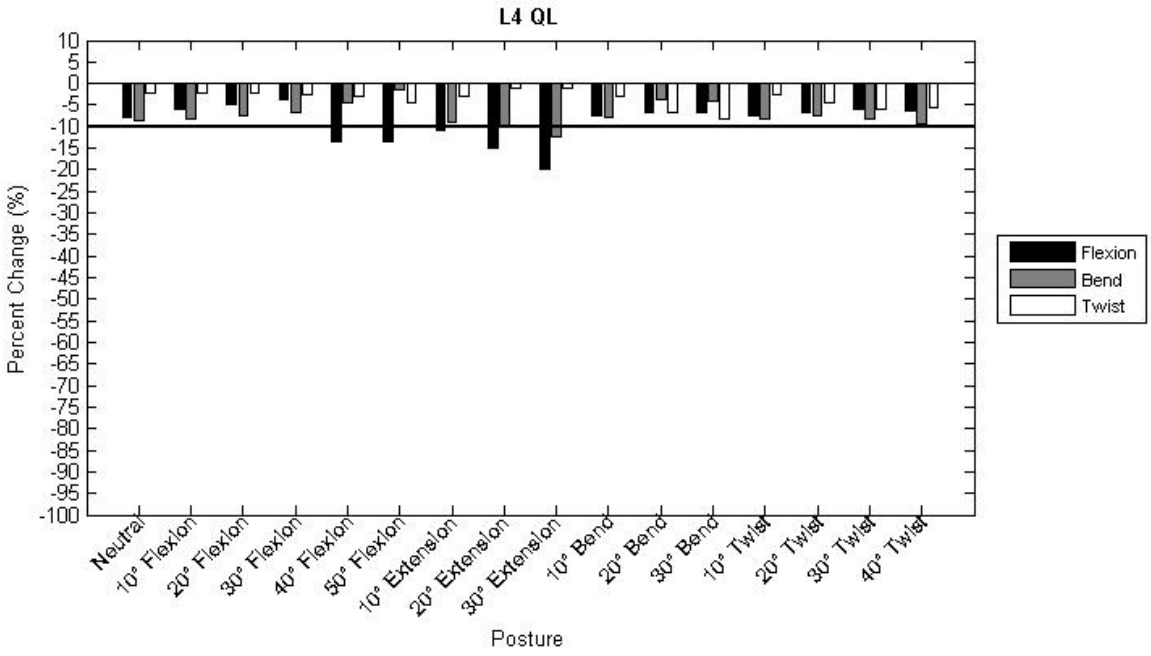

Figure 25 - Percent change in the L4 EVs when reducing the QL muscle activation to 0% MVC while all other muscles remained active to 50% MVC for all postures. Negative change represents a lower EV when the QL muscle activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

At the L4 level, the Psoas muscle (figure 26) influenced only one axis of stability/stiffness in each posture. In the deep flexion postures, the flexion axis was affected with 12.0% and 12.1% change in the EV, respectively. The twist axis was influenced in the deep bend, and deep twist postures, with less than a 19% change in the L4 T EV. For all other postures, the bend axis was affected, with changes in the EV of less than 20% except for the 30º extension posture where there was a 27.4% change.

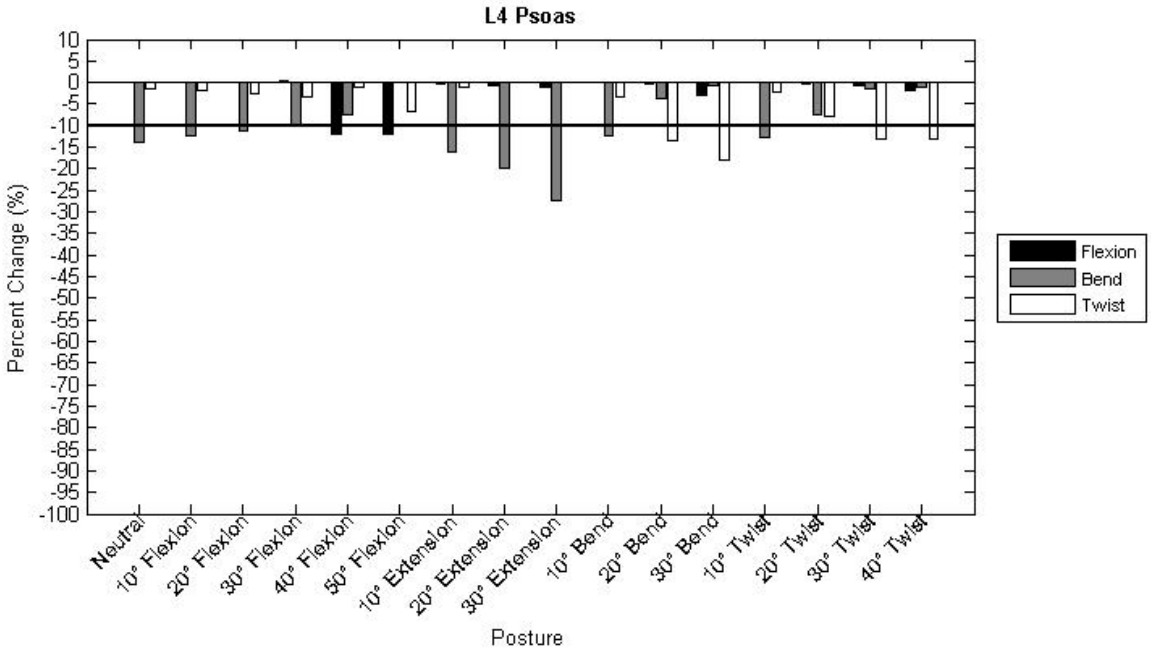

Figure 26 - Percent change in the L4 EVs when reducing the Psoas muscle activation to 0% MVC while all other muscles remained active to 50% MVC for all postures. Negative change represents a lower EV when the Psoas muscle activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

The LD muscle (figure 27) never resulted in a biologically significant change in the flexion axis of stability/stiffness at the L4 level. In the twist axis, it only resulted in a biologically significant change in the 40° and 50° flexion postures, with a 15.3% and 16.4% change in the L4 EV, respectively. In all postures, the bend axis was influenced the most, but there was only a biologically significant change in the flexion, twist, 10° bend, and 20º bend postures. In all postures, there was less than a 17% change in the L4B EV except for the 50º flexion posture where there was a 23.6% change.

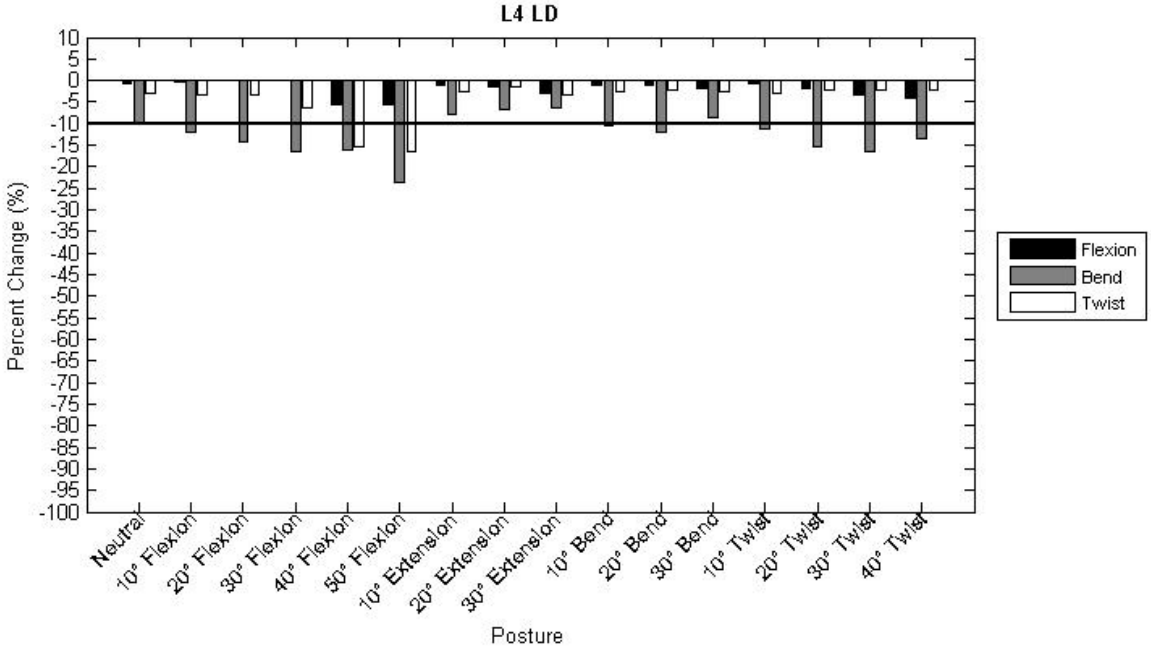

Figure 27 - Percent change in the L4 EVs when reducing the LD muscle activation to 0% MVC while all other muscles remained active to 50% MVC for all postures. Negative change represents a lower EV when the LD muscle activity was reduced to 0% MVC. The thick black line highlights the point where changes were considered biologically significant.

In summary, the major findings regarding the plane of stability at the L4 lumbar level were that muscles preferentially influence different planes of stability/stiffness, assuming that a 10% change in the EV indicated biological significance. With most postures, the erector spinae and Mult muscles most

influenced the flexion axis, while the abdominal muscles had the greatest influence on the bend axis and occasionally the twist axis.

4.1.3 Hypothesis 3: Effect of Posture on Eigenvalues

When determining if EVs were affected by posture, all postures were compared to the neutral posture for the condition where all muscles were active to 50% MVC. Similar to the previous comparisons, a change of 10% or greater between the neutral posture and posture of interest was considered biologically significant. A negative change indicated that the EV for the posture being examined was smaller than that of the neutral posture and vice versa.

Postures that were close to neutral rarely resulted in a biologically significant change in the EVs. There was no influence on any EV in the 10° flexion posture (figure 28) and the 20° flexion posture (figure 28) only had a biologically significant difference from the neutral posture for the L4F and L5B EVs, where there was a change in the EV of 10.5% and 18.1%, respectively. The 10° extension posture (figure 29) had a biologically significant effect on all the L1 EVs and the L5F EV. In all cases, the 10° extension posture resulted in less than a 17% change in the EV. The 10° bend posture (figure 30) had a small influence of 13.8% and 14.1% change on the L4T and L5B EVs, respectively. Finally, the 10° twist posture (figure 31) only had a biologically significant effect on the L5B EV, with a 10.9% change.

The EVs influenced by the remaining flexion postures were dependent on the posture (figure 28). The 30° flexion posture influenced multiple EVs, including L1F, L3F, L3B, L4F, L5F and L5B. When averaged across these EVs, there was a 19.5% ± 8.0% change for the 30° flexion posture. The 40° flexion posture resulted in a biologically significant change for majority of the EVs, only not influencing the L2F, L4B and L5T EVs. The 50° flexion posture influenced all EVs except L5T. When averaged across all EVs that resulted in a biologically significant change, there was a 25.1% ± 8.7% and 33.2% ± 10.0% change for the 40° and 50° flexion postures, respectively.

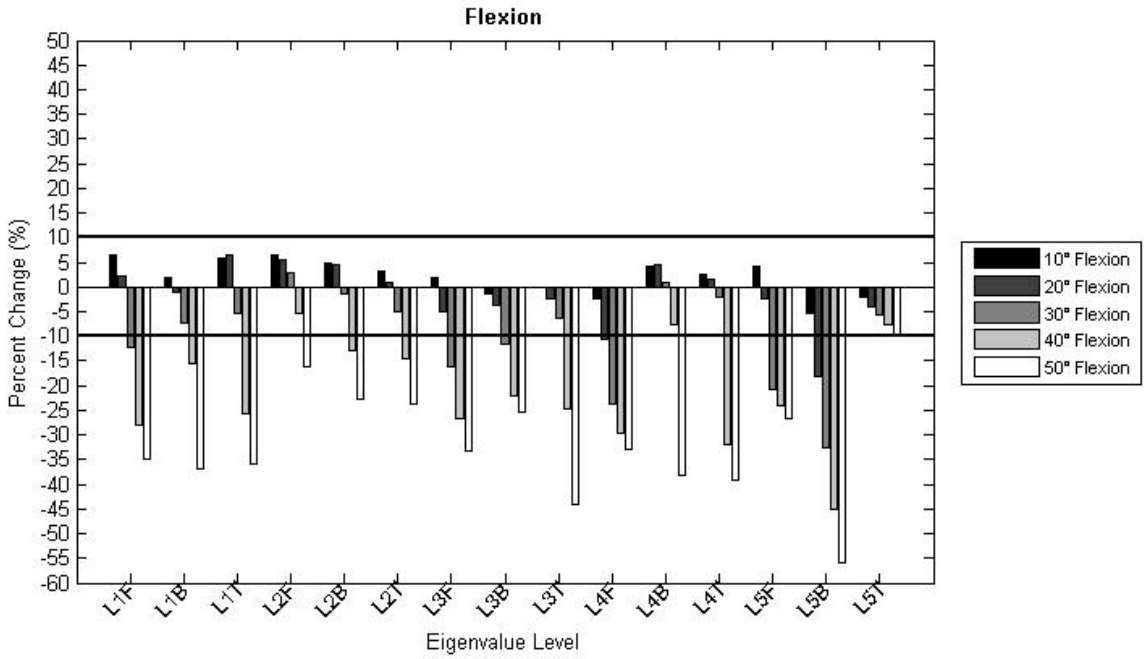

Figure 28 - Percent change of all EV levels of the flexion postures compared to the neutral posture. Negative change represents a lower EV in the measured posture. The thick black lines highlight the points where changes were considered biologically significant.

The 20° and 30° extension postures (figure 29) both caused a biologically significant effect on the majority of the EVs. The 20° extension posture had no influence on the L3T, L4F and L5T EVs, while the 30° extension posture did not influence the L5T EV. When averaged across the EVs where a biologically significant change occurred between the extension and neutral postures, there was a 23.2% ± 7.5% and 38.1% ± 11.8% change for the 20° and 30° extension postures, respectively. The 30° extension posture had the largest influence on all EVs except the L3T, L4F, L5B and L5T EVs, which were most influenced by the 50° flexion posture and the L4B EV, which was most influenced by the 30º bend posture.

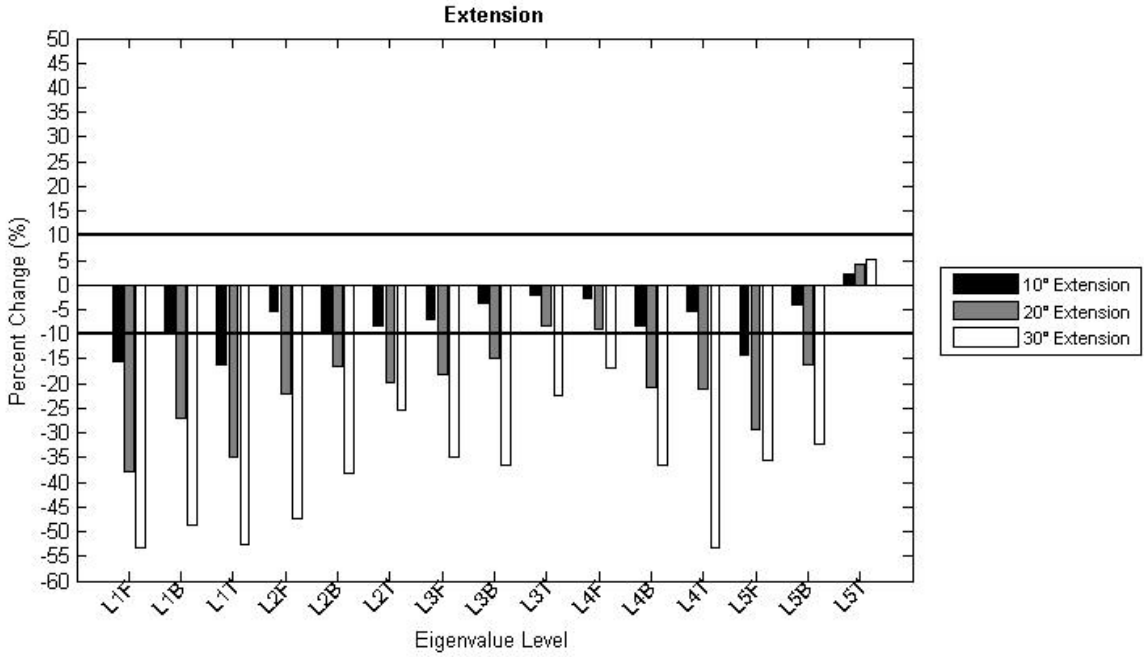

Figure 29 - Percent change of all EV levels of the extension postures compared to the neutral posture. Negative change represents a lower EV in the measured posture. The thick black lines highlight the points where changes were considered biologically significant.

The bend postures also had a biologically significant effect on multiple EVs (figure 30). The 30° bend posture influenced all EVs except L5T. The L1F EV resulted in a 35.8% change, L4B resulted in a 56.8% change, L4T resulted in a 48.0% change and the L5B eigenvalue resulted in a 39.9% change for the 30º bend posture. All other EVs had less than a 20% change for the 30° bend posture. The 20° bend posture influenced fewer EVs, only having a biologically significant effect on L1F, L3F, L4B, L4T and L5B. When averaged across these EVs, the 20° bend posture resulted in a 26.1% ± 11.7% change. The 20° bend posture also resulted in a -10.9% change for the L5T EV, indicating that the L5T EV was higher in the 20º bend posture than the neutral posture. For all EVs, the 30° bend posture had a greater influence than the 20° bend posture.

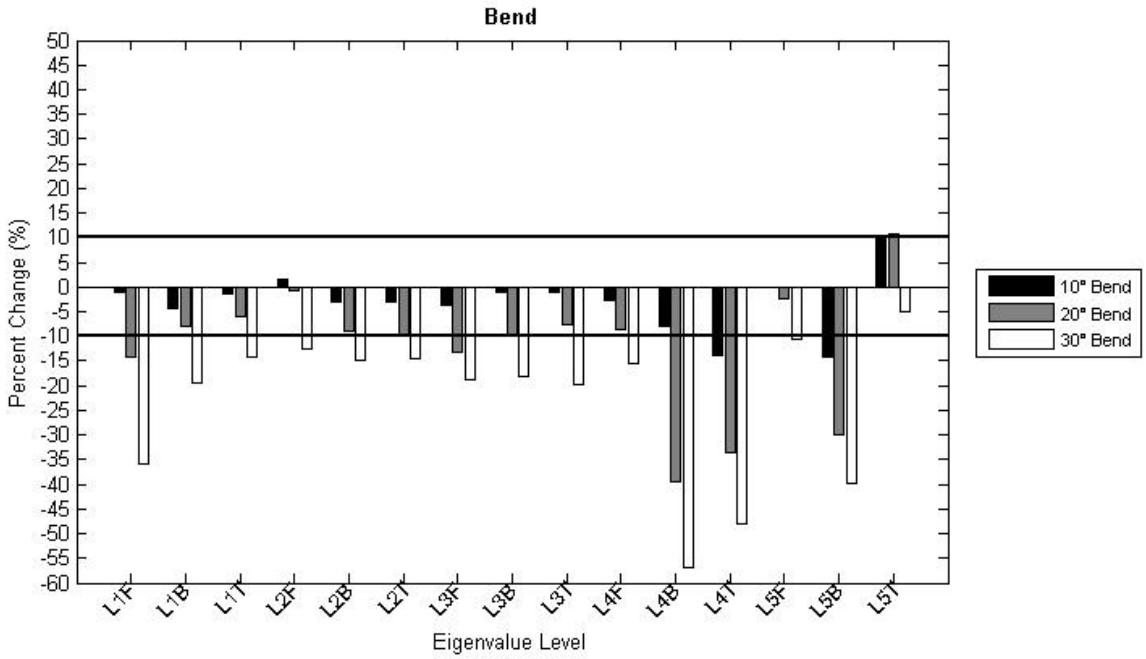

Figure 30 - Percent change of all EVs of the bend postures compared to the neutral posture. Negative change represents a lower EV in the measured posture. The thick black lines highlight the points where changes were considered biologically significant.

The remaining twist postures (figure 31) only influenced the L4B, L4T and L5B EVs, as well as the L1T EV for the 40° twist posture. For each EV, the magnitude of change increased as the degree of twist increased. The L1T EV for the 40° posture resulted in a 10.1% change. When averaged across the L4B, L4T and L5B EVs, there was a 17.5% ± 5.6% change for the 20° twist posture, 26.9% ± 2.0% change for the 30° twist posture, and a 33.8% ± 9.2% change for the 40° twist posture. All twist postures also resulted in a negative change for the L5T EV, indicating the twist postures resulted in a higher EV than the neutral posture, indicating more stability/stiffness in the L5T EV when in a twisted posture. The L5T EV resulted in changes of -11.1%, -23.2%, -31.6% and -38.9% for the 10°, 20°, 30° and 40° twist postures, respectively.

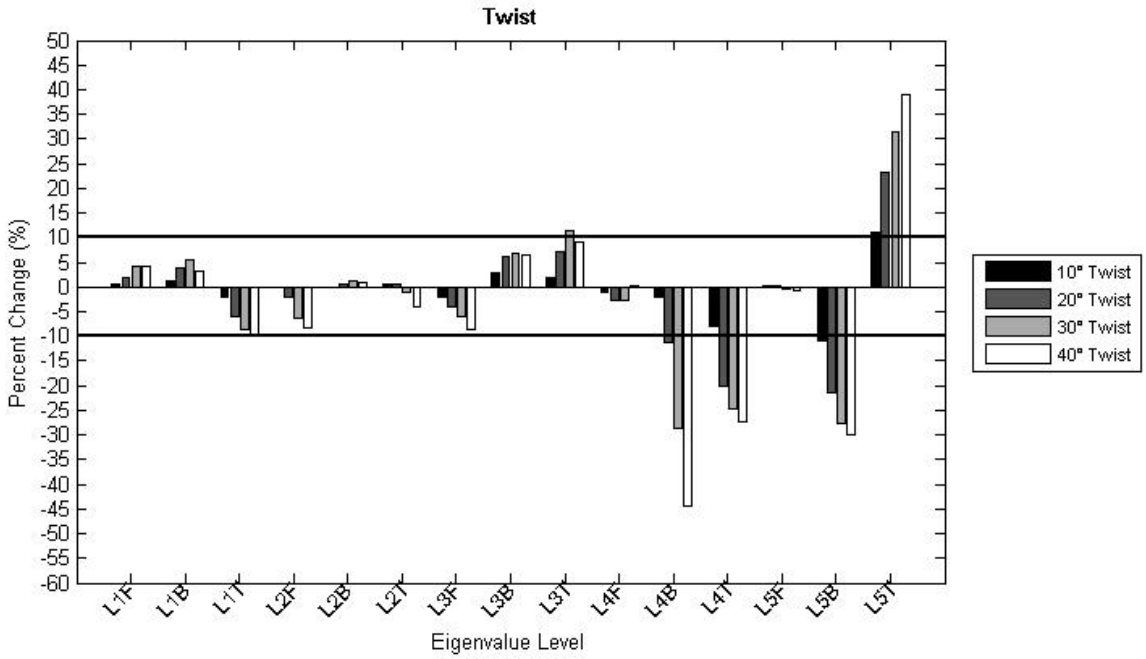

Figure 31 - Percent change of all EVs of the twist postures compared to the neutral posture. Negative change represents a lower EV in the measured posture. The thick black lines highlight the points where changes were considered biologically significant.

In summary, the major findings regarding hypothesis 3 were that postures close to neutral resulted in little change in the EVs. The postures further from neutral resulted in decreased EVs, indicating less stability/stiffness. The lowest EVs were found in the 30º extension, 50º flexion and 30º bend postures.

4.1.4 Hypothesis 4: Effect of 100% Muscle Activation on Eigenvalues

Boosting muscle activity from 50% MVC to 100% MVC, in most cases, had little effect on the EVs, although when there was a biologically significant effect, no EV increased more than 20%, except for the L1F EV with the 20° extension posture for the Mult muscle. In this situation, there was a +24.0% change. The positive change indicated that the EV was increased when muscle activation was increased

to 100% MVC. Figures displaying the effect of increasing muscle activity to 100% MVC for all EVs and all postures can be found in Appendix C.

At the L4 level, where the spine model is most robust, very few EVs were influenced by increasing a muscle activation to 100% MVC. The L4F EV resulted in a biologically significant change in the 30° flexion posture when the Mult muscle was altered. The EO muscle had a biologically significant effect on the 30° twist posture for the L4B EV. Finally, for the L4T EV, the Ilio muscle resulted in a biologically significant change for the 30° and 40° twist postures and the IO muscle resulted in a biologically significant change for the neutral posture. All other muscle and posture combinations did not have a biologically significant influence on the L4 level EVs.

4.1.5 Phase 1 Part 2: Actual Data

Four subjects performed a walking trial where they carried a 15 kg load in each hand while EMG and kinematic data were collected. The EMG and kinematic data were input into the anatomically detailed spine model and the sensitivity analysis performed in phase 1, part 1 was repeated using this actual data. This carrying task resulted in relatively low levels of muscle activation at less than 13% MVC (table 4). The averaged lumbar spine angles were $7.1^{o} \pm 1.9^{o}$ flexion, $4.0^{o} \pm 2.2^{o}$ left bend, and $4.0^{o} \pm 2.9^{o}$ right twist. There was a statistically significant interaction effect between EV levels and activation level for each muscle, as measured by repeated measures ANOVA tests. The EV level/activation level interactions that resulted in a statistically significant difference were dependent on the muscle. The p-values were only examined in detail if the EV level/activation level combination resulted in greater than a 10% change in the EV because this level was considered to be biologically significant, as described previously. Using this assumption, the TrA never resulted in a biologically significant change. For this reason, this muscle was not discussed, despite the statistically significant differences found in many EV levels. Comparisons made were 0% MVC of a specific muscle to the unaltered actual EMG pattern

(0/A), and the unaltered actual EMG pattern to 100% MVC of a specific muscle (A/100). All p-values are displayed in Appendix D.

Table 4 - EMG during the left foot stance phase of a walking trial carrying a 15 kg load in each hand (Mean ± SD).

Muscle	Right EMG (% MVC)	Left EMG (% MVC)
RA	1.23 ± 0.51	1.32 ± 0.55
EO	5.08 ± 2.98	3.77 ± 1.77
IO	12.90 ± 10.58	11.11 ± 5.07
UES	5.46 ± 5.48	3.99 ± 4.05
LES	8.85 ± 3.93	6.01 ± 2.62
LD	4.65 ± 2.44	3.92 ± 3.46

At the L1 lumbar level, altering the activation of muscles resulted in few biologically and statistically significant changes in the EVs. In every plane of stability, only the Mult muscle resulted in a biologically and statistically significant change. For the L1F EV (figure 32 A), the Mult muscle had a 58.0% change for the 0/A comparison. For the L1B EV (figure 32 B), the Mult muscle resulted in a biologically and statistically significant change for the A/100 comparison, with a 42.7% change in the EV. For the L1T EV (figure 32 C), there was a 49.5% change in the EV for the Mult muscle in the A/100 comparison. In all situations, the 0% MVC condition resulted in the smallest EV and the 100% MVC condition had the largest EV.

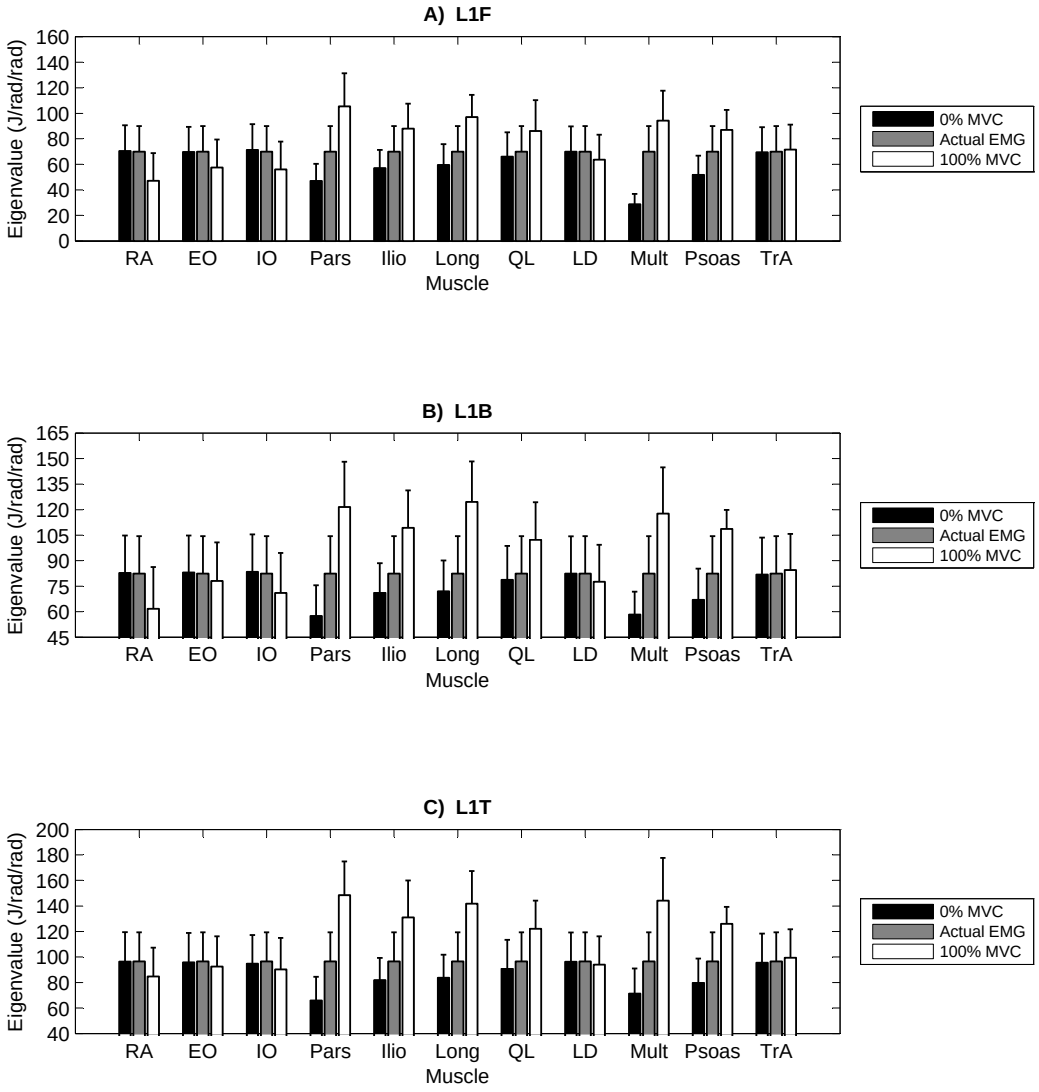

Figure 32 - (A) L1F, (B) L1B, and (C) L1T EVs calculated during the left foot stance phase of a walking trial carrying a 15 kg load in each hand using actual EMG patterns, reducing a specific muscle activation to 0% MVC and boosting the activation of a specific muscle to 100% MVC (mean ± SD).

At the L2 level, the Pars, Long, Mult and Psoas muscles were the only muscles that had a biologically and statistically significant influence on the EVs. In all planes of stability/stiffness at the L2 lumbar level, there were no statistically significant changes for the 0/A comparison. For the L2F EV (figure 33 A), the A/100 comparison resulted in changes for the Pars and Mult muscle. The Pars muscle resulted in a 65.7% change and the Mult muscle resulted in a 50.3% change in the L2F EV for the 0/A comparison. For the L2B EV (figure 33 B), there was a 77.6% change with the Pars muscle, 63.0% change with the Long muscle, 56.0% change with the Mult muscle and a 29.8% change with the Psoas muscle for the A/100 comparison. Finally, for the L2T EV (figure 33 C), the Long muscle caused an 89.3% change, the Pars muscle caused an 84.2% change the Mult muscle resulted in an 83.7% change and the Psoas muscle resulted in a 30.6% change in the EV. In all these situations, the 100% MVC condition had the largest EV.

For the L3F EV (figure 34 A), the 0/A comparison had no statistically significant differences. For the A/100 comparison, there was a biologically and statistically significant change in the EV for the Mult (168.7%), Pars (133.4%), Ilio (111.0%), Long (82.4%), and Psoas (33.8%) muscles. Similarly, for the L3B EV (figure 34 B), there were no statistically significant changes for the 0/A comparison. For the A/100 comparison, the Mult muscle resulted in a 173.5% change, the Pars muscle had a 140.3% change, the Ilio muscle caused a 95.4% change, the Long muscle resulted in a 63.9% change and the Psoas muscle had a 27.0% change in the EV, all of which were statistically significant. Finally, for the L3T EV (figure 34 C), there was a biologically and statistically significant change of 18.0% for the Mult muscle with the 0/A comparison. For the A/100 comparison, there were biologically and statistically significant changes for the Mult (148.3%), Pars (121.5%), Ilio (72.6%), Long (49.1%), QL (36.0%) and Psoas (21.0%) muscles. In all situations, the 100% MVC condition had the largest EV and the 0% MVC condition had the smallest EV.

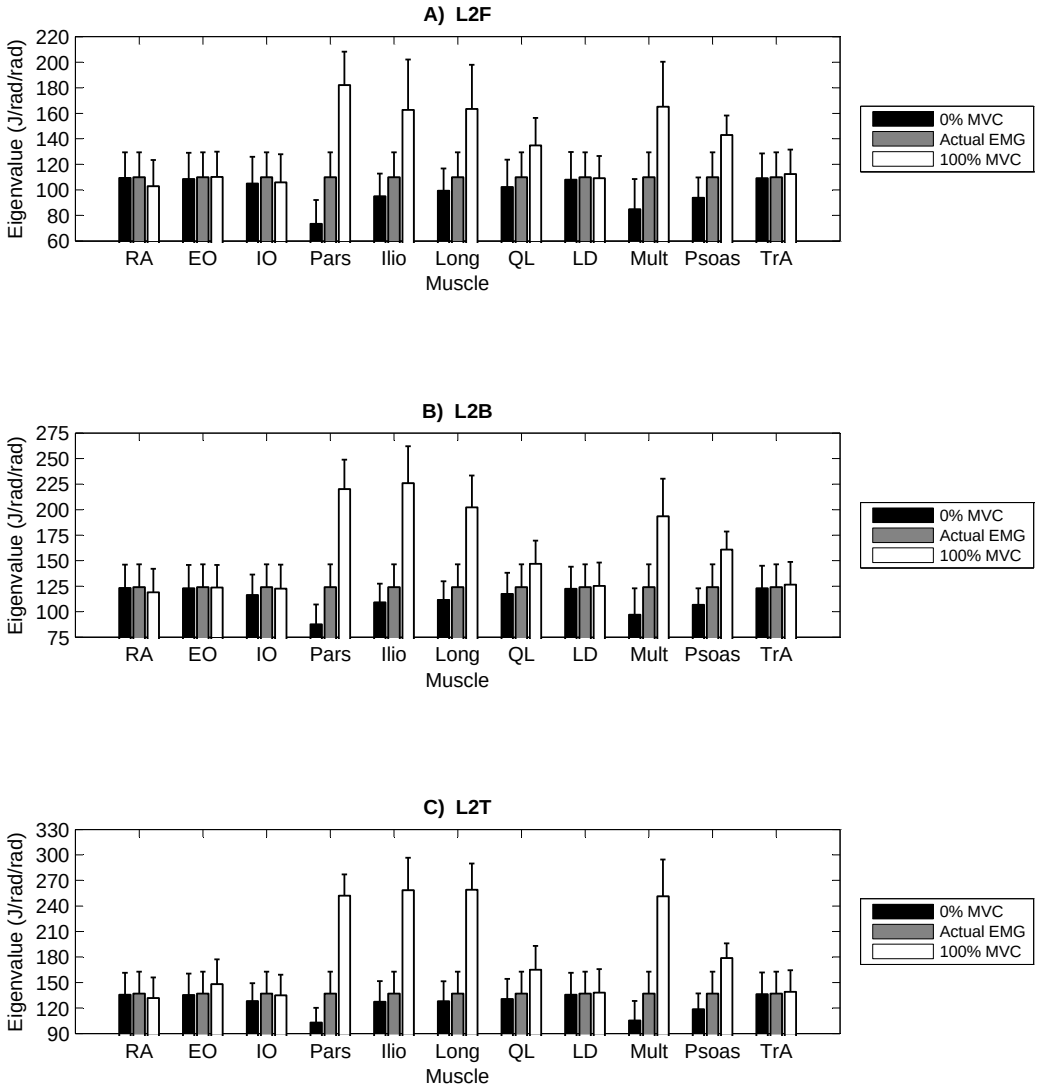

Figure 33 - (A) L2F, (B) L2B, and (C) L2T EVs calculated during the left foot stance phase of a walking trial carrying a 15 kg load in each hand using actual EMG patterns, reducing a specific muscle activation to 0% MVC and boosting the activation of a specific muscle to 100% MVC (mean ± SD).

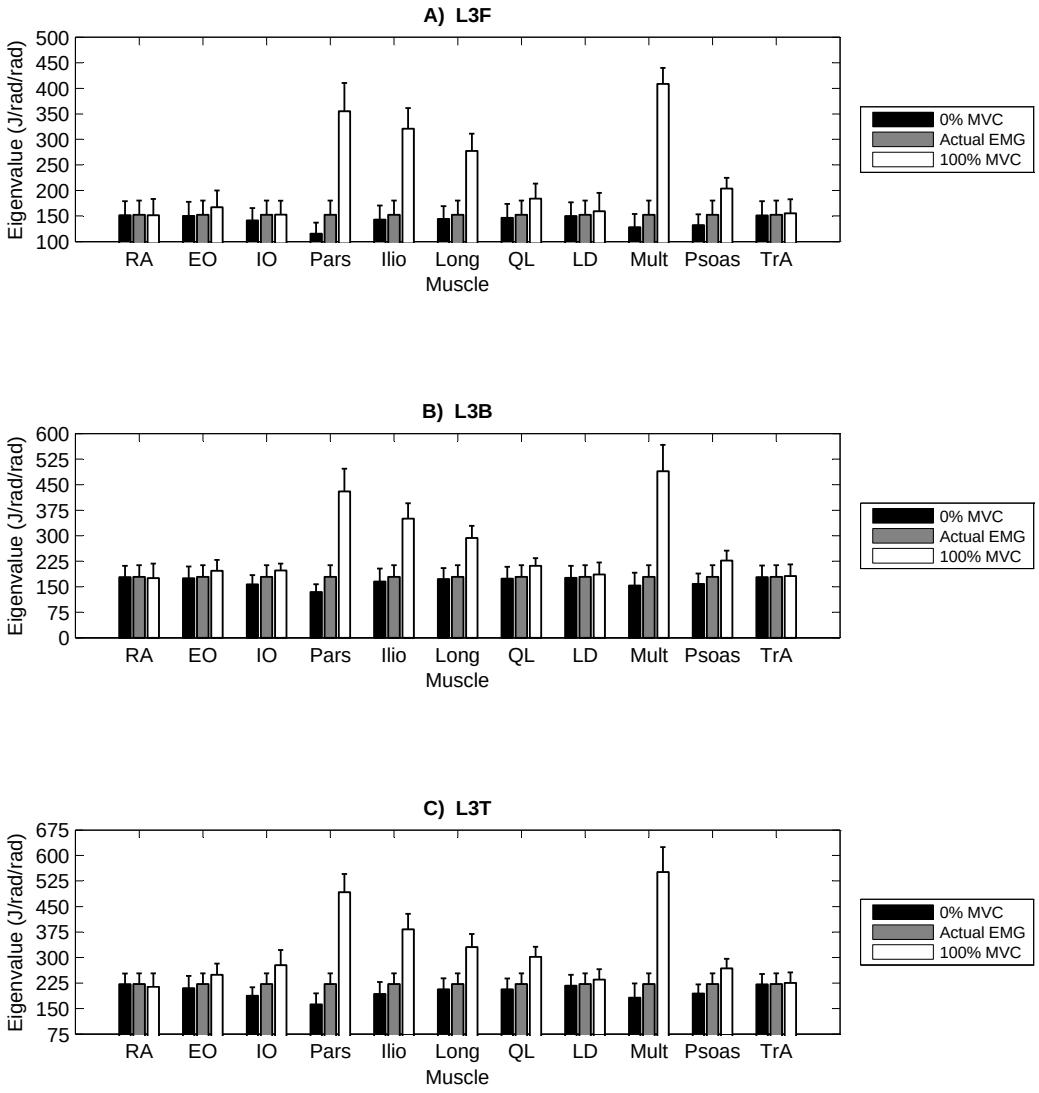

Figure 34 - (A) L3F, (B) L3B, and (C) L3T EVs calculated during the left foot stance phase of a walking trial carrying a 15 kg load in each hand using actual EMG patterns, reducing a specific muscle activation to 0% MVC and boosting the activation of a specific muscle to 100% MVC (mean ± SD).

At the L4 level, many muscles had a biologically and statistically significant influence on the EVs. For the L4F EV (figure 35 A), the 0/A comparison resulted in a biologically and statistically significant change of 14.7% for the Mult muscle. For the A/100 comparison, there was a biologically and statistically significant change in the L4F EV for the Mult (139.5%), Pars (133.2%), Ilio (60.9%), Long (42.6%), QL (36.3%), and Psoas (31.8%) muscles.

For the L4B EV (figure 35 B), there were biologically and statistically significant changes for the IO, EO, and Psoas muscles at 51.4%, 43.5% and 21.6% change in the EV, respectively, for the 0/A comparison. For the A/100 comparison, there were biologically and statistically significant differences for all muscles except for the RA, EO and IO muscles. Of the remaining muscles, the largest change was seen with the Pars muscle at 38.2% change in the L4B EV.

Finally, for the L4T EV (figure 35 C), there was a biologically and statistically significant change for the 0/A comparison with the IO and Pars muscle, with changes in the EV of 42.1% and 12.3%, respectively. There was also a statistically significant change with the Psoas muscle, but this muscle did not have a biologically significant influence on the EV. When considering the A/100 comparison with the L4T EV, there was a biologically and statistically significant change for all muscles. The muscle with the largest influences on the L4T EV were the Ilio, Pars and IO muscles, with 62.9%, 62.8% and 43.7% change in the EV, respectively. The muscle with the smallest influence was the Psoas muscle with a 17.2% change in the L4T EV. In all situations for the L4 EV, the 100% MVC condition had the largest EV and the 0% MVC condition had the smallest EV.

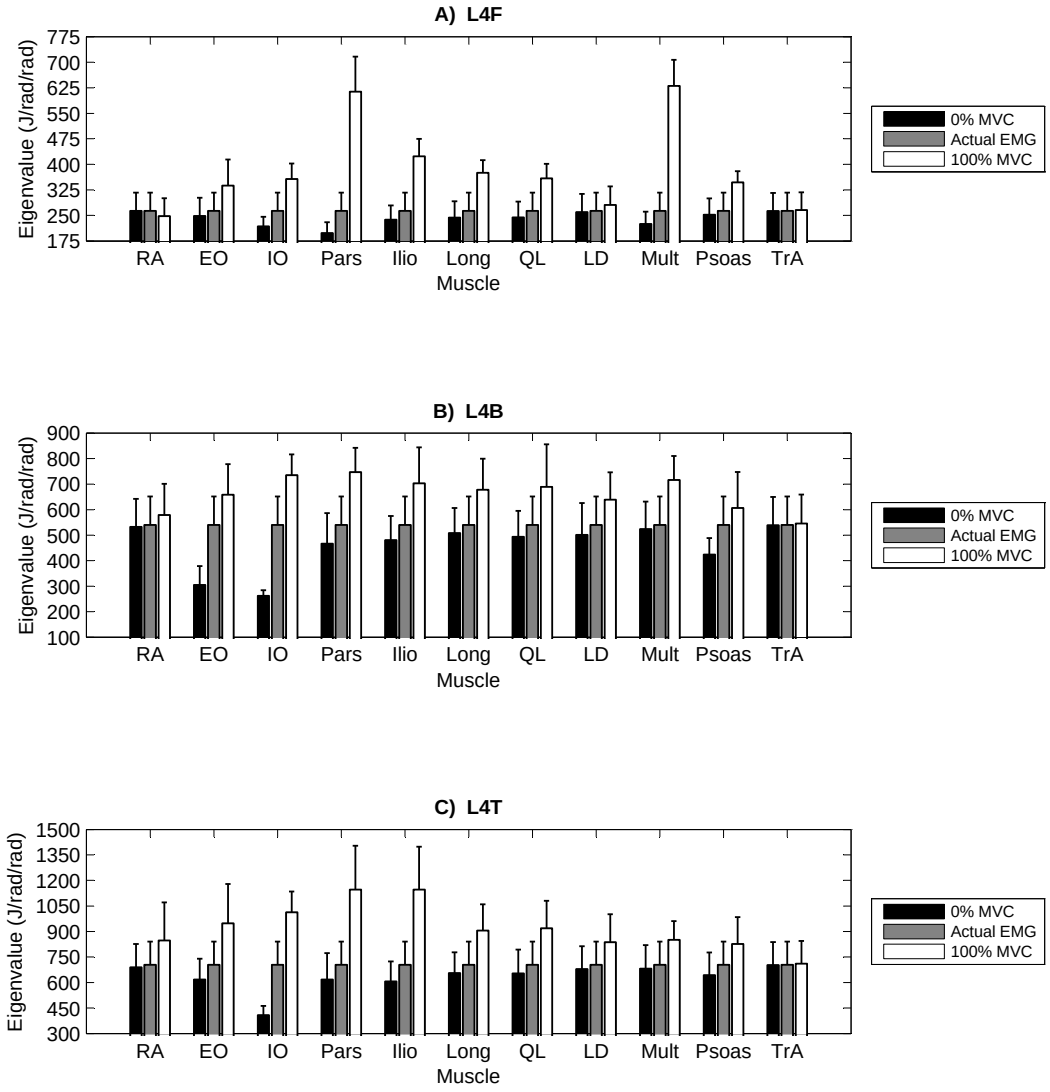

Figure 35 - (A) L4F, (B) L4B, and (C) L4T EVs calculated during the left foot stance phase of a walking trial carrying a 15 kg load in each hand using actual EMG patterns, reducing a specific muscle activation to 0% MVC and boosting the activation of a specific muscle to 100% MVC (mean ± SD).

The L5 level EVs resulted in a statistically significant change for many comparisons. For the L5F EV (figure 36 A) with the 0/A comparison, there was a statistically significant difference for the IO, Pars and Mult muscles, but the Mult muscle did not result in a biologically significant change. The IO and Pars muscles caused a 36.6% and 11.1% change in the L5F EV, respectively, when the muscle was removed. All muscles resulted in a biologically and statistically significant change for the L5F EV when considering the A/100 comparison. The Ilio, Pars and Long muscles resulted in the largest changes in the EV, at 81.4%, 55.9% and 51.7%, respectively, followed by the IO and EO muscles at 48.9% and 47.1% change in the L5F EV. The Mult muscle had the smallest influence on this EV, with a change of 22.7%.

For the L5B EV (figure 36 B), there was a statistically significant change with the IO, LD and Mult muscles for the 0/A comparison, but only the IO muscle resulted in a biologically significant change. The IO muscle caused a 42.2% change in the L5B EV when reduced to 0% MVC. For the A/100 comparison, all muscles had a biologically and statistically significant influence on the L5B EV. With this comparison, the largest changes were seen with the IO, RA and Ilio muscles, at 238.1%, 124.4%, and 111.8% change in the L5B EV, respectively. The Psoas and Mult muscles had the smallest influence on the L5B EV, with 10.2% and 13.9% change in the EV, respectively.

For the L5T EV (figure 36 C), there was a biologically and statistically significant change for the IO and EO muscles when considering the 0/A comparison, with a 39.7% and 11.2% change in the EV, respectively. The LD and QL muscles also resulted in a statistically significant difference, but not a biologically significant difference. For the A/100 comparison, there was a statistically significant change for all muscles, but the Mult and Psoas muscles did not have a biologically significant change. Of the remaining muscles, the EO, IO and RA muscles had the greatest change, at 208.8%, 206.3% and 133.7% change in the L5T EV. The smallest change was seen with the Long muscle at 19.7% change in the EV. In all situations, the 100% MVC condition had the largest EV and the 0% MVC condition ha the smallest EV.

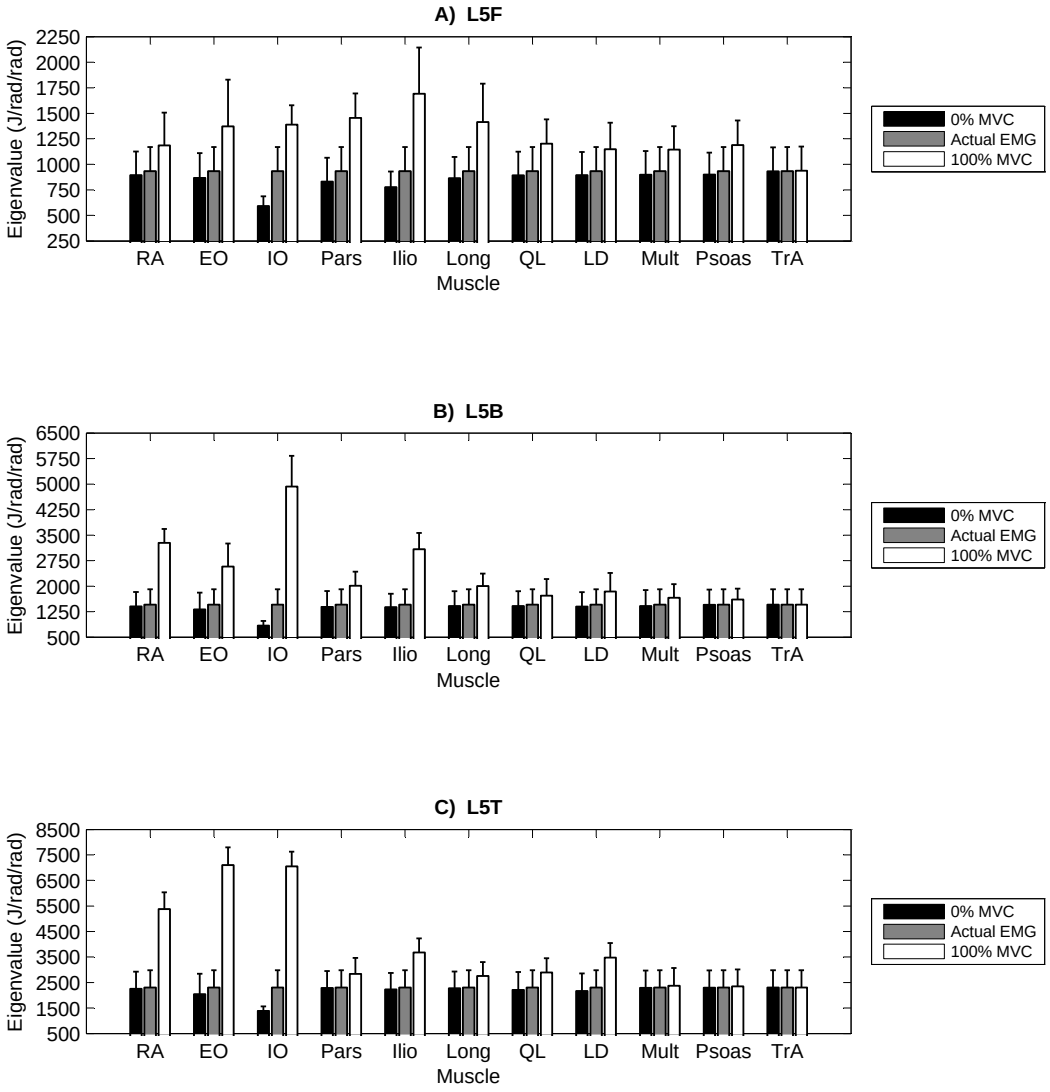

Figure 36 - (A) L5F, (B) L5B, and (C) L5T EVs calculated during the left foot stance phase of a walking trial carrying a 15 kg load in each hand using actual EMG patterns, reducing a specific muscle activation to 0% MVC and boosting the activation of a specific muscle to 100% MVC (mean ± SD).

In summary, there were few biologically and statistically significant changes when comparing the 0% MVC condition to the actual EMG condition. However, when comparing the 100% MVC condition to the actual EMG condition, the erector spinae and Mult muscles influenced most EVs, while the

abdominal muscles only influenced the L4 and L5 level EVs. In most situations, the single muscles affected all planes of stability/stiffness, but the abdominal muscles typically had a larger influence on the bend and twist axes than the flexion axis, while the erector spinae and Mult muscles typically had a greater influence on the flexion axis than the bend and twist axes.

4.2 Phase 2: Case Studies

For Phase 2 of this thesis, four case studies were analyzed to test the final hypotheses regarding the links between pain, intervention and whether these influence the EVs and other biomechanical variables. Each subject had a different pain presentation and different biomechanical reactions to the suggested interventions. For this reason, each subject will be discussed individually. Only the EVs at the L4 and L5 level were examined in detail because of the anatomical robustness included in the model at this level. Similar to phase 1, a biologically significant change was assumed to occur for any variable that changed more than 10%. The time-history plots for all trials and variables can be found in Appendix E.

4.2.1 Subject 1

Subject 1 performed the heel drop test using unbraced muscles and two interventions: an abdominal brace and stiffening with the LD muscles, as described in section 3.6 of this document. Using the unbraced pattern, this subject reported a NPS of 1. When using the abdominal brace intervention, the NPS increased to 2, while when using the LD intervention the NPS decreased to 0. In other words, the abdominal brace strategy increased pain while the shoulder muscle strategy reduced pain. The EMG patterns collected indicated that the subject performed the interventions sufficiently. The mean EMG activity of each muscle increased by more than 15% when comparing the muscle patterns from the interventions with the unbraced muscle pattern (absolute magnitudes in table 5). The abdominal brace intervention showed that the subject increased the activity of the right and left LD muscles the most, with an increase of 1655.2% from 4.9 %RVC to 85.1 %RVC and 914.1% from 7.6 %RVC to 76.6 %RVC,

respectively. The four erector spinae muscles (right and left UES and LES) increased an average of 251.6% ± 189.3%. The RA muscle activity increased the most of the abdominal muscles, with a percent change of 197.6% and 284.7% for left and right RA, respectively. The right and left IO and EO muscles increased an average of 99.2% ± 51.2% with the abdominal brace intervention. The LD muscles had the largest change in EMG for the LD intervention, increasing the muscle activity by 2849.9% from 7.6 %RVC to 222.8 %RV and 2828.8% from 4.9 %RVC to 142.1 %RVC for the left and right LD muscles, respectively. The erector spinae muscle activity increased more than with the abdominal intervention, while the abdominal muscle activity had a smaller increase. With the LD intervention, the erector spinae muscle activity increased by an average of 601.7% ± 658.2% and the abdominal muscle activity increased by an average of 54.5% ± 35.6%.

Table 5 - EMG activity for Subject 1 for the heel drop task (Mean ± SD).

Muscle	**Unbraced (%RVC)**		**Abdominal Brace (%RVC)**		**LD intervention (%RVC)**	
	Right	**Left**	**Right**	**Left**	**Right**	**Left**
LD	4.9 (2.4)	7.6 (3.4)	85.1 (42.4)	76.6 (42.3)	142.1 (27.3)	222.8 (82.6)
UES	2.4 (1.3)	5.4 (1.4)	15.4 (4.9)	12.1 (5.2)	40.2 (8.5)	34.9 (14.2)
LES	14.5 (11.9)	12.6 (8.9)	37.9 (18.1)	36.5 (12.6)	34.4 (8.0)	34.9 (7.7)
RA	13.2 (5.0)	14.2 (3.5)	50.8 (18.7)	42.4 (13.2)	28.3 (19.7)	24.8 (12.0)
EO	35.5 (10.3)	42.9 (11.9)	66.3 (17.3)	116.6 (17.6)	55.1 (14.0)	58.9 (12.4)
IO	46.3 (16.1)	33.4 (8.4)	86.1 (23.2)	50.8 (11.0)	55.2 (18.0)	42.2 (8.1)

After inputting the EMG patterns into the spine model, assuming neutral posture through the entire trial, it was found that there was an increase in the mean of all EVs, L4/L5 compression and L4/L5 anterior to posterior (AP) shear for both interventions compared to the unbraced condition. For the EVs, the L4 EVs were influenced more than the L5 EVs, with the twist axis being the most influenced for both

the abdominal and LD interventions (figure 37). The L4 and L5F EVs increased more with the LD intervention than the abdominal intervention, while the opposite occurred for the L5B and L5T EVs. The mean L4T EV increased 92.3% from 1496.1 J/rad^2 to 2877.3 J/rad^2 for the abdominal intervention, and increased 129.0% from 1496.1 J/rad^2 to 3426.8 J/rad^2 for the LD intervention. All other EVs increased to a lesser degree.

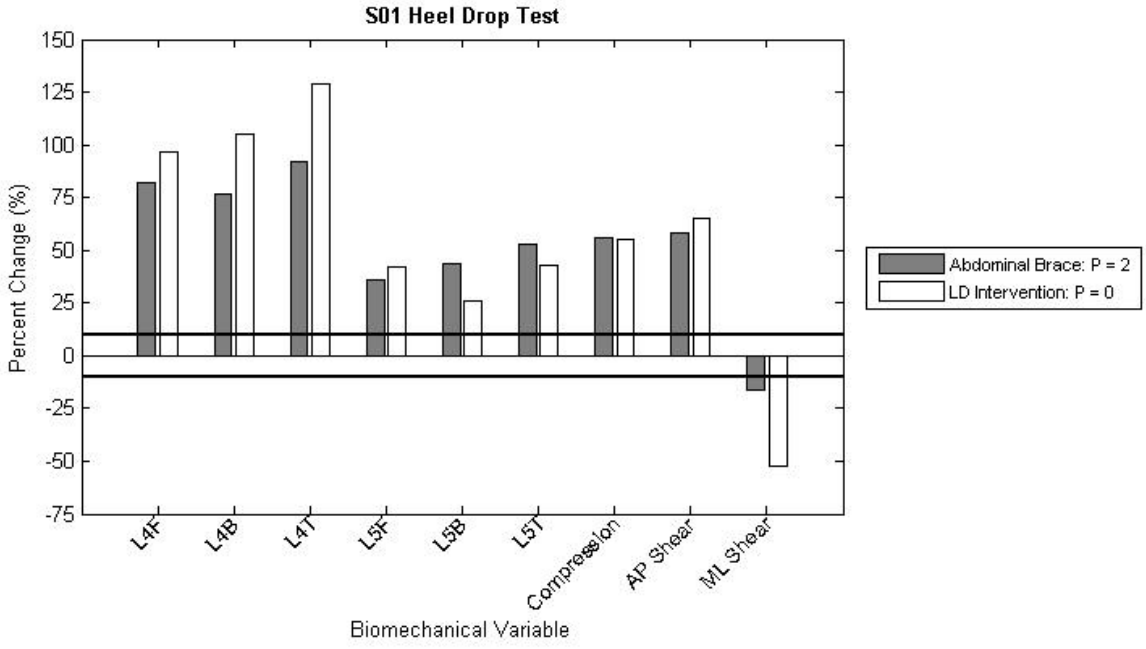

Figure 37 - Percent change of the L4 and L5 level EVs, L4/L5 compression, L4/L5 AP shear and L4/L5 ML shear between the unbraced condition and the applied interventions for the heel drop task for Subject 1. Positive change represents a higher magnitude of the biomechanical variable when using the intervention. The thick black lines highlight the points where changes were considered biologically significant.

Mean L4/L5 compression did not change between interventions, but did increase compared to the unbraced muscle condition, resulting in a 56.1% increase from -3372.8 N to -5264.0 N for the abdominal intervention and a 54.9% increase from -3372.8 N to -5226.1 N for the LD intervention (figure 37). The mean L4/L5 AP shear also increased when using an intervention. Using the abdominal intervention, it

increased 58.3% from -459.3 N to -727.0 N, while using the LD intervention the mean AP shear increased 65.1% from -459.3 N to -758.2 N (figure 37). However, the mean medial to lateral (ML) shear decreased 16.0% from 55.6 N to 46.7 N for the abdominal intervention and 52.1% from 55.6 N to 26.7 N for the LD intervention (figure 37).

In summary, using the unbraced condition as a base, both the abdominal strategy, which increased pain, and the LD stiffening strategy, which reduced pain, were linked with increased EVs, L4/L5 compression, and L4/L5 AP shear and decreased L4/L5 ML shear, although the pain changes were not clinically significant. However, the mean L4/L5 AP shear increased more and the mean L4/L5 ML shear decreased more with the LD stiffening strategy than the abdominal brace strategy. For Subject 1, the L4 level EVs were the most influenced variables by the suggested interventions.

4.2.2 Subject 2

Subject 2 performed three tasks: the heel drop test, a squat task, and lifting a 45 lb bar from a height of 45 cm with the tasks and interventions used described in section 3.6 of this document. For the squat task, the subject reported an NPS of 4 when using his initial and unbraced pattern and an NPS of 3 when using the LD intervention. The mean EMG activity showed that all muscles had increased activation (table 6). The right and left LD muscles increased 94.7% from 11.0 %RVC to 21.4 %RVC and 176.5% from 17.6 %RVC to 48.7 %RVC, respectively. The mean EMG activity of the abdominal muscles increased by an average of 62.0% ± 29.2% and the mean EMG activity of the erector spinae muscles increased by an average of 54.7% ± 32.2%.

Table 6 - EMG activity for Subject 2 for the squat task (Mean ± SD).

Muscle	Unbraced (%RVC)		LD intervention (%RVC)	
	Right	Left	Right	Left
LD	11.0 (4.1)	17.6 (8.0)	21.4 (5.3)	48.7 (12.1)
UES	43.3 (20.4)	23.6 (12.7)	72.6 (28.0)	45.8 (15.1)
LES	44.4 (21.6)	48.9 (17.1)	57.8 (21.1)	62.0 (21.0)
RA	32.0 (6.0)	31.6 (6.8)	43.8 (11.8)	42.0 (9.8)
EO	59.0 (9.8)	48.7 (10.8)	116.8 (27.8)	94.0 (30.5)
IO	43.5 (17.8)	44.1 (11.4)	61.1 (15.9)	75.2 (14.5)

The mean EVs at the L4 and L5 level all increased when using the LD intervention (figure 38). On average, the L4 eigenvalues increased by 28.2% ± 5.3% and the L5 eigenvalues increased by 26.9% ± 2.0%. The largest change was seen in the L4T EV at a 31.6% increase from 1975.4 J/rad^2 to 2599.7 J/rad^2. The mean L4/L5 compression and shear also increased (figure 38), with the compression increasing 22.8% from -3590.4 N to -4409.2 N when using the LD intervention. Mean L4/L5 AP shear increased 12.6% from -786.6 N to -886.0 N and mean L4/L5 ML shear increased 76.8% from 81.7 N to 144.5 N. Finally, the lumbar flexion angle decreased 28.6% from -11.5º to -8.2º and the excursion of the lumbar flexion angle decreased 13.6% from 24.3° to 21.0° when using the intervention (figure 38).

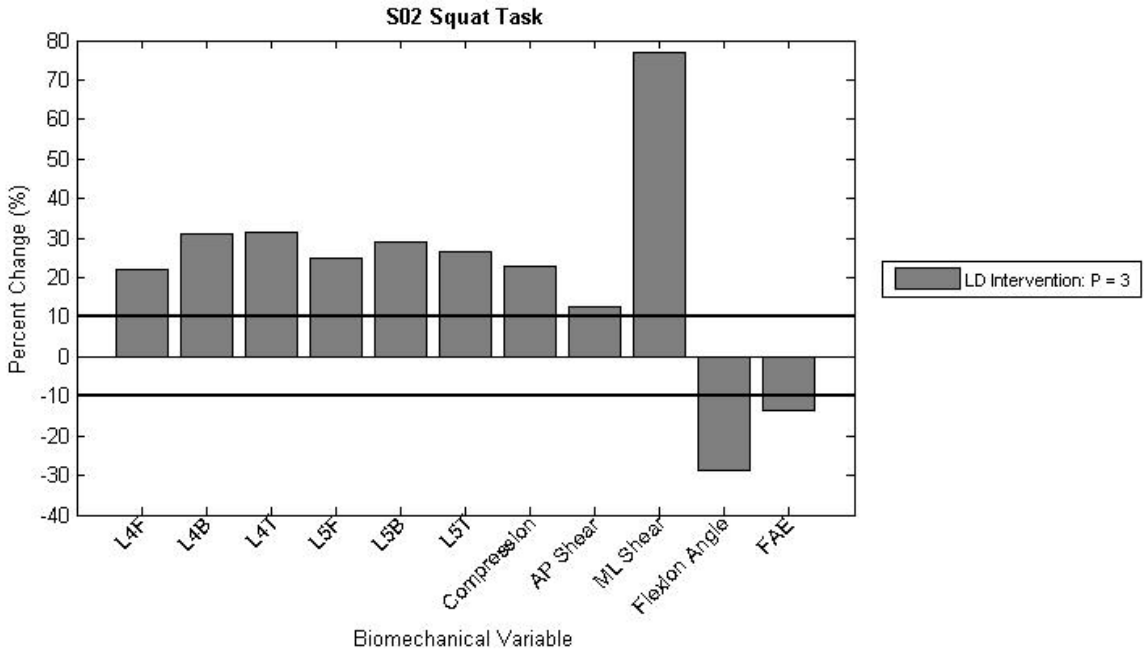

Figure 38 - Percent change of the L4 and L5 level EVs, L4/L5 compression, L4/L5 AP shear, L4/L5 ML shear, lumbar flexion angle and flexion angle excursion (FAE) between the unbraced condition and the LD intervention for the squat task for Subject 2. Positive change represents a higher magnitude of the biomechanical variable when using the intervention. The thick black lines highlight the points where changes were considered biologically significant.

For the lift bar task, the subject reported an NPS of 5 using the initial and unbraced pattern and an NPS of 3 when using the hip hinge plus LD intervention. The mean EMG activity indicated that all muscles had a higher activation when using the intervention except for the LLES muscle, which had less activation, and the RIO and RLES muscles, which did not have a biologically significant change in muscle activation (table 7). The left EO muscle had the largest change in muscle activation at a 140.3% increase from 35.3 %RVC to 84.9 %RVC, while the remaining muscle activations increased in the range of 35% to 113%, with the smallest change seen with the RLD muscle.

Table 7 - EMG activity for Subject 2 for the lift bar task (Mean ± SD).

Muscle	Unbraced (%RVC)		Hip Hinge + LD intervention (%RVC)	
	Right	Left	Right	Left
LD	23.7 (13.7)	50.5 (33.1)	32.0 (21.2)	72.3 (47.0)
UES	32.0 (18.7)	29.2 (21.7)	68.0 (36.9)	54.4 (32.4)
LES	58.2 (32.5)	76.8 (32.6)	60.0 (35.4)	64.1 (25.5)
RA	32.6 (14.9)	30.0 (13.0)	44.7 (26.2)	42.7 (22.7)
EO	54.1 (17.9)	35.3 (12.8)	88.6 (57.8)	84.9 (59.6)
IO	47.3 (11.6)	41.8 (19.0)	44.8 (43.6)	62.6 (57.6)

For the lift bar task, the mean EVs at the L4 and L5 level all increased when using the hip hinge + LD intervention (figure 39), with the L5F EV having the greatest change with a 46.0% increase from 2048.4 J/rad^2 to 2990.5 J/rad^2. The remaining L4 and L5 level EVs increased in the range of 17% to 29%. Mean L4/L5 compression, AP shear, and ML shear also increased when using the hip hinge + LD intervention (figure 39). The mean compression increased 20.9% from -3399.9 N to -4111.3 N, mean AP shear increased 14.8% from -921.2 N to -1057.2 N, and mean ML shear increased 141.5% from 50.8 N to 122.7 N. Finally, the mean lumbar flexion angle did not have a biologically significant change and the excursion of the flexion lumbar spine angle was 41.0° using the subject's initial pattern and 35.7° using the hip hinge + LD intervention, indicating a 13.0% decrease when using the intervention (figure 39).

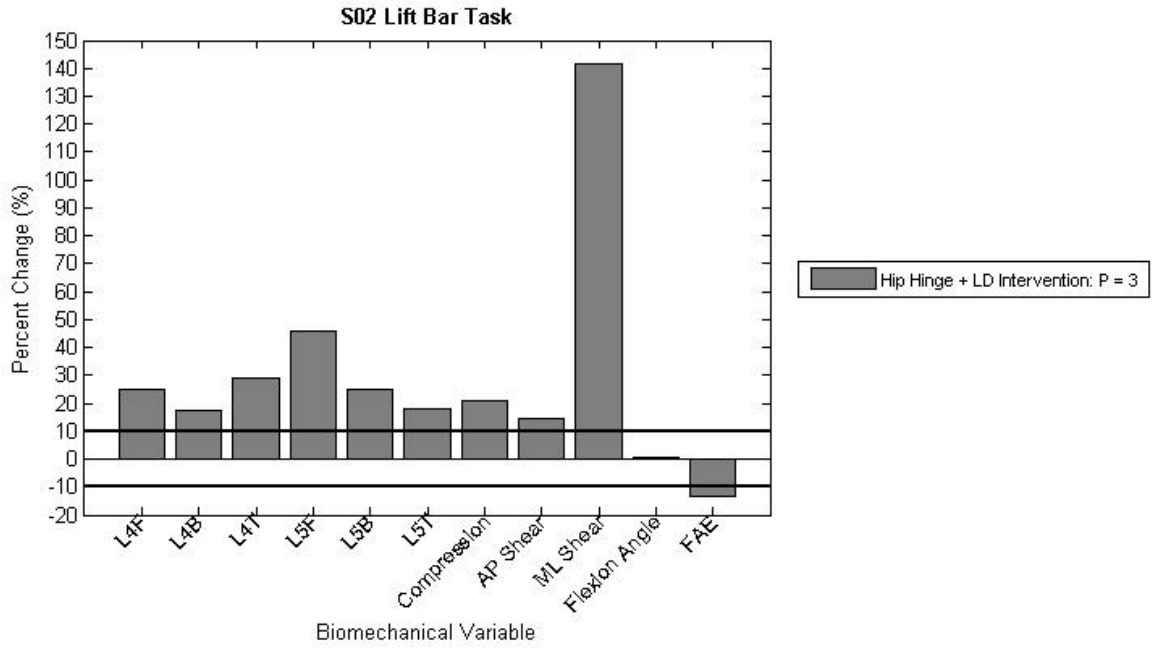

Figure 39 - Percent change of the L4 and L5 level EVs, L4/L5 compression, L4/L5 AP shear, L4/L5 ML shear, lumbar flexion angle and flexion angle excursion (FAE) between the unbraced condition and the hip hinge + LD intervention for the lift bar task for Subject 2. Positive change represents a higher magnitude of the biomechanical variable when using the intervention. The thick black lines highlight the points where changes were considered biologically significant.

For the heel drop task, the subject reported an NPS of 5 for the unbraced condition. For any given intervention, the NPS score decreased, reported at 4, 2, 2 and 1 for the mild brace, robust brace, LD, and brace + LD interventions, respectively. With every intervention, the mean EMG activity of all muscles increased compared to the unbraced condition (table 8). There was an average increase of 58.4% ± 26.8% in the abdominal muscle activity for the mild brace condition, while for the robust brace condition the mean abdominal muscle activity increased by 304.9% ± 119.1%. For the erector spinae muscle activity, there was an average increase of 51.8% ± 31.0% for the mild brace condition and an average increase of 219.1% ± 94.6% for the robust brace condition. For the right and left LD EMG activity, there was an average increase of 446.8% ± 222.8% for the mild brace condition and an average

increase of 1643.7% ± 730.8% for the robust brace condition. For the LD condition, the mean abdominal muscle activity increased 212.3% ± 26.0%, the mean erector spinae muscle activity increased 283.4% ± 263.2% and the mean LD muscle activity increased 2516.3% ± 1180.8%. Lastly, for the brace + LD condition, the mean abdominal EMG increased 107.2% ± 40.6%, the mean erector spinae EMG increased 239.9% ± 194.8% and the mean LD EMG increased 2112.3% ± 867.0%.

Table 8 - EMG activity for Subject 2 for the heel drop task (Mean ± SD).

Muscle	**Unbraced (%RVC)**		**Mild Brace (%RVC)**		**Robust Brace (%RVC)**		**LD Intervention (%RVC)**		**Brace + LD (%RVC)**	
	Right	**Left**	**Right**	**Left**	**Right**	**Left**	**Right**	**Left**	**Right**	**Left**
LD	3.6 (2.5)	4.8 (2.5)	14.2 (2.2)	33.5 (3.9)	44.7 (12.7)	107.5 (66.0)	64.8 (7.8)	164.1 (17.7)	58.2 (8.2)	134.3 (14.7)
UES	7.9 (4.0)	4.1 (2.0)	14.6 (4.9)	6.7 (1.6)	28.8 (8.5)	16.7 (10.2)	34.8 (6.6)	30.1 (4.1)	37.6 (5.6)	22.1 (2.7)
LES	4.8 (3.1)	15.5 (5.2)	7.2 (3.1)	17.2 (3.6)	15.3 (3.1)	29.2 (4.8)	9.4 (1.9)	25.8 (5.8)	9.7 (4.8)	22.6 (7.5)
RA	19.2 (7.2)	20.9 (9.4)	24.7 (5.3)	25.9 (7.7)	71.5 (51.7)	103.2 (77.6)	50.2 (7.0)	69.1 (19.7)	39.3 (9.7)	53.8 (19.6)
EO	33.5 (16.8)	36.8 (30.5)	61.0 (8.1)	68.2 (20.5)	121.3 (79.9)	221.2 (236.9)	106.7 (24.6)	118.4 (71.4)	76.7 (26.9)	85.1 (95.4)
IO	40.9 (20.5)	31.6 (7.6)	70.7 (13.6)	49.8 (6.3)	121.8 (80.3)	95.5 (79.9)	134.2 (27.5)	99.1 (21.5)	61.1 (21.5)	54.0 (26.2)

For the heel drop task, the mean L4 and L5 EVs increased in all situations (figure 40). The L4 EVs increased an average of 48.4% ± 1.6%, 107.8% ± 12.1%, 120.7% ± 17.2% and 105.4% ± 29.1% for the mild brace, robust brace, LD and brace + LD interventions, respectively. The L5 EVs increased an average of 42.4% ± 4.6% for the mild brace condition, 78.2% ± 11.0% for the robust brace condition, 83.6% ± 8.8% for the LD condition and 55.2% ± 6.6% for the brace + LD condition. The largest change was always seen in the L4T EV and the smallest change was always seen in the L5T EV.

The mean L4/L5 compression increased when using all heel drop test interventions (figure 40). For the mild brace condition, there was a 40.5% increase from -2142.0 N to -3009.1 N and there was an 85.2% increase from -2142.0 N to -3967.7 N for the robust brace condition. For the LD condition, compression increased by 87.0% from -2142.0 N to -4005.7 N and for the brace + LD condition it increased by 62.7% from -2142.0 N to -3484.1 N. The L4/L5 AP shear resulted in increases of 29.9% from -296.1 N to -371.8 N for the mild brace condition, 58.1% from -286.1 N to -452.5 N for the robust brace condition, 27.6% from -286.1 N to -365.0 N for the LD condition and 70.8% from -286.1 N to -488.8 for the brace + LD conditions (figure 40). L4/L5 ML shear resulted in the largest increase of the measured biomechanical variables, excluding the mean lumbar flexion angle (figure 40), resulting in a 291.3% increase from -16.5 N to 31.6 N for the mild brace condition and a 601.4% increase from -16.5 N to 82.8 N for the robust brace condition. For the LD and brace + LD conditions there was an increase of 431.0% from -16.5 N to 54.7 N and 388.2% from -16.5 N to 47.6 N, respectively, for ML shear. The mean lumbar flexion angle increased from -0.005º in the unbraced condition to 0.4º with the mild brace, 2.5º with the robust brace, 5.9º with the LD intervention and -2.3º with the brace + LD intervention, thus resulting in a very large percent change between the unbraced condition and the other interventions. The lumbar flexion angle excursion resulted in a decrease in the range of 52% to 67% for the various interventions (figure 40). With the unguarded condition, the flexion angle excursion was 8.62°, while for the mild brace and robust brace condition the excursion was 3.80° and 3.58°, respectively. For the LD

condition, the subject had a flexion angle excursion of 2.89° and for the brace + LD condition, there was an excursion of 4.13° in the lumbar flexion angle.

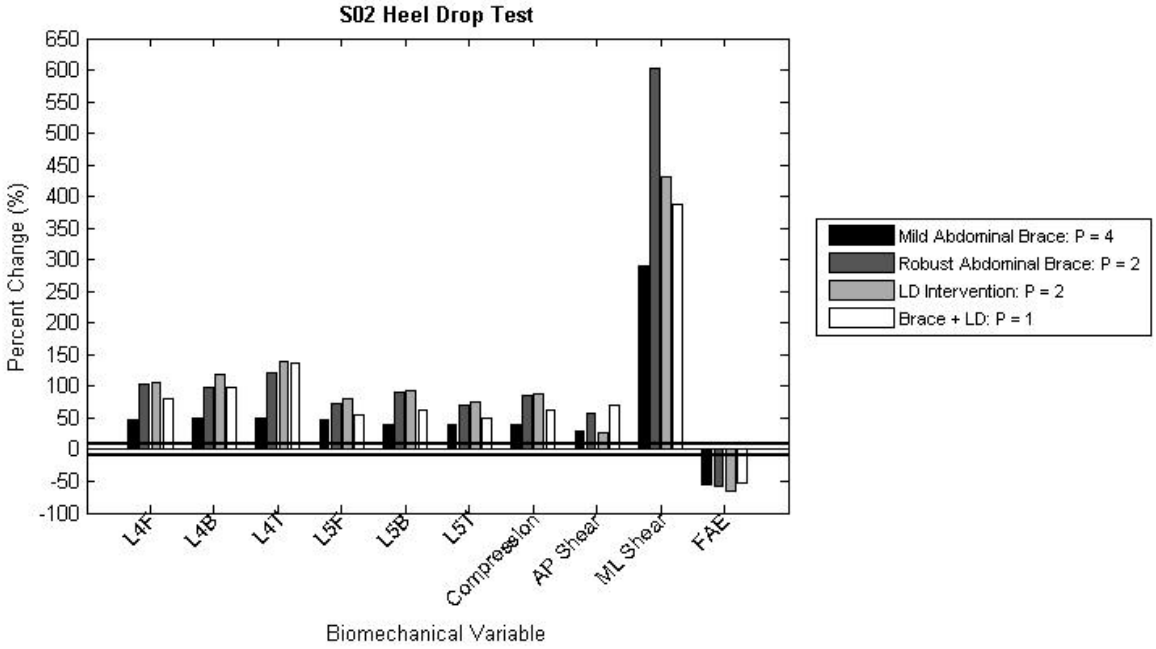

Figure 40 - Percent change of the L4 and L5 level EVs, L4/L5 compression, L4/L5 AP shear, L4/L5 ML shear and flexion angle excursion (FAE) between the unbraced condition and the various interventions tested for the heel drop test for Subject 2. Positive change represents a higher magnitude of the biomechanical variable when using the intervention. The thick black lines highlight the points where changes were considered biologically significant.

In summary, using the unbraced condition as a base, the interventions, which decreased pain, caused a biologically significant change in all measured biomechanical variables. The L4 and L5 level EVs, L4/L5 compression and L4/L5 ML and AP shear increased, while the flexion angle excursion decreased. In all situations for Subject 2, excluding the mean lumbar flexion angle, the L4/L5 ML shear was the most influenced by the suggested intervention.

4.2.3 Subject 3

Subject 3, a volleyball player with Olympic experience, performed four tasks: 1) heel drop test, 2) squat, 3) jump from a stool, and 4) one-step approach spike, as described in section 3.6. The subject performed the heel drop test while unbraced and when using an abdominal brace, reporting NPS scores of 5 and 0, respectively. The mean EMG activity indicated that the abdominal brace intervention did increase the abdominal muscle activity (table 9). Of the abdominal muscles, the left and right RA muscle activities were influenced the least, at an increase of 15.1% and 51.9% for left and right, respectively. However, the oblique muscle activity increased by 120.4%, 223.7%, 224.6% and 540.6% for RIO, REO, LIO and LEO, respectively. The RLD muscle activity also increased by 244.7% and the LLES muscle activity increased by 17.6%, while the muscle activity of the RUES and LUES muscles had a decrease in mean EMG activity by 58.8% and 65.9%, respectively (table 9). The muscle activity of the RLES and LLD muscles did not have a biologically significant change when using the intervention (table 9).

Table 9 - EMG activity for Subject 3 for the heel drop task (Mean ± SD).

Muscle	Unbraced (%RVC)		Abdominal Brace (% RVC)	
	Right	Left	Right	Left
LD	2.7 (0.7)	3.6 (0.7)	9.2 (7.3)	3.3 (1.8)
UES	12.8 (2.1)	11.5 (2.1)	5.3 (4.4)	3.9 (1.8)
LES	5.3 (1.2)	3.1 (0.8)	7.4 (4.4)	3.7 (1.2)
RA	26.4 (7.6)	47.8 (4.6)	40.1 (12.2)	55.1 (1.7)
EO	4.5 (3.8)	4.5 (4.9)	14.6 (5.0)	28.7 (8.2)
IO	28.6 (22.9)	17.5 (10.7)	63.1 (30.9)	56.9 (21.6)

For the heel drop task, the EVs at the L4 and L5 level all resulted in a significant increase when using the abdominal brace intervention except for the L4T EV, with the L5 EVs increasing more than the

L4 EVs (figure 41). The L4F and L4B EVs increased by 13.4% and 14.9%, respectively, when using the intervention. The L5F, L5B and L5T EVs increased by 54.0%, 65.6% and 44.1%, respectively. The mean L4/L5 compression increased 37.8% from -1326.2 N to -1828.2 N when using the abdominal brace (figure 41). Mean L4/L5 AP shear increased 40.7% from -195.3 N to -274.9 N, while mean L4/L5 ML shear decreased 33.4% from 46.1 N to 30.6 N when using the intervention (figure 41). The mean lumbar flexion angle decreased 272.9% (figure 41) from 1.07° to -1.86°, indicating the subject moved into more flexion. The flexion angle excursion increased 37.5% (figure 41) from 1.84° to 2.53°.

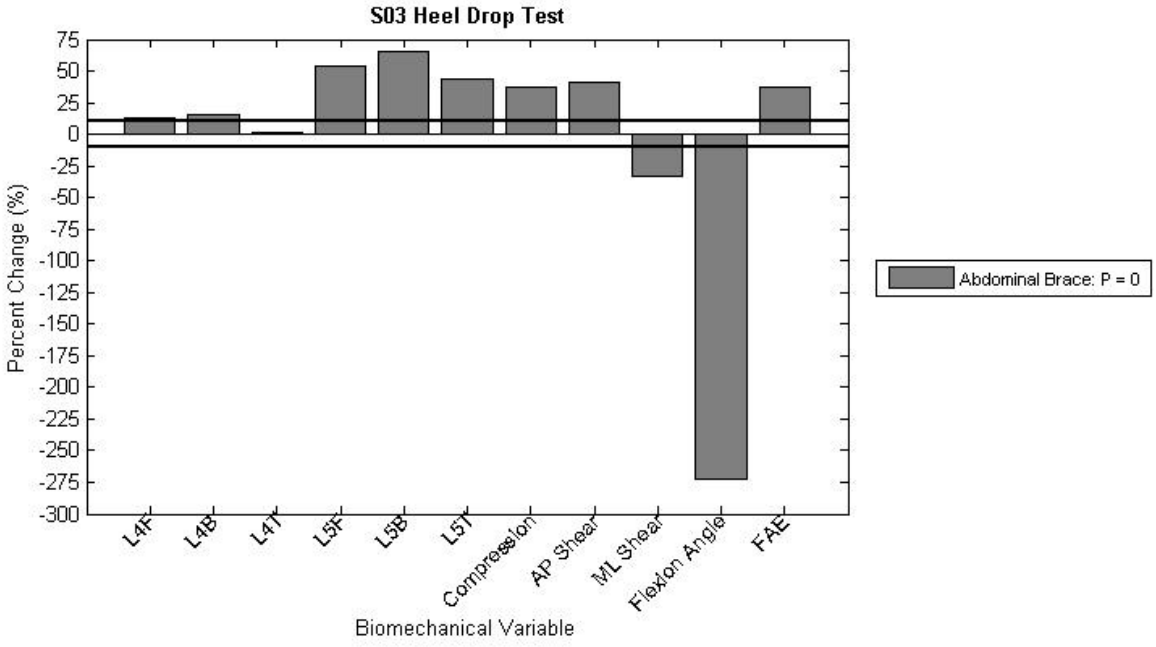

Figure 41 - Percent change of the L4 and L5 level EVs, L4/L5 compression, L4/L5 AP shear, L4/L5 ML shear, lumbar flexion angle and flexion angle excursion (FAE) between the unbraced condition and the abdominal brace intervention for the heel drop test for Subject 3. Positive change represents a higher magnitude of the biomechanical variable when using the intervention. The thick black lines highlight the points where changes were considered biologically significant.

For the squat task, Subject 3 reported an NPS of 6 when performing the task with a slouch in the initial and unbraced pattern. Using the hip hinge with LD activation, the reported NPS score was 0. The

mean EMG activity showed that the LRA, RIO and RLES muscle activity did have a biologically significant change, but the activity for the remainder of the muscles increased (table 10). The RLD and LLD muscle activity increased the most, showing a 435.5% increase from 23.9 %RVC to 127.9 %RVC and 664.4% increase from 14.0 %RVC to 107.3 %RVC, respectively. The RUES and LUES muscle activity increased 240.0% and 192.8%, respectively, followed by the LEO muscle activity at a 145.3% increase. The LIO, RRA, REO and LLES muscle activity increased in the range of 15% to 92%.

Table 10 - EMG activity for Subject 3 for the squat task (Mean ± SD).

Muscle	**Unbraced (%RVC)**		**Hip Hinge + LD (% RVC)**	
	Right	**Left**	**Right**	**Left**
LD	23.9 (17.4)	14.0 (9.4)	127.9 (125.3)	107.3 (100.1)
UES	22.9 (11.1)	18.1 (13.4)	77.9 (46.7)	53.0 (26.4)
LES	36.9 (9.1)	35.4 (8.6)	40.2 (12.2)	41.1 (16.6)
RA	28.8 (5.0)	49.9 (4.9)	55.0 (24.7)	50.2 (5.8)
EO	13.4 (4.1)	10.5 (3.7)	17.3 (18.5)	25.8 (25.9)
IO	35.7 (5.7)	29.0 (5.0)	36.3 (19.6)	38.1 (24.0)

The L4 and L5 level EVs all increased when using the hip hinge + LD intervention for the squat task (figure 42). The largest change was seen in the L5F EV with a 66.7% increase from 1502.7 J/rad^2 to 2504.8 J/rad^2, while the remaining L4 and L5 level EVs increased in the range of 26% to 34%. The mean L4/L5 compression and ML shear also increased by 27.2% from -2225.8 N to -2832.0 N and 120.2% from 7.9 N to 17.5 N, respectively (figure 42). However, the mean L4/L5 AP shear decreased 12.6% from -590.3 N to -516.1 N (figure 42). The lumbar flexion angle also decreased 76.6% (figure 42) from -15.7º to -3.7º when using the hip hinge intervention. Finally, the lumbar flexion angle excursion decreased 69.4% (figure 42) from 18.9° to 5.8°, moving toward a more neutral posture.

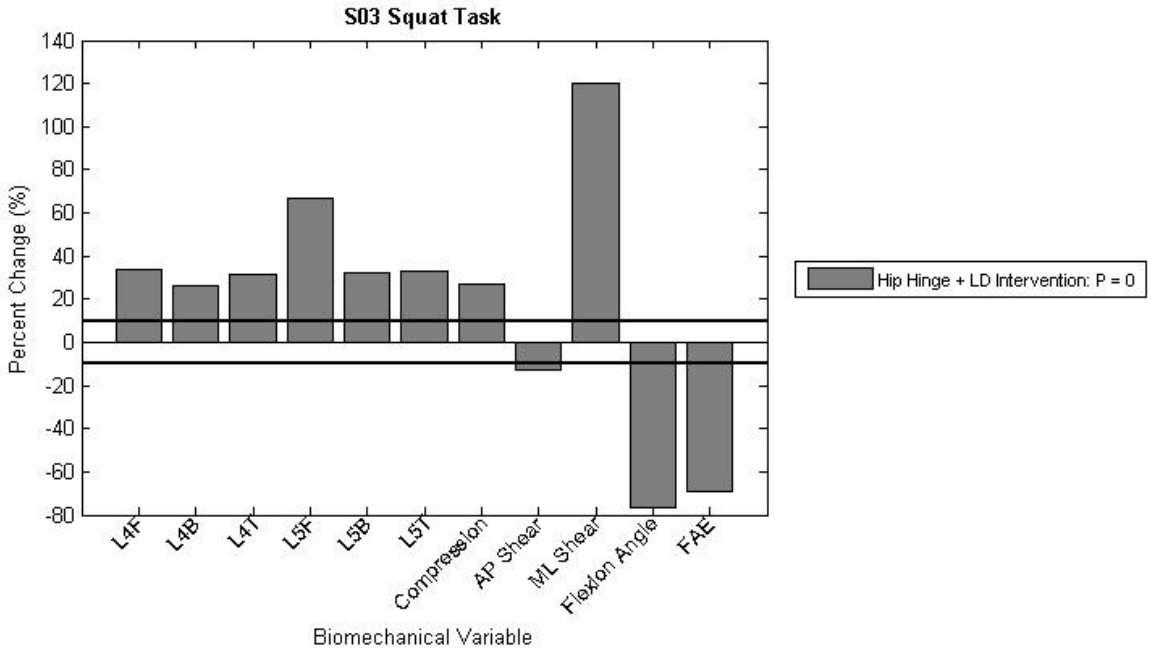

Figure 42 - Percent change of the L4 and L5 level EVs, L4/L5 compression, L4/L5 AP shear, L4/L5 ML shear, lumbar flexion angle and flexion angle excursion (FAE) between the unbraced condition and the hip hinge plus LD activation intervention for the squat task for Subject 3. Positive change represents a higher magnitude of the biomechanical variable when using the intervention. The thick black lines highlight the points where changes were considered biologically significant.

The jump from stool task was performed using the subject's initial and unbraced pattern, as well as two interventions: wtih an abdominal brace and a LD intervention. For the unbraced condition, the subject reported an NPS score of 7, while with both interventions the NPS score decreased to 0. The mean EMG activity for the brace condition resulted in a biologically significant increase compared to the unbraced condition for all muscles except RUES and RLES (table 11), which had a 20.5% and 23.5% decrease, respectively. The largest increases in muscle activity were in the RRA muscle at 148.3% from 293.2 %RV to 728.2 %RVC and LUES muscle at 135.3% from 18.6 %RVC to 43.8 %RVC. The muscle activity of the LLES and LLD muscles increased the least, at 15.1% and 54.5%, respectively. The muscle

activity of the remaining muscles increased in the range of 62% to 120% for the brace condition. For the LD condition, all muscles resulted in increased mean EMG activity except the RLES muscle that did not change and the LRA muscle (table 11), which had a 13.5% decrease. The largest increases in muscle activity for the LD intervention were 447.8% from 24.0 %RVC to 131.5 %RVC for the LEO muscle, 374.6% from 60.9 %RVC to 289.0 %RVC for the REO muscle and 286.8% from 16.7 %RVC to 64.8 %RVC for the RLD muscle. The smallest increases in muscle activity were in the LLES and RIO muscles at 41.8% and 98.9%, respectively, while the remaining muscle activities increased in the range of 110% to 198% for the LD intervention.

Table 11 - EMG activity for Subject 3 for the jump from stool task (Mean ± SD).

Muscle	Unbraced (%RVC)		Abdominal Brace (% RVC)		LD intervention (% RVC)	
	Right	Left	Right	Left	Right	Left
LD	16.7 (21.6)	30.3 (9.9)	29.4 (25.1)	46.8 (11.2)	64.8 (61.4)	72.1 (60.6)
UES	14.4 (16.6)	18.6 (7.8)	11.5 (5.5)	43.8 (18.3)	33.6 (24.3)	55.5 (23.4)
LES	15.0 (6.5)	16.2 (5.5)	11.5 (2.3)	18.6 (5.8)	13.8 (3.2)	22.9 (3.8)
RA	293.2 (128.7)	44.5 (14.8)	728.2 (213.5)	72.4 (14.7)	620.5 (535.8)	38.5 (19.9)
EO	60.9 (15.1)	24.0 (8.6)	99.1 (33.6)	52.7 (18.7)	289.0 (215.9)	131.5 (78.2)
IO	43.9 (18.6)	59.4 (14.6)	88.7 (29.6)	100.4 (44.0)	87.3 (40.0)	141.2 (64.9)

The mean EVs at the L4 and L5 level all resulted in a biologically significant increase when using both interventions compared to the unbraced condition (figure 43). For the brace condition, the L4 level EVs increased an average of 20.2% ± 8.3% and the L5 level EVs increased an average of 29.3% ± 3.8%. For the LD condition, the L4 level EVs increased an average of 49.0% ± 12.0% and the L5 level EVs increased an average of 40.8% ± 7.6%. The mean L4/L5 compression and AP shear resulted in a biologically significant increase for both interventions compared to the unbraced condition (figure 43).

For the brace intervention, AP shear increased 23.8% from -305.1 N to -377.8 N, while compression increased 21.0% from -2133.0 N to -2582.0 N. For the LD intervention, AP shear increased 33.5% from -305.1 N to -407.5 N, while compression increased 37.2% from -2133.0 N to -2926.6 N. However, the mean ML shear decreased by 28.8% from -61.0 N to -43.4 N with the brace intervention, while there was a 110.2% increase in ML shear from -61.0 N to -128.1 N with the LD intervention (figure 43). The mean lumbar flexion angle increased 98.8% and 97.7% for the brace and LD interventions, respectively (figure 43). The mean lumbar flexion angle was -2.5° in the unbraced condition and it increased to -5.0° for both the brace and LD conditions, moving into more flexion. The flexion angle excursion increased 38.0% from 5.0º to 6.9º for the brace condition and 33.9% from 5.0º to 6.7º for the LD condition (figure 43).

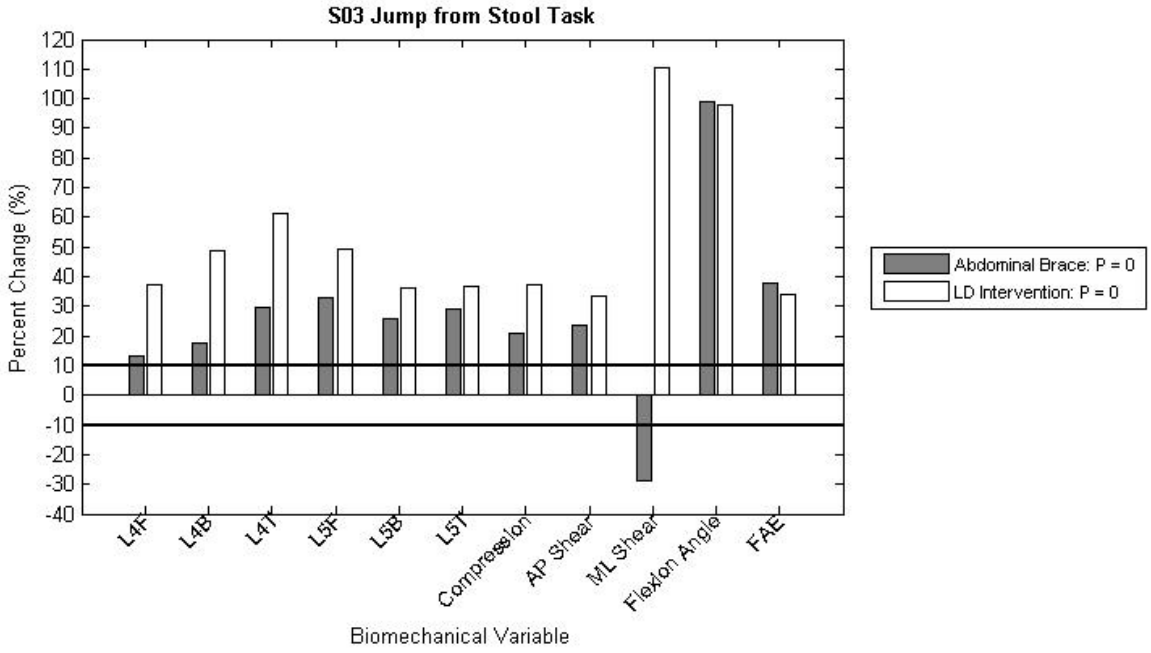

Figure 43 - Percent change of the L4 and L5 level EVs, L4/L5 compression, L4/L5 AP shear, L4/L5 ML shear, lumbar flexion angle and flexion angle excursion (FAE) between the unbraced condition and the suggested interventions for the jump from stool task for Subject 3. Positive change represents a higher magnitude of the biomechanical variable when using the intervention. The thick black lines highlight the points where changes were considered biologically significant.

Subject 3 also performed a one-step approach spike using her initial, unbraced pattern and using an abdominal brace. Using her initial pattern she reported an NPS of 4, while using the abdominal brace the NPS score decreased to 0. The intervention did not have a large influence on the mean EMG activity for all muscles (table 12). There was a biologically significant increase in mean EMG activity in the LRA, RRA, REO and RLD muscles at 23.8%, 80.0%, 30.9% and 31.0% change, respectively. The muscle activity of the LIO, RIO and LLD muscles resulted in decreases of 10.1%, 15.9% and 14.3%, respectively. The mean EMG activity of the remaining muscles did not have a biologically significant change.

Table 12 - EMG activity for Subject 3 for the one-step approach spike task (Mean ± SD).

Muscle	**Unbraced (%RVC)**		**Abdominal Brace (% RVC)**	
	Right	**Left**	**Right**	**Left**
LD	80.1 (29.6)	91.5 (42.8)	104.9 (40.5)	78.4 (37.7)
UES	92.2 (35.1)	67.0 (34.9)	91.9 (39.8)	72.6 (36.4)
LES	88.4 (50.9)	83.3 (62.8)	86.8 (57.6)	78.3 (47.7)
RA	926.6 (1048.2)	47.3 (28.1)	1667.9 (1941.7)	58.6 (51.2)
EO	162.5 (142.4)	311.3 (273.0)	212.8 (212.2)	290.4 (278.5)
IO	96.3 (30.1)	213.0 (118.7)	80.9 (30.6)	191.6 (113.7)

The EVs at the L4 level did not have a biologically significant change, while at the L5 level only the L5B EV had a biologically significant change (figure 44). This EV resulted in a 15.5% increase when using the abdominal brace compared to the unbraced condition. The mean L4/L5 compression and AP shear also did not have a biologically significant change, while the L4/L5 ML shear increased by 406.3% (figure 44) from -20.6 N to -104.4 N. There was not a biologically significant change in the lumbar

flexion angle excursion when using the intervention (figure 44), but the mean flexion angle increased 32.4% (figure 44) from -4.8° to -6.4°, moving towards more flexion.

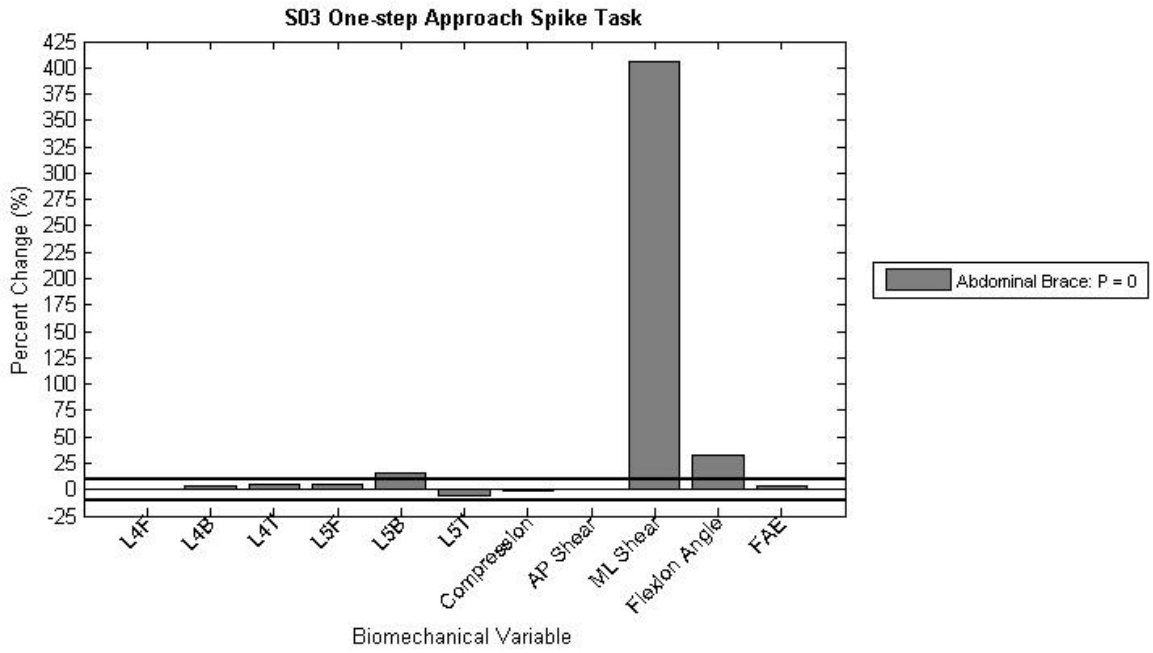

Figure 44 - Percent change of the L4 and L5 level EVs, L4/L5 compression, L4/L5 AP shear, L4/L5 ML shear, lumbar flexion angle and flexion angle excursion (FAE) between the unbraced condition and the suggested interventions for the one-step approach spike for Subject 3. Positive change represents a higher magnitude of the biomechanical variable when using the intervention. The thick black lines highlight the points where changes were considered biologically significant.

In summary, using the unbraced condition as a base, the interventions, which significantly decreased pain in all tasks, caused a biologically significant change in majority of measured biomechanical variables for every task except the spike task. The L4 and L5 level EVs and L4/L5 compression increased when using the interventions for all tasks. The L4/L5 AP shear increased for the heel drop and jump tasks, but decreased for the squat task. The L4/L5 ML shear decreased for the squat, spike and the abdominal brace intervention of the jump task, while it increased for the heel drop and LD intervention of the jump task. With all interventions and all tasks, the mean lumbar flexion angle moved

into more flexion, while the mean flexion angle excursion increased for the heel drop and jump tasks, but decreased for the squat task. For Subject 3, the flexion angle and L4/L5 ML shear variables were typically the most influenced by the interventions tested.

4.2.4 Subject 4

Subject 4 performed 3 tasks: sit to stand, stand to sit and a squat, as described in section 3.6. For the sit to stand task, the subject reported an NPS of 3 using the initial and unbraced pattern. The reported NPS score decreased to 0 for both the abdominal brace + hip hinge intervention and the spread floor intervention. For the brace + hip hinge intervention, the mean EMG activity increased for the left and right LD and EO muscles, while it decreased for the left and right LES and IO muscles. There was no biologically significant change for the left and right UES and RA muscles in the brace + hip hinge condition (table 13). The muscle activity of the right and left LD muscles increased 127.5% from 3.8 %RVC to 8.7 %RVC and 93.9% from 10.9 %RVC to 21.1 RVC, respectively, while the muscle activity of the right and left EO muscles increased 48.1% from 42.0 %RVC to 62.2 %RVC and 187.5% from 34.9 %RVC to 100.3 %RVC, respectively. The right and left LES mean EMG activity decreased 28.2% and 35.6%, respectively, and the right and left IO mean EMG activity decreased 54.0% and 49.9%, respectively, for the brace + hip hinge intervention. For the spread floor intervention, the LIO and RIO mean EMG activity did not have a biologically significantly change, but the muscle activity of the remaining muscles resulted in a biologically significant increase (table 13). The muscle activity of the LES and RA muscles had the smallest increase, with the right and left muscles averaging at 26.7% ± 5.9% for the LES muscles and 53.5% ± 2.5% for the RA muscles. The mean EMG activity of the LD muscles had the greatest increase at 1030.8% from 3.8 %RVC to 43.2 %RVC for the RLD muscle and 237.1% from 10.9 %RVC to 36.6 %RVC for the LLD muscle. The right and left UES muscles increased an average of 138.2% ± 26.6% and the EO muscle increased 160.1% on the right side and 316.3% on the left side.

Table 13 - EMG activity for Subject 4 for the sit to stand task (Mean ± SD).

Muscle	Unbraced (%RVC)		Abdominal Brace + Hip Hinge (% RVC)		Spread Floor intervention (% RVC)	
	Right	Left	Right	Left	Right	Left
LD	3.8 (4.0)	10.9 (5.4)	8.7 (11.0)	21.1 (15.8)	43.2 (20.0)	36.6 (12.6)
UES	12.9 (9.8)	12.9 (6.1)	11.7 (6.3)	13.7 (7.2)	28.4 (13.5)	33.1 (12.7)
LES	54.8 (30.3)	59.8 (28.4)	39.4 (24.2)	38.5 (30.0)	71.8 (27.0)	73.3 (33.7)
RA	32.6 (20.7)	38.1 (20.6)	29.9 (18.7)	35.3 (19.0)	49.4 (11.8)	59.1 (14.7)
EO	42.0 (27.0)	34.9 (15.8)	62.2 (26.3)	100.3 (36.2)	109.3 (18.9)	145.2 (44.7)
IO	62.8 (40.2)	52.1 (31.4)	28.9 (15.4)	26.1 (12.9)	62.5 (17.7)	47.3 (13.5)

The mean EVs at the L4 and L5 level resulted in a biologically significant increase when using both the abdominal brace + hip hinge intervention and the spread floor intervention (figure 45). For both interventions, the bend axis had the largest increase for both the L4 and L5 level. For the brace + hip hinge condition, the L4B EV increased 49.3% from 232.5 J/rad^2 to 347.0 J/rad^2 and the L5B EV increased 50.1% from 659.7 J/rad^2 to 989.9 J/rad^2. For the spread floor intervention, the L4B EV increased 105.4% from 232.5 J/rad^2 to 477.4 J/rad^2 and the L5B EV increased 88.2% from 659.7 J/rad^2 to 1241.6 J/rad^2. The mean L4/L5 compression did not result in a biologically significant change for the brace + hip hinge condition, but resulted in a 41.4% increase (figure 45) from -856.6 N to -1212.0 N for the spread floor intervention. The mean L4/L5 AP shear decreased 29.0% from -326.5 N to -231.7 N in the brace + hip hinge condition and did not have a biologically significant change in the spread floor condition (figure 45). The mean L4/L5 ML shear decreased 78.5% from -17.6 N to -3.8 N in the brace + hip hinge intervention and increased 28.1% from -17.6 N to -22.5 N for the spread floor intervention (figure 45). The mean lumbar flexion angle decreased 45.7% and 42.4% for the brace + hip hinge and spread floor

interventions, respectively, for the sit to stand task (figure 45). The mean flexion angle in the unbraced condition was -40.9°, while using the brace + hip hinge and spread floor interventions the mean flexion angle decreased to -22.2° and -23.6°, respectively. The lumbar flexion angle excursion also decreased when using the interventions (figure 45). With the brace + hip hinge intervention, the flexion angle excursion decreased 41.7% from 53.7° to 31.3°. Using the spread floor intervention, the flexion angle excursion decreased 38.2% from 53.7° to 33.2°.

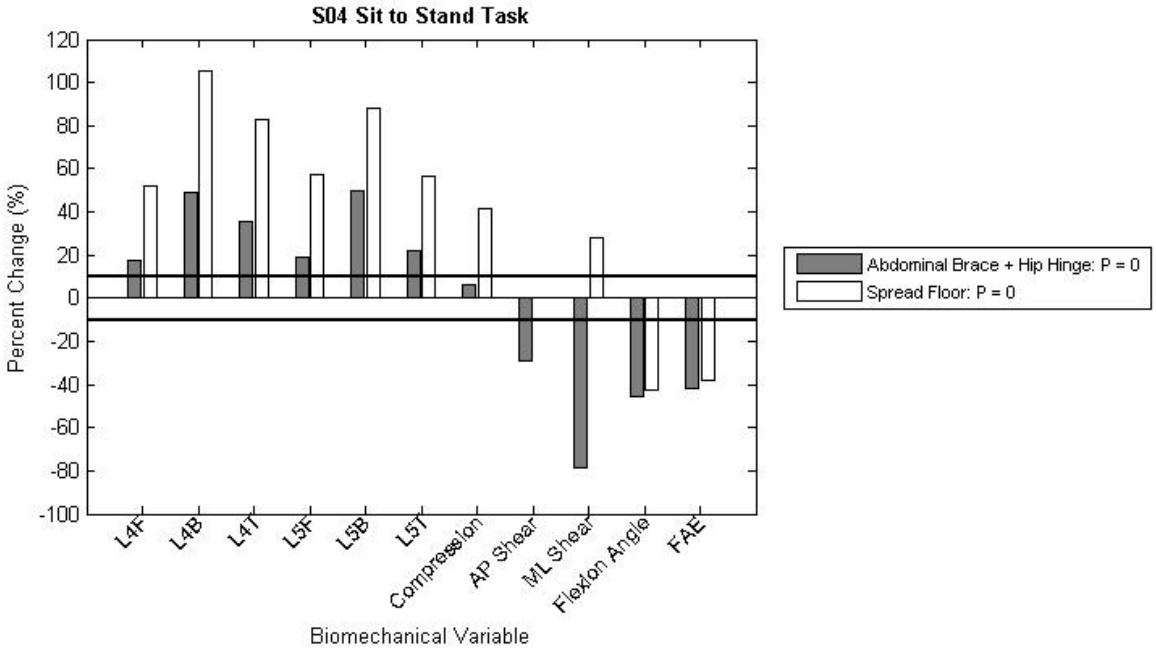

Figure 45 - Percent change of the L4 and L5 level EVs, L4/L5 compression, L4/L5 AP shear, L4/L5 ML shear, lumbar flexion angle and flexion angle excursion (FAE) between the unbraced condition and the suggested interventions for the sit to stand task for Subject 4. Positive change represents a higher magnitude of the biomechanical variable when using the intervention. The thick black lines highlight the points where changes were considered biologically significant.

For the stand to sit task, the subject reported an NPS score of 2 when using her initial and unbraced pattern and reported an NPS score of 0 for both the brace + hip hinge and spread floor interventions. For the brace + hip hinge intervention, the mean EMG of all muscles had a biologically significant increase except for the RUES muscle (table 14). The muscle activity of the left and right EO muscles had the largest increases at 236.4% from 25.0 %RVC to 84.0 %RVC and 211.2% from 22.0 %RVC to 68.4 %RVC, respectively. The muscle activity of the left and right LD muscles also increased a large amount, increasing 193.6% and 163.1%, respectively. The RIO muscle activity also increased more than 100% at a 105.6% increase. For the spread floor intervention, the mean EMG activity of RLD and RUES muscles did not have a biologically significant change and the EMG activity of the LUES muscle resulted in a decrease of 20.0%. The mean EMG activity of the remaining muscles resulted in a biologically significant increase (table 14), with the muscle activity of the RIO and LEO muscles having the largest change at 178.1% from 18.4 %RVC to 51.1 %RVC and 74.3% from 25.0 %RVC to 43.5 %RVC, respectively. The other muscles had a mean EMG activity increase in the range of 24% to 67%.

Table 14 - EMG activity for Subject 4 for the stand to sit task (Mean ± SD).

Muscle	**Unbraced (%RVC)**		**Abdominal Brace + Hip Hinge (% RVC)**		**Spread Floor intervention (% RVC)**	
	Right	**Left**	**Right**	**Left**	**Right**	**Left**
LD	11.0 (9.9)	10.2 (6.1)	29.0 (21.1)	29.9 (16.2)	11.9 (9.3)	14.7 (6.7)
UES	14.2 (8.8)	18.8 (12.4)	15.4 (5.2)	21.8 (15.9)	13.5 (4.4)	15.1 (4.6)
LES	32.8 (20.0)	42.4 (29.8)	58.7 (20.2)	64.5 (26.1)	48.2 (17.3)	52.8 (24.7)
RA	21.2 (14.4)	27.7 (11.0)	32.2 (11.1)	39.6 (10.9)	143.4 (9.8)	36.6 (8.2)
EO	22.0 (14.1)	25.0 (9.6)	68.4 (14.2)	84.0 (16.8)	34.2 (9.0)	43.5 (18.9)
IO	18.4 (18.5)	23.9 (11.5)	37.8 (14.7)	29.5 (10.3)	51.1 (29.2)	39.9 (19.7)

The variables output from the spine model demonstrated very similar trends to those seen in the sit to stand task. The mean L4 and L5 level EVs had a biologically significant increase when using both interventions (figure 46). Of the L4 level EVs, the L4B EV had the largest increase at 138.5% increase from 180.9 J/rad^2 to 431.3 J/rad^2 for the brace + hip hinge intervention and 129.4% increase from 180.9 J/rad^2 to 415.0 J/rad^2 for the spread floor intervention. For the L5 level EVs, the largest increase was seen in the L5B EV at 118.2% increase from 473.9 J/rad^2 to 1033.9 J/rad^2 for the brace + hip hinge intervention and 134.2% increase from 473.9 J/rad^2 to 1110.0 J/rad^2 for the spread floor intervention. The mean L4/L5 compression also increased when using the interventions (figure 46), with the brace + hip hinge intervention increasing 48.9% from -729.1 N to -1085.6 N. For the spread floor intervention, the mean compression increased 43.0% from -729.1 N to -1042.4 N. The mean L4/L5 AP shear increased 10.5% from -285.3 N to -315.3 N for the brace + hip hinge intervention, but did not have a biologically significant increase for the spread floor intervention (figure 46). For mean L4/L5 ML shear, the brace + hip hinge intervention caused a 59.8% decrease from -29.3 N to -11.8 N and the spread floor intervention caused a 80.5% decrease from -29.3 N to -5.7 N (figure 46). The mean lumbar flexion angle decreased with both interventions, at a 36.4% and 45.6% decrease for the brace + hip hinge and spread floor interventions, respectively (figure 46). In both situations, the mean flexion angle moved towards a more neutral posture, from -41.2º with the unbraced pattern to -26.2º and -22.4º for the brace + hip hinge intervention and spread floor intervention, respectively. The lumbar flexion angle excursion also decreased with the interventions, with a 13.8% decrease from 40.3º to 34.8º for the brace + hip hinge intervention and 22.5% decrease from 40.3º to 31.3º for the spread floor intervention (figure 46).

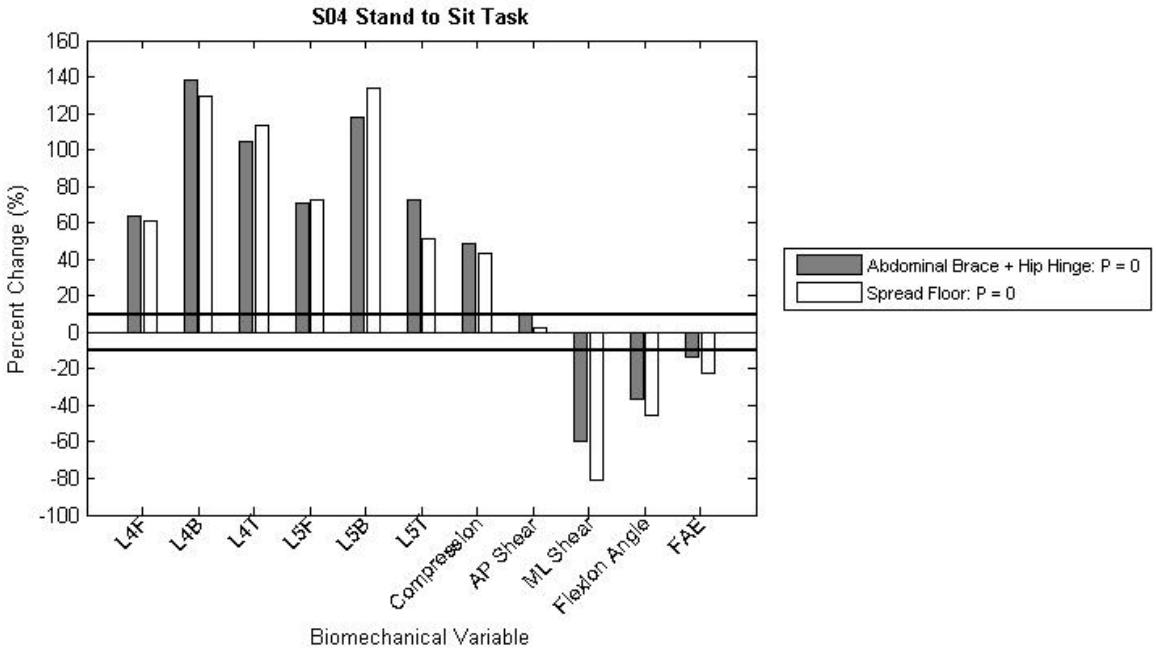

Figure 46 - Percent change of the L4 and L5 level EVs, L4/L5 compression, L4/L5 AP shear, L4/L5 ML shear, lumbar flexion angle and flexion angle excursion (FAE) between the unbraced condition and the suggested interventions for the stand to sit task for Subject 4. Positive change represents a higher magnitude of the biomechanical variable when using the intervention. The thick black lines highlight the points where changes were considered biologically significant.

For the squat task, subject 4 reported an NPS of 2 when using the initial and unbraced pattern and an NPS of 0 when using the hip hinge intervention. The mean EMG activity indicated that there was a biologically significant increase in muscle activity for all muscles when using the hip hinge intervention compared to the unbraced condition (table 15). The muscle activity of the RLES and LLES muscles had the smallest increases at 37.5% and 46.9%, respectively. The muscle activity of the RIO and REO muscles increased the most at 310.9% increase from 16.7 %RVC to 68.5 %RVC and 248.5% increase from 28.8 %RVC to 100.4 %RVC, respectively. The mean EMG activity of the remaining muscles increased in the range of 95% to 237% when using the hip hinge intervention.

Table 15 - EMG activity for Subject 4 for the squat task (Mean ± SD).

Muscle	Unbraced (%RVC)		Hip Hinge (% RVC)	
	Right	Left	Right	Left
LD	6.5 (7.0)	19.7 (11.6)	19.9 (17.4)	60.8 (26.6)
UES	18.8 (11.1)	17.6 (5.5)	36.6 (22.9)	38.8 (28.0)
LES	58.6 (28.3)	69.8 (30.4)	80.5 (28.7)	102.5 (44.2)
RA	23.0 (25.8)	29.4 (14.7)	59.5 (28.8)	64.8 (25.4)
EO	28.8 (22.0)	19.9 (14.3)	100.4 (54.5)	67.0 (22.5)
IO	16.7 (15.0)	26.9 (12.0)	68.5 (45.8)	55.2 (30.8)

The mean EVs at the L4 and L5 lumbar levels always resulted in a biologically significant increase when using the hip hinge intervention compared to the unbraced condition (figure 47). The L4T EV had the largest increase at 117.4% from 255.7 J/rad^2 to 555.8 J/rad^2, followed by the L5B EV at 116.6% from 537.7 J/rad^2 to 1164.4 J/rad^2. The lowest changes in the EVs were a 53.1% increase from 120.9 J/rad^2 to 185.2 J/rad^2 in the L4F EV and a 73.4% increase from 1086.3 J/rad^2 to 1884.0 J/rad^2 in the L5T EV. The mean L4/L5 compression and AP shear also increased, with a 56.7% increase from -770.2 N to -1206.6 N and a 22.4% increase from -295.1 N to -361.1 N, respectively, when using the intervention (figure 47). The mean L4/L5 ML shear decreased 54.3% (figure 47) from -30.3 N to -13.8 N when using the hip hinge intervention compared to the unbraced condition. The mean lumbar flexion angle decreased 31.8% from -37.5° to -25.6° when using the intervention and the lumbar flexion angle excursion also decreased, with a 30.2% decrease from 52.0° to 36.3° (figure 47). Both the lumbar flexion angle and flexion angle excursion indicate the subject moved towards a more neutral posture when using the hip hinge intervention than during the unbraced condition.

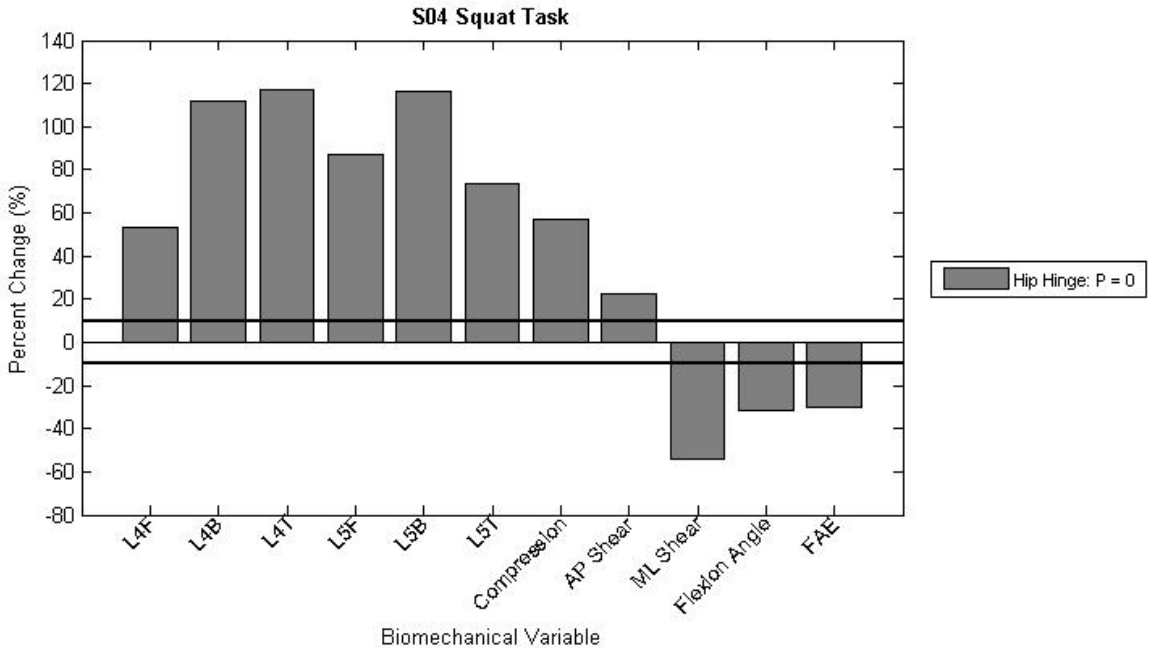

Figure 47 - Percent change of the L4 and L5 level EVs, L4/L5 compression, L4/L5 AP shear, L4/L5 ML shear, lumbar flexion angle and flexion angle excursion (FAE) between the unbraced condition and the hip hinge interventions for the squat task for Subject 4. Positive change represents a higher magnitude of the biomechanical variable when using the intervention. The thick black lines highlight the points where changes were considered biologically significant.

In summary, using the unbraced condition as a base, the interventions, which significantly decreased pain in all tasks, caused a biologically significant change in majority of measured biomechanical variables for every task. The L4 and L5 level EVs and L4/L5 compression increased when using the interventions for all tasks. The L4/L5 AP shear increased for the squat and stand to sit task, but decreased for the brace + hip hinge intervention with the sit to stand task. For the L4/L5 ML shear, there was a decrease in every situation except the spread floor intervention with the sit to stand task. For all interventions and all tasks, the mean lumbar flexion angle and lumbar flexion angle excursion decreased, moving towards a more neutral posture. For Subject 4, the L4 and L5 level EVs in the bend and twist axes were typically the most influenced by the suggested interventions.

Chapter 5
Discussion

This discussion is organized to first specifically address the phase 1 hypotheses followed by a general discussion regarding the overall conclusions from the sensitivity analysis portion, or phase 1, of the thesis. Second, the phase 2 hypotheses are addressed as they relate to each individual subject, followed by the overall conclusions obtained from the case studies portion, or phase 2, of the thesis. Finally, there is an integrated discussion of the thesis limitations and a general summary of the thesis.

5.1 Phase 1: Sensitivity Analysis

Phase 1 of this thesis was performed to learn the behaviours of the various EVs. A sensitivity analysis was performed to determine if there are links between muscles and EVs when using the anatomically detailed spine model developed by McGill and colleagues. For example, if a biomechanist could assess EVs, could they advise a clinician to attempt to activate a particular muscle in a patient. As it turned out, there was no link between specific EVs and specific muscles, as discussed below in relationship with the specific hypotheses.

5.1.1 Hypothesis 1: Individual muscles affect specific EVs, but no one muscle can be associated with one EV level

Assuming that a 10% change in the EVs represented a biologically significant difference, individual muscles did affect specific EVs, but no one muscle could be associated with one EV level (i.e. one EV level was affected by multiple muscles), supporting hypothesis 1. The muscles that caused a biologically significant change in the EVs when removed were dependent on posture. However, in most situations, the Mult, Pars, Ilio and Long muscles tended to cause changes in the EVs at the L1, L2 and L3 levels. At the L4 level, the Pars and Ilio muscles were still important, but the IO and EO muscles resulted in a larger change. At the L5 level, the abdominal muscles resulted in the largest changes, with the Ilio

muscle still being important for the L5F EV. It is also important to note that across all EVs, the TrA muscle never resulted in a biologically significant change, and in the L4B, L4T and L5 EVs the Mult muscle did not cause a biologically significant change when removed.

One strategy that is commonly reported as an effective way to improve spine stability/stiffness is to try and activate the TrA and Mult muscles (Richardson et al. 1992; Richardson & Jull 1995; O'Sullivan et al. 1997; França et al. 2010). Typically, this is accomplished using the abdominal hollowing technique where the individual draws in their abdominal wall (Richardson et al. 1992). A second method to improve spine stability/stiffness is to use an abdominal brace to try and activate all of the muscles of the torso, including the RA, EO, IO and erector spinae muscles (McGill 2003; Grenier & McGill 2007; Vera-Garcia et al. 2007; Stanton & Kawchuk 2008). There has been controversy over which of these methods results in a larger increase in stability/stiffness and decrease in pain in LBP subjects.

Grenier & McGill (2007) examined the effects of abdominal hollowing and abdominal bracing on lumbar stability and compressive loads, as calculated by a comprehensive lumbar spine model. This study found that bracing always resulted in a greater stability than hollowing, with equal compressive loads for both the bracing and hollowing interventions. Stanton & Kawchuk (2008) also reported a higher stiffness when using bracing over hollowing. Here, the researchers used posteroanterior stiffness at the L4 level as the indicator of stability. In addition, though there was not a significant difference, the bracing condition was trending to have more TrA activity than the hollowing condition, and the RA, EO, IO and upper and lower erector spinae muscles also had more activation using the bracing technique than the hollowing technique. Both these studies were performed on asymptomatic individuals.

França et al. (2010) compared the effects of segmental stabilization versus strengthening of the torso muscles for six-weeks in individuals with chronic LBP felt between T12 and the gluteal fold. The segmental stabilization intervention focused on the TrA and Mult muscles, while the torso strengthening intervention focused on the RA, IO, EO and erector spinae muscles. Although both interventions were

found to significantly decrease pain, the segmental stabilization protocol did so to a greater degree. The segmental stabilization protocol also resulted in a higher TrA activation capacity, but this may have been a function of the exercises prescribed, since the prescribed exercises for the torso strengthening protocol did not address the TrA muscle. It could be argued that the torso strengthening protocol in the França et al. (2010) study was designed to worsen symptoms as it involved bent-leg sit-ups, bent-leg sit-ups with a twist, bent-leg raise and a prone exercise involving trunk extension, with a high focus on increasing the strength of the RA muscle. These exercises were potentially inappropriate for the population studied due to the high compression imposed on the low back combined with bending. Axler & McGill (1997) found that sit-ups result in high low back compression values, with 3350 N compression for bent-leg sit-up, 2964 N compression for cross-knee curl-up and 1767 N compression for bent-leg raise, as reported by McGill (2007). The action limit for low back compression is 3300 N (NIOSH 1981) indicating that repetitive loading above this elevates the risk of injury. Therefore, the prescribed sit-up exercise is over this limit, and the sit-up with a twist is approaching the limit. In addition, the trunk extension exercise has been found to submit the low back to over 4000 N compression (Callaghan et al. 1998), which is well over the action limit stated. In addition, compression plus bending has been shown to cause disc herniations (Callaghan & McGill 2001). These high compressive loads together with repeated bending under load imposed on the low back during the torso strengthening protocol may have been a source of pain for the individuals prescribed this intervention, which resulted in the segmental stabilization intervention having lower pain scores than the torso strengthening intervention. A more appropriate torso strengthening protocol may have included using the 'big three' exercises, including curl-ups, side bridge and the 'bird dog' exercise. These three exercises ensure a positive stability index while not incurring excessive spine load. The curl-ups aim to train the RA muscle, the bird dog trains the back extensors, including the Long, Ilio and Mult muscles, and the side bridge trains the QL, IO, EO and TrA muscles

(McGill 2007). Using these exercises would effectively strengthen the torso muscles while reducing low back loads, potentially changing the results of the França et al. (2010) study.

The results of the sensitivity analysis showed that the TrA and Mult muscles did not have an effect on the EVs at the lower lumbar levels, but the abdominal and erector spinae muscles did have a large effect at these levels, thus it is logical that an abdominal brace should be used to increase stability/stiffness in the lower lumbar levels. Therefore, if an individual has LBP associated with instability in the lumbar spine, they will most likely benefit more from strengthening the torso muscles than focusing on strengthening the TrA and Mult muscles alone. These results agree with the results found by Grenier & McGill (2007) and Stanton & Kawchuk (2008). Further, Stanton & Kawchuk (2008) found that the bracing condition activates the TrA muscle as much as the hollowing technique. This implies that the bracing technique would be as efficient as the hollowing technique to activate the TrA muscle and is arguably the better method due to the numerous other muscles that are trained.

At the upper lumbar levels, the Mult and erector spinae muscles have the largest effect on the EVs, while the abdominal muscles have a smaller effect at these levels. This may be a function of the anatomical robustness of the spine model used. The spine model was designed as an L4/L5 model; therefore the most anatomical robustness is at the L4 and L5 levels. It is possible that the results of this sensitivity analysis would be different if the model was more anatomically detailed at the upper lumbar levels.

5.1.2 Hypothesis 2: Specific muscles do affect specific planes of stability/stiffness

Assuming that a change of 10% or greater in the EVs constituted a biologically significant difference, muscles did affect the plane of stability/stiffness, supporting hypothesis 2. In most situations, the erector spinae and Mult muscles affected the flexion axis to the greatest degree. In certain postures at the L1 and L2 levels, they had the largest change in the bend axis, while at the L3 and L4 level they sometimes affected the twist axis the most. At the L4 and L5 lumbar levels, when the abdominal muscles

had a biologically significant change, they had the greatest effect on the bend axis and occasionally the twist axis.

Given the functions of the erector spinae muscles, it was anticipated that they would have the largest effect on the axis they did. Since the erector spinae muscles are primarily extensor muscles, it would be expected that they would have the most effect on the flexion/extension axis, as was seen. Similarly, the IO and EO muscles would be expected to affect the bend and twist axes, as the oblique muscles act to twist (McGill 1991a; McGill 1991b) and laterally bend (McGill 1992) the torso. Given that the muscles appear to affect the stability/stiffness axis in which they act, it was no surprise that the TrA muscle did not have a biologically significant effect on any EV since the TrA muscle compresses the abdomen and does not cause spine motion in any plane.

5.1.3 Hypothesis 3: EVs are affected by posture

A common approach to reduce LBP is to ensure the individual remains in spinal postures close to neutral to ensure shear support, ensure the ability to withstand compressive forces and reduce the risk of ligamentous damage and disc herniation (McGill 2007). In this sensitivity analysis, hypothesis 3 was supported, as posture was found to have a biologically significant effect on the EVs when compared to neutral, assuming a change of 10% or greater in the EVs represented a biologically significant difference. The twist postures affected the least number of EVs, indicating that it is not as important to stay close to neutral in the twist axis as it is in the flexion/extension or bend axes. In all situations, the postures further from neutral resulted in larger magnitude changes than the postures close to neutral. For most EVs, the largest change was seen in the 30° extension posture. The only EVs that this did not occur were the L3T, L4F, L5B and L5T EVs, where the largest change was seen in the 50° flexion posture. The L4B EV also was not highest in the 30° extension posture, but the 30° bend posture. This implies that extreme flexion and extension postures have the most detrimental effect on stability/stiffness.

It has been previously found that spine mechanics and load carrying abilities are affected by the degree of lordosis. For example, there is a smaller moment arm for the extensor muscles (Tveit et al. 1994) and a decreased tolerance to compression (Gunning et al. 2001) with a more flexed posture. In addition, flexion angles over 75% of the full range resulted in significantly higher intradiscal pressure (Adams et al. 1994). It has also been found that in flexed postures the load is transferred from muscles to passive tissue, increasing the likelihood of a disc herniation (McGill 1997). Further, McGill et al. (2000) found that flexing the lumbar spine reduces the cosine of the Long and Ilio muscles, which diminishes the ability of these muscles to resist the anterior shear forces introduced during flexion. This implies that there would be a larger shear load when in flexed postures than neutral postures.

Extension of the lumbar spine also causes changes in spine mechanics. First, extended postures cause an articulation of the spinous processes that result in transmission of high compressive forces (Adams et al. 1988). Further, due to the load-bearing apophyseal joints in extension, damage could occur at compressive loads as low as 500 N (Adams et al. 1994). These authors also found that the distribution of compressive stress is shifted from a peak in the anterior annulus during neutral postures to a large peak in the posterior annulus. In addition, there is a 40% decrease in nucleus pressure when in extension than in neutral postures (Adams et al. 1994). It has also been found that degenerated discs in extension usually showed an increase in compressive stress in the posterior annulus, but occasionally decreased the compressive stress (Adams et al. 2000). Further, extended discs show a decrease in foramen area increasing the likelihood of nerve root compression (Inufusa et al. 1996), which is one source of pain in individuals with LBP. This implies that extended postures may be detrimental for most individuals, but beneficial for others.

The sensitivity analysis on the spine model is in accordance with the previously mentioned research. In addition to ensuring normal spine mechanics and tissue mechanics, stability/stiffness is also

higher when in neutral postures than in flexed or extended postures. These results support the idea that individuals should remain in a neutral posture, especially if they are plagued by LBP.

5.1.4 Hypothesis 4: Overactivating muscles by increasing muscle activation to 100% MVC negatively affects the EVs.

Increasing the muscle activation of single muscles from 50% MVC to 100% MVC did not have a major effect on most EVs and postures, but when there was an effect, there was an increase in the EV, thus hypothesis 4 was refuted, at least when using the 10% change in EV criterion as the level of biological significance. However, it is possible that increasing the activation to 100% MVC from an initially lower muscle activation level would affect the EVs.

Stiffness has been found to have a non-linear relationship with muscle force in the cat soleus muscle (Joyce & Rack 1969), human elbow flexors (Pousson et al. 1990) and rat gastrocnemius muscle (Ettema & Huijing 1994). In particular, it has been found that in the cat soleus muscle, stiffness remains constant with muscle forces in the range of 25-100% of maximum (Hoffer & Andreassen 1981). Since muscle stiffness is related to stability, it stands to reason that there would be a non-linear force-stability relationship. Further, Brown & McGill (2005) found that the contribution to stability of an individual muscle peaks at a critical force level, after which it may even be detrimental to stability. The results of increasing the activation from 50% MVC to 100% MVC indicate that 50% MVC may have been over this critical force level for some muscles.

It has been found that using the abdominal brace results in the highest muscle activity of the torso muscles at less than 35% MVC (Stanton & Kawchuk 2008). This result and the concept of non-linear force-stiffness relationships indicate that it would be beneficial to perform the sensitivity analysis using a lower initial muscle activation level and determining the effect on EVs when decreasing the activation to 0% MVC and increasing it to 100% MVC.

5.1.5 Hypothesis 5: The relationship between muscles and specific EVs obtained during simulation remains with real subjects performing loaded tasks.

Four subjects performed a walking task where they carried a 15 kg load in each hand to obtain the actual data set for the sensitivity analysis. Based on the observation that this task was performed at approximately 7º flexion, 4º bend and 4º twist, it would be expected that the results of this sensitivity analysis would be similar to those obtained from the 10º flexion posture using the simulated data set. Assuming a change of 10% or greater in the EVs represented a biologically significant change, the results from the actual data and simulated data in the 10º flexion posture were similar, supporting hypothesis 5.

Due to the low levels of muscle activation with the actual data set, it is not surprising that reducing the muscle activity to 0% MVC did not have a biologically significant effect on most EVs. When there was a biologically significant change between actual EMG and 0% MVC, it was most often seen in the L4 and L5 EVs, indicating that these EVs are most sensitive, as would be expected given the anatomical robustness of the model at the L4 and L5 lumbar levels. For this reason, the actual EMG to 100% MVC for the actual data and the 50% MVC to 0% MVC for the theoretical data set were compared. Although there were occasionally differences in the specific muscles that affected each EV, the overall conclusions were the same. In both situations, the L1, L2, and L3 levels and the L4F EVs were most influenced by the erector spinae and Mult muscles, while the remaining EVs were most influenced by the abdominal muscles.

These results imply that activating all muscles to 50% MVC does not have a large influence on the interpretation of which EVs and muscles are linked. Although this thesis only tests one posture with actual EMG, it is not anticipated that there would be a large difference between the theoretical and actual data sets in different postures.

5.1.6 Phase 1 Summary

Results from phase 1 indicate that activating the abdominal muscles has the largest influence on the lower level EVs, while the erector spinae muscles most affect the upper level EVs. Further, the abdominal muscles most affect the bend or twist axes, while the erector spinae muscles most affect the flexion axis. Postures more than 10° from neutral have a negative effect on the stability/stiffness and increasing the activation from 50% MVC to 100% MVC does not typically result in large change in stability/stiffness.

Stokes et al. (2011) also used a model to assess the effects of selective activation of the TrA, RA or IO and EO muscles on the spinal stability when the trunk was loaded. It was found that activating the TrA and oblique muscles to 10% MVC had a small increase in stability, but increasing it further to 20% MVC had little effect. In addition, selectively activating the RA muscle did not have an effect on stability. These results imply that activating the entire abdominal wall will have a larger increase in stability than selectively activating the abdominal muscles, similar to the results found in the present work.

5.2 Phase 2: Case Studies

Phase 2 of this thesis allowed insight into the relationship between pain and a number of different biomechanical variables. Due to individuality in pain presentation, a series of case studies was conducted. The relationships found will be discussed for each subject, followed by an overall summary of the findings.

5.2.1 Hypothesis 6 and 7: Coaching and cueing specific movement patterns and motor patterns would alter pain in low back pain patients, and the changes in pain would be reflected in changes in EVs.

Based on the assumption that a difference of 2 points in the NPS score constituted a clinically significant change in pain, hypothesis 6 was conditionally supported, as there was a clinically significant

decrease in pain for most subjects and most tasks when using a suggested intervention that altered specific movement and motor patterns. Given that there was a change in pain with most interventions, hypothesis 7 was conditionally accepted, as these changes in pain were reflected in the EVs in most situations. However, the EVs were not always the most influenced biomechanical variable when comparing the interventions with the unbraced patterns. The most influenced variable was sometimes able to be predicted by the individual subjects' pain presentation, but this was not always the case, as discussed below.

Based on the observation that subject 1 was compression intolerant, it would be expected that mean L4/L5 compression would be the most influenced variable when using the intervention. Although L4/L5 compression did increase when using both interventions, the L4 level EVs were the most influenced by the interventions for this subject. However, there was not a clinically significant difference in pain levels between the unbraced condition and the interventions. Based on these results, it is possible that the heel drop test does not only test compression, but it is also possible the mechanism of pain cannot be determined through modeling and the clinical pain presentation may not match the output of the spine model.

For subject 2, it would be expected that performing tasks in a posture closer to neutral would decrease pain based on the observation that this subject was flexion intolerant. Although this was found to occur in all situations except one, the variable that was typically influenced the most by the interventions, and subsequent decrease in pain, was the L4/L5 ML shear. In all situations the L4/L5 ML shear increased, but remained well below the proposed shear action limit of 500 N (Norman et al. 1998). Although this action limit is based on AP shear, there is no known limit for ML shear so this limit was used as a reference. The mean lumbar flexion angle was the most influenced variable in the heel drop task, but this was likely a function of the very small degree of flexion employed during the initial trial when the subject performed the heel drop task. For this subject, it is difficult to determine the

biomechanical variable associated with the decrease in NPS score reported when using the suggested interventions.

For subject 3, it would be expected that the lumbar flexion angle would trend towards more flexion with decreased pain, due to the initial conclusion that this subject was extension intolerant. For this subject, the lumbar flexion angle always moved into more flexion and was typically one of the most influenced variables when using the suggested interventions, which was associated with decreased pain. The other variable that had a major change when using the interventions was the L4/L5 ML shear, which typically increased but remained well below the reference shear action limit of 500 N.

Based on the conclusion that subject 4 had instability at the L5 level, it would be expected that a decrease in pain would be associated with an increase in stability/stiffness in the EVs representing the L4/L5 and L5/S1 joints. These results were seen in all tasks for subject 4, as the EVs at the L4 and L5 lumbar level were the most influenced biomechanical variable when using the suggested interventions, which were associated with decreased pain.

An interesting case was with subject 3 with the spike task. In this situation, there was very little change in the EMG activity except for in the right RA muscle. There was also little change in the flexion angle, except for 0% movement to 50% movement (right foot toe-off to halfway between right foot toe-off and both feet leaving the ground) where the subject moved into more flexion with the intervention (Appendix E). With these small changes in EMG activity and posture, few variables showed a biologically significant change, with only the L5B EV having a small increase and ML shear resulting in a large increase when using the intervention. These results indicate that the subject performed the spike task in nearly the same way every time. This is not surprising because high-level athletes practice the specific skills and tasks needed for the sport with the aim to obtain automation of the task. With automation of the task, it would be expected that the biomechanical variables would be similar each time the task was performed. This appeared to be the case with subject 3, as both times she performed the

spike task, the EMG and spine angles were very similar. The surprising part with this is that the pain level decreased with the intervention when there did not seem to be a biomechanical change. This indicates that there was another variable that was responsible for the pain reduction that was not quantified in this study.

5.2.2 Phase 2 Summary

A common assumption of low back pain is that motions, postures and loads are responsible for tissue damage/irritation that lead to pain. Therefore, altering the motions, postures and loads would be expected to decrease pain (McGill 2007). Based on the subjects analyzed, the compression and AP shear almost always increased with the intervention that was chosen. Using the (NIOSH 1981) action limit of 3300 N for compression and the AP shear action limit of 500 N (Norman et al. 1998), the compression and AP shear often approached or even exceeded the limits. This may be a function of the RVC task and gain factor such that in reality, these values were not as high. These observations show that the loads were altered when using the interventions, although they changed the in the opposite direction than what was expected.

Motions and postures were also altered when using the interventions. In all subjects, if the flexion angle was over approximately 15°, it was always decreased towards a neutral posture. In the squat and lift bar tasks where spine motion occurred, it was also seen that there was less spine motion and the lumbar spine remained closer to a neutral posture. As mentioned previously, ensuring a neutral posture and proximal stiffness is beneficial so that more shear (McGill et al. 2000) and compression (Gunning et al. 2001) can be tolerated and there is less chance of a disc herniation (McGill 1997).

Based on the case studies conducted, altering motions, postures and loads reduced pain intensity, but there does not appear to be one biomechanical variable that was common for reducing pain; all variables examined appeared to play a part in the pain reduction. In fact, the biomechanical variable that would be expected to change based on clinical assessment did not always react in the expected way, as

seen with subject 1. The overall conclusion obtained from this phase of the thesis is that a common goal for clinicians may be to ensure the flexion angle is close to neutral. However, clinicians cannot be instructed on what muscles to preferentially activate based on a biomechanical analysis; the treatment/interventions must be decided on an individual basis through careful clinical assessment and sometimes trial and error.

5.3 Limitations

There are a number of limitations in this thesis that should be noted. First, all variables measured were based on a model, which was influenced by the architecture of the muscles and vertebrae. As mentioned throughout this document, the model is more anatomically robust at the lower lumbar levels, indicating that the results obtained at the upper lumbar levels may be less accurate. In addition, the activity of some muscles is driven from different muscle activation profiles that are accessible with surface EMG electrodes. In this spine model, the Pars, QL and Mult muscles are driven by the EMG activity at the LES site and the Ilio and Long muscles are driven by the EMG activity measured at the UES site. In addition, the Psoas and TrA muscles are driven by the IO activation profile. These assumptions are necessary due to the difficultly in collecting EMG activity of these deep muscles. However, previous work by McGill et al. (1996) suggested that the error is low with these assumptions.

A second limitation is the assumptions regarding biological significance. For this thesis, a change of 10% in any given variable was assumed to be biologically significant and a difference in the NPS score of 2 was considered clinically significant, as stated by Bijur et al. (2003). The value of 10% was chosen because it visually appeared to be a natural cut-off point when examining the results of the sensitivity analysis. It is unlikely that these assumptions affected the results of phase 2 because there was rarely a case where the variable changes were not clinically or biologically significant. For phase 1, there may have been a change in the specific muscles or postures that affected each EV, but the overall conclusions obtained would likely not have changed. In addition, interpreting the EVs collectively is difficult to begin

with. By nature, the EV is nonlinear; therefore one cannot say how much more or less stable the spinal system is based on the EVs alone.

For phase 1 of the thesis, one major limitation is the muscle activation level chosen. A value of 50% MVC was chosen to ensure a large change in EMG activity when increasing it to 100% MVC and decreasing it to 0% MVC, i.e. the muscle knockout model. It is unlikely that choosing a different value would have a large effect on the end conclusions of the sensitivity analysis because it is expected that knocking a muscle out to 0% MVC would have the same effect regardless of the starting activation; there would only be a different magnitude of change. There may have been a different result when increasing the activation to 100% MVC because this procedure had little change when the starting activation was 50% MVC and had a much larger effect when using the actual EMG patterns that had lower initial muscle activation levels.

A second limitation to phase 1 is that the EMG was assumed to be the same across all muscles, which is not a typical physiological response. However, having the same MVC for all muscles was important for the sensitivity analysis to determine the effect of each muscle individually on each of the EVs. This limitation was also addressed when using the actual EMG patterns of four healthy subjects. In addition, removing a muscle completely is not really relevant in real life. Even if force was zero in a single muscle, for example from severing the aponeurosis, cross-talk would still occur due to force and stiffness transmission through connecting tissues (Brown & McGill 2009).

For phase 1 part 2, a limitation was that only 4 subjects were used and the task performed resulted in less than 20% MVC muscle activation except for one subject who had 27% MVC for one muscle. In most situations, the muscle activation was even less than 10% MVC. In future it may be beneficial to examine actual EMG patterns that required more muscle activation for this sensitivity analysis. Further, one subject did not have any EMG activity of the right UES, so the Ilio and Long muscles did not actually

have actual EMG patterns. Not surprisingly, for this subject knocking these muscles to 0% MVC also did not often have an effect.

Another limitation to phase 1 of this thesis is that only the EVs were evaluated. In future, it would be beneficial to evaluate other variables such as the total stability index, L4/L5 compression and L4/L5 shear when a muscle knockout model is used. This may also give insight into the relationship between muscles and these other variables as well as the relationship between each of the variables.

One major limitation to phase 2 is that the results were based on a series of case studies; however this was also a strength. If multiple subjects could be classified into groups based on the type of pain presentation, a pattern may have emerged regarding the variables that change for each group. For example, it may have been seen that flexion intolerant individuals would move into more extension when they have pain reduction, while extension intolerant individuals would do the opposite. However, based on the case studies presented, this would not likely occur because in most situations no single variable stood out as being the most important; all variables changed. The variables also did not always change in the expected way based on the pain classification, as seen with subject 1. Thus, back pain, when examining details of behaviours, may be best studied as a series of case studies.

There were multiple limitations to the analysis of subject 1. First, only one task was analyzed so a trend within the subject could not be seen. Although a limitation, this probably did not have a large effect on the end result because the other subjects also showed that no single variable was the sole contributor to a reduction in pain. Second, due to technical difficulties during the data collection for this subject, kinematic data was unable to be collected so the posture was assumed to be completely neutral. It is unlikely that this had a major effect on the results because the sensitivity analysis (phase 1) showed that there is only an effect of posture at spine angles greater than 10° in any axis. The heel drop test is performed in an upright posture and it is unlikely that subject 1 had spine angles of over 10°. Further, the reaction forces and moments were unable to be calculated because of the lack of kinematic data. This

meant that a muscle gain could not be calculated for the spine model so the gain was assumed to be 1. This would not affect the results because the muscle gain is linear and affects the variables in the same way. Since variables were only compared within subjects, assuming a gain of 1 would show the same results as if a gain based on forces and moments was calculated.

A further limitation to phase 2 is the concept of expectation. The individuals were referred to Professor McGill under the impression that they would have the best chance at reducing pain after a consultation. Expectation may alter low back pain through promoting a physiological response, as has been seen in some studies using a placebo group, changing the individual's understanding of what is causing them pain or decreasing the anxiety surrounding the low back pain (Bialosky et al. 2010). Any of these factors, along with other expectation related factors, may have altered the reported pain intensities. However, Professor McGill historically follows up annually with each patient to see if results are transient. About 70% report continued and maintained improvement over the following two years of his consultation (Stu McGill, personal communication, October 3, 2011).

5.4 Thesis Conclusions

This thesis provides evidence that increasing the activation of the abdominal wall results in an increase in stability, and by default stiffness, and subsequent decrease in pain intensity for some individuals. This is consistent with the notion that stiffening a lumbar motion segment that is painful is pain reducing. It also provides evidence that ensuring a more neutral spine increases stability/stiffness and often decreases pain. This suggests that less passive tissue load and probably less stimulation of pain sensors with motion is helpful. The results indicate that the intervention required to decrease pain is case dependent and there does not appear to be one biomechanical variable that is responsible for pain reduction. This implies that treating patients is not only a science in terms of ensuring increased stability/stiffness relating movement to more distal locations in the skeletal chain, but an art in terms of determining the best way to increase stability/stiffness while decreasing pain.

In summary, major global findings are that EVs appear to give insight into whether stability/stiffness is changing due to changes in muscle activation and posture. However, the magnitude of individual EVs do not indicate the "amount" of change in stability/stiffness, nor does it appear that specific EVs are linked to specific muscles to guide clinical interventions. Rather, clinical interventions alter a host of variables such as joint compression, shear, posture, movements and ultimately joint stability/stiffness, but the links to pain appear to be specific to the individual. One could interpret this as meaning that, when studying the details of back pain, the best study design is a case studies series. Further, provocative testing of the patient, altering motions, postures and loads, to both exacerbate pain and identify the painful combinations and then reduce/eliminate pain by avoiding the exacerbator while still performing the same functional tasks appears to have efficacy. Little appears to be gained in terms of clinical utility from knowledge of the EV.

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Appendix A

Effect muscles on EVs for various postures

Each figure represents the percent change in one EV when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for various postures. The EV being examined is the large title at the top of each page, the posture being examined is the title of each graph and the muscle that was removed is along the x-axis. Negative change represents a lower EV when a single muscle's activity was reduced to 0% MVC. A change of 10% or greater in the EV was considered biologically significant. The thick black lines highlight these points. These figures are supplemental to those in section 4.1.1 (hypothesis 1 results), which display only the neutral posture.

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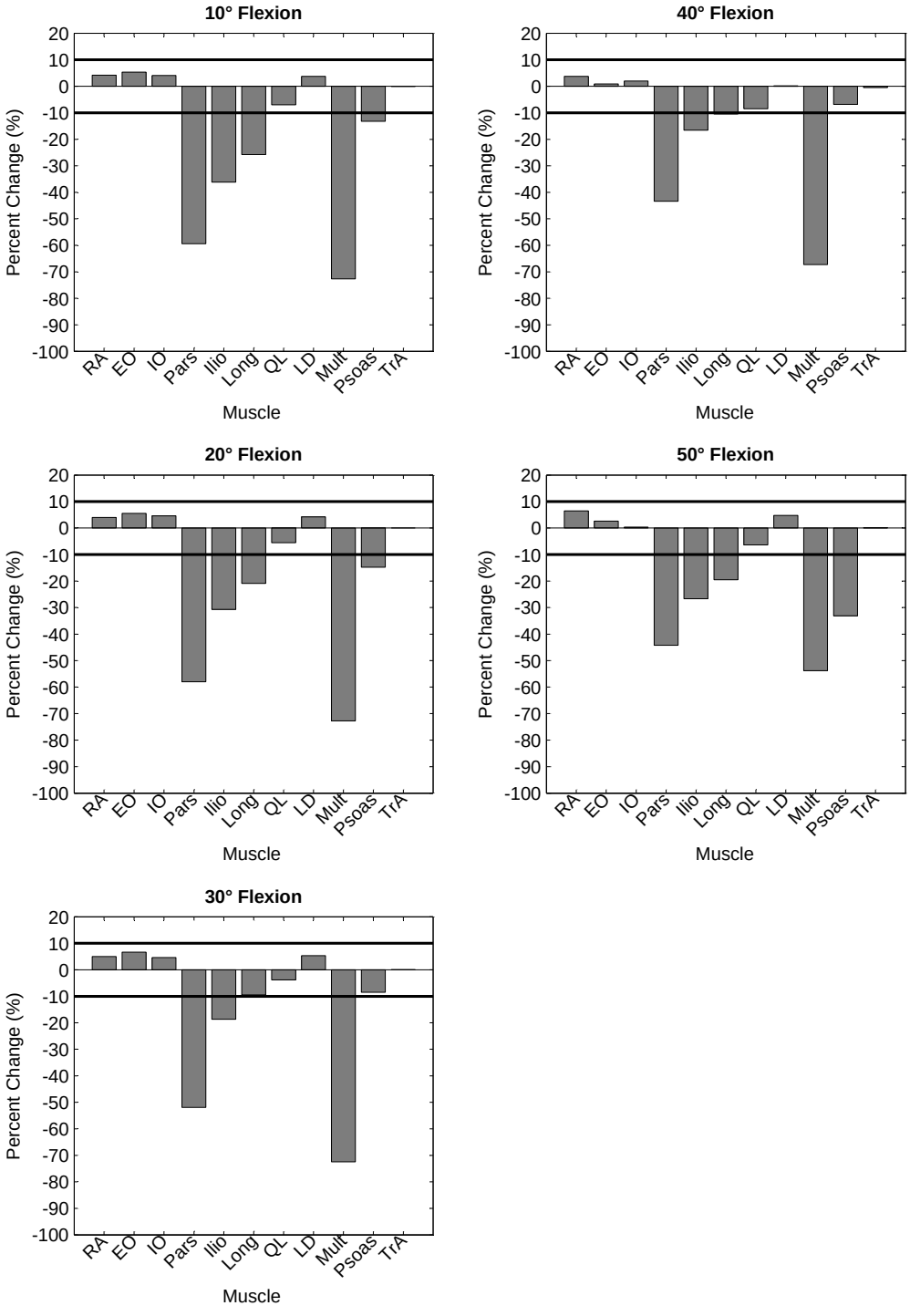
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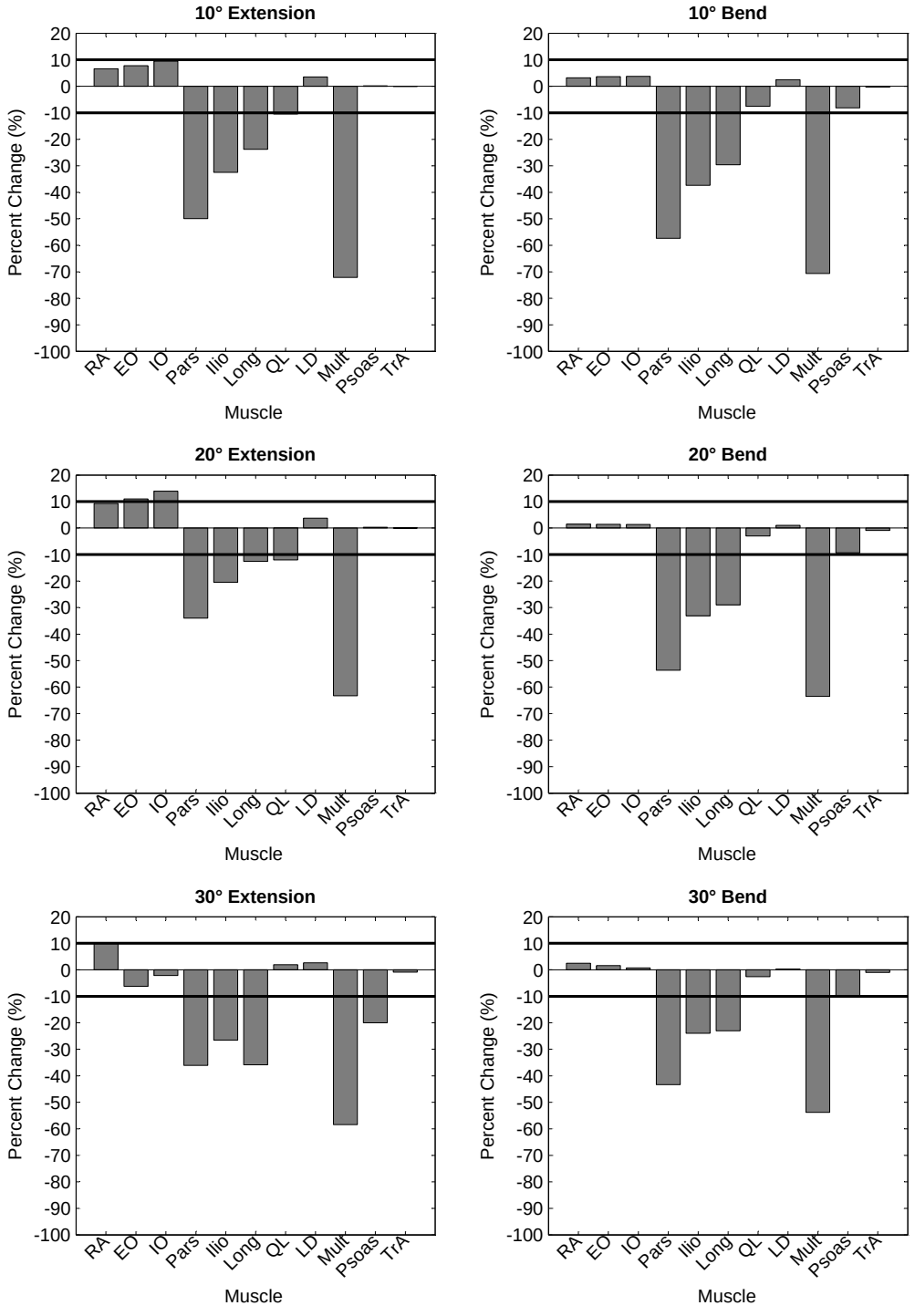
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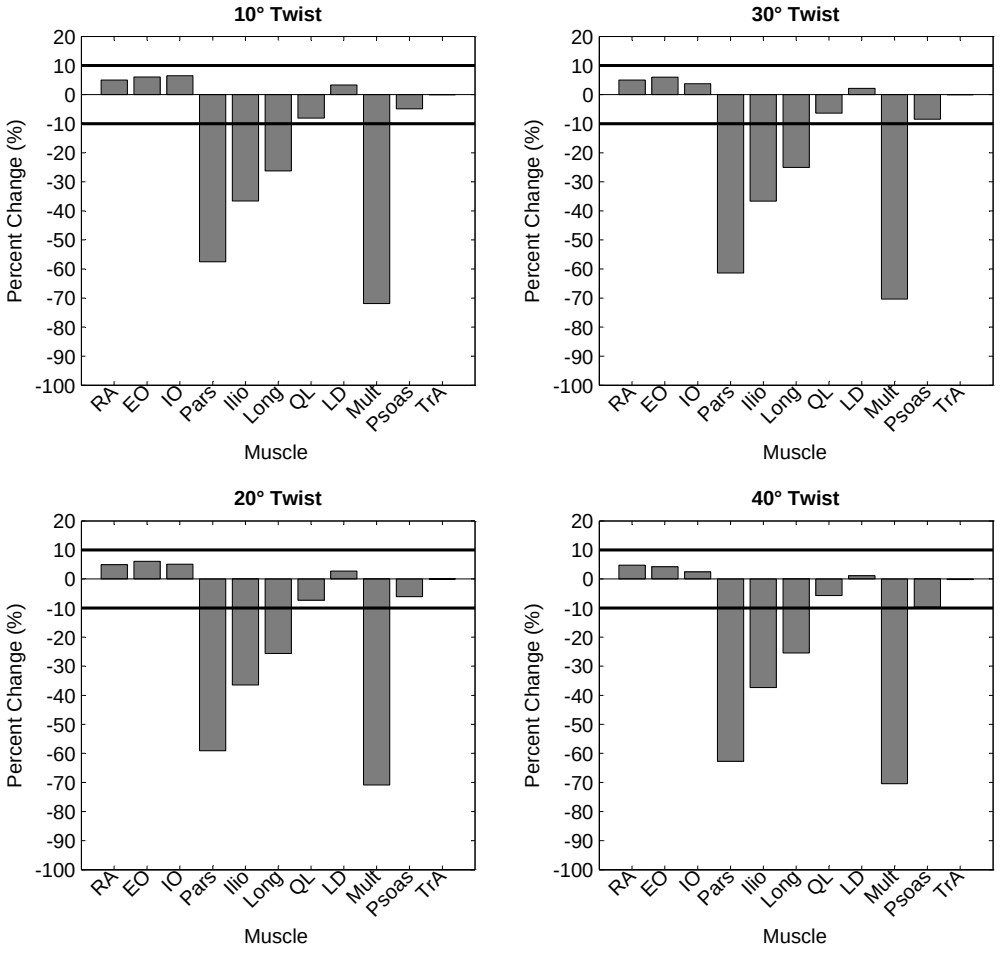
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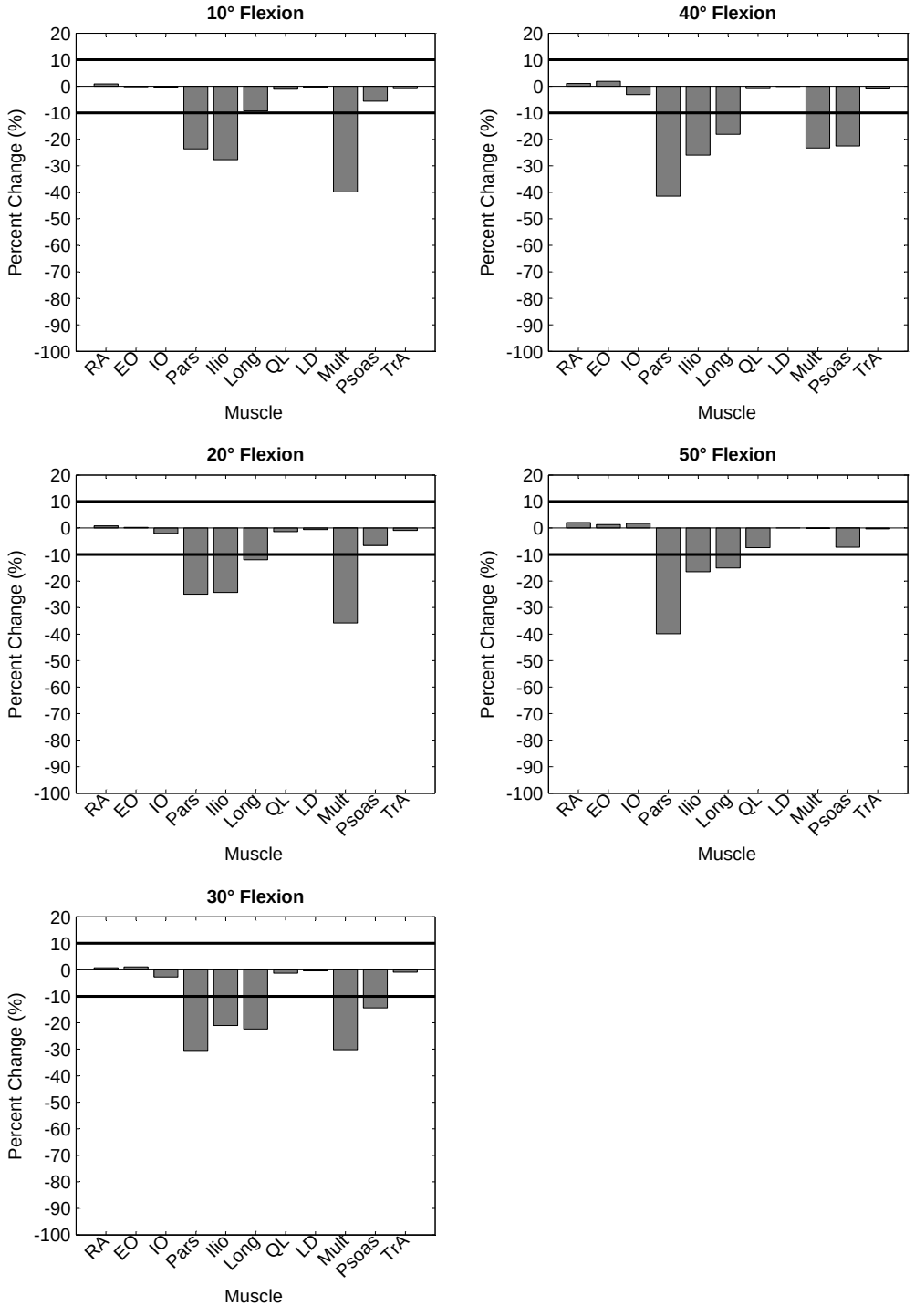
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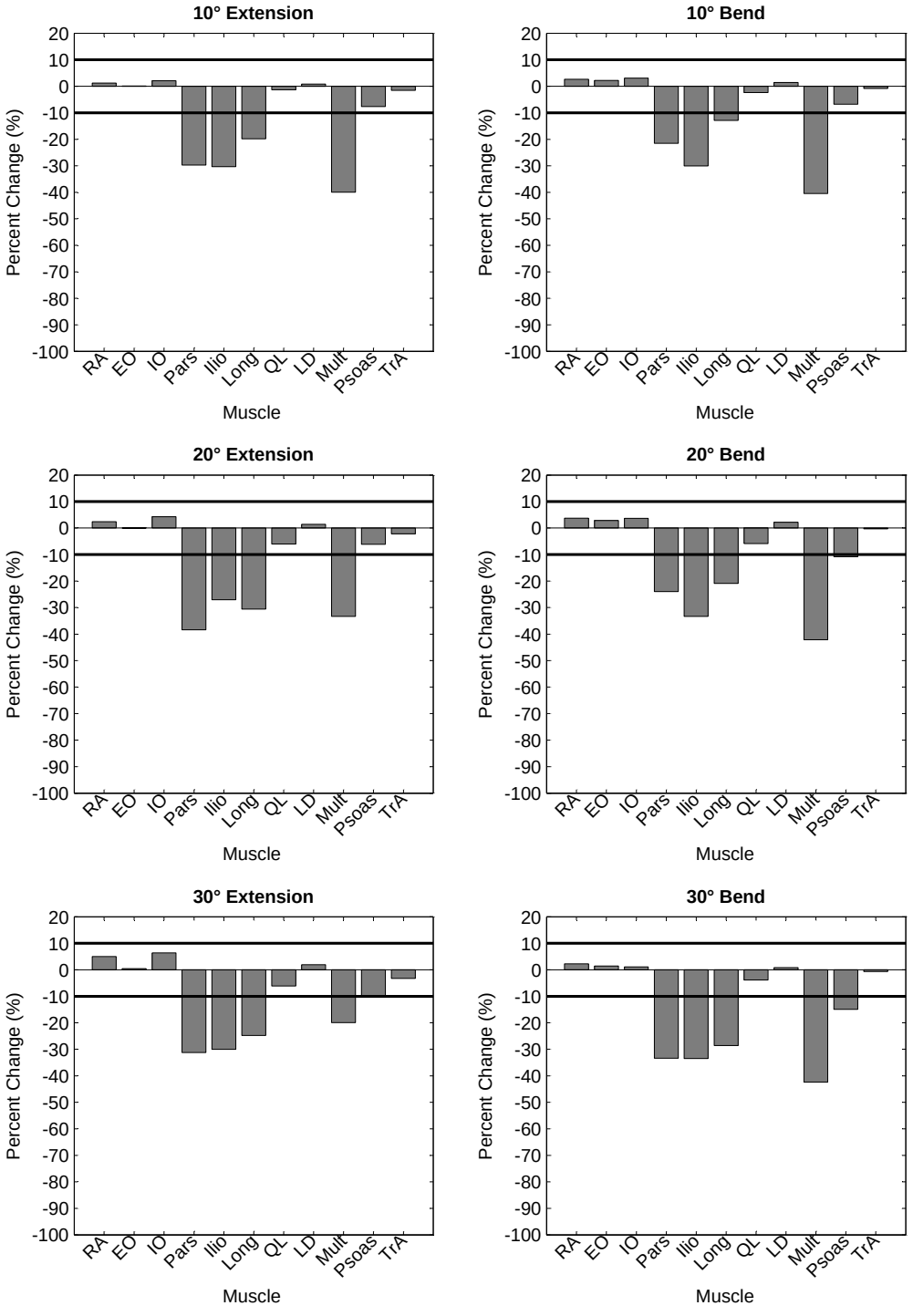
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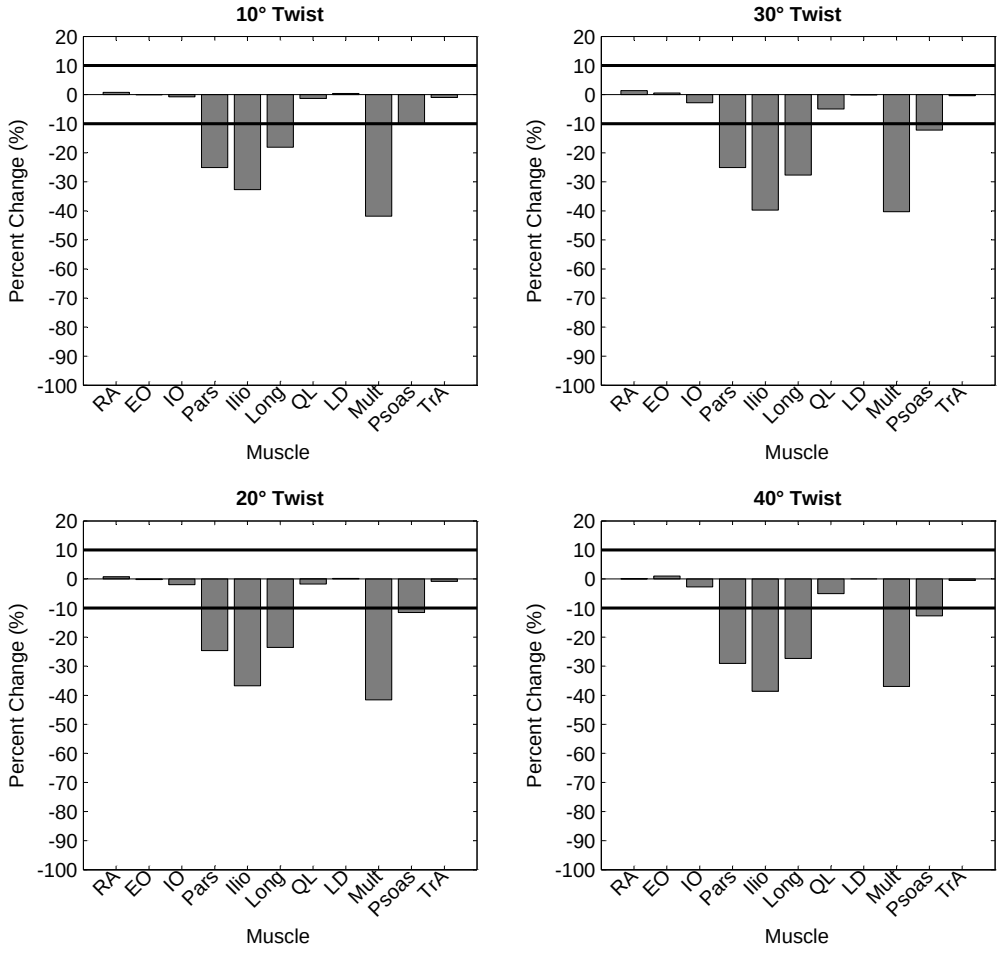
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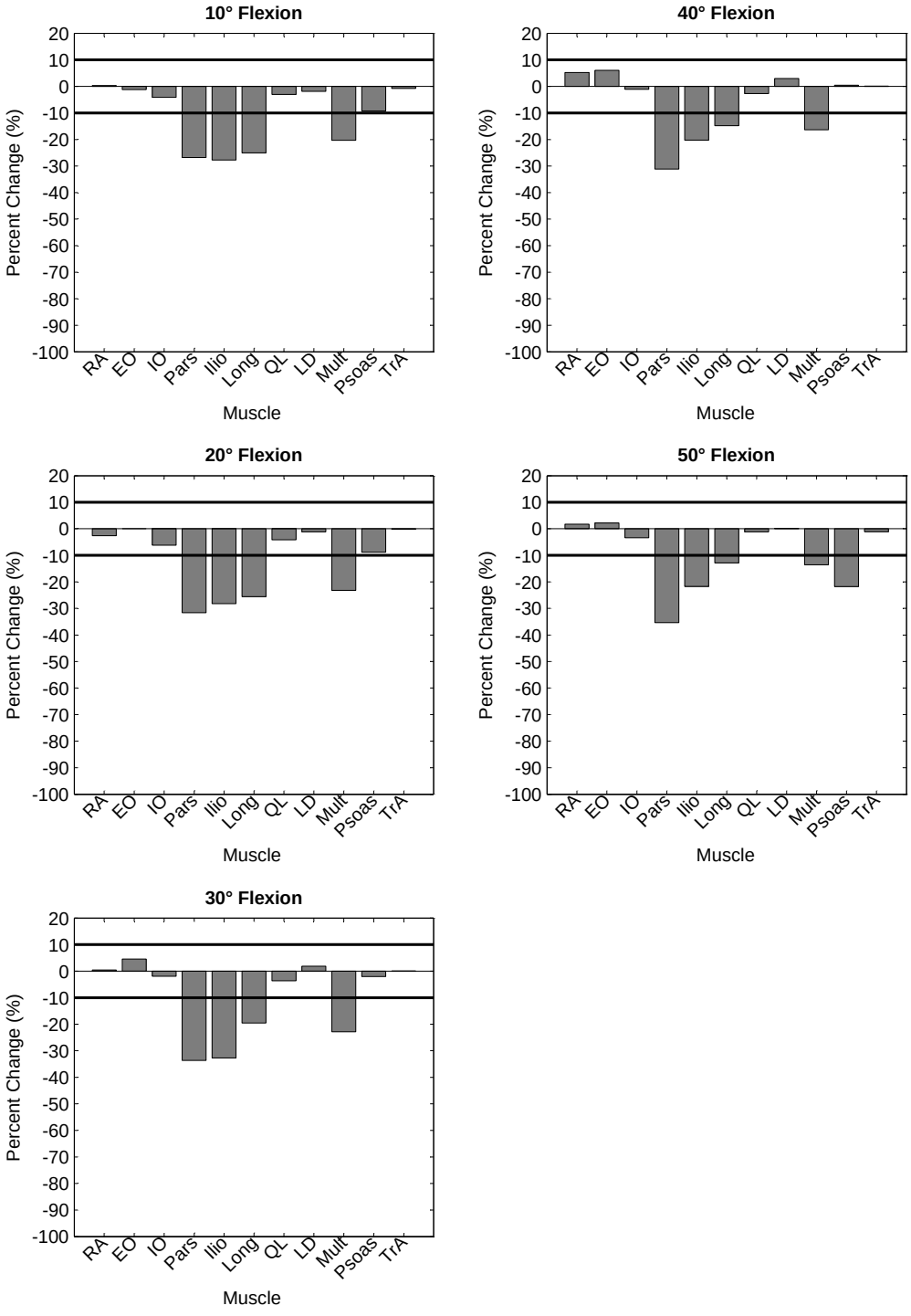
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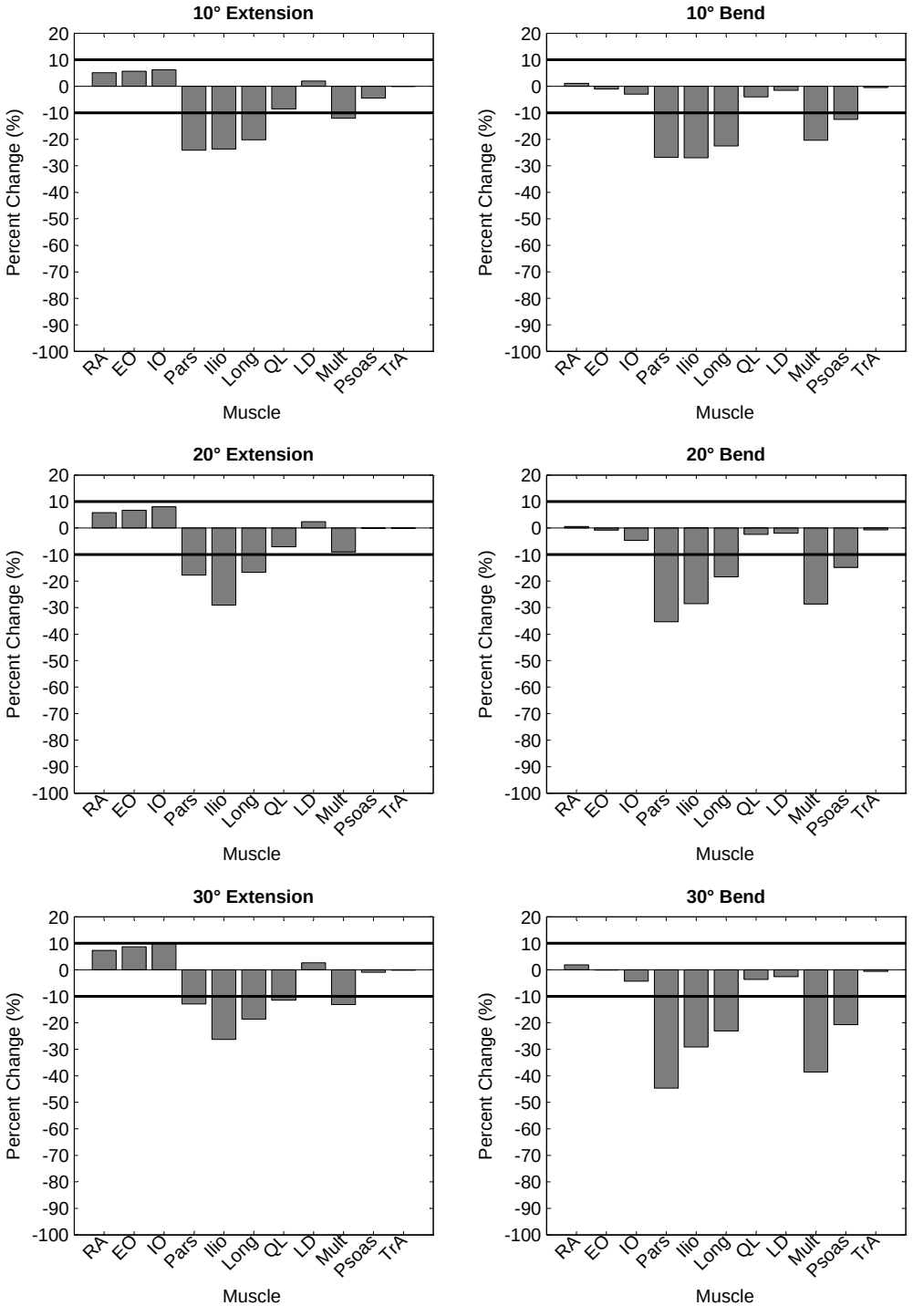
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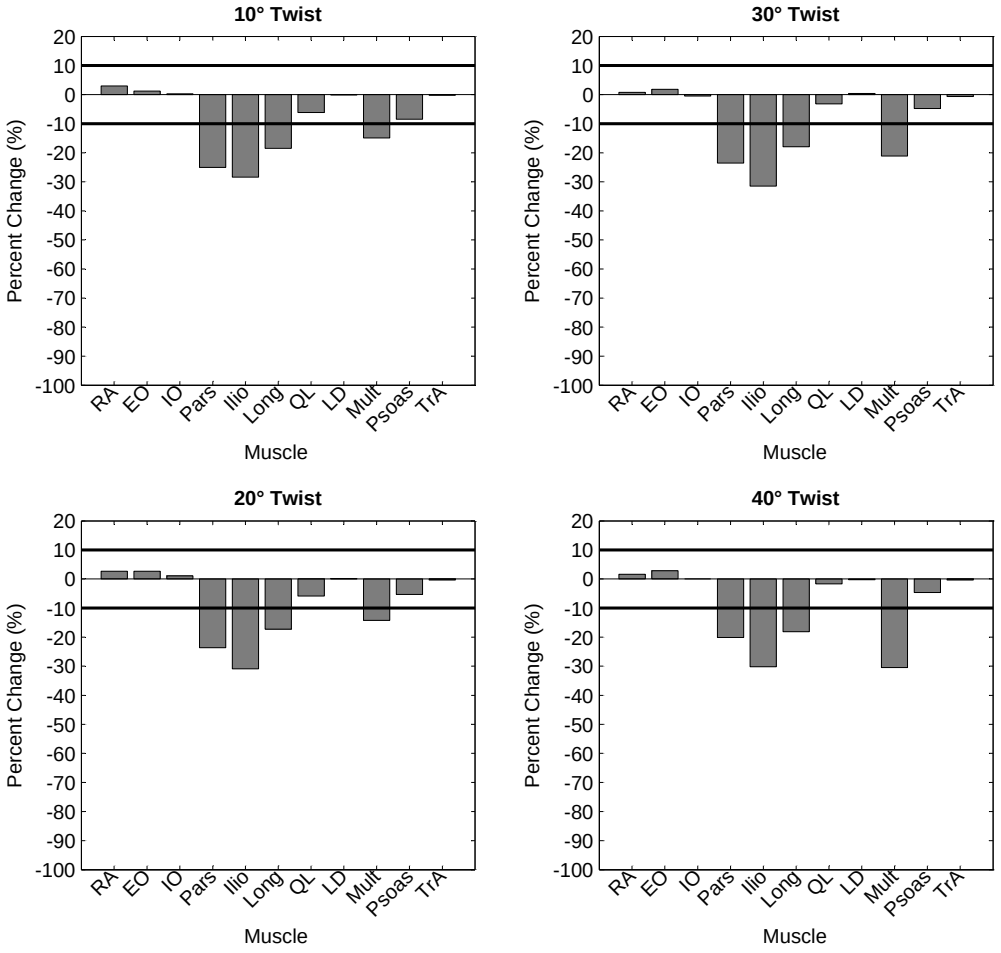
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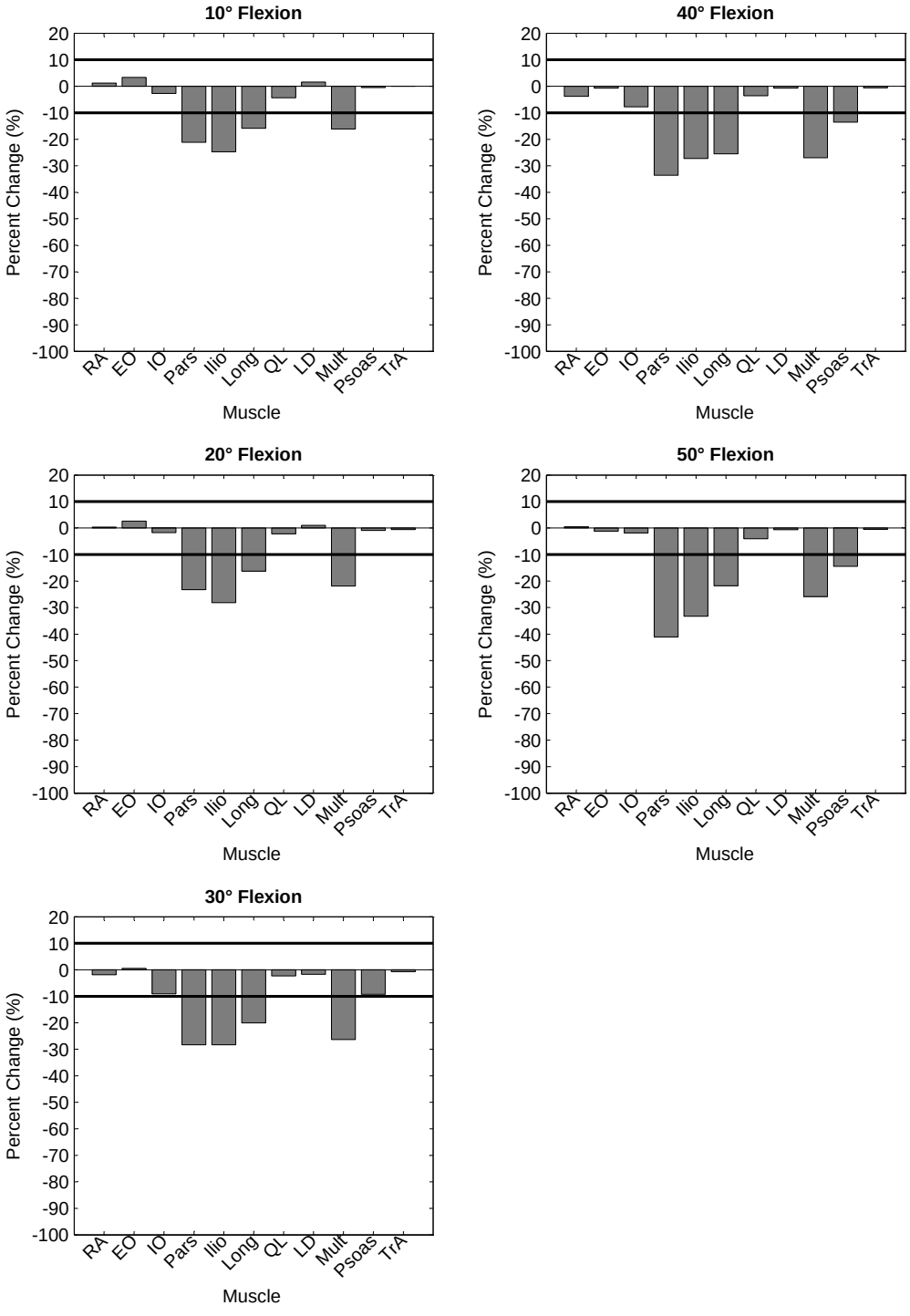
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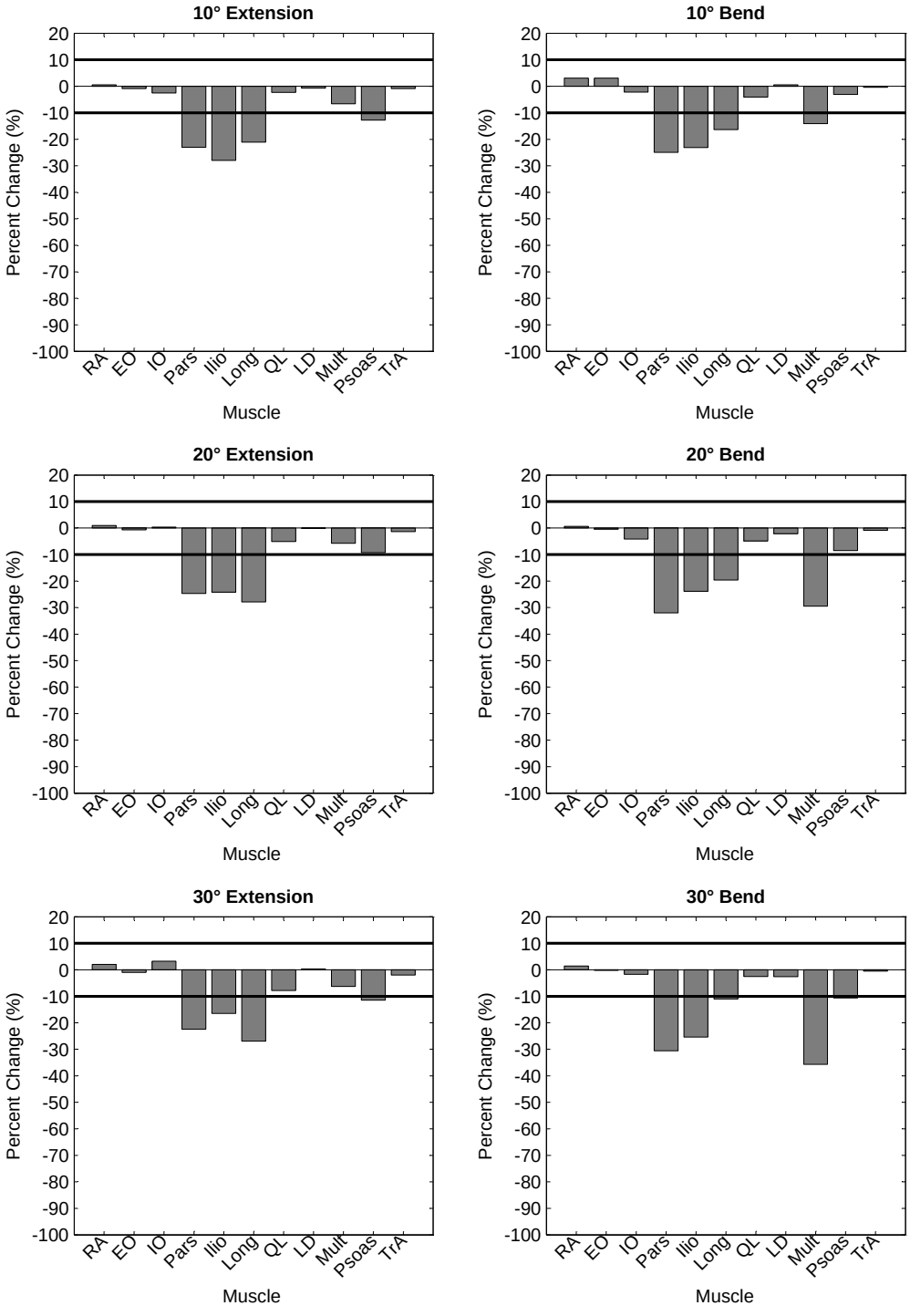
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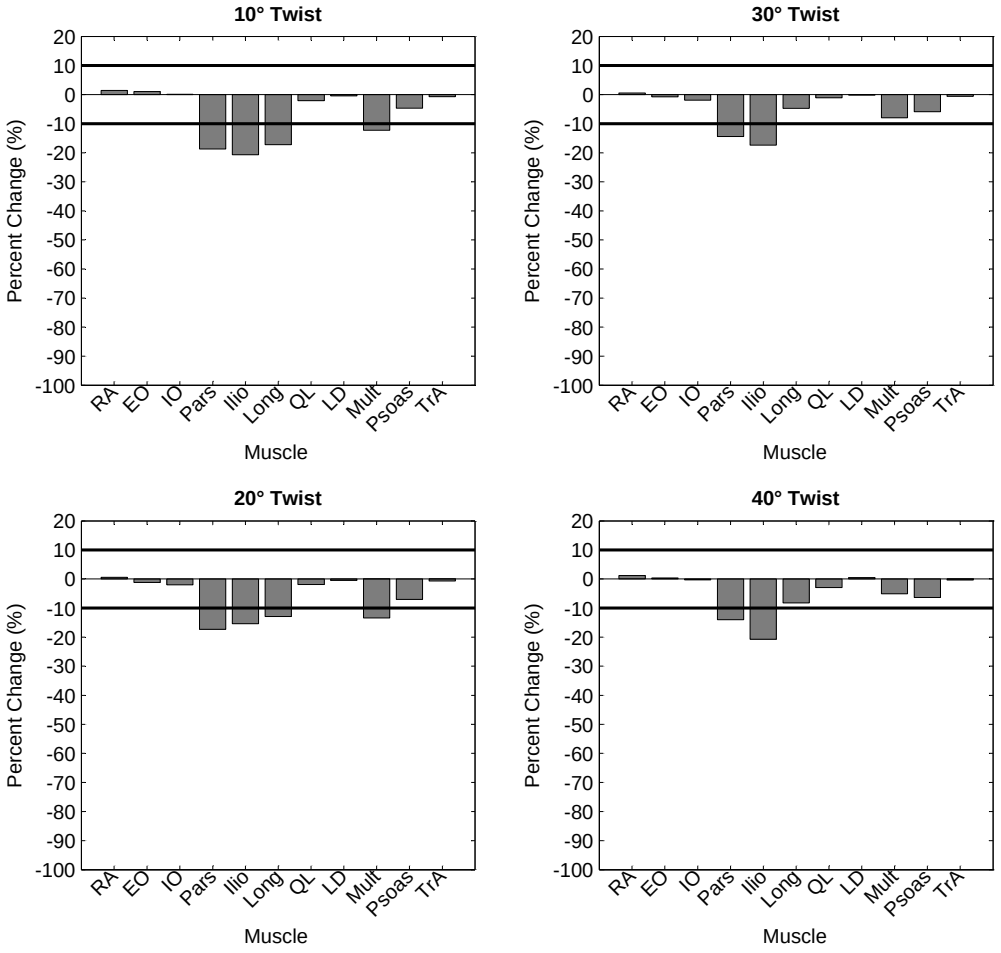
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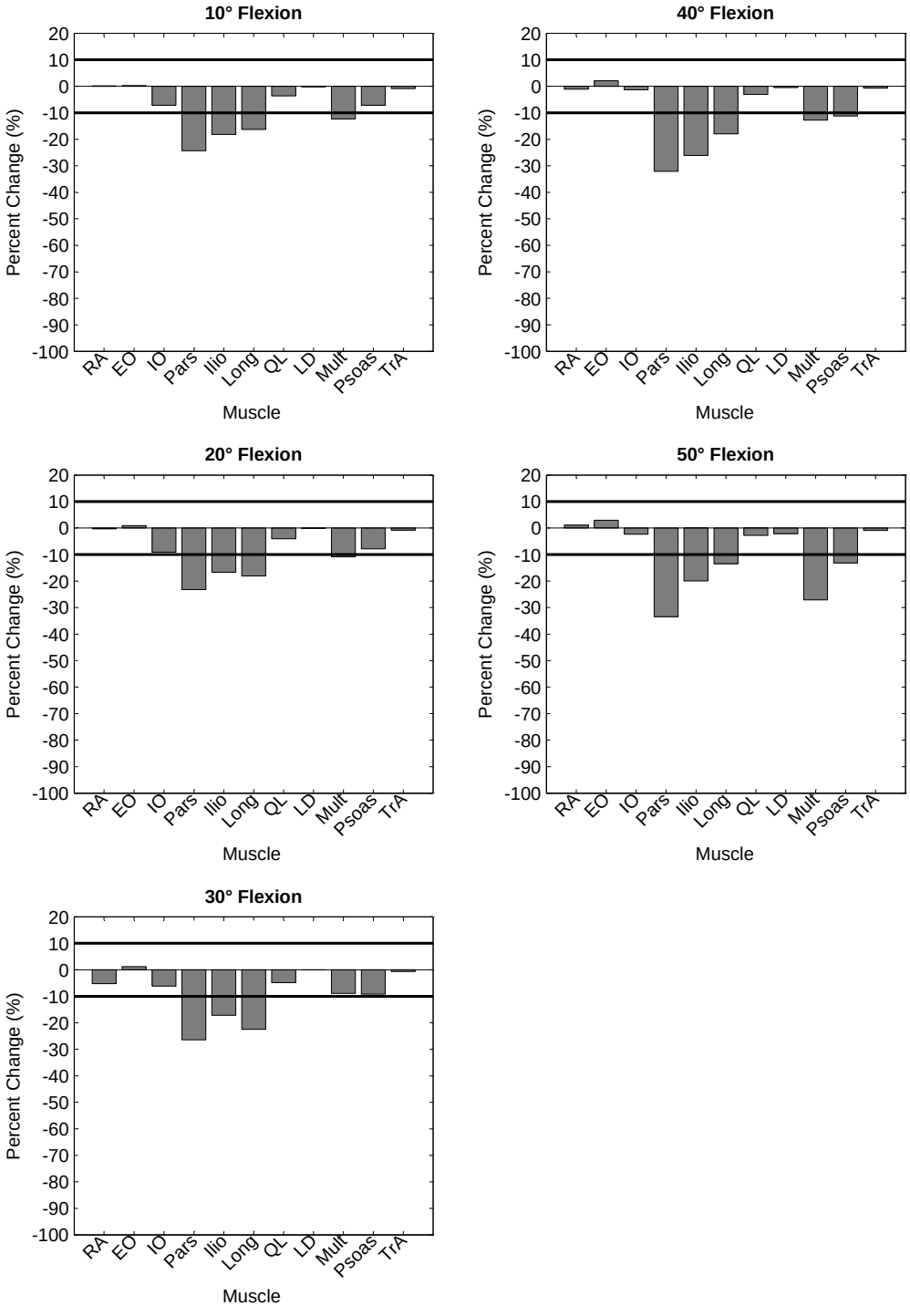
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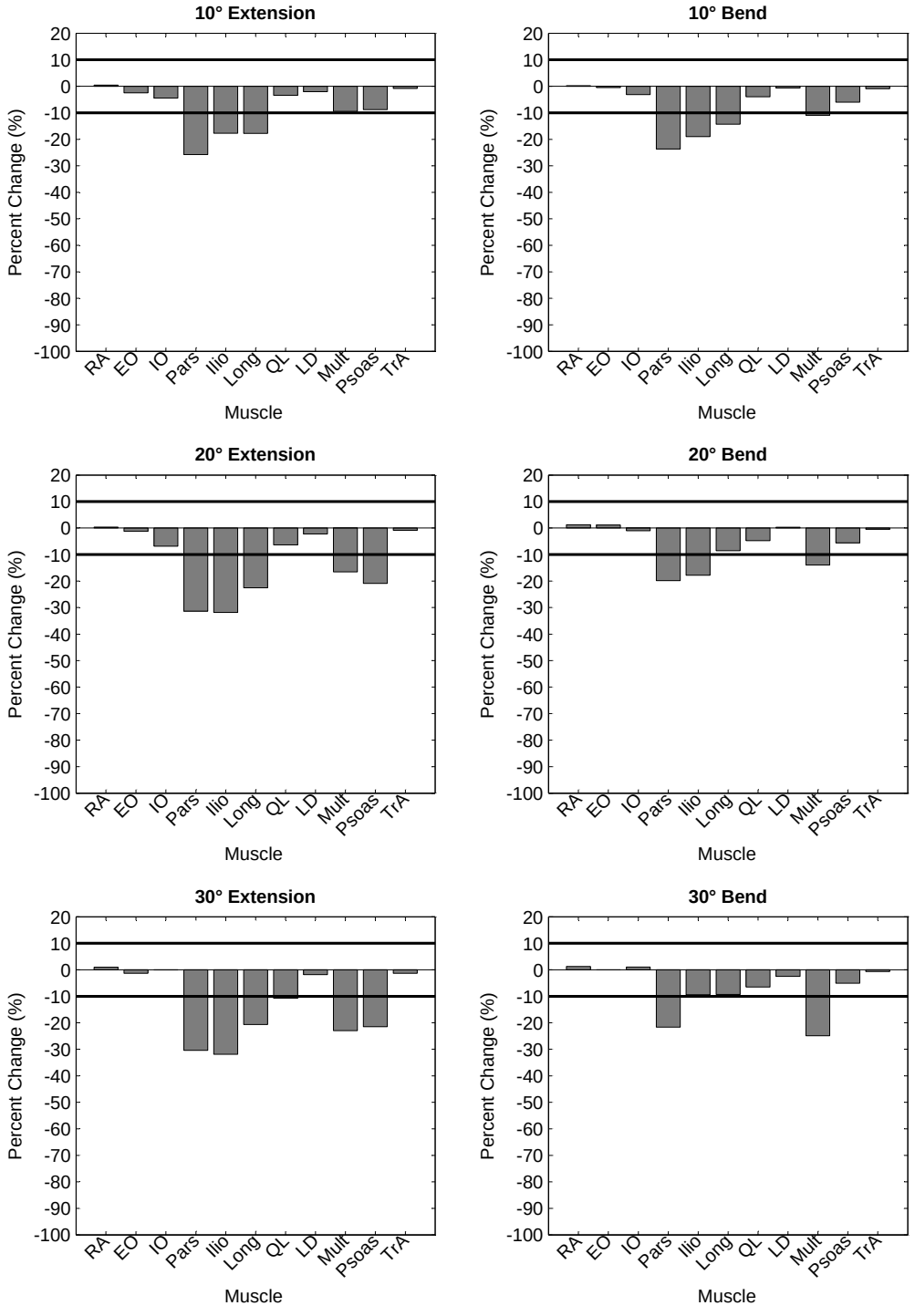
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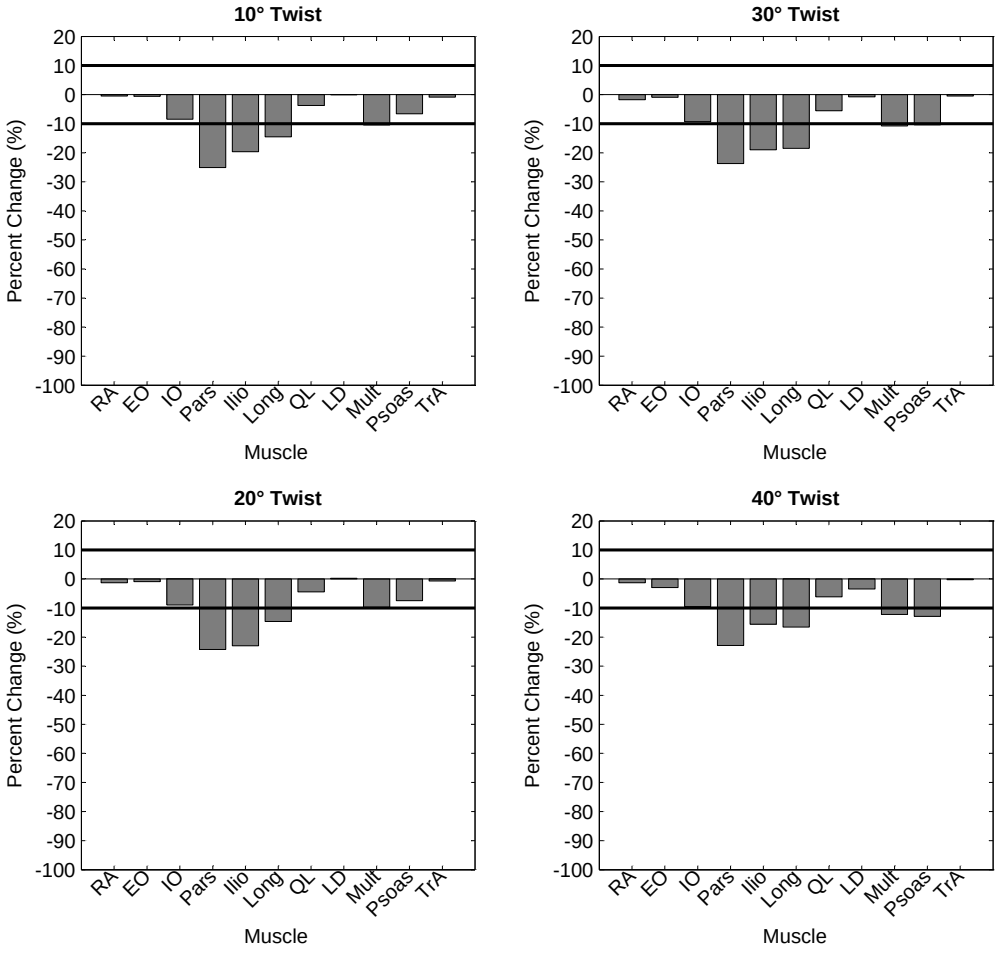
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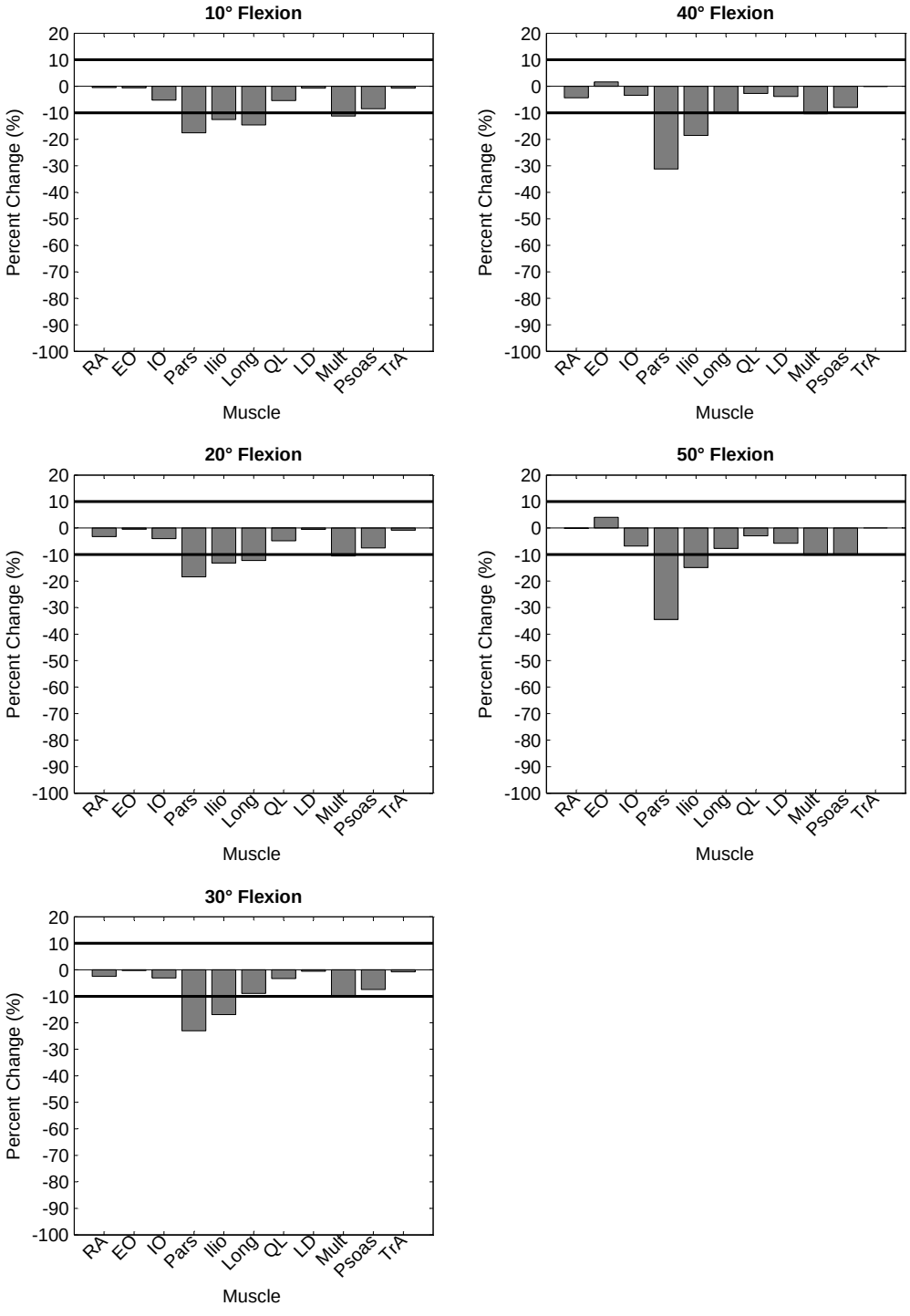
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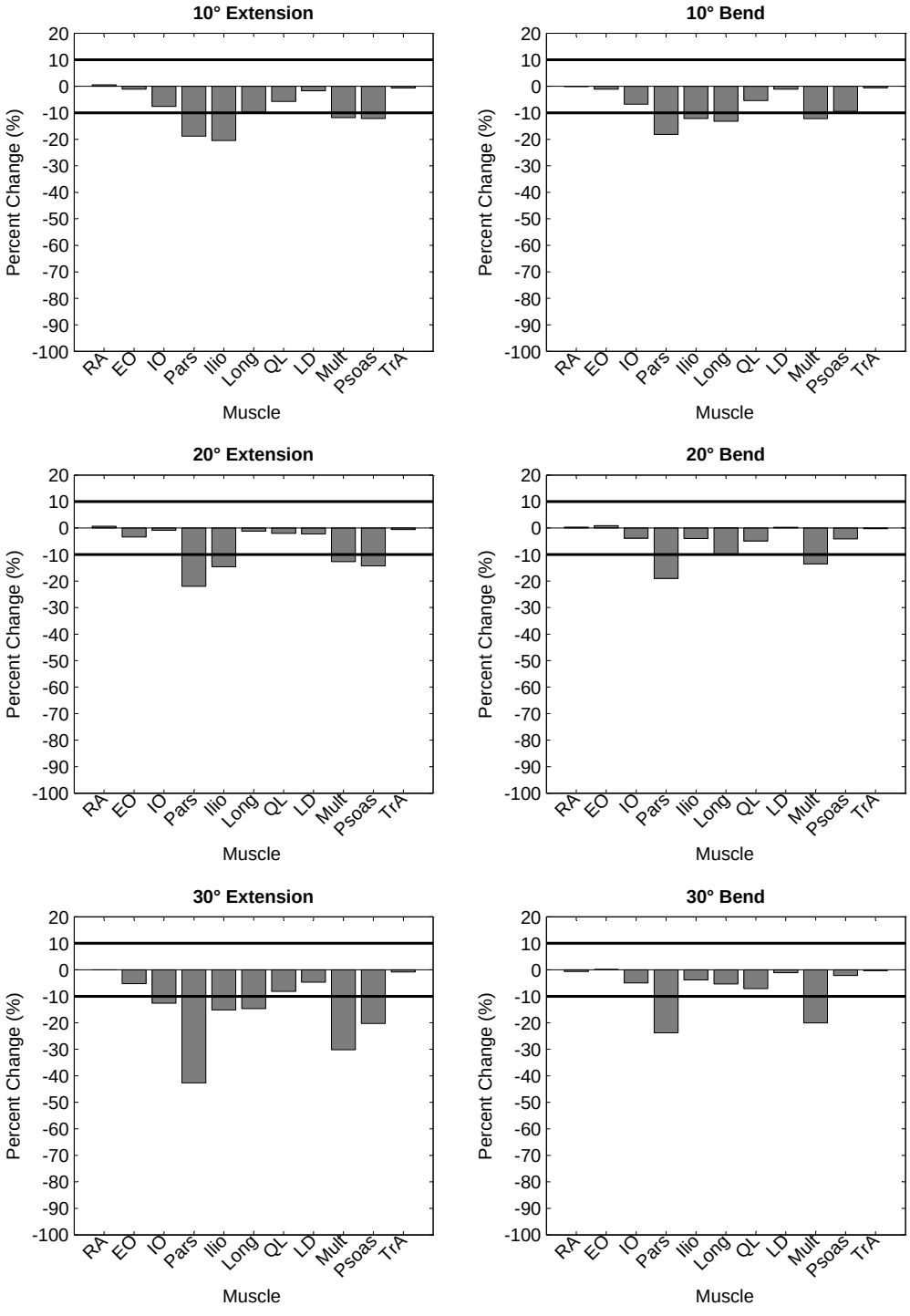
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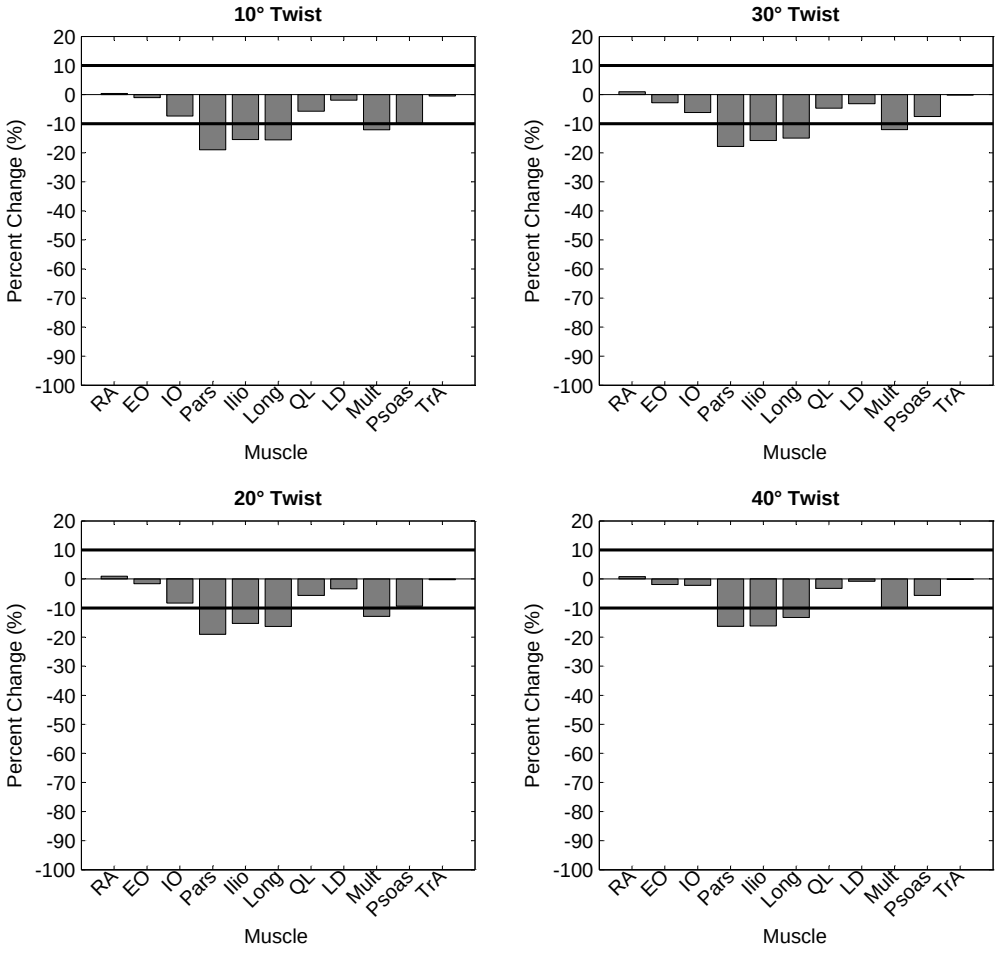
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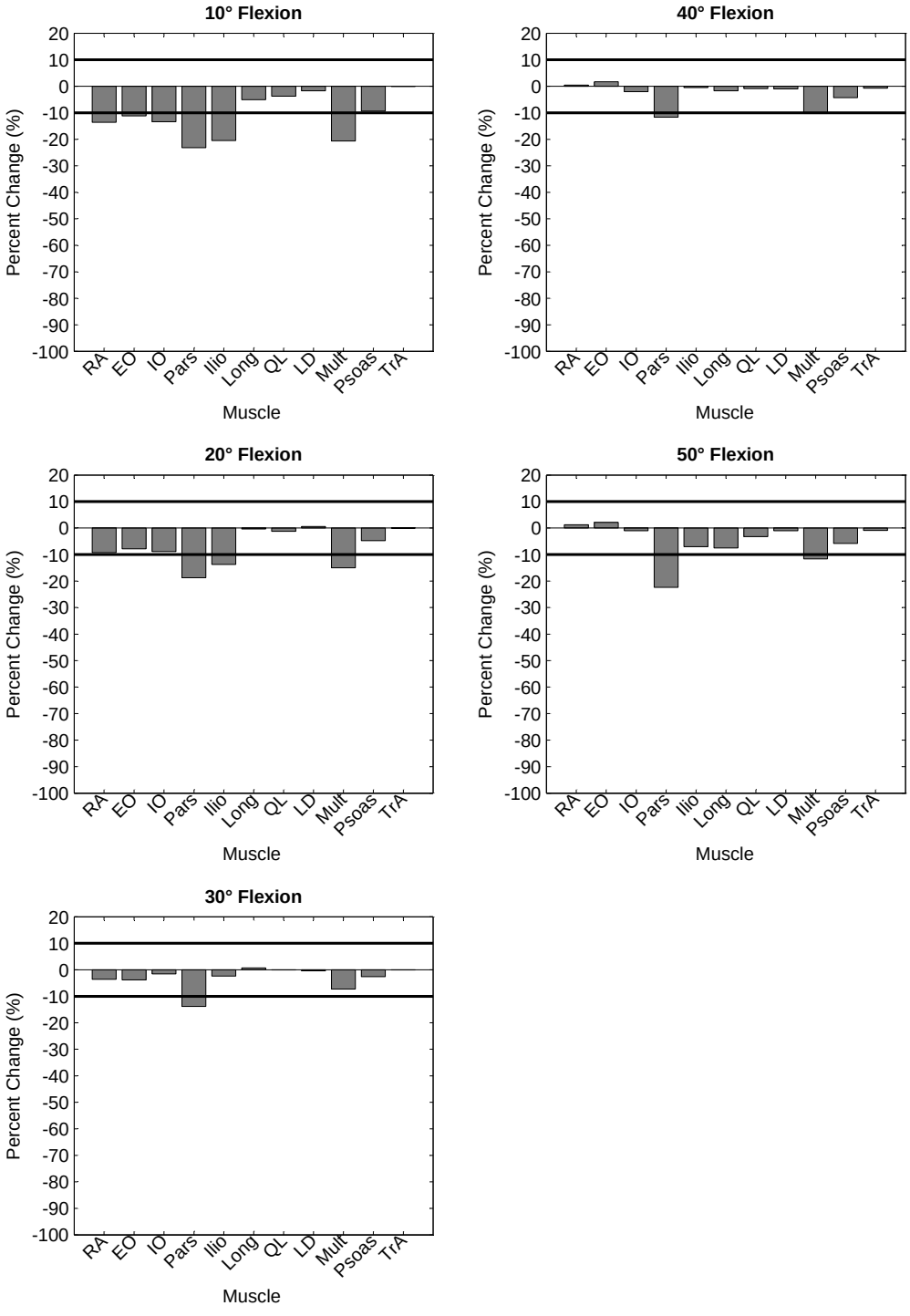
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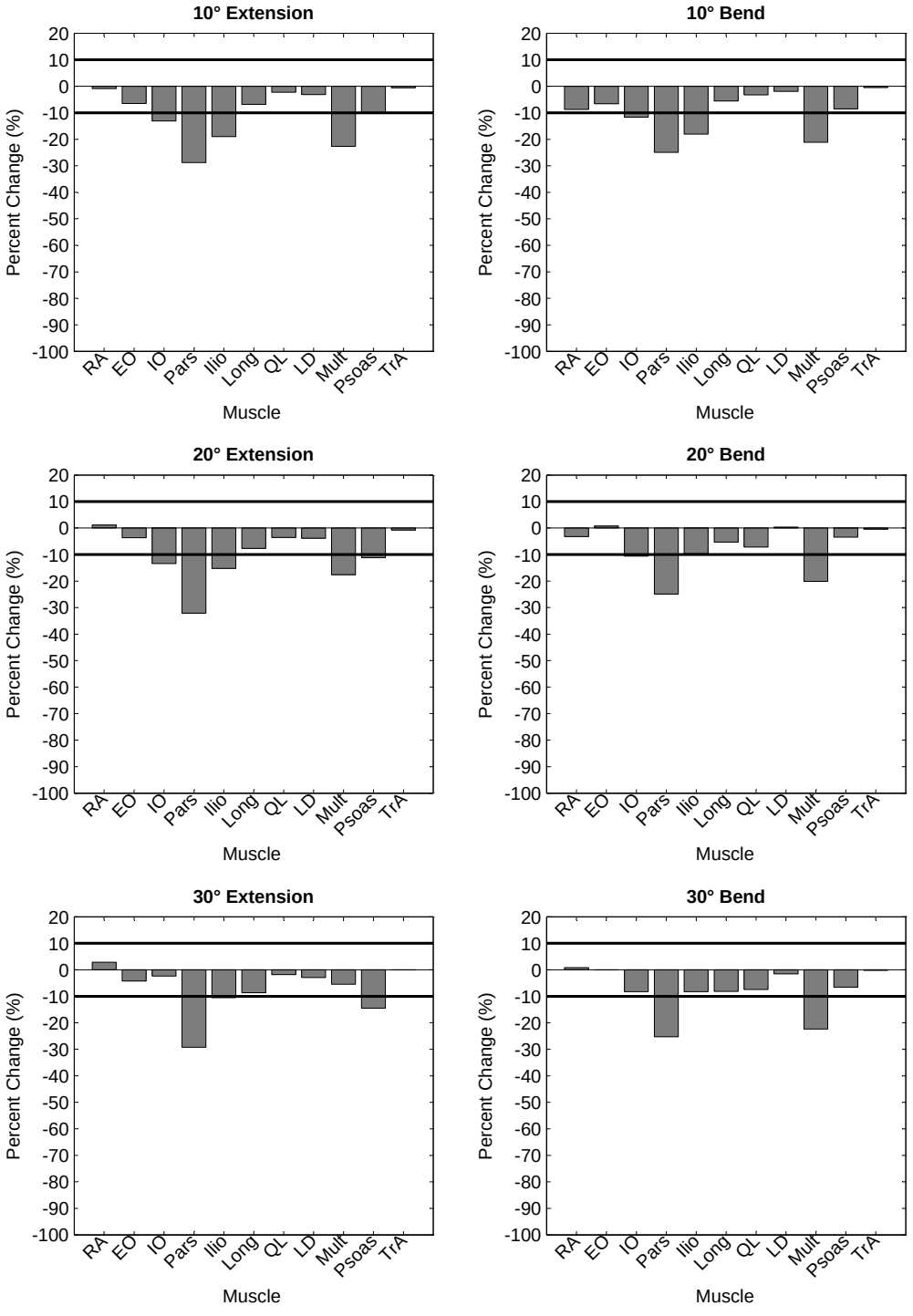
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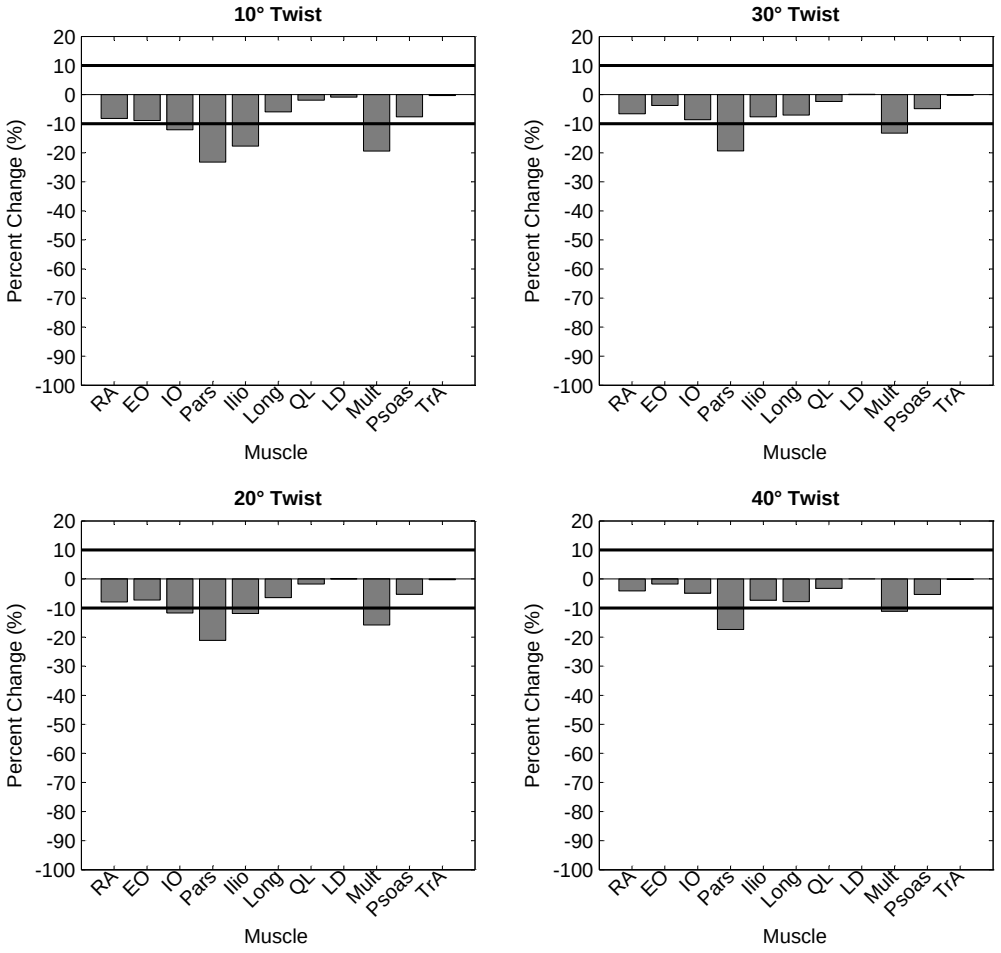
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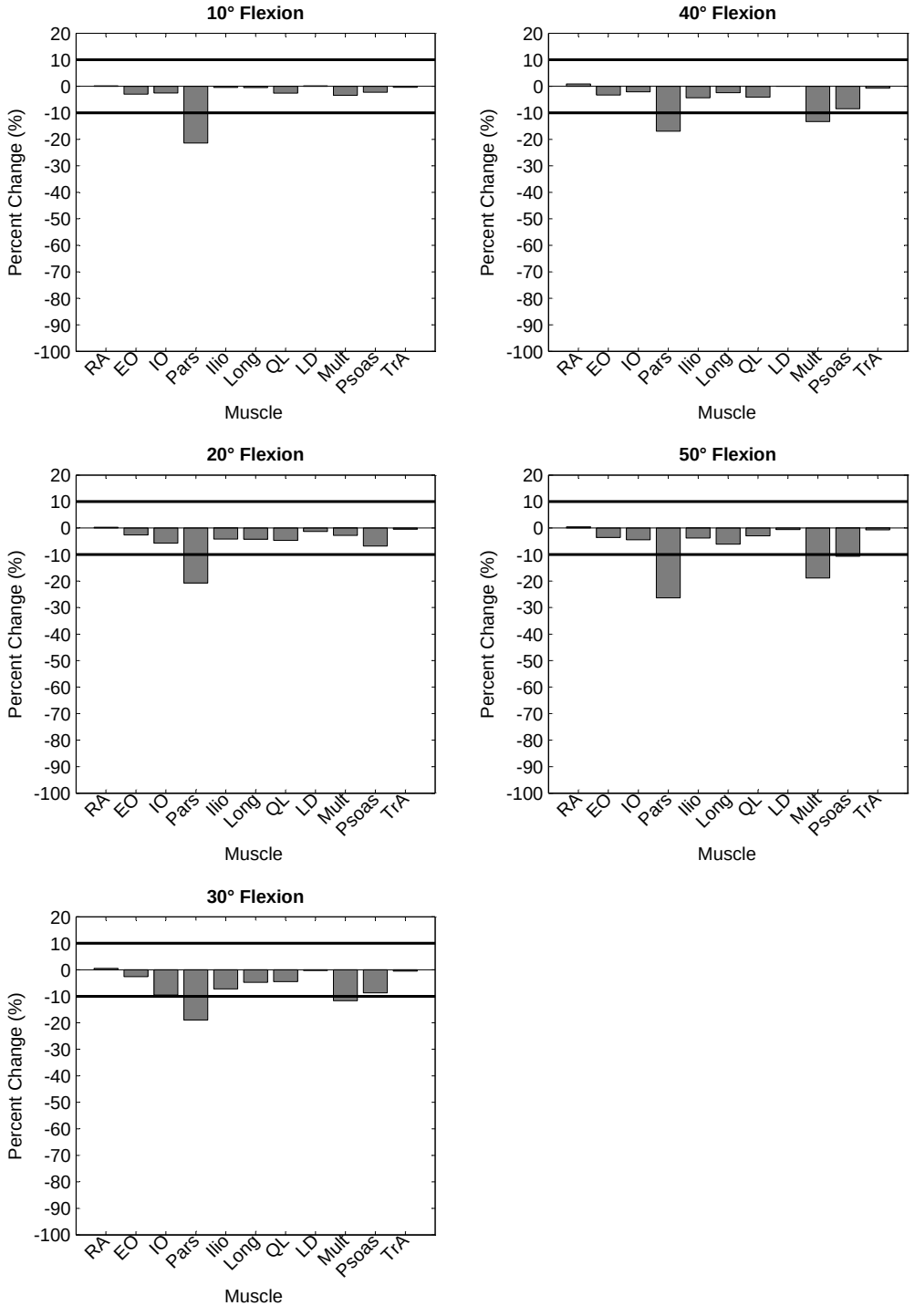
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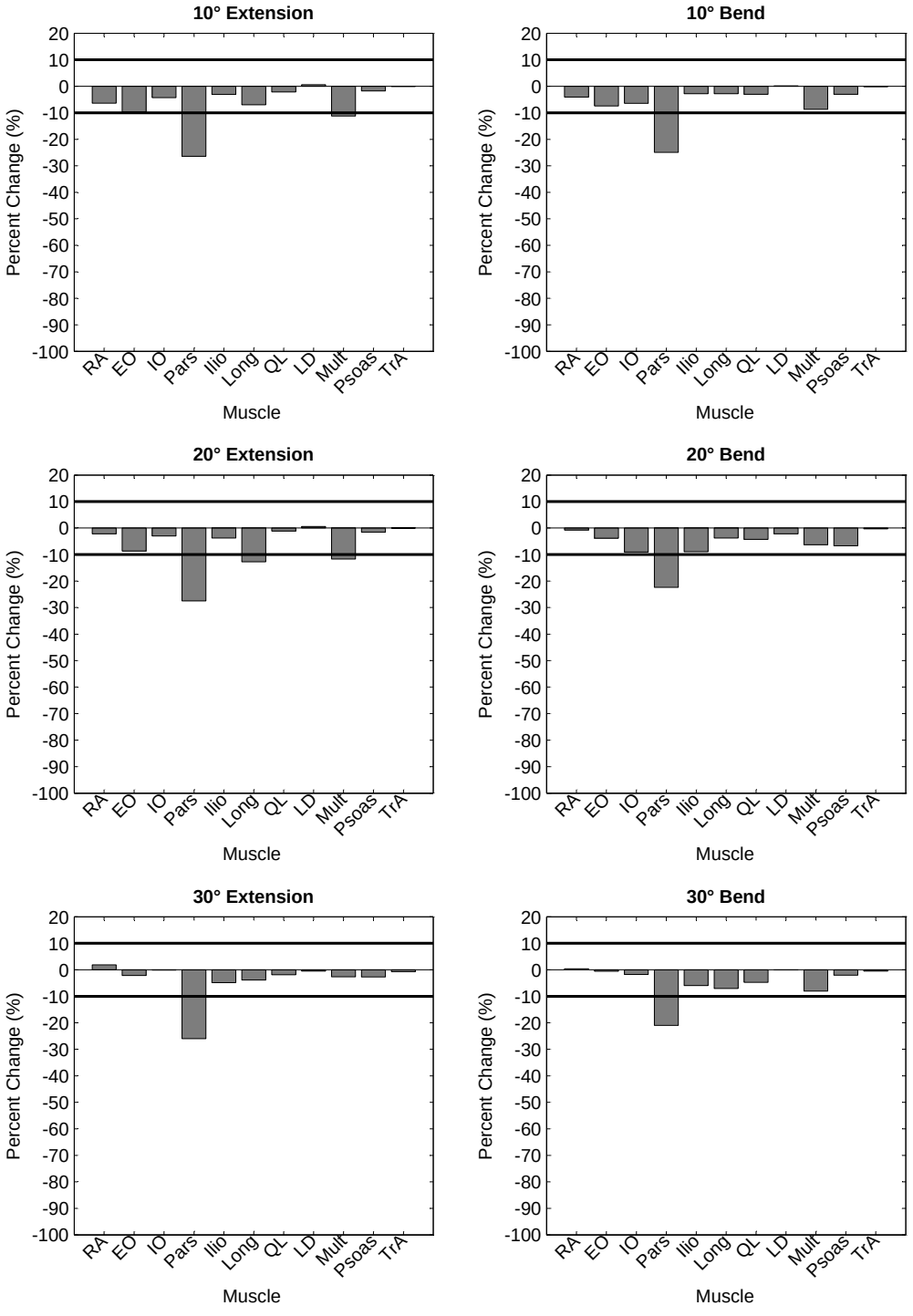
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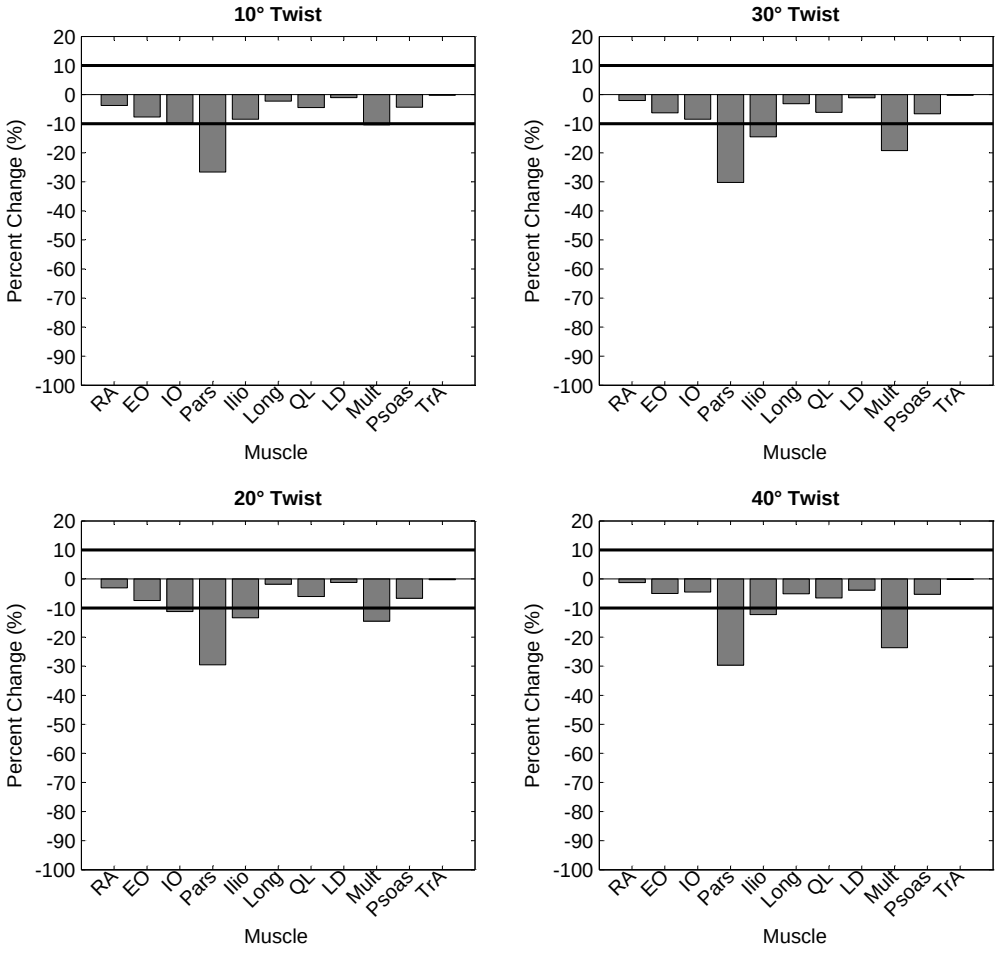
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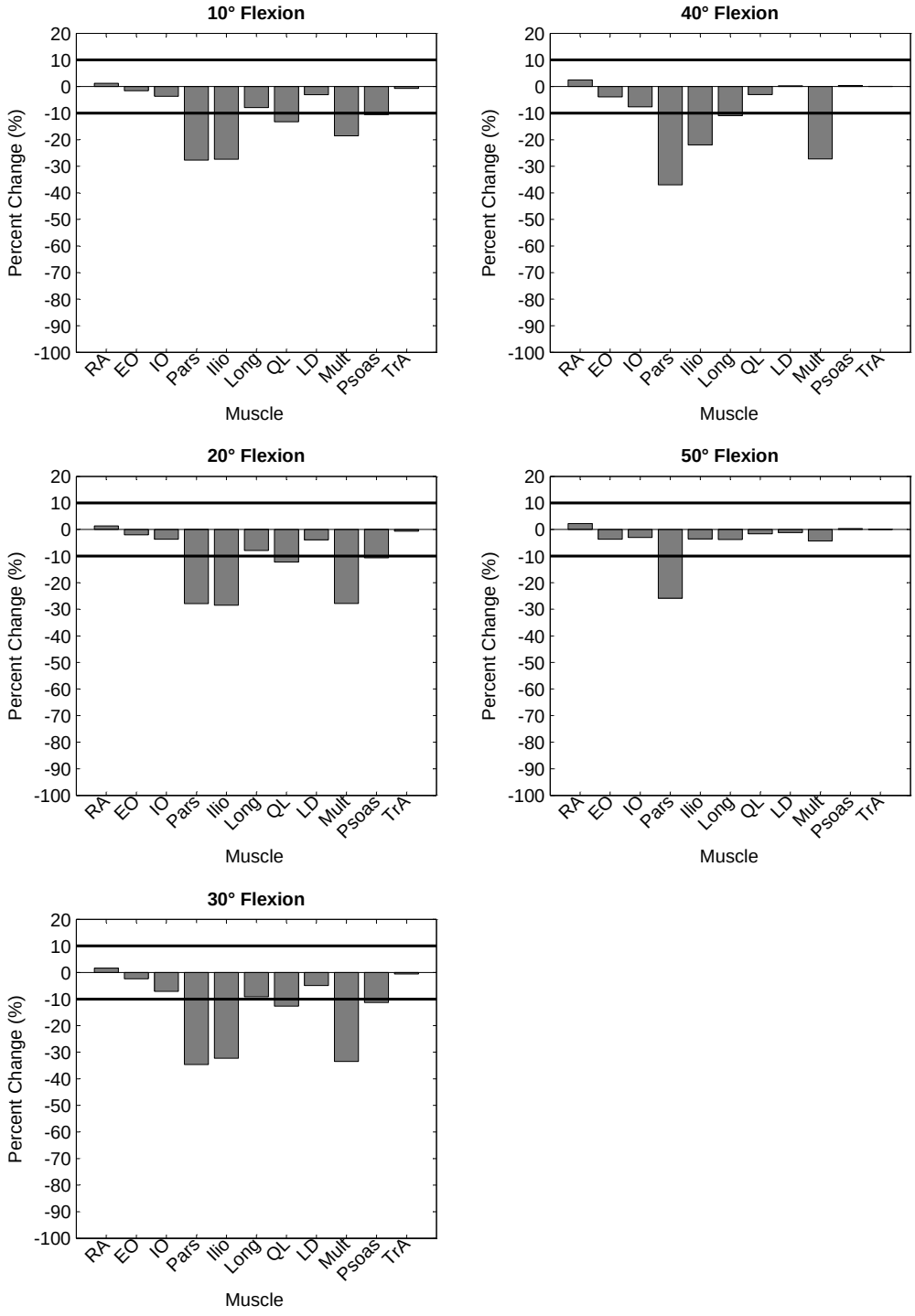
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

L3T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40° Flexion
20° Flexion
50° Flexion
30° Flexion
Percent Change (%)
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10
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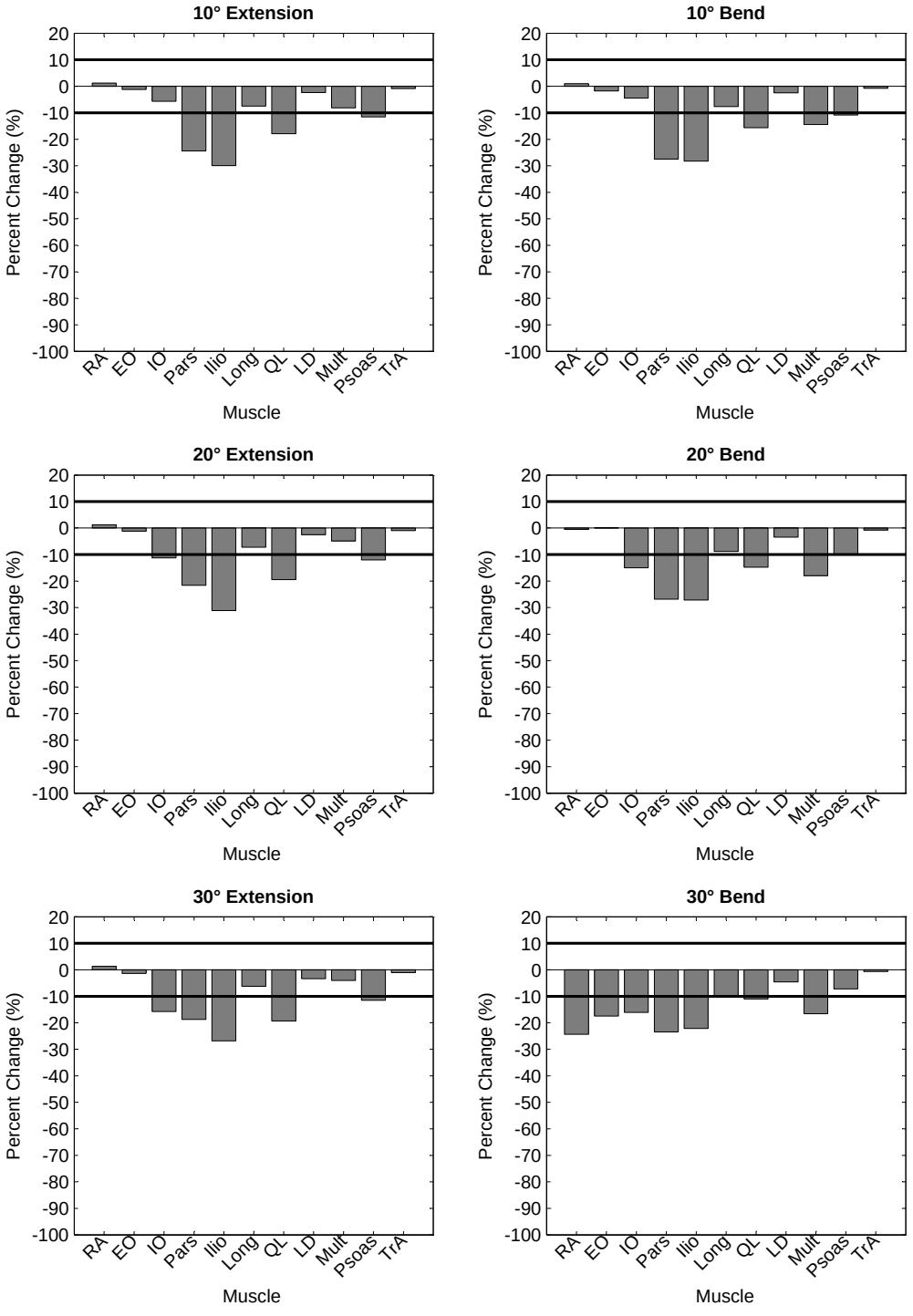
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

L3T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10° Bend
20° Extension
20° Bend
30° Extension
30° Bend
Percent Change (%)
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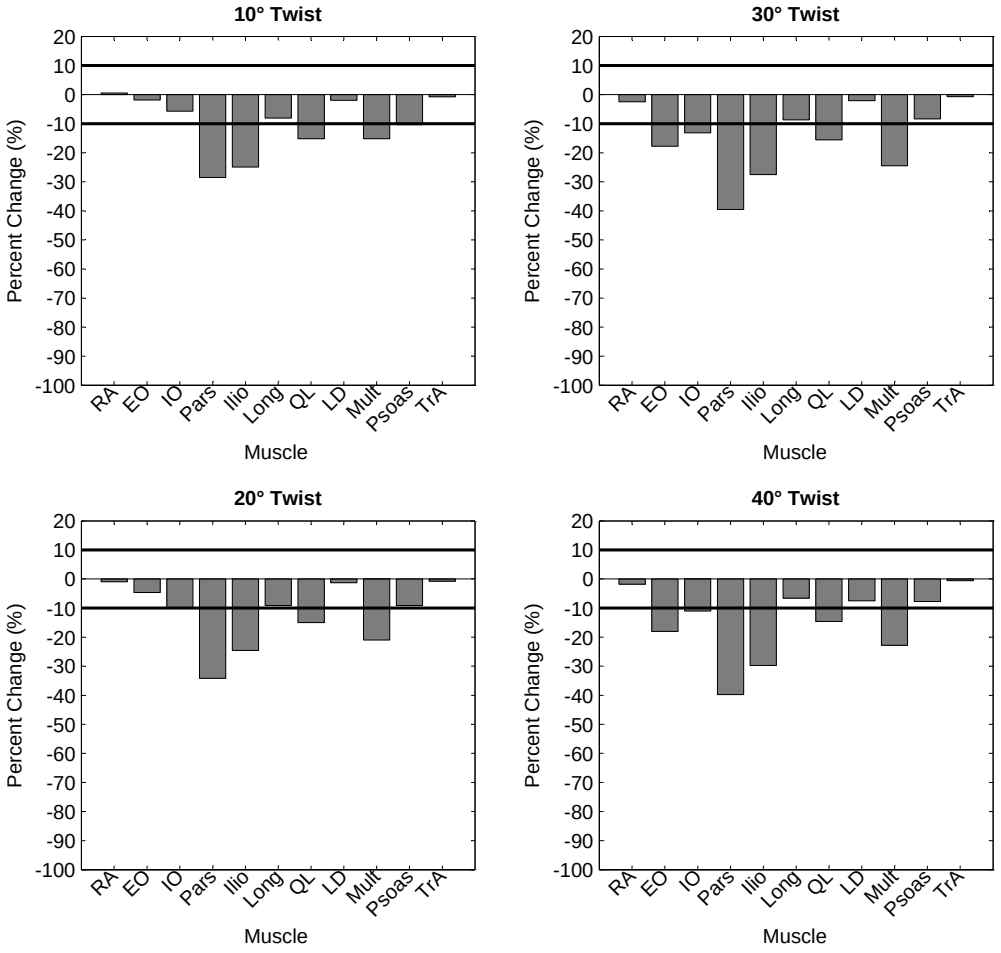
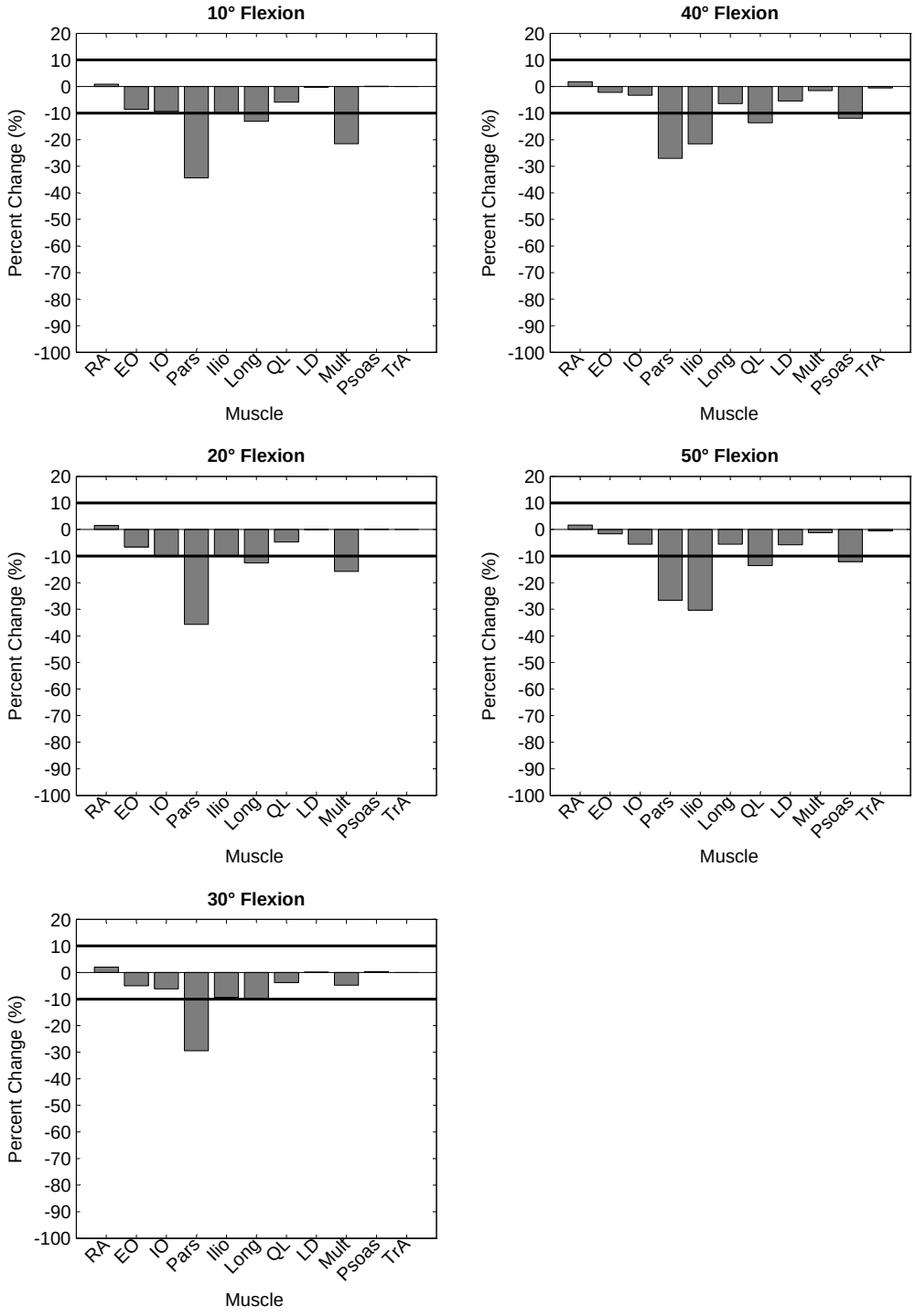
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

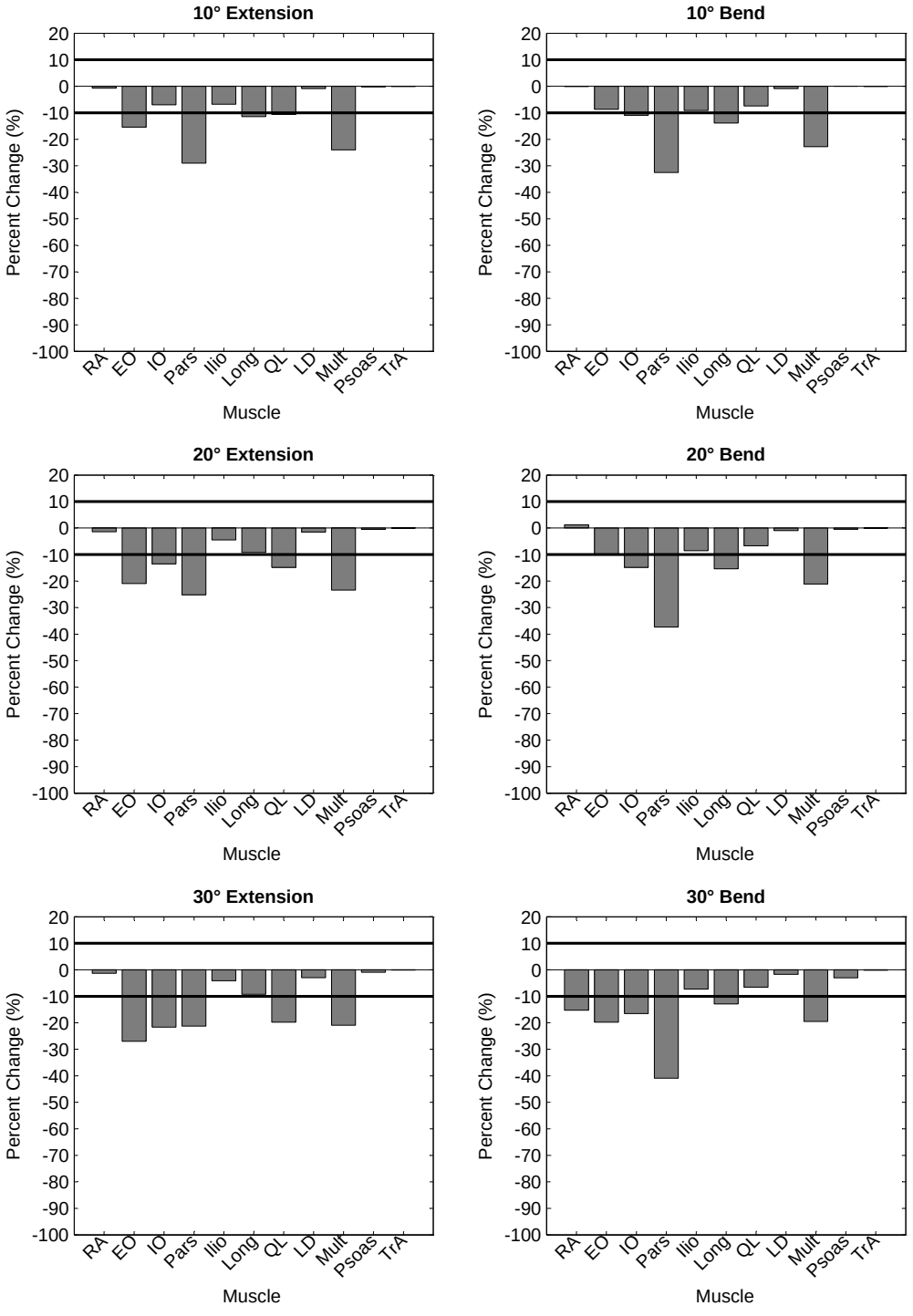
L3T


10° Twist
30° Twist
20° Twist
40° Twist
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

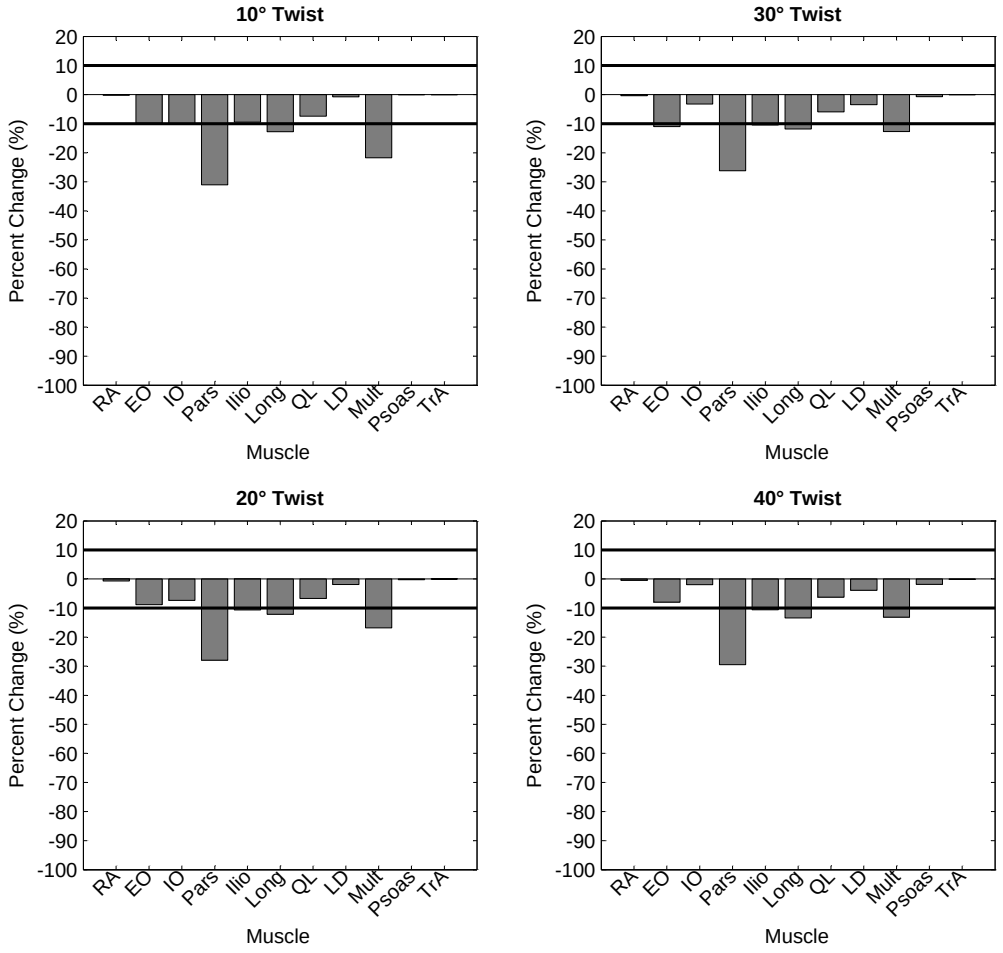
L4F


10° Flexion
40° Flexion
20° Flexion
50° Flexion
30° Flexion
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

L4F


10° Extension
10° Bend
20° Extension
20° Bend
30° Extension
30° Bend
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

L4F


10° Twist
30° Twist
20° Twist
40° Twist
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
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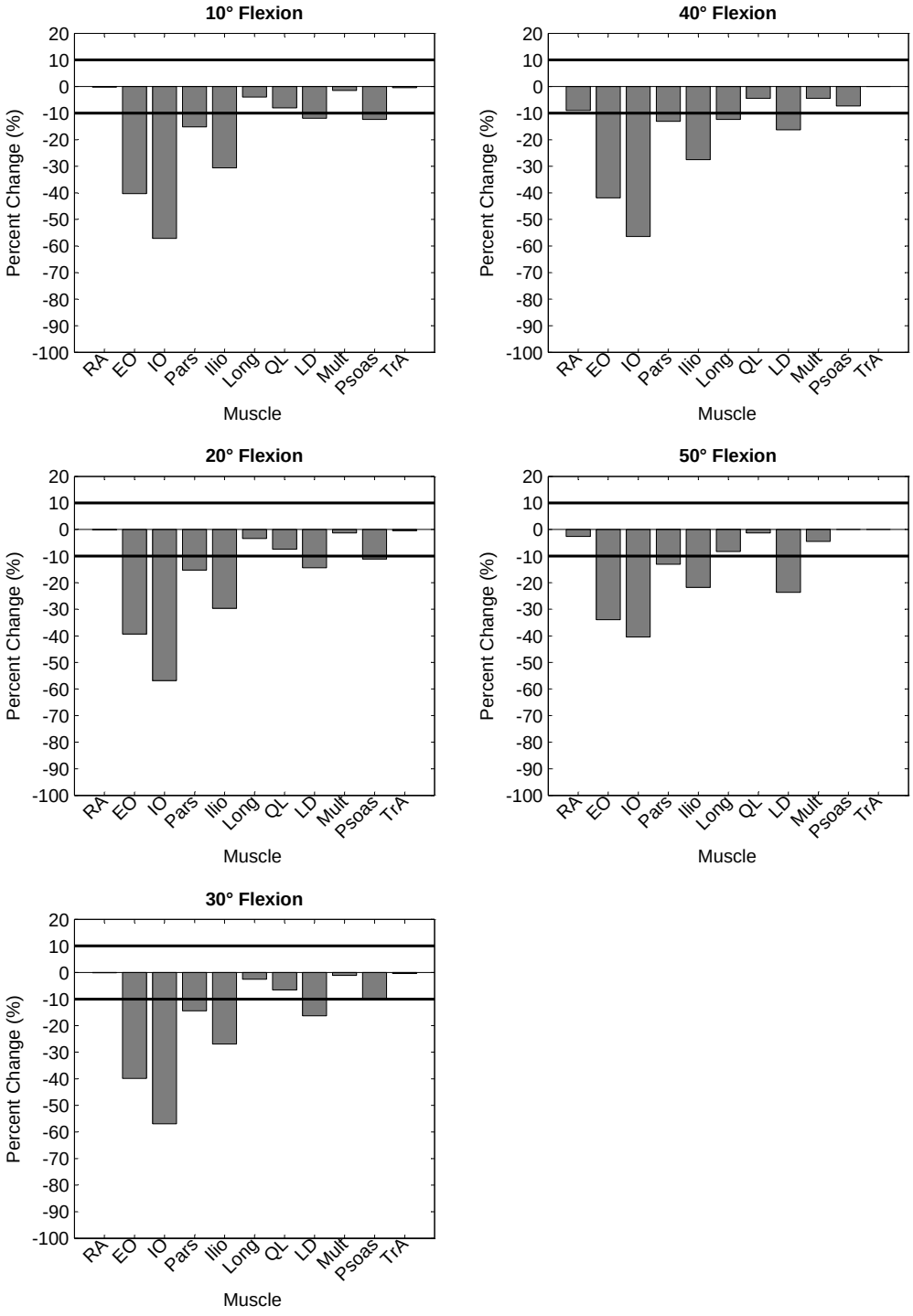
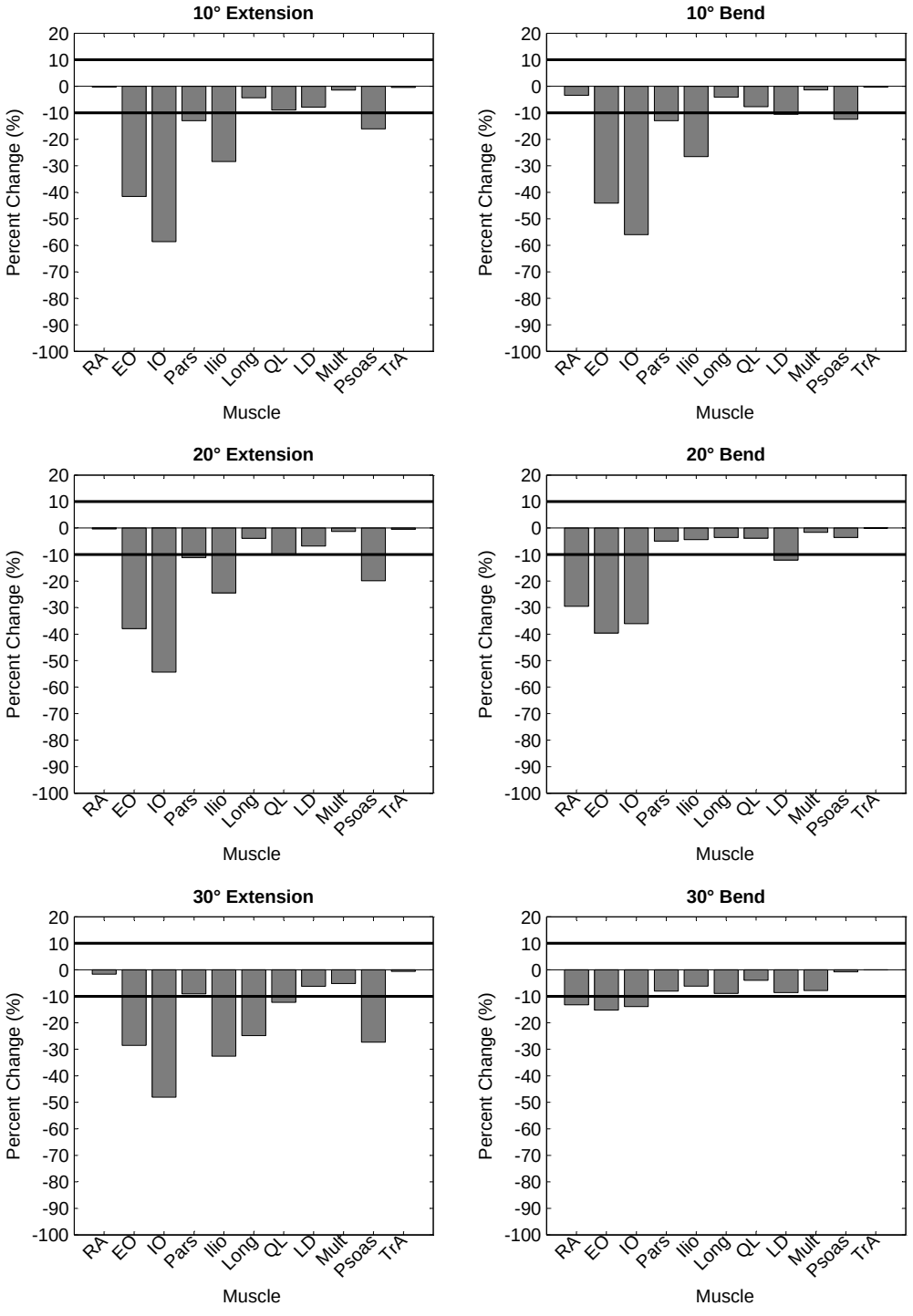
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

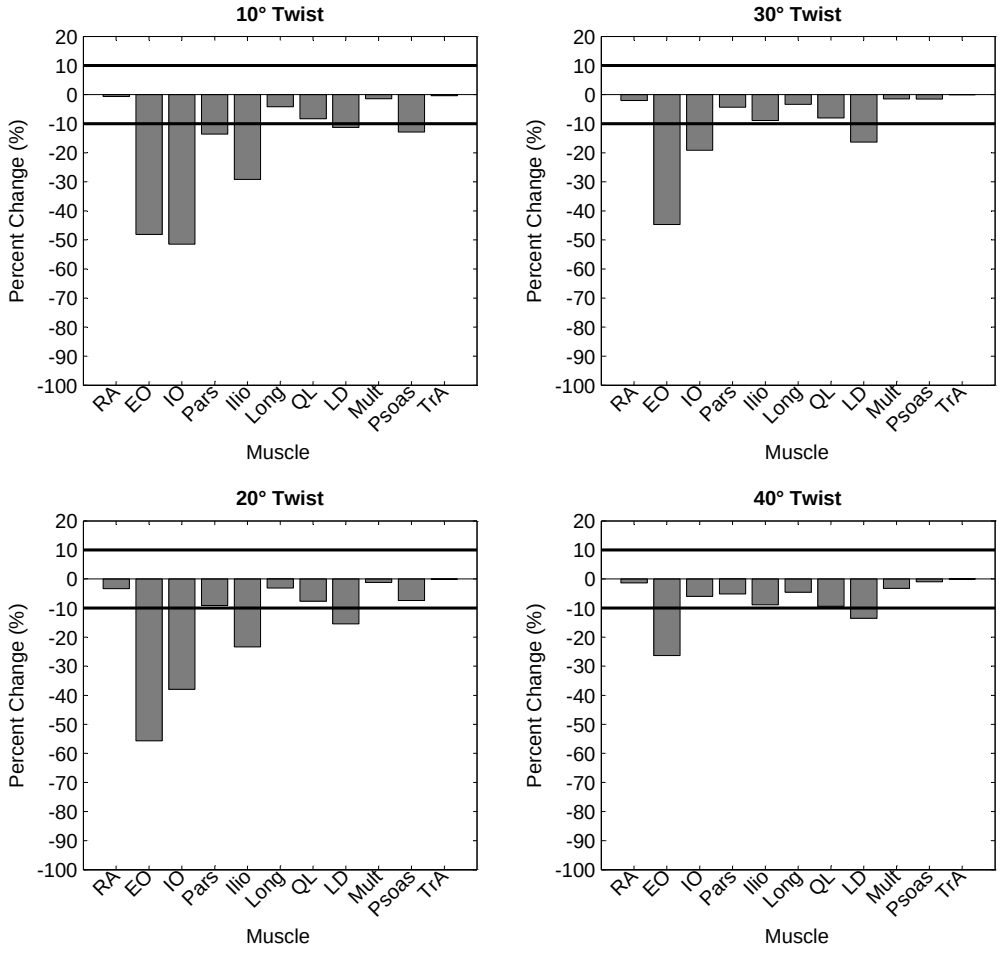
L4B


10° Flexion
40° Flexion
20° Flexion
50° Flexion
30° Flexion
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

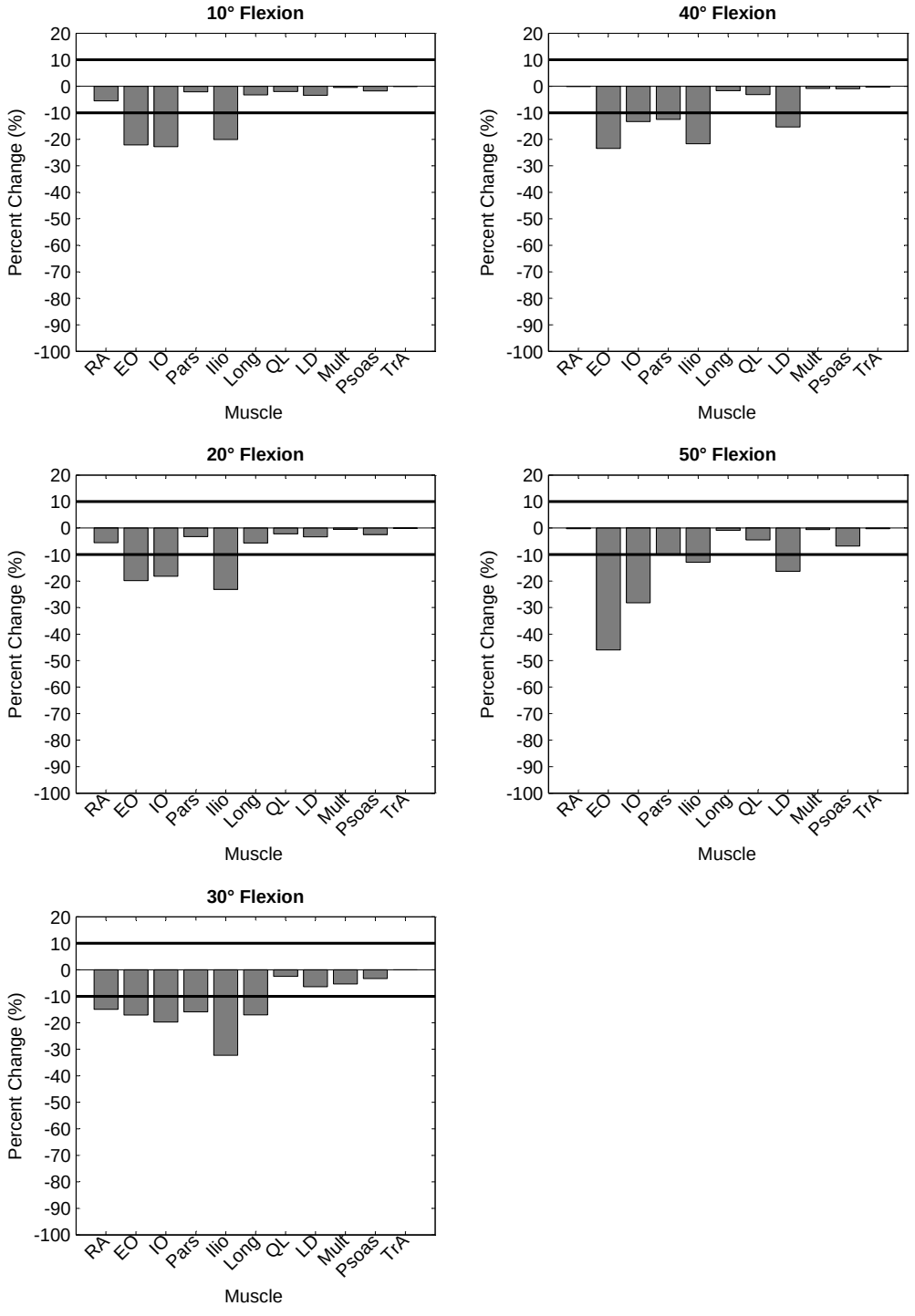
L4B


10° Extension
10° Bend
20° Extension
20° Bend
30° Extension
30° Bend
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

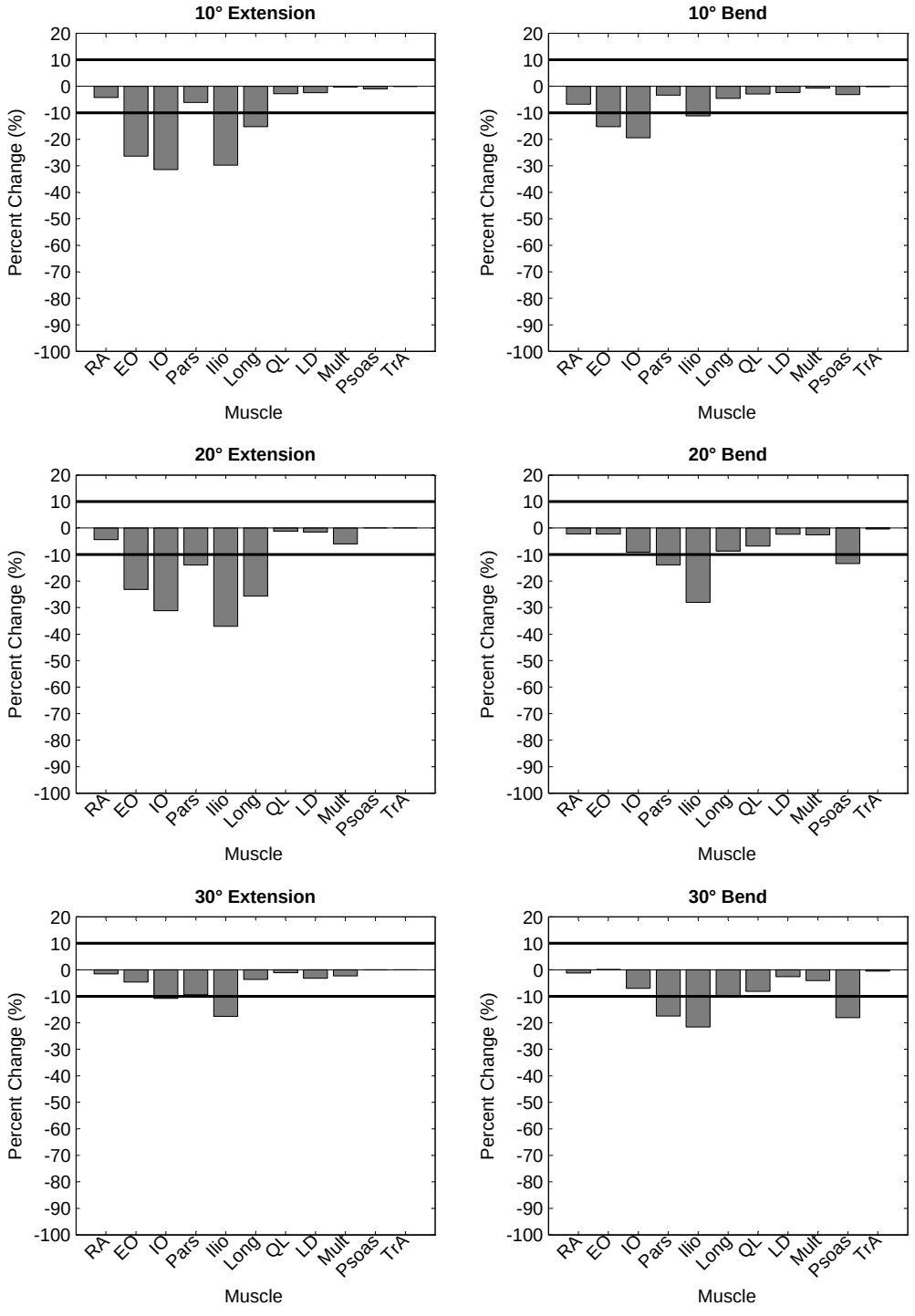
L4B


10° Twist
30° Twist
20° Twist
40° Twist
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

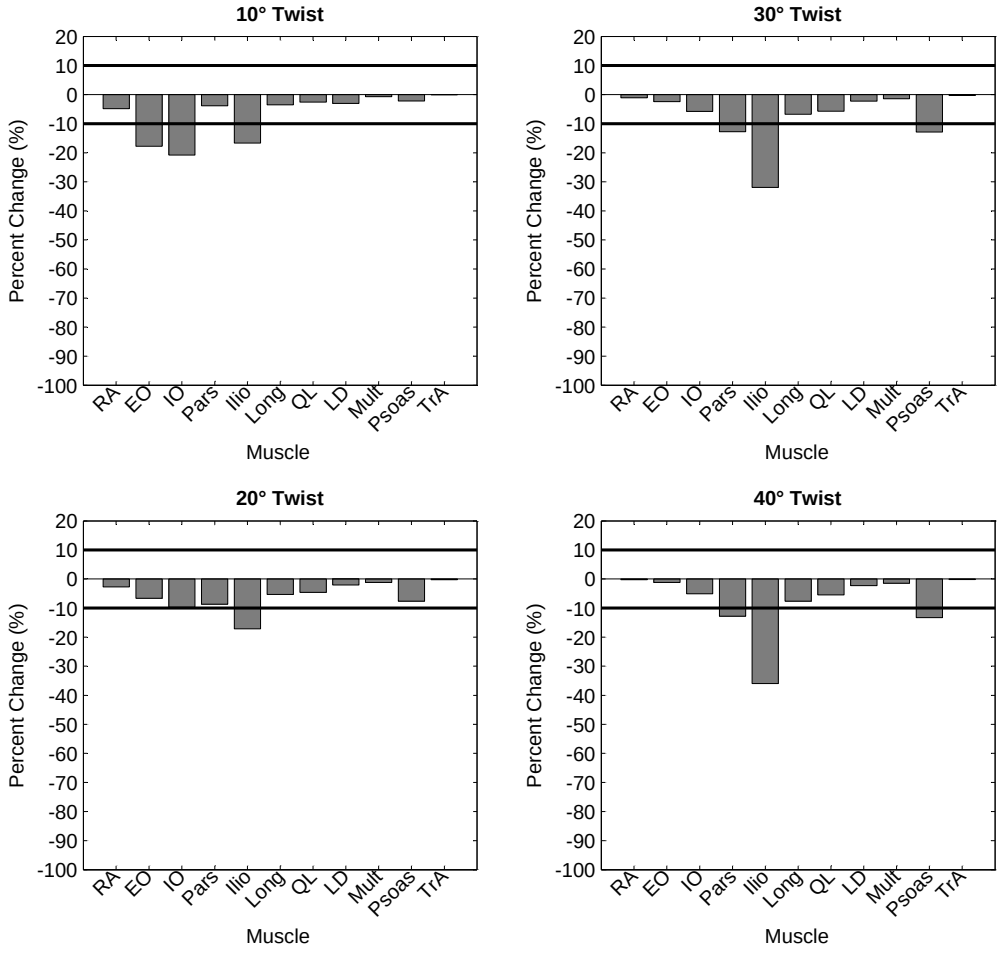
L4T


10° Flexion
40° Flexion
20° Flexion
50° Flexion
30° Flexion
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

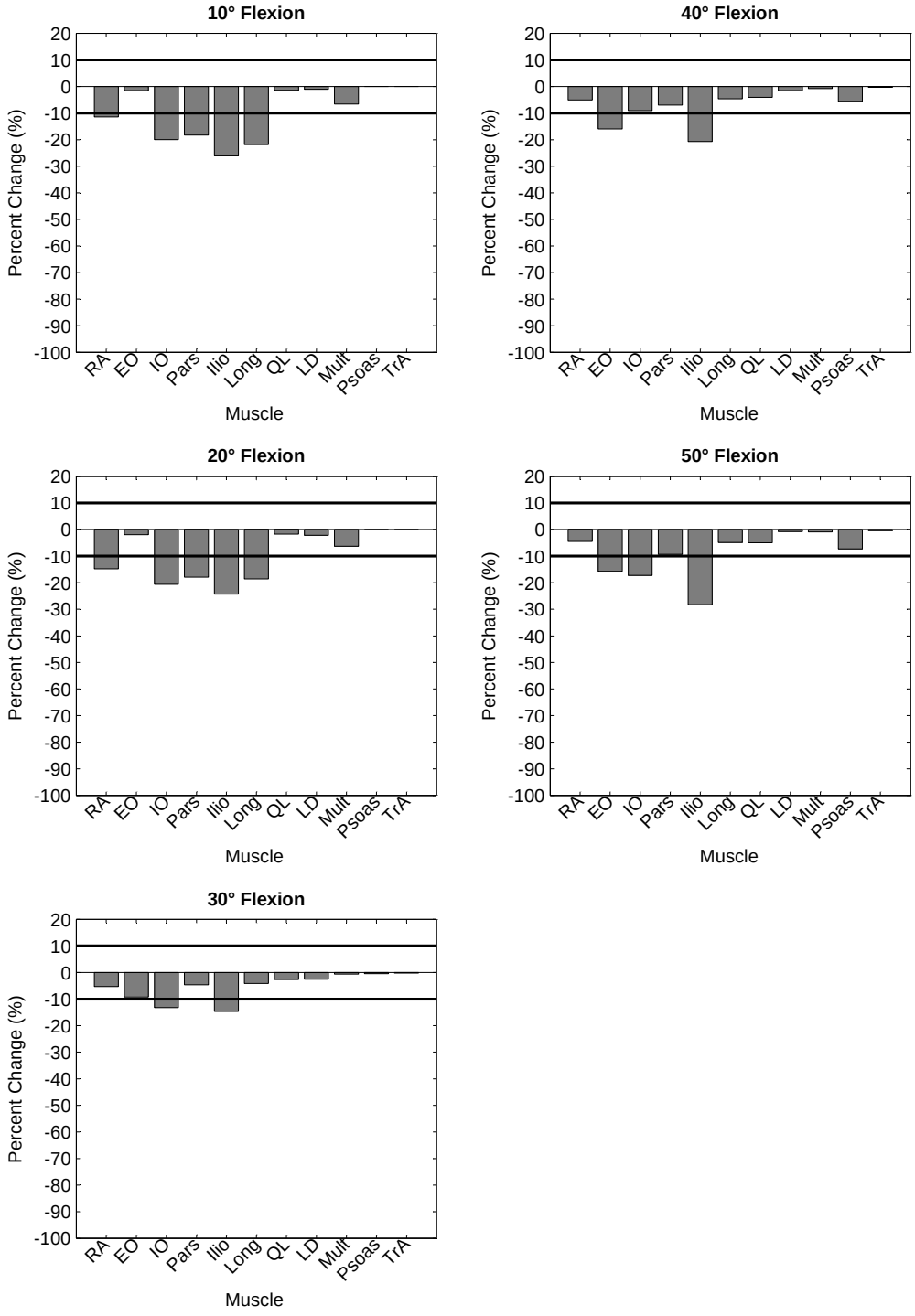
L4T


10° Extension
10° Bend
20° Extension
20° Bend
30° Extension
30° Bend
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

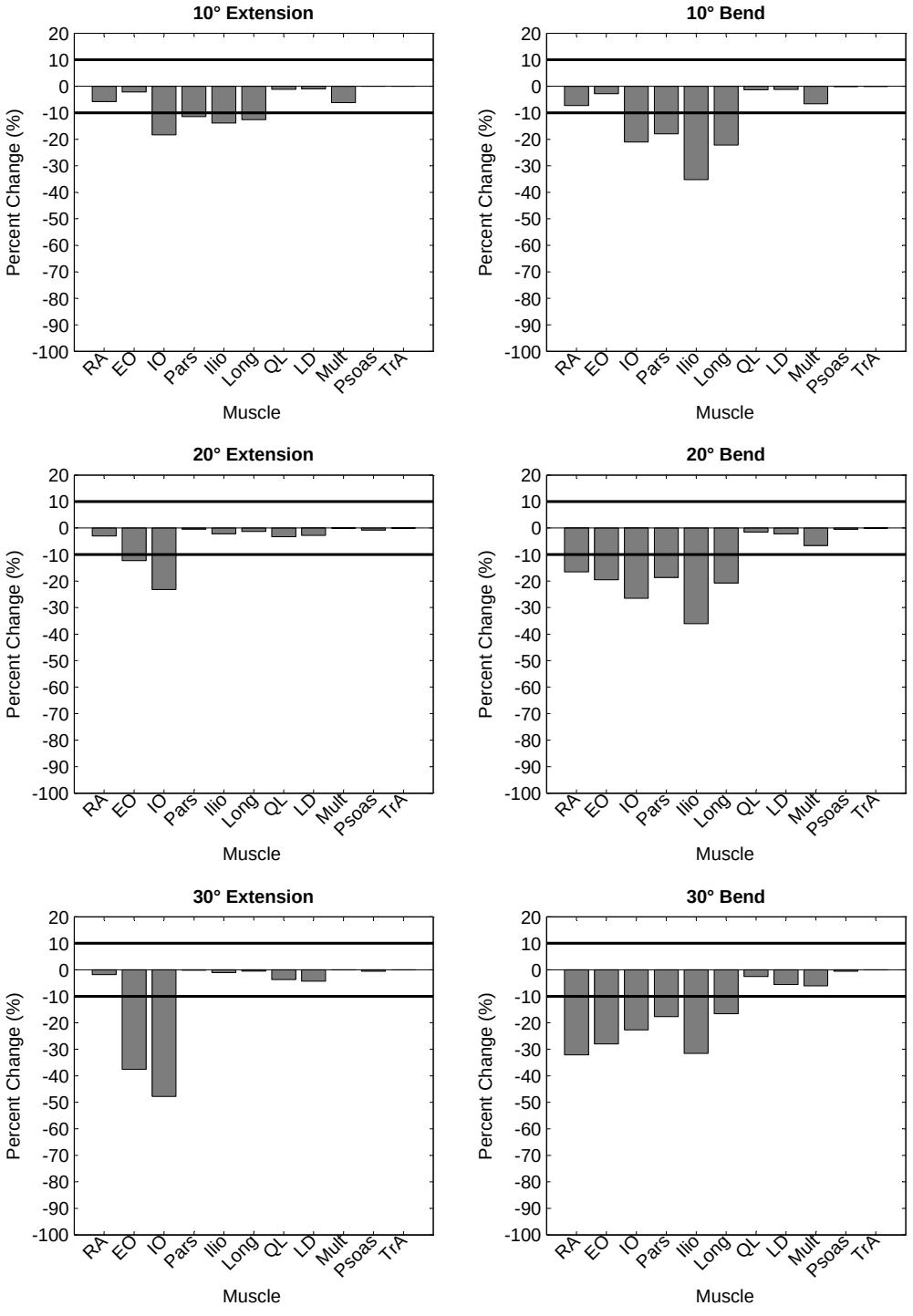
L4T


10° Twist
30° Twist
20° Twist
40° Twist
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

L5F


10° Flexion
40° Flexion
20° Flexion
50° Flexion
30° Flexion
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

L5F


10° Extension
10° Bend
20° Extension
20° Bend
30° Extension
30° Bend
Percent Change (%)
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10
0
-10
-20
-30
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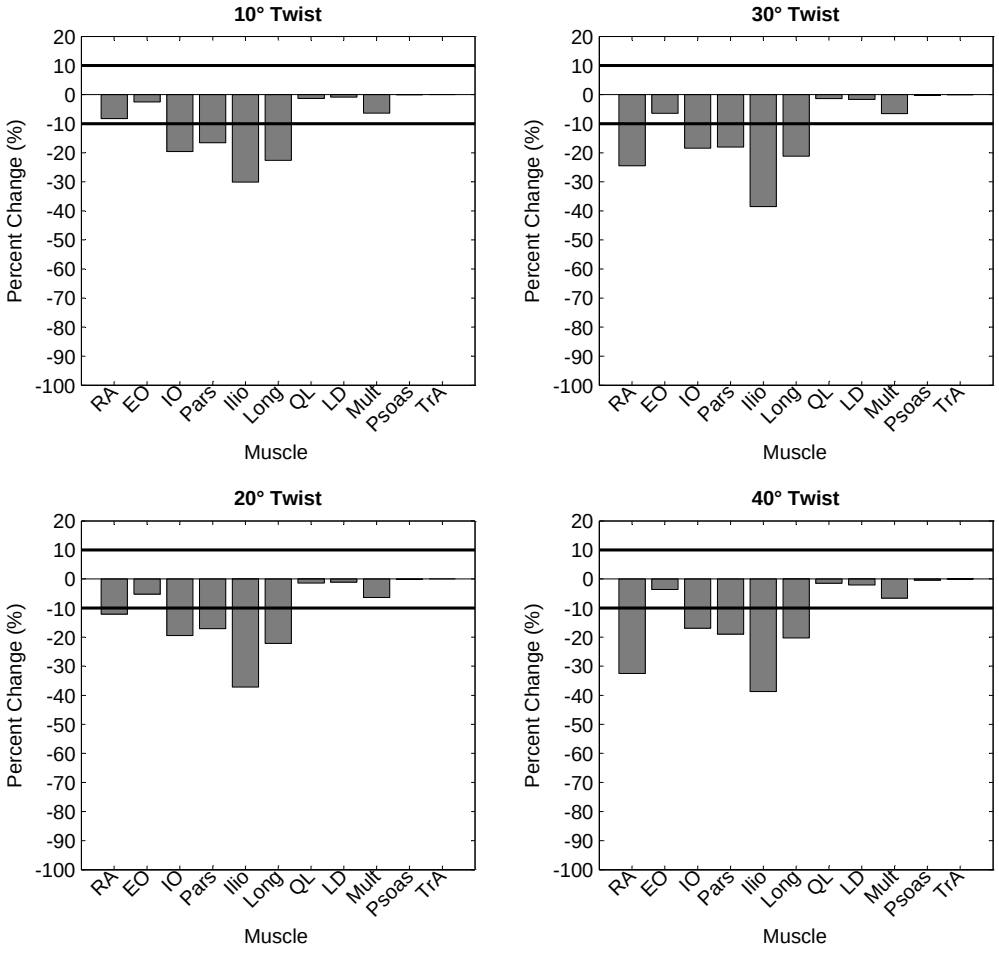
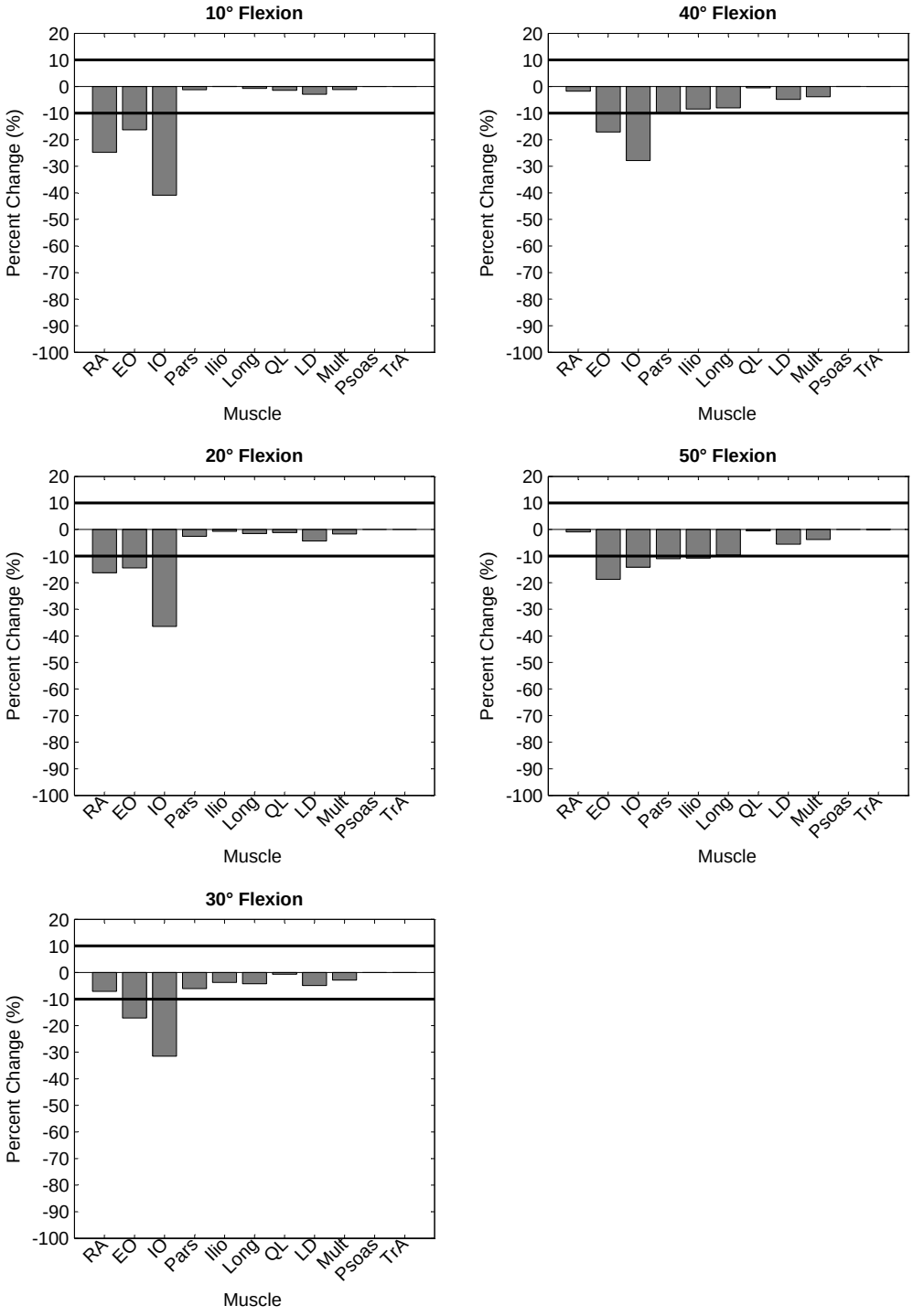
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

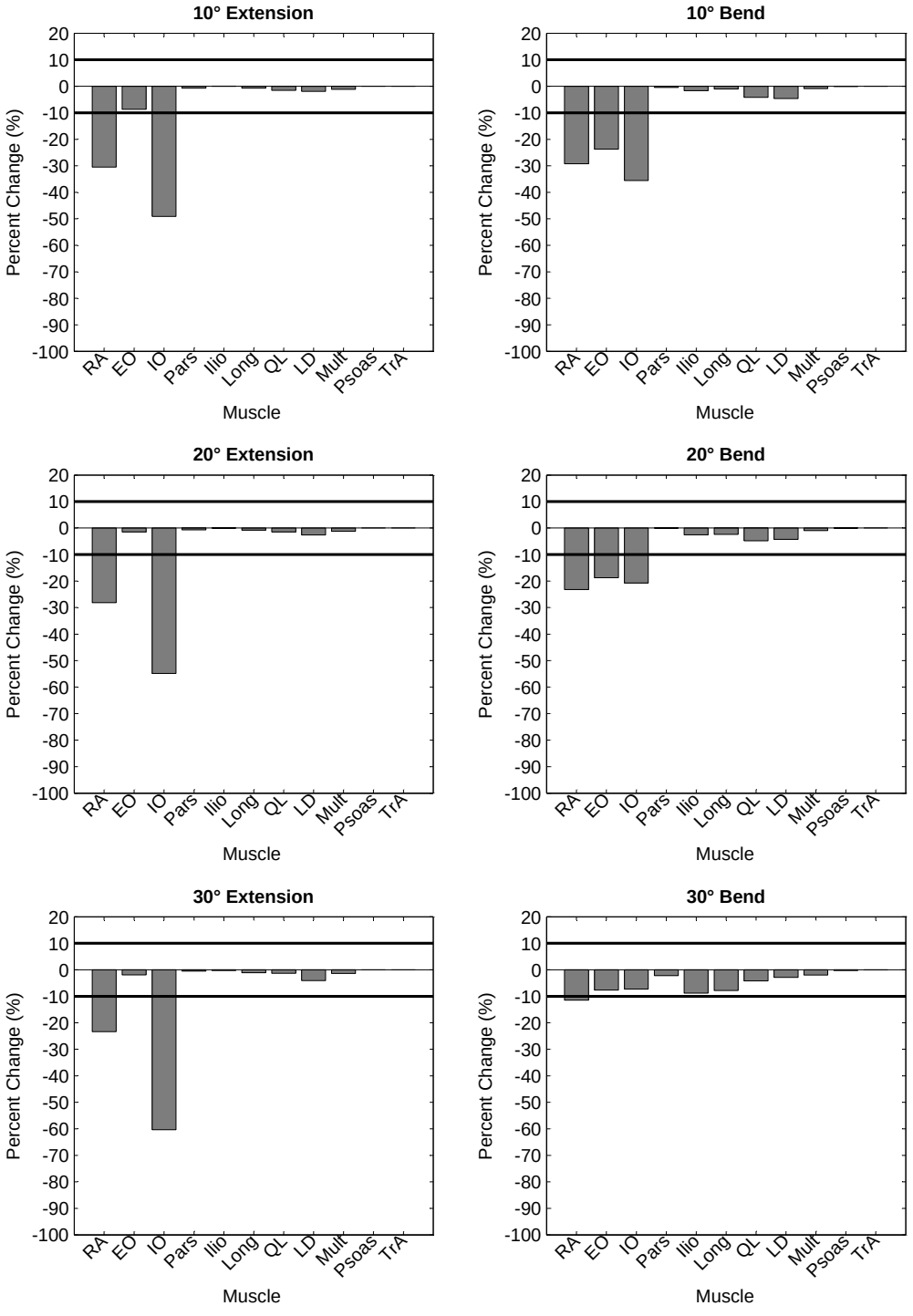
L5F


10° Twist
30° Twist
20° Twist
40° Twist
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

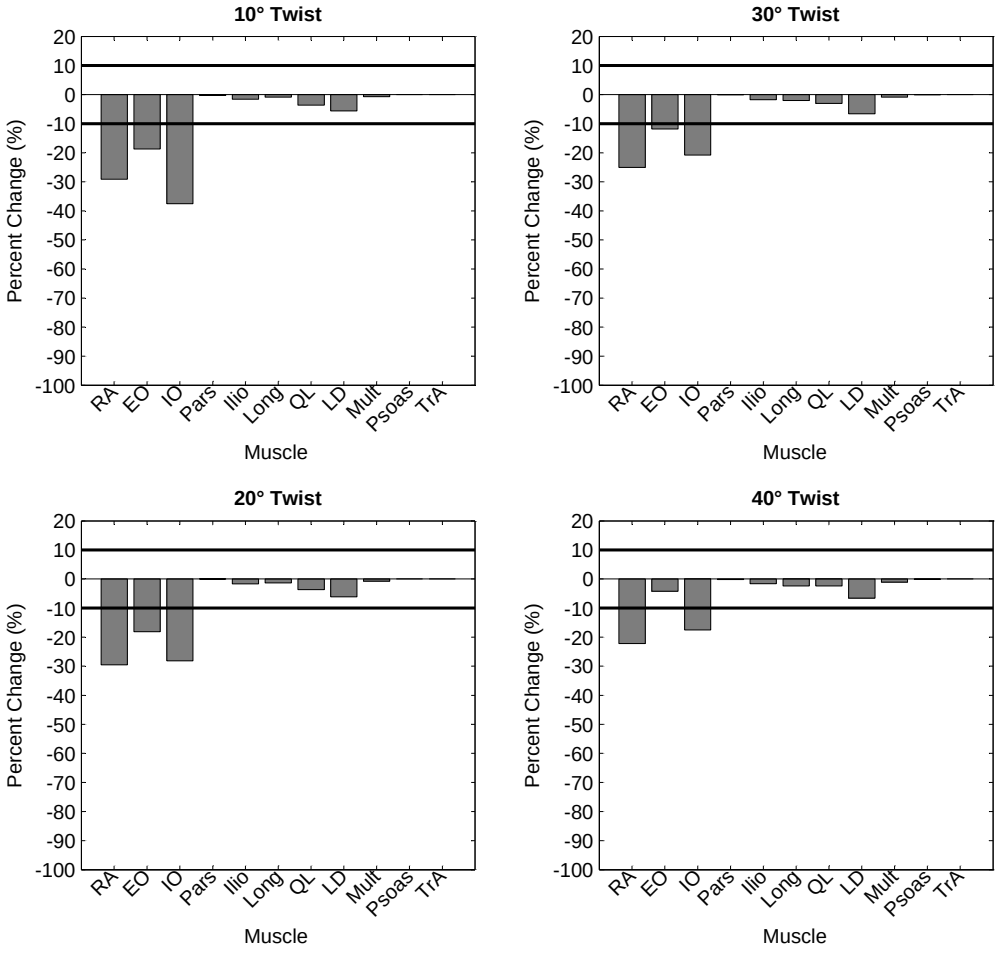
L5B


10° Flexion
40° Flexion
20° Flexion
50° Flexion
30° Flexion
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

L5B


10° Extension
10° Bend
20° Extension
20° Bend
30° Extension
30° Bend
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

L5B


10° Twist
30° Twist
20° Twist
40° Twist
Percent Change (%)
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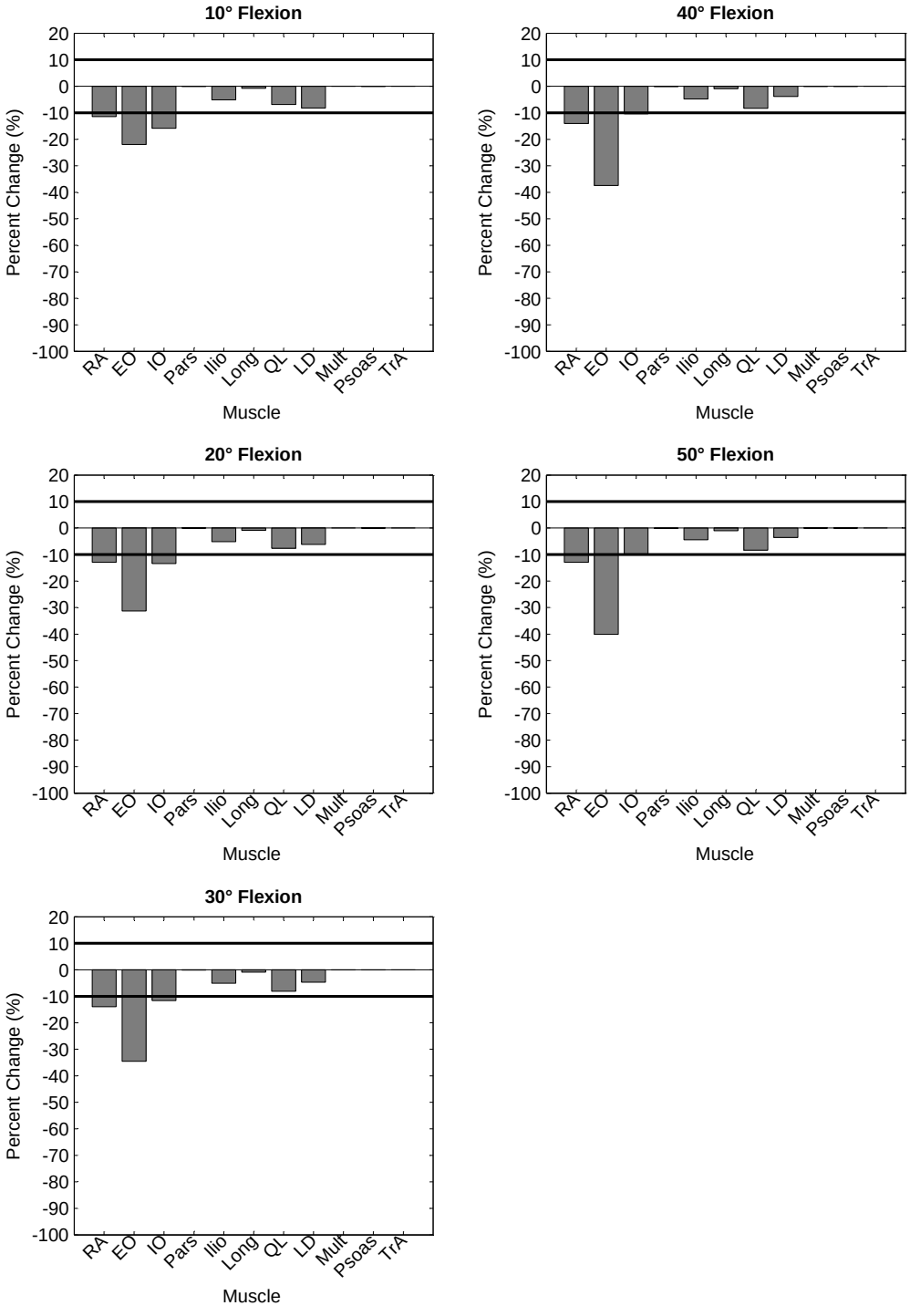
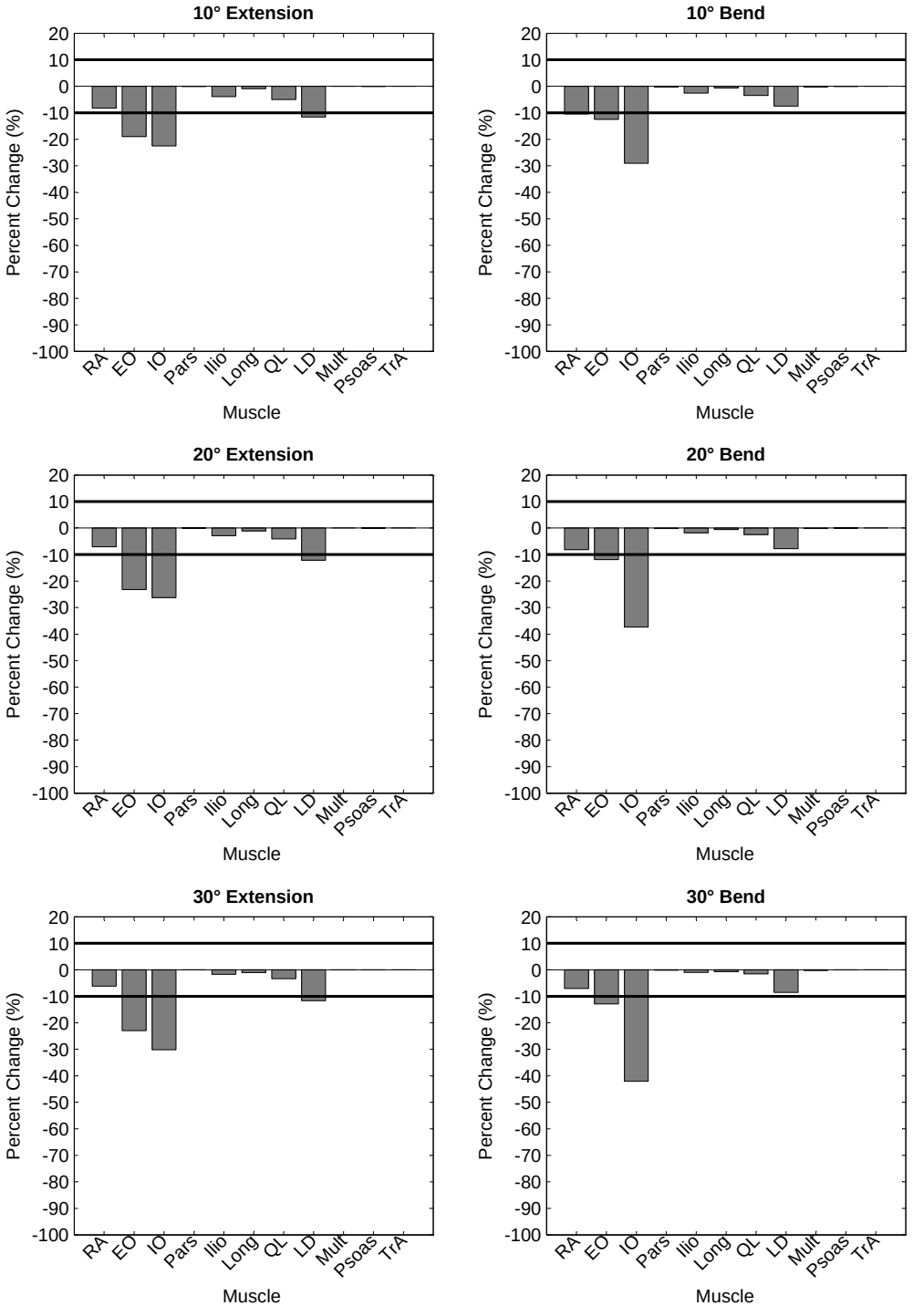
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

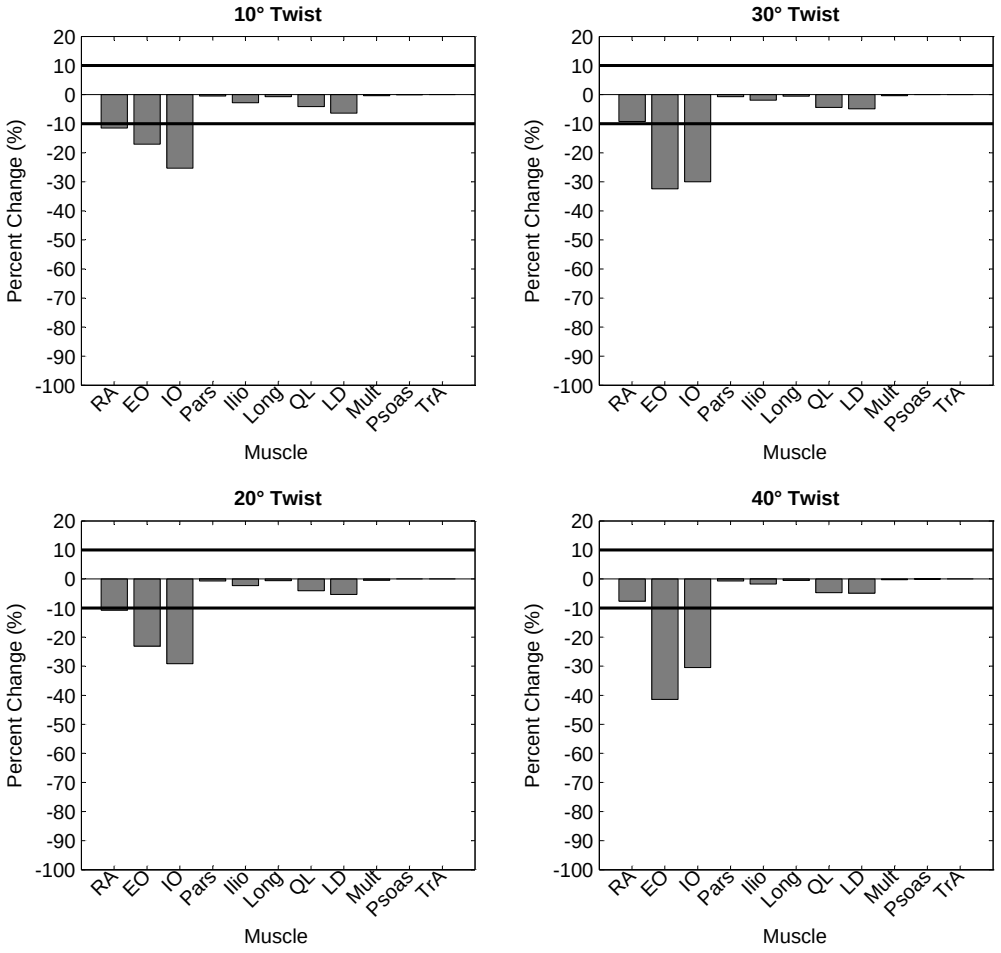
L5T


10° Flexion
40° Flexion
20° Flexion
50° Flexion
30° Flexion
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

L5T


10° Extension
10° Bend
20° Extension
20° Bend
30° Extension
30° Bend
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

L5T

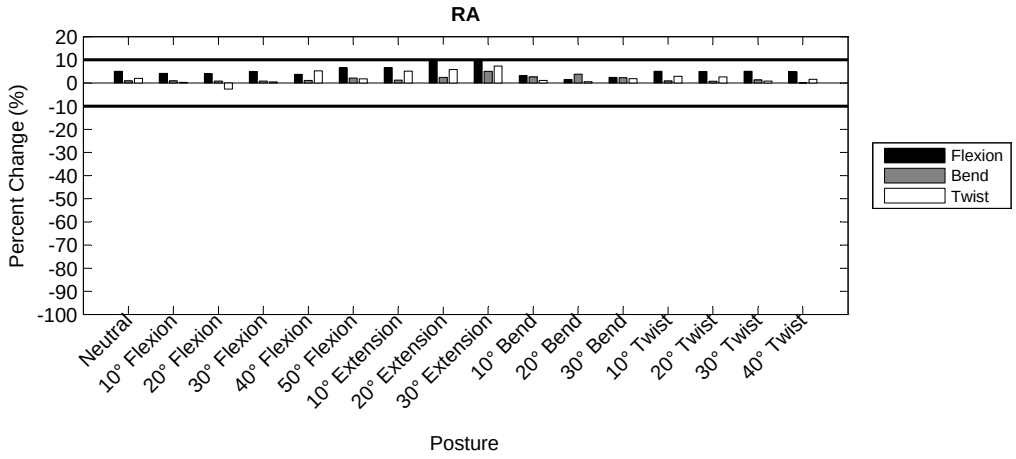

10° Twist
30° Twist
20° Twist
40° Twist
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
RA
EO
IO
Pars
Ilio
Long
QL
LD
Mult
Psoas
TrA
Muscle

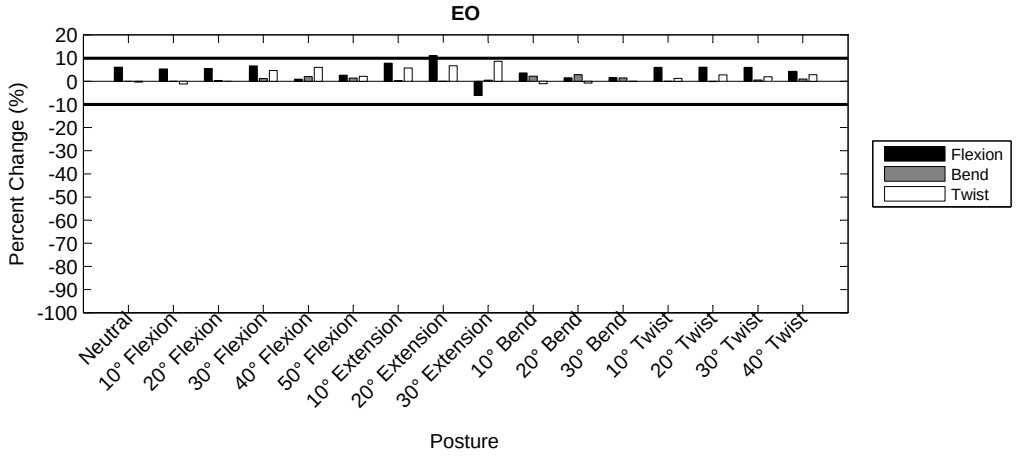
Appendix B

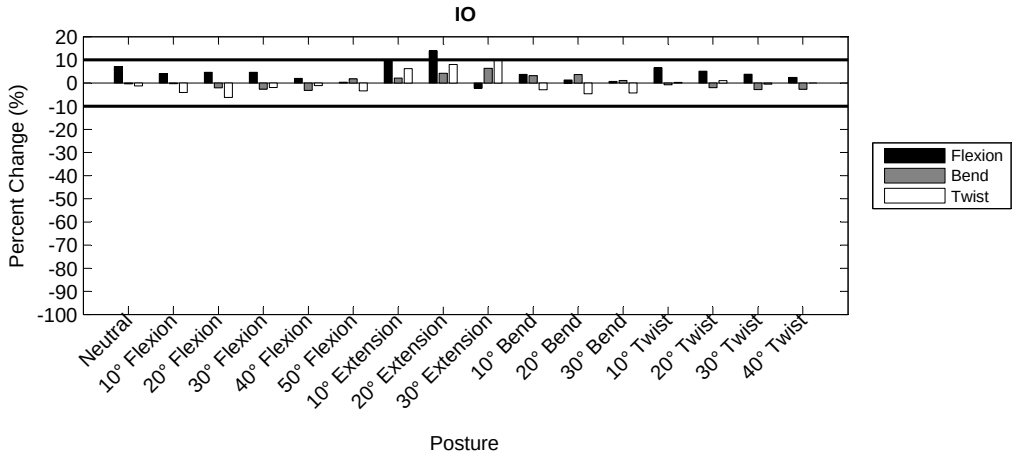
Effect of specific muscles on plane of stability/stiffness

Each figure represents the percent change in three EVs when reducing single muscle activation to 0% MVC while all other muscles remained active to 50% MVC for various postures. The EV level being examined is the large title at the top of each page, the plane of stability/stiffness is in the legend, the muscle that was removed is the title of each graph and the posture being examined is along the x-axis. Negative change represents a lower EV when a single muscle's activity was reduced to 0% MVC.A change of 10% or greater in the EV was considered biologically significant. The thick black lines highlight these points. These figures are supplemental to those in section 4.1.2 (hypothesis 2 results), which display only the L4 level EVs.

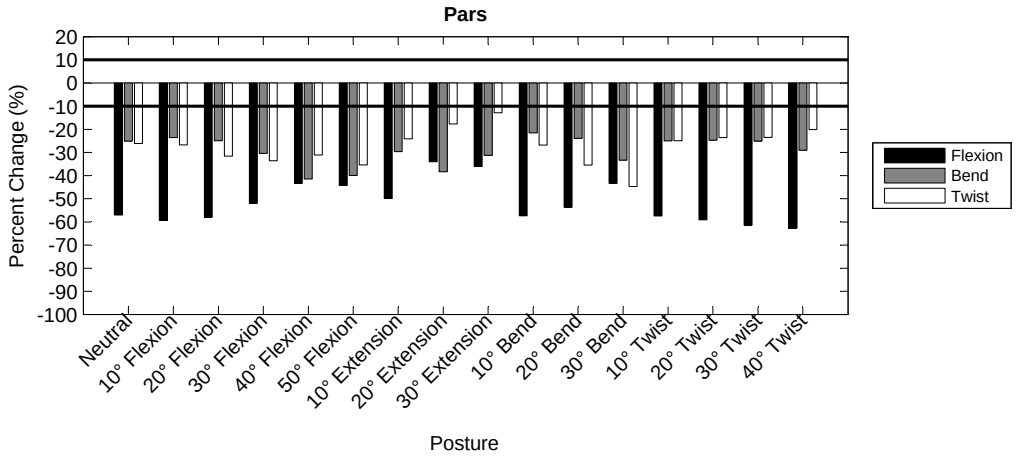
L1 Lumbar Level (0 %MVC)

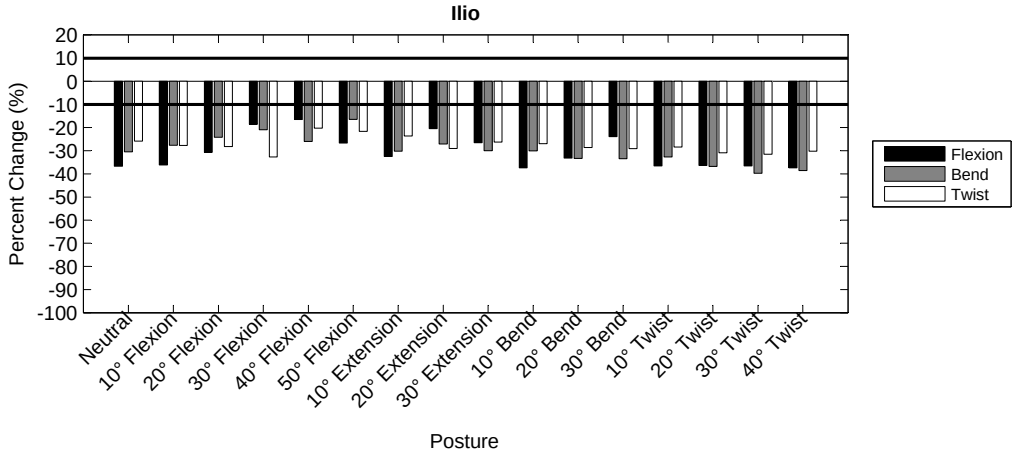


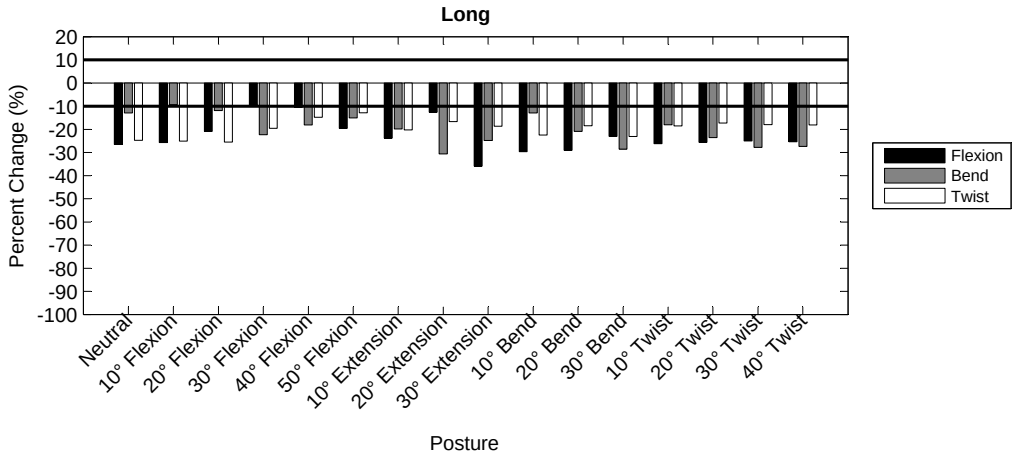




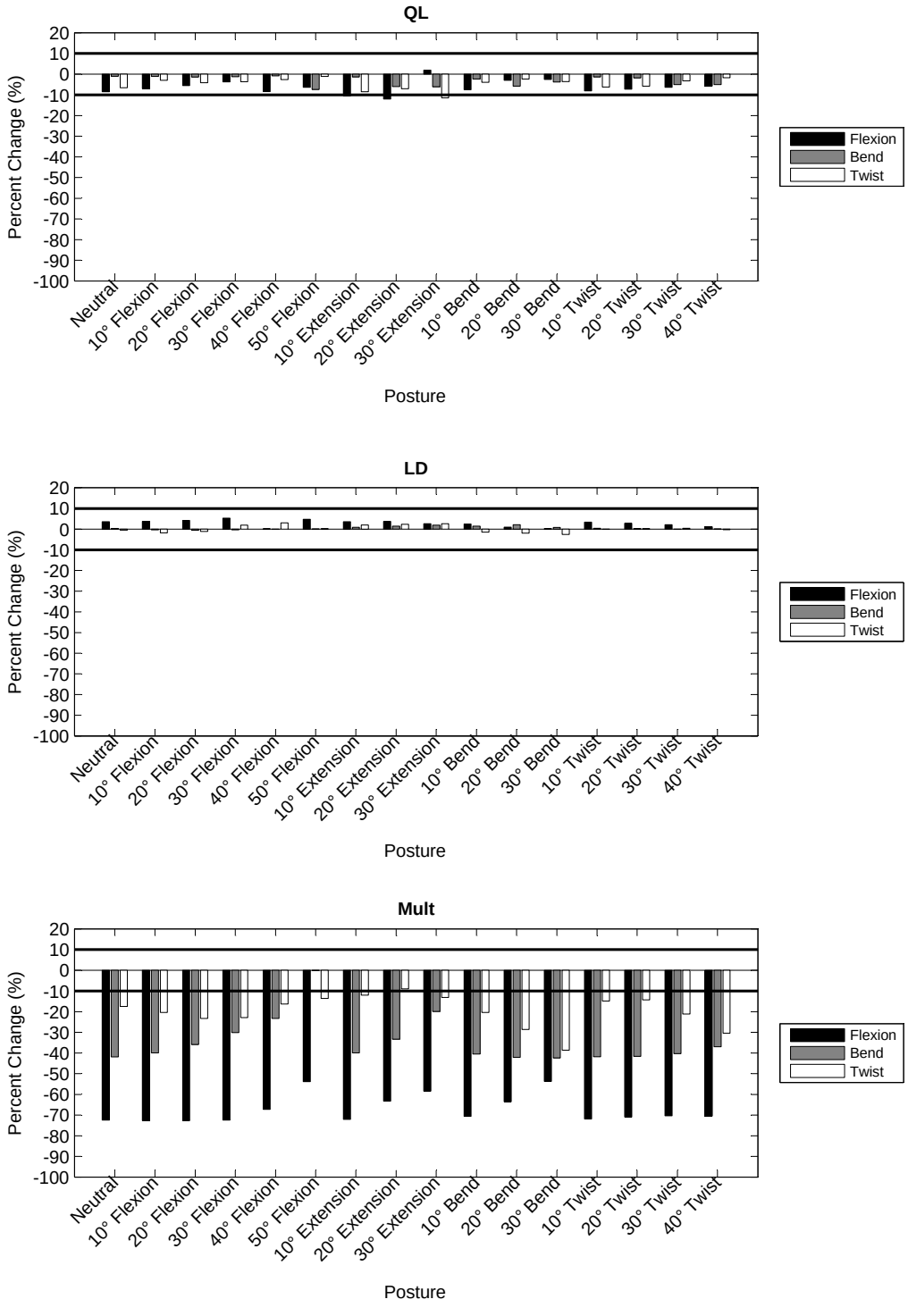
L1 Lumbar Level (0 %MVC)

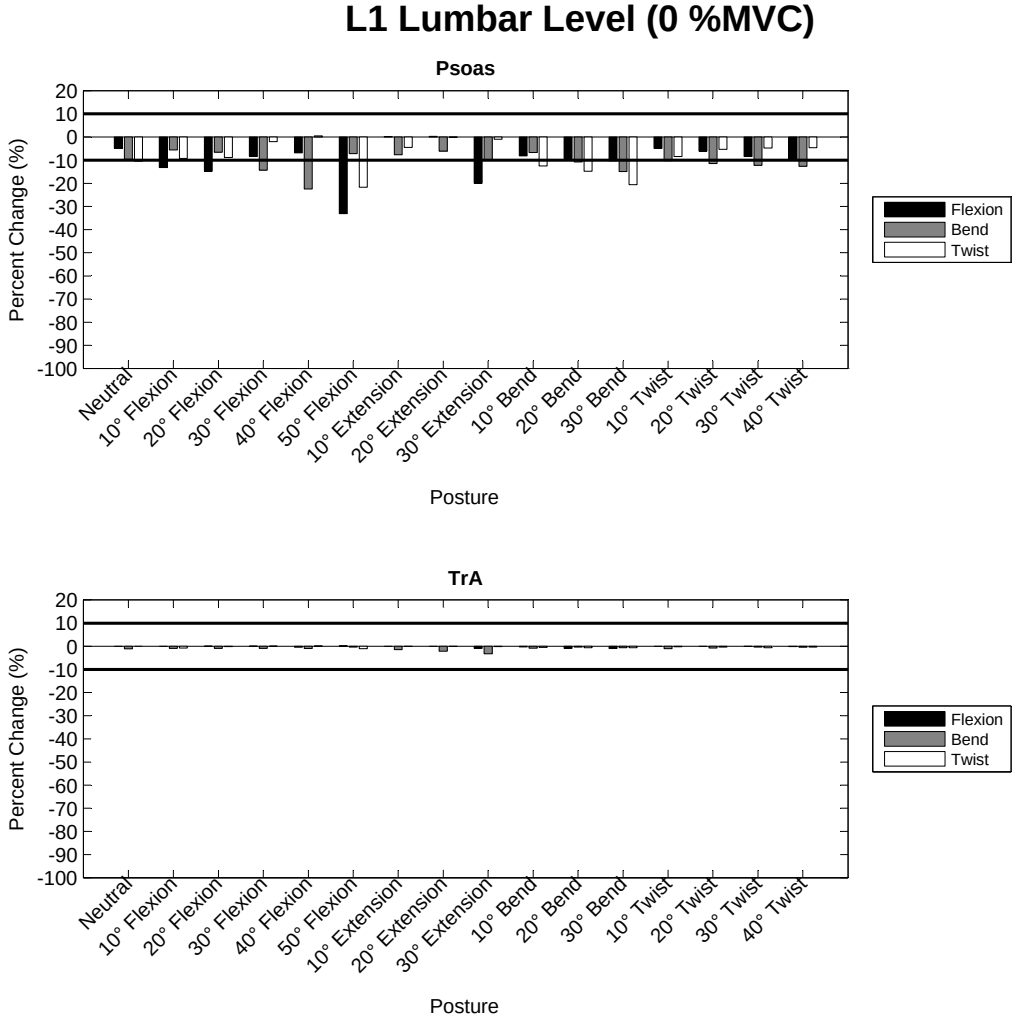




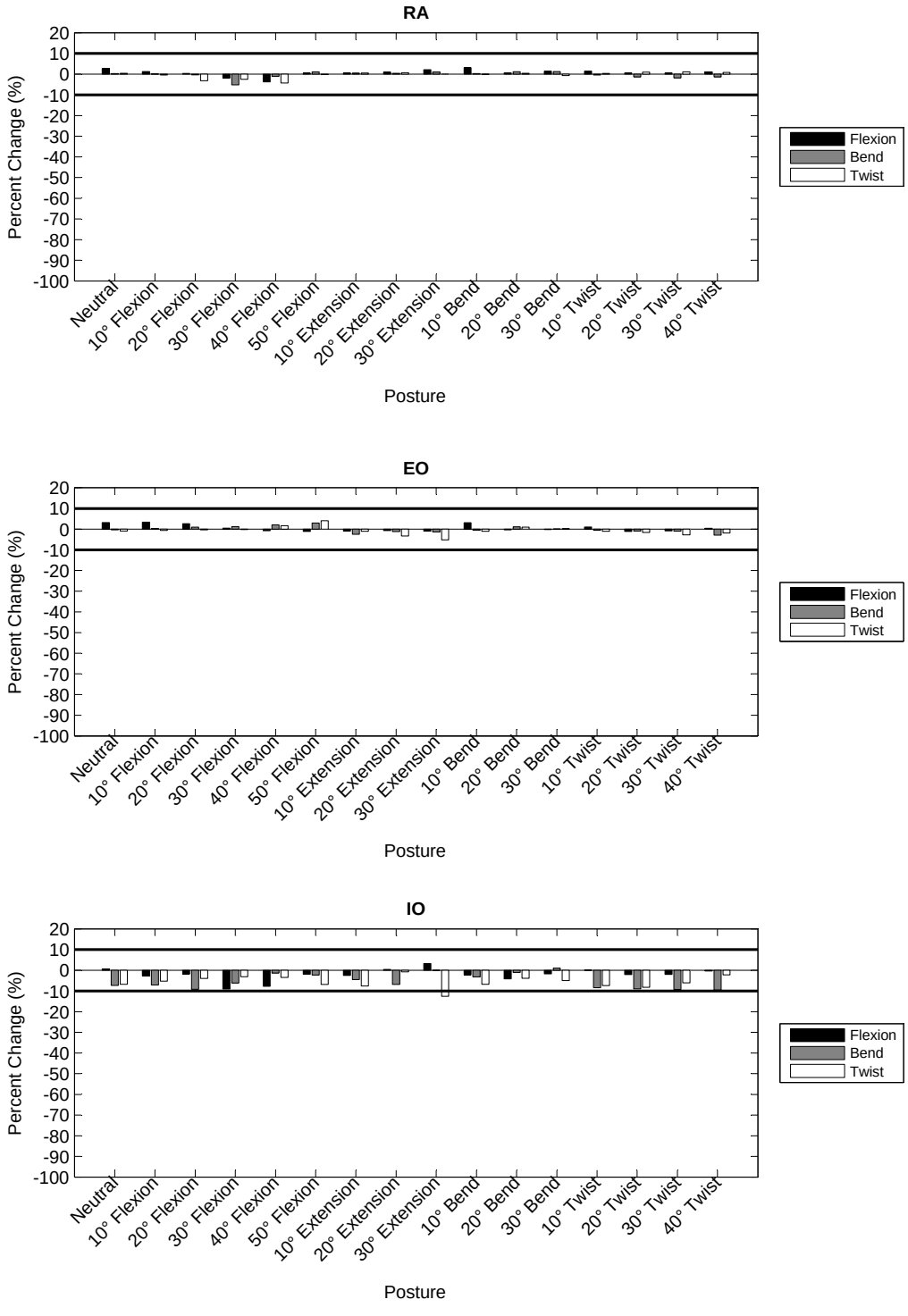


L1 Lumbar Level (0 %MVC)

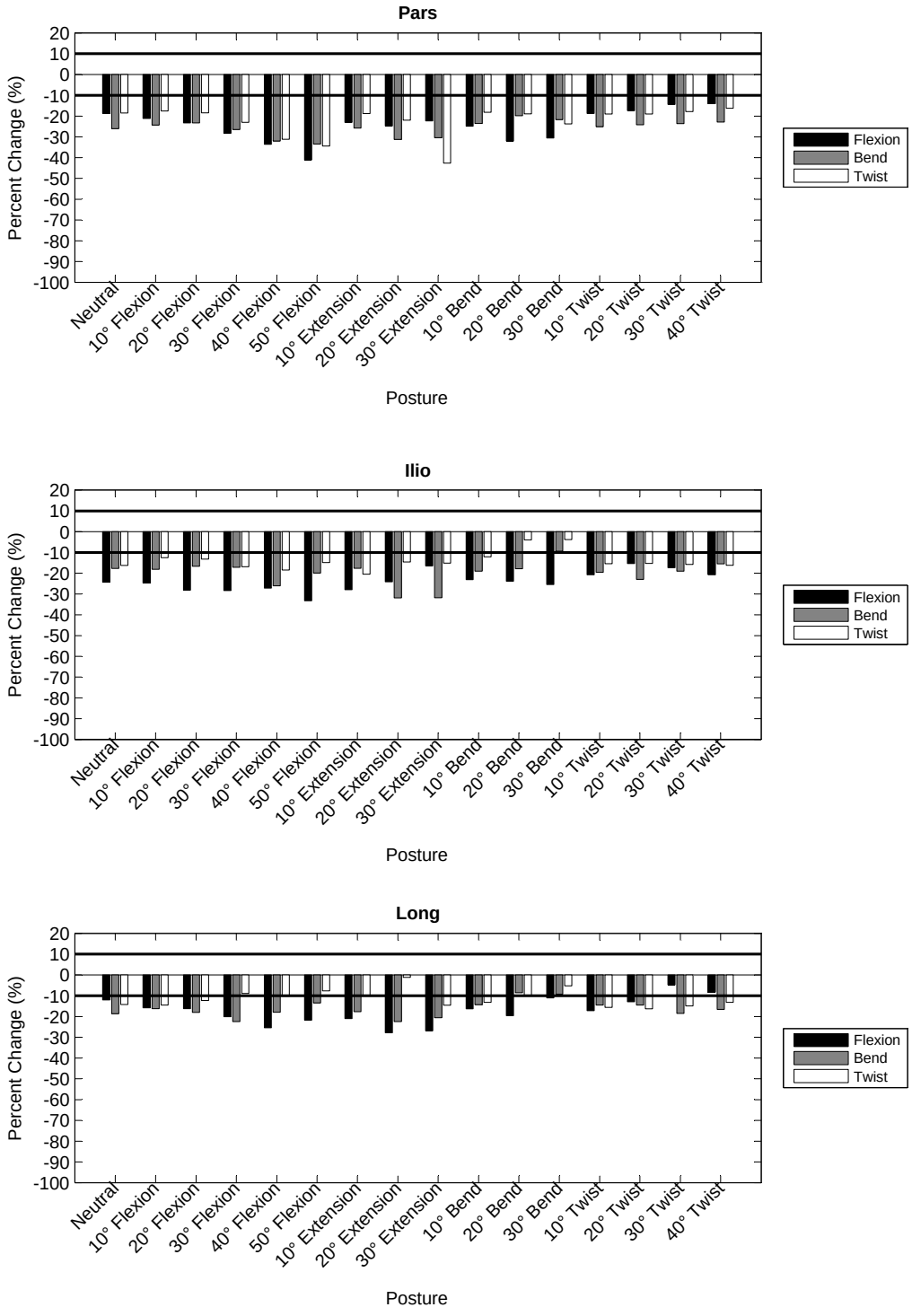



L1 Lumbar Level (0 %MVC)
Psoas
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
Neutral
10° Flexion
20° Flexion
30° Flexion
40° Flexion
50° Flexion
10° Extension
20° Extension
30° Extension
10° Bend
20° Bend
30° Bend
10° Twist
20° Twist
30° Twist
40° Twist
Posture
Flexion
Bend
Twist
TrA
Percent Change (%)
Posture
Flexion
Bend
Twist

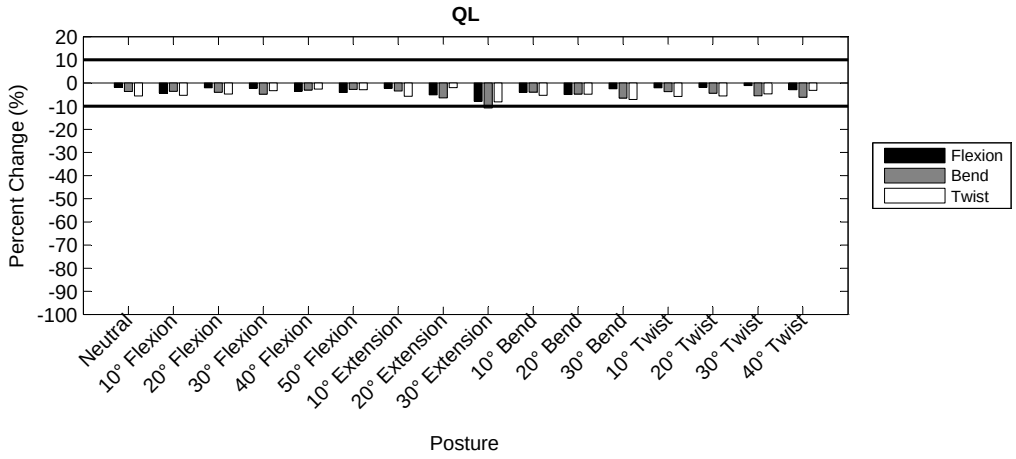
L2 Lumbar Level (0 %MVC)

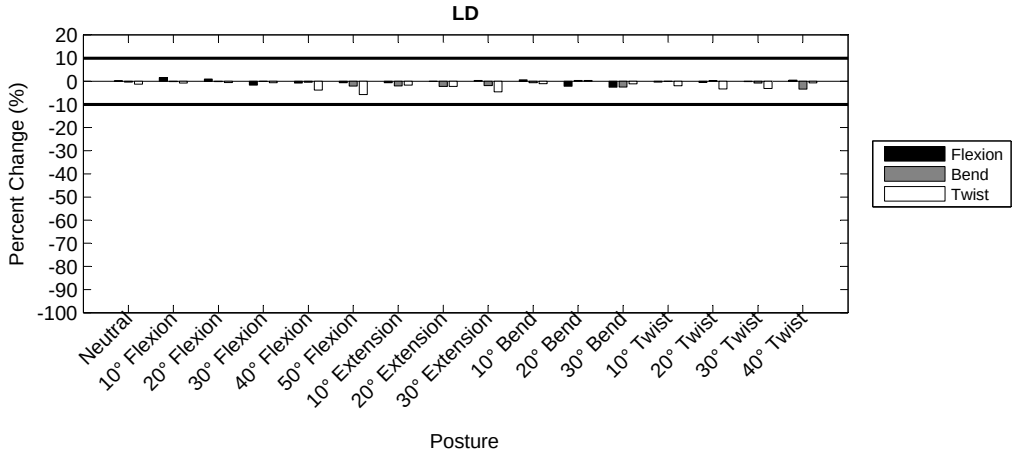


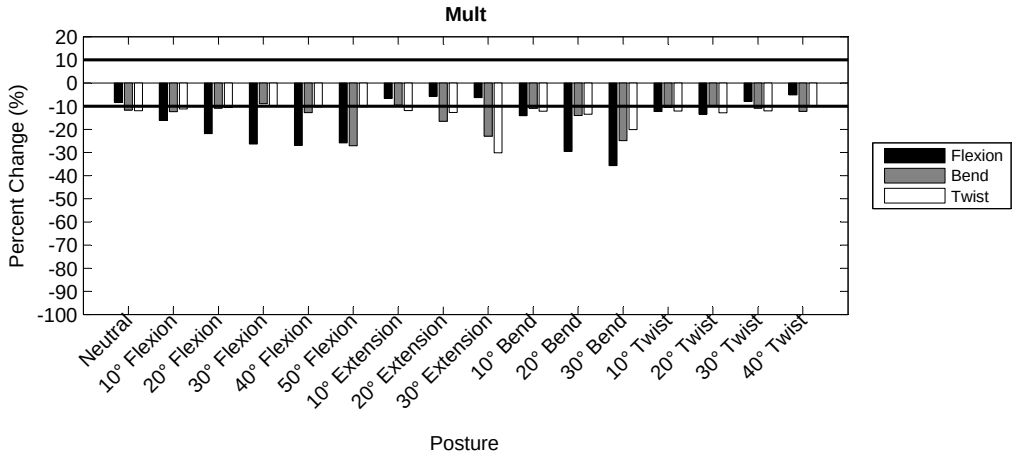
L2 Lumbar Level (0 %MVC)

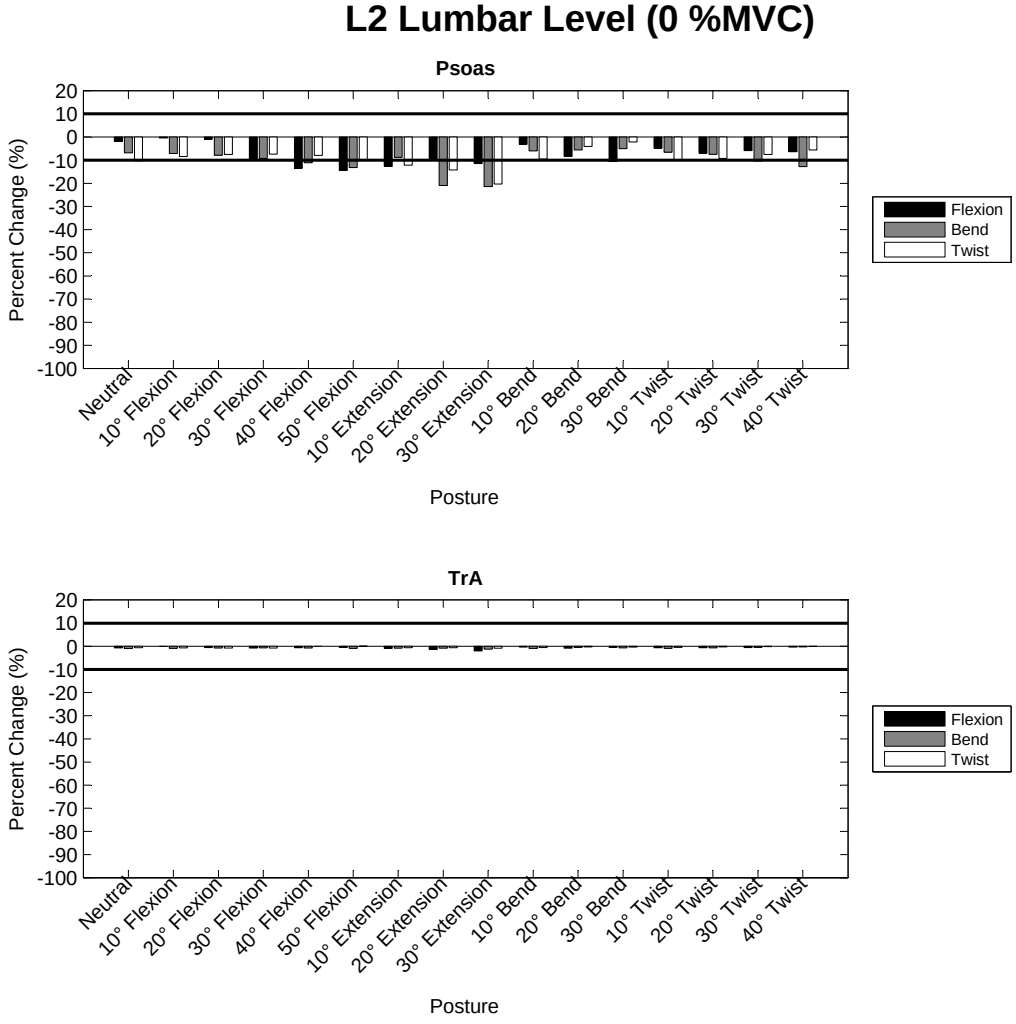


L2 Lumbar Level (0 %MVC)

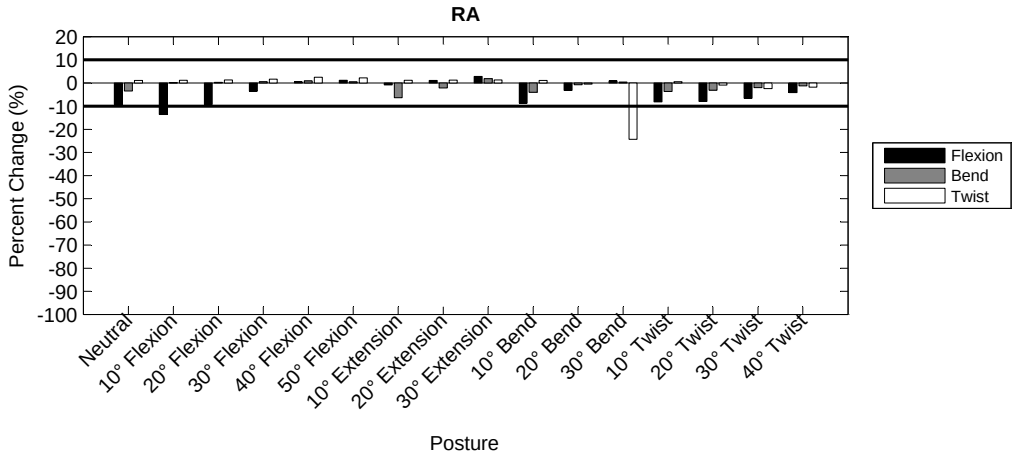


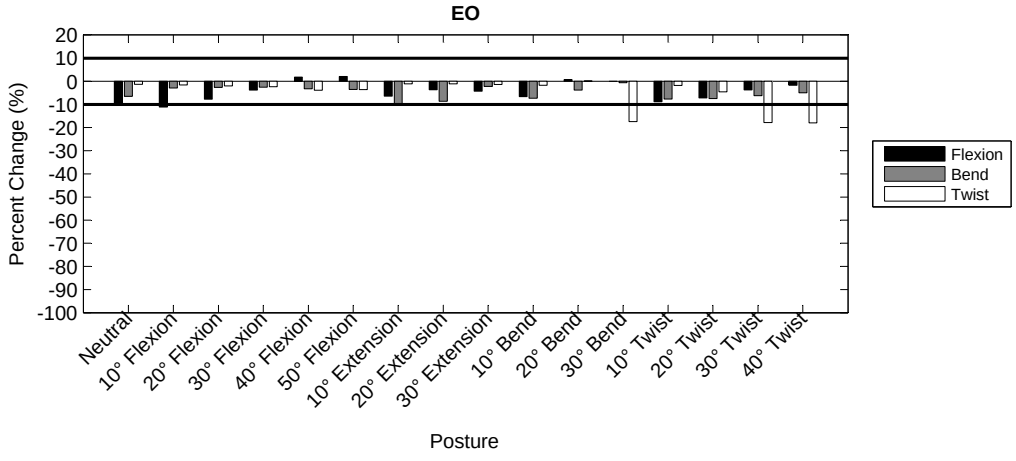


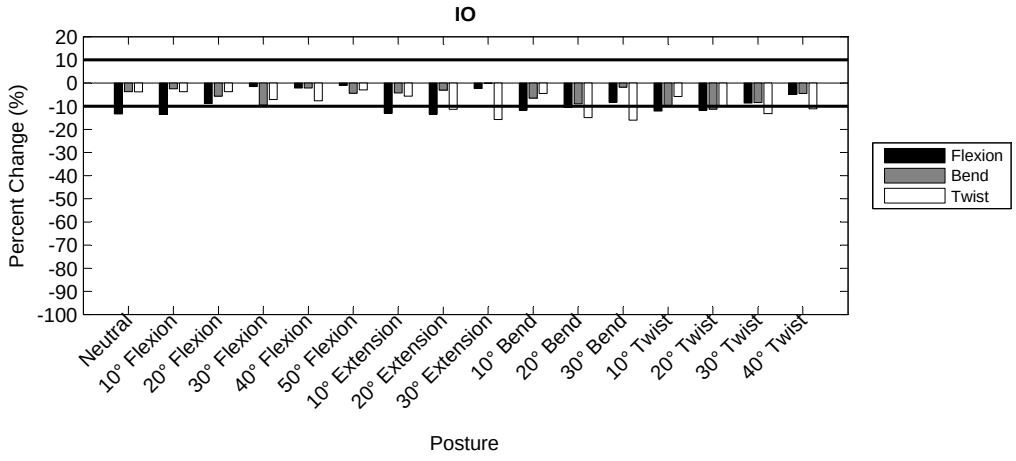


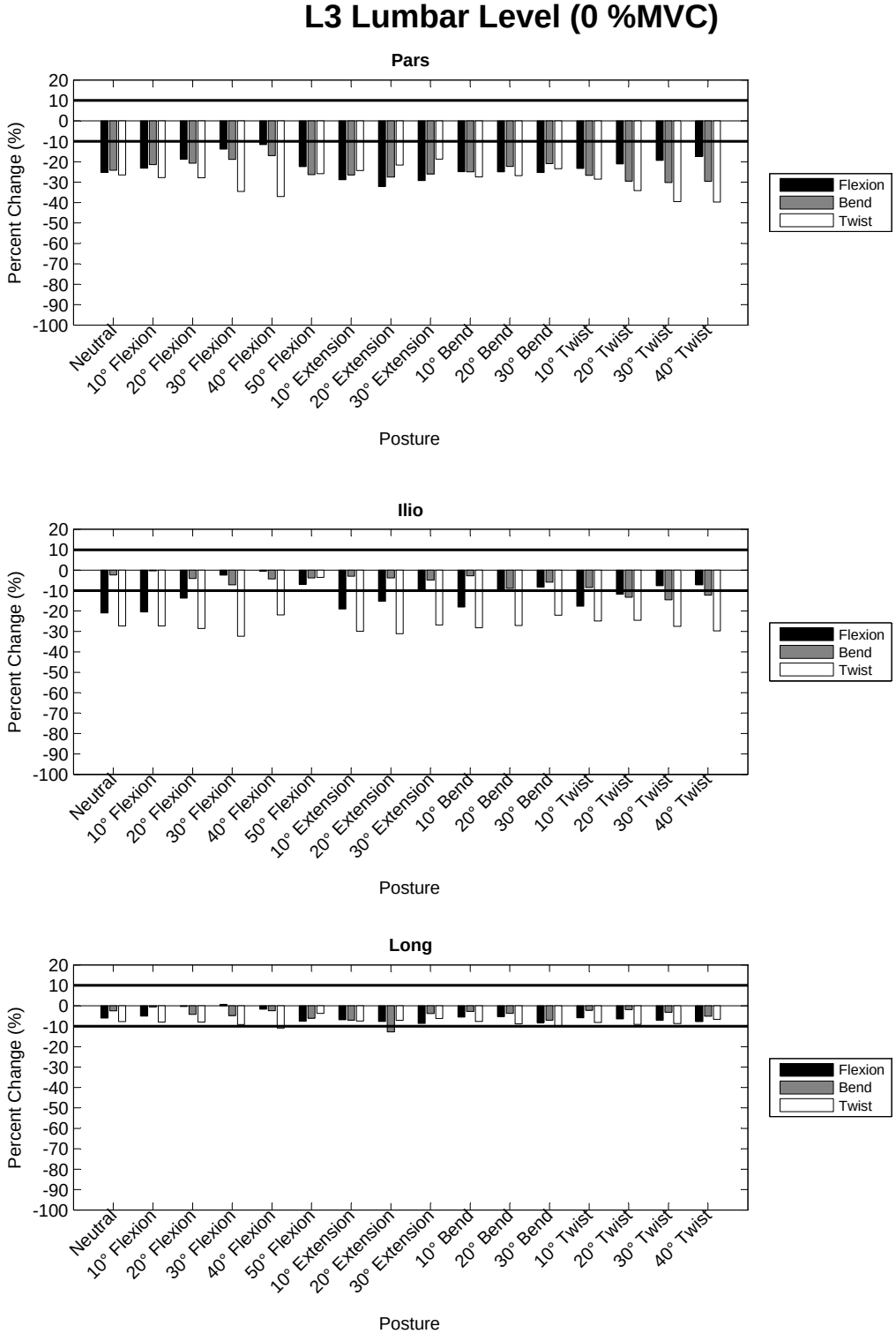

L2 Lumbar Level (0 %MVC)
Psoas
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
Neutral
10° Flexion
20° Flexion
30° Flexion
40° Flexion
50° Flexion
10° Extension
20° Extension
30° Extension
10° Bend
20° Bend
30° Bend
10° Twist
20° Twist
30° Twist
40° Twist
Posture
Flexion
Bend
Twist
TrA
Percent Change (%)
Posture
Flexion
Bend
Twist

L3 Lumbar Level (0 %MVC)

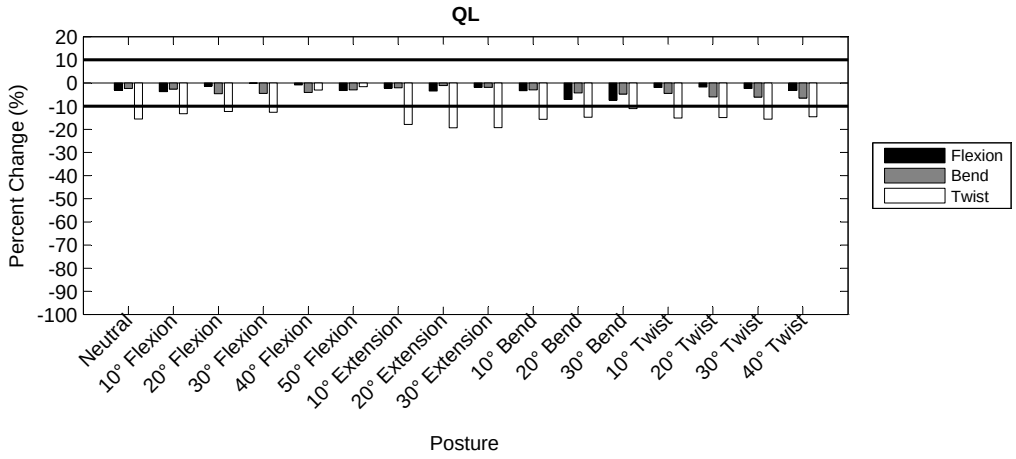


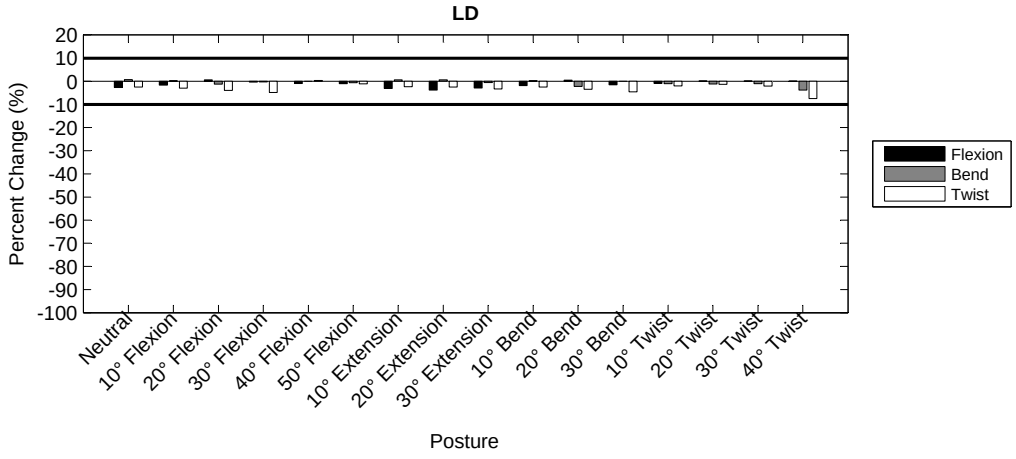


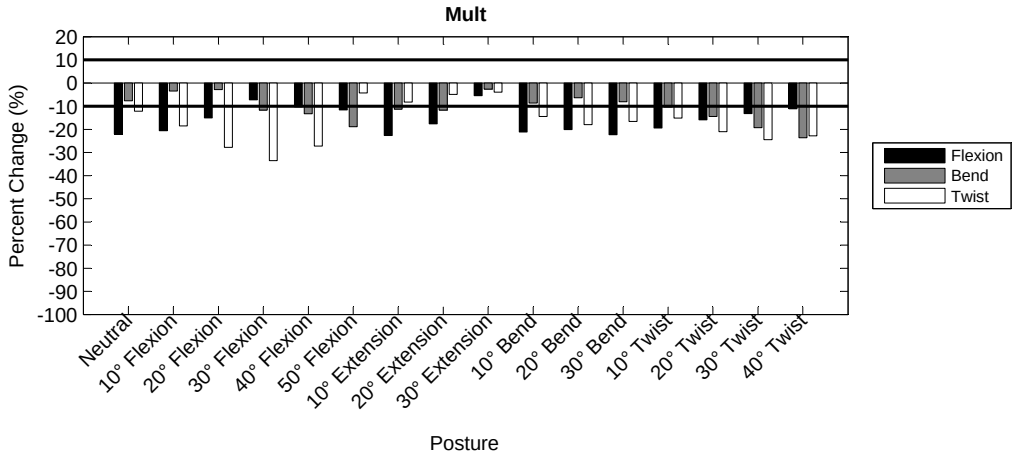



L3 Lumbar Level (0 %MVC)
Pars
Ilio
Long
Percent Change (%)
20
10
0
-10
-20
-30
-40
-50
-60
-70
-80
-90
-100
Neutral
10° Flexion
20° Flexion
30° Flexion
40° Flexion
50° Flexion
10° Extension
20° Extension
30° Extension
10° Bend
20° Bend
30° Bend
10° Twist
20° Twist
30° Twist
40° Twist
Posture
Flexion
Bend
Twist

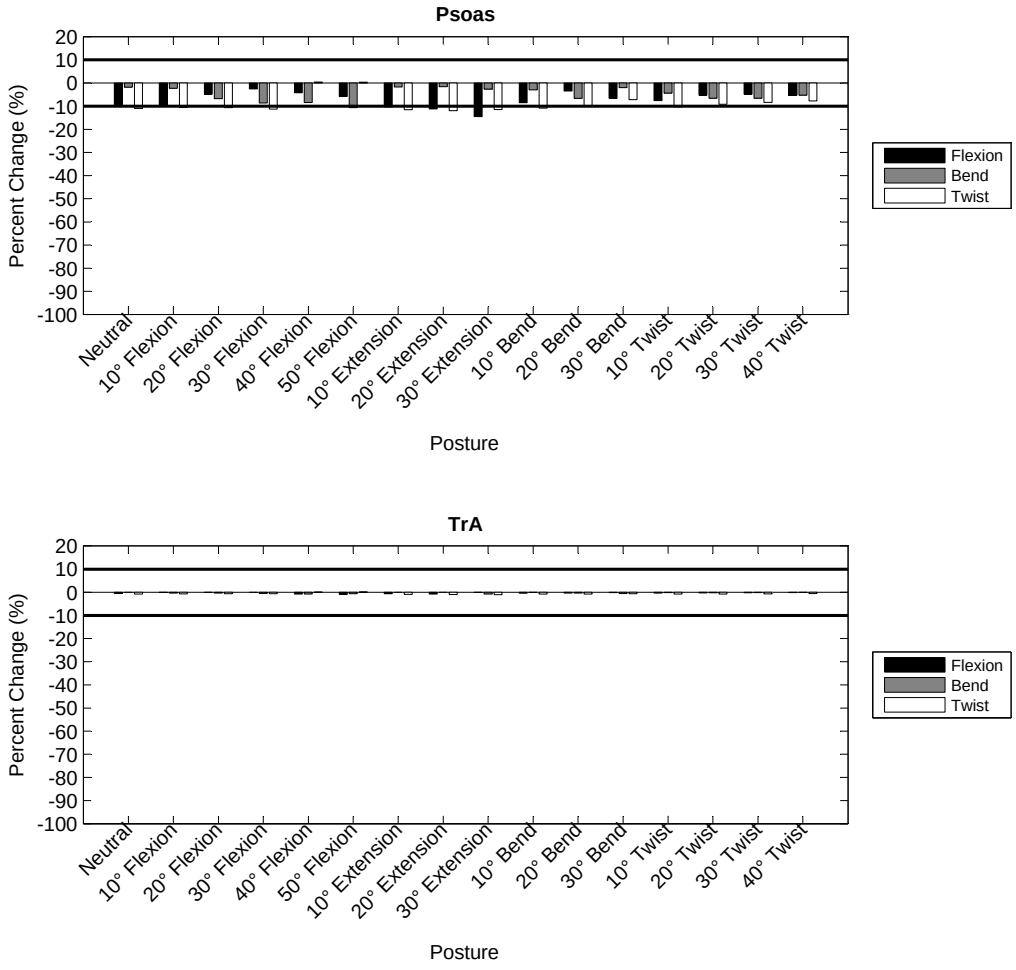
L3 Lumbar Level (0 %MVC)



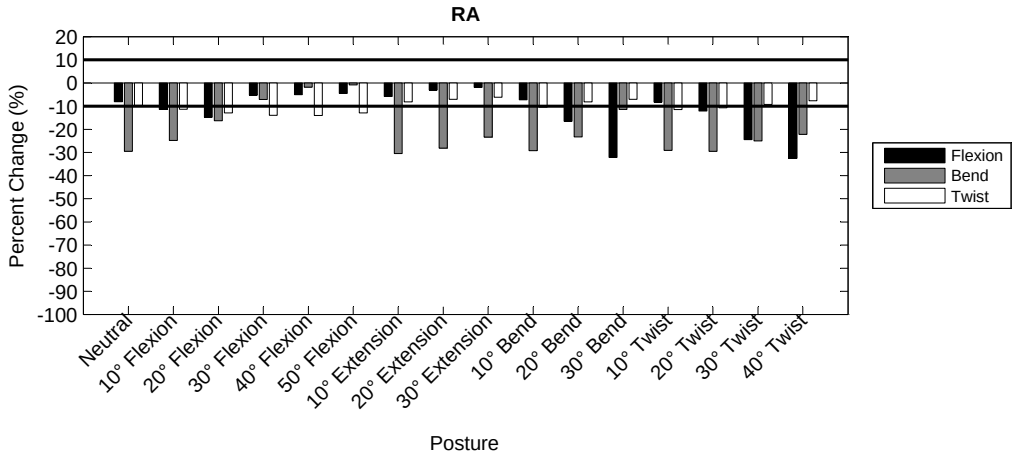


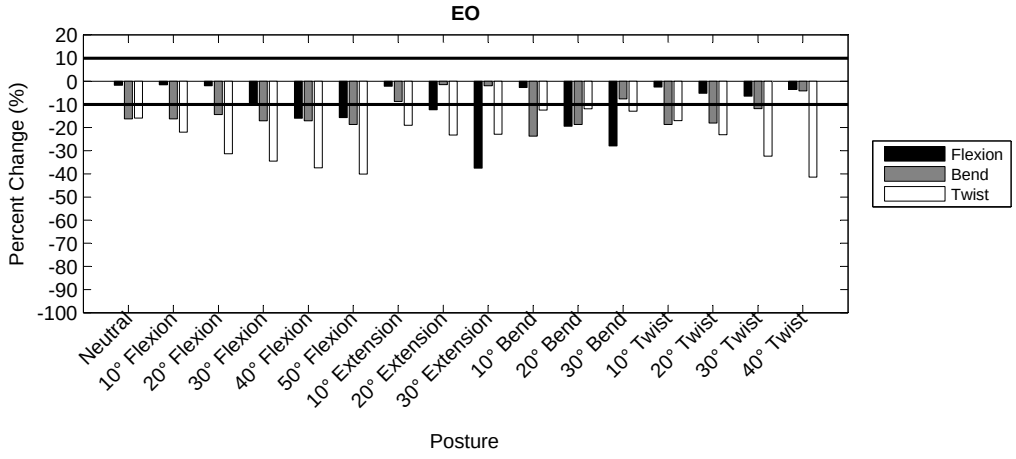


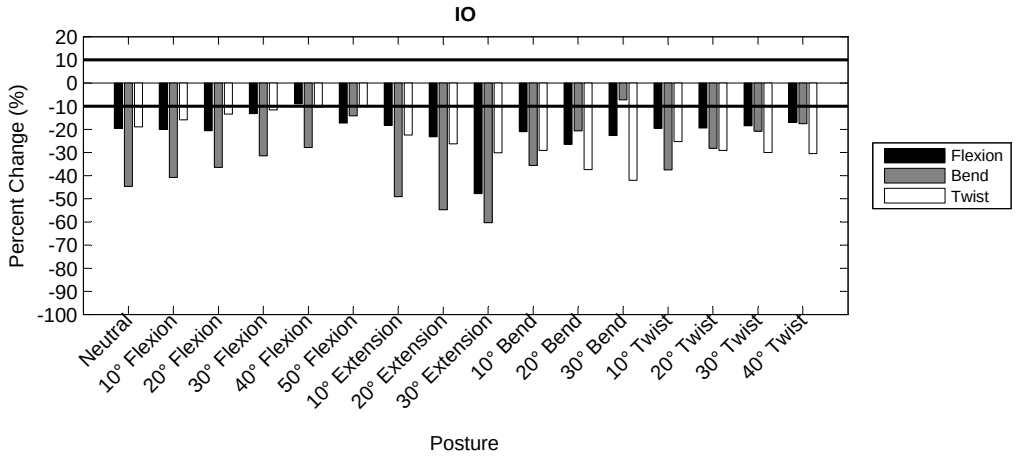
L3 Lumbar Level (0 %MVC)



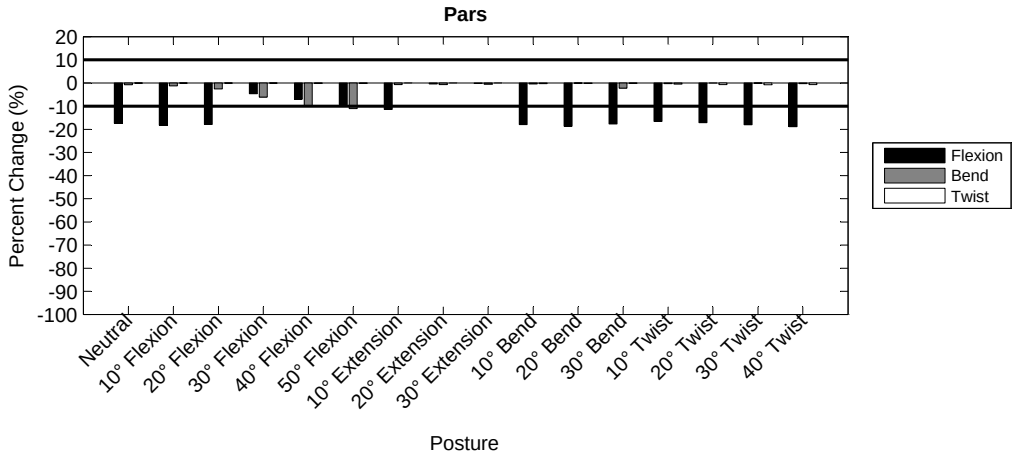
L5 Lumbar Level (0 %MVC)

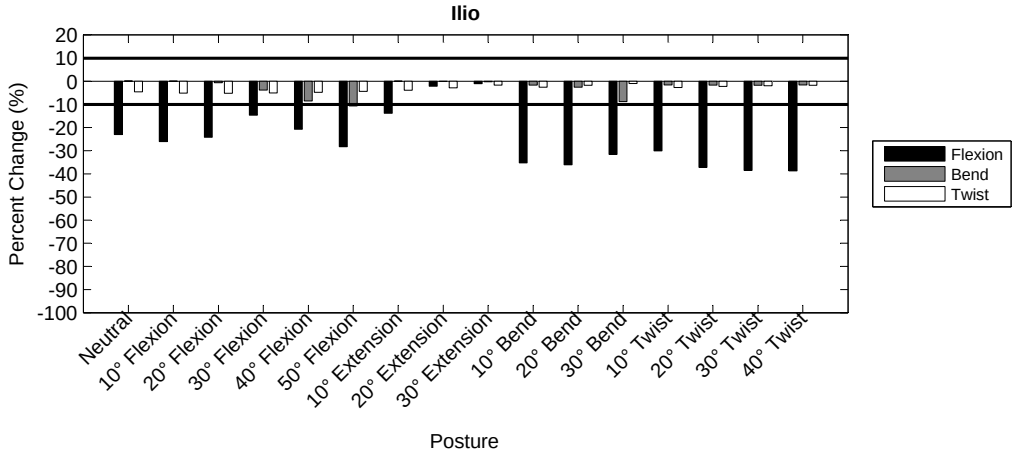


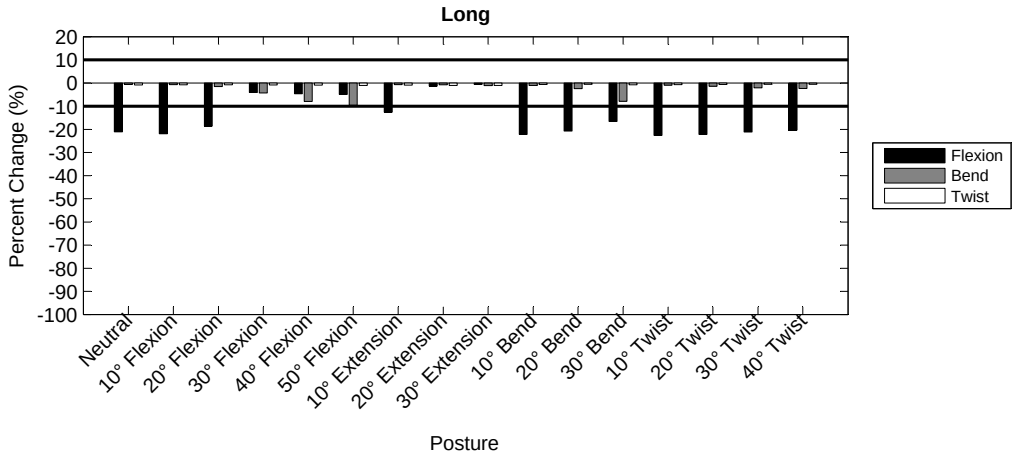




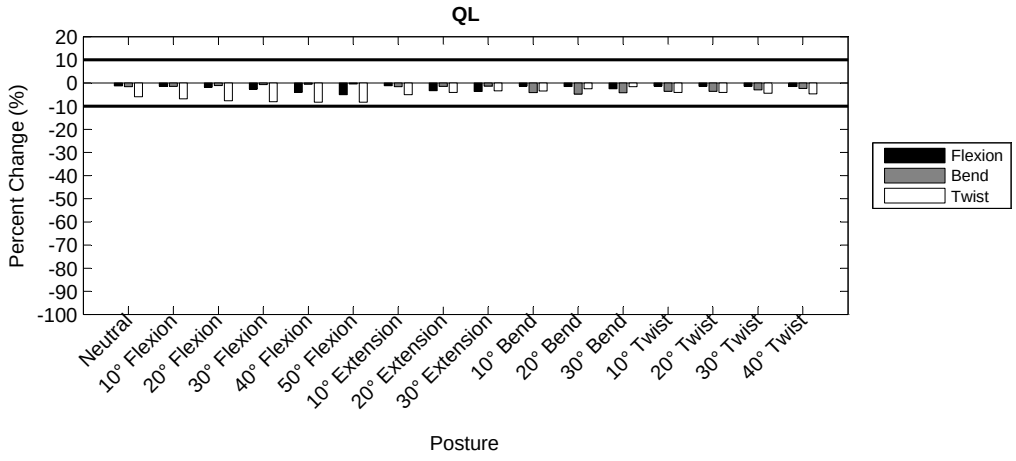
L5 Lumbar Level (0 %MVC)

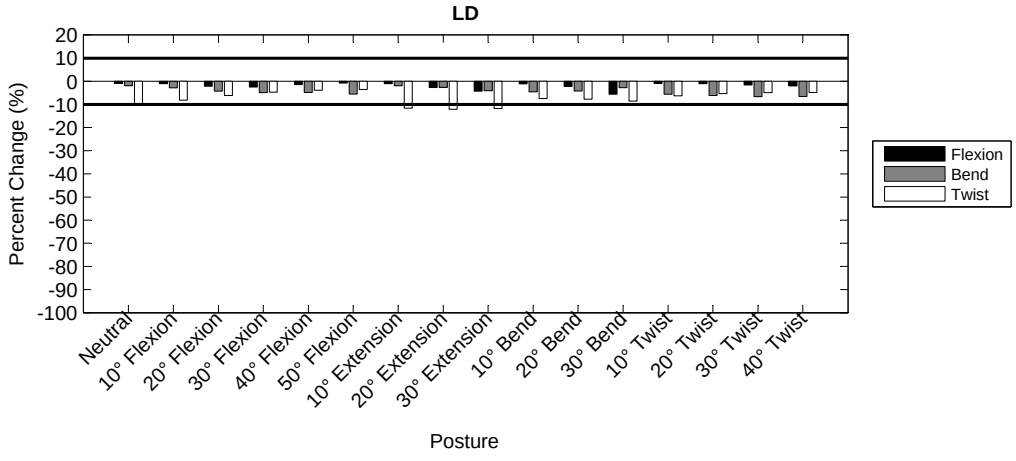


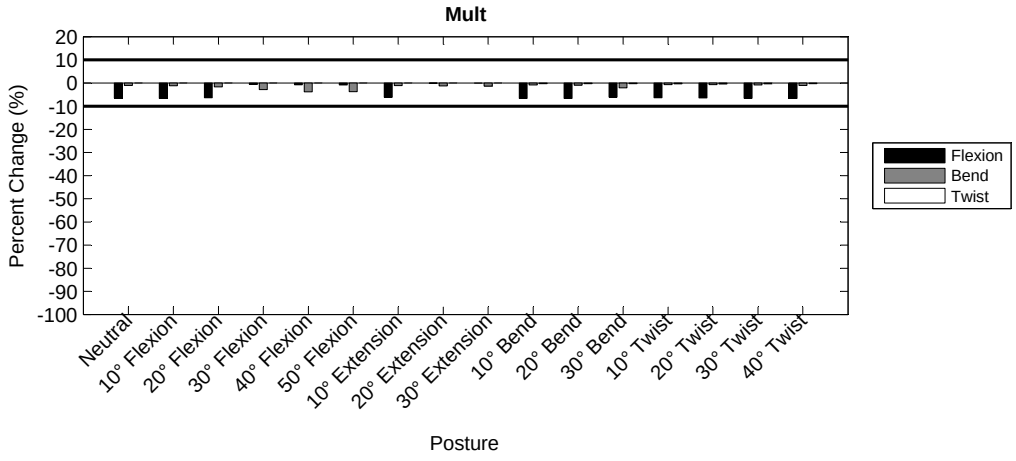




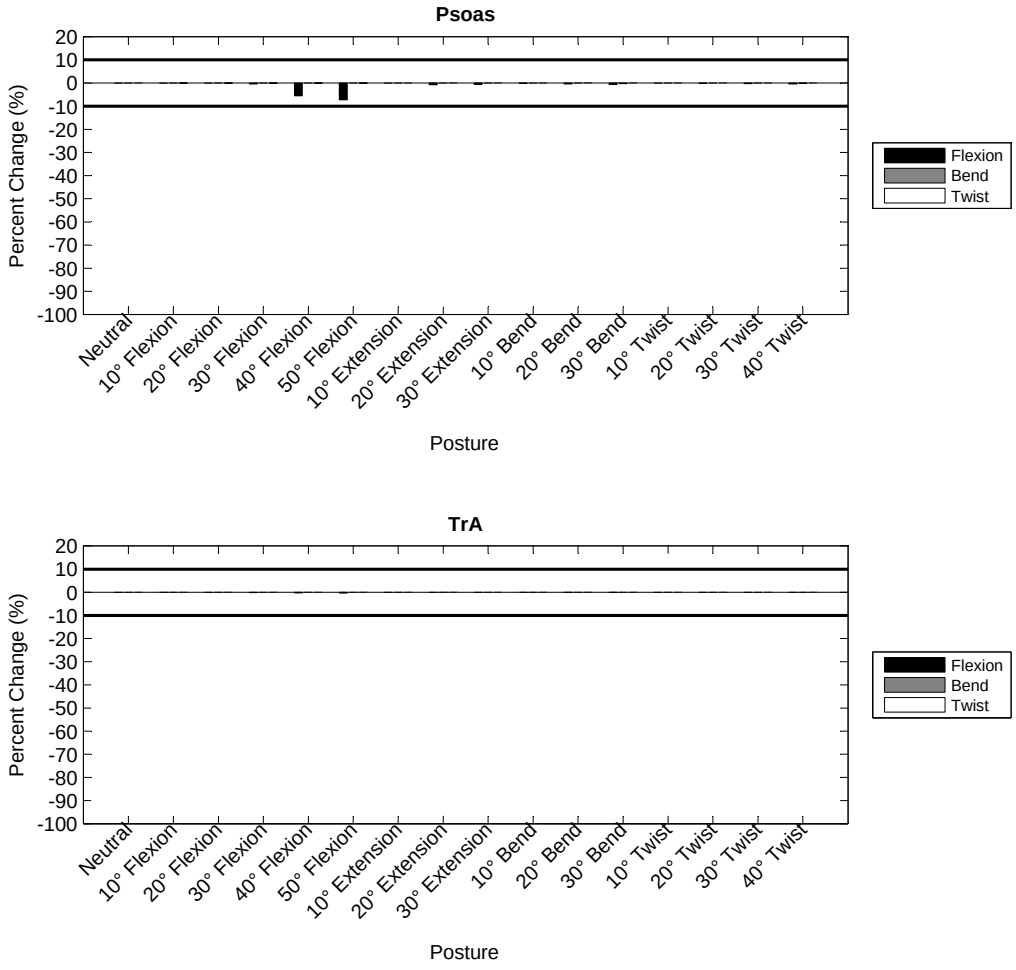
L5 Lumbar Level (0 %MVC)







L5 Lumbar Level (0 %MVC)

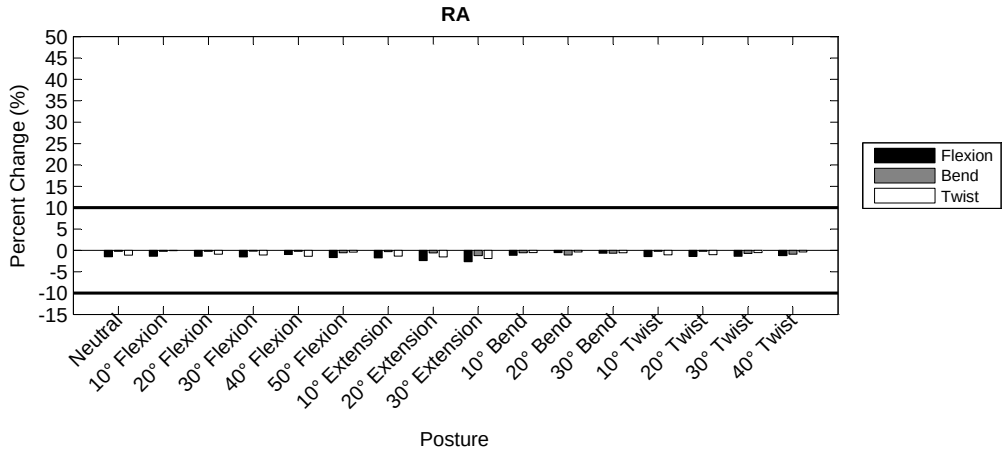


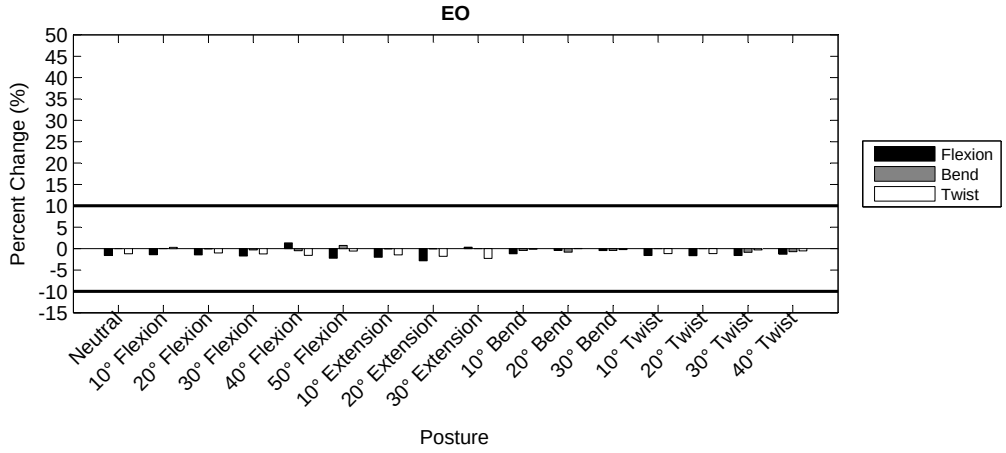
Appendix C

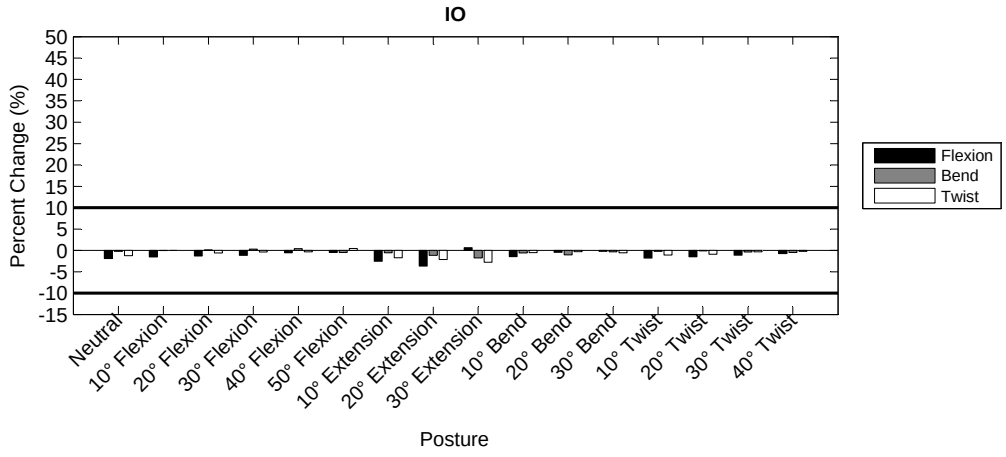
Effect of 100% muscle activation of single muscles on EVs

Each figure represents the percent change in three EVs when increasing single muscle activation to 100% MVC while all other muscles remained active to 50% MVC for various postures. The EV level being examined is the large title at the top of each page, the plane of stability/stiffness is in the legend, the muscle that was increased is the title of each graph and the posture being examined is along the x-axis. Positive change represents a higher EV when a single muscle's activity was increased to 100% MVC.. A change of 10% or greater in the EV was considered biologically significant. The thick black lines highlight these points. These figures are supplemental to section 4.1.4 (hypothesis 4 results).

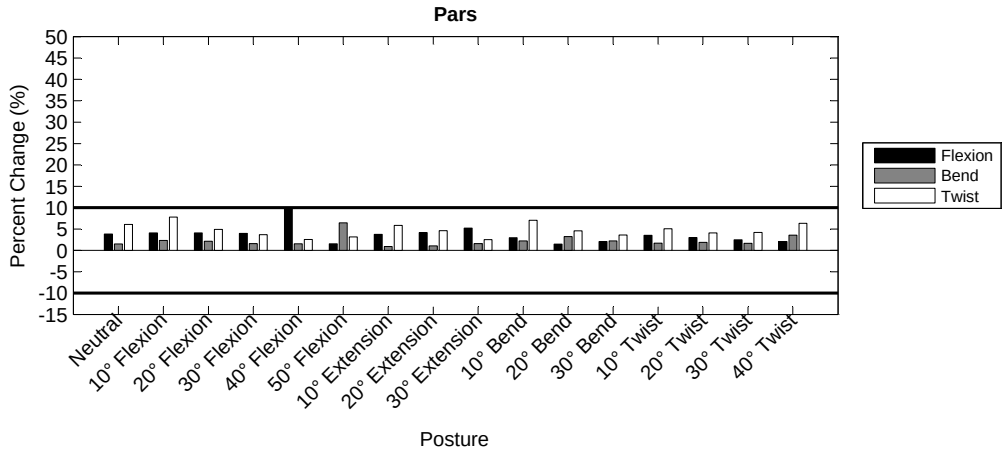
L1 Lumbar Level (100 %MVC)

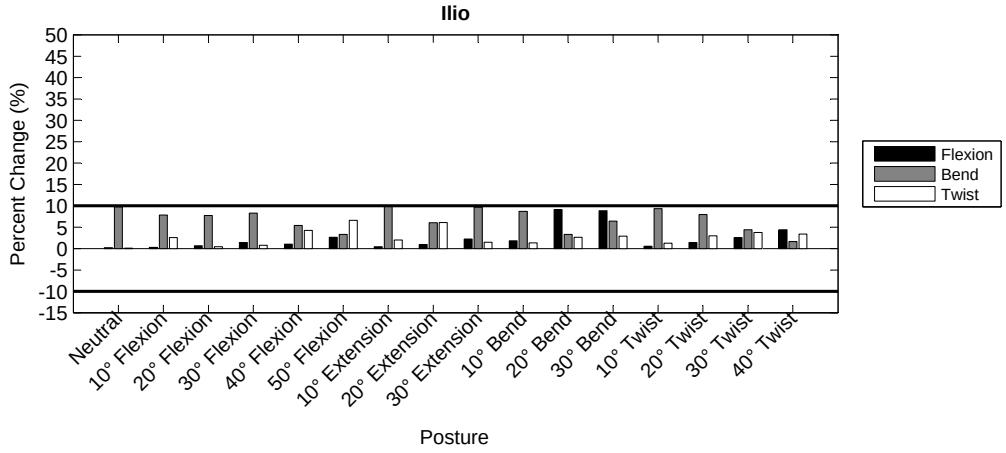


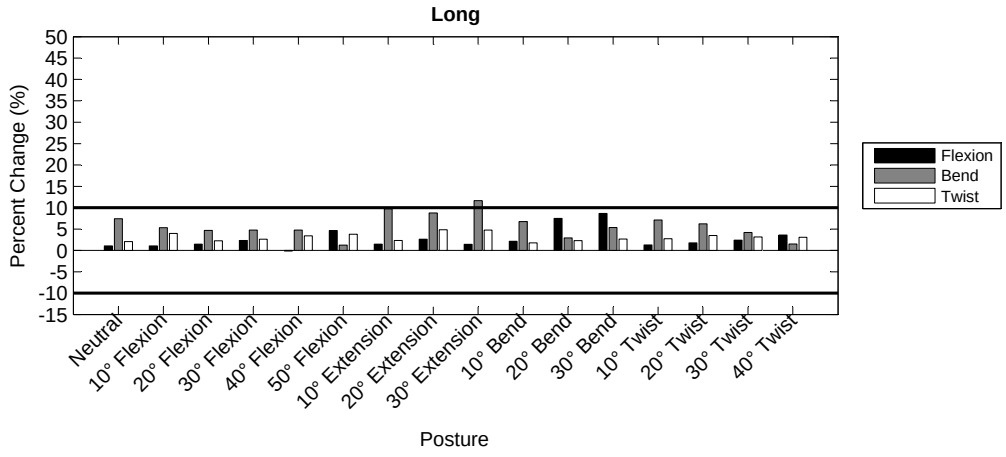




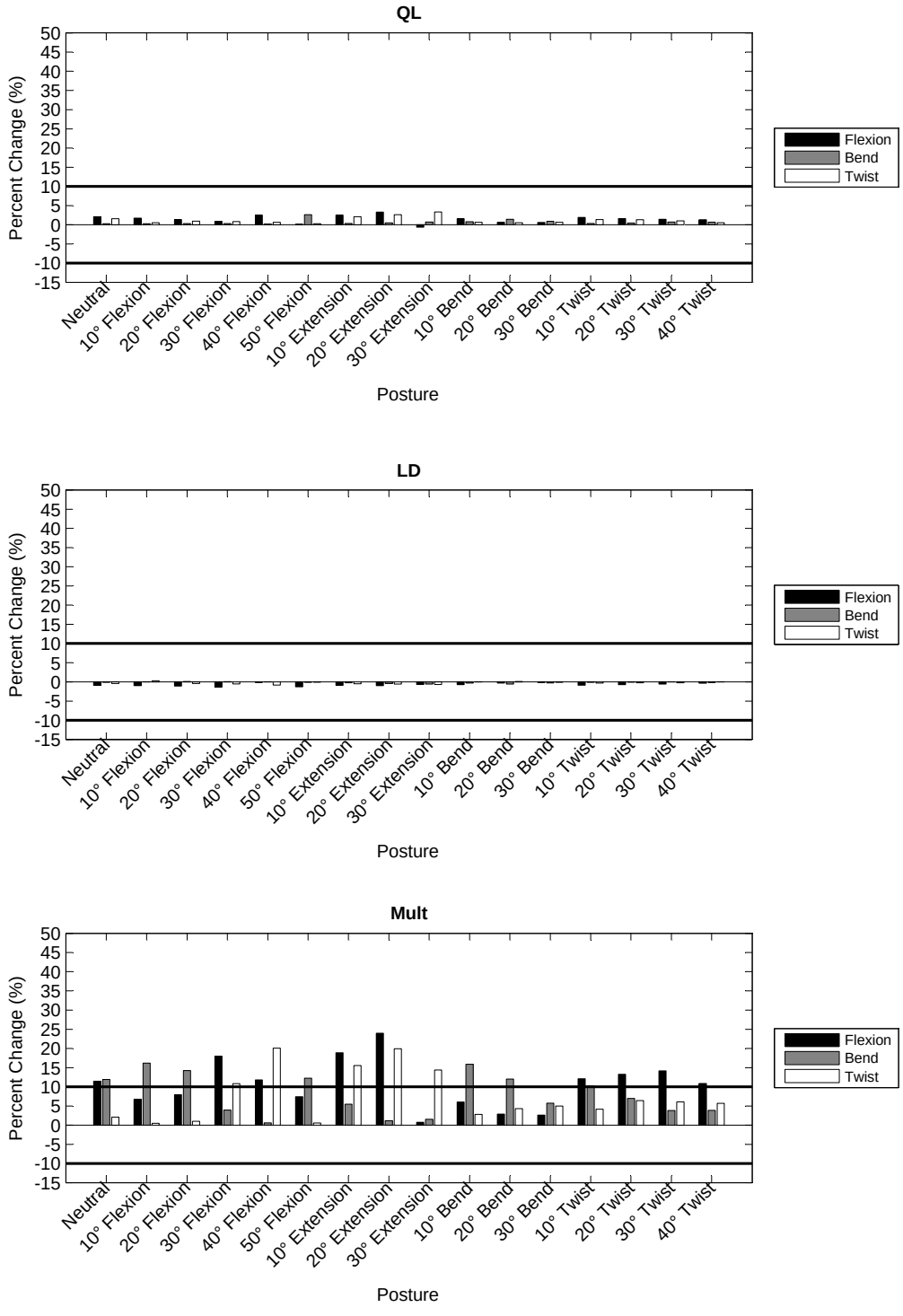
L1 Lumbar Level (100 %MVC)



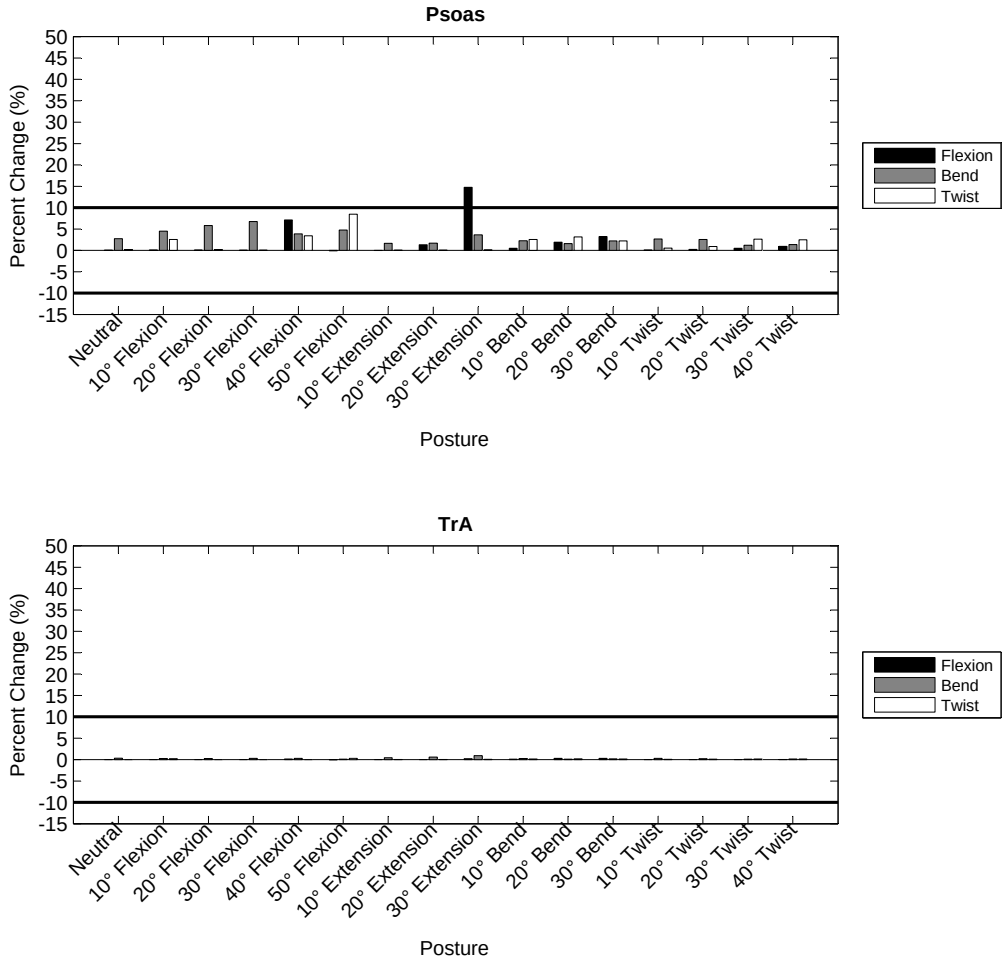




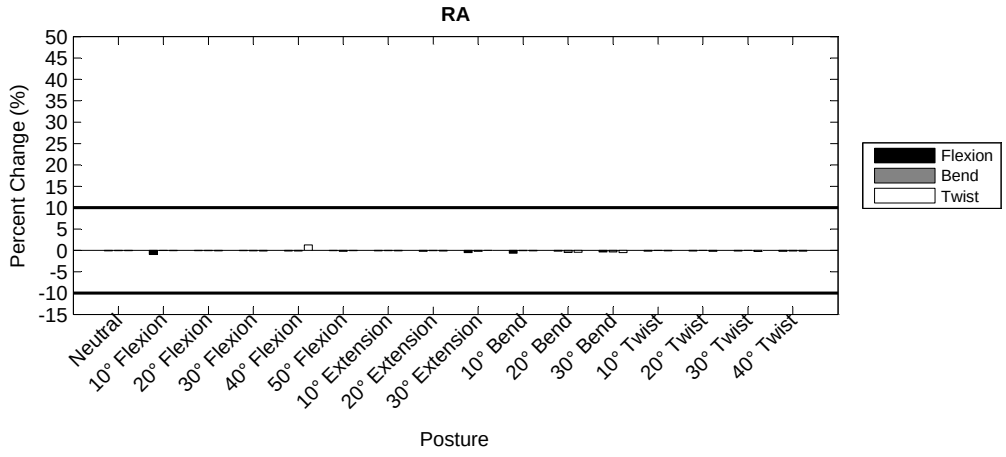
L1 Lumbar Level (100 %MVC)

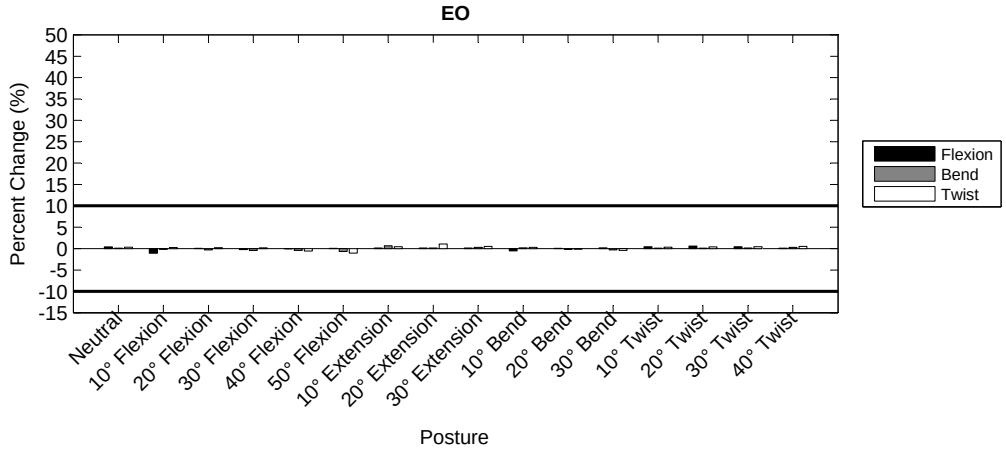


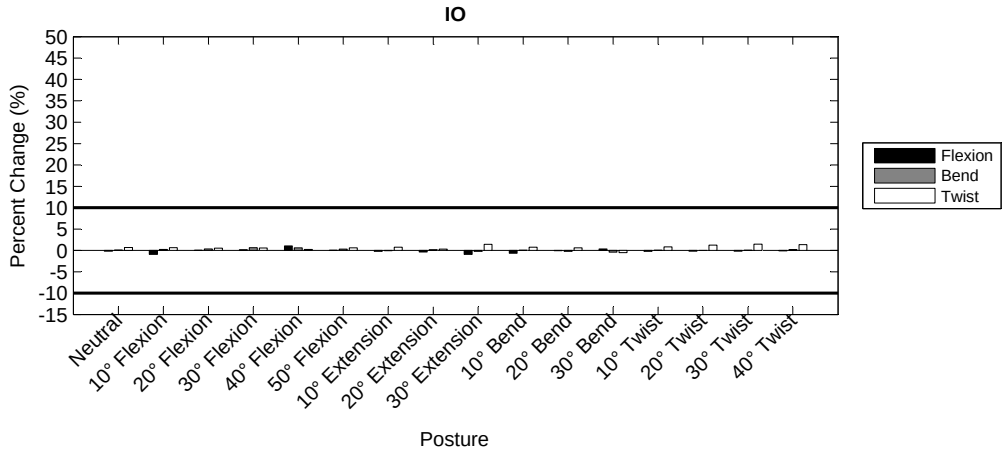
L1 Lumbar Level (100 %MVC)



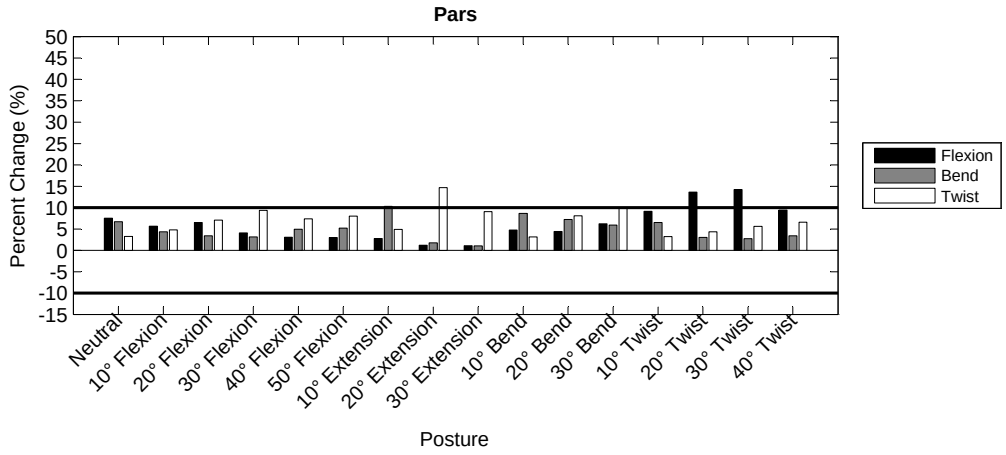
L2 Lumbar Level (100 %MVC)

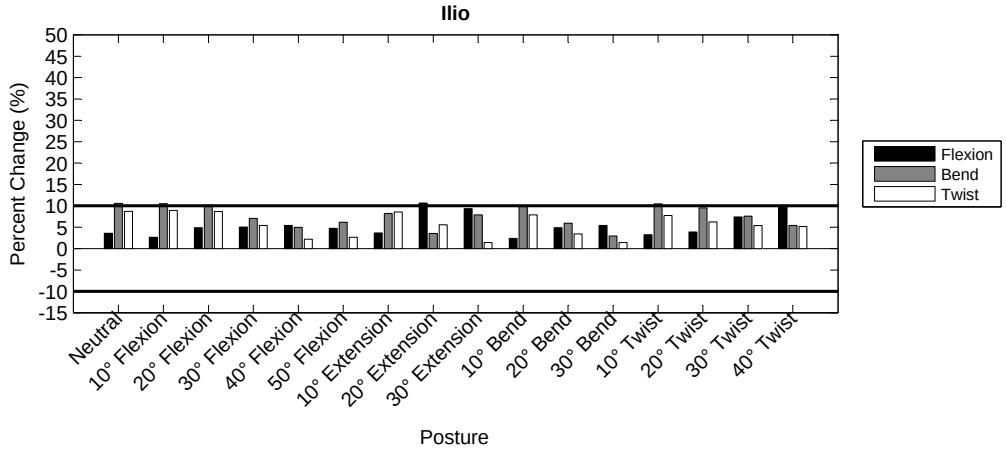


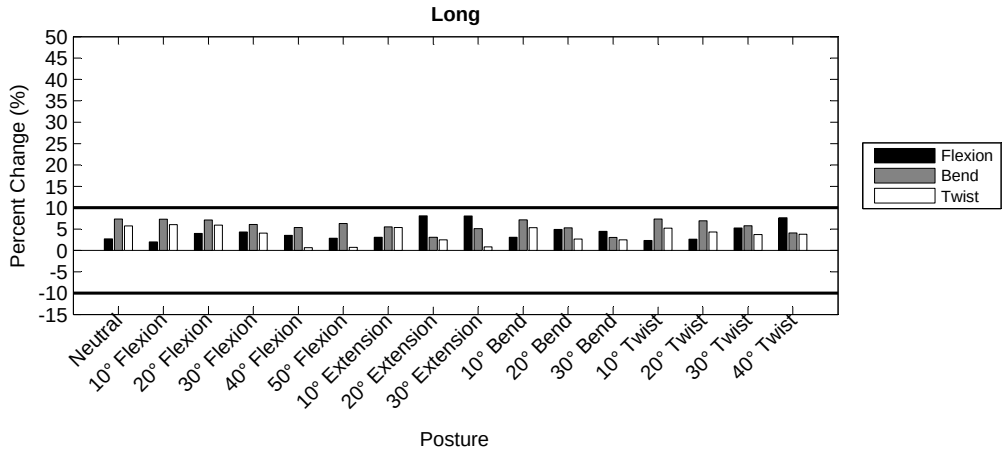




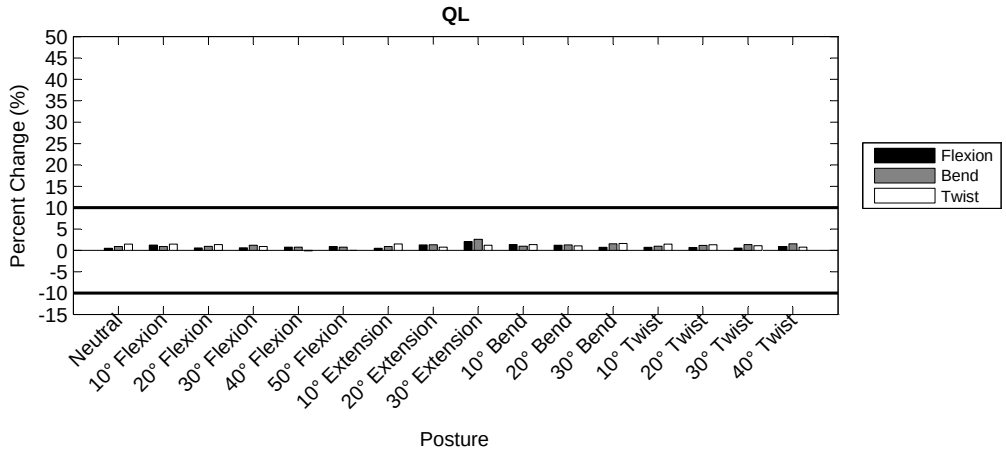
L2 Lumbar Level (100 %MVC)

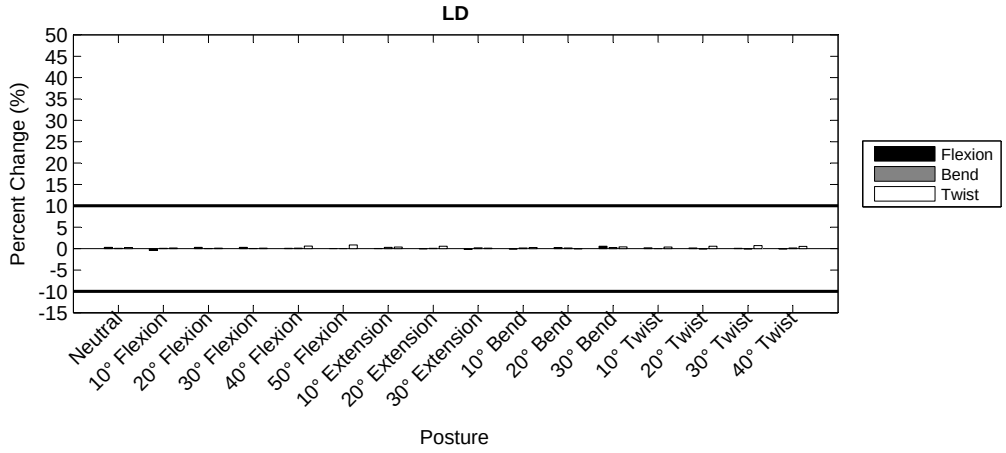


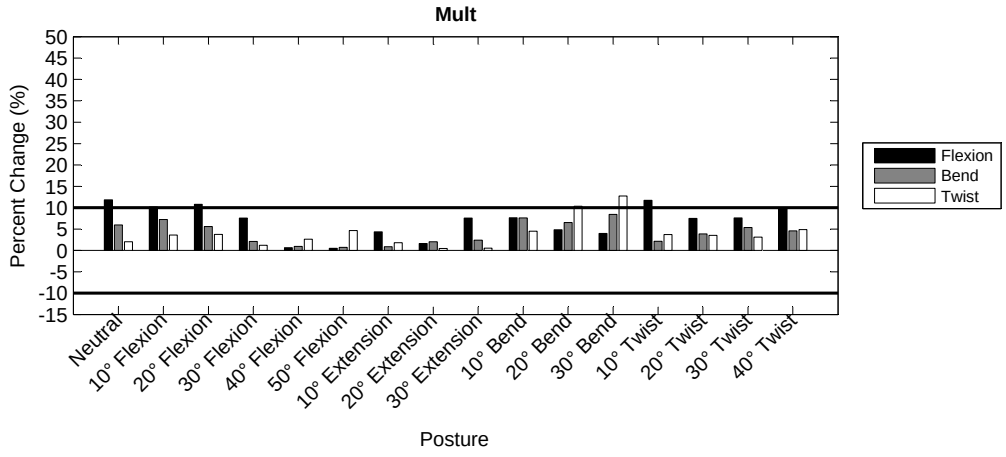




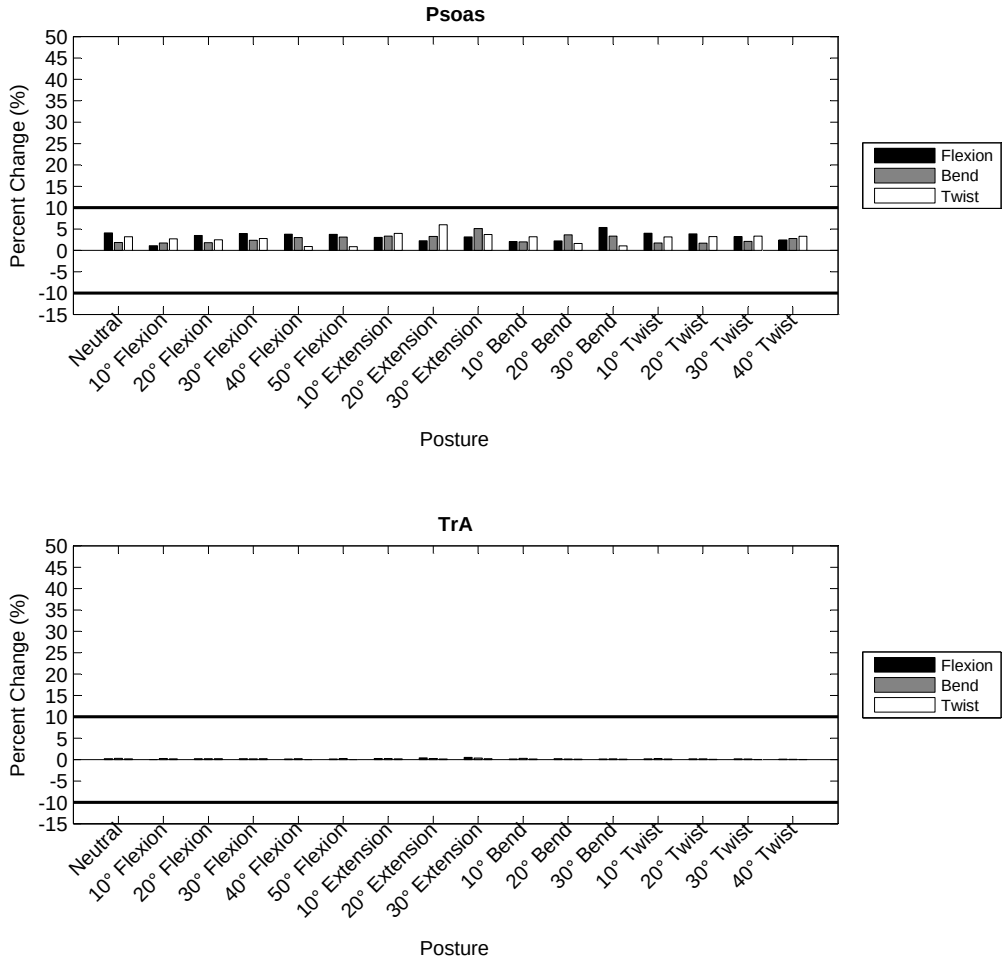
L2 Lumbar Level (100 %MVC)



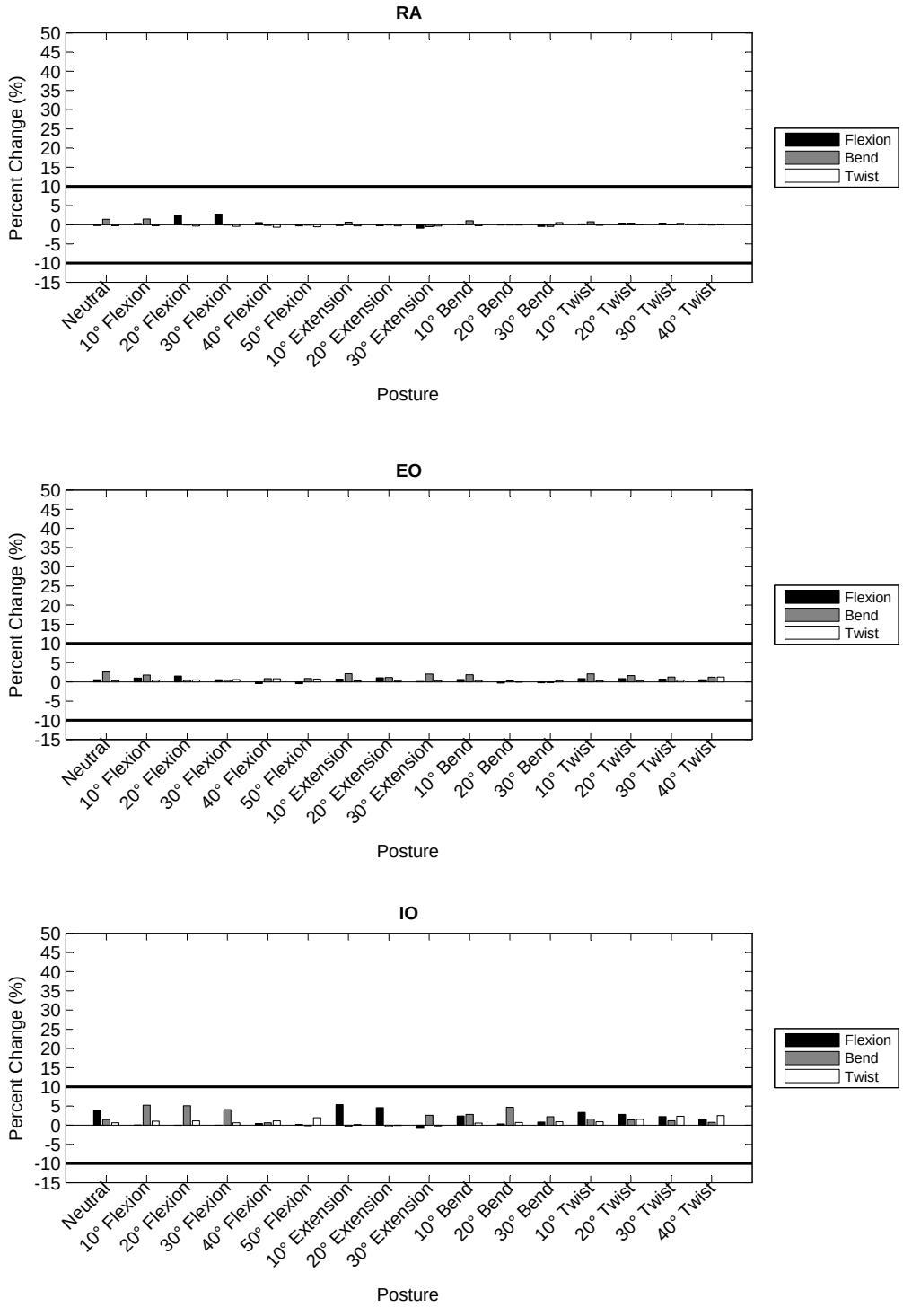




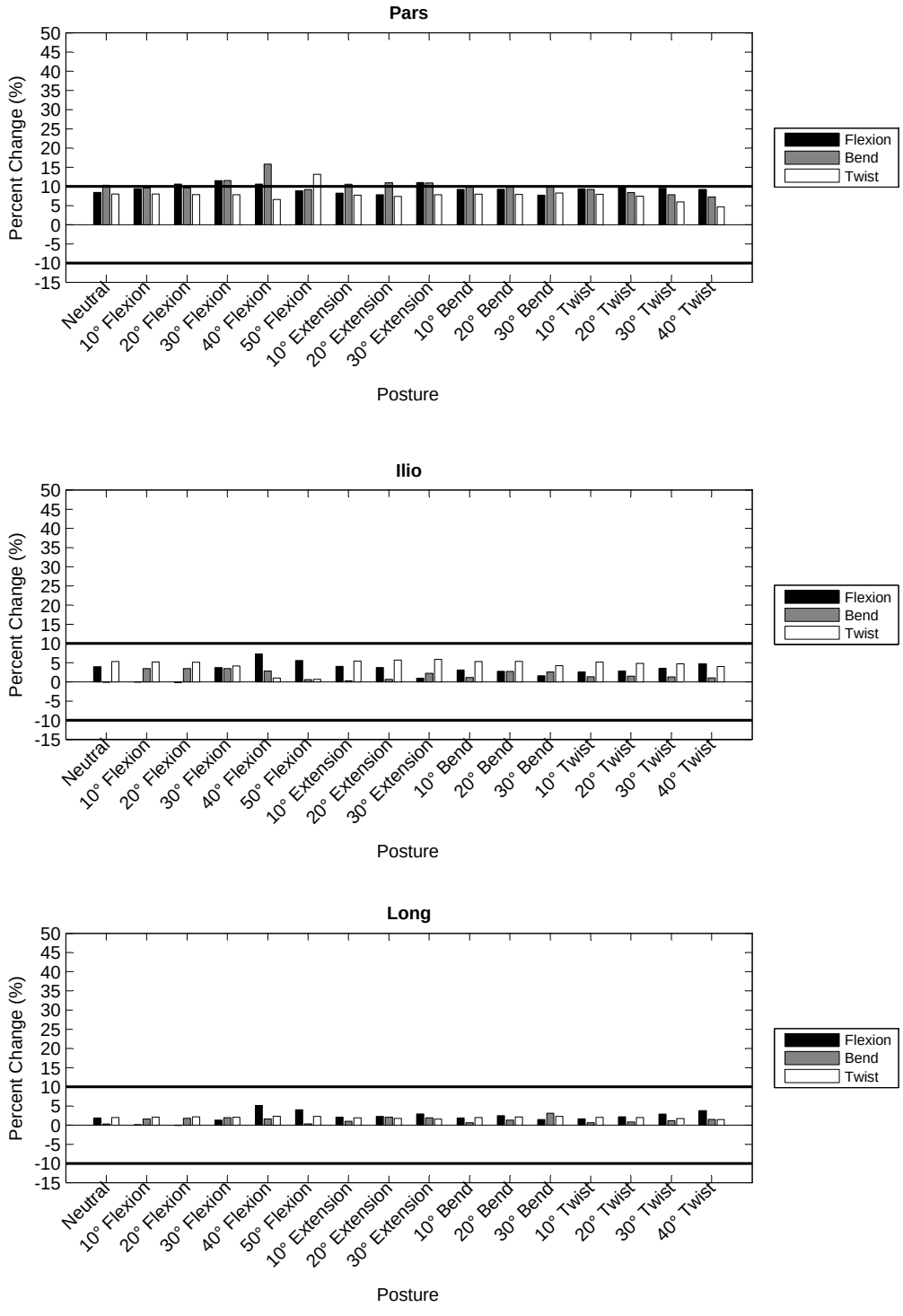
L2 Lumbar Level (100 %MVC)



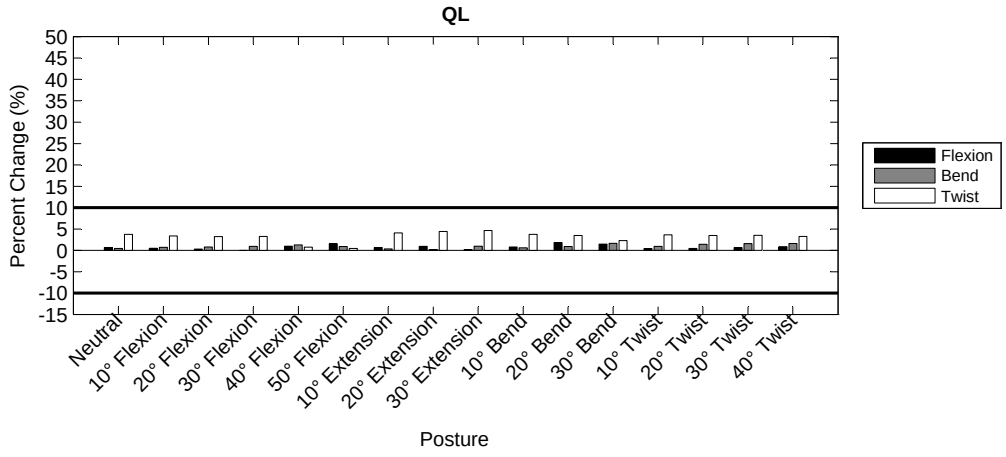
L3 Lumbar Level (100 %MVC)

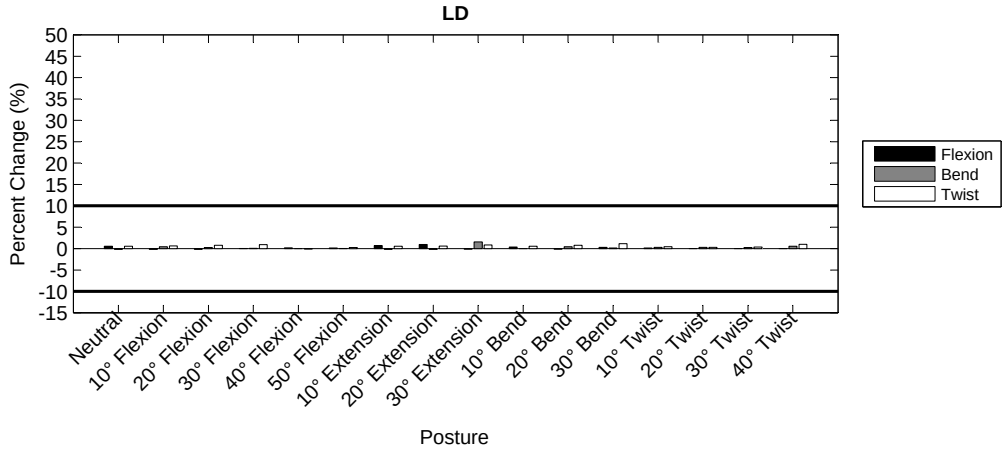


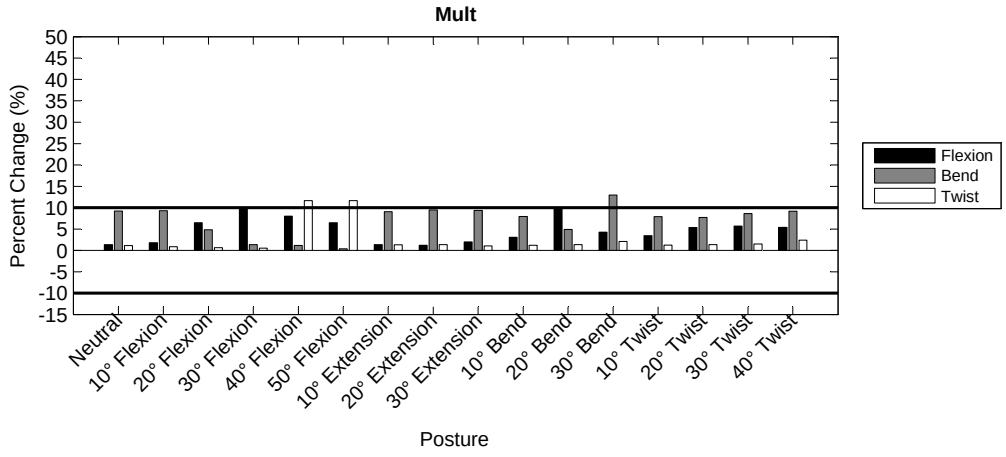
L3 Lumbar Level (100 %MVC)



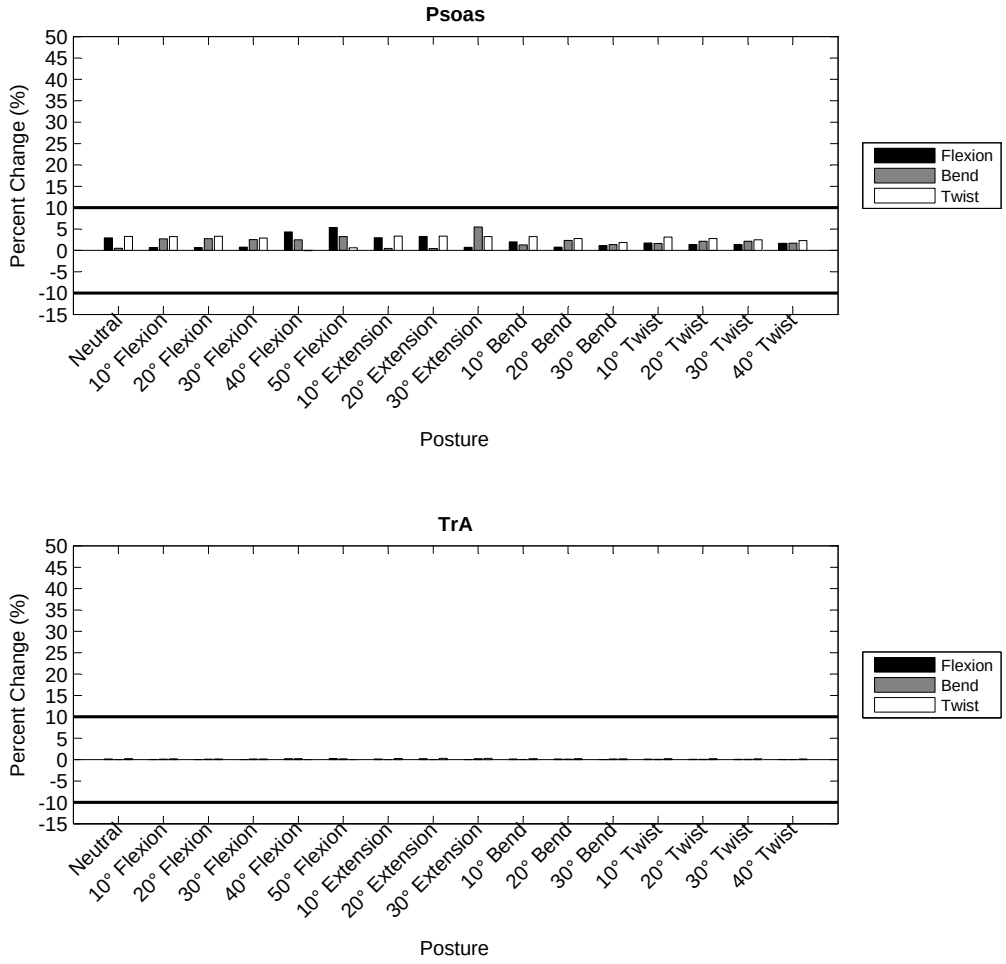
L3 Lumbar Level (100 %MVC)



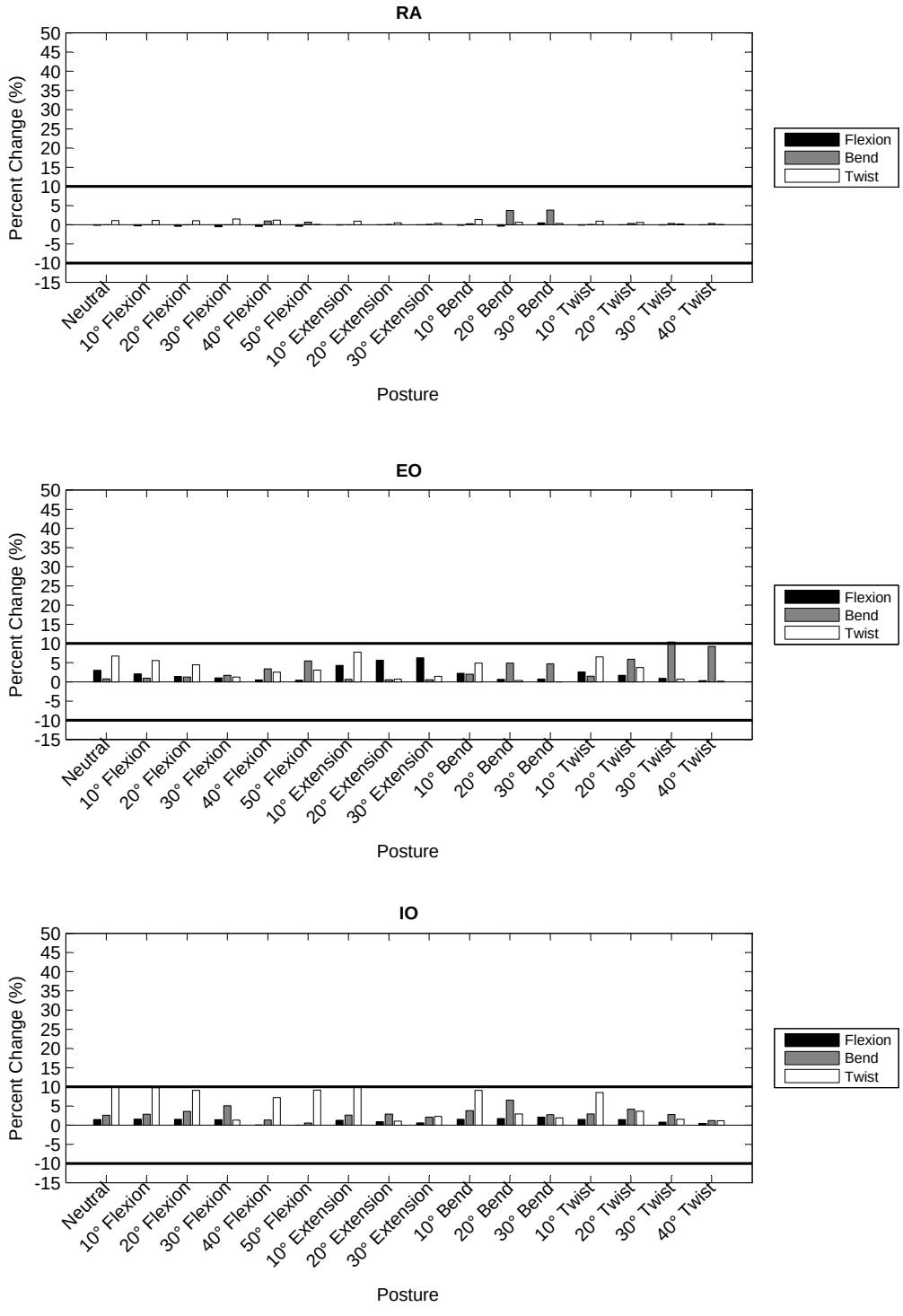




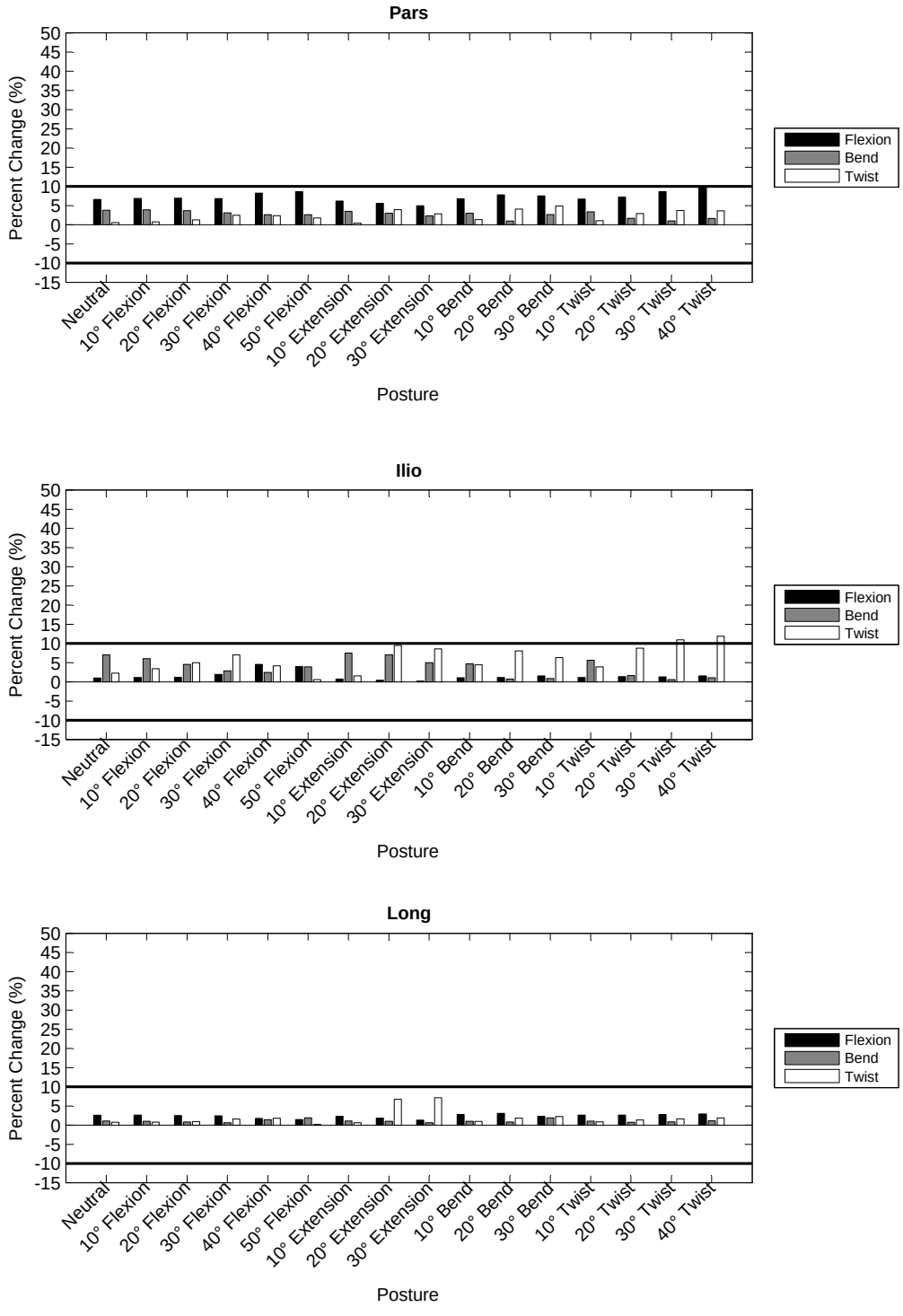
L3 Lumbar Level (100 %MVC)



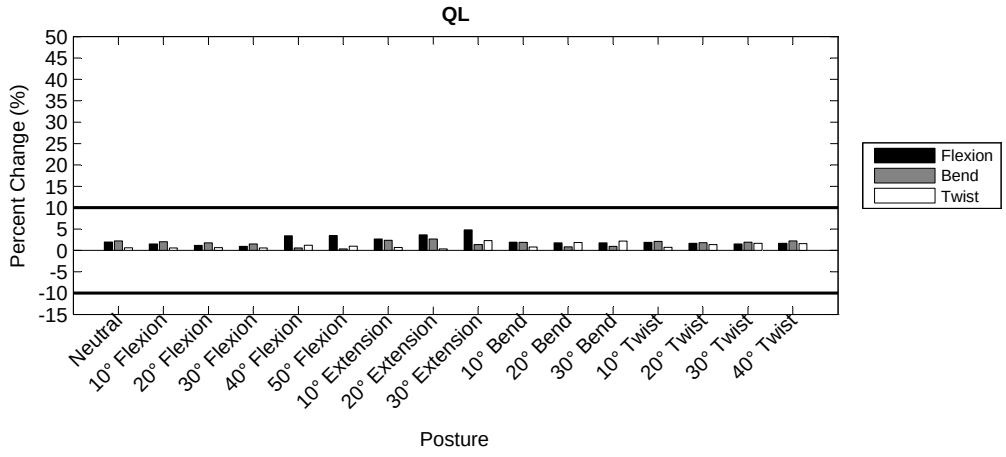
L4 Lumbar Level (100 %MVC)

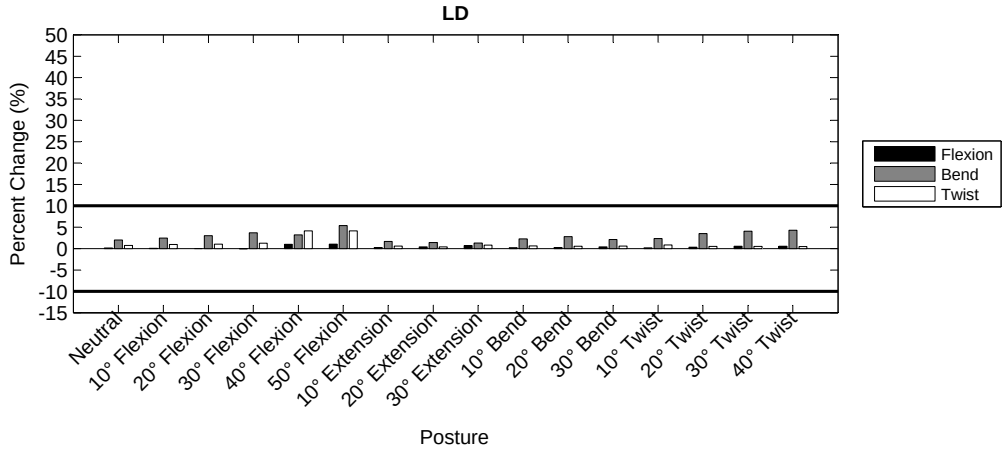


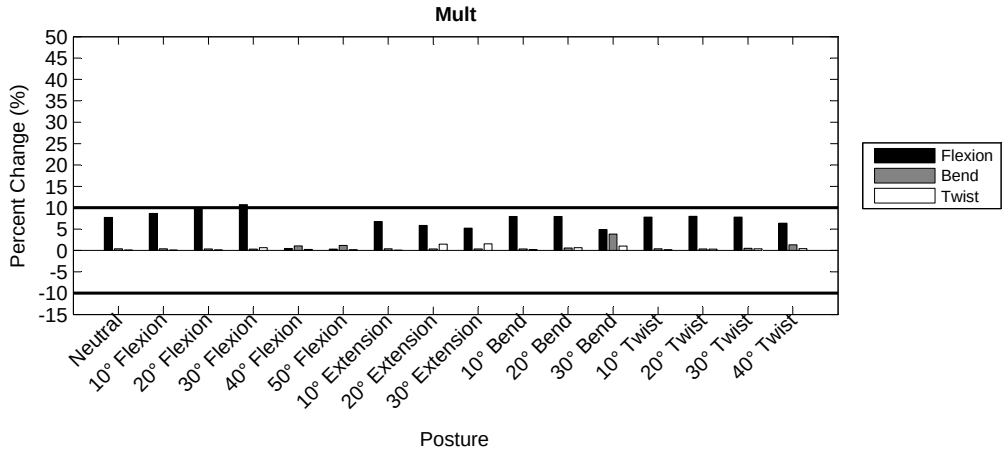
L4 Lumbar Level (100 %MVC)



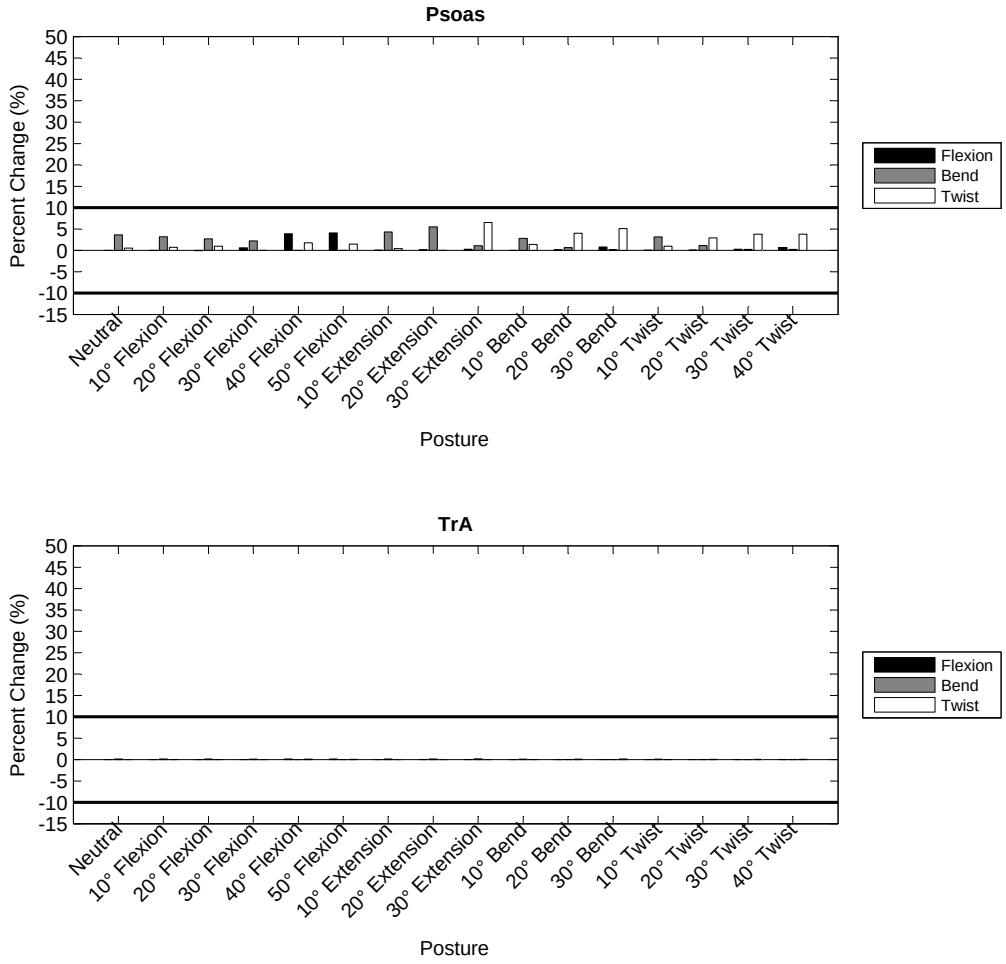
L4 Lumbar Level (100 %MVC)



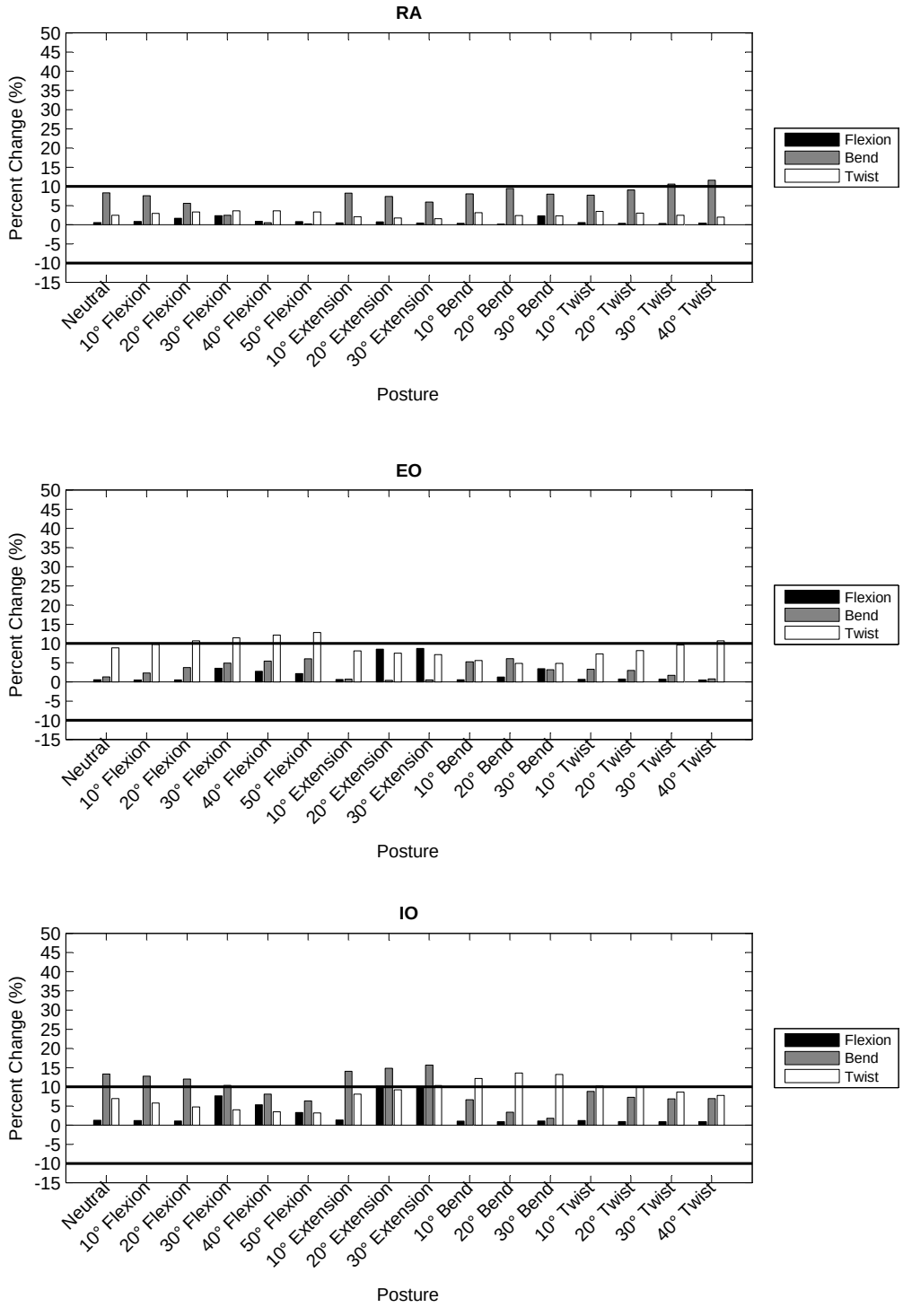




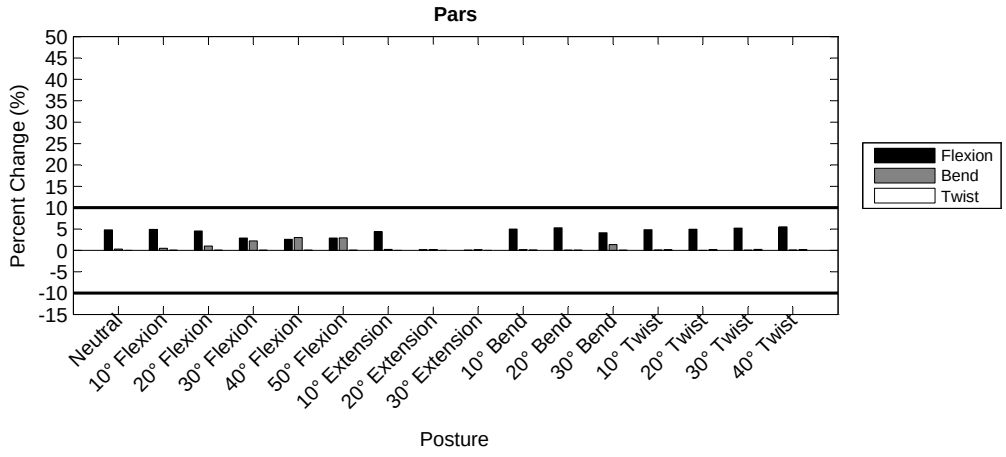
L4 Lumbar Level (100 %MVC)

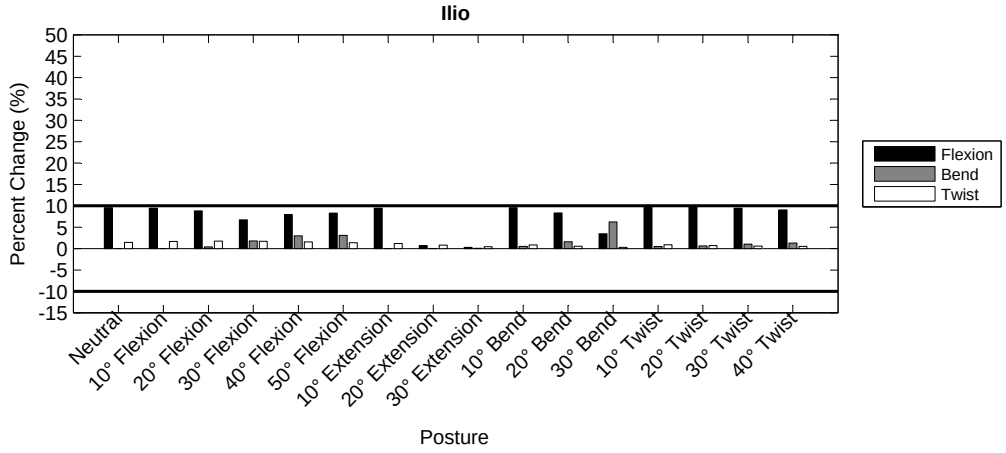


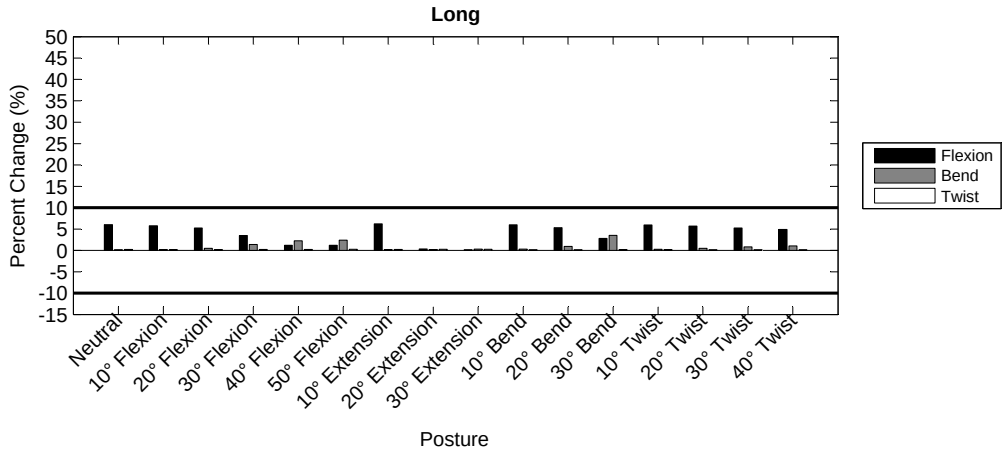
L5 Lumbar Level (100 %MVC)



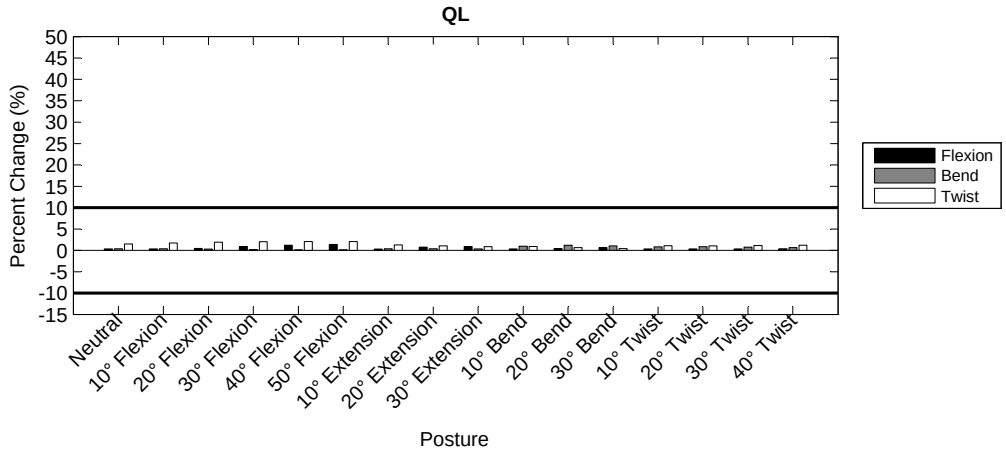
L5 Lumbar Level (100 %MVC)

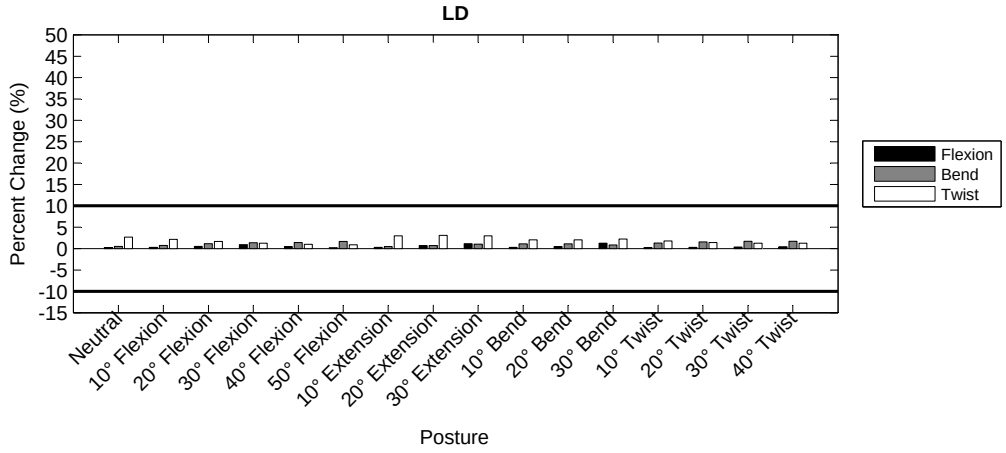


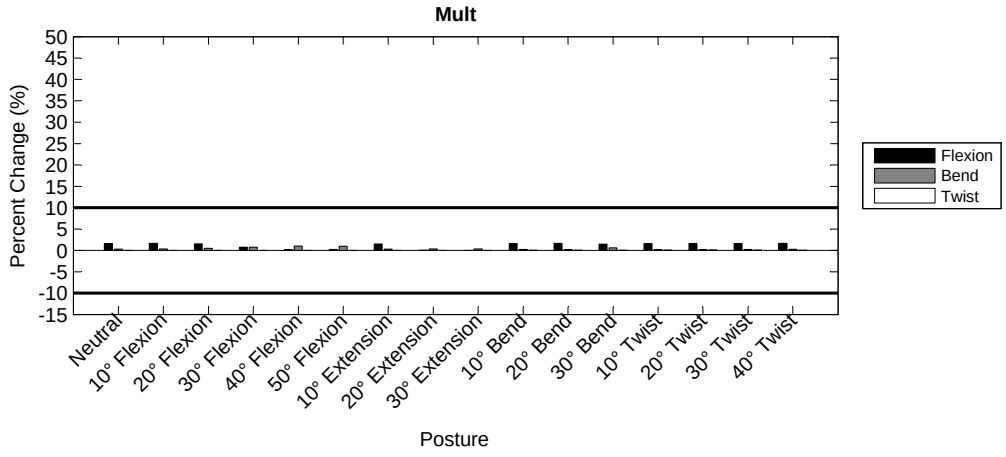




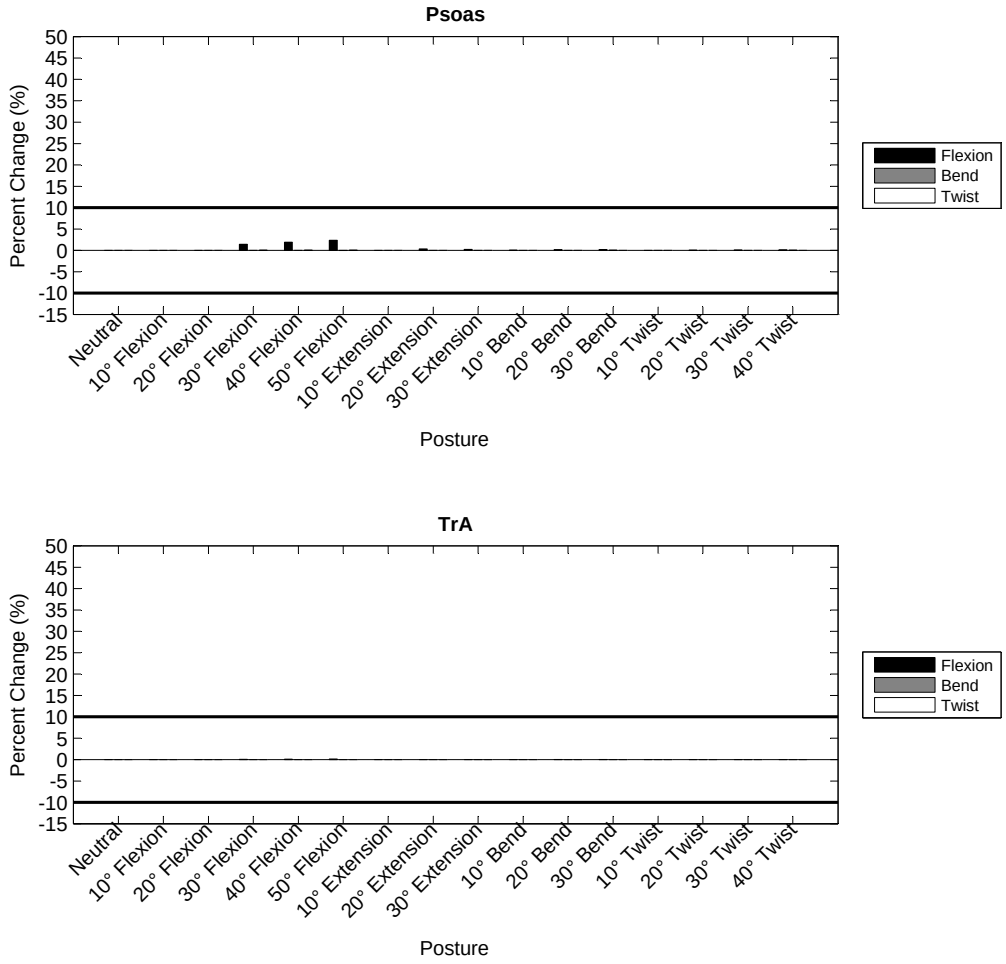
L5 Lumbar Level (100 %MVC)







L5 Lumbar Level (100 %MVC)



Appendix D

p-values for the actual data set sensitivity analysis

L1 Lumbar Level

	Comparison	RA	EO	IO	Pars	Ilio	Long	QL	LD	Mult	Psoas	TrA
L1F	0-A	0.9942	0.9978	0.9926	*0.5689*	*0.8719*	*0.7721*	0.8857	0.9984	***0.0133***	*0.3169*	0.4301
	A-100	*0.7095*	*0.8943*	*0.9186*	*0.3807*	*0.8218*	*0.4522*	*0.5588*	0.8086	*0.1388*	*0.3488*	**0.0308**
L1B	0-A	0.995	0.9947	0.9945	*0.5382*	*0.8874*	*0.773*	0.8926	0.9979	*0.1438*	*0.3954*	0.4022
	A-100	*0.7345*	0.9627	*0.9331*	*0.3349*	*0.7374*	*0.2435*	*0.4769*	0.8497	***0.0335***	*0.1512*	**0.006**
L1T	0-A	0.9994	0.9947	0.9897	*0.4511*	*0.8551*	*0.7248*	0.8363	0.9928	*0.1271*	*0.3552*	0.2015
	A-100	*0.8487*	0.9668	0.9633	*0.2013*	*0.6657*	*0.2092*	*0.356*	0.9253	***0.0044***	*0.1045*	**0.0001**

L2 Lumbar Level

	Comparison	RA	EO	IO	Pars	Ilio	Long	QL	LD	Mult	Psoas	TrA
L2F	0-A	0.9932	0.9887	0.9718	*0.3678*	*0.8516*	0.7709	0.7842	0.9411	*0.1258*	*0.3776*	0.2731
	A-100	0.9079	0.998	0.9759	***0.007***	*0.5109*	*0.1386*	*0.367*	0.9743	***0.0011***	*0.0702*	**0.0009**
L2B	0-A	0.9909	0.9905	0.9556	*0.3685*	*0.8533*	*0.7282*	0.8128	0.9528	*0.1016*	*0.3463*	0.162
	A-100	0.9338	0.9982	0.9922	***0.0194***	*0.2057*	***0.0321***	*0.4062*	0.9632	***<0.0001***	***0.0439***	**0.0004**
L2T	0-A	0.9856	0.987	0.9483	*0.403*	0.9056	0.803	0.8236	0.9659	*0.0572*	*0.3137*	0.2925
	A-100	0.9319	0.904	0.9892	***0.0054***	*0.1314*	***0.001***	*0.3117*	0.9576	***<0.0001***	***0.0229***	**0.0035**

* Shaded cells = biologically and statistically significant difference
* Italicized cells = biologically significant difference
* Bolded cells = statistically significant difference

L3 Lumbar Level

	Comparison	RA	EO	IO	Pars	Ilio	Long	QL	LD	Mult	Psoas	TrA
L3F	0-A	0.9919	0.9852	0.9351	*0.3624*	0.9102	0.8301	0.8338	0.9316	*0.1412*	*0.2627*	0.152
	A-100	0.9883	0.8759	0.9975	***<0.0001***	***0.0375***	***0.0008***	*0.2492*	0.7806	***<0.0001***	***0.0055***	**<0.0001**
L3B	0-A	0.9862	0.9672	*0.8703*	*0.2725*	0.8633	0.8463	0.8517	0.9254	*0.1238*	*0.2561*	0.2738
	A-100	0.9524	*0.8479*	*0.8897*	***<0.0001***	***0.0352***	***0.002***	*0.2435*	0.7812	***<0.0001***	***0.0089***	**0.0029**
L3T	0-A	0.9932	0.896	*0.801*	*0.1414*	*0.7169*	0.6665	0.5853	0.8713	***0.0161***	*0.1253*	0.1224
	A-100	0.8928	*0.7699*	*0.6832*	***<0.0001***	***0.0466***	***0.0031***	***0.0049***	0.6138	***<0.0001***	***0.0116***	**<0.0001**

L4 Lumbar Level

	Comparison	RA	EO	IO	Pars	Ilio	Long	QL	LD	Mult	Psoas	TrA
L4F	0-A	0.9997	0.8759	*0.7358*	*0.1094*	0.75	0.5832	0.4863	0.8877	***0.0198***	0.5402	0.3887
	A-100	0.8001	*0.4284*	*0.4906*	***<0.0001***	***0.048***	***0.0024***	***0.0009***	0.4948	***<0.0001***	***<0.0001***	**0.0016**
L4B	0-A	0.9044	***0.0138***	***0.043***	*0.0708*	*0.4575*	0.3676	0.0932	0.1325	0.3087	***<0.0001***	**0.009**
	A-100	0.5295	*0.2087*	*0.1529*	***<0.0001***	***0.0446***	***0.0002***	***<0.0001***	***0.0002***	***<0.0001***	***0.0004***	**<0.0001**
L4T	0-A	0.798	*0.3555*	***0.0311***	***0.0341***	*0.2255*	0.1749	0.0722	0.3215	0.1566	**0.0012**	**0.0063**
	A-100	***0.022***	***0.0108***	***0.0252***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	**<0.0001**

* Shaded cells = biologically and statistically significant difference
* Italicized cells = biologically significant difference
* Bolded cells = statistically significant difference

L5 Lumbar Level

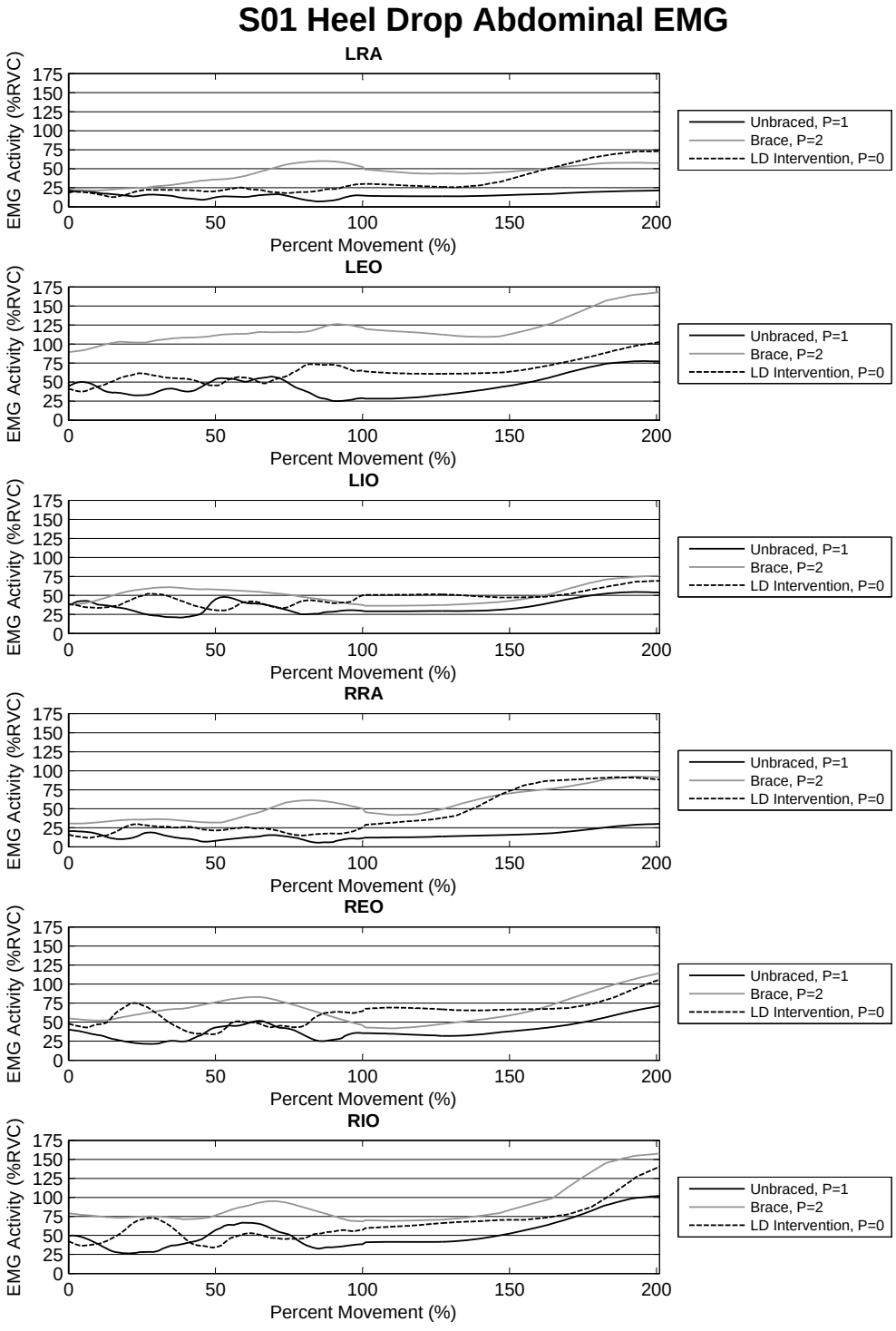
	Comparison	RA	EO	IO	Pars	Ilio	Long	QL	LD	Mult	Psoas	TrA
L5F	0-P	0.5202	0.488	***0.0135***	***0.0125***	*0.0534*	0.0587	0.1555	0.1273	**0.0363**	0.0745	0.084
	P-100	***<0.0001***	***<0.0001***	***0.0011***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	**<0.0001**
L5B	0-P	0.3995	0.1385	***<0.0001***	0.1025	0.3236	0.2638	0.1203	**0.0233**	**0.024**	0.7298	0.7483
	P-100	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	0.2201
L5T	0-P	0.4528	***0.0069***	***<0.0001***	0.7039	0.3726	0.4464	**0.001**	**<0.0001**	0.5431	0.7827	0.8135
	P-100	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	***<0.0001***	**<0.0001**	**0.0136**	0.579

* Shaded cells = biologically and statistically significant difference
* Italicized cells = biologically significant difference
* Bolded cells = statistically significant difference

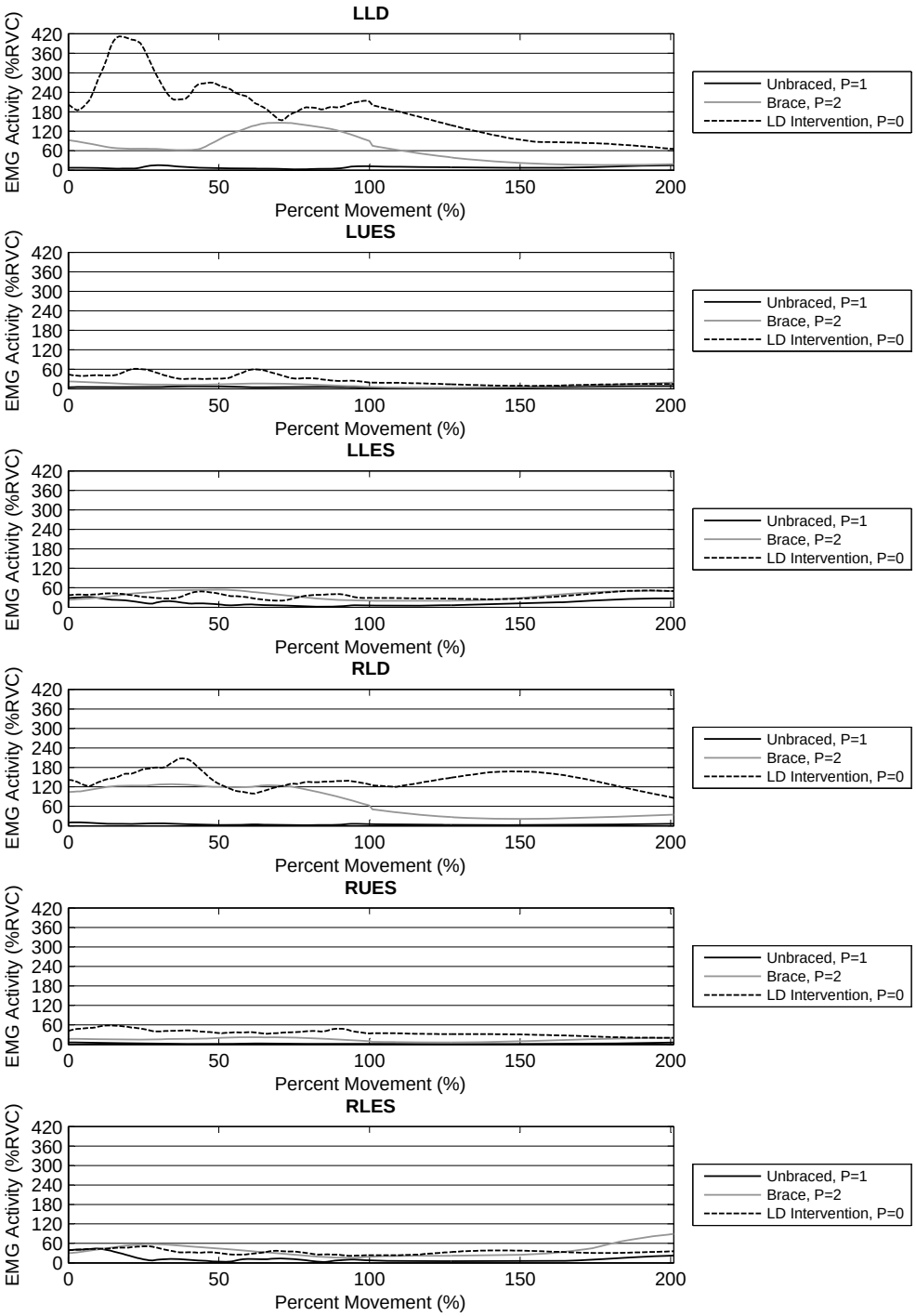
Appendix E
Time histories of Case Studies

Each figure contains the time history of a trial prior to an intervention and after using the intervention. In all situations, the solid black line represents the unbraced condition. The interventions used are specified in the legend. The x-axis displays the percentage of movement, with endpoints as described in table 2 in section 3.6. The subject and task being examined is the large title at the top of each page, along with the general variables included on the page (i.e. EMG, Eigenvalues, etc.). The specific variable being examined (i.e. RRA, RIO, L4F, etc.) is the title of each graph. These figures are supplemental to those in section 4.2 (case studies results), which display the percent change in each of the biomechanical variables.

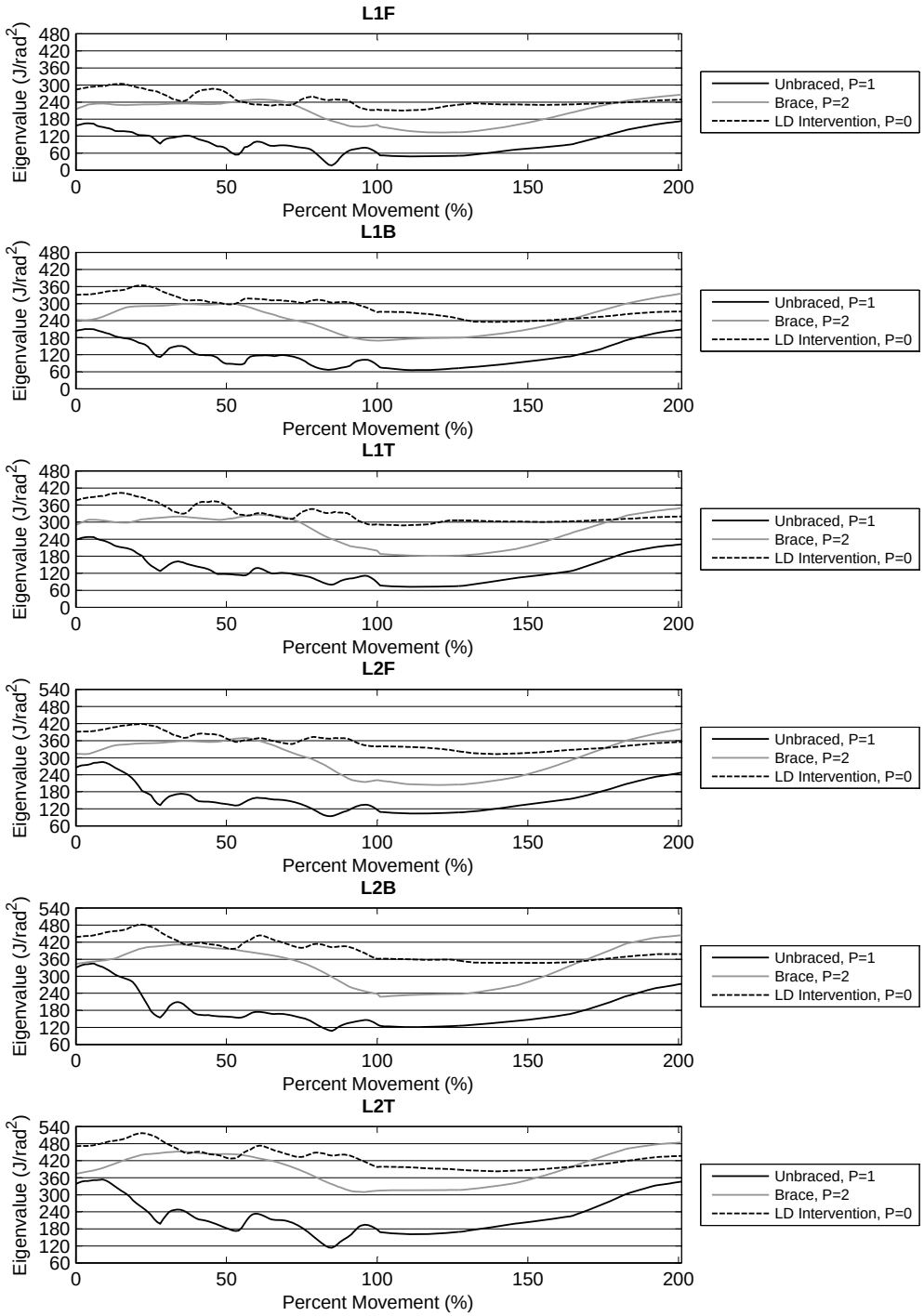
S01


S01 Heel Drop Abdominal EMG
LRA
LEO
LIO
RRA
REO
RIO
EMG Activity (%RVC)
Percent Movement (%)
0
25
50
75
100
125
150
175
0
50
100
150
200
Unbraced, P=1
Brace, P=2
LD Intervention, P=0

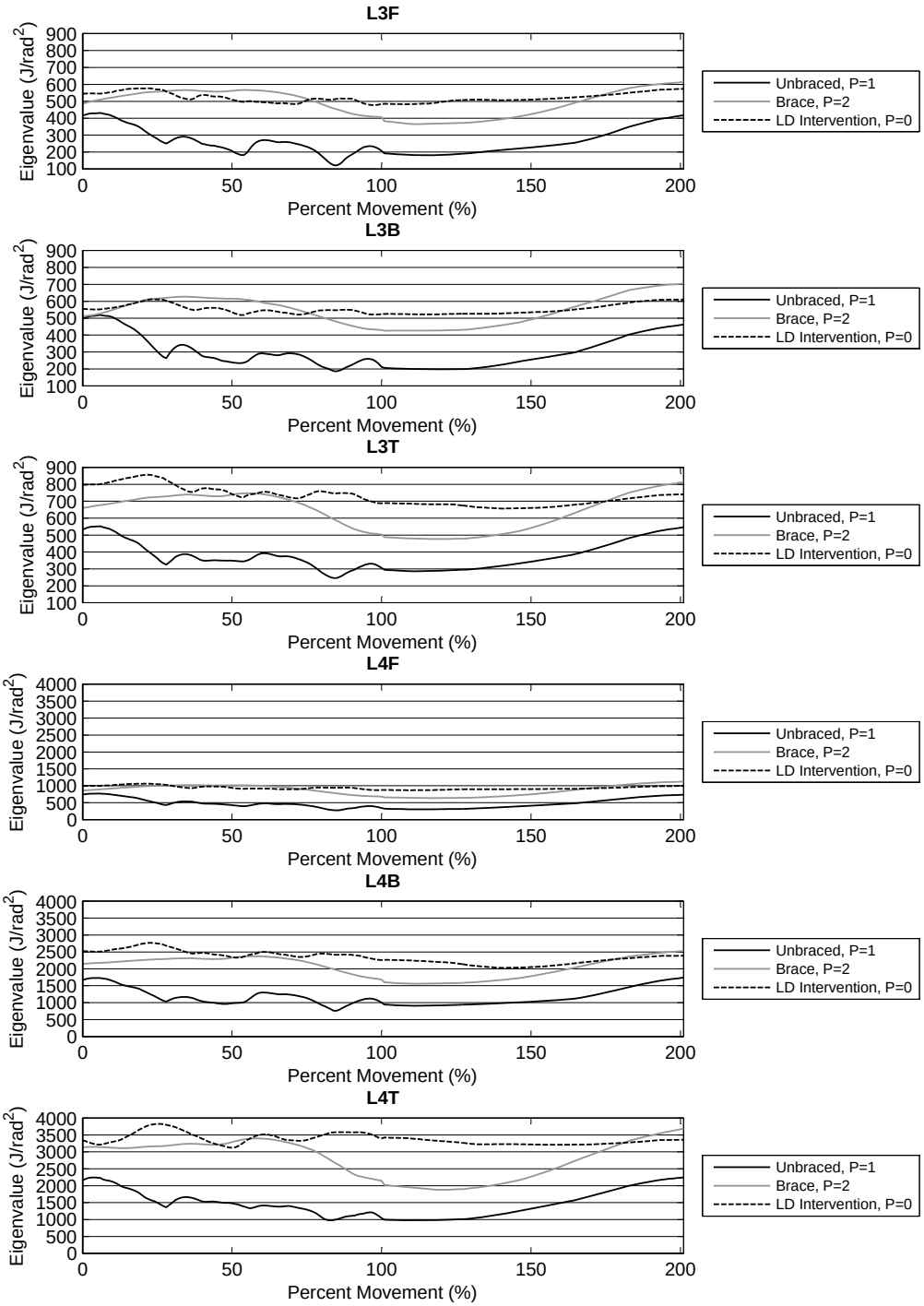
S01 Heel Drop Back EMG



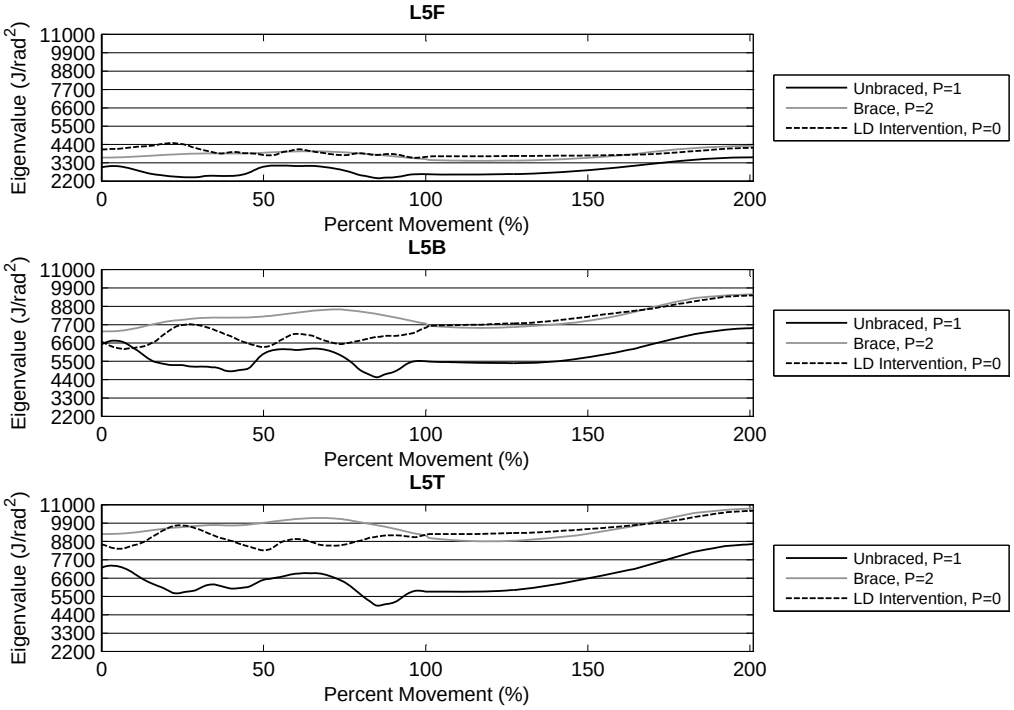
S01 Heel Drop Eigenvalues



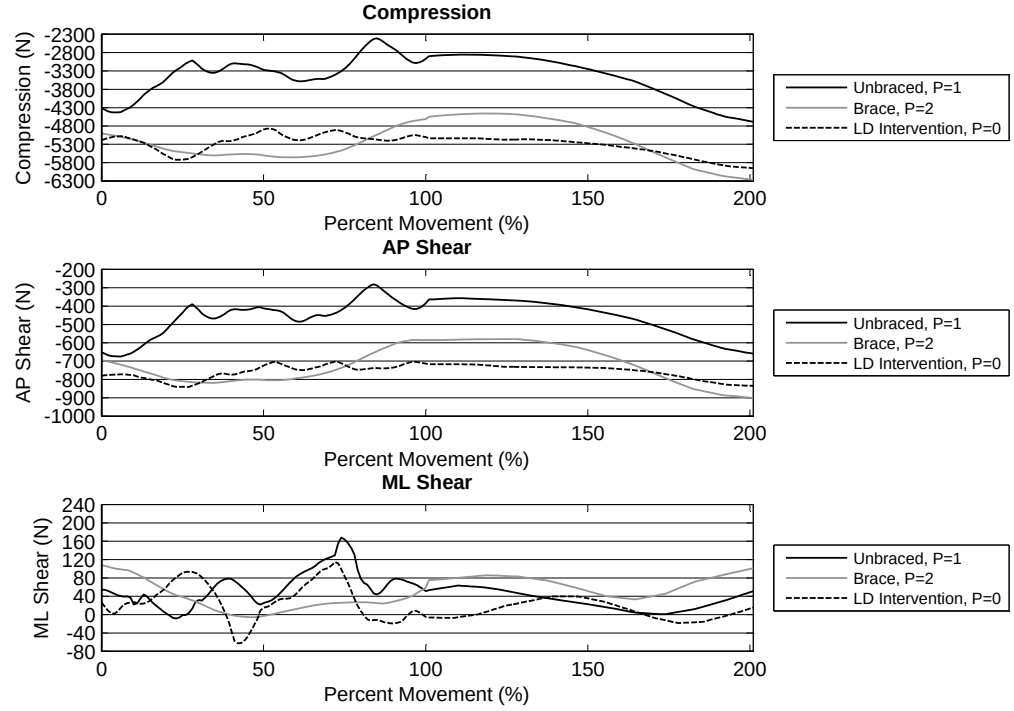
S01 Heel Drop Eigenvalues



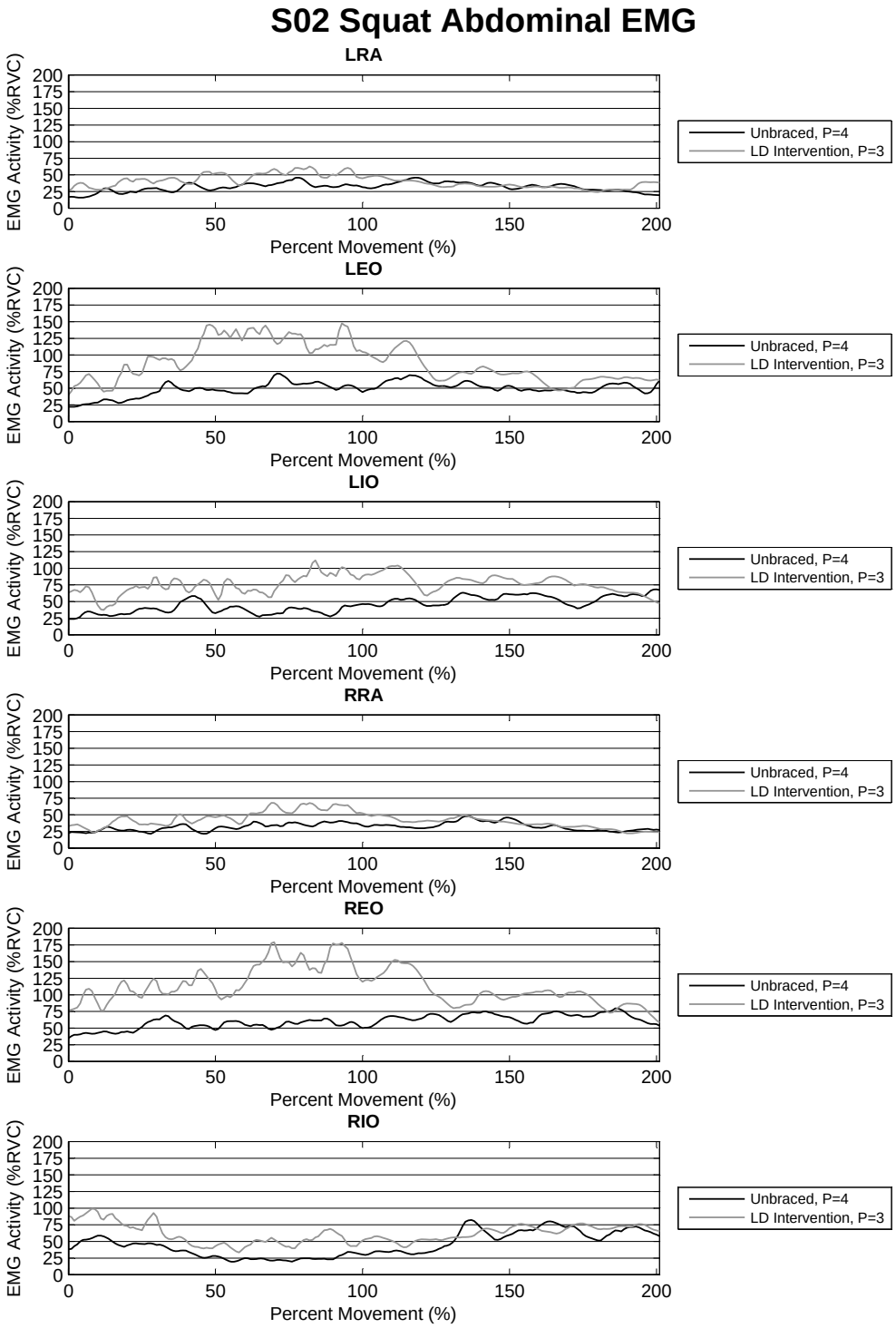
S01 Heel Drop Eigenvalues

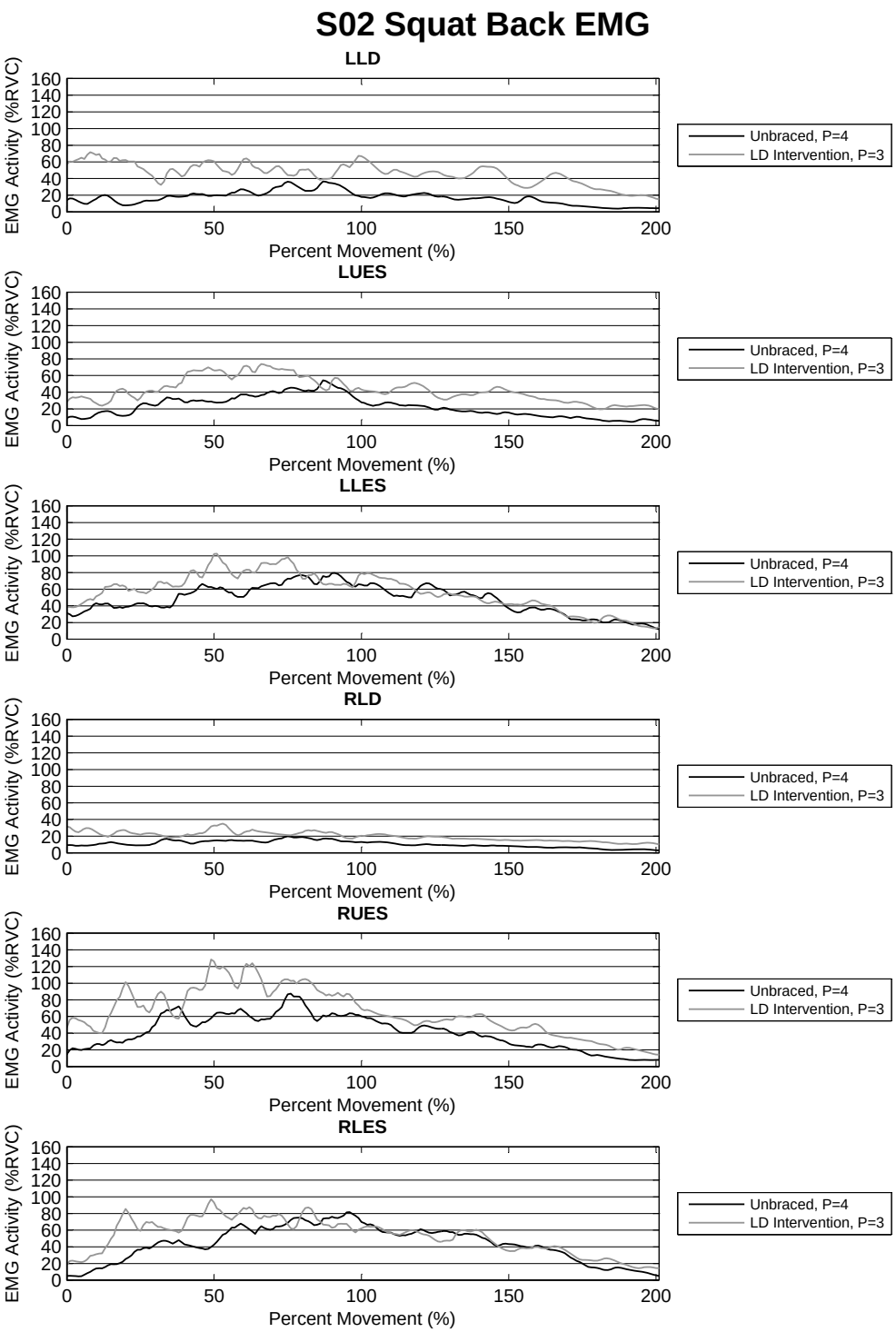


S01 Heel Drop Compression & Shear

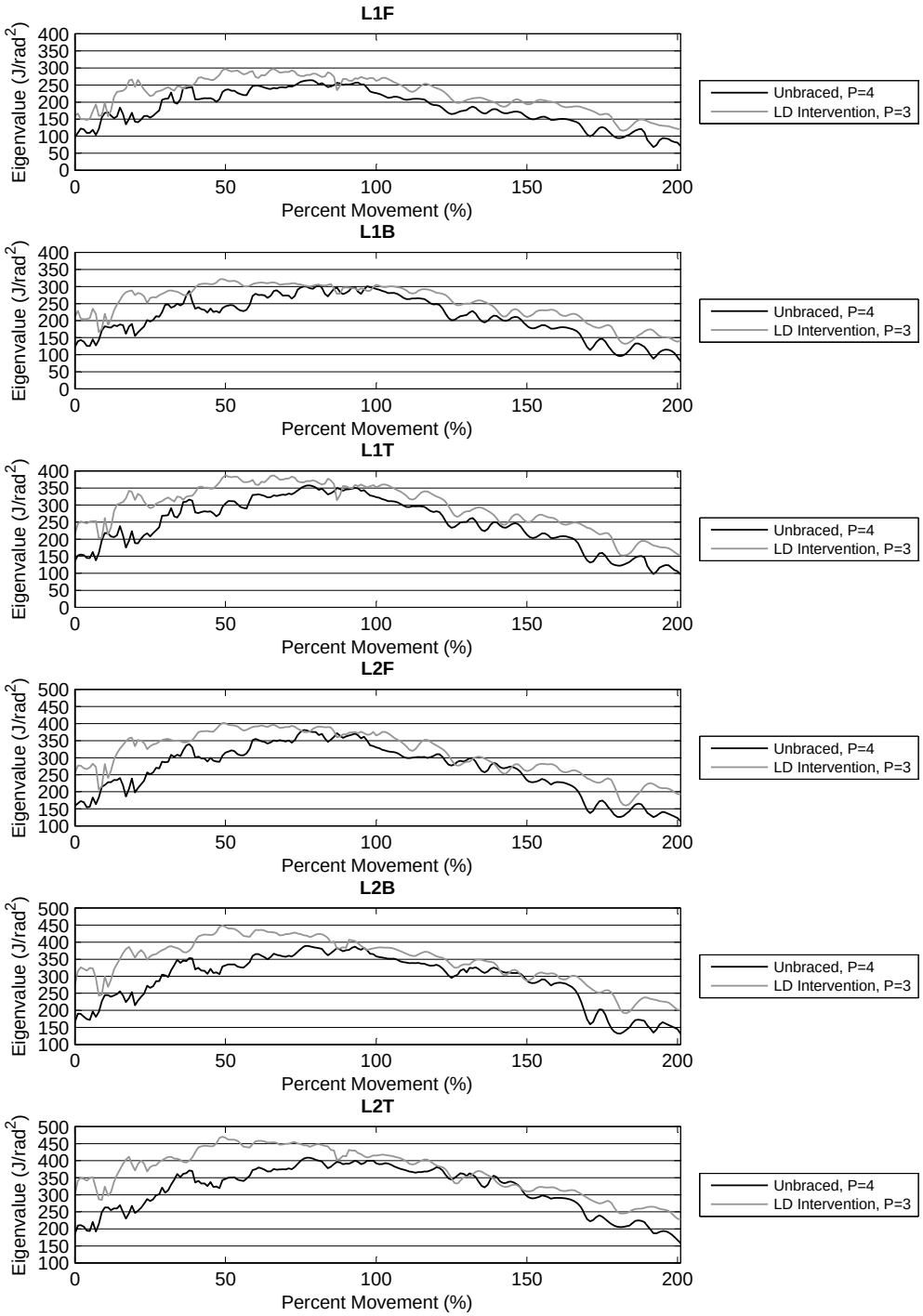


S02

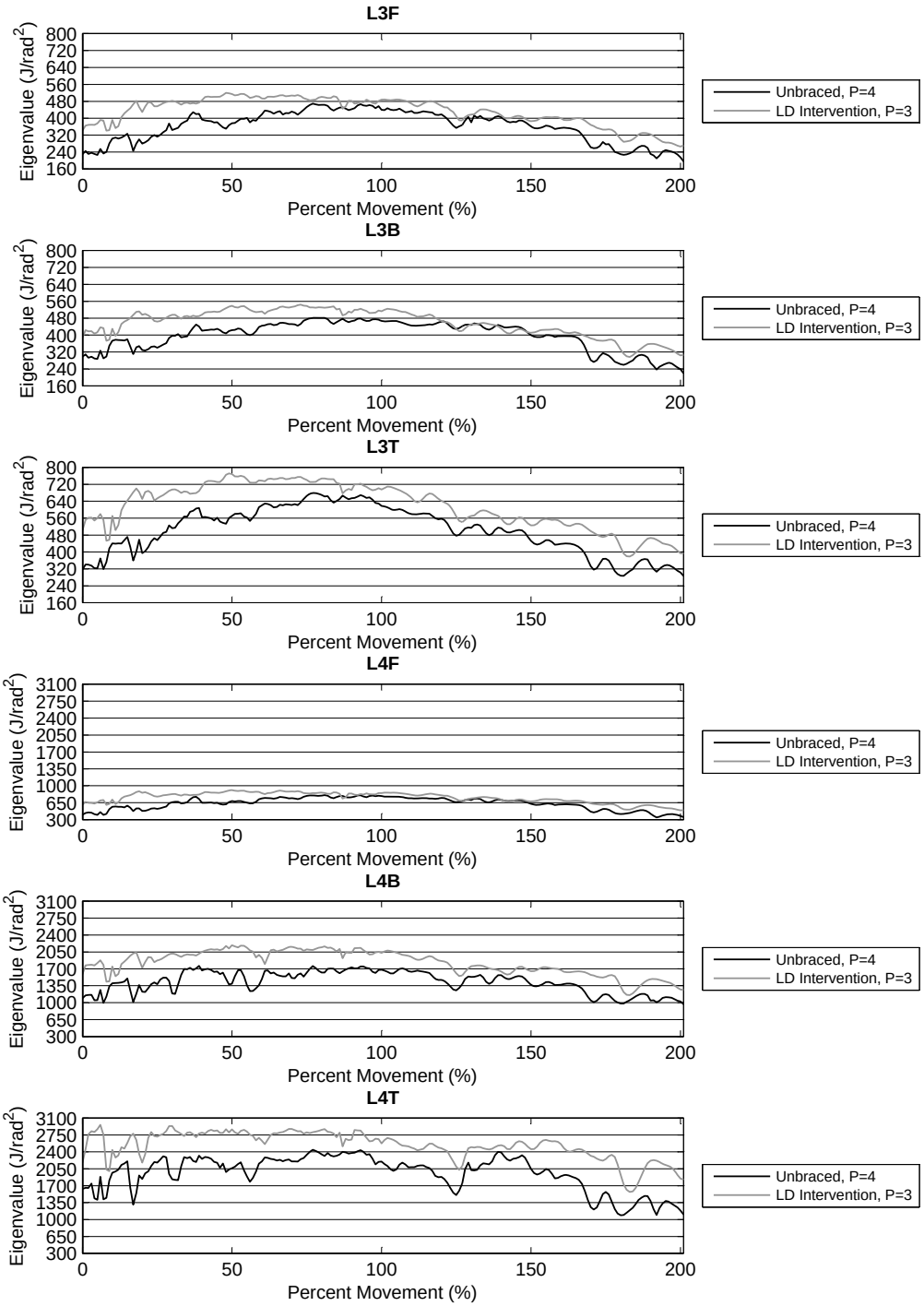

S02 Squat Abdominal EMG
LRA
LEO
LIO
RRA
REO
RIO
EMG Activity (%RVC)
Percent Movement (%)
0
25
50
75
100
125
150
175
200
Unbraced, P=4
LD Intervention, P=3

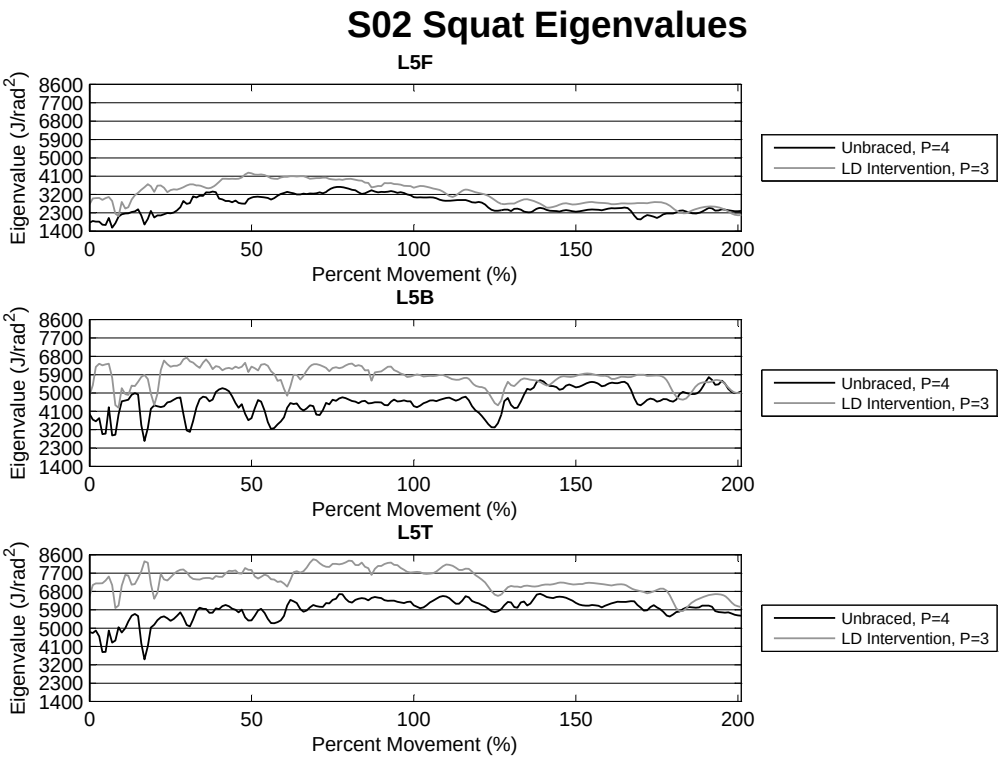

S02 Squat Back EMG
LLD
LUES
LLES
RLD
RUES
RLES
EMG Activity (%RVC)
Percent Movement (%)
0
20
40
60
80
100
120
140
160
0
50
100
150
200
Unbraced, P=4
LD Intervention, P=3

S02 Squat Eigenvalues

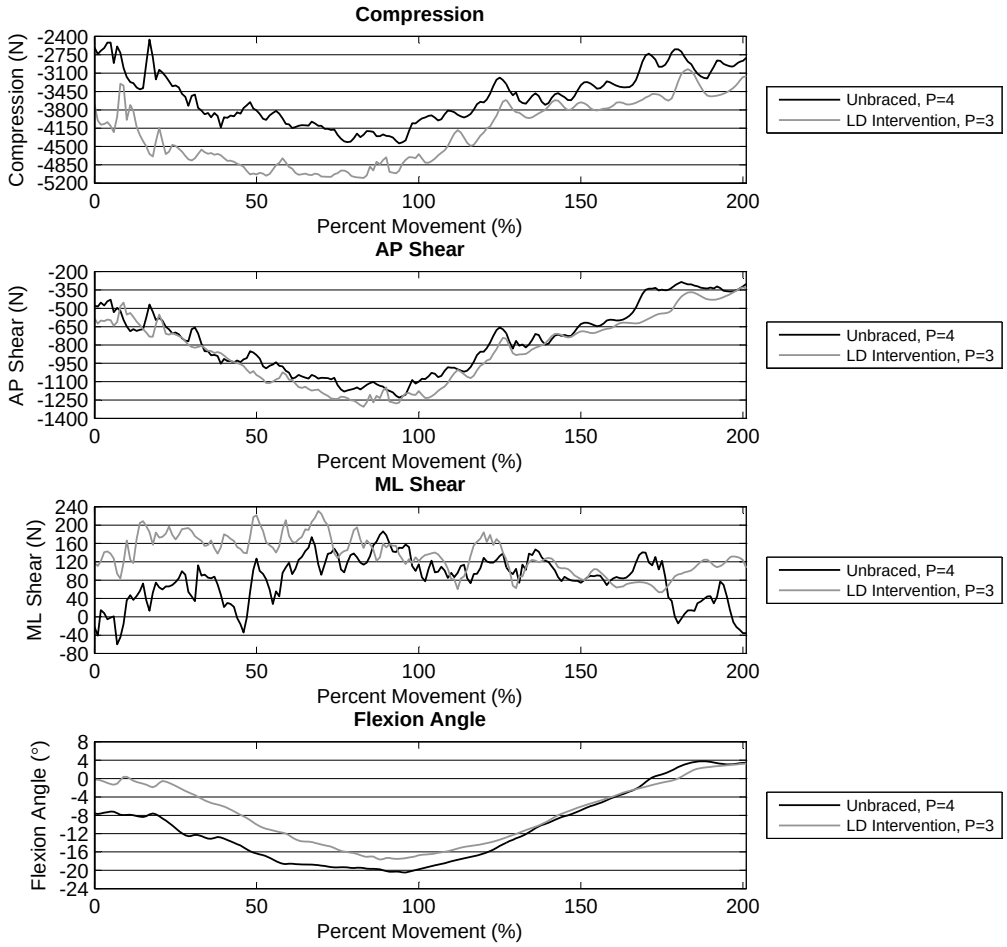


S02 Squat Eigenvalues

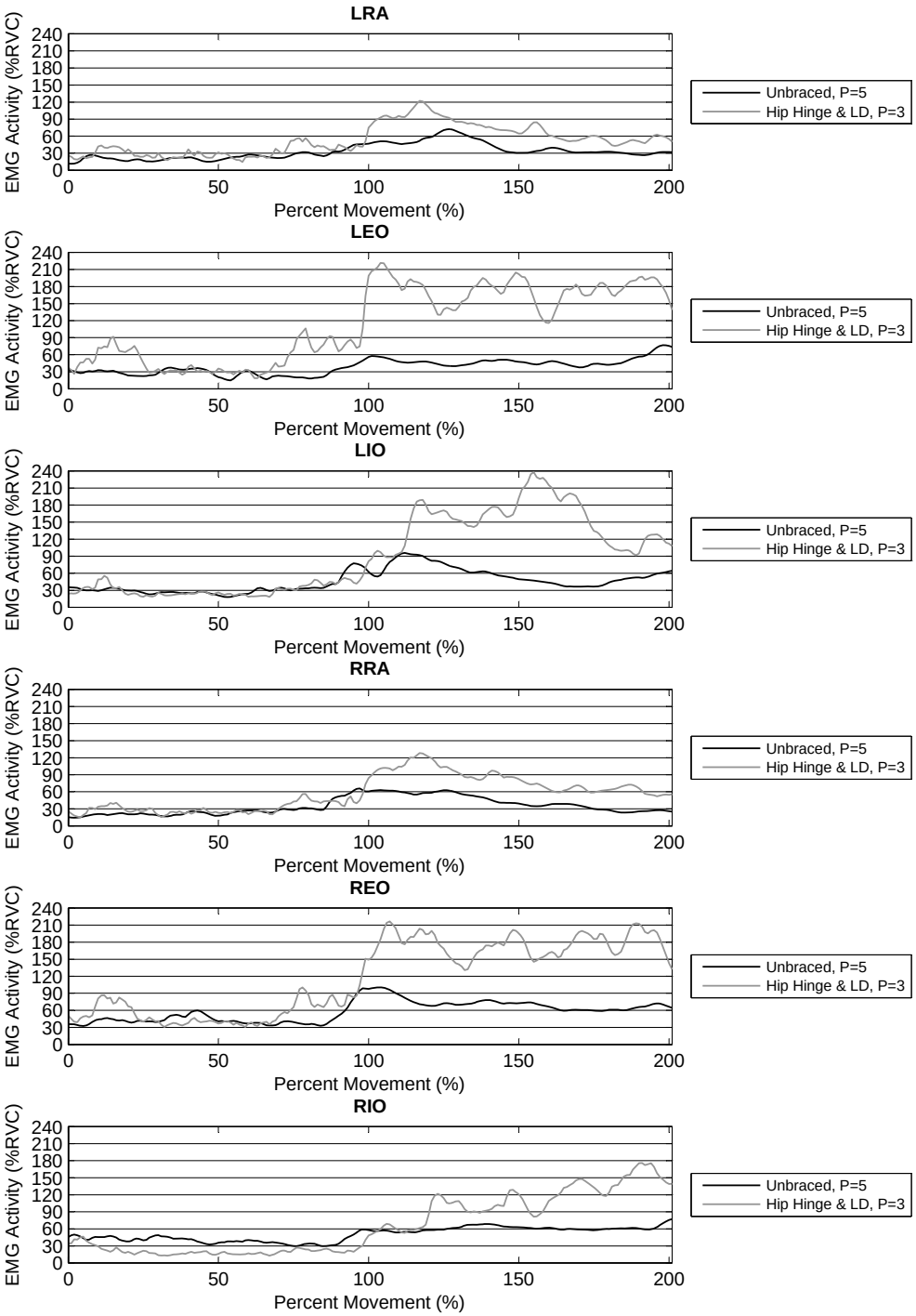


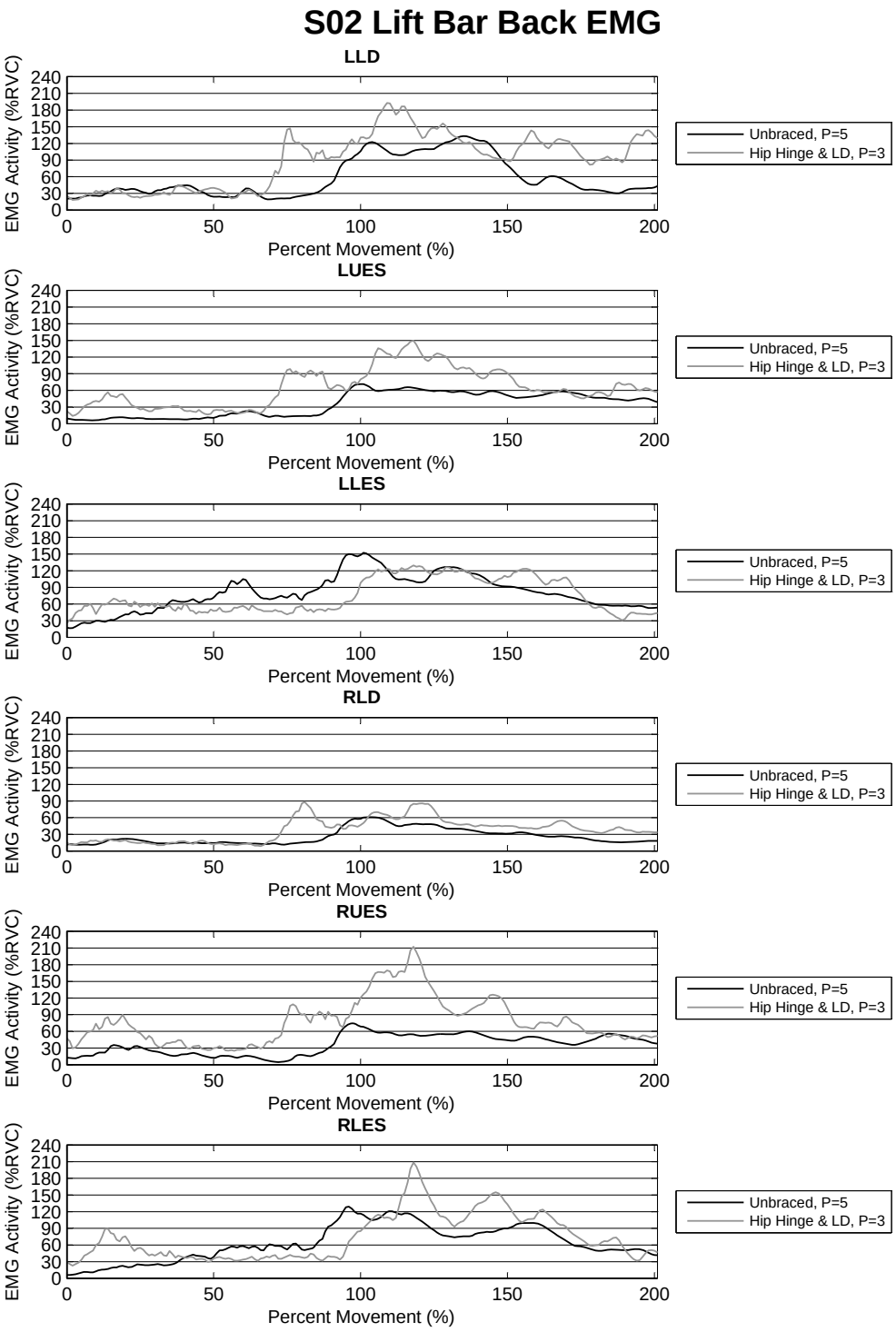

S02 Squat Eigenvalues
L5F
L5B
L5T
Eigenvalue (J/rad^2)
8600
7700
6800
5900
5000
4100
3200
2300
1400
0
50
100
150
200
Percent Movement (%)
Unbraced, P=4
LD Intervention, P=3

S02 Squat Compression, Shear & Flexion Angle

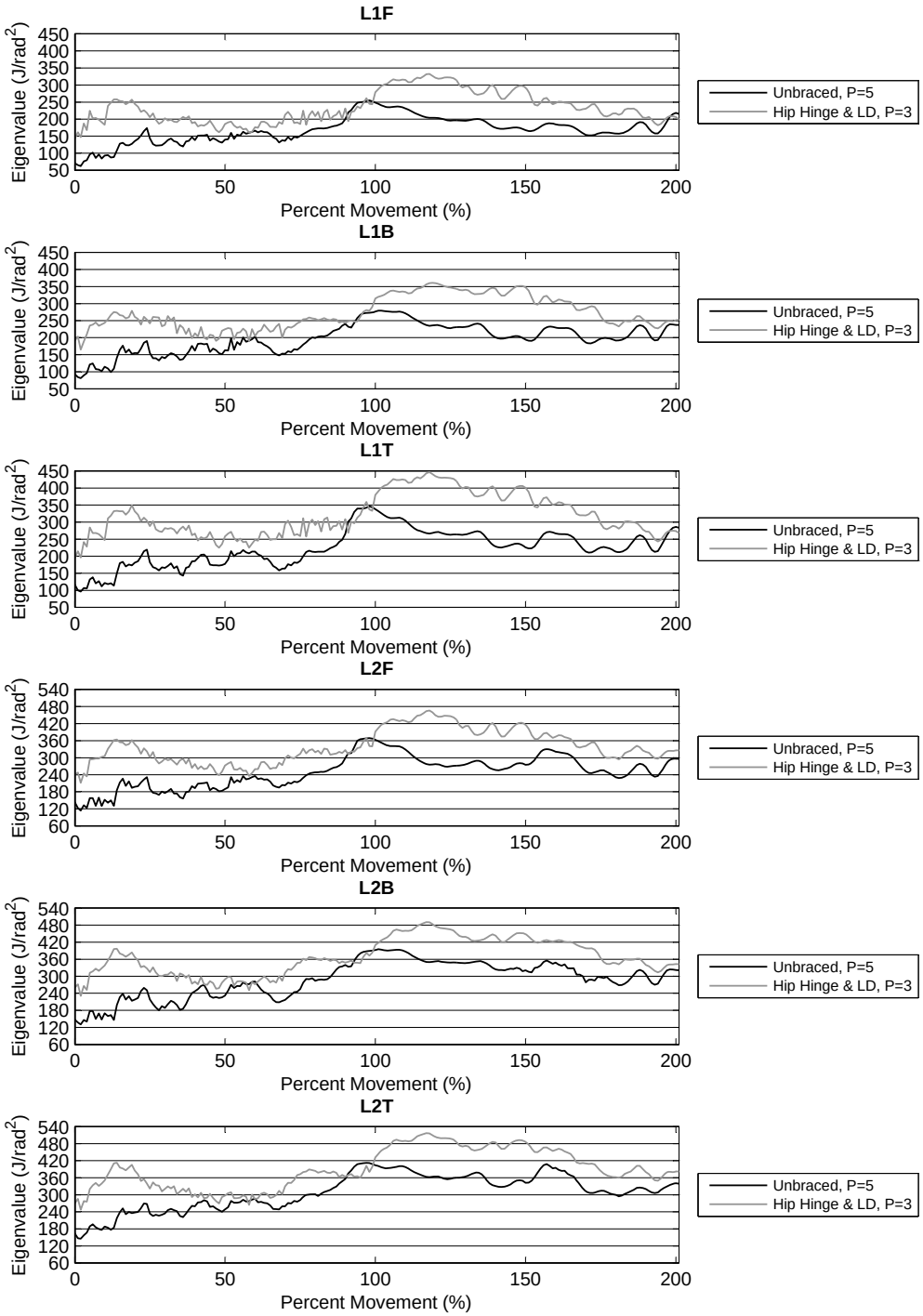


S02 Lift Bar Abdominal EMG

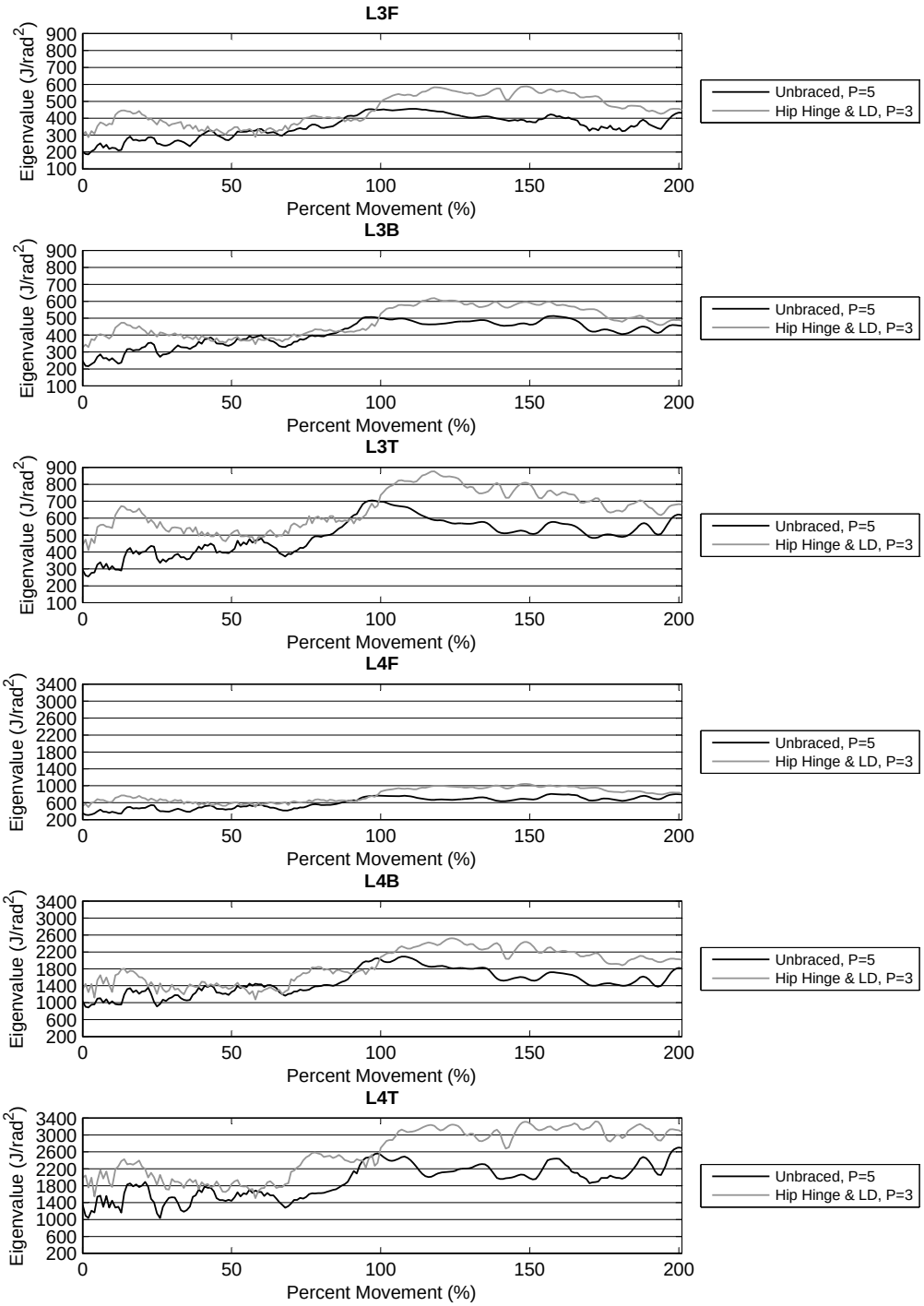



S02 Lift Bar Back EMG
LLD
LUES
LLES
RLD
RUES
RLES
EMG Activity (%RVC)
Percent Movement (%)
0
30
60
90
120
150
180
210
240
0
50
100
150
200
Unbraced, P=5
Hip Hinge & LD, P=3

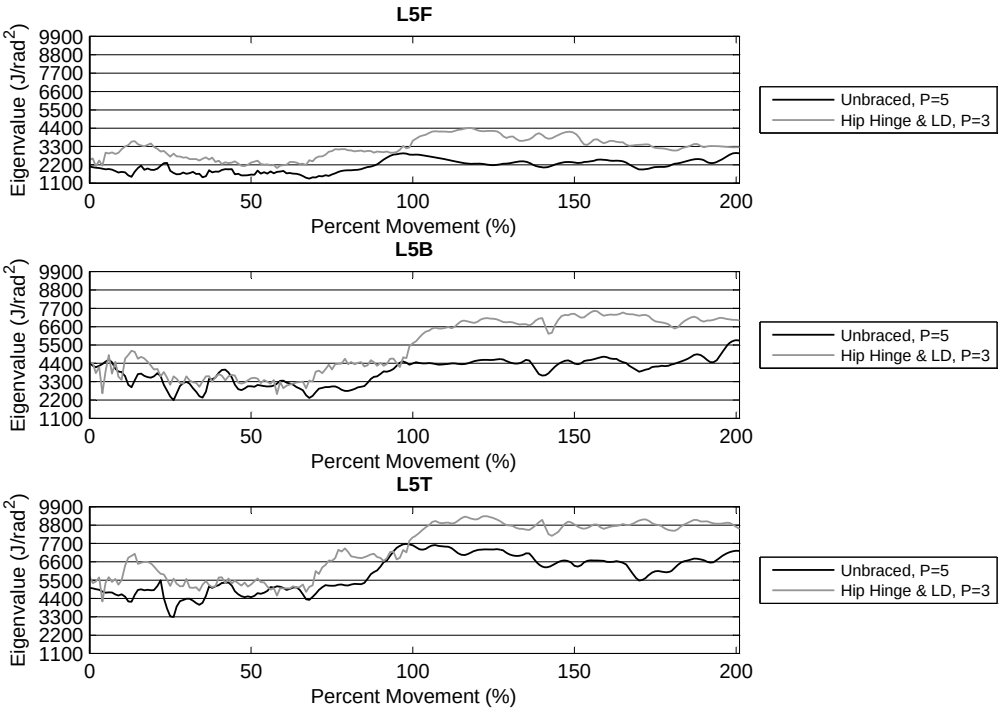
S02 Lift Bar Eigenvalues



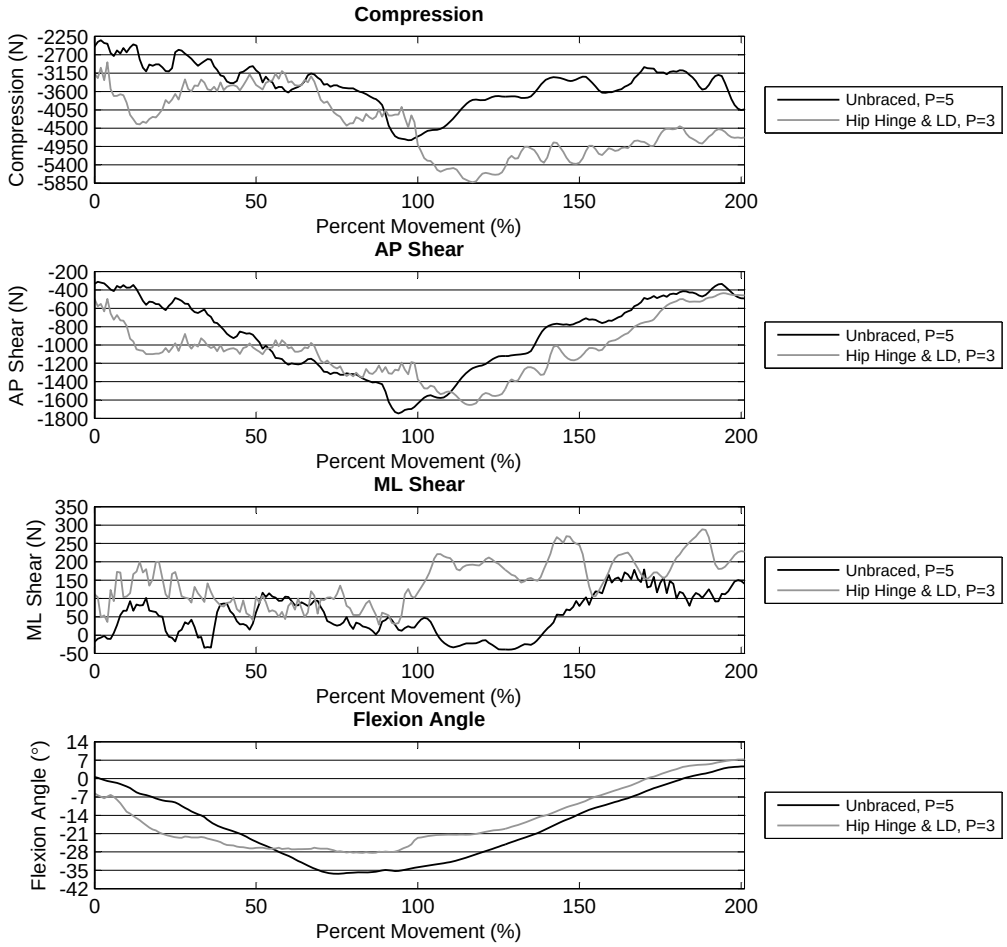
S02 Lift Bar Eigenvalues



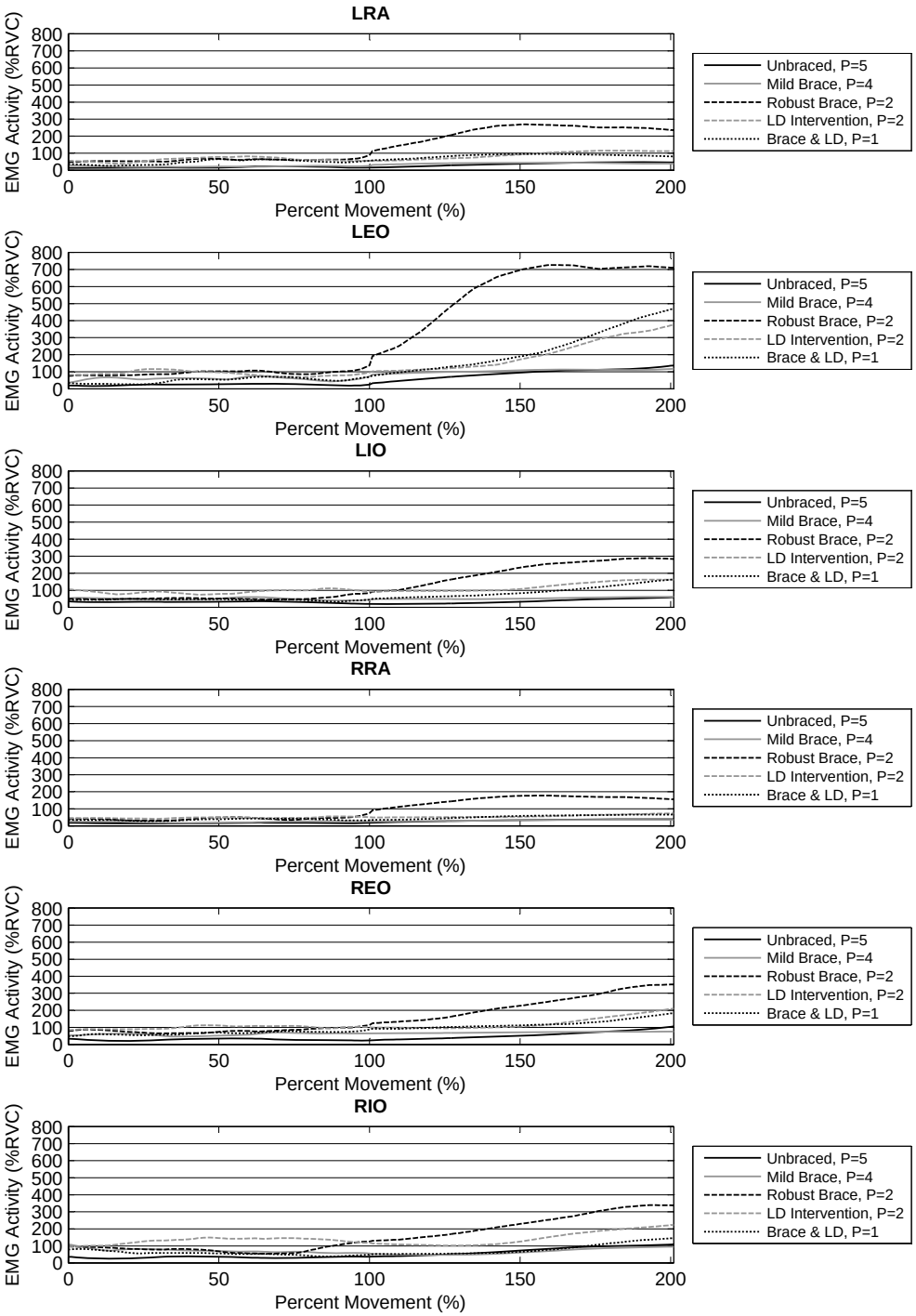
S02 Lift Bar Eigenvalues



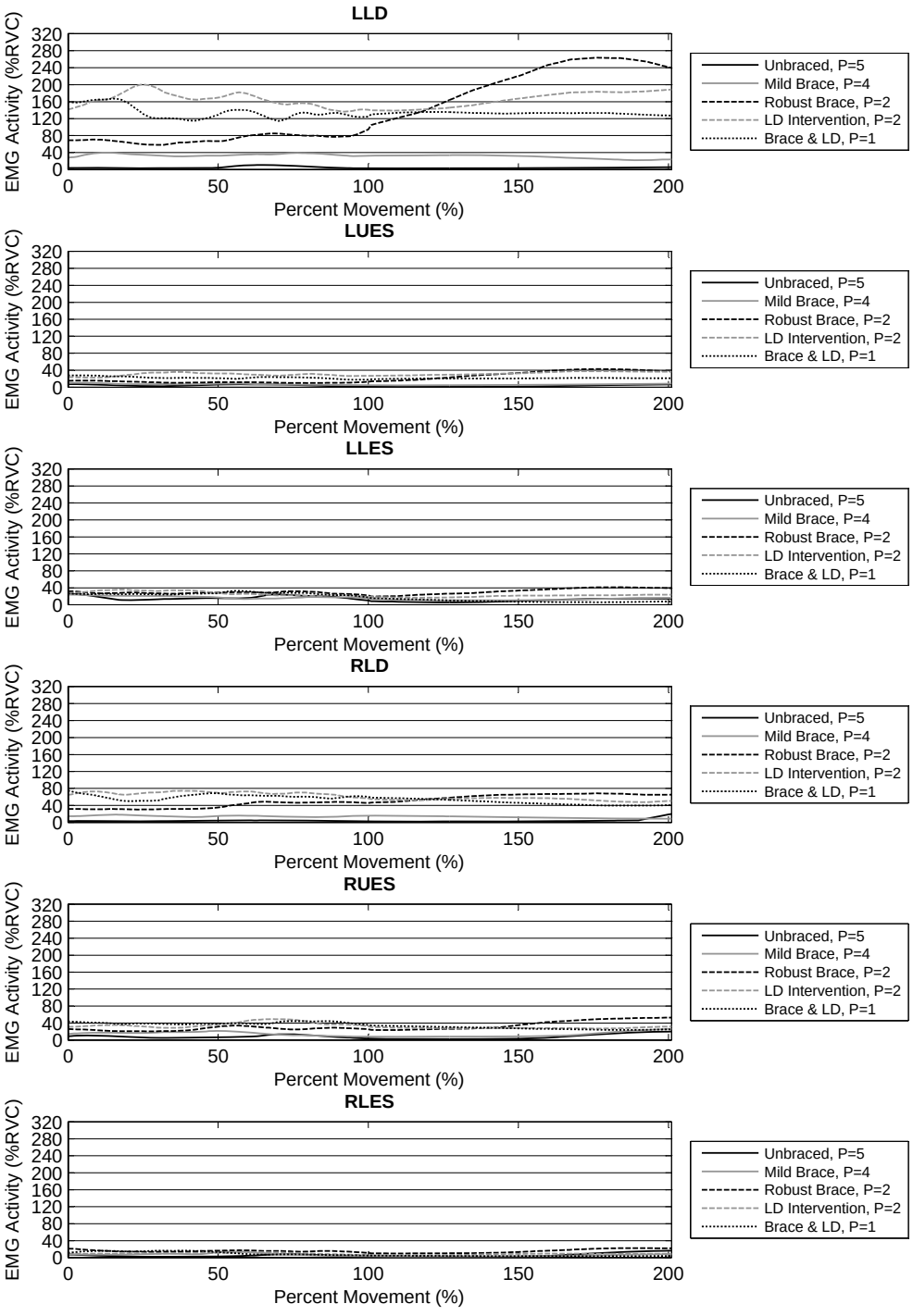
S02 Lift Bar Compression, Shear & Flexion Angle



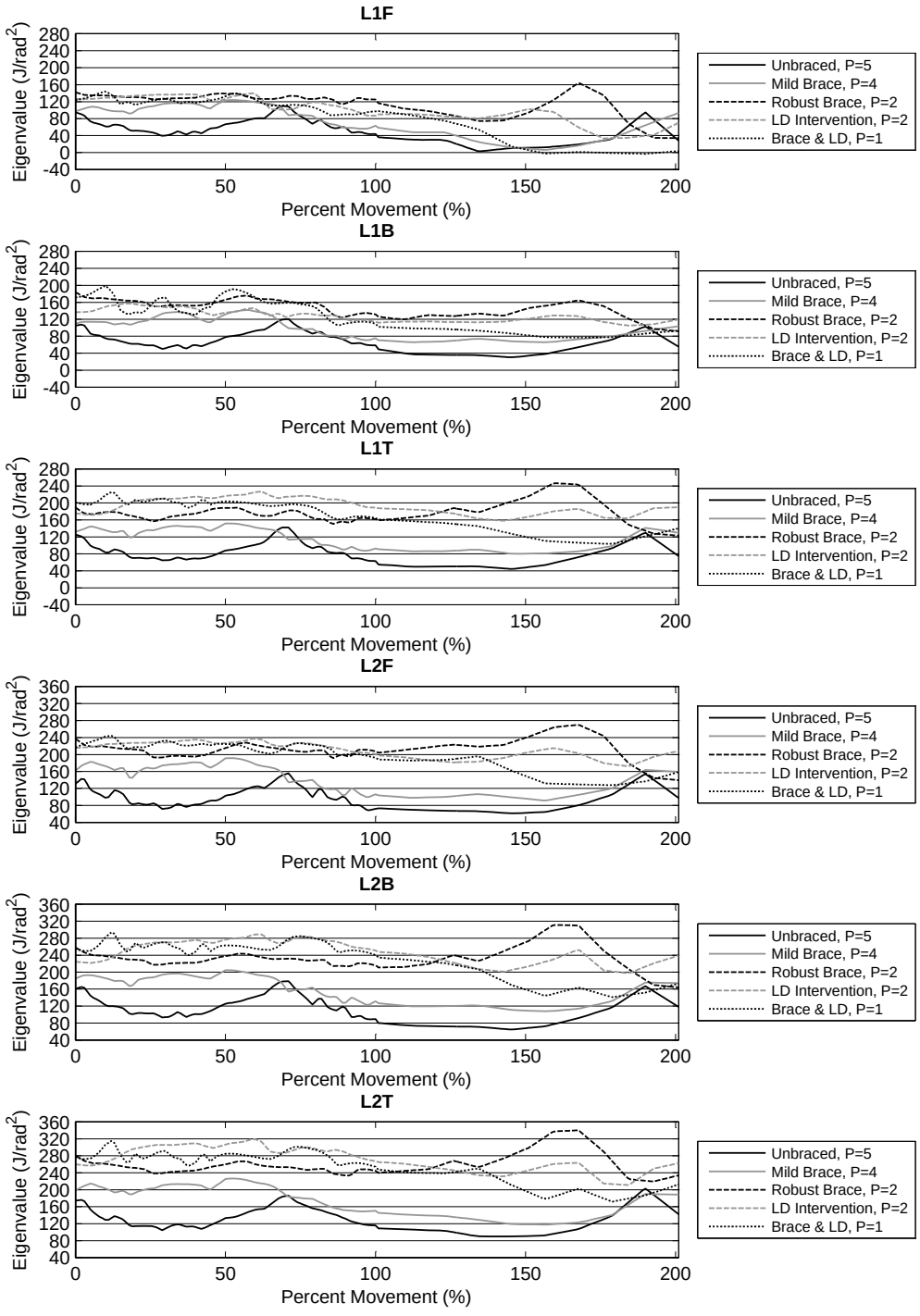
S02 Heel Drop Abdominal EMG



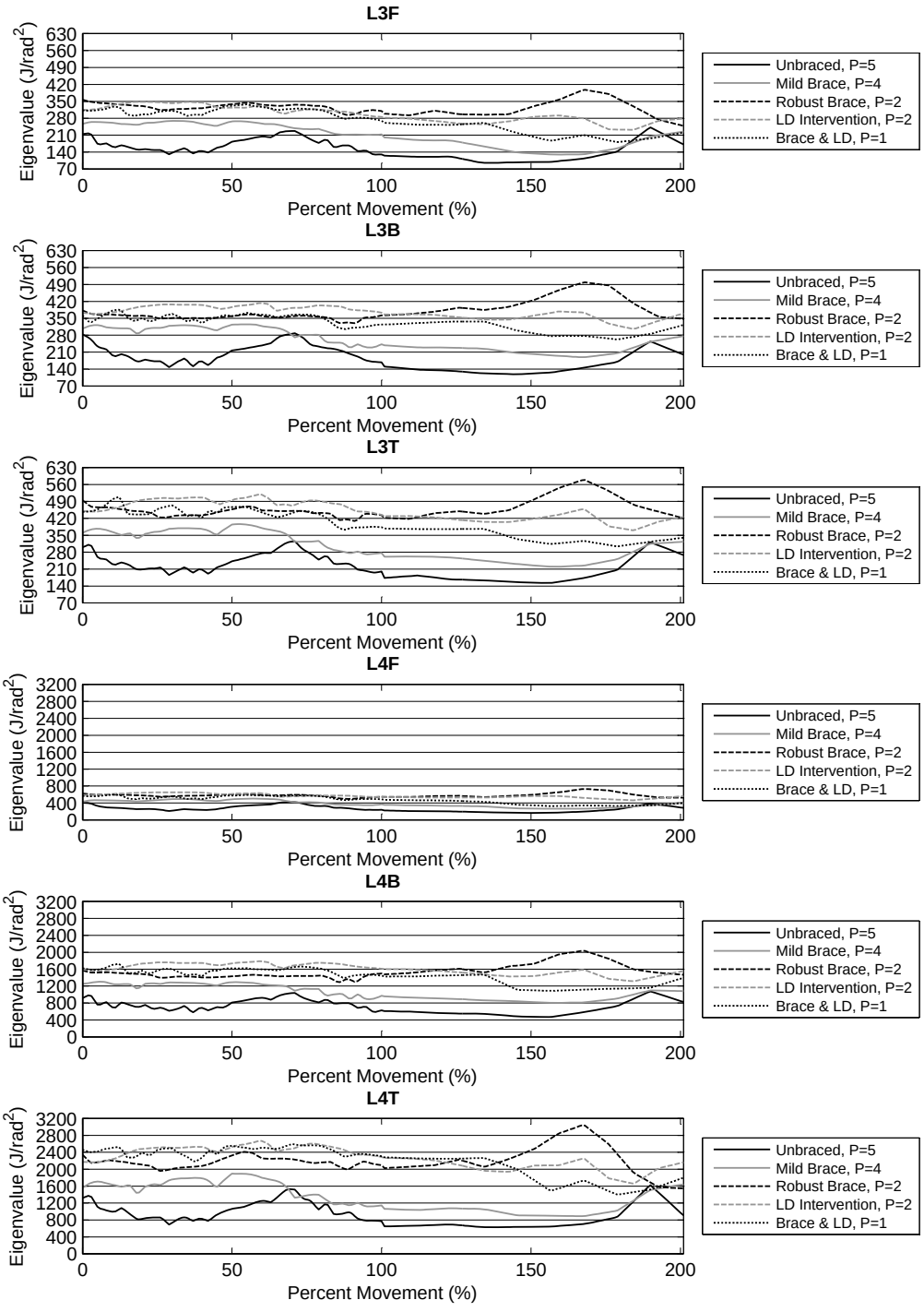
S02 Heel Drop Back EMG



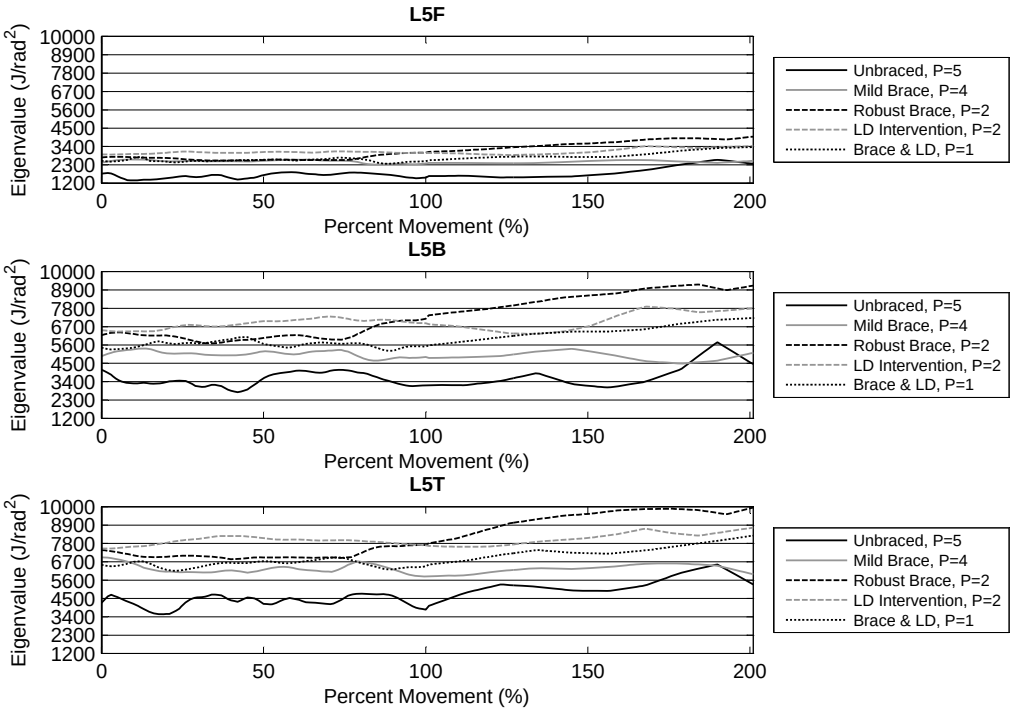
S02 Heel Drop Eigenvalues



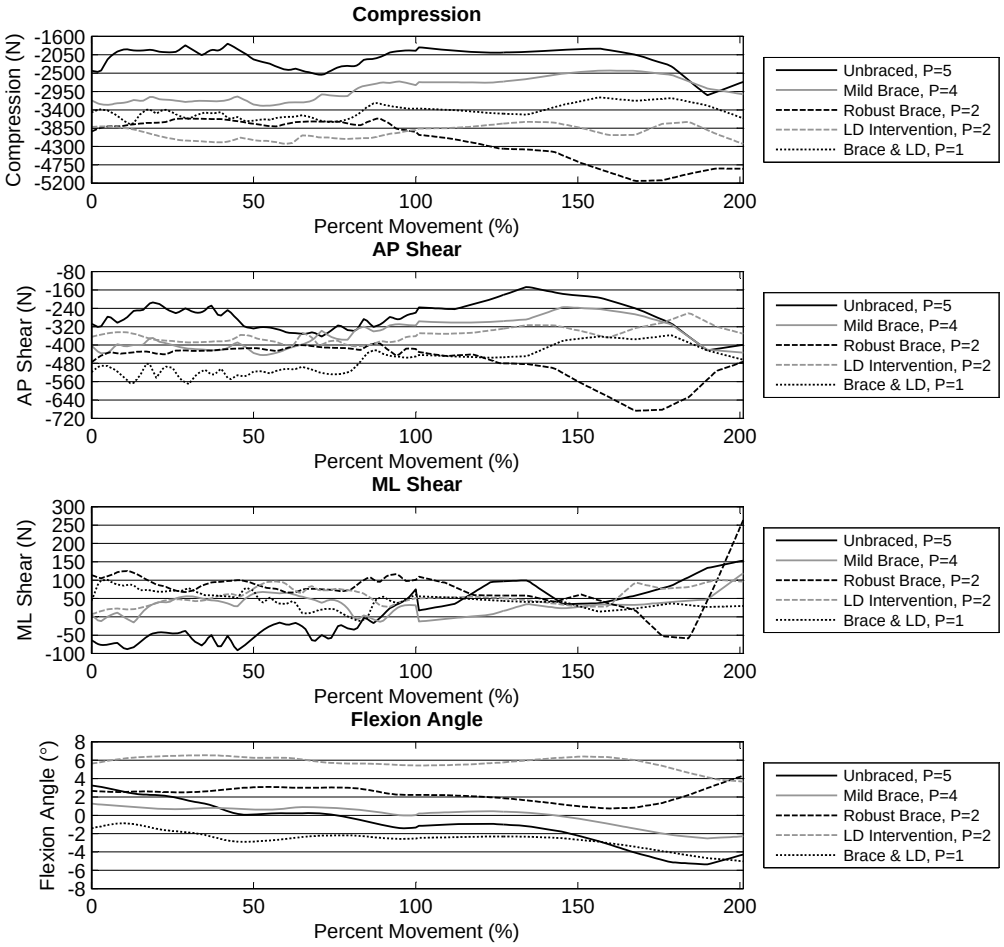
S02 Heel Drop Eigenvalues



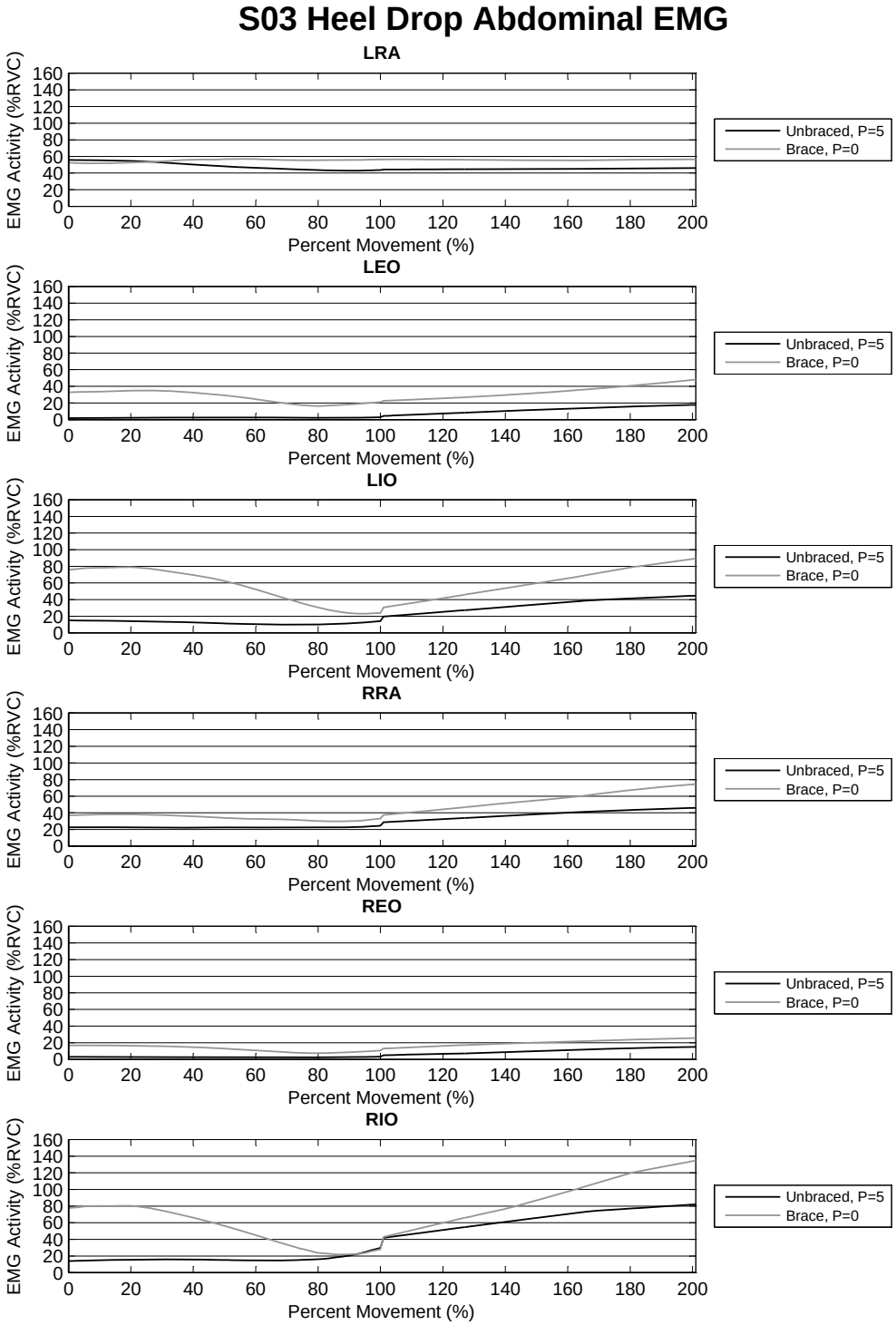
S02 Heel Drop Eigenvalues



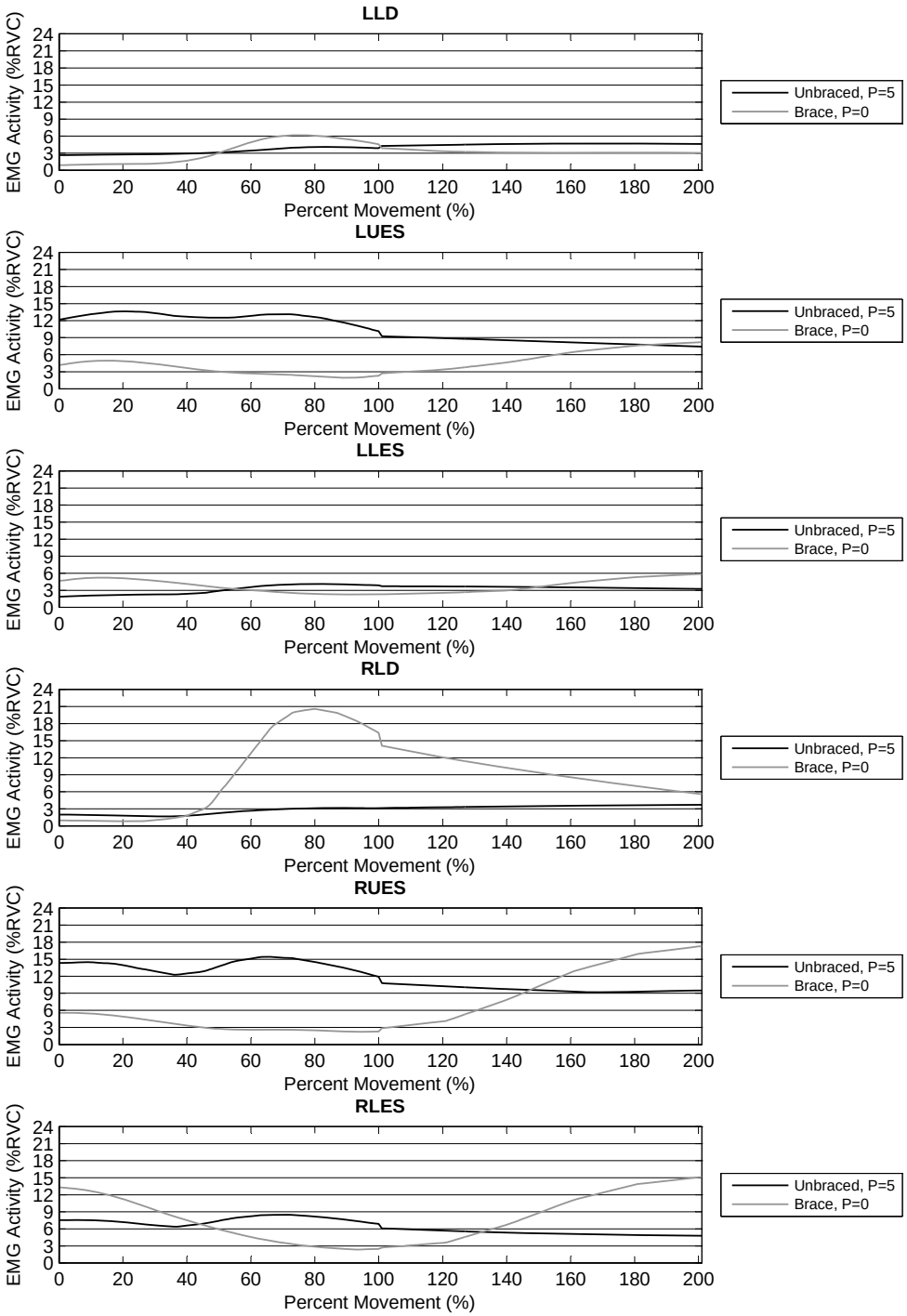
S02 Heel Drop Compression, Shear & Flexion Angle



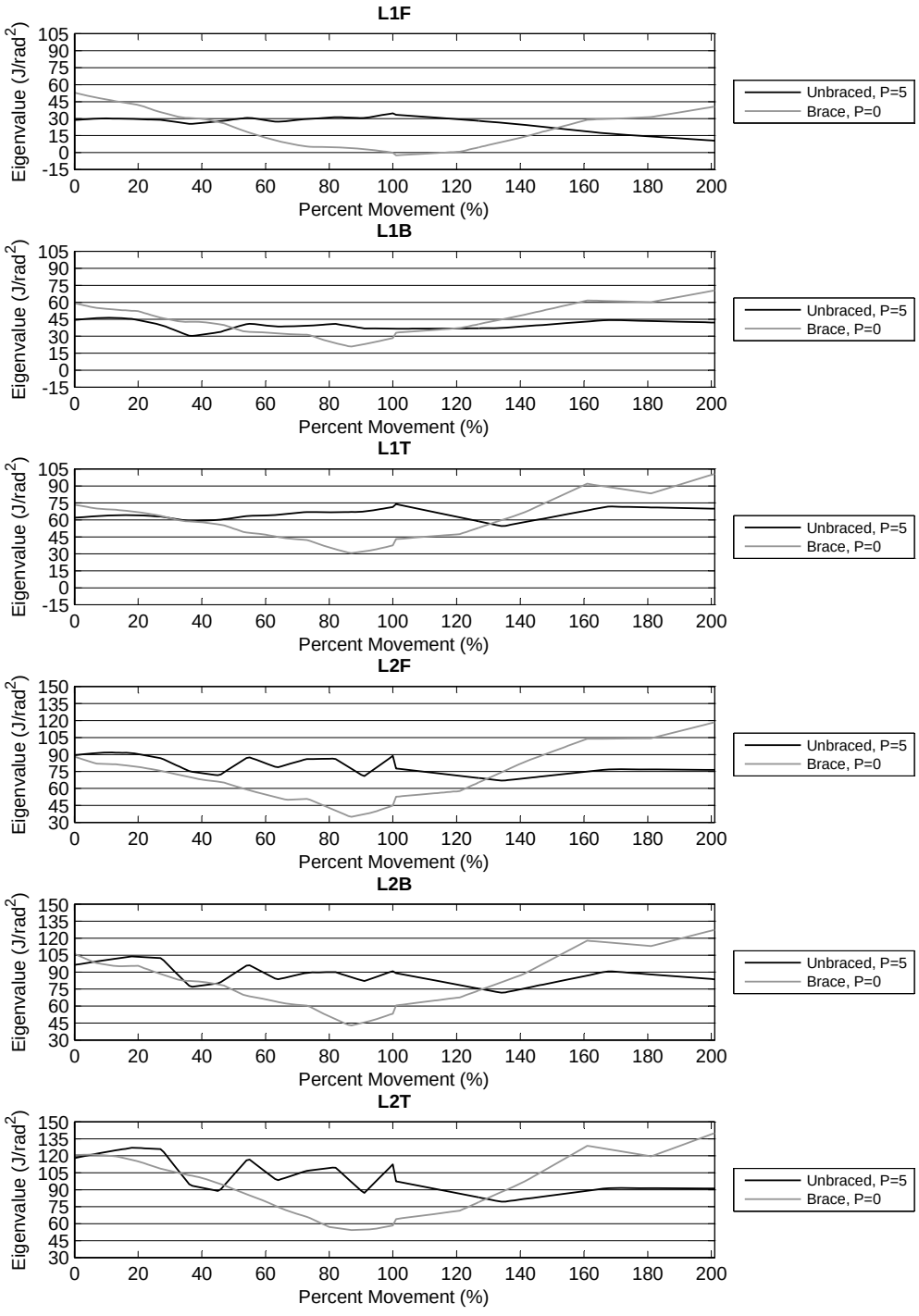
S03


S03 Heel Drop Abdominal EMG
LRA
LEO
LIO
RRA
REO
RIO
EMG Activity (%RVC)
Percent Movement (%)
Unbraced, P=5
Brace, P=0

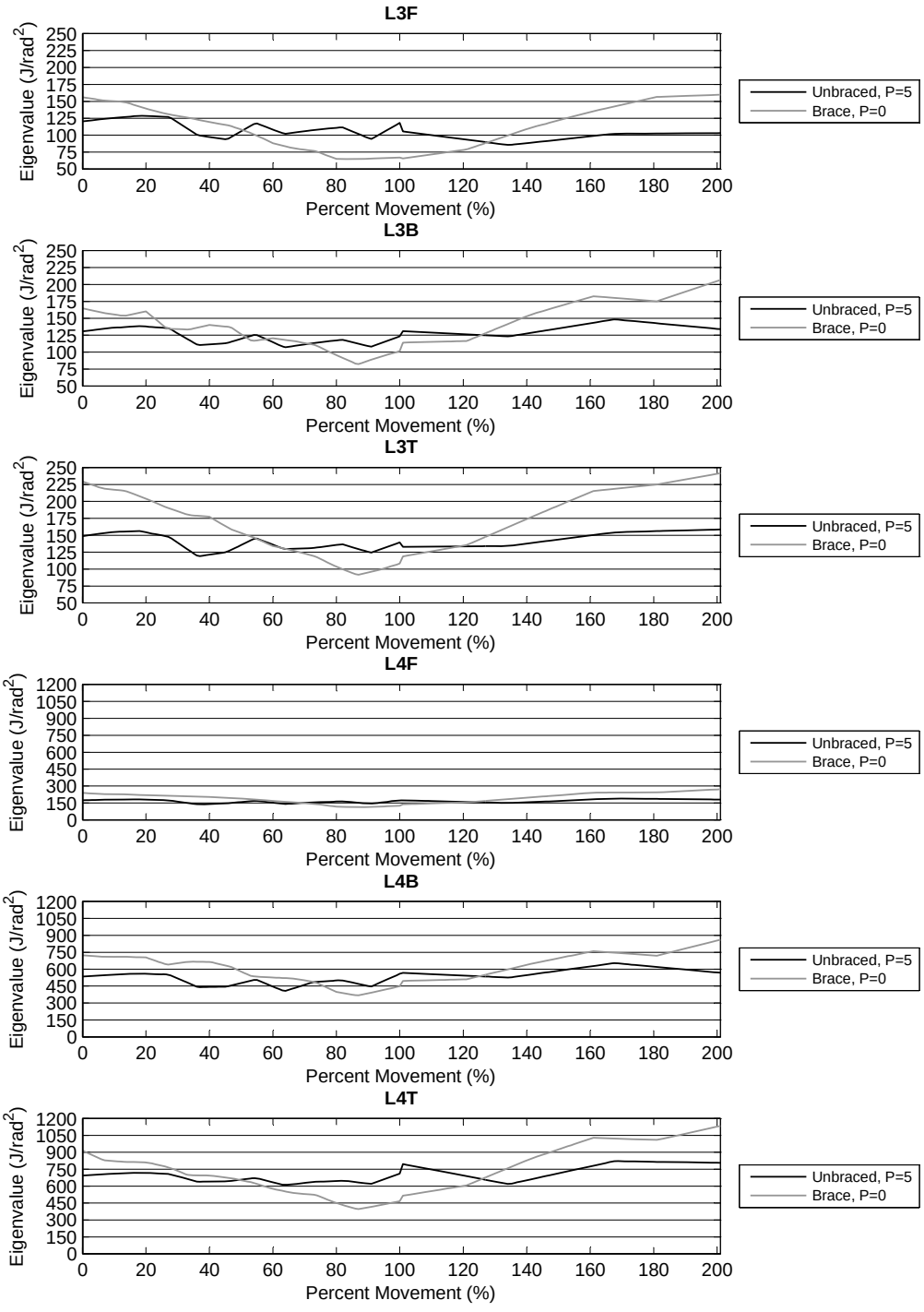
S03 Heel Drop Back EMG



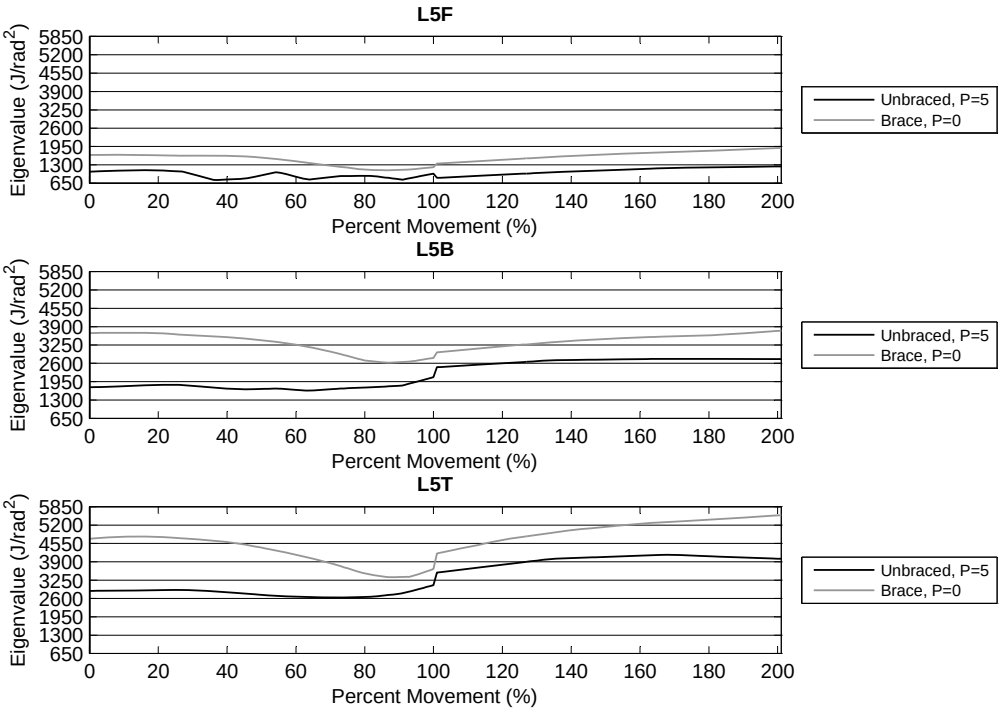
S03 Heel Drop Eigenvalues



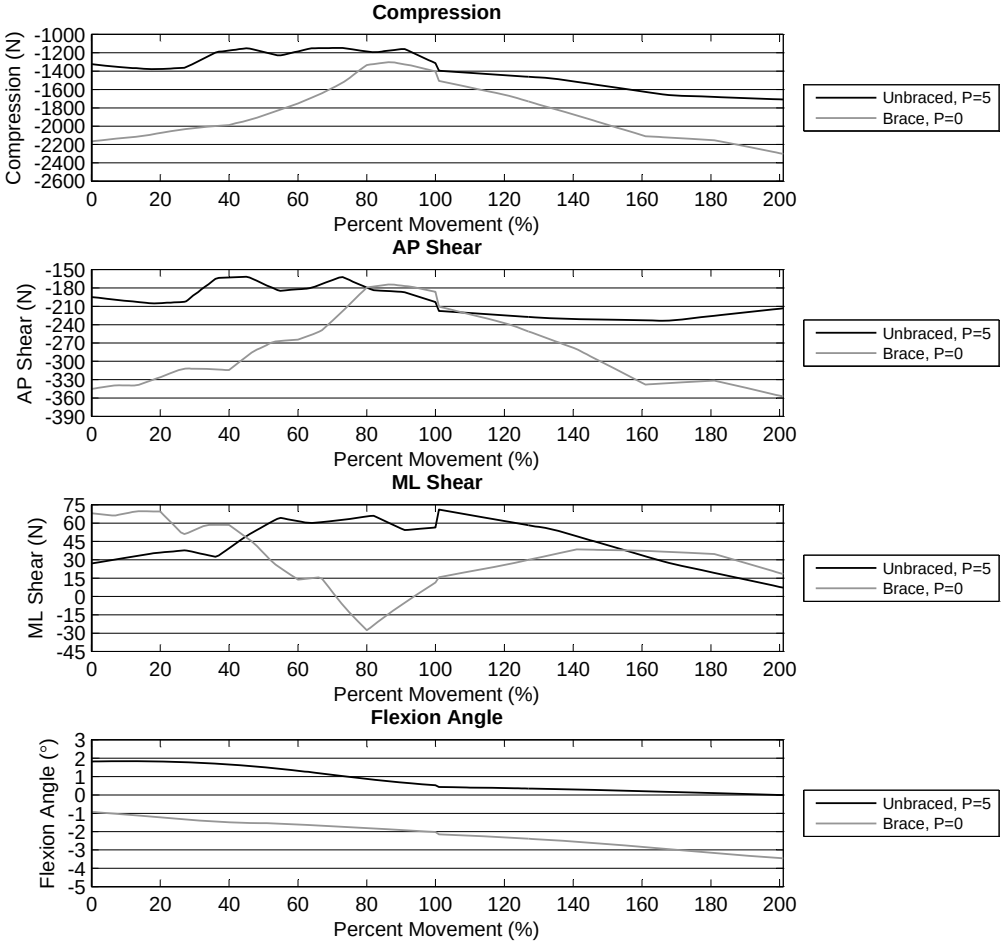
S03 Heel Drop Eigenvalues



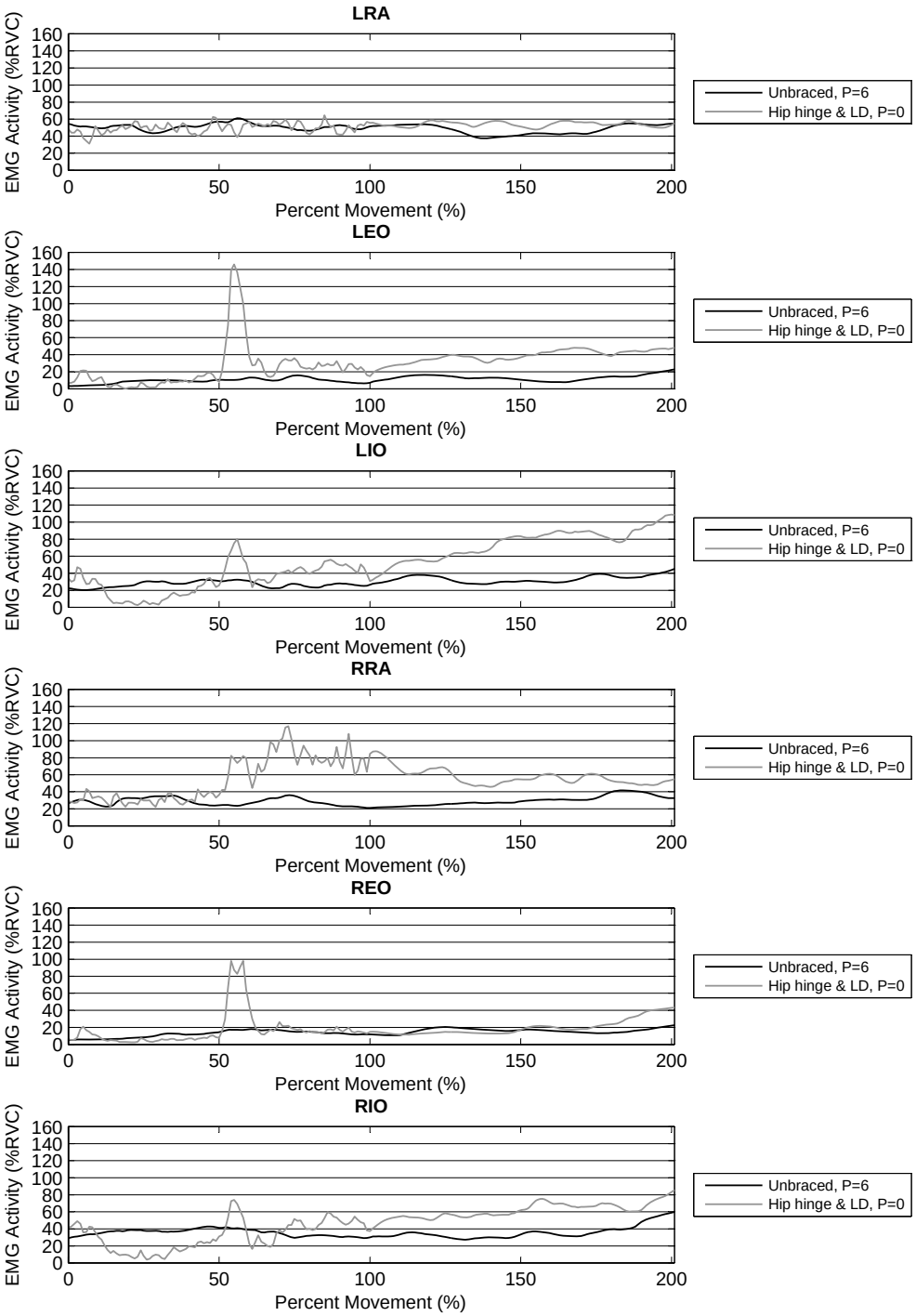
S03 Heel Drop Eigenvalues



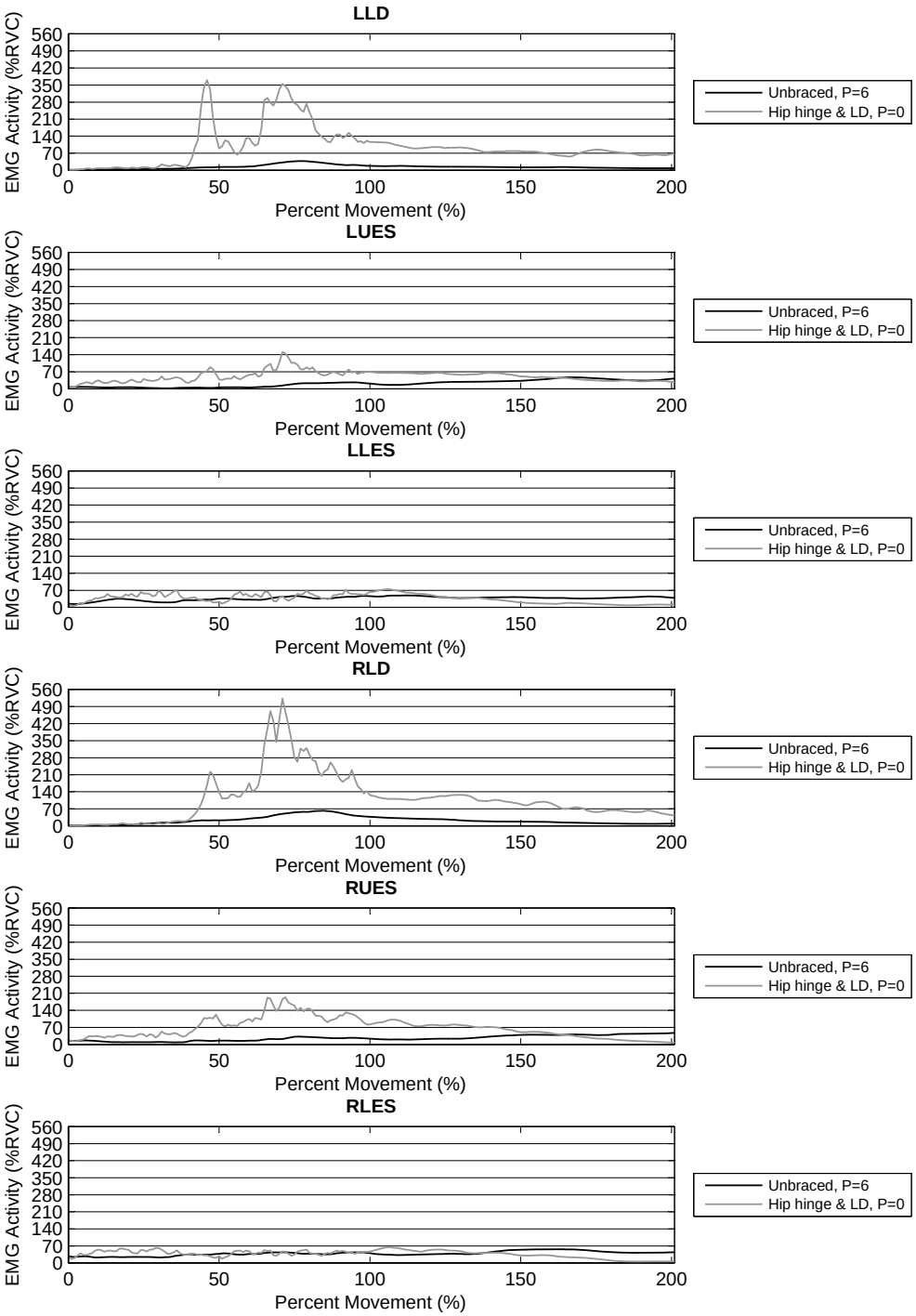
S03 Heel Drop Compression, Shear & Flexion Angle



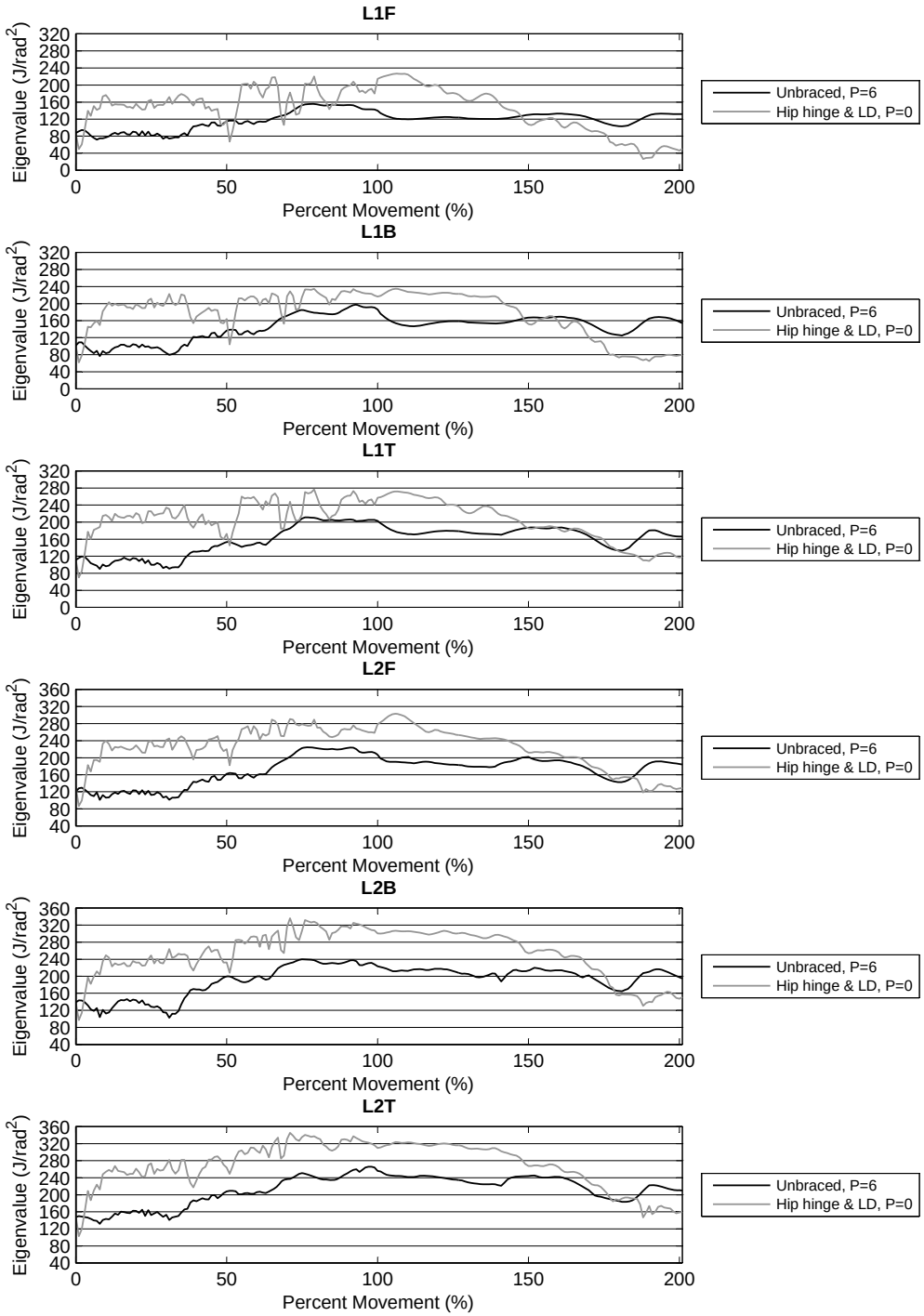
S03 Squat Abdominal EMG



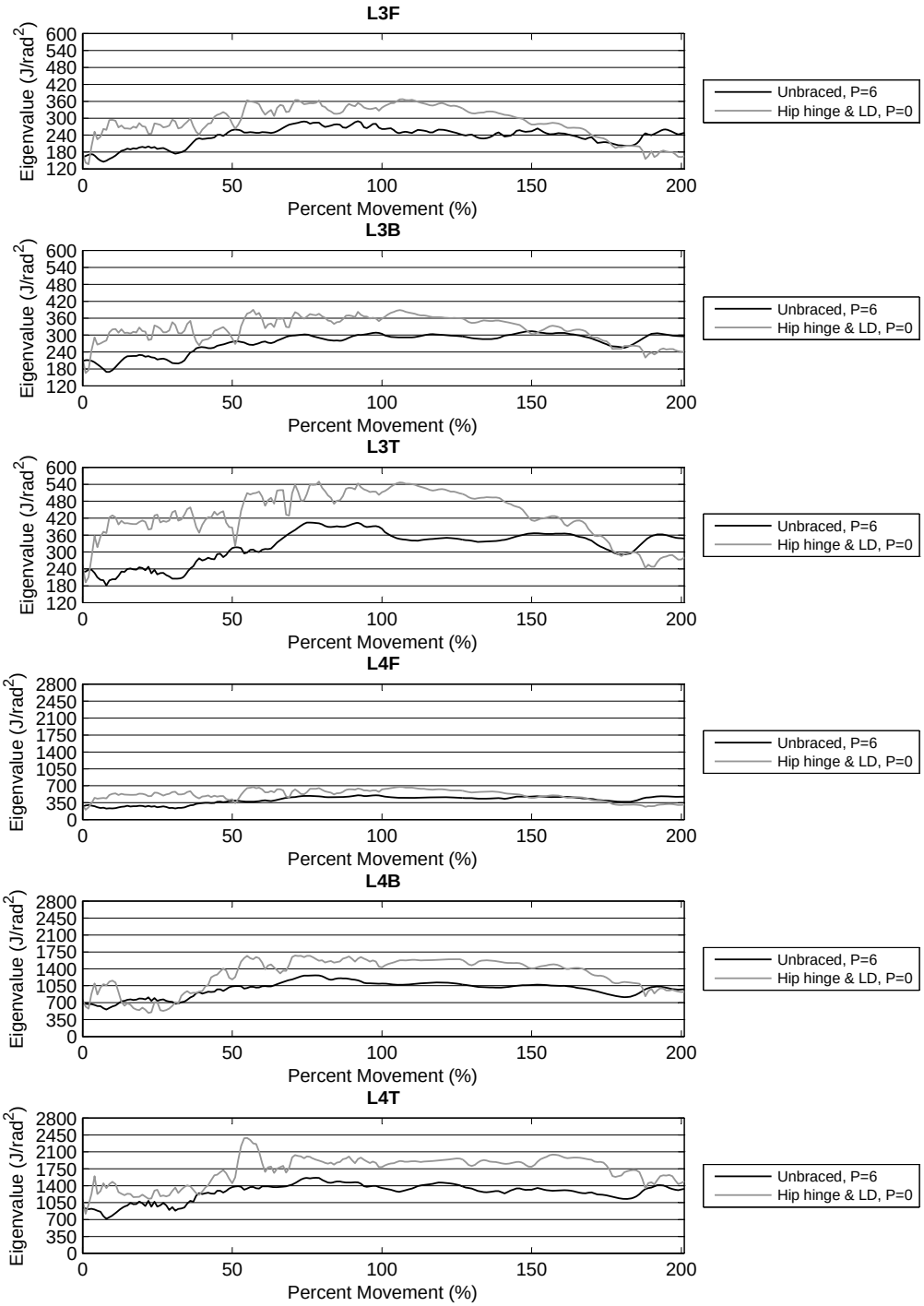
S03 Squat Back EMG

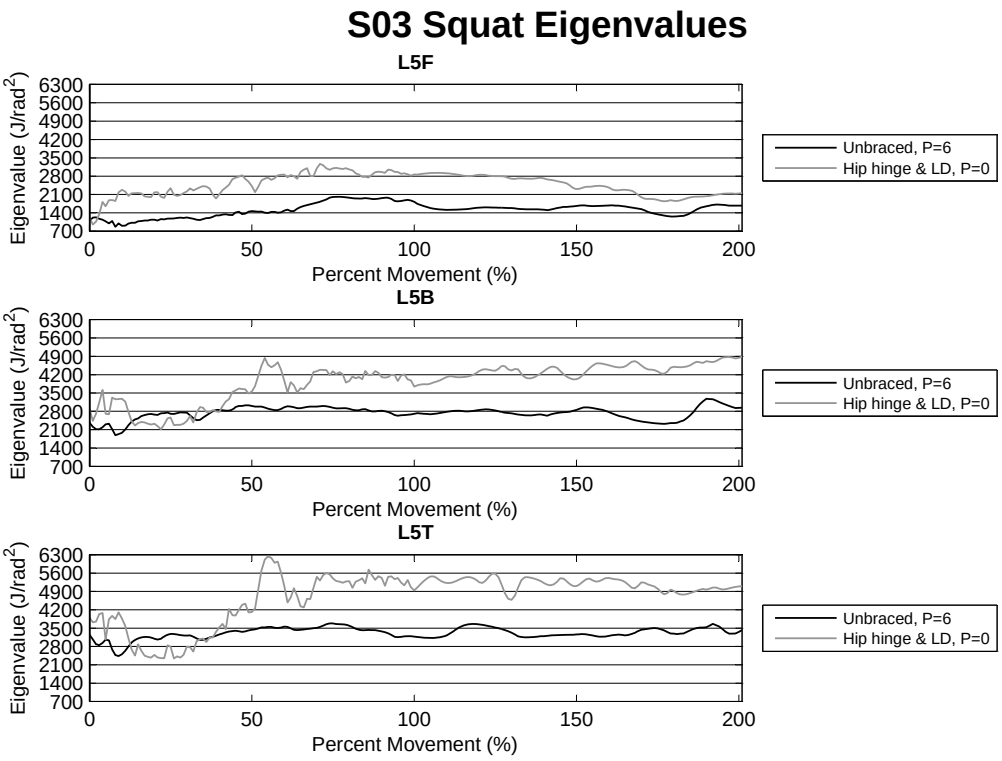


S03 Squat Eigenvalues

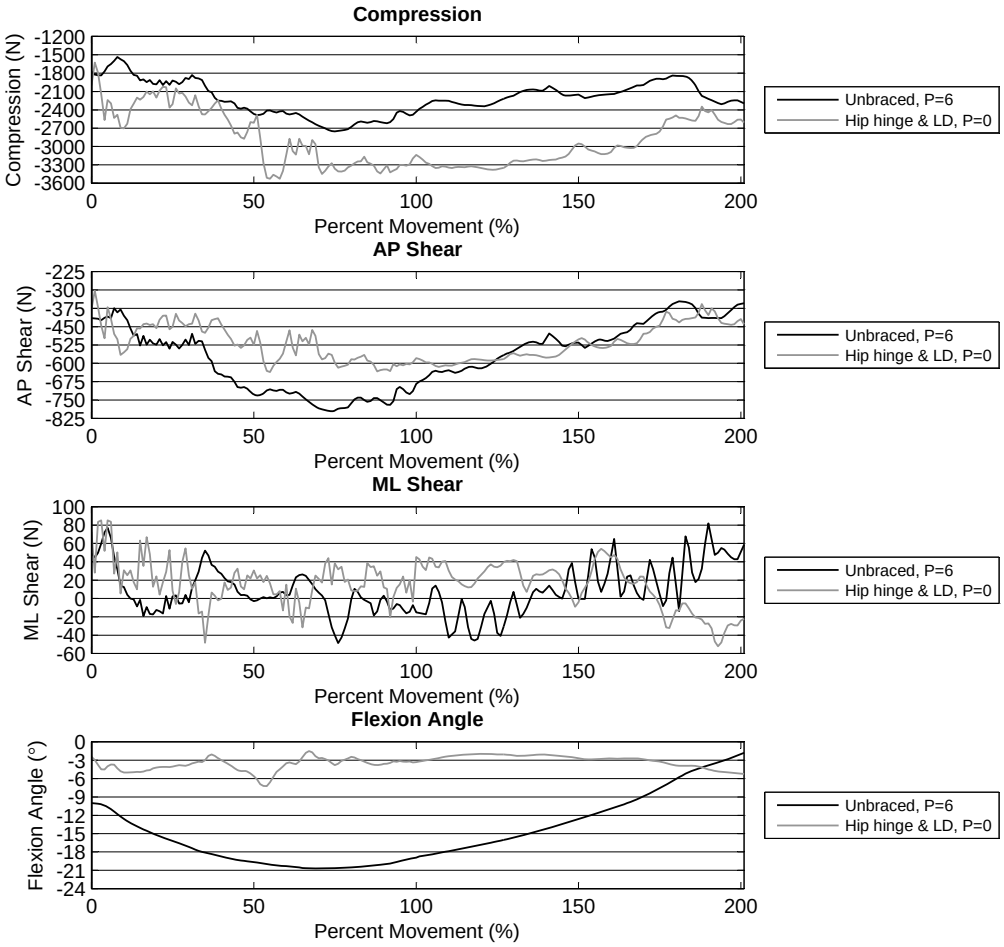


S03 Squat Eigenvalues

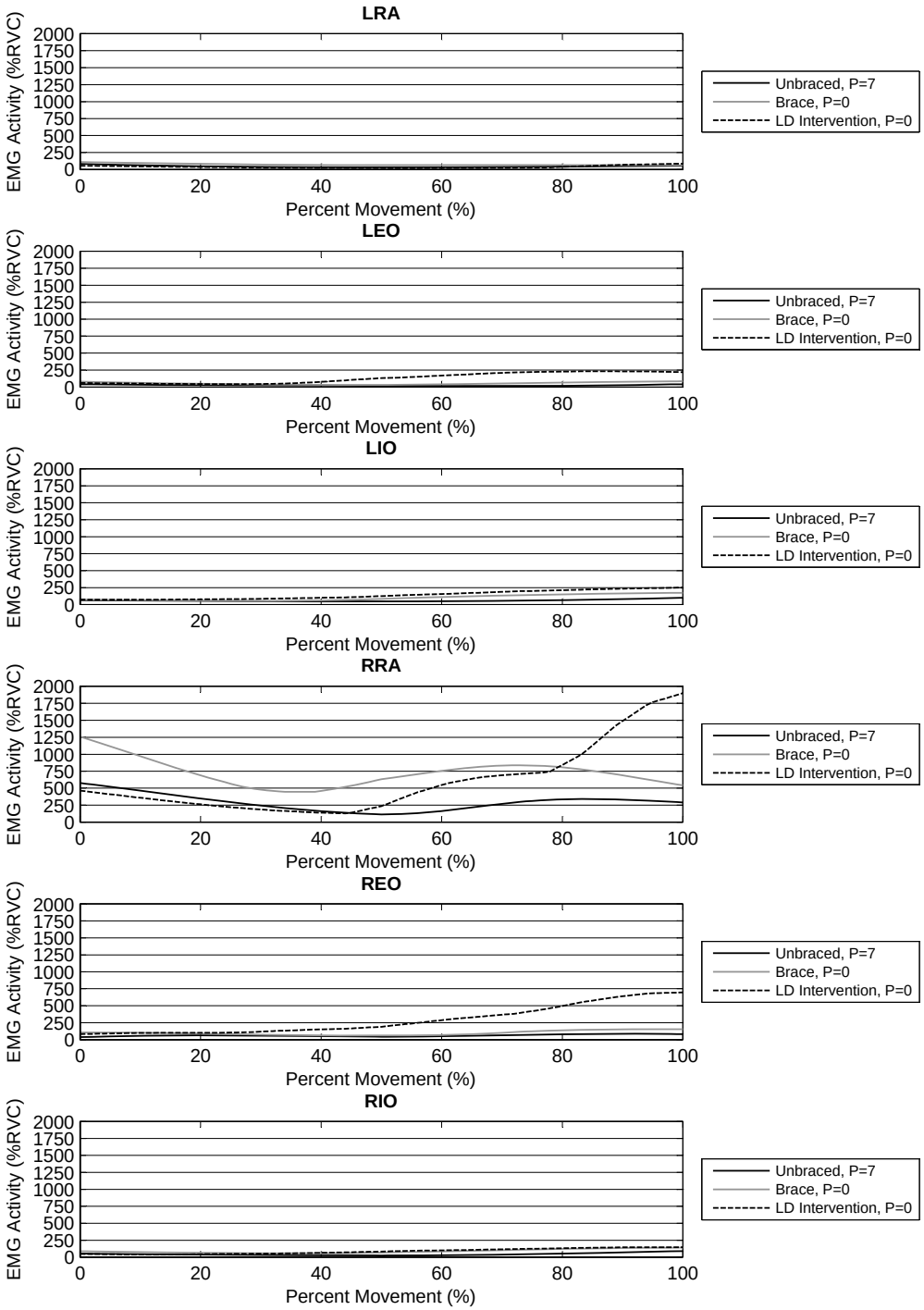



S03 Squat Eigenvalues
L5F
L5B
L5T
Eigenvalue (J/rad^2)
6300
5600
4900
4200
3500
2800
2100
1400
700
0
50
100
150
200
Percent Movement (%)
Unbraced, P=6
Hip hinge & LD, P=0

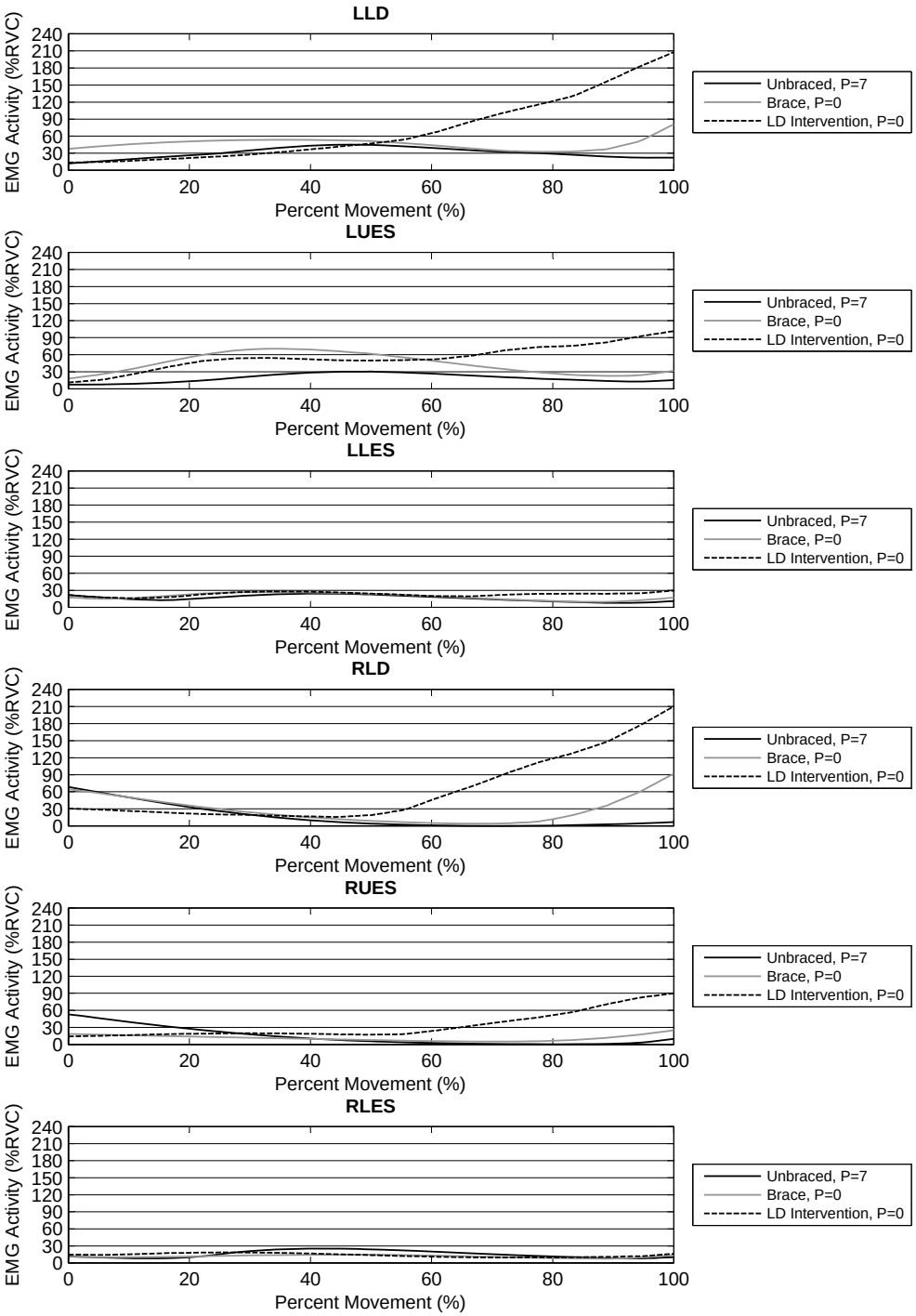
S03 Squat Compression, Shear & Flexion Angle



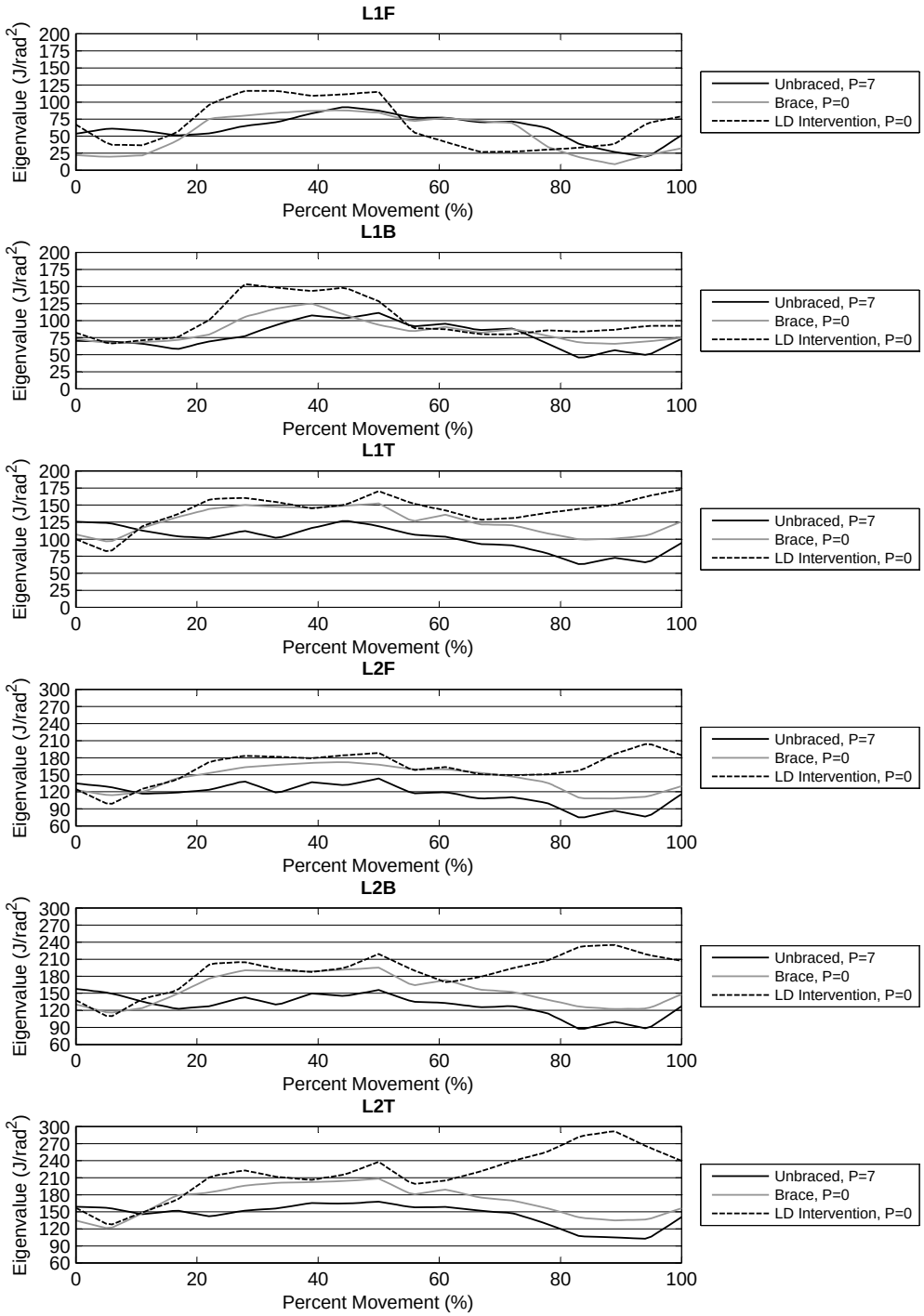
S03 Jump from Stool Abdominal EMG



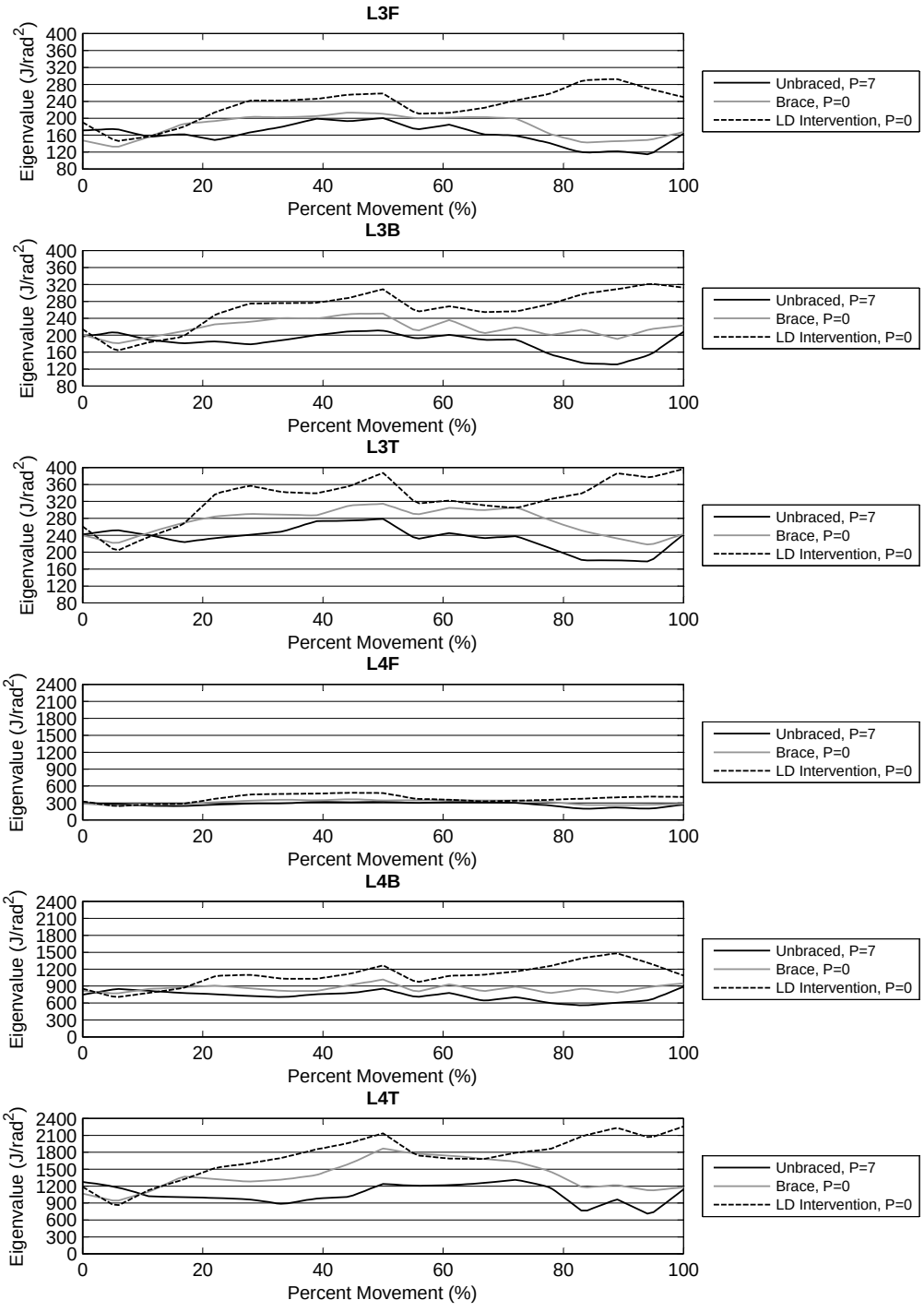
S03 Jump from Stool Back EMG



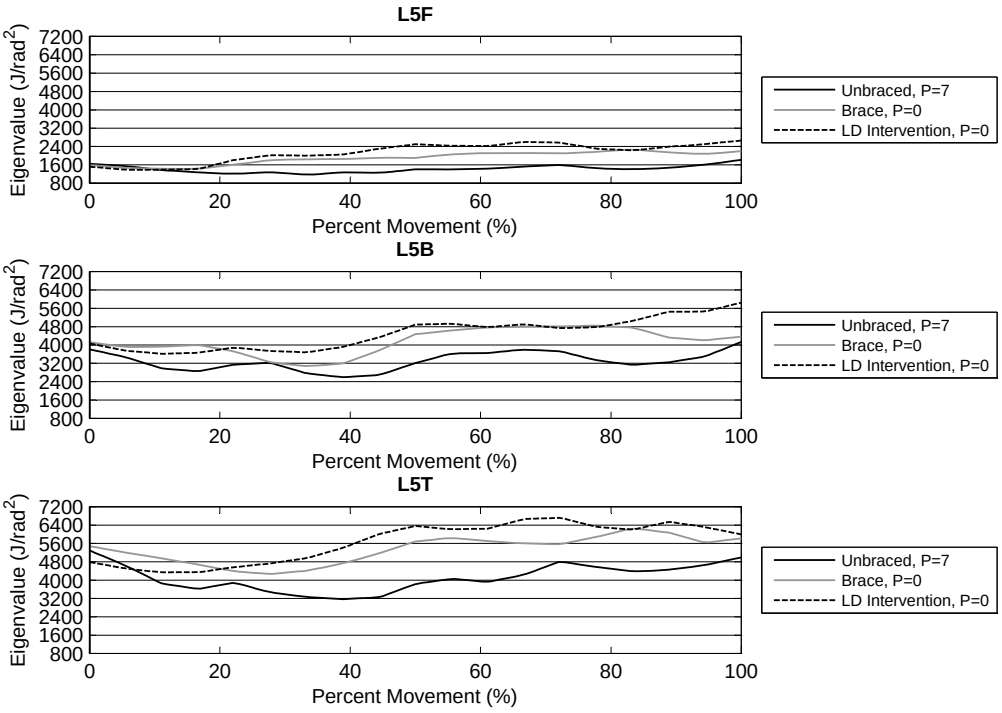
S03 Jump from Stool Eigenvalues



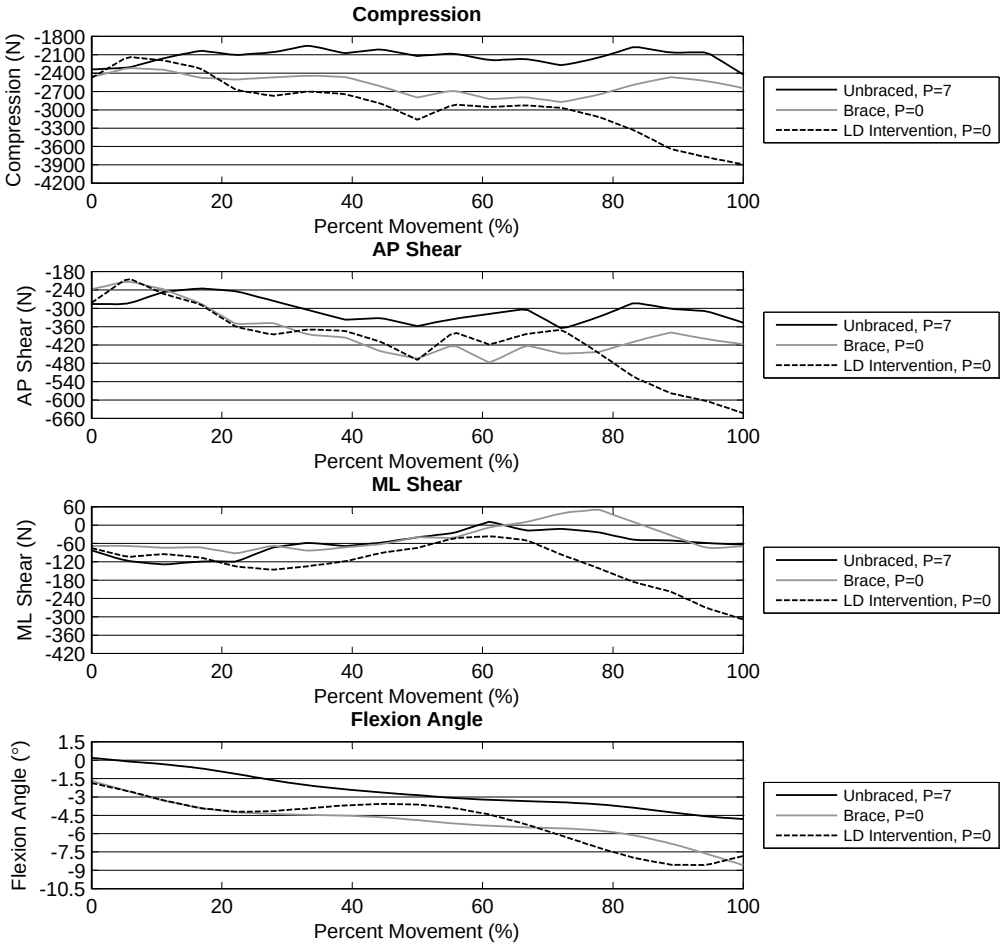
S03 Jump from Stool Eigenvalues



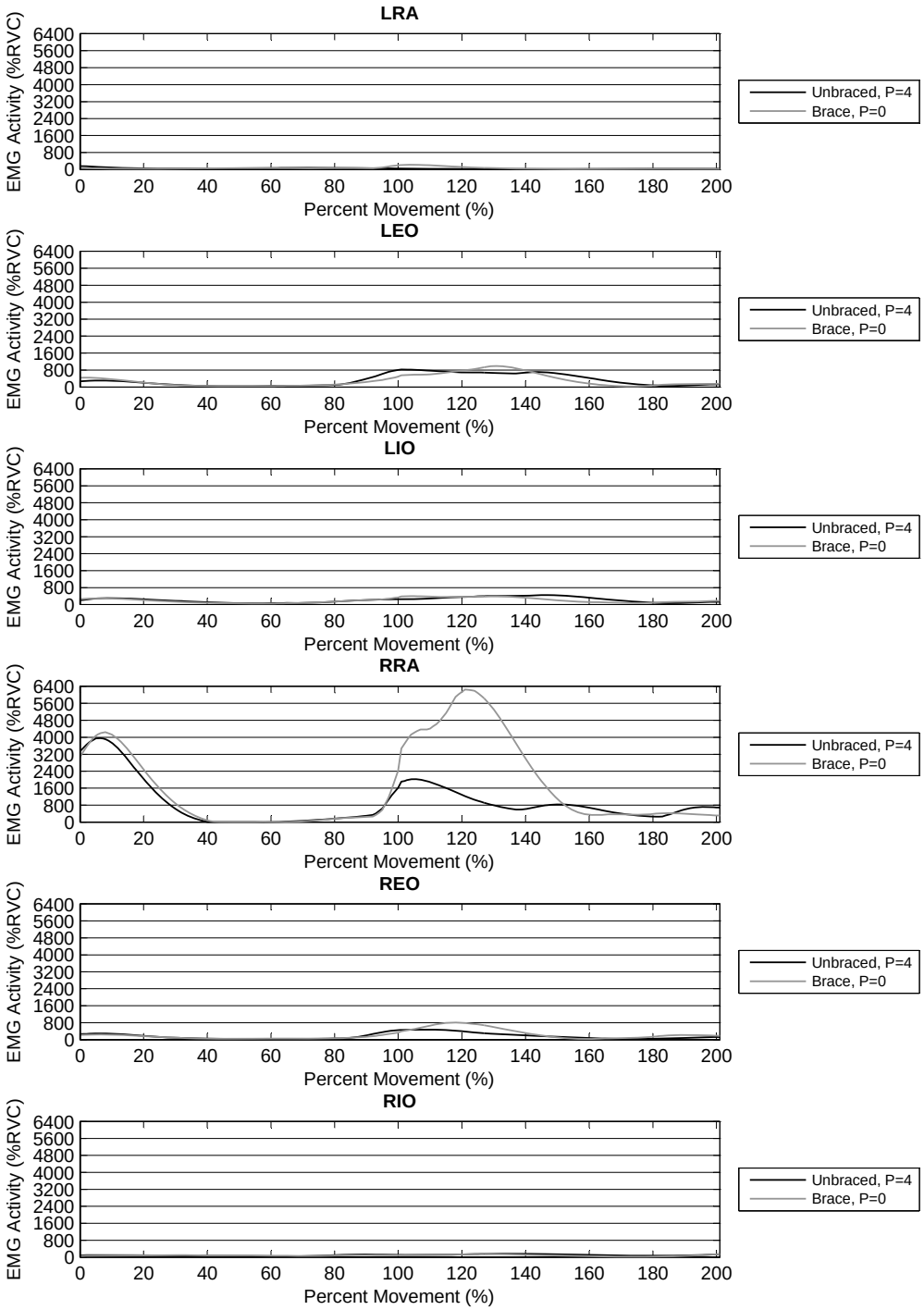
S03 Jump from Stool Eigenvalues



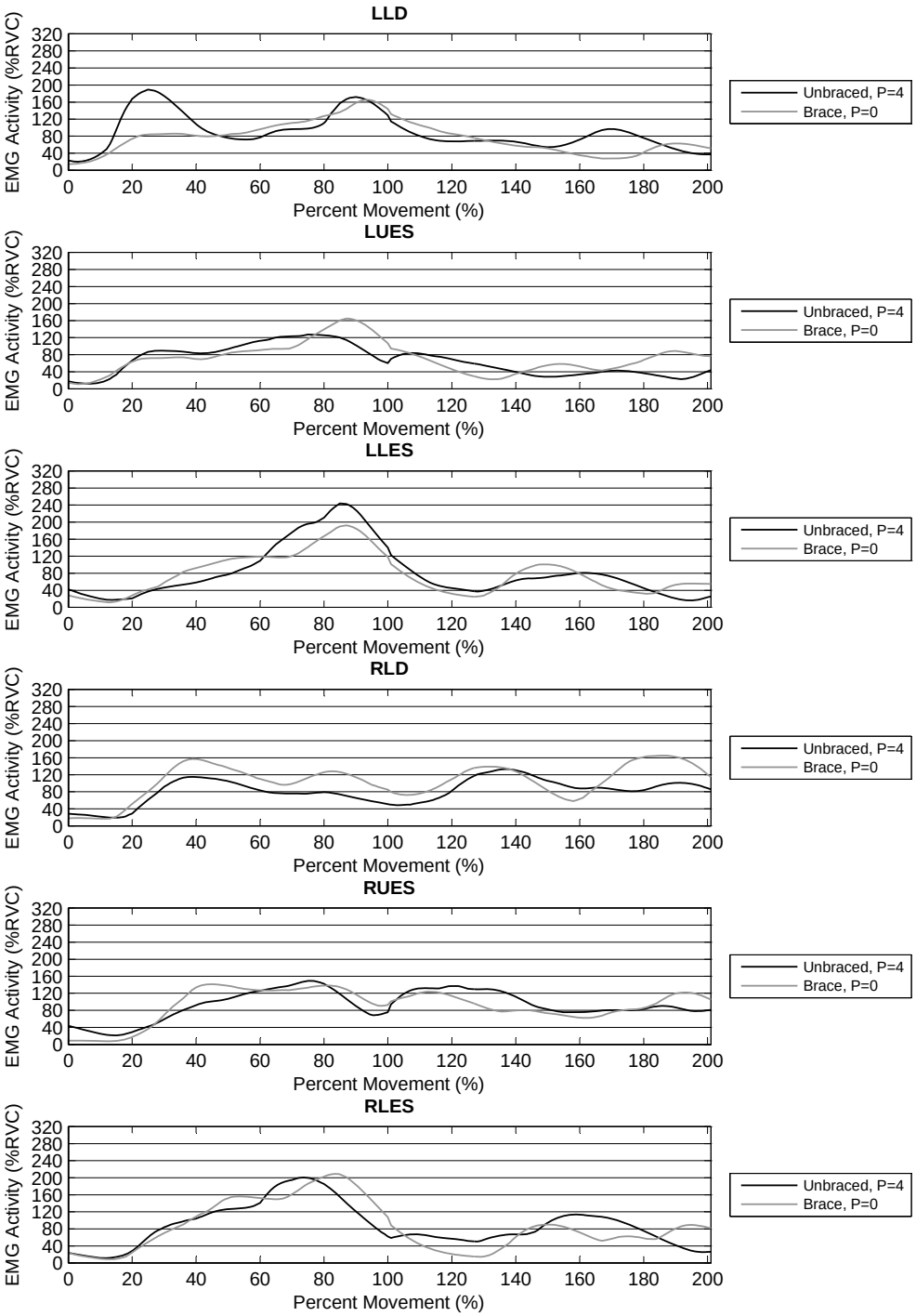
S03 Jump from Stool Compression, Shear & Flexion Angle



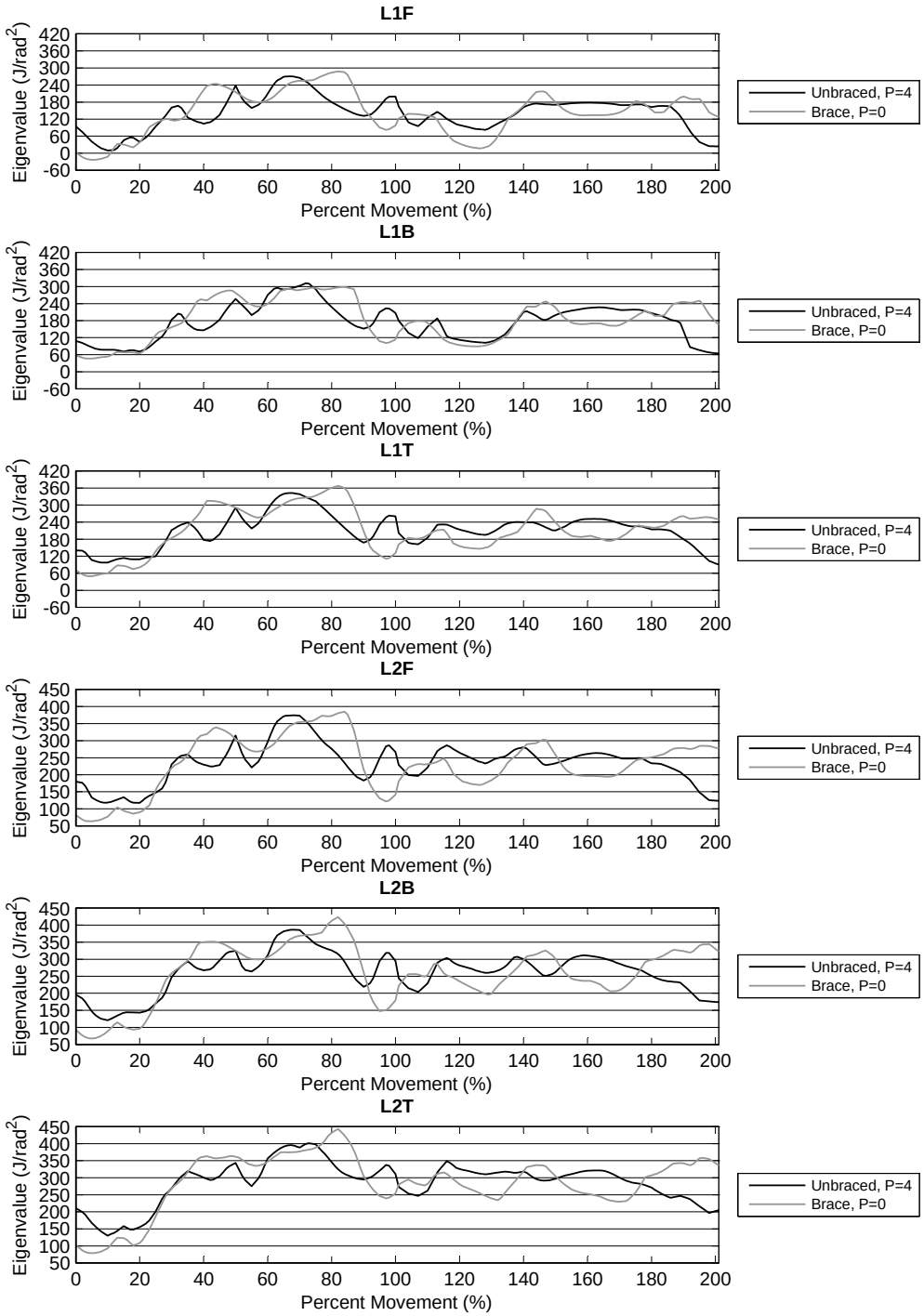
S03 One-step Spike Abdominal EMG



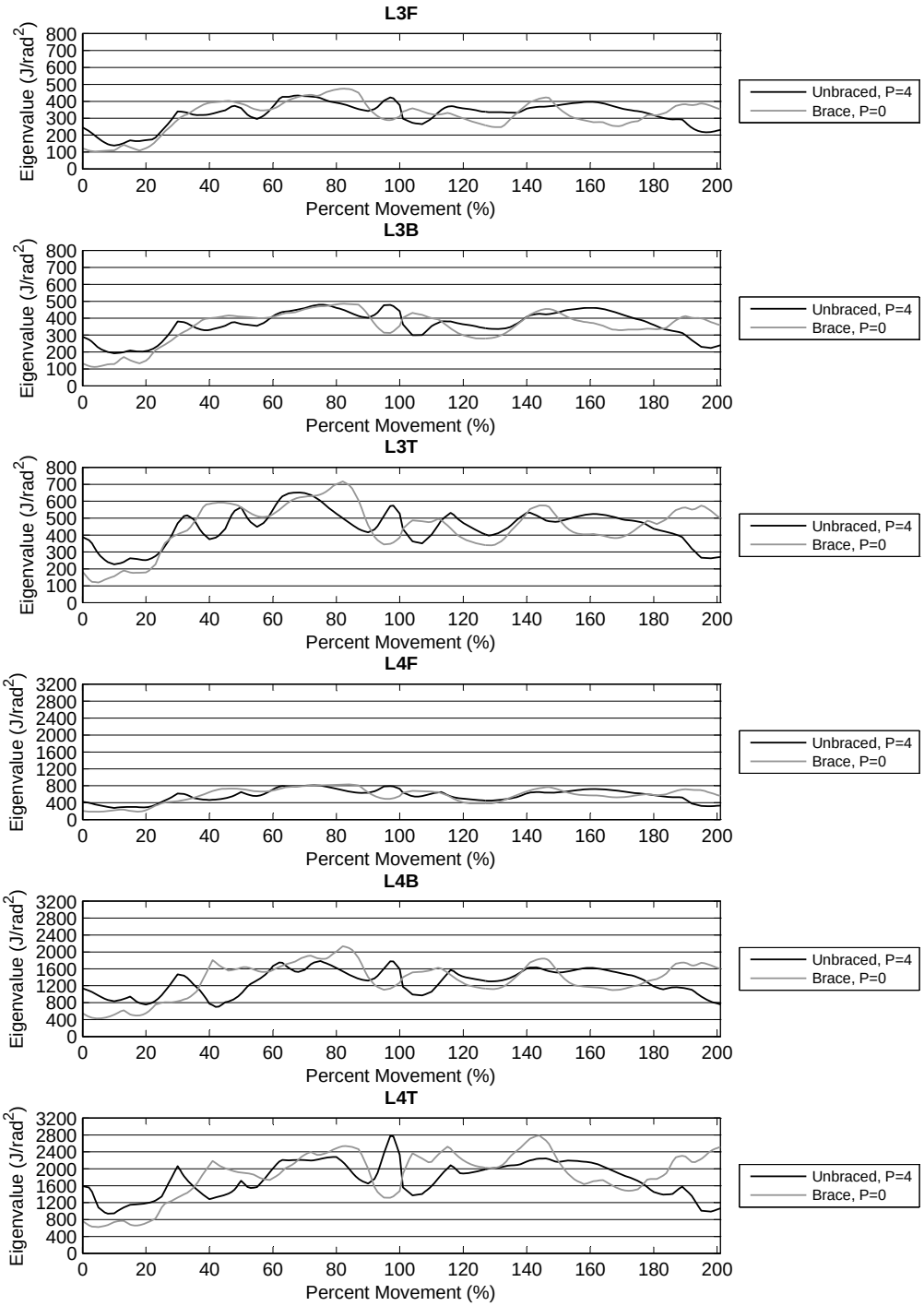
S03 One-step Spike Back EMG

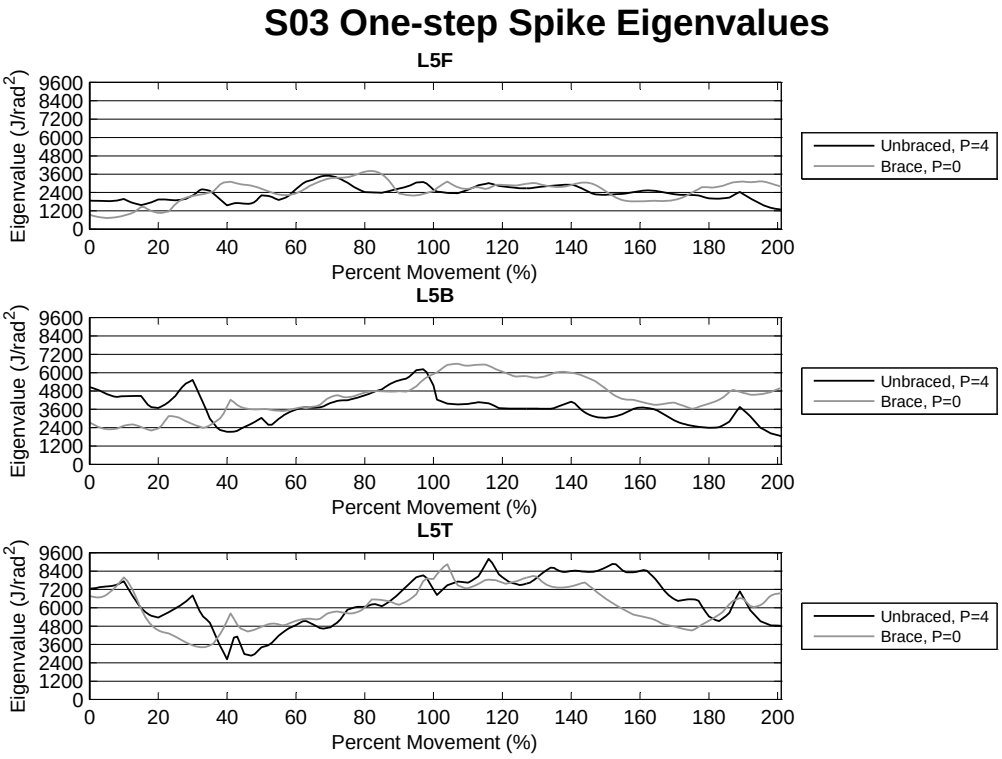


S03 One-step Spike Eigenvalues

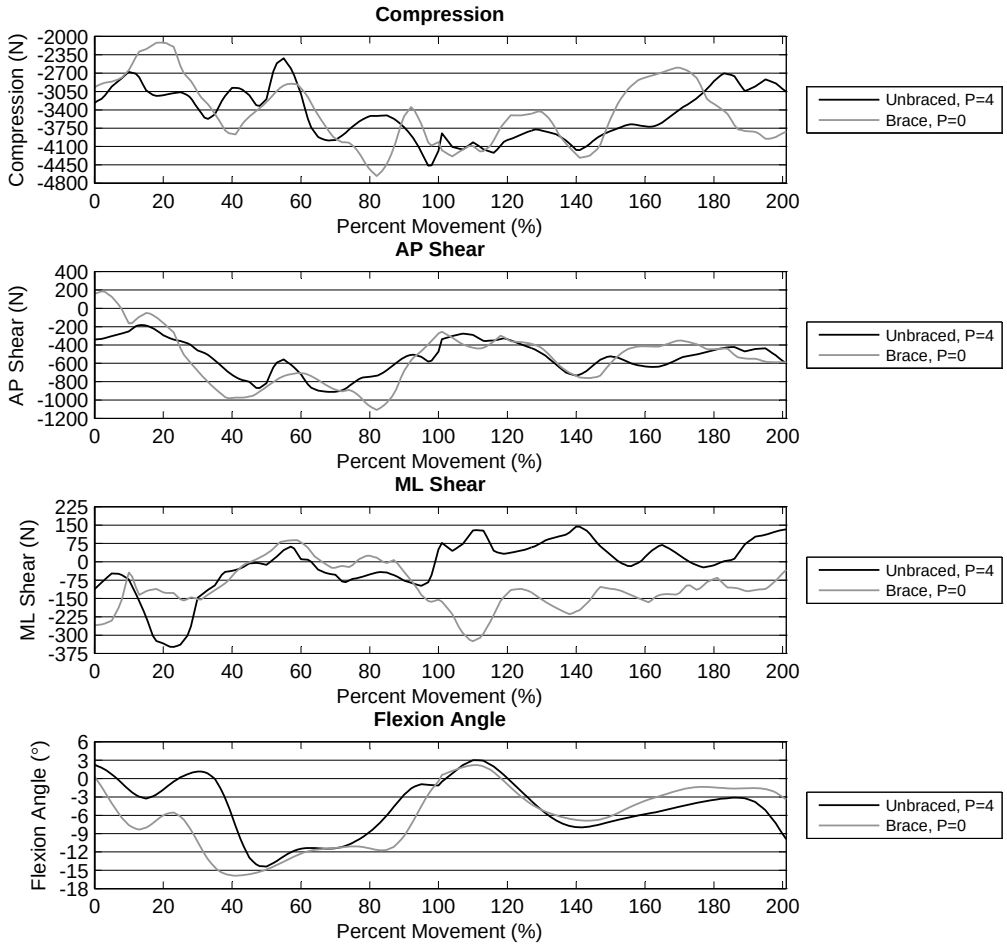


S03 One-step Spike Eigenvalues

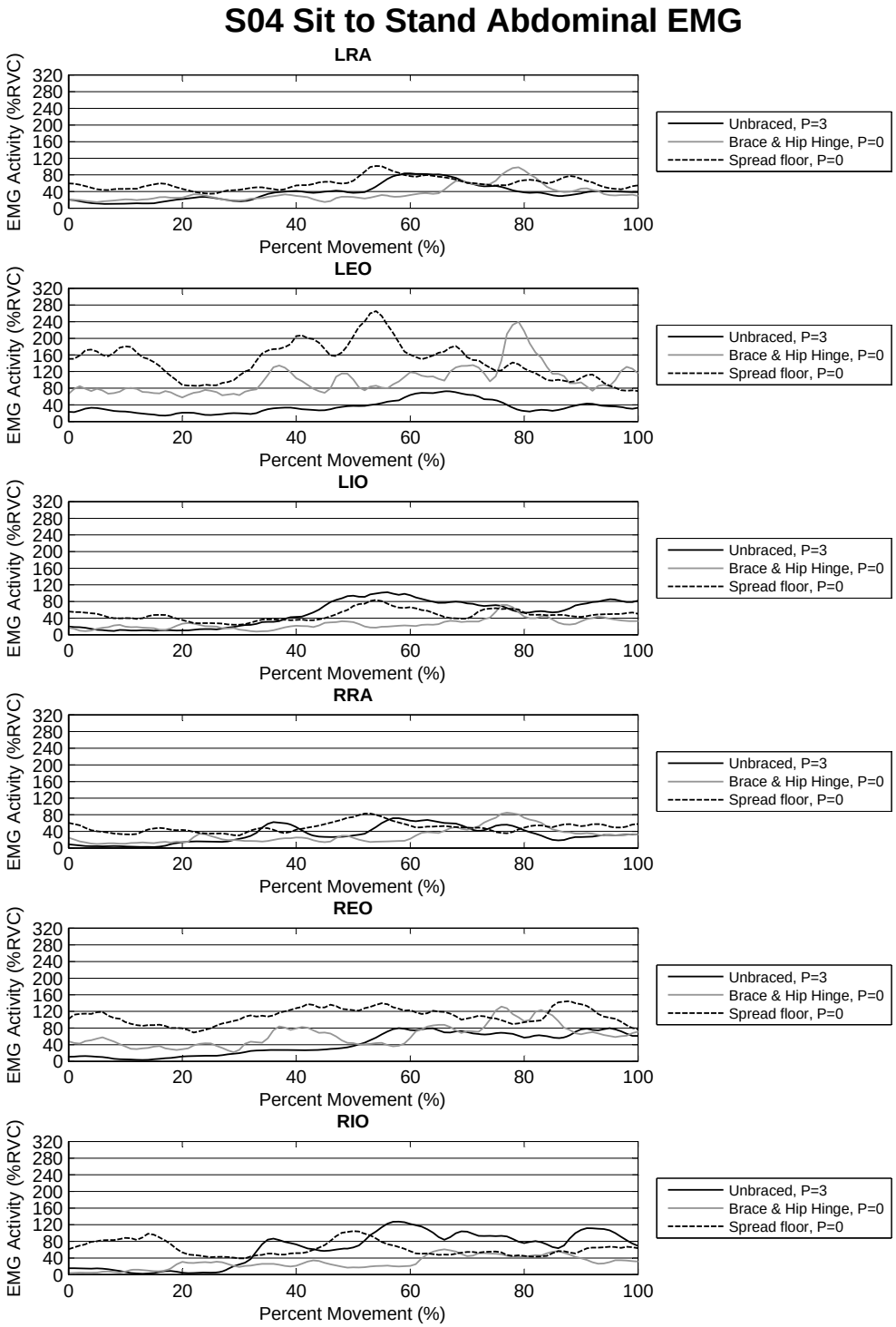



S03 One-step Spike Eigenvalues
L5F
L5B
L5T
Eigenvalue (J/rad2)
Percent Movement (%)
Unbraced, P=4
Brace, P=0
0
1200
2400
3600
4800
6000
7200
8400
9600
0
20
40
60
80
100
120
140
160
180
200

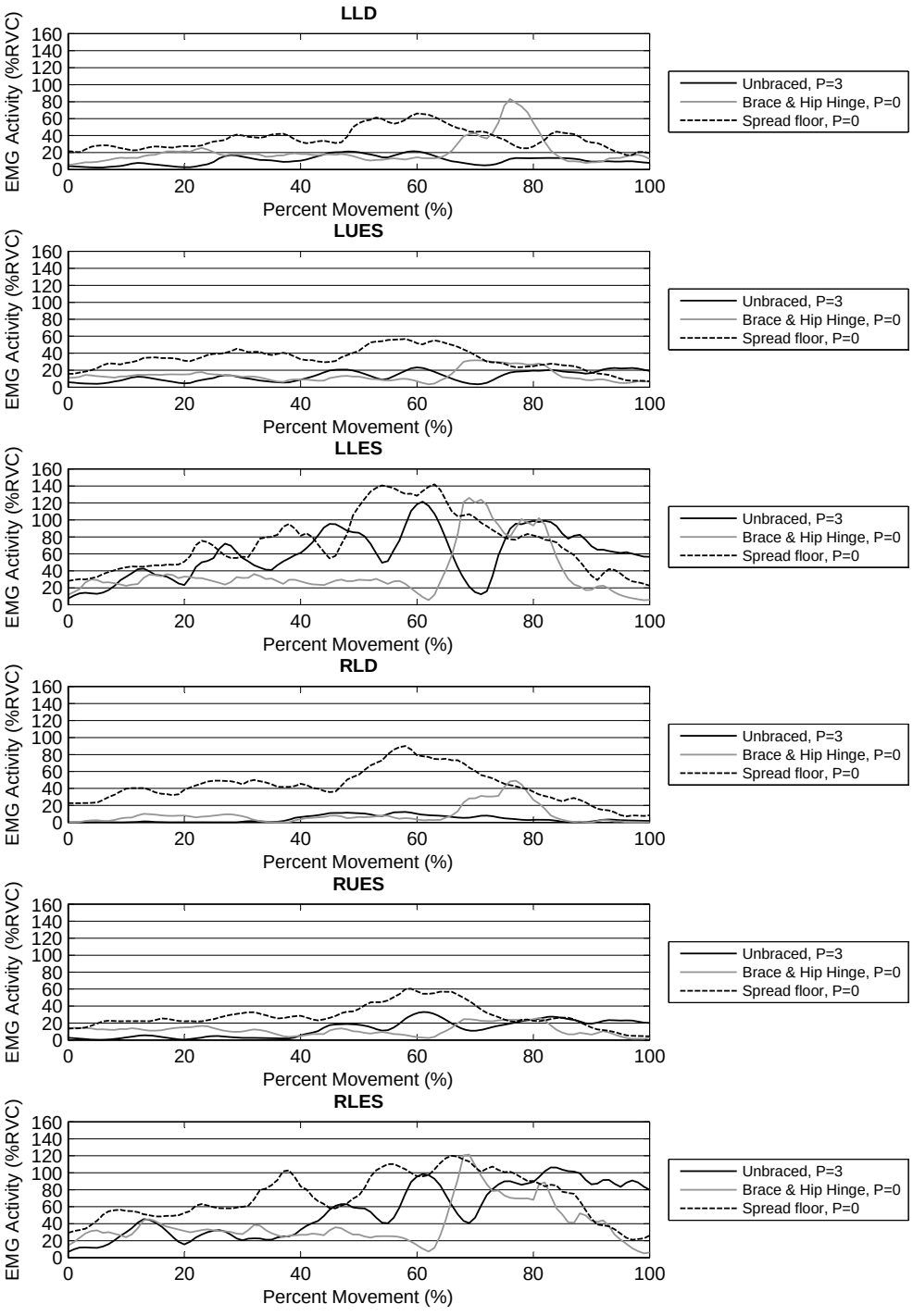
S03 One-step Spike Compression, Shear & Flexion Angle



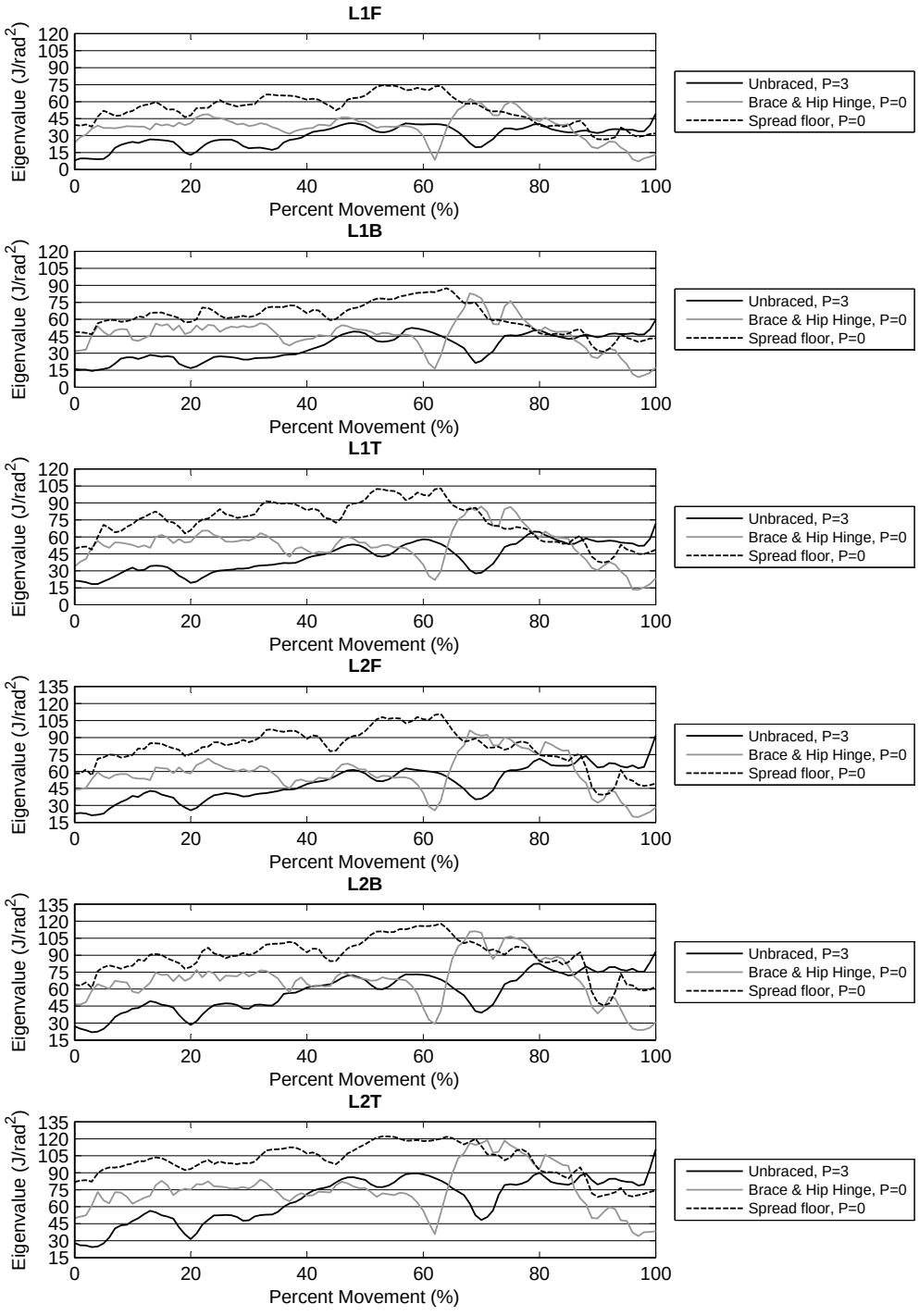
S04


S04 Sit to Stand Abdominal EMG
LRA
LEO
LIO
RRA
REO
RIO
EMG Activity (%RVC)
Percent Movement (%)
0
40
80
120
160
200
240
280
320
0
20
40
60
80
100
Unbraced, P=3
Brace & Hip Hinge, P=0
Spread floor, P=0

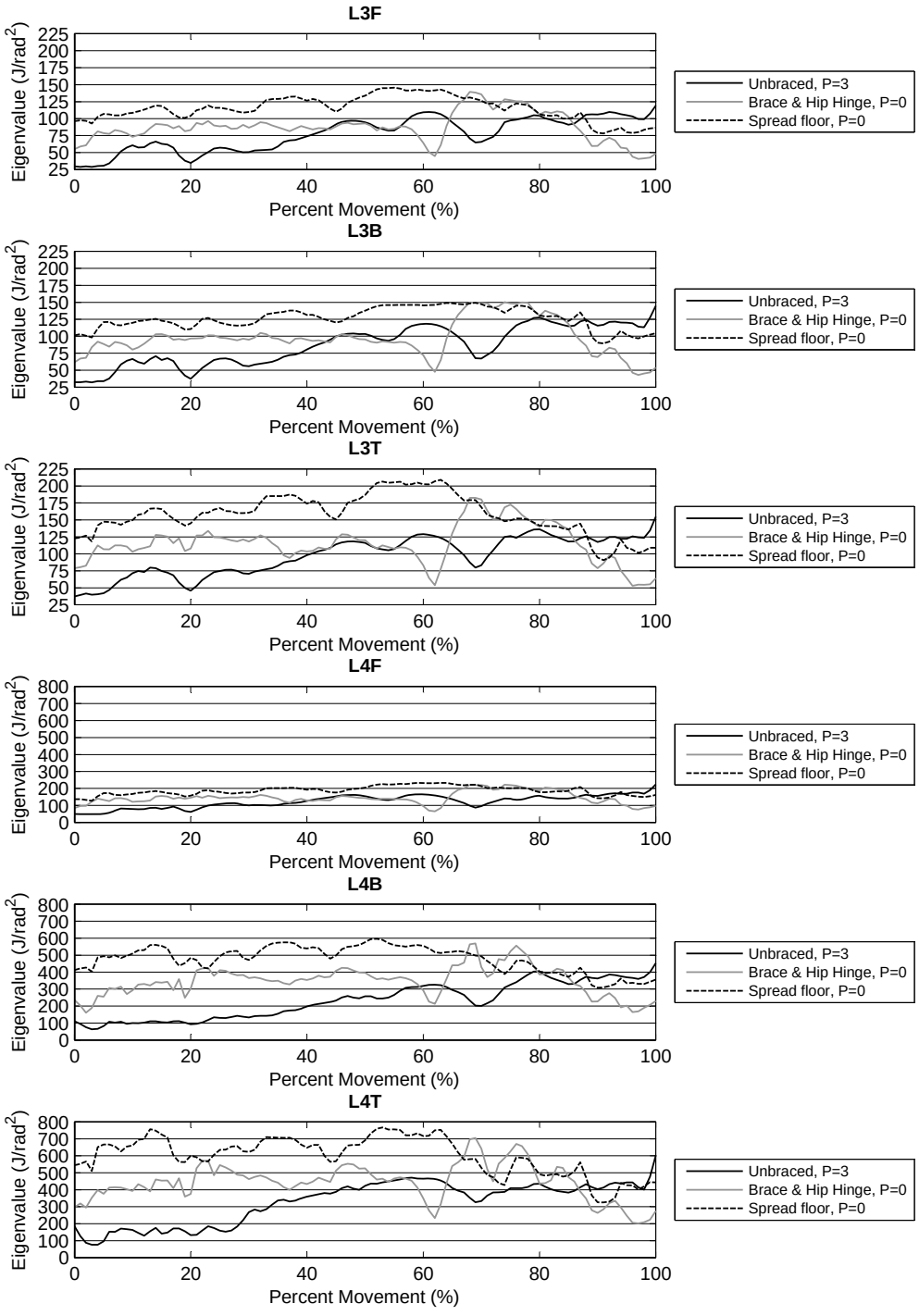
S04 Sit to Stand Back EMG



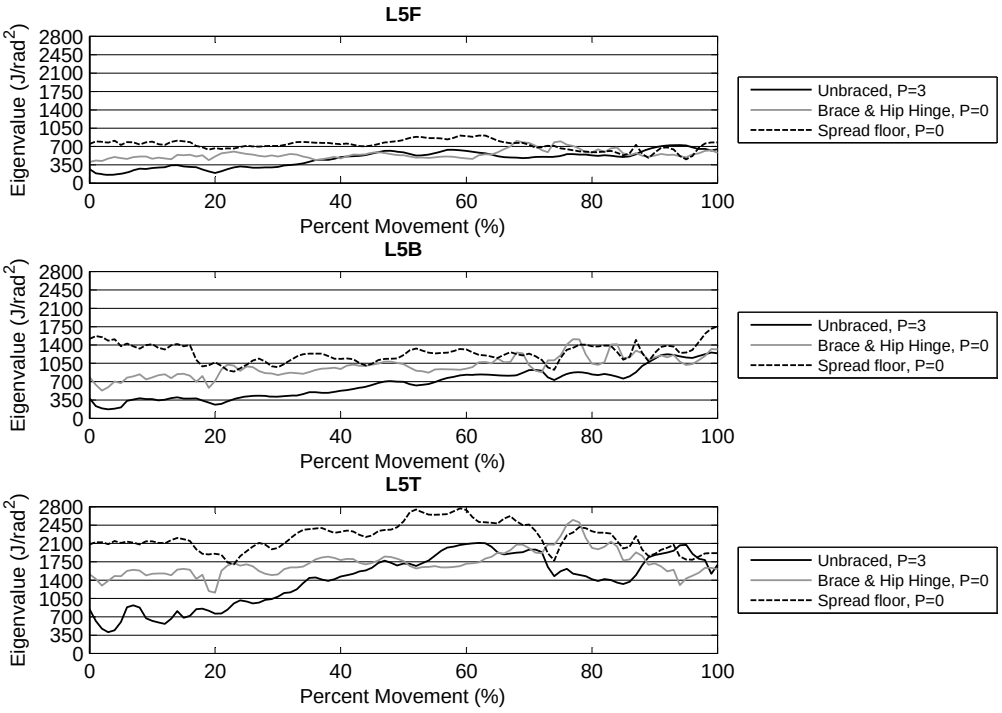
S04 Sit to Stand Eigenvalues



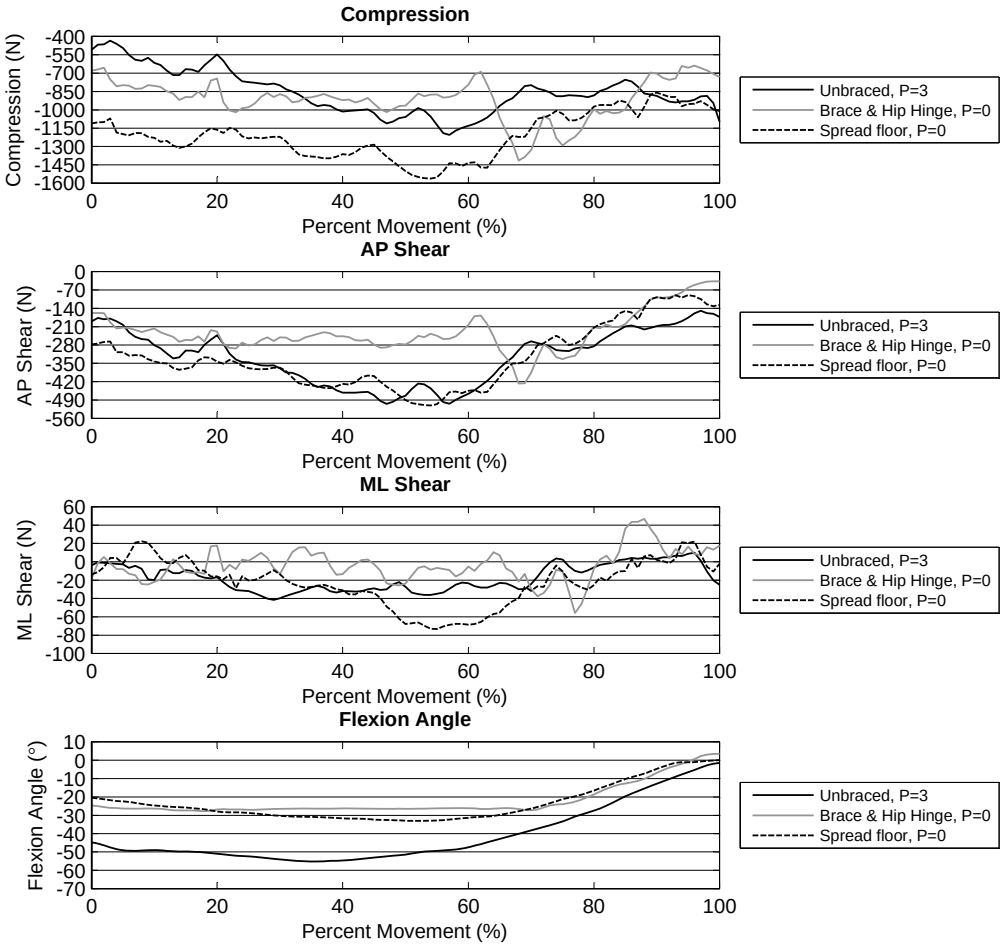
S04 Sit to Stand Eigenvalues



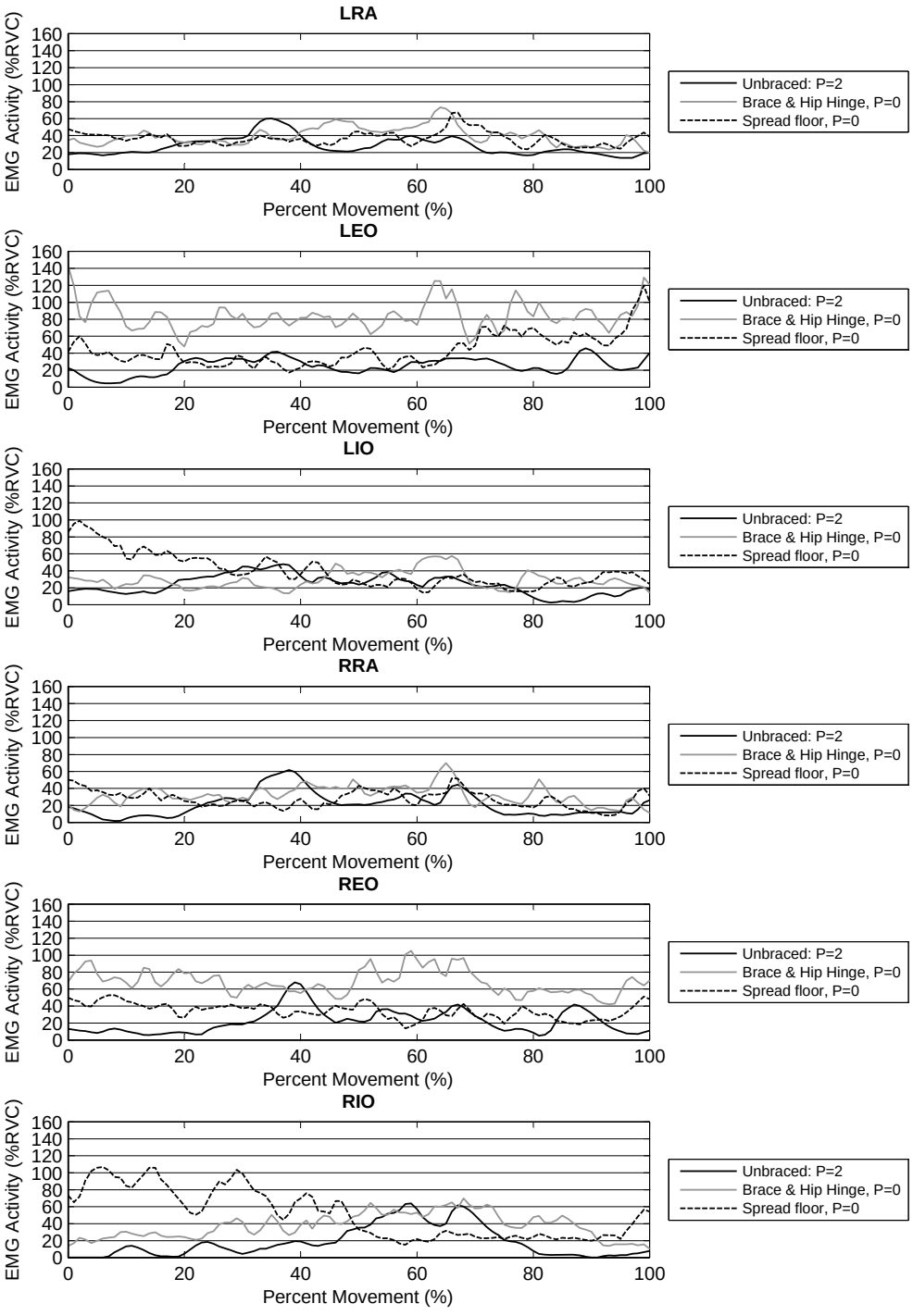
S04 Sit to Stand Eigenvalues



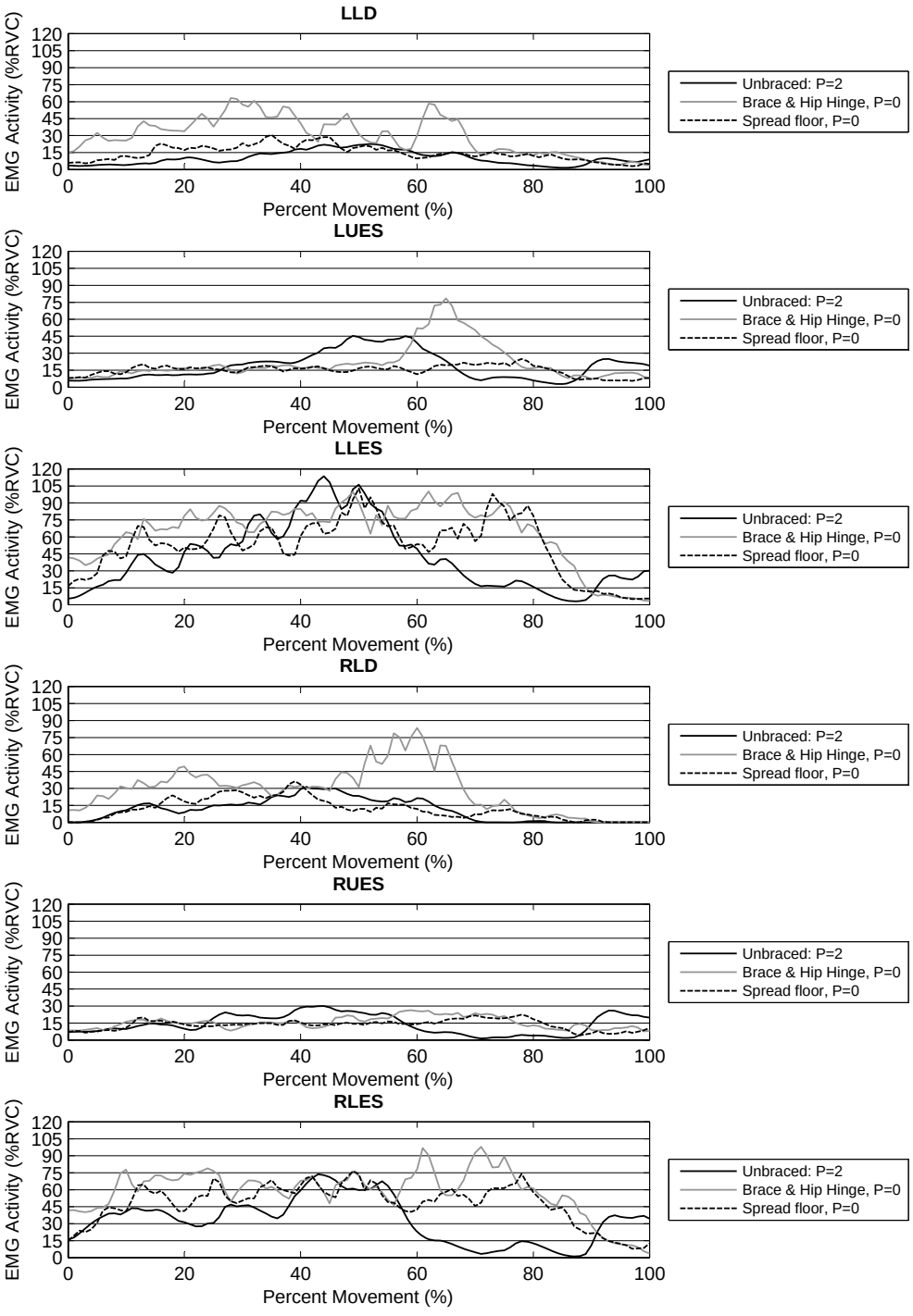
S04 Sit to Stand Compression, Shear & Flexion Angle



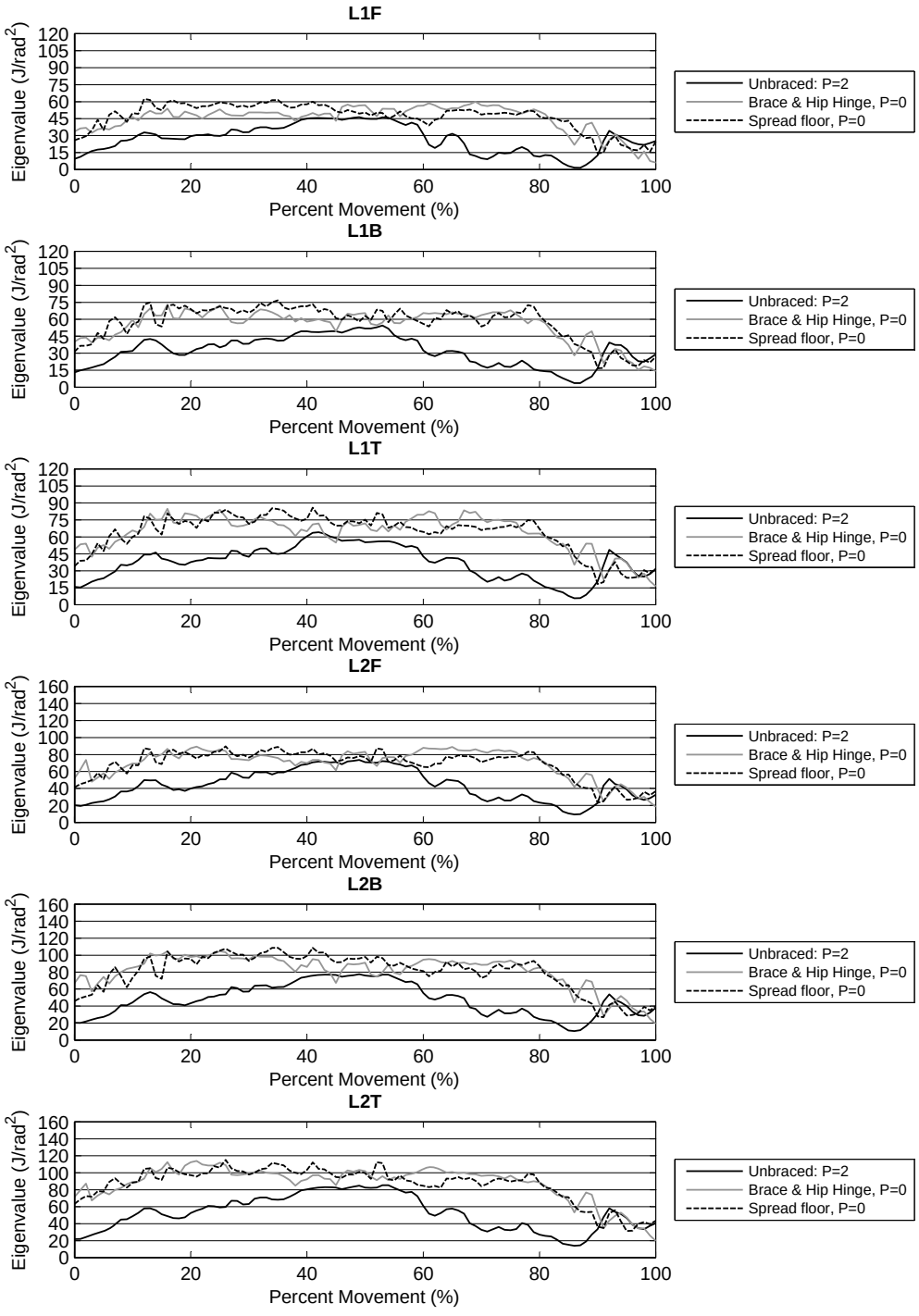
S04 Stand to Sit Abdominal EMG



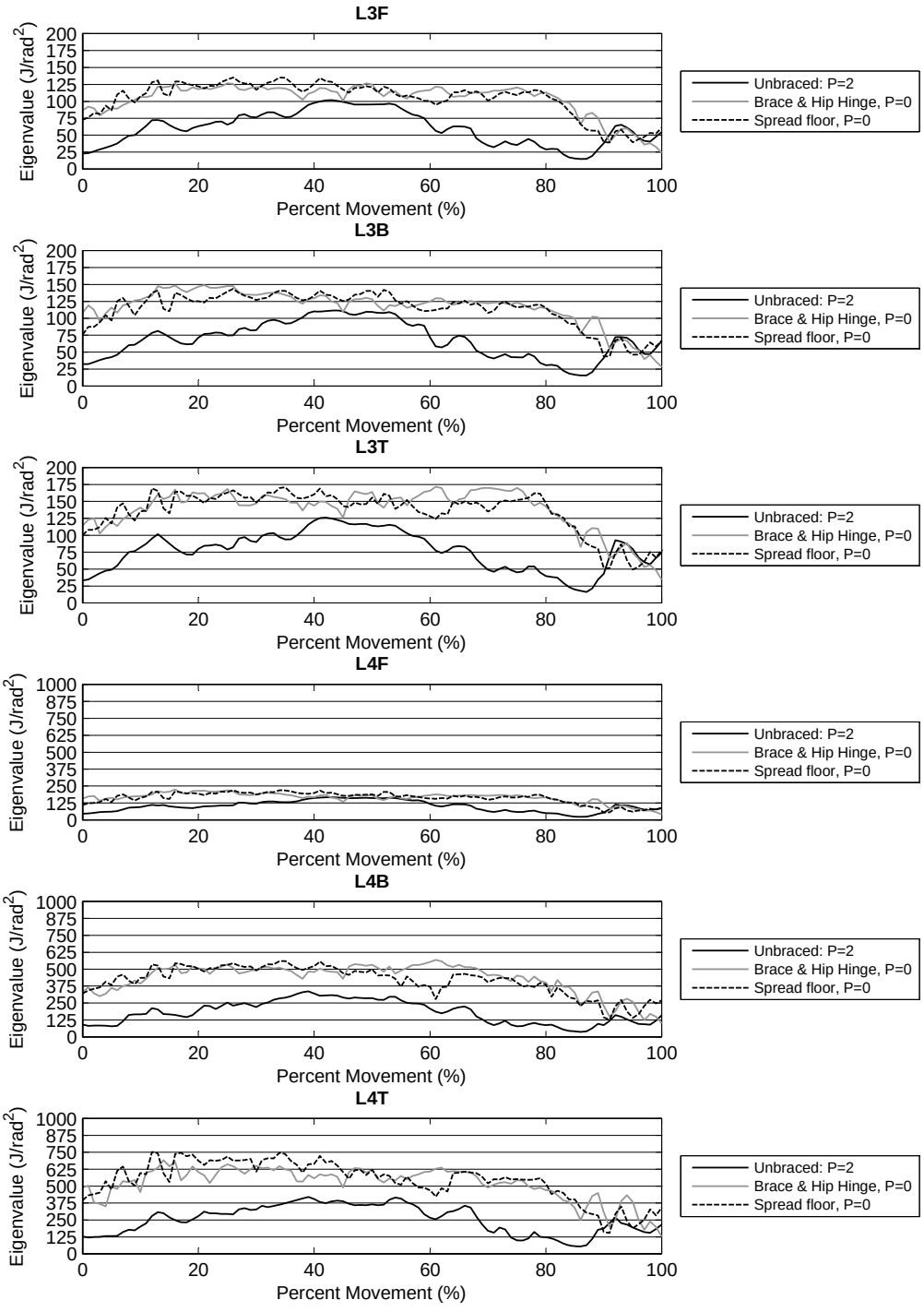
S04 Stand to Sit Back EMG



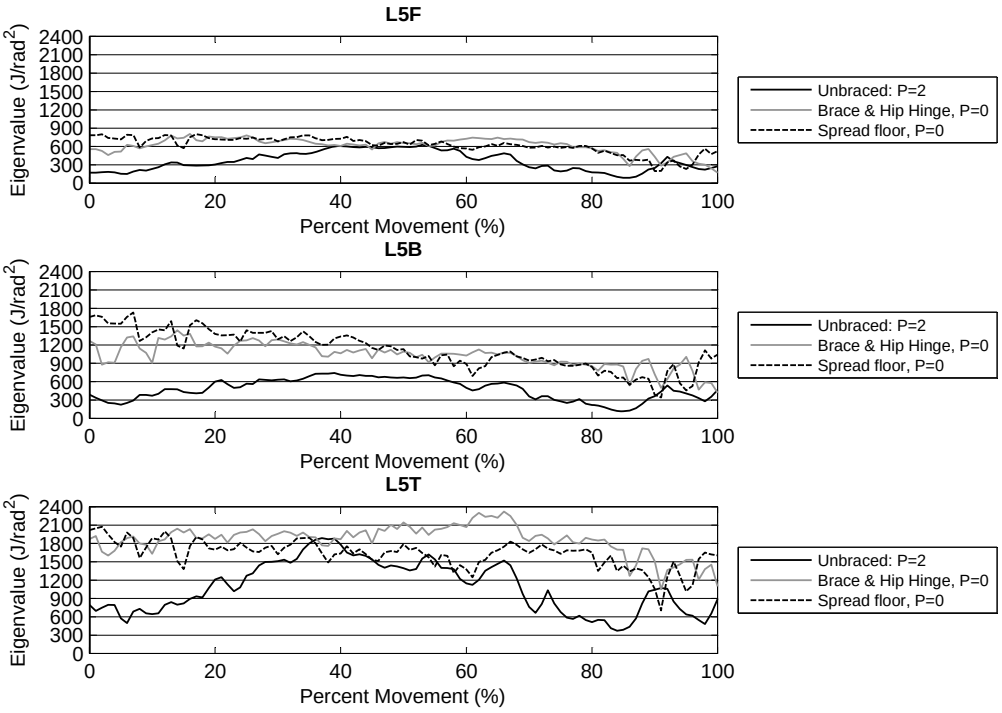
S04 Stand to Sit Eigenvalues



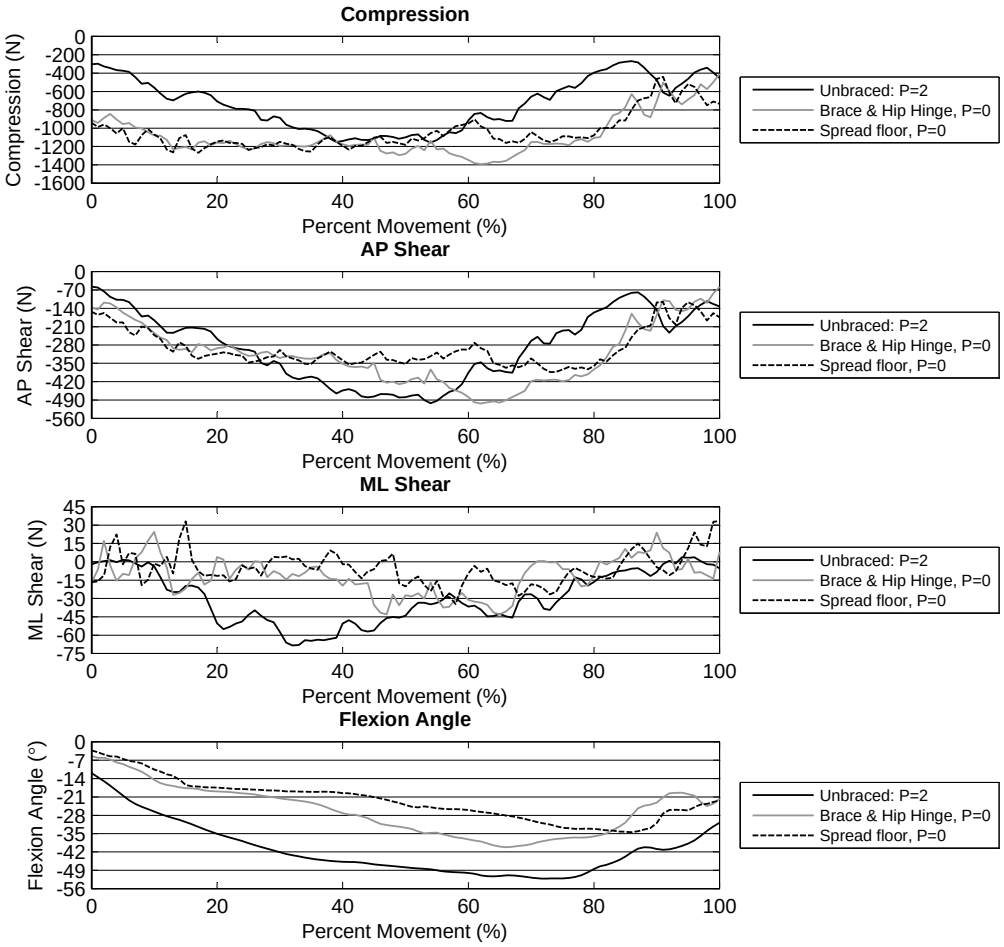
S04 Stand to Sit Eigenvalues



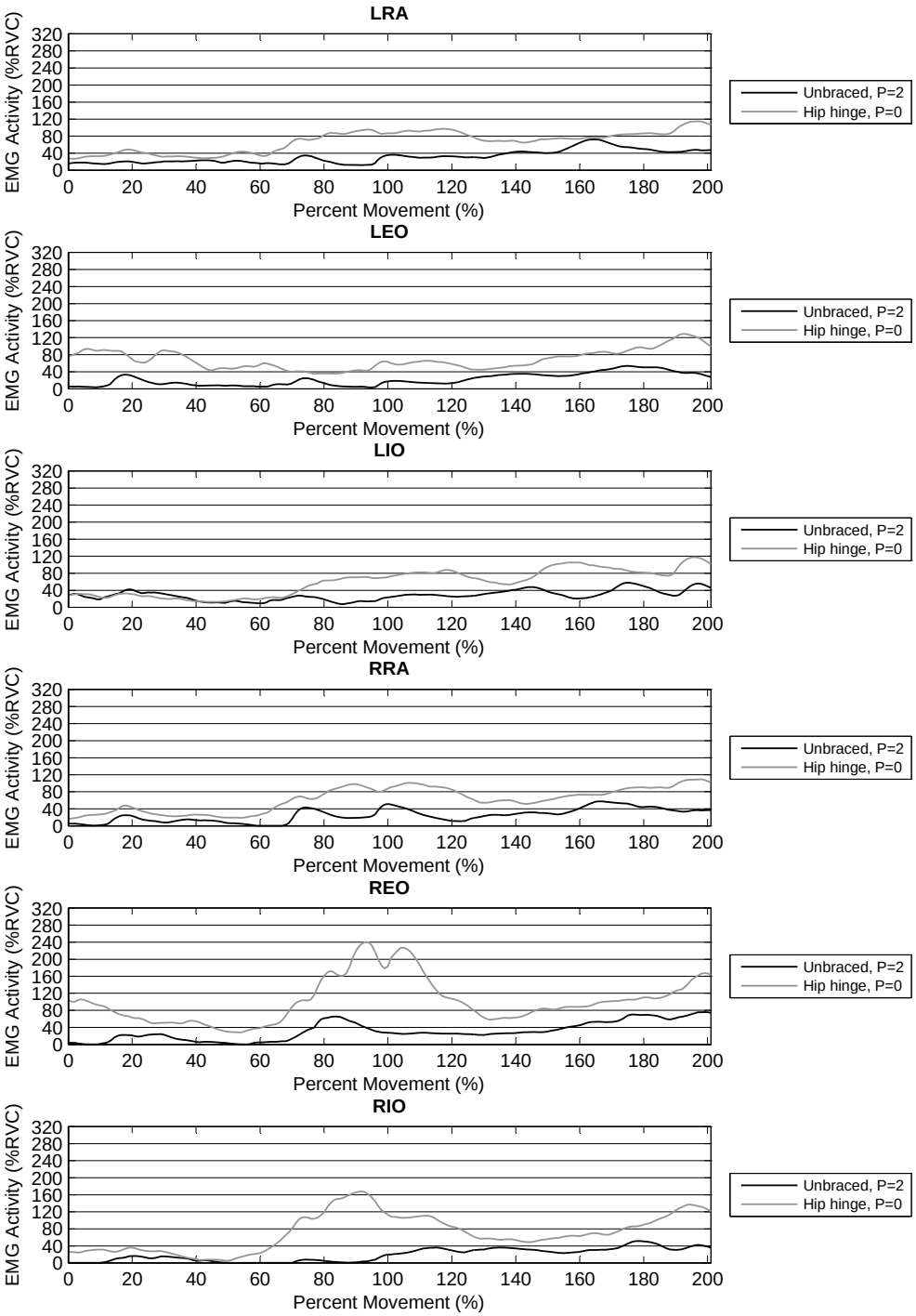
S04 Stand to Sit Eigenvalues



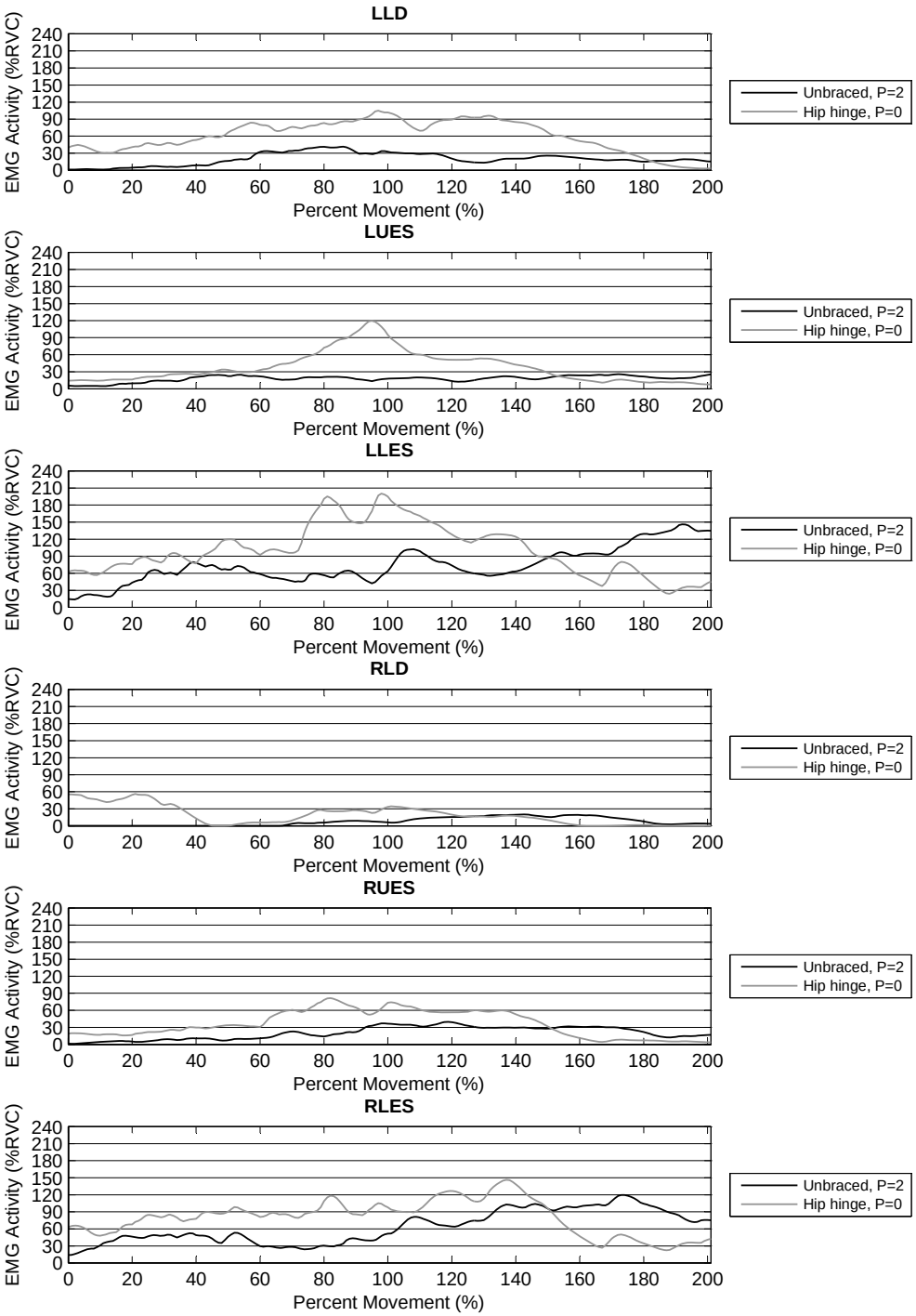
S04 Stand to Sit Compression, Shear & Flexion Angle



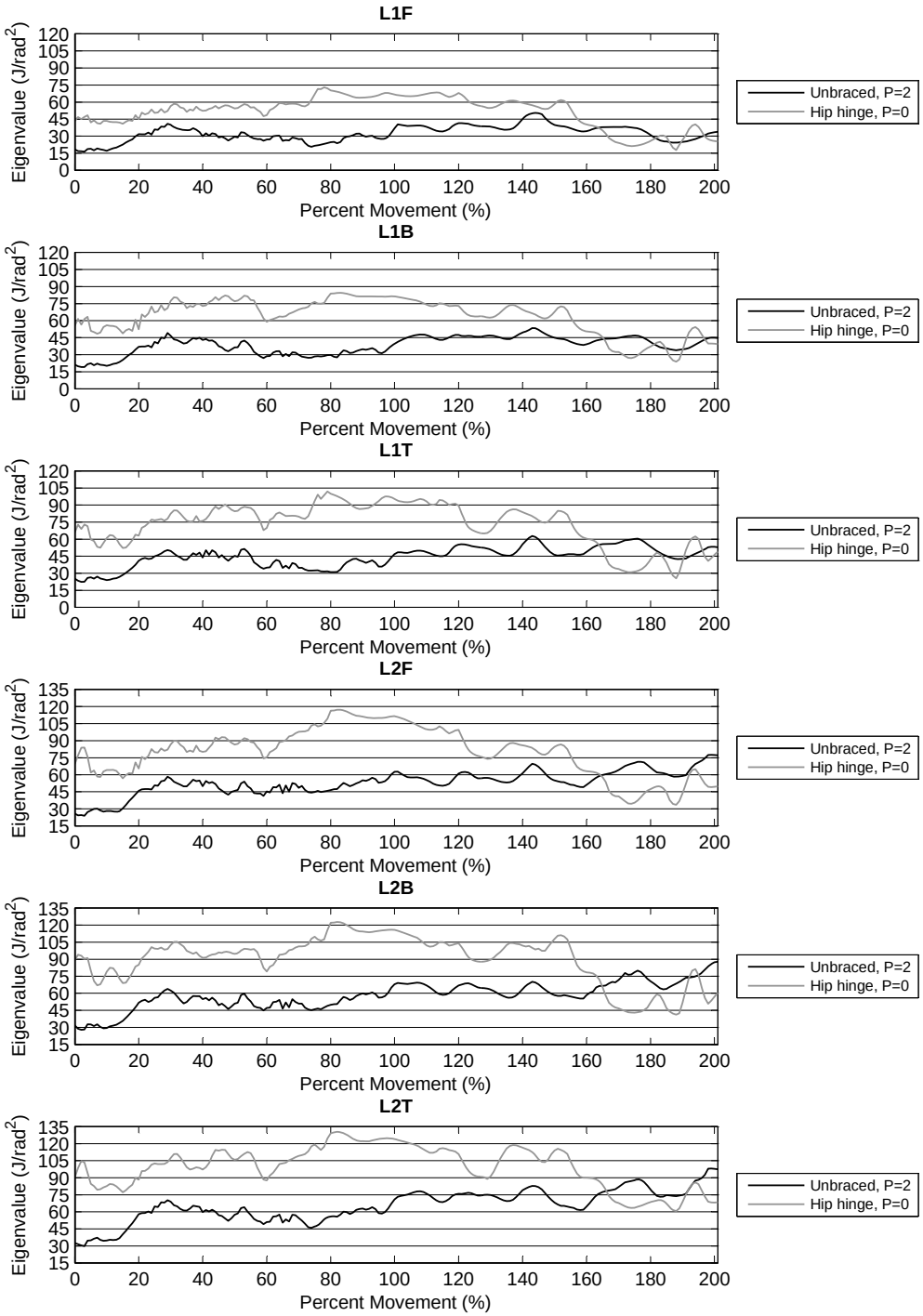
S04 Squat Abdominal EMG



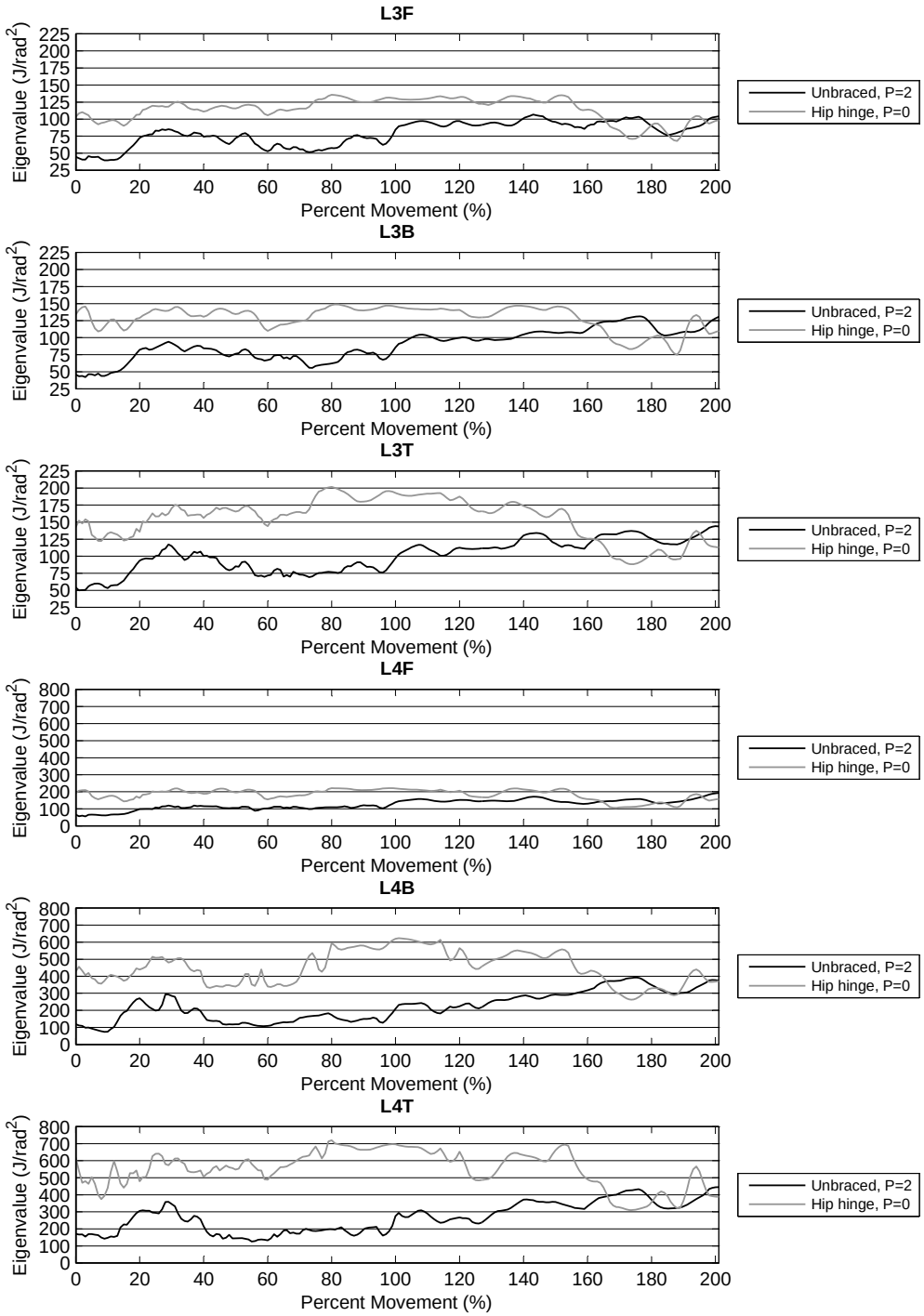
S04 Squat Back EMG

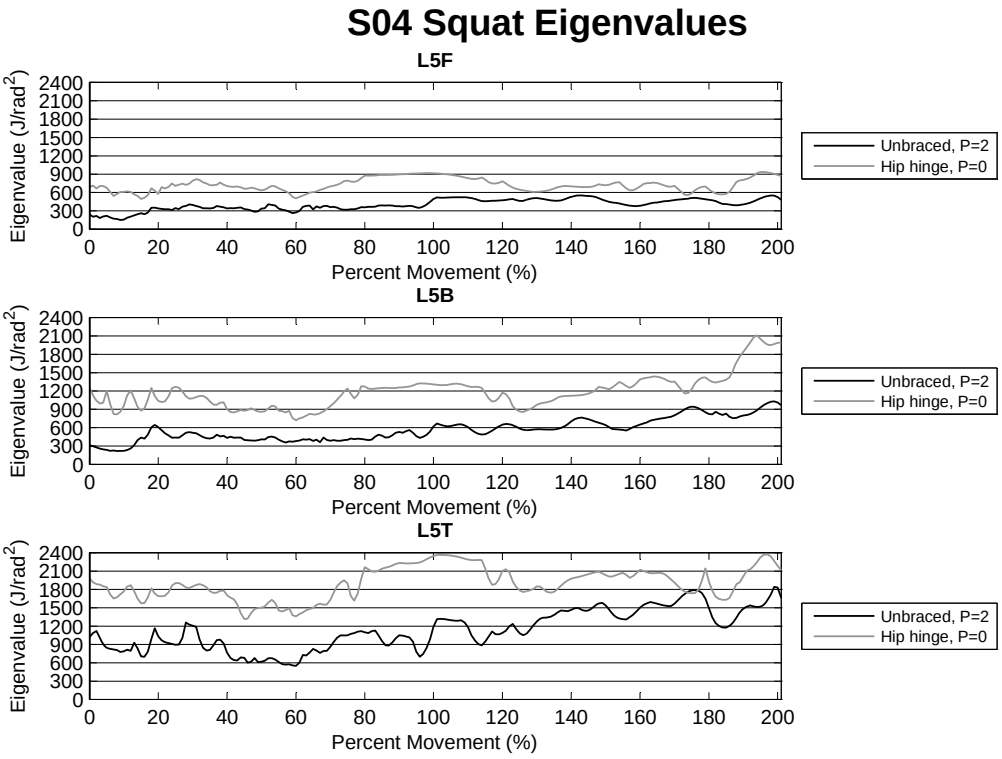


S04 Squat Eigenvalues



S04 Squat Eigenvalues




S04 Squat Eigenvalues
L5F
L5B
L5T
Eigenvalue (J/rad2)
Percent Movement (%)
Unbraced, P=2
Hip hinge, P=0

S04 Squat Compression, Shear & Flexion Angle